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Editorial

The year 2020 has been considered as the year zero. This is because all activities all over the world has stopped due to deadly corona virus pandemic. There is no activity which gas functioned normally. Health care activities have been more severely affected. Doctors (whether in Medical Colleges or in the periphery services) have been fighting tooth and nail against the Covid-19 disease. Many doctors and paramedical workers have died. Many doctors have closed their clinic. Many hospitals were closed also. The non-Covid patients suffered more than the Covid patients due to lack of public transport system and closure of the clinic and hospitals. Now the incidence of Covid-19 cases has come down in some countries like our country. Still then doctors are not feeling safe to run the clinic. Here I shall mention some guidelines which can be followed by the doctors to open their clinic. Here are these guidelines.

1. If you have got two adjacent rooms connected by a door cover the door with a transparent plastic sheet. You sit in one room and the patient will sit in the other room. Pull your chair and table near the door and put the chair for the patient on the other side. So between the doctor and the patient the plastic sheet will work like a shield. If you do not have two rooms, you can partition one room into two parts by hanging plastic sheets. You sit in one part and the patient should sit in the other part. The height of the partition should be not less than 6.5feet, so that even if a tall patient coughs or sneezes it will not come to the other side. Don't allow anybody to come to your side. The medical representatives can meet you only from the other side.
2. In the plastic sheet make a U shaped cut which will create a flap. Through this flap you can transfer things like prescription or investigation reports with the patient or others on the other side. Even, you can examine the patient through this flap.
3. If your room is provided with AC arrange your table in such way that air from Ac will flow from your side to the other side; not in the reverse direction. If you do not have AC- arrange a wall fan behind you or put a pedestal fan behind you - so that air will blow away from you; not towards you.
4. Don't run the ceiling fan. It will disperse the virus particles to all parts of the room.
5. Don't dry sweep both the rooms. As you know these viral particles are quite heavy, so they quickly settle on to the ground or other surfaces. Dry sweeping will send the viral particles to air from the ground. Rather mop the floor with hypochlorite solution.
6. Do not keep many things on your table top. It will be difficult to sanitize all of them daily.
7. Before you begin your clinic wipe the table top with sanitizer.
8. Wear good quality mask and other personal protective devices. A full PPE kit is not required.

   Time to time sanitize your hand with sanitizer-particularly after handling outside things.

   See that only the patient himself comes for consultation. If very much required allow one more person. See that both of them wear masks covering nose and mouth.
9. Before entry they should sanitize their hands and if possible sanitize their documents in a UV sanitizer. Don't allow the patient to carry unnecessary things with him.
10. Patients having fever for less than 10days should be asked to attend the fever clinic run by the government or ensure Covid test before proceeding further in his treatment.
11. At no time you should open your mask. So stop taking any food or drink in the clinic.
12. Do not allow many people come for consultation on any day. They will make a crowd which is against the government guidelines.

I feel that with all these arrangements and precautions doctors can safely take care of the non-covid patients.

Dr. Kashinath Padhiary

The Assistant Editor speaks…...

The State APICON of this year was kept suspended for a long time. Finally, under the guidance of national API it was decided to hold it in the form of a virtual conference. This decision was also taken within a short period. Information was given to different members through Whatsapp or e-mail. It is obvious that within this short period (around 20days) it might not have been possible to write suitable articles by our members. In spite of that we have tried to bring out the OPJ with the limited articles we have received. Hope that after the situation becomes normal a full- fledged OPJ will come out. There might be some errors in editing. Hope the valuable readers will rectify themselves. Long live Odisha API.

Dr Ashok Kumar Behera

Review Article

SICKLE CELL DISEASE: AN UPDATE

Pradeep Kumar Mohanty

INTRODUCTION:

Sickle cell disease refers to symptomatic forms of sickle cell hemoglobinopathy characterized by inheritance of Hb S gene in homozygous form or in combination with other abnormal haemoglobin variants. First case of sickle cell anaemia was reported by James B. Herrick in the year 1910 [1]. After thisnumberof medical scientists contributed their research findings on detection,pathophysiology and management to enrich the knowledge base and transfer this to clinical practice. Presently there is considerable improvement in survival of the affected persons through improved accessibility, supportive care, penicillin prophylaxis, vaccines against pneumococciand hydroxyurea therapy.

EPIDEMIOLOGY:

Globally the sickle cell hemoglobinopathy is distributed widely in continents of Africa, Americas,Middle eastAsia and Indian sub-continent. In 2010, a century after the first case was detected, the number of babies born with sickle cell disease was estimated to be nearly 3,00,000 per year and for India this was 44,425 births[2.3].

In India sickle cell disease is found to be more concentrated in states Gujarat, Maharashtra, Madhya Pradesh,Chhatisgarh and Odisha. Although initially identified intribes of Nilgiri hills later it was also found to be prevalent in non-tribal populationas well including scheduled castes and caste-Hindus [4].

In Odisha state sickle cell disease is most prevalent in western districts. In a recent screening program 39,000 residential school children were screened for sickle hemoglobinopathy in whom sickle gene prevalence was found to be around nine percent[Unpublished data of Odisha Sickle Cell Project (OSCP)]. The number of sickle cell disease and traits registered at the nodal centre of the Odisha sickle cell Project, VIMSAR stands at 22454 and 30107 respectively as on 4.12.2020. SCD is mostly found among agharia, kulitaand chasa castes as well as in scheduled castes and tribes [5]. In fact, the first report published from Odisha was from agharia communitywhich practiced consanguineous marriage. Most of the SCD cases in India belonged to Asian/ Arab-Indian haplotype[6,7].

Sickle cell diseaseputs considerable financial burden on family in terms of cost incurred on travel, repeated hospitalization, drugs and loss of wages. Further the children with SCD are subjected to loss of school days and poor academic performance. Presently most of the states of India provide a monthly financial grant to SCD affected individuals towards travel and other expenses.

Molecular defect and basis of sickling process

Sickle haemoglobin (Hb S) is formed as a result of replacement of glutamic acid by valine at 6th position of â-globin chain which is due to a point mutation at codon 6 of â-globin gene (GAT to GTG). Hb Shas a tendency to undergo gelation in deoxygenated state to form rigid long polymers that gives the RBC a shape similar to a "sickle " ; in arterial circulation most of these sickled RBCs revert to normal shape due to reoxygenation of Hb S and dissolution of polymers. Polymerized Hb S forms haemoglobin tetramers which takes up a helical structure with multiple layers. Each layer consists of 14 Hb tetramers with a core of four strands surrounded by 10 additional strands as inferred from electron microscopy and X-ray diffraction.

Factors which determine polymerization of Hb S are oxygen tension, Hb Sconcentrationand presence of

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other haemoglobin variants. Increase in 2'3-diphosphoglycerate (DPG) and decrease in pH leads to decrease in oxygen affinity of Hb S and increase in amount of deoxygenated Hb Sresulting in enhanced polymer formation. Mean deoxy-Hb concentration more than 20.8 gm/dl favours polymerization as is presence of HbS, HbC and HbD. HbA and HbF interfere with polymerization of deoxy Hb S[8].

Spectrum of Sickle cell disease:

Homozygous (Hb SS) and symptomatic compound heterozygous states are referred to as sickle cell disease.Compound heterozygotes found in Odisha state include Hb Sâ0 thalassemia, Hb Sâ+thalassemia, H SDpunjab disease,Hb SC disease, Hb SE disease, Hb S-Tianshui, Hb S-Lepore, Hb S-Hofu and Hb S-Westdale etc.

PATHOPHYSIOLOGY:

The deoxygenated Hb S brings up several structural and functional changes in RBC membrane. Initially the sickle cell can revert to biconcave disc like shape on oxygenation but fails to do so after repeated such cycles of deoxygenation and oxygenation. Loss of membrane following repeated sickling and unsickling leads to formation of irreversible sickle cells prone to haemolysis.Shortening of life span of sickle RBCs leads to anaemia and tempting the marrow to compensate by producing more erythrocytes.Reorientation of membrane phospholipids with phosphatidylserine getting exposed on surfacemakes the sickle RBCs sensitive to complement mediated haemolysis and may initiate clotting. Membrane changes also leads to activation of ca^{2+}-activated potassium channel (Gardo's channel) and K-Cl cotransport channel leading to loss of K+ and water from cell and cellular dehydration that promote sickling.Reversible sickle cell expresses adhesion molecules on their surface which make them abnormally adherent to endothelium, leukocytes and platelets culminating in vaso-occlusion [9,10]. In addition,there occurs activation endothelium and release of inflammatory cytokines leading a proinflammatory state [10]. Free haemoglobin in plasma increases viscosity of blood and scavenges nitric oxide (NO), a molecule with multitudes of functions, including vasodilatation [8]. Further, there occurs increased oxidant stress following release of'heme'as well as by ischemia-reperfusion injury . Activation of coagulation has also been noted[11].

Hemolysis in Sickle Cell Disease:

Hemolysis is mainly extravascular mediated through phagocytosis by activated monocytes and macrophages and by physical trapping of sickle cells in splenic pulp. Intravascular hemolysis also occurs from exposure of phosphatidyl serine molecules on surface of sickle cell RBCs making them more sensitive to complement mediated lysis or may be shearinduced [8]. in addition to producing a hemolytic anemia and mild hyperbilirubinemiait also plays role in vaso-occlusion,inflammation and oxidant stress.

VASO-OCCLUSION:

Vaso-occlusion is one of the major pathogenetic process in SCD giving rise to acute painful crisis as well as chronic organ damage. Studies in mice and humans have brought to the fore possible new mechanisms underlying vaso-occlusion which include adhesive interactions between sickled RBC – endothelium, neutrophil-endothelium-sickled RBC,platelet-neutrophils and platelet-endothelium [9,10,11].Further it was observed that heme released into plasma due to hemolysis can promote adhesion through activation of endothelial cells, expression of adhesion molecules on their surfaceand leukocyte recruitment [11]. Heme also activates neutrophils to produce neutrophil extracellular traps (NETs) which can underlie lung injury when exposed to TNF-á [11].

Adhesive interactions between sickled RBC (SS-RBC)and endothelium [9]

SS-RBC expresses on its surface á4â1 integrin, ICAM-4,B-CAM/LU and CD36 which bind to molecules expressed on endothelial cells directly or through plasma proteins(fibronectin,thrombo-spondin,ultra-large vonWillbrand factor)and lead to endothelial activation.Molecules expressed on endothelial cells are V-CAM-1, E-and P-selectins and ávâ3 Integrins.These interactions result in increased levels of oxidant molecules and activation of NF–kB which in turn upregulate genes of molecules such as E- section,VCAM-1 and ICAM-1 on endothelial surface. ICAM-4, an adhesive receptor of RBC bind to endothelial ávâ3 Integrin being activated by epinephrine[9,10].

Adhesive interaction between neutrophils and endothelium

Neutrophils activated by microbiota signaling crawl on endothelial surface and interact with endothelial E-selection and P-selectin which lead to expression áMâ2 integrin (Mac-1) on leading edge of the neutrophils capable of binding sickled red cells.This leads to trapping of cells in circulation and occlusion in post-capillary venules. Various molecules have been developed to interfere with Mac-1 expression which had the potential to prevent or reverse the vaso-occlusion [11].

Role of inflammation

SCD is characterized by presence of a state of inflammation mediated by activated monocytes, endothelialcells, neutrophils, sickled erythrocytes, invariant natural killer T (iNKT) cells and platelets.Endothelial cells get activated by monocytes,platelets, adhesion of sickle red cells,heme mediated release of placental growth factor (PlGF) from RBCs and proinflammatory cytokines (TNF-á,IL-6,IL-1â, GM-CSF, IL-3, endothelin-1,PGE2).Endothelial cell activation leads to nuclear factor NF-KB pathway activation resulting in expression of adhesion molecules ICAM-1,VCAM-1,E-Selection and P-Selection which promotes neutrophil recruitment and vaso-occlusion. Monocytes are activated by platelets and PlGFleading to secretion of TNF-á IL1-â, IL-8 and monocytes chemoattractantprotein-1 [12,13,14].PlGFrelease is induced from RBCS through TLR-4 pathway by heme.

An activated phenotype of iNKT Cells are found in SCD which increased during vaso-occlusion and responsible for pulmonary inflammation and dysfunction through secretion of IFN-˜ and induction of chemokine CXCR3 [15]. Activated iNKT cells also express increased adenosine 2A receptor ($A_{2A}R$) which has anti-inflammatory property [16].Highmobility group box-1 (HMGB-1), a danger associated molecular pattern (DAMP) molecular released from activated immune cells and necrotic cells and found to be elevated in SCD can promote inflammation through TLR-4 signalingpathway [17].

These molecules and receptors expressed in and foundin SCD are now novel experimental targets for intervention of which few have been approved for use by FDA.

Clinical Presentation in Sickle Cell Disease:

Clinical presentation in sickle cell disease is variable and range from nearly asymptomatic to mild to moderate or severe depending ongenetic modifiers and environmental factors. However, most patients remain symptomatic throughout their life. The acute manifestations which disturb the steady state of SCD include infections, dactylitis (hand foot syndrome) and splenic sequestration crisis, aplastic crisis, hemolytic crisis, acute chest syndrome, stroke, priapism, avascular neurosis of femoral and humeral head, abdominal crisis and sickle cell hepatopathy [8].

Vaso-occlusive crisis (VOC) or acute painful crisis (APC) is the most common manifestation and its frequency is associated with decreased survival. In most typical form, it presents with pain long bones of extremities, ribs, vertebrae and around the joints lasting for about 4-10 days with a tendency to recur[8]. Its variants include dactylitis in children and abdominal crisis due to mesenteric and splenic infarctions. Common precipitating factors of VOC are physical and mental exertion, exposure to cold or hot environment, fever, dehydration and discontinuation of hydroxyurea.Acute chest syndrome (ACS) is characterized by chest pain, fever, hypoxemia and dyspnea, new pulmonary infiltration, leukocytosis and acute anemia [8]. ACS is potentially life threatening sometimes associated with precipitous fall in oxygen saturation. Stroke not common in Arab-Indian haplotype is usually ischemic type affecting children but can occur in adults when it may be hemorrhagic. Priapism is uncommon but associated withconsiderable suffering. Avascular necrosis of femoral head is not uncommon in Indian subsets of SCD and presents with unilateral (rarely bilateral) hip pain with limping [Odisha sickle cell project data]. Sickle cell hepatopathy is characterized byrise of serum bilirubin to very high levels (100 mg/dl) in absence of viral hepatitis and other causes of hepatitis; the conditions which are included under are acute hepatic sequestration and intrahepatic cholestasis [18,19].

Splenic sequestration crisis is a life-threatening manifestation of SCD. Children present with acute enlargement of spleen, acute anemia with Hb level falling by > 2.0 g/dl and hypovolemic shock which can lead to fatality unless recognized and treated promptly.

Aplastic crisis characterized by transient suppression of erythropoiesis and acute anemia following infection by Parvo virus B19 infection.

Chronic complications of SCD include delayed growth and development (not in all) and delayed attainment of puberty and menarche, gall stones, chronic pulmonary hypertension, leg ulcer, cognitive dysfunction and ocular changes. Other renal changes include micro- and macro-albuminuria, impaired urinary concentration, renal papillary necrosis and nephrotic syndrome. Chronic leg ulcers are infrequent in our patients, takes a long time to heal.

Pregnancy and Surgery in SCD:

Pregnancy is getting safer with better outcomes. However, there can be fetal and maternal complications as well as increased frequency of sickle cell related complications. There is increased incidence of pre-eclampsia and eclampsia. Preterm labor, low birth weight and neonatal jaundice are found in infants born to SCD mothers. In pregnancy, there is increased frequency of pyelonephritis, hematuria and anemia; there is increased incidence of vaso-occlusive crisis during pregnancy and child birth.

Surgery and general anaesthesia predispose SCD patients to vaso-occlusive crisis and acute chest syndrome. Need for surgery may arise out of sickle cell-related or unrelated problems. Splenectomy, cholecystectomy, hip arthroplasty may be needed for the patient. Caution is to be exercised in raising hemoglobin to 10g/dl and close observation of patient during surgery and later inpost-operative period.

Laboratory Diagnosis of SCD:

Diagnosis methods used are sickle cell slide tests or solubility test, hemoglobin electrophoresis in acidic or alkaline pH, cation exchange high performance liquid chromatography (CEHPLC) or isoelectronic focusing or PCR based tests and genetic sequencing. Sickle cell slide test is used to detect sickle cell in peripheral blood by including hypoxia which can be enhanced by addition of sodium metabisulfite; it is used as a screening test[8]. Hemoglobin electrophoresis in agarose gel at alkaline pH 8.6 detects different bands of hemoglobin at different positions from point of application; presence of Hb S or SF band in absence of Hb A band indicates presence of sickle cell disease.

HPLC is an automated system that has revolutionized the hemoglobinopathy diagnosis. Various hemoglobin variants appear after different time intervals from the time of presenting the processed samples of blood to the system. The time of appearance of specific hemoglobin is called retention time of that hemoglobin. In each of the time window after beginning of the testThis apart the morphology and amount calculated from area under curveof the graph on the system generated paper identifies the hemoglobin variants and quantity of Hb in percentage; however, interpretation must be done by an expert.Below are shown the windows and retention times of various hemoglobin variants and a chromatogram homozygous sickle cell anemia.

Table1.Manufacturer assigned retention times ˜ Bio-Rad Variant II â-thal short program (Hercules, CA)

WINDOW	Retention time in minutes	Hb variants	Retention time in minutes
Hb F window	0.98-1.20	Hb-F	1.05
Hb A0 window	1.90 -3.10	Hb A	2.41
Hb A2 window	3.30-3.90	Hb A2	3.62
Hb D window	3.90-4.30	Hb D	4.19
Hb S window	4.30-4.70	Hb S	4.5
Hb C window	4.90-5.30	Hb C	5.0

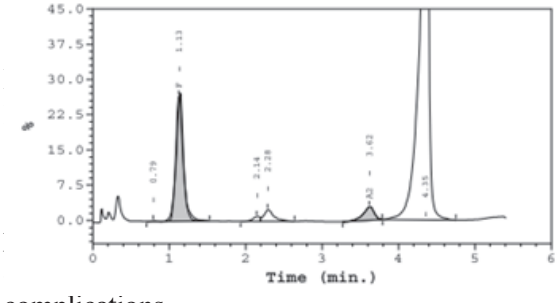
Peak Name	Calibrated Area %	Area %	Retention Time (min)	Peak Area
P1	---	0.1	0.79	992
F	18.1*	---	1.13	267944
Unknown	---	0.7	2.14	10936
Ao	---	2.3	2.28	34724
A2	2.9	---	3.62	43642
S-window	---	76.2	4.35	1148844

Total Area: 1,507,081

F Concentration = 18.1* %
A2 Concentration = 2.9 %

*Values outside of expected ranges

Analysis comments:



complications.

MANAGEMENT IN STEADY STATE:

Penicillin prophylaxis and vaccines: Diagnosis of SCD can be done soon after the HbF is replaced by adult Hb as early as 3 months but usually by 6 months. Infection with capsulated organisms particularly pneumococcus or H- influenzae is common in childhood. Prevention of infection is done by oral penicillin prophylaxis upto 5 years of age [21]. Pneumococcal vaccine is given to children and adults with SCD. Other vaccines recommended are H-influenzae type b vaccine, hepatitis B vaccine and influenza vaccine [R].

Folic acid: Folic acid supplementation is done to meet the demand of accelerated erythropoiesis to compensate forhemolysis. Recommended dose for adult is 1 mg daily.

Hydroxyurea: Hydroxyurea(HU) is an S-phase cytotoxic agent used first in malignant diseases. In the year 1984 Platt OS et al found HU to induce HbF and later several studies substantiatedits usefulness in SCD [22,23]. This was approved for use in 1998. Hydroxyurea use has revolutionized the management of SCD with decrease in morbidity and mortality. HU decreases the frequency of painful crisis, prevents acute chest-syndrome and decreases frequency of blood transfusion; it was used as an alternative to blood transfusion for prophylaxis against stroke [R]. HU increases level of Hb, Hb F, F-cell numbers and MCV; it decreases WBC, neutrophil and platelet counts, and serum bilirubin level.

Mechanisms action of hydroxyurea:

1. Induction of fetal hemoglobin: HU causes intermittent cytotoxic suppression of erythroid progenitors and signaling of stress erythropoiesis leading to recruitment of erythroid progenitors with more F-cells. Further, it generates nitric oxide (NO) and increase NO-dependent soluble guanyl cyclase level leading to increased cGMP, increased gamma gene expression and more Hb F[24].
2. Reduces leukocyte and platelet count and interferes vaso-occlusion.
3. HU decreases expression of red cell and endothelial adhesion molecules VCAM-1 and ICAM-1.
4. May produce local vasodilation mediated by NO.
5. Increases MCV and improves rheology.

Usual dose is 15-35 mg/kg/day depending on the response and tolerance to HU. In India **low fixed dose HU** at 10 mg/kg/day was found to be equally effective by several researchers including our center [25,26,27]. HU dose is reduced in presence of CKD to 5-10 mg/Kg/ day.

Indications for hydroxyurea has been expanded and include the following [28]:

1. Adults with SCA with three or more moderate VOC in a 12-months period.
2. Adults with SCA with sickle cell associated pain interfering with daily activities and quality of life.
3. Adults with SCA with severe and/or recurrent ACS.
4. Adults with SCA with symptomatic chronic anemia interfering with daily activities and quality of life.
5. Infants 9 months or older, children and adolescents with SCA may be offered HU irrespective of clinical severity of SCD related complications.
6. In adults and children with SCD who have CKD and taking erythropoietin.
7. In those with HbSâ$^{+}$ thalassemia or HbSC with recurrent sickle cell associated pain interfering with daily activities of life on advice of sickle cell expert.

Contraindicationsof HU include pregnancy and lactation, absolute neutrophil count(ANC) less than 2000/cubic mm., total platelet count (PLT)less than 80,000/cubic mm., Hb < 5 g/dl, hypersensitivity to HU in past, severe hepatic disease.

Safety profile and toxicities: HU is relatively safe; toxicities include transient reversible myelosuppression; HU is teratogenic in animal studies and found reduce sperm count and mortality in mice; so avoided in pregnancy and used with a watch sperm parameters in males who are unmarried or have not completed family.

Consent and monitoring: Informed consent must be taken from patient or his guardian before prescribing hydroxyurea; patient must be ready for periodic monitoring of toxicities and response to therapy.

HU should be discontinued when ANC < 2000/cubic mm., PLT < 80,000 /cubic mm. and Hb < 5 g/ dl. HU can be restarted after hematological recovery starting with lower dose and increasing gradually.

Response to hydroxyurea to occur takes three to six months. This can be known from clinical and laboratory parameters as well as increase in Hb F level.

Blood transfusion: Before HU blood transfusion was the only treatment proven to modify the clinical outcome of SCD. Its use must be rationalized. In steady state repeated transfusion is most appropriately indicated in primary and secondary prophylaxis of stroke; it is also indicated as top-up transfusion during pregnancy and child birth with Hb< 7.0 g/dl, in perioperative period to keep the Hb level at 10 g/dl and in case of symptomatic anemia limiting daily activities of life. In acute complications simple or exchange transfusion is indicated in ACS, acute splenic sequestration, aplastic crisis, stroke, hepatic sequestration, Intra-hepatic cholestasis and Multi-system organ failure[28].

Chronic complications of blood transfusionincludeiron overload, alloimmunization, delayed hemolytic transfusion reactions and hyper-hemolytic syndrome of SCD. **Hyperhemolysis syndromeof SCD** is characterized by falling of post-transfusion hemoglobin level below that of pre-transfusion level; it is potentially life threatening and occurs in 3 to5 % of transfusions; presents with fever, jaundice,pain, hemoglobinuria, increased bilirubin and LDH and reticulocytopenia [29].Treatment found to be of benefit include combination of IVIG with methyl prednisolone, rituximab, erythropoietin, eculizumab and therapeutic plasma exchange.

Recently approved agents for use in SCD are L-glutamine, Voxelotor and Crizanlizumab. These drugs may supplement hydroxyurea in future or play important role patient who are not candidates for contraindications or those having no response to HU.

L-glutamine:A precursor of NAD, Glutathione and arginine is an antioxidant approved by FDA in 2017 for se in SCD for individuals above five years of age; long term safety data is not available. It should be avoided in cases of hepatic and renal derangements.Can be used add on to optimal hydroxyurea treatment or if hydroxyurea is not tolerated can be started as primary treatment. Dose is 5-15 gm twice a day with 240 ml of beverage.

Voxelotor: Approved on November 25, 2019, by FDA for adults and pediatric patients 12 years of age and older with sickle cell disease it is an inhibitor of deoxygenated sickle hemoglobin polymerization, which is the central abnormality in sickle cell disease.It increases hemoglobin and decreases hemolysis.The recommended voxelotor dose is 1500 mg orally once daily with or without food. It is relatively safe with adverse effects like headache, diarrhea, abdominal pain, nausea, rash, fatigue and pyrexia,hypersensitivity and potential laboratory interference. Voxelotor may interfere with measurement of Hb subtypes (HbA, HbS, and HbF) by high performance liquid chromatography.

Crizanlizumab: it is a P- selectin inhibitor approved on November 15, 2019, FDA; reduces the frequency of vaso-occlusive crises (VOCs) in adults and pediatric patients aged 16 years and older with sickle cell disease.Most common adverse reactions (>10%) are nausea, arthralgia, back pain, and pyrexia.The recommended dose is 5 mg/kg intravenously over a period of 30 minutes on week 0, 2, and every 4 weeks thereafter.

Management of acute complications:

Infections are usually due capsulated organisms. Pneumococcal sepsis and meningitis, meningitis due to H. influenzae and meningococci can occur and treated with parenteral ceftriaxone. Osteomyelitis is not common. Vaso-occlusive crisis is managed by prompt administration of opioid analgesics and/or NSAIDsat earliest within 30 minutes. NSAIDs must be used with caution in SCD as in older patients underlying CKD might be present. NSAIDs are notrecommended during pregnancybefore 12 weeks and after 28 weeks. Oral or IV fluids may be prescribed as are antibiotics. Close watch on patients with VOC is essential as the course can get complicated with ACS or acute anemia. ACS should be preferably treated in ICU. Supplementary oxygen to maintain saturation above 95%,simple or exchange transfusion, antibiotics (ceftriaxone and oral macrolide) are to be initiated and the patient needs to beclosely monitored [28]. Mechanical ventilation may

be needed. Acute avascular necrosis is managed by analgesics and avoidance of weight bearing. Aplastic crisis and splenic sequestration crises are managed with blood transfusion. Stroke is managed in same manner as in other cases except that transfusion is given. Priapism is managed with analgesics and fluids.

Management of chronic complications:

Chronic problems such as CKD managedas in other cases; dose of HU is to be modified to 5-10 mg/kg/day. Pulmonary hypertension does not respond well to drugs. Cholecystectomy may be needed for gall stones.Leg ulcer are treated by topical agents.

Hematopoietic stem cell transplantation

It is the only curative treatment but only few cases have been transplanted until recently because of inherent challenges in SCD. These are non-availability of HLA matched healthy siblings, transplant related morbidity and graft failure. Improvement in scenario is expected with reduced intensity safe regimens and better graft survival.

Gene therapy

Gene therapy intended to correct the mutation in hematopoietic stemcellsare tried with CRISPR/CAS9 gene editing system and has been found to be successful in few cases [30]. But this has tobe translated into clinical practice and affordableand free from adverse effects to live up to the expectations of SCD patients.

Many novel agents are presently undergoing trial targeting various molecules and pathway involved in pathophysiology of SCD. Hb F inducing agents, antioxidants, anti-inflammatory agents, antiplatelet agents and anticoagulants, selectin inhibitors, antiadhesive agents are in pipeline [10,11].

Prognosis:

Many children now reach adulthood and pursue their livelihood meaningfully with improved quality of daily living by using hydroxyurea and regular follow up. Acute complications remain a challenge which can punctuate the life early. Mortality is mainly due to severe VOC, acute chest syndrome, infections, stroke, splenic sequestration crisis, sickle cell hepatopathy,hyperhemolytic crisis, end stage renal disease and pulmonary hypertension. Thoughtful monitoring, counselling and management with newer drugs, improvement in transplant procedure as well as gene therapy may improve survival further in SCD.

Prevention of SCDand its complications:

Preventing birth of SCD child is a goal for every country and state to limit disease burden as well as to provide adequate care to those affectedwith available resources. SCD is amenable for prevention through various means. Premarital screening followed byavoidance of marriage between sickle cell traits and/or disease havenot been successful in past. Prenatal diagnosis remains a feasible option adapting which countries like Cyprus has lowered their burden of thalassemia major to near zero.

Pre-implantation genetic diagnosis:

Fetal cells are collected from blastocyst between three to five days and subjected to DNA analysis to know the genotype. If the test shows normal hemoglobin genotype the couple can continue pregnancy; if found to have SCD genotype they can take the decision for discontinuation.

Prenatal diagnosis:

Here also fetal cells are collected but the timing and procedures are different. Chorionic villus sampling (CVS) is the preferred method done usually between 10-12 weeks of pregnancy to allow the couple to make a informed decision regarding continuation or termination of pregnancy. Providing free pre-natal diagnosis to couples is planned for Odisha state under collaborative program with Christian Medical College, Vellore which will be helpful in this regard.

Newborn screening:

Early detection and intervention can save the life many children from morbidity and mortality.

REFERENCES:

1. Herrick JB. Peculiar elongated and sickle-shaped red blood corpuscles in a case of severe anemia. *Arch Intern Med (Chic).* 1910; VI(5):517–521. doi:10.1001/archinte.1910.00050330050003
2. Piel FB. Patil AP, Howes RE, Nyangiri OA, Gething PW et al. Global epidemiology of sickle haemoglobin in neonates: a contemporary geostatistical model-based map and population estimates.Lancet 2013; 380:142-151.
3. Piel FB, Hay SI, Gupta S, Weatherall DJ, Williams T. Global burden of sickle cell anaemia in children under

five, 2010-2050: modelling based on demographics, excessmortality and interventions. PLoS Med. 2013;10(7):e 1001484. Doi:10.137/J. Pmed. 1001484. Epub 2013 July 16. Accessed on 07.12.2020.

4. Kar B C, Devi S,Dash K C, Das M. The sickle cell gene is widespread in India, *Transactions of The Royal Society of Tropical Medicine and Hygiene* 1987; 81(2) :273–275.

5. Nanda BK, Panda GK, Naik UP, Nanda CN, Praharaj KC.Haemoglobin-S in Agharia community of Orissa. J Indian Med Assoc. 1967 Feb 16;48(4):150-2.

6. Kulozik AE, Wainscoat JS, Serjeant GR, Kar BC, Al-Awamy B, Essan GJ, Falusi AG, Haque SK, Hilali AM, Kate S, et al. Geographical survey of beta S-globin gene haplotypes: evidence for an independent Asian origin of the sickle-cell mutation. Am J Hum Genet. 1986 Aug;39(2):239-44.

7. Patel DK, Mashon RS, Patel S, Das BS, Purohit P, Bishwal SC. Low dose hydroxyurea is effective in reducing the incidence of painful crisis and frequency of blood transfusion sickle cell anaemia in patients from eastern India. Hemoglobin. 2012;36(5):409-420

8. Henkins JS, Wang WC. Sickle cell anemia and other sickling syndromes. In: Greer JP,

Rodgers GM, Glader BE, Arber DA, Means RT (Jr), List AF, Appelbaum FR,Dispenzieri A, Fehniger TA (eds). Wintrobe's clinical hematology. 14th edition.Philadelphia: Wolters Kluwer; 2019. p823-865.

9. Kaul DK, Finnegan E, Barabino GA. Sickle red cell-endothelium interactions. Microcirculation. 2009;16(1):97-111.

10. Manwani D, Frenette PS. Vaso-occlusion in sickle cell disease: pathophysiology and novel targeted therapies. Blood. 2013;122(24):3892-3898.

11. Zhang D, Xu C, Manwani D, Frennete P S. Neutrophils, platelets and inflammatory pathways at the nexus of sickle cell disease pathways. Blood 2016;127(7): 801-809.

12. Wun T, Cordoba M, Rangaswami A, CheungAW, Paglieroni T. Activated monocytes and

platelet-monocyte aggregates in patients withsickle cell disease. Clin Lab Haematol. 2002;24(2):81-88.

13. Selvaraj SK, Giri RK, Perelman N, Johnson C, Malik P, Kalra VK. Mechanism of monocyte activation and expression of proinflammatory cytochemokines by placenta growth factor. Blood 2003;102(4):1515-1524.

14. Perelman N, Selvaraj SK, Batra S, et al. Placenta growth factor activates monocytes and correlates with sickle cell disease severity.Blood. 2003;102(4):1506-1514

15. Wallace KL, Marshall MA, Ramos SI, et al. NKT cells mediate pulmonary inflammation and dysfunction in murine sickle cell disease through production of IFN-˜ and CXCR3 chemokines. Blood. 2009;114(3):667-676.

16. Wallace KL, Linden J. Adenosine A2A receptors induced on iNKT and NK cells reduce pulmonary inflammation and injury in mice with sickle cell disease. Blood. 2010;116(23):5010-5020.

17. Xu H, Wandersee NJ, Guo Y, et al. Sickle cell disease increases high mobility group box 1: a novel mechanism of inflammation. Blood. 2014; 124(26):3978-3981.

18. Irizarry K, Rossbach HC, Ignacio JR, Winesett MP, Kaiser GC, Kumar M, Gilbert-Barness E, Wilsey MJ Jr. Sickle cell intrahepatic cholestasis with cholelithiasis. PediatrHematol Oncol. 2006 Mar;23(2):95-102. doi: 10.1080/08880010500456964. PMID: 16651237.

19. Hatton CS, Bunch C, Weatherall DJ. Hepatic sequestration in sickle cell anaemia. *Br Med J(Clin Res Ed)*. 1985;290(6470):744-5.

20. Joutovsky A, Hadzi-Nesic J,Nardi M A. HPLC Retention Time as a Diagnostic Tool for Hemoglobin Variants and Hemoglobinopathies: A Study of 60000 Samples in a Clinical Diagnostic Laboratory, Clinical Chemistry 2004; 50(10): 1736–1747

21. Woods G, Pegelow C, Kelleher J, Presbury G, Zarkowsky H, Vichinsky E, Iyer R, Lobel JS, et al.Prophylaxis with oral penicillin in children with sickle cell anemia. A randomized trial. N Engl J Med. 1986 Jun 19;314(25):1593-9.

22. Platt OS, Orkin SH, Dover G, Beardsley GP, Miller B, Nathan DG. Hydroxyurea enhances fetal hemoglobin production in sickle cell anemia. *J Clin Invest*. 1984;74(2):652-656.

23. Charache S,Terrin M L, Moore R D, Dover J G, Barton F B, Eckert S Vet al. Effect of hydroxyurea on the frequency of painful crisis in sickle cell anemia. N. Eng. J. Med. 1995; 332: 1317-22

24. Cokic VP, Smith RD, Beleslin-Cokic BB, Njoroge JM, Miller JL, Gladwin MT, Schechter AN. Hydroxyurea induces fetal hemoglobin by the nitric oxide-dependent activation of soluble guanylyl cyclase. J.Clin.Invest. 2003; 111:231–239. [PubMed: 12531879].

25. Italia K et al. Hydroxyurea in sickle cell disease – A study of clinico-pharmacological efficacy in the Indian haplotype. Blood cells molecules and diseases. 2009; 42(1): 25 -31.

26. Jain DL et al. Low fixed dose hydroxyurea in severely affected Indian children with Sickle cell disease. Hemoglobin 2012; 36(4): 323-32.

27. Patel DK et al. Low dose hydroxyurea is effective in reducing the incidence of painful crises and frequency of blood transfusion in sickle cell Anemia patients from Eastern India. Hemoglobin 2012 Aug. 13 (E.pub ahead of print)

28. Evidence based management of sickle cell disease. Expert panel report 2014.US Department of health and human services. NIH.NHLBI.

29. Banks M, Shikle J. Hyperhemolysis syndrome in patients with sickle cell disease. Arch Pathol Lab Med. 2018; 142:1425–1427.

30. Ferrari G, Cavazzana M, MavilioF.Gene Therapy Approaches to Hemoglobinopathies.HematolOncolClin North Am. 2017 Oct;31(5):835-852. doi: 10.1016/j.hoc.2017.06.010.

31. Kangpu Xu, PhD; Zhong Ming Shi, MD; Lucinda L. Veeck, MLT, DSc; Mark R. Hughes, MD, PhD; Zev Rosenwaks, MD.First Unaffected Pregnancy Using Preimplantation Genetic Diagnosis for Sickle Cell Anemia.JAMA. 1999;281(18):1701-1706.

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Review Article

FINGER PULSE OXYMETER : AN INESCAPABLE CLINICAL DIGNOSTIC TOOL FOR PHYSICIAN AKIN TO STETHOSCOPE

Surgeon Captain Dr. Sambhu Dutta

INTRODUCTION :

In response to most unfortunate comment by Eric Topol, MD, of Scripps Translational Science Institute in La Jolla,**The stethoscope's 200th birthday should also be its funeral,"** , Professor Emeritus Valentin Fuster from Mount Sinai Hospital in New York City wrote in the famous Journal of American College Cardiology On 08 Mar 2016 , Quote "In my view, practically and economically, echocardiography systems are not and will never be poised to totally eradicate the stethoscope, as it is not possible for every clinician to possess a handheld echocardiography within and outside the United States," "Thus, we cannot discontinue the important training that takes place during physical exam, which can be aided through the amplified sounds of a stethoscope" Unquote.

As a true companion to time tested stethoscope in our day today clinical bed side medicine and critical care practice the role of **finger pulse oximeter** can not be overlooked as it has truly turned out to be a inescapable instrument in the hand of physician ,intensivist and internists, nurses, paramedics more so especially in this Covid era. Pulse oximetry is a Saviour noninvasive gadget for monitoring a person's oxygen saturation. Pulse oximetry is ubiquitously used for monitoring oxygenation in the critical care setting. By forewarning the clinicians about the presence of hypoxemia, pulse oximeters may lead to a quicker treatment of serious hypoxemia and possibly circumvent serious complications. {1}

Principles of Latest Pulse Oximetry

The pulse oximeter measures the saturation of hemoglobin in arterial blood, which is a measure of the average amount of oxygen bound to each hemoglobin molecule plus any dyshemoglobins. The percentage saturation is given as a digital readout together with an audible signal varying in pitch depending on the oxygen saturation. The pulse oximeter also displays the pulse rate in beats per minute, averaged over 5 to 20 seconds, or the digital value is modified with each beat-to-beat measurement. An additional value is available on some pulse oximeters—the perfusion index (PI) value, which is an indication of relative perfusion at the sensor site.Even some high end pulse oximeter give PPV indices (Pulse Pressure Variation) an imprtant parameter of fluid volume resuscitation to guide fliud / inotrope therapy.

Oxygen is carried in the bloodstream primarily bound to hemoglobin. One molecule of hemoglobin can carry up to four molecules of oxygen, and it is then 100% saturated with oxygen. The average percentage saturation of a population of hemoglobin molecules in a blood sample is the oxygen saturation of the blood. In addition, a very small quantity of oxygen is carried dissolved in the plasma. Oxygen transported with the plasma is not measured by pulse oximetry.

A pulse oximeter consists of a sensor, together with an oximeter unit, displaying a waveform, the oxygen saturation, and the pulse rate, and/or PI value as well as PPV(In high end versions). The sensor is placed on a peripheral tissue bed such as a digit, ear lobe, or nose. Proper sensor selection and placement are critical to accurate and continuous measurements in the presence of low perfusion. Within the sensor are two or three light emitting diodes (LEDs). The diodes emit light, which is both visible and invisible to the human eye and passes through the tissues to a photodetector.

The pulse oximeter identifies the absorbancy of the pulsatile fraction of blood—defined as the arterial component—from the absorbancy due to nonpulsatile venous or capillary blood and other tissue pigments. Identification and isolation of the pulsatile component

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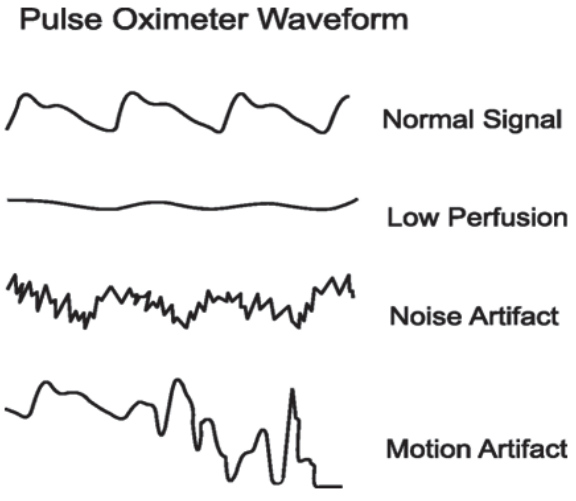
are difficult in the presence of low perfusion in the patient. Recent advances in pulse oximetry technology have reduced the effects of low perfusion on pulse oximeter function.



Common pulsatile signals on a pulse oximeter. (Top panel) Normal signal showing the sharp waveform with a clear dicrotic notch. (Second panel) Pulsatile signal during low perfusion showing a typical sine wave. (Third panel) Pulsatile signal with superimposed noise artifact giving a jagged appearance. (Bottom panel) Pulsatile signal during motion artifact showing an erratic waveform.

Limitations of pulse oximetry

Shape of oxygen dissociation curve, Dyshemoglobins, Carboxyhemoglobin, Methemoglobin, Dyes, Low Perfusion State, Skin pigmentation, Anemia, Nail polish, Motion artifact

CLINICAL USE OF PULSE OXYMETER

Pulse oximeters provide noninvasive analysis of the arterial hemoglobin oxygen saturation. **Two principles** are involved:

1. Differential light absorption by hemoglobin and oxyhemoglobin; and 2. Identifying pulsatile component of signal. Pulse oximetry does not provide a direct indication of patients' ventilation, only of oxygenation.

<u>Pulse oximetry can provide an early warning of hypoxemia</u>. Pulse oximetry has been shown to be reliable in titrating the fractional inspired oxygen concentration (F_IO_2) in patients requiring mechanical ventilation; aiming for a SpO_2 of 92 % is reasonable for ensuring satisfactory oxygenation. The ratio of SpO_2 to F_IO_2 (S/F) can be used as a surrogate for the ratio of PaO_2 to F_IO_2 (P/F). {2}

Studies have shown that the presence of pulse oximetry may reduce the number of arterial blood gas samples obtained in the ICU and in the emergency department. In the largest randomized trial involving more than 20,000 perioperative patients, rates of incidence of hypoxemia (SpO_2 of less than 90 %) were 7.9 % in patients who were monitored with pulse oximetry and only 0.4 % in patients without an oximeter. {2}

COVID SEVERITY AND PROGNOSTICATION

Across the world healthcare systems are dealing with COVID 19. One of the main manifestations of this infection is varied degree of involvement of lung causing a spectrum of illness from mild lower respiratory tract infection to severe Adult Respiratory Distress Syndrome (ARDS). One of the important clinical parameters is to identify hypoxia early to initiate higher level of care at the earliest. However, presence of silent or latent hypoxia has made this task a challenge in COVID 19. A simple 6-minute walk test (6MWT) to look for inducible hypoxia for a patient who looks comfortable and is not hypoxic at rest, helps in early detection of hypoxia and initiating early higher-level care. The 6MWT also helps in looking for discharge preparedness of patient. This simple tool has immense clinical applicability to ensure safe care of COVID 19 patients. One of the important clinical parameters is to identify hypoxia early to initiate higher level of care at the earliest. However, presence of silent or latent hypoxia has made this task a challenge in COVID 19. A simple 6-minute walk test (6MWT) to look for inducible hypoxia (fall in baseline SPO2 by 3%) for a patient who looks comfortable and is not hypoxic at rest, helps in early detection of hypoxia and initiating early higher-level care. {3}

Use of perfusion index from pulse oximetry

Pulse Index (P.I.) is provided in the newer pulse oxymetry a vital clinical point of care marker to detect the state of peripheral perfusion status.

PI is an indicator of the relative strength of the pulsatile signal from pulse oximetry and has been found

Case Report

A CASE OF THYROTOXIC HYPOKALEMIC PALSY IMPROVED BY IV CORTICOSTEROID

Saumya Ranjan Maharana[1], Ganeswar Sethi[2]

ABSTRACT

Thyrotoxic hypokalemic palsy is a rare complication of hyperthyroidism characterized by sudden onset weakness of all the four limbs associated with hypokalemia .It is typically seen in young Asian males.(1) But here we report a case of middle aged female patient hospitalized with a chief complaint of sudden onset weakness of all the four limbs of 1 day duration.

Patient was evaluated for the acute onset flaccid Quadriplegia. Evaluation during admission revealed severe hypokalemia with serum potassium level of 1.7meq/l. Potassium chloride infusion was given in view of hypokalemic periodic palsy but patient did not show much improvement clinically as well as biochemically (no significant improvement in serum potassium level).But administration of IV corticosteroid resolved the muscle weakness , also corrected the serum potassium level. Thyroid function test showed elevation in freeT3 & freeT4 level with suppression of TSH. Patient was put on antithyroid drug which led the complete reversal of clinical signs & symptoms. Hence, a diagnosis of thyrotoxic hypokalemic palsy was made, which is a reversible cause of acute onset flaccid quadriplegia & highindex of suspicion as well as timely interventions are required to prevent its life threatening complications.

INTRODUCTION

Thyrotoxic hypokalemic palsy is a rare, but serious complication of hyperthyroidism. It has been reported to occur in 1.8% to 1.9% of thyrotoxic patients of Asia(2). Hypokalemia in the setting of hyperthyroidism is the cornerstone of this condition. Hypokalemia occurs as a result of transcellular shift of potassium into the intracellular compartment .This intracellular shift is attributed to both direct & indirect activation of Na+/K+ ATPase by the thyroid hormones, resulting in increased uptake of K+ by muscles.(3) Definitive treatment of hyperthyroidism results in complete resolution of paralytic episode.(4)

Here we present a case of thyrotoxic hypokalemic palsy who presented with an episode of sudden onset quadriplegia associated with hypokalemia.

CASE REPORT

A middle aged female patient was hospitalized with a chief complaint of sudden onset weakness of all the four limbs of 1 day duration. She felt the weakness in her limbs while she got up from the bed in the morning. Lower limbs were affected more than the upper limbs. There was no history suggestive of sensory system involvement. She had intact bladder & bowel functions. She gave a history of feeling of nervousness, excessive anxiety, sweating, tremor & palpitation of around 4 months duration. She denied that she had never experienced similar kind of illness in past. None of her family members suffered from similar attack.

On examination : Patient was conscious, oriented but a bit anxious.

Her vitals were : pulse rate-140/min, regular in rhythm, blood pressure 98/50 mmHg in right arm supine position, Oxygen saturation was 95% in ambient air. Respiratory rate was 16/min and Temperature was 98.9 degree Fahrenheit. There was mild pallor, tremors in her hands, but no apparent thyromegaly or exophthalmos.

CNS

(i) Higher functions & cranial nerves were found to be intact.

(ii) Motor system examination revealed power of 2/5 in both lower limbs ,3/5 in both upper limbs.

(iii) Tone-hypotonia

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(IV) Deep tendon reflexes were absent in both upper & lower limbs.

Examination of the respiratory system did not reveal any abnormality

Cardiovascular system examination showed Tachycardia & loud 1st heart sound

On laboratory investigations : complete blood count (CBC) was within normal limits. serum creatinine 1.3mg/dl. However, serum potassium -1.7meq/l, serum phosphate- 1.9mg/dl & serum magnesium- 1.06mg/dl. Thyroid function test report was awaited.

ECG showed the features of hypokalemia with prominent u waves in mid precordial leads with prolongation of QT interval.

Treatment : Potassium chloride infusion was given in view of hypokalemic palsy but patient did not show any improvement. Later on intravenous corticosteroid [hydrocortisone 50mg i.v. 6hrly] was administered suspecting thyrotoxic hypokalemic palsy. Just after 1 day the patient showed dramatic improvement.

Thyroid function tests showed increase in freeT3 & freeT4 level with suppression of Serum TSH level. Based upon that methimazole 10mg & propranolol 40mg were advised.

DISCUSSION

Thyrotoxic hypokalemic palsy is a rare & potentially life threatening condition. The majority of cases are seen in hyperthyroidism due to Graves'disease, however toxic adenoma, thyroiditis, toxic multinodular goitre, amiodarone induced thyroiditis, levothyroxine intoxication & TSH producing pituitary adenoma, all have been associated with thyrotoxic hypokalemic palsy.(5)

Thyrotoxic hypokalemic palsy usually presents in the 3rd to 5th decades of life , sporadic inheritance & is always a consequence of thyrotoxicosis.(6) Our patient was a middle aged female with hyperthyroidism.

Thyrotoxic hypokalemic palsy is a rare complication of hyperthyroidism characterized by hypokalemia & acute muscle weakness. (7)The patient in our case report also presented with sudden onset weakness of all the 4 limbs of 1 day duration.

The severity of muscle weakness depends on the degree of hypokalemia .(8) Thyroid hormone mediated stimulation of Na+/k+ ATPase pump leads to enhanced intracellular shift of k+ producing hypokalemia.(5)

Management of thyrotoxic hypokalemic palsy involves the correction of hypokalemia with potassium chloride (Kcl) infusion

.In most cases less than 50 mmol of kcl is needed. (5) But in our case Kcl infusion neither corrected the hypokalemia nor produced any clinical improvement, rather IV corticosteroid was able to correct the hypokalemia with rapid reversal of muscle weakness. Probable explanation might be inhibition of peripheral conversion of T4(Levothyroxine) to T3(tri-iodothyronine) ,consequence loss of thyroid hormone mediated stimulation of the pump resulting in inhibition of Na+/k+ ATPase pump driven intracellular shift of K+, producing correction of hypokalemia as well as improvement in muscle weakness.

Even high dose of oral propranolol [3-4mg/kg] alone has been reported to rapidly abort the paralysis .(9)

Definitive management of thyrotoxic hypokalemic palsy includes restoration of euthyroid state for prevention of future attacks of the paralysis either with radioiodine ablation or with thyroidectomy. In our case patient was treated with Antithyroid drug (methimazole).However, the use of antithyroid drugs lead to relapse in 56%patients within 7 months. (10)

CONCLUSION

Thyrotoxic hypokalemic palsy can be the 1st manifestation of thyrotoxicosis & it can be life threatening as complications like atrial fibrillation due to excess circulating thyroid hormones, acute hypercapnic respiratory failure, colonic pseudo obstruction secondary to hypokalemia may occur. Hence it is essential to diagnose & treat thyrotoxic hypokalemic palsy in time.

REFERENCES :

1. Okinaka S, Shizume K, Iino S, Watanabe A, Irie M, Noguch A. Kuma S, kuma K et al.The association of periodic paralysis and hyperthyroidism in Japan. J Clin Endocrinol Metab. 1957 Dec; 17(12):1454-9.

[PubMed] [Ref list

2. Falhammar H, Thorén M, Calissendorff J Thyrotoxic periodic paralysis: clinical and molecular aspects. Endocrine. 2013 Apr; 43(2):274-84. [PubMed] [Ref list]

3. Kung AW: Clinical review: Thyrotoxic periodic paralysis: a diagnostic challenge. J Clin Endocrinol Metab. 2006 Jul; 91(7):2490-5. [PubMed] [Ref list]

4. Garla VV, Gunturu M, Kowuru KR, Salim SA. Thyrotoxic periodic paralysis: case report and review of the literature. Electron Physician. 2018 Aug 25;10(8):7174–9. doi: 10.19082/7174. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list

6. Meseeha M, Parsamehr B, Kissell K, Attia M. Thyrotoxic periodic paralysis: a case study and review of the literature. J Community Hosp Intern Med Perspect. 2017 Jun 6;7(2):103–6. doi: 10.1080/20009666.2017.1316906. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list]

7. Majhi S, Mehta KD, Rohil V. Thyrotoxic hypokalaemic periodic paralysis in a man from Nepal. BMJ Case Rep. 2009; 2009:pii: bcr05.2009.1836. doi:101136/bcr.05.2009.1836. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list]

8. Tachamo N, Lohani S, Nazir S, Juliano N. Paralysis that easily reverses: a case of thyrotoxic periodic paralysis. BMJ Case Rep. 2017 Jan 30;2017:pii: bcr2016218951. doi: 10.1136/bcr-2016-218951. [PMC free article] [PubMed] [CrossRef] [Google Scholar] [Ref list]

Lin SH, Lin YF

Am J Kidney Dis. 2001 Mar; 37(3):620-3.

[PubMed] [Ref list]

10. Abbas MT, Khan FY, Errayes M, Baidaa AD, Haleem AH. Thyrotoxic periodic paralysis admitted to the medical department in Qatar. Neth J Med. 2008;66(9):384–

8. [PubMed] [Google Scholar]

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Case Report

TETRALOGY OF FALLOT AND HYPOTHYROIDISM

Kamalakanta Sahoo[1], Bipin Kishore Kullu[2]

ABSTRACT

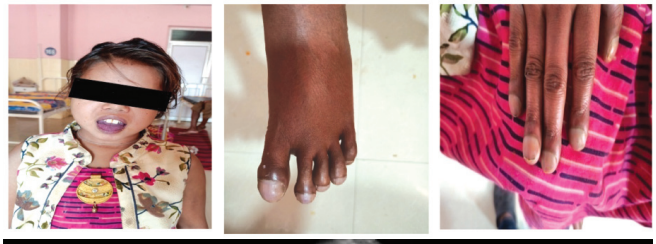
We report a case of an young female presenting with one episode of GTCS and having Tetralogy of Fallot,hypothyroidism,other features suggestive of DiGeorge syndrome.

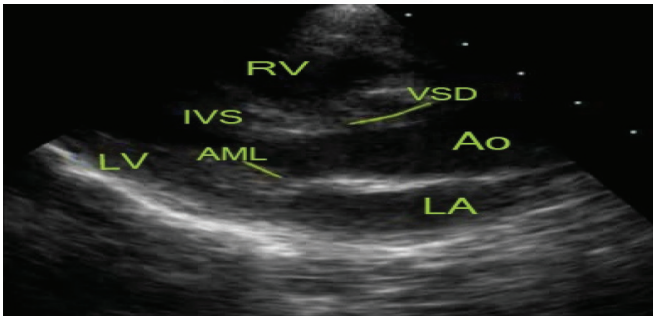
INTRODUCTION

Patients with congenital heart disease (CHD) have higher prevalence of thyroid dysfunction due to embryonic and genetic coexistence[1]. CHD patients have an increased risk for both congenital hypothyroidism (10-fold higher) and acquired mild hypothyroidism (3-fold higher). Unrecognized mild hypothyroidism may negatively affect the outcome of CHD, suggesting that thyroid function should be repeatedly checked. Thyroid autoimmunity and 22q11.2 micro-deletions account for small percentage of these cases, and still unknown mechanisms underline such a strong association[2].

CASE REPORT

An 18-year-old female girl was brought to the emergency department of VIMSAR, Burla after experiencing a generalized tonic-clonic seizure. The seizure lasted for 10 minutes that began while she was brushing teeth. Her family did not report any prodromal complaints or symptoms. There was no history of seizures in the past. The patient did not attain menarche. On examination, she had short stature (Height-147cm), facial dysmorphism, central cyanosis, clubbing, non-pitting edema, mental retardation. Her vital parameters were stable. Secondary sexual characteristics were poorly developed. Investigations shows polycythemia (RBC-9.04millions/mm^3,Hb19.9g/dl), hypocalcemia(0.5mg/dl),hypothyroidism(FT3-1.56pg/ml,FT4-0.67ng/dl,TSH-37.41uIU/ml).CT scan of brain showed cerebritis. Echocardiography findings were large non-restrictive VSD, overriding of aorta , pulmonary stenosis, right to left shunt and good LV function-features of Tetralogy of Fallot.





DISCUSSION

The clinical picture is highly suggestive of DiGeorge syndrome and needs chromosomal study. Physicians should recognize the association in adults presenting with any combinations of hypothyroidism, cardiac defects, hypocalcaemia and neuropsychiatric disorders. Pathognomonic facial dysmorphism or short stature can be the key to diagnosis. The first description in the 1960s in children with DiGeorge syndrome presented with the clinical triad of immunodeficiency, hypoparathyroidism and congenital heart disease. The syndrome is now known to have a heterogeneous presentation that includes multiple additional congenital anomalies and later-onset conditions; such as palatal, gastrointestinal and renal abnormalities, autoimmune disease, variable cognitive delays, behavioral abnormalities and psychiatric illness. Cardiovascular abnormalities become evident in the prenatal or neonatal

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period and are often the initial manifestation that leads to diagnosis[3]. Most abnormalities are cono-truncal heart defects — defined as malformations of the outflow tract — and include Tetralogy of Fallot (with or without pulmonary atresia), truncus arteriosus, interrupted aortic arch type B (between the left carotid and the left subclavian arteries) and ventricular septal defect. Anomalies of the aortic arch and/or of the pulmonary arteries may occur as isolated entities (~40%) or in association with cono-truncal defects (~60%)[4], contributing to the relative specificity of the cardiovascular patterns of DiGeorge syndrome[5-7].Aortic arch anomalies most frequently include a right-sided or a double aortic arch with or without aberrant subclavian arteries sometimes resulting in a vascular ring (~13%)[8]. Pulmonary artery anomalies include diffuse hypoplasia and discontinuity with or without major aorto-pulmonary collateral arteries. Overall, CHD represents the main cause of mortality (~87%)[9-10]in children with 22q11.2DS. Lack of recognition of the condition and/or lack of familiarity with genetic testing methods, together with the wide variability of clinical presentation, delays diagnosis. Early diagnosis, preferably prenatally or neonatally, could improve outcomes, thus stressing the importance of universal screening. Management requires a multidisciplinary approach.

REFERENCES

1. Lerner RK, Gruber N, Pollak U. Congenital Heart Disease and Thyroid Dysfunction: Combination, Association, and Implication. World J Pediatr Congenit Heart Surg. 2019 Sep;10(5):604-615. doi: 10.1177/2150135119857704. PMID: 31496400.
2. E. Passeri, M. Frigerio, T. De Filippis, R. Valaperta, P. Capelli, E. Costa, L. Fugazzola, F. Marelli, P. Porazzi, C. Arcidiacono, M. Carminati, B. Ambrosi, L. Persani, S. Corbetta, Increased Risk for Non-Autoimmune Hypothyroidism in Young Patients with Congenital Heart Defects, *The Journal of Clinical Endocrinology & Metabolism*, Volume 96, Issue 7, 1 July 2011, Pages E1115–E1119
3. McDonald-McGinn DM, Sullivan K. Chromosome 22q11.2 deletion syndrome (DiGeorge syndrome/ velocardiofacial syndrome). Medicine. 2011; 90:1–18.
4. Peyvandi S, et al. 22q11.2 deletions in patients with conotruncal defects: data from 1,610 consecutive cases. Pediatr. Cardiol. 2013; 34:1687–1694.
5. Keyte AL, Alonzo-Johnsen M, Hutson MR. Evolutionary and developmental origins of the cardiac neural crest: building a divided outflow tract. Birth Defects. Res. C Embryo Today. 2014; 102:309–323.
6. Neeb Z, Lajiness JD, Bolanis E, Conway SJ. Cardiac outflow tract anomalies. Wiley Interdiscip. Rev. Dev. Biol. 2013; 2:499–530.
7. Lewin MB, et al. A genetic etiology for interruption of the aortic arch type B. Am. J. Cardiol. 1997; 80:493–497.
8. McElhinney DB, McDonald-McGinn D, Zackai EH, Goldmuntz E. Cardiovascular anomalies in patients diagnosed with a chromosome 22q11 deletion beyond 6 months of age. Pediatrics. 2001; 108:E104.
9. McDonald-McGinn DM, et al. Phenotype of the 22q11.2 deletion in individuals identified through an affected relative: cast a wide FISHing net! Genet. Med. 2001; 3:23–29.
10. Costain G, Chow EW, Silversides CK, Bassett AS. Sex differences in reproductive fitness contribute to preferential maternal transmission of 22q11.2 deletions. J. Med. Genet. 2011; 48:819–824.

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Case Report

CEREBRAL VENOUS SINUS THROMBOSIS WITH MULTIFOCALINTRACEREBRAL AND SUB ARACHNOID HAEMORRHAGE IN A HIV INFECTED PATIENTWITH POLYCYTHEMIA

Ansuman Mishra, Rina Mohanty, Chitta Ranjan Khatua, Biswaranjan Prusty, Asisha Kumar Pattanayak, Pankaj Kumar Maravi, Putul Bara, Ajaya Kumar Biswal, Kamalakhya Samantaraya, Deepak Kumar Nayak, Snigdha Das, Utsav Kumar Senapati.

ABSTRACT

Cerebral venous sinus thrombosis (CVST) is an uncommon cause of stroke with the incidence of about 0.5 % of all strokes. It is caused by a wide range of etiologies such as hypercoagulable conditions (deficiency of protein C or protein S, factor V Leiden mutation, presence of anticardiolipin antibody, antithrombin gene mutation), polycythemia and HIV infection.Furthermore, the clinical presentation of CVST with hemorrhage constitutes a diagnostic challenge. Here we report such an unusual case in a young HIV infected male who presented with acute onset status epilepticus. Imaging revealed superior sagittal sinus thrombosis, right transverse sinus thrombosis, parietal lobe & occipital lobe hemorrhage with sub arachnoid hemorrhage and laboratory examination revealed polycythemia. He improved symptomatically during his hospital stay following conservative treatment. Our report highlights such a rare presentation of CVST with cerebral and subarachnoid hemorrhage(SAH) in HIV infected patient.

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is a rare variety of cerebrovascular disease that can occur at any age. It occurs when a blood clot forms in any of the venous sinuses of the brain. Approximately 5 people per million are affected by CVST and it accounts for approximately 0.5% of all stroke events. A wide range of etiologies have been implicated in the causation of CVST[1]. CVST may develop in relation to infections of the adjacent ear and paranasal sinuses or to bacterial meningitis[2]. More common are non-infectious venous occlusions resulting from hypercoagulable disorders like deficiency of protein C or protein S, factor V Leiden mutation, presence of anticardiolipin antibody and antithrombin gene mutation[3]. Some of the rare causes of CVST are Polycythemia, HIV infection and heavy alcohol intake.

Thrombosis is a serious complication of polycythemiaand can lead to death in up to 8.3% of patients as reported by Ferro et al. The most likely mechanisms are hyper viscosity and sluggish blood flow[4]. Due to increased hematocrit in polycythemia, there is increased blood viscosity and consequently an increased incidence of thrombotic phenomena. With increase in the plasma red cell volume in polycythemia, the viscosity of blood increases, leading to complications of the hypercoagulable state such as stroke, cortical sinus thrombosis, acute coronary syndrome, pulmonary embolism and deep vein thrombosis[5]. Polycythemia can be primary (caused mainly by mutation in the JAK2 gene) or secondary. Chronic hypoxia causes secondary polycythemia by increasing serum erythropoietin levels leading to excess production of erythrocytes from the bone marrow[6].

Although venous thrombotic events (VTEs) are frequent among HIVpatients, few cases of CVST have been reported. Mechanisms for the observed hypercoagulability in HIV infected patients are multifactorial and include the presence of antiphospholipid antibodies and deficiency of natural anticoagulants such as protein C, protein S, heparin cofactor II, and antithrombin[7].

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Approximately, one-third of CVST patients develops intracerebral hemorrhage (ICH). Presentation with ICHand SAH may be challenging and a common pitfall in diagnosing CVST. Identification of these patients is critical given that the pathophysiology underlying hemorrhage in such cases is distinct from other causes of ICH, and this has important treatment implications.Intracerebral hemorrhage in the context of CVST is generally associated with poorer outcomes compared to only CVST.

So, here we present a rare case of CVST presenting with ICH and SAH in a young HIV infected male patient with polycythemia.

Case:

A 25 year old male patient presented with repeated seizures and altered sensorium since last 6 hours. On thorough history taking it was revealed that he had been suffering from headache and intermittent vomiting since last 3 months. Headache was mild and generalized which gradually increased in severity. It was associated with dizziness and vomiting which was projectile and bilious in nature. There was no history of limb weakness or paresthesia. He denied any history of fever, trauma or any loss of consciousness, prior to this episode. He did not have any similar complaint in the past. He isnot a known case of hypertension or type 2 diabetes or dyslipidemia. He was taking some analgesics and antiemetics from a local health center for the past 3 months. There was no family history of strokes, sudden deaths or clotting disorders.

On examination, patient was confused, disoriented and afebrile. His vitals were normal. GCS was 9/15. His palpebral conjunctiva was dusky red colored and both of his bulbar conjunctiva was congested and edematous. There was no icterus or clubbing or lymphadenopathy or pedal edema. Pupils were well reactive to light and Fundoscopy did not reveal papilledema. There was no neck stiffness. Examinations of other systems were unremarkable.

On laboratory examination, Hemoglobin was 20.5 g/dl; hematocrit was 62.5%; total leucocyte count was 9520/µL; total platelet count was 2.18 X 10^5; ESR was 08 mm/hr; RBS was 150mg/dl; serum urea was 27mg/dl; serum creatinine was 1.1 mg/dl; serum total bilirubin was 4.3 mg/dl; serum unconjugated bilirubin was 0.8 mg/dl; SGOT was 98 U/L; SGPT was 76 U/L; ALP was 328 U/L; serum protein 8.4 g/dl; serum albumin was 4.3 g/dl. Serum erythropoietin, JAK2 V617F mutation and tests for different clotting factors could not be done.

His NCCT brain revealed hyperdensity in superior sagittal sinus, right transverse sinus and right sigmoid sinus with bilateral parietal lobe hemorrhage and subarachnoid hemorrhage. His MRI brain with venography revealed superior sagittal, right transverse and sigmoid sinus thrombosis with venous infarcts in bilateral parietal lobes.

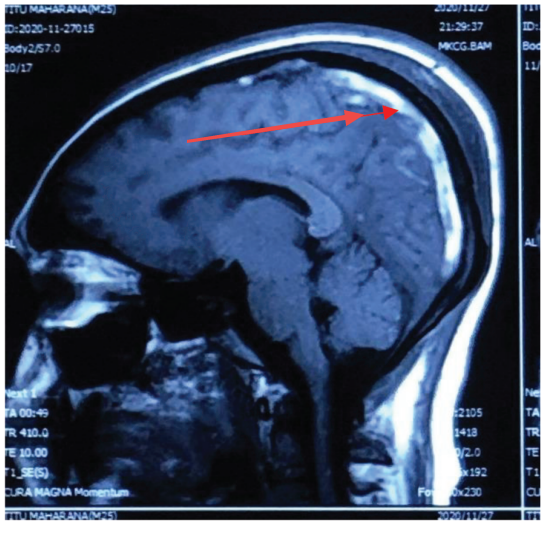

Figure1: MRI brain showing T1 hyperintensity along superior sagittal sinus, suggestive of thrombosis.(red arrow)

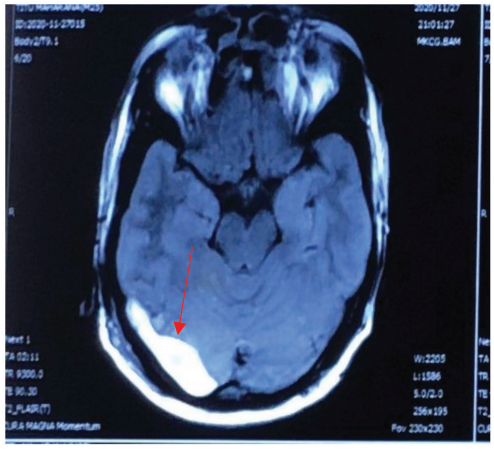

Figure2: MRI brain showing FLAIR hyperintensity along right transverse sinus, suggestive of thrombosis.(red arrow)

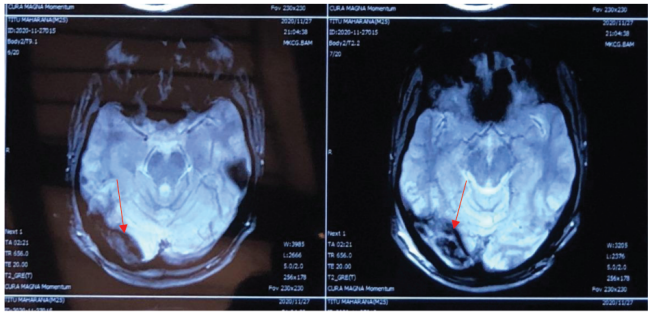

Figure3: MRI brain T2 wieghtedimage showing no flow along the right transverse sinus suggestive of thrombosis. (red arrows)

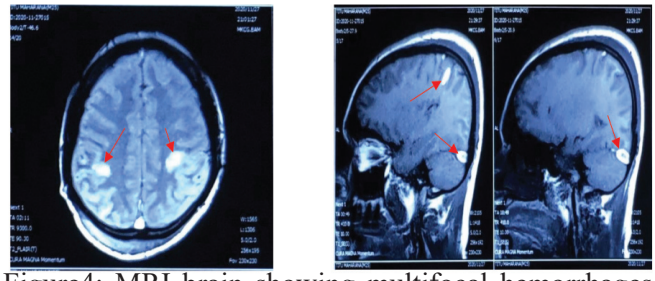

Figure4: MRI brain showing multifocal hemorrhages involving both parietal lobe and occipital lobe. (red arrows)

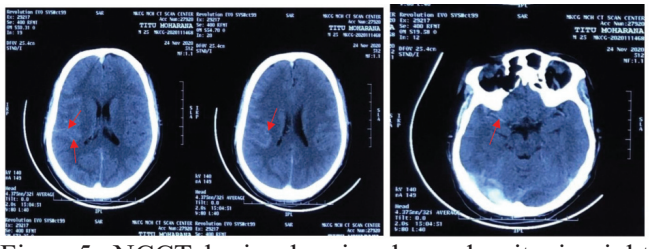

Figure5: NCCT brain showing hyperdensity in right cerebral cortical sulci and right sylvian fissure, suggestive of sub arachnoid hemorrhage. (red arrows)

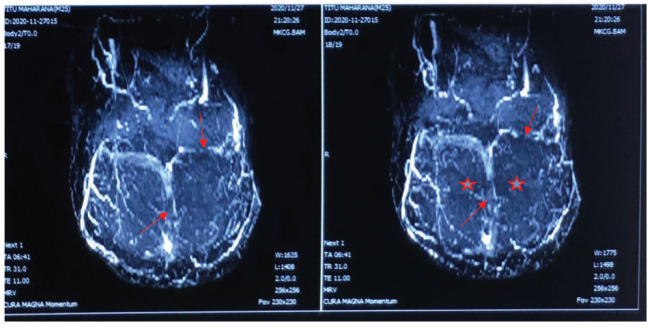

Figure6: MR Venography of brain showing occlusion of superior sagittal sinus, right transverse sinus (red arrows) with presence of bilateral venous infarcts in parietal lobe. (star marks)

During his hospital stay, he was treated symptomatically with anti convulsants, mannitol, iv fluids and antibiotics. Gradually he showed signs of improvement without any further convulsions.His headache and vomiting also decreased and soon after he was able to carry out his day to day activities without any hesitation.

He was not given any anticoagulants after careful consideration of the risks and benefits of anticoagulation in the presence of ICH and SAH.He was later started on HAART. After 2 weeks he was discharged with anti convulsants and HAART. He is doing well on follow up after 2 months.

DISCUSSION

The patient presented with repeated seizures and altered sensorium with ahistory of headache and intermittent vomiting for last 3 months. On conservative treatment patient's sensorium gradually improved. On routine investigations HIV infection and polycythemia was detected. We couldnot do specific tests to detect hypercoagulable disorders as these facilities were not available in our center.

Some studies have reported a high prevalence of antibodies against protein S among HIV infected patients, leading to significantly low protein S activity in about 31%-76% of patients. Although protein S deficiency is not correlated with HIV disease severity it appears that thrombosis is highly correlated with low CD4 counts, the presence of opportunistic infections, malignancies, or autoimmune disorders[8]. In the current case, the patient had a CD4 count of 426 and there was no evidence of any opportunistic infection.

Cerebral venous sinus thrombosis (CVST) is anuncommon cause of stroke in young males.In the causes of intracerebral hemorrhage (ICH), CVST is not mentioned in standard text[9]. Although CVST is observed with increasing frequency in daily practice only very few case reports have been made with ICH and SAH[2].In patients with CVST, spontaneous intracranial hemorrhage is accounted for 30% to 40%. CVST-induced ICH includes simple cerebral hemorrhage and venous infarction hemorrhage.In the present case, bleeding in the b/l occipital and parietal lobes was considered a venous infarction hemorrhage, which may be attributed to the blockade of venous

sinuses[1]. Since the patient did not have any history or risk factors that could explain ICH, we suppose the elevation of venous pressure leading to congestion and dilation of upstream capillaries and venules could have caused the hemorrhage.The distribution of SAH associated with CVST typically differs from the characteristic pattern of SAH with arterial origins.Specifically, when SAH is localized at the cerebral convexity and spares the basal cisterns and skull base, CVST should be considered.The exact mechanism of cortical CVST-induced SAH is unknown, although rupture of venous parenchymal hemorrhagic infarcts into the subarachnoid space is a possibility.This mechanism is potentially applicable to the current patient because signs of multifocal hemorrhage were observed on her CT scans. Another possible mechanism is venous hypertension and subsequent rupture of dilated, thin-walled, bridging subarachnoid cortical veins devoid of smooth muscle fibers[1].

Case reports have been done between CVST and Polycythemia; CVST in PLHA patients; CVST in binge alcohol drinkers; but as per our knowledge no such case reports have been made on both CVST and ICH with SAH in HIV infected patients with Polycythemia.

The 2011 American Heart Association/American Stroke Association guidelines recommend that once imaging confirms CVST, anticoagulation should be initiated regardless of whether ICH is evident at presentation. However, some patients with CVS and ICH present a quintessential example of the controversy regarding anticoagulation treatment, thereby emphasizing a careful consideration of the risks and benefits of anticoagulation in patients with combined CVST and ICH[1]. The patient in the present study had SAH and multifocal hemorrhage; therefore, the extension and severity of the bleed indicated that anticoagulation should not be initiated[4]. Fortunately, the patient experienced successful recovery after 2 weeks of treatment without any anticoagulation.

CONCLUSION:

CVST is anuncommon cause of stroke in young males and its presentation with multifocal ICH with SAH is highly uncommon.HIV infection and Polycythemia are rare causes for occurrence of the CVST. We conclude that CVST with multifocal ICH and SAH in the presence of HIV infection and polycythemia is very rare and our case is the first to present such an unusual combination. Early recognition and diagnosis of these allow the clinicians to commence appropriate treatment early and thereby reduce the associated morbidity.

Such rare causes could be easily missed while evaluating a patient of CVST. So patients presenting with CVST with or without hemorrhages should be evaluated for the presence of HIV infection and Polycythemia.

REFERENCES:

1. Cerebral venous sinus thrombosis presenting with multifocal intracerebral hemorrhage and subarachnoid hemorrhage A case report Jing Sun, MD, PhDa ,Zhidong He, MDb , Guangxian Nan, MD, PhDa.
2. Adam and Victor's Principles of Neurology, 11th edition.
3. Oda S, Shimoda M, Hoshikawa K, et al. Cortical subarachnoid hemorrhage caused by cerebral venous thrombosis. Neurol Med Chir (Tokyo) 2011;51:30–6.
4. Cerebral venous thrombosis and secondary polycythemia in a case of nephrotic syndrome; Shankar Prasad Nagaraju1, Manohar Bairy1, RavindraPrabhu Attur1, Charudutt Jayant Sambhaji2.
5. Malignant Cerebral Venous Sinus Thrombosis in Polycythemia; Rohan Mahale, Anish Mehta, Kiran Buddaraju, Abhinandan K. Shankar, and SrinivasaRangasetty.
6. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. Lancet Neurol. 2007;6:162–170. [PubMed] [Google Scholar].
7. Pedraza S, Rovira A, Saballs M, Capdevila J. Thrombosis of the superior longitudinal sinus in patients with AIDS - A review of the literature] Revista de neurologia. 1997;25(138):247. [PubMed] [Google Scholar].
8. Crum-Cianflone NF, Weekes J, Bavaro M. Review: thromboses among HIV-infected patients during the highly active antiretroviral therapy era. AIDS patient care and STDs. 2008;22(10):771–8. [PMC free article] [PubMed] [Google Scholar].
9. Intracerebral haemorrhage due to cerebral venous sinus thrombosis; J. Kalita, DM, V. Bansal, DM, U.K. Misra, DM, S.K. Jain, MD.

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Case Report

A RARE CASE OF ARTESUNATE RESISTANCE PLASMODIUM FALCIPARUM MALARIA IN ODISHA

Subrat Ku Dutta[1], PK Thatoi[2], Bhagyasree Panda[2], NR Mohanty[3], Abinash Swain[4], Samir Hota[4]

BACKGROUND

Odisha is endemic for severe falciparum malaria.According to WHO ACT (Artemisinin based combination therapy) is the gold standard therapy for severe falciparum malaria.Currently ACT resistance has been limited to Great Mekong sub-region i.e. Cambodia,Thailand,Myanmar,Vietnam,India –China border. In India very few cases of artesunate resistance have been reported in Odisha Andhra border in 2016.

2. CASE PRESENTATION

In Odisha we are reporting a case of artesunate resistance Plasmodium falciparum malaria which was being treated with Artesunate full dosage but didn't improve clinically; subsequently received Quinine sulfate 600mg and got improved clinically.

3. CONCLUSION

We thus highlighted the need of increase monitoring and surveillance to identify artemisin based combination therapy and resistance in other parts of India. It is also essential to ensure rationale use of existing antimalarial drugs so we can have novel weapon to fight against these parasitic disease without fail. We also emphasize the new research in this field to find out novel drugs in ACT resistance.

Key words-ACT; Plasmodium falciparum; Drug Resistant; Malaria.

Abbreviations used-ACT-Artemisinin based Combination Therapy, WHO-World Health Organisation, DCT-Direct Coombs Test, ICT-Indirect Coombs Test, USG-Ultra sonography, CBC-Complete Blood Count, DHFR-Dihydro folate reductase

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INTRODUCTION:

The emergence and spread of drug resistance malaria represents a considerable challenge in controlling malaria.[1]The World Health Organisation(WHO) recommends the use of artemisinin based combination therapy(ACT) against Plasmodium falciparum malaria to ensure high cure rates and prevent resistance against artemisinin compounds.(2)However resistance to artemisinin has emerged in Thai-Cambodian and Thai-Myanmar border.(3)There have been 2 case reports of artesunate resistance in India occurring in Kolkata and Mumbai.(4)

Now, the success of a new treatment policy is dependent on the adherence of health providers to the guideline and the compliance of patients with the recommended treatment. Equally, information on current provider and patient practices is needed to inform and improve future treatment policy [5]. But our recent report suggests the inappropriate treatment practices continue in spite of the availability of evidenced-based guidelines. Here we are reporting 1 case of artesunate resistance plasmodium falciparum malaria responded to Quinine.

CASE REPORT

An eighteen year old male from Jajpur, Odisha presented with fever with chills, rigor and altered sensorium for 2days without any episode of vomiting, convulsion. Patient has no similar episode in past. Patient has no past history of seizure disorder, diabetes, respiratory illness, and trauma. No family history of similar illness. Patient was not addicted to tobacco, alcohol or any illicit drugs. There was no history of drug allergy. The patient was on no medication.

On examination patient was febrile, temperature was 102degree Fahrenheit, heart rate 120 beats per min, blood pressure 130/80mm of hg and oxygen

saturation 94%while the patient was breathing ambient air; the body mass index (the weight in kilograms divided by square of the height in meters)was 24.Patient was drowsy but responding to painful stimulus. Cranial nerve examination was normal .Rest of physical examination was normal. Examination of respiratory, cardiovascular, GI system appears to be normal. All routine tests came normal shown in {Table 1}.Malaria parasite slide test came positive.(fig.1).Patient was put on Inj. Artesunate 2.4mg/kg Intravenous on 0hour,12hour,24hour and patient was put on empirical antibiotics Intravenous Ceftriaxone 1gram twice daily. On day 2,Patient sensorium didn't improve heartrate was 120beats per minute and blood pressure came as 90/50mm of hg so we presume it to be septic shock and order for serum Procalcitonin and change the antibiotic to IV Piperacillin+Tazobactum 4.5gm in 100ml normal saline thrice daily. .Patient sensorium improved a bit but remained confused so presuming it to be meningitis or encephalitis we performed lumbar puncture. The value of csf protein was 32mg/dl, sugar 56mg/dl, total cell count 3/hpf. Staining for mycobacterium came negative. Despite the treatment with Intravenous Artesunate patient remained febrile. Other reports like Urine culture sensitivity revealed no organism, Echocardiography came normal systolic and diastolic function, Ultrasound abdomen pelvis was normal. On Day 3 Patient's Complete blood count report came Hb as 7gm%.S ferritin came as >2000,DCT +ve, ICT-ve. On doing bone marrow biopsy increase no. of Reticulum cells studded with coarse pigment (malaria pigment)picture attached below

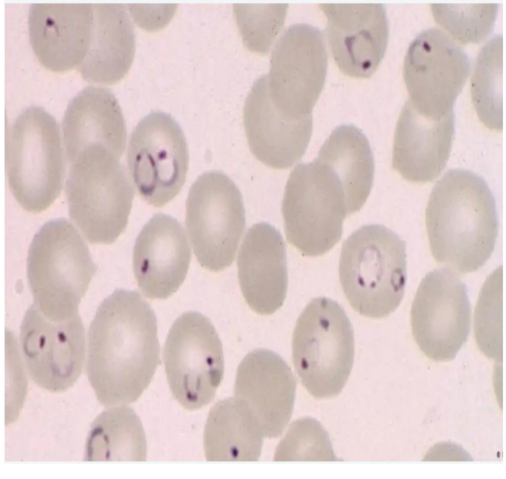

Figure 1.Slide of malaria parasite.

Image showing multiple ring shaped plasmodium falciparum malaria.

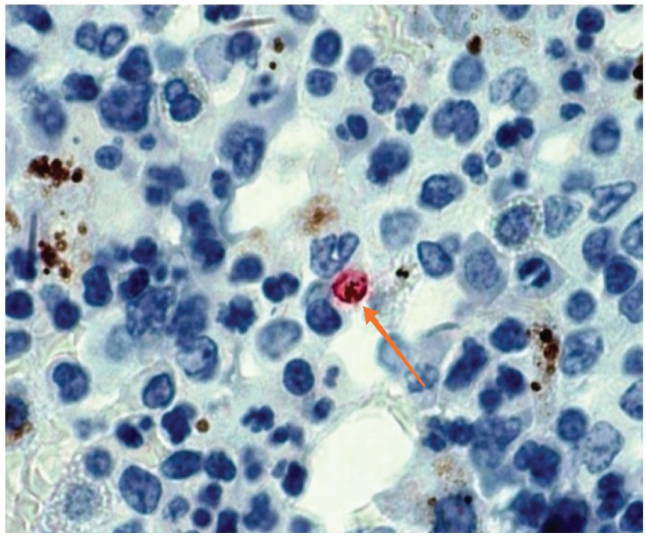

Figure 2. Bone marrow biopsy picture; arrow showing multiple reticulum studded with coarse pigment (hemozoin).

Table 1.Laboratory data*

Variables	Reference ranges adults	On admission	Day 3
Hematocrit(%)	36-46	36.7	48
Hemoglobin(g/dl)	12-16	10.8	7
White cell count(per microliter)	4500-11000	10,520	12400
Differential count(permicroliter)			
Neutrophils	1800-7700	9080	10800
Lymphocytes	1000-4800	700	1100
Monocytes	200-1200	700	980
Eosinophils	0-900	0	56
Platelet count (per microliter)	150,000-400,000	279,000	300,000
Prothrombin time(sec)	11.5-14.5	13.0	14
Serum ferritin(microgram/liter)	20-300	550	2000
Serum Sodium(milligram/decilitre)	135-145	137	142
Serum Pottasium(milligram/decilitre)	3.5-5	4.2	4.4
Glucose(milligram/decilitre)	<200	115	140
C reactive protein(milligram/decilitre)	<8	42	34
CSF cellcount(cells/hpf)	<5	3	
Protein	15-45	35	
Sugar	40-80	60	
Acid fast bacillus		absent	
Cryptococcal antigen		negative	

As patient's clinical signs and symptoms didn't improve after intravenous artesunate for 7 days. Hence we started Tab Quinine sulfate 600mg thrice daily after food. Patient's fever got improved after that and remained afebrile and got cured fully. Patient was being discharged after 14 days of hospitalization and asked for follow up.

This shows there is resistance of artesunate which responded to Tab Quinine Sulfate orally subsequently.

DISCUSSION

This is the first clinical case reported as artesunate resistance malaria in Odisha. A significant failure rate of artesunate (10%) has been observed in Odisha though no specific mutation has been observed in pfATPase6 gene.[6]In view of this above ,a thorough research on artesunate resistance should be done. Rationality of inclusion of Sulfadoxin and pyrimethamine with Artemesinin compounds raises a serious question as dhfr mutation is most common among isolates of plasmodium falciparum patients .The mutation is also found in C59R gene.[6]

Artemisinin compounds are highly potent, rapidly eliminated drugs and clear parasitemia most rapidly when compared to any other antimalarial.[7]In 2005 WHO recommended that artemisinin based combination be used as the 1st line therapy for severe falciparum malaria in malaria endemic countries.[1]In 2010 India revised its antimalarial policy and recommended ACT for all cases of falciparum malaria.

With widespread use of these drugs, Artemesinin resistance has emerged in Cambodia and other parts globally. It has also been seen in Southeast Asia due possible reasons being; widespread availability of artemisinin mono-therapies, poor quality drugs, unregulated use of antimalarial agents and unusual genetic structures of parasites.[8]Resistance to ACT should be suspected if there is no clinical or parasitological response after 72 hrs of commencing of therapy in the absence of vomiting or diarrhoea.[2]This report emphasizes the need for increased monitoring and surveillance to identify artemisinin resistance in other parts of India also. There are no new antimalarial drugs available to us for several years. Hence it is essential to ensure rationale use of the few remaining effective drugs[4].

REFERENCES

1. Guidelines for the Treatment of Malaria. 2nd ed. World Health Organization: South-East Asia; 2010. ISBN: 9789241547925
2. World Health Organisation. Global Malaria Programme. Geneva: 2016. Artemisinin and artemisinin-based combination therapy resistance
3. Dondorp AM, Nosten F, Yi P, Das D, Phyo AP, Tarning J, et al. Artemisinin resistance in Plasmodium falciparum malaria. N Engl J Med. 2009;361:455–67
4. . Gogtay NJ, Kadam VS, Karnad DR, Kanbur A, Kamtekar KD, Kshirsagar NA. Probable resistance to parenteral artemether in Plasmodium falciparum: Case reports from Mumbai (Bombay), India. Ann Trop Med Parasitol. 2000;94:519–20
5. . Wasunna B, Zurovac D, Goodman CA, Snow RW (2008) Why don't health workers prescribe ACT? a qualitative study of factors affecting the prescription of artemether-lumefantrine. Malar J 7:29.
6. Das S, Chakraborty SP, Tripathy S, Hati AK, Roy S (2012) Association between prevalence of pyrimethamine resistance and double mutation in pfdhfr gene in West Bengal, India. Asian Pacific J Trop Dis 5: 31-35.
7. Operational Manual for Malaria Elimination in India. New Delhi: 2016. Directorate of National Vector Borne Disease Control Programme Directorate General of Health Services Ministry of Health & Family Welfare Government of India.
8. Bhattacharyya N, Mukherjee H, Bose D, Roy S, Das S, Tripathy S, et al. Clinical case of artesunate resistant Plasmodium falciparum malaria in Kolkata: A first report. J Trop Dis. 2014;2:12

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Case Report

COMPLETE HEART BLOCK IN RHEUMATOID ARTHRITIS

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INTRODUCTION

Rheumatoid arthritis (RA) is a symmetric, inflammatory, peripheral polyarthritis of unknown etiology.Extraarticular manifestations involving skin, eye, lungs, heart, and others occurs in approximately 40 percent of patients with rheumatoid arthritis (RA) over the course of the disease.

Cardiac involvements in RA include pericarditis, valvulitis, myocarditis. Clinically apparent pericarditis and myocarditis are uncommon disorders in patients with RA.There is an increased risk of coronary artery disease and heart failure (HF) in patients with rheumatoid arthritis (RA), and there may be an increased risk of atrial fibrillation (AF).

Atrioventricular block is rare in RA, usually complete, and does not respond to anti-inflammatory and immunosuppressive therapy.

Here we report a case of Complete atrioventricular block in RA.

CASE REPORT

We present a male patient aged 63 years who came to the emergency department with the complains of one episode of syncope. He was managed in the local hospital and subsequently referred to this hospital.

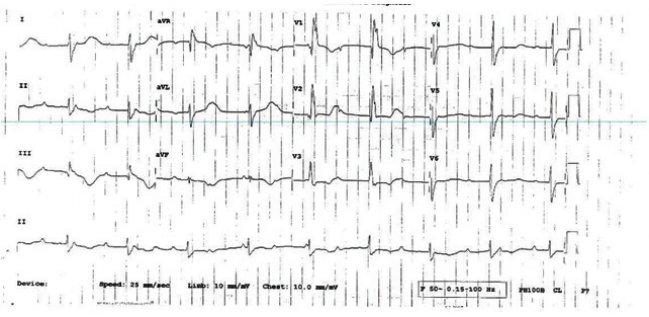
He gave h/o pain & swelling of multiple small & big joints of both upper & lower limbs since 2 years, and was being treated with NSAIDs off &on.

Patient was conscious, well oriented, Obese, Pulse- 53/min, regular, BP- 150/70 mm of Hg, SpO2- 96% in room air, Heart sounds normal, no murmur, Chest- Bilateral vesicular breath sound, Abdomen – soft & non tender, no hepatosplenomegaly, CNS- No focal neurological deficit.

MSK examination revealed – Early morning stiffness - > 30 minutes, Tender joint count – 12/28, Swollen joint count- 3/28, No deformity.

Investigations revealed- CBC:TLC-11,500, Hb-11.6 gm, TPC-1.54 lcs,ESR: 80 mm, Urea: 33.6 mg, Creatinine: 1.18 mg,LFT: Bilirubin(T) 0.56 mg, AST-66.3 U , ALT- 51.4 U ,ALP- 205 U , Albumin – 3.5 gm, PT/INR: 1.04, APTT: 24.4 sec, Urine R &M: No abnormality, TSH: 1.48 mIU,HBsAg : Negative , Anti HCV: Negative , HIV: Negative , RF: 114.82 IU , Anti CCP antibody:111.30 U

ECG: Complete Atrioventricular block,



ECHO: No RWMA, No MR, EF- 60% ,

CAG: Normal Coronaries

USG abdomen: Normal Study, CXR PA View: No abnormality detected

X Ray of both hands AP did not reveal any erosion.

Diagnosis:

Rheumatoid Arthritis(Seropositive)with High Disease Activity

(DAS 28 ESR- 6.3) , Complete Heat Block

Sr. Consultant in Int. Medicine & Rheuamtology,
LVPEI & CARE, Bhubaneswar

TREATMENT:

Treated with Permanent Pacemaker (DDDR-MRIC),csDMARD& low dose oral steroid. Patient was followed intermittently. Disease activity came down and was doing well with DMARDS and PPI.

DISCUSSION:

Approximate incidence of complete heart block in RA mentioned by Ahern et al. is 1 in 1000 patients of RA, whereas Raskar and Cosh found the incidence of 1 in 1600 patients.

Primary infiltration of the AV node or other conducting tissue by mononuclear cells or rheumatoid granulomas can be revealed in patients with conduction disorders and RA. Other potential mechanisms are vasculitis of the arterial supply to conductive tissue, hemorrhage into a rheumatoid nodule or extension of an inflammatory lesion from the aortic or mitral valve. Rarely, these lesions may be due to amyloid deposition.

Usually,AV block is detected in long standing erosive and high disease activity RA , as published in literature case reports.

In our case the duration of polyarthritis was only two years, nonerosive and the patient presented with Complete heart block before he was diagnosed as RA.

REFERENCES:

1. Charcot JL. Clinical Lectures on Senile and Chronic Diseases. Vol. 95. London: Sydenham Society; 1881. p. 172-5.
2. Handforth CP, Woodbury JF. Cardiovascular manifestations of rheumatoid arthritis. Can Med Assoc J 1959;80:86-90.
3. Ahern M, Lever JV, Cosh J. Complete heart block in rheumatoid arthritis. Ann Rheum Dis 1983;42:389-97
4. Seferoviæ PM, Ristiæ AD, Maksimoviæ R, Simeunoviæ DS, RistiæGG, Radovanoviæ G, *et al*. Cardiac arrhythmias and conductiondisturbances in autoimmune rheumatic diseases. Rheumatology(Oxford) 2006;45 Suppl 4:iv39-42.
5. Owlia MB, Mostafavi Pour Manshadi SM, Naderi N. Cardiac manifestations of rheumatological conditions: A narrative review. ISRN Rheumatol2012;2012:463620. 7. Maisch B, Ristiæ AD. Immunological basis of the cardiac conduction and rhythm disorders. Eur Heart J 2001;22:813-24. 8. Cimaz R, Duquesne A. Neonatal lupus syndromes. Arch Pediatr2006;13:473-8.
6. Maisch B, Ristiæ AD. Immunological basis of the cardiac conduction and rhythm disorders. Eur Heart J 2001;22:813-24. 8. Cimaz R, Duquesne A. Neonatal lupus syndromes. Arch Pediatr2006;13:473-8
7. Cimaz R, Duquesne A. Neonatal lupus syndromes. Arch Pediatr2006;13:473-8.

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Case Report

DYSAUTONEMIA IN HIV

Debabrata Rath[1], Umashankar Mishra[2], Abinash Mishra[3]

ABSTRACT

Autonomic dysfunction in HIV patients is an uncommon problem. The exact mechanism by which HIV modulates autonomic function is unknown. It is hypothesized that HIV promotes a sympathetic imbalance, contributing to a virus-friendly Th1-biased immune environment. Here we found a case of 30 yr age male patient complaining of repeated blackout & fall since 2 months. All systemic examinations were normal. During examinations related to autonomic dysfunction it was found that the patient had severe autonomic dysfunction. Under high salt diet, fludrocotisone, pyridostigmine therapy patient condition gradually improved. Our report highlights such rare case of autonomic dysfunction in HIV patients.

INTRODUCTION:

Autonomic neuropathy is unusual amongst the neuropathies. HIV can cause neuropathies however dysautonemia is a rare phenomenon & very few cases have been reported in world till date. dysautonomia can result in symptoms such as orthostatic intolerance, gastrointestinal abnormalities, and male sexual dysfunction, which substantially impacts quality of life [1]. human immunodeficiency virus-infected individuals with symptomatic autonomic neuropathy have increased morbidity [2]. in the pre-highly active antiretroviral therapy (art) era, ad in patients infected with hiv was associated with longer duration of infection, uncontrolled hiv viremia, and exposure to older antiretroviral medications [3].

CASE STUDY

Young adult of 30 years with known HIV presented with repeated Blackout & fall since 2 months. Patient was apparently alright 2 months back to start with he developed repeated blackouts and falls on standing which was aggravated with any movements. It was relieved on supine position and at rest. There was no history of excess water loss as diarrhoea, vomiting, in urination or sweating. Patient was taking ART since 2 months. During the hospital stay patient was taking Fludricortisone and Pyridostigmine**.**

General examination: No pallor, icterus, cyanosis, clubbing, lymphadenopathy, edema. JVP not raised, no thyromegaly.

***Systemic examination*:** Cardiovascular system, respiratory system, gastro intestinal system and central nervous system examination reveals no abnormality.

Methods (Tests for autonomic system)

Blood pressure at different posture was routinely taken before starting therapy. Different tests for ANS were done.

		Blood Pressure (mm of HG)	Pulse (beats/min
SLEEPING		100/70	72
SITTING		88/62	74
STANDING	10 SEC	60/40	65
	20 SEC	Not Recordable	Not Recordable

Head Up Tilt Test

✓ Tilt test is done a specialized table by passive tilting.

✓ Supine BP 96/70

✓ Tilt at an angle of 60 degree

- o At 1 min 88/70
- o At 15 min 82/72
- o At 30 min 80/68

• There was a blackout after 20 seconds of standing.

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Valsalva Maneuver

Phase	BP	Pulse
1	72/50	73
2	80/48	70
3	70/50	75
4	70/62	81

Cold pressor test

• Hand immersed in COLD water. (Due to pain and temperature fibres there should be sympathetic activation and increase in BP and HR). BP was 92/70.

Investigations

• All routine tests like CBC, RFT, LFT, FBS were normal. ECG Of different posture was done. There was Strain pattern with T inversion seen on standing position which showed probable decrease in blood supply to heart. There was erectile dysfunction in the patient for 2months. To rule out other causes of dysautonemia other investigations were done. MRI of brain and spinal cord, Thyroid function test, Nerve conduction study, echocardiography, serum cortisol assay were done. All were normal.

Patient was kept under high salt diet, fludrocotisone, pyrigostigmine therapy and there wass improvement on BP while standing comparing previous episodes.

Discussion:

The case clearly shows features of early ANS involvement HIV. The exact mechanism by which HIV modulates autonomic function is unknown [4]. It is hypothesized that HIV promotes a sympathetic imbalance, contributing to a virus-friendly Th1-biased immune environment [5]. Clinically, AD in HIV-positive patients is consistent as a spectrum of HIV-associated neuropathies [6, 7]. Pathologic studies involving the jejunal mucosa of HIV-positive patients demonstrates that damage to the autonomic nerve fibres occurs early in the course of HIV infection [7]. Dysautonemia in HIV is very rare and few cases reported. Clinical manifestations of ANS are diverse and complex. HIV patient though have multiple neuropathic features but autonomic dysfunction is rare, severe dysautonemia of this type is very rare as we have presented here. Jessica Robinson-Papp described in 2013 that autonomic dysfunction is common in HIV, often symptomatic. Pradheep C Mathew, Shishir Nagesh Duble in 2014 found that Cardiac autonomic nervous dysfunction is a common showing symptom such as bowel and bladder dysfunction, impotence, syncope and sweating abnormalities. M Mahesh , M Shashidhara in 2016 published that there was significant autonomic nervous system dysfunction in both HIV without AIDS and HIV with AIDS. Similar to the studies given above we also found similar findings in this patient. Hence All HIV should be thoroughly examined with BP & valsalva to Rule out dysautonemia which is sometimes fatal.

CONCLUSION

HIV associated Autonomic Dysfunction is very rare among HIV patients. The patients having this dysfunction have high morbity and mortality. So autonomic dysfunction should be diagnosed as early as possible in HIV patients. All HIV patients should be screened for autonomic dysfunction.

REFERENCES

1. Montaner JS, Lima VD, Harrigan PR et al. . Expansion of HAART coverage is associated with sustained decreases in HIV/AIDS morbidity, mortality and HIV transmission: the “HIV Treatment as Prevention” experience in a Canadian setting. PLoS One 2014; 9:e87872.
2. Robinson-Papp J, Sharma SK. Autonomic neuropathy in HIV is unrecognized and associated with medical morbidity. AIDS Patient Care STDS 2013; 27:539–43.
3. Lee KA, Gay C, Portillo CJ et al. . Symptom experience in HIV-infected adults: a function of demographic and clinical characteristics. J Pain Symptom Manage 2009; 38:882–93.
4. Rogers RC, Viard E, Hermann GE. CXCL12 sensitizes vago-vagal reflex neurons in the dorsal medulla. Brain Res2013; 1492:46–52.
5. Yun AJ, Lee PY, Bazar KA. Modulation of autonomic balance by tumors and viruses. Med Hypotheses 2004; 63:344–51.
6. Boger MS, Hulgan T, Haas DW et al. . Measures of small-fiber neuropathy in HIV infection. Auton Neurosci2012; 169:56–61.
7. Griffin GE, Miller A, Batman P et al. . Damage to jejunal intrinsic autonomic nerves in HIV infection. AIDS 1988; 2:379–82.

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Case Report

NUMB CHIN SYNDROME IN SICKLE CELL DISEASE; A RARE CASE REPORT

Deepak Kumar[1], Pradeep Kumar Mohanty[2], Sagnika Tripathy[3], Deepak Kumar Panigraphy[4], Baldev Prasad Kar[5], Jitendra Panigraphy

ABSTRACT

Numb chin syndrome (NCS) is a rare but important clinical entity. NCS presents as anaesthesia or paraesthesia over the chin along the distribution of inferior alveolar nerve and its mental and incisor branch.NCS has spontaneous onset with no history of trauma, infection or obvious odontogenic cause and usually associated with malignancy (1). Here we report a case of sickle cell disease with vaso-occlusive crisis with NCS.

INTRODUCTION

Globally, sickle cell disease (SCD) is one of the mostcommonhaemoglobinopathy resulting from a point mutation at codon no 6 of beta globin gene leading altered structure of hemoglobin and resultant sickling of red cell in hypoxic condition. (2)

Due to HbS polymerisation there is formation of long fibers that increase cellular rigidity and distort the erythrocyte membrane, leading to erythrocyte sickling.Vaso-occlusive crisis is the commonest of all sickle cell crisis and is characterised by ischemicpain, tenderness in long bones and joints, fever, and tachycardia.Numb chin syndrome is also called as mental neuropathy is due to damage of inferior alveolar nerve or its branch, mental nerve and is mostly associated with cancer.NCS is a very uncommon neuropathy associated with SCD.In india very few no of cases have been reported till now.

CASE REPORT

A31-year-old man withhistory of sickle cell disease presented to the emergency department for severe pain in the limbs suggestive of vaso-occlusive painful crisis with subsequent feeling of numbness of chin and lower lip. In general examination BP was 124/78 mm of Hg, Pulse rate was 86/min, respiratory rate was 24/min. There was no cervical lymphadenopathy or visible neck massfound on examination. Oral examination revealed normal soft tissue and salivary glands. On examination there is absence of pain, touch and temperature sensation over the skin of the chin and lower lip. Sensation over rest of the facial skin was intact. There was no restriction of movement around temporo-mandibular joint of both sides. InCardiovascular system examinationeverything found to normal except tachycardia, inGastrointestinal systemexamination there was presence of splenomegaly, inrespiratory system examination there wasmild tachypnoea with rate of 32 per minute. Laboratory tests revealed Hemoglobin-6.2 gm/dl, level of Serum LDH-1548 IU/LESR- 98mm/1 hour. All radiological imaging such as X-ray of skull and chest PA view were within normal limits. Ultrasound of abdomen and pelvis showed splenomegaly.

DISCUSSION

NCS is usually unilateral and is associated with altered sensation along the anatomical boundary of the inferior alveolar nerve, mostly in the mental division. The inferior alveolar nervewhich is branch of mandibular nerve follows a long course in the mandibular canal, under the teeth roots, and is prone to compression and ischemia when this narrow canal is infiltrated. It terminates by dividing into incisive branch and mental nerve. NCS is not associated with any motor or taste disturbance. It may have local odontogenic causes such as infection, trauma, local anaesthesia for dental procedures and odontogenic neoplasms. (3)It may also be a consequence of radiotherapy to the mandible causing direct damage to the nerve or by ischaemia (4).It may be caused by compression of the nerve through the infratemporal fossa and skull base or

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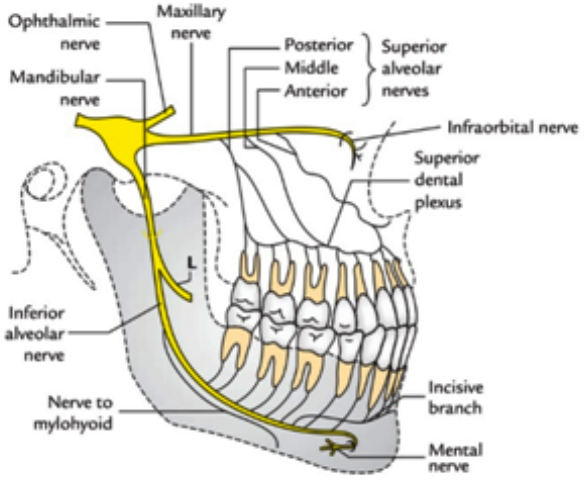
compression of the nerve root through its intracranial course by direct causes or indirectly through a rise in intracranial pressure. Selective demyelination of the nerve in MS can cause NCS as an isolated neurological sign.

Other non-malignant causes of a numb chin are -sickle cell disease,mandibular trauma, odontogenicinfection, mandibulareosteomyelitis, severe mandibularatrophy, benign sensory trigeminalneuropathy, radiotherapy, post-vaccination vasculitis, connective tissue vasculitis, systemicamyloidosis, sarcoidosis, HIV, diabetes mellitus,syphilis,vertebrobasilar insufficiency,lymes disease.

Konotey-Ahulu were firstfisrst to report mental neuropathyin 5 patients,out of wich 3 patient were having hemoglobin C disease and 2 withsickle cell disease.[5]

Numb chin syndrome in sickle cell crisis is is due to infarction of the inferior alveolar nerve at or near the mental foramen where the mental nerve has a bending course before the exit from canal. [6]Other possibilities are osteomyelitis of the mandible has also been shown as a cause of numb chin syndrome in a patient with sickle cell disease. [7]



CONCLUSION

Though raresickle cell disease should be considered one of the differential diagnosis of numb chin syndrome or mental neuropathy.

Conflicts of interest-The author declares no conflicts of interest.

REFERENCE

1. Laurencet F M, Anchisi S, Tullen E, Dietrich P Y. Mental neuropathy: report of five cases and review of the literature. *Crit Rev Oncol Hematol* 2000; **34**: 71–79.
2. Serjeant GR, Serjeant BE. Nomenclature and genetics of sickle cell disease, In Sickle Cell Disease, Oxford Univ Press, 3rd ed. 2001:31–38.
3. Mallefert J F, Gazet-Maillefert M P, Tavernier C, Farge P. Numb chin syndrome. *Joint Bone Spine* 2000; **67**: 86–93.
4. Lossos A, Siegal T. Numb chin syndrome in cancer patients: etiology, response to treatment and prognostic significance. *Neurology* 1992; **42**: 1181–1184.
5. Konotey-Ahulu FI. Mental-nerve neuropathy: a complication of sickle cell crisis. Lancet. 1972;2(7773):388.
6. Kirson LE, TomaroAJ.Mental nerve paresthesia secondary to sicklecell crisis. Oral Surg Oral Med Oral Pathol. 1979;48(6):509-512.
7. Patton LL, Brahim JS, Travis WD. Mandibular osteomyelitis in a patient with sickle cell anemia: report of case. J Am Dent Assoc.1990;121(5):602-604.

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Case Report

PARAPHENYLENEDIAMINE POISONING IN INDUSTRIAL HOSPITAL : AN EXPERIENCE

Merry Puspa Anita Lakra, Suprava Manjari Pradhan, Chinni Krishna Kasi Rajyabardhan Pattnaik, Sanjib Mohanty

ABSTRACT

Suicidal poisoning is more common in India because poisons are easily available and inexpensive. Paraphenylenediamine (PPD) poisoning is rare in our area. PPD, an important component of hair dye, causes extensive angioedema of the upper airway leading to severe respiratory distress. It also causes rhabdomyolysis and acute renal failure. There is no specific antidote for it and the main mode of treatment is supportive. We report three cases of PPD poisoning in our hospital in the last 8 years. Out of these, two survived due to timely reporting and adequate management in the form of early intubation, fluid therapy and haemodialysis.

Keywords - *PPD poisoning, airway protection, fluid therapy, dialysis*

INTRODUCTION

Suicidal poisoning by ingestion of pesticides, certain plant products and different drugs is common in this region of India. We encountered around 531 cases of poisoning in last 5 years (unpublished data). Most commonly seen in our setting is poisoning with organophosphorus compounds, mainly due to consumption by the oral route. Cases with poisoning via the parenteral route[1] or exposure of body surface while spraying has also been seen. Hair dye is inexpensive, easily available, and accessible in the community. The toxic components in hair dye include PPD (paraphenylenediamine), propylene glycol, resorcinol, sodium ethylene diamine tetraacetic acid and preservatives, of which the main poison is PPD [C6H4(NH2)2].[2] PPD is mainly absorbed via the mucous membrane of the digestive tract on ingestion. 100 ml of hair dye contains 12g of PPD, and the toxic dose is >3g and the fatal dose is e”7g via oral route.[3] Intoxication presents with laryngeal oedema, rhabdomyolysis and eventually renal failure, neurotoxicity, and acute toxic hepatitis.

We present 3 cases that were encountered in the last 8 years. Out of these 2 survived due to timely reporting and adequate management.

Case 1

A 17-year-old female presented with swelling of tongue and difficulty breathing for 2 hours. The attendant gave a history of ingestion of approximately half a bottle of hair dye containing PPD 6 hours prior to admission. As she was having severe respiratory distress, respiratory rate 25/min with oxygen saturation (SPO2) of 82%, immediate endotracheal intubation was done to protect the airways and fluid therapy with administration of corticosteroids and adrenaline. Her laryngeal and tongue oedema reduced on day 4 following which she was successfully extubated. Subsequently, urine output decreased and lab investigations revealed increasing levels of blood urea nitrogen (BUN) and serum creatinine. She underwent haemodialysis on day 3 that continued for 3 hours/day until day 10. Thereafter she had adequate urination with a gradual decrease in BUN and serum creatinine. She was discharged on day 20.

Day	Urea (mg %)	Creatinine (mg %)	Na+ (mmol/L)	K+ (mmol/L)	Bilirubin (mg %)	ALT (U/L)	CPK (IU/L)	Hemoglobin (gm %)	TLC (mm^3)
1	11	0.9	137	3.1	0.6	130	13668	15	25200
3	95	4.1	140	6.8					
5	96	5	128	3.4					
10	66	3.5	130	3.6					

Case 2

A 42-year-old male presented with severe facial oedema with increased salivation and breathing difficulty for 1 hour. He had a history of ingestion of hair dye containing PPD 4 hours back. The patient was administered corticosteroids and adrenaline along with

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other supportive measures. Endotracheal intubation was attempted but failed due to severe oedema of the tongue and oral cavity. Although tracheostomy was planned, it was not possible because of the extension of oedema to involve the neck. Meanwhile, bag and mask ventilation was provided. The patient ultimately succumbed after an hour.

Case 3

A 22-year female presented with swelling of tongue, stridor and respiratory distress for 2 hours. She had ingested hair dye containing PPD about 5-6 hours prior to admission. Immediately endotracheal intubation was done along with administration of corticosteroids, adrenaline and appropriate fluid therapy. Her renal parameters remained stable and she was having adequate urination. Her tongue and oral cavity oedema visibly decreased after 48 hours and she was successfully extubated. She was discharged on day 6.

Day	Urea (mg %)	Creatinine (mg %)	Na+ (mmol/L)	K+ (mmol/L)	Bilirubin (mg %)	ALT (U/L)	CPK (IU/L)	Hemoglobin (gm %)	TLC (mm^3)
1	20	0.9	135	3.6	0.8	100	6000	14	12200

DISCUSSION

In hair dye poisoning, the main toxic compound is PPD. It causes inflammation characterized by 3 stages - immunodepression in the first 3 days, proinflammatory stage due to rhabdomyolysis from days 3 to 6, and immunomodulating stage due to oxidative metabolism from day 6 onwards. This systemic inflammatory reaction is known as cytotoxic cell effect. The clinical triad consists of angioneurotic oedema of the face and neck with hoarseness of voice and stridor, rhabdomyolysis with dark coloured urination, and acute kidney injury. The earliest presentation in these patients following ingestion of the dye is a hard, swollen, and protruding tongue with an edematous bull neck. The stages of angioedema are facial and lip oedema (stage 1), soft palate oedema (stage 2), lingual oedema (stage 3), and laryngeal oedema (stage 4). The toxic effect of PPD is due to its oxidized product quinonediamine which is responsible for the severe localized reaction causing oropharyngeal oedema and subsequent respiratory distress leading to respiratory arrest.[4] Therefore, early and adequate airway protection is essential to save the life of the patient.

There are multiple mechanisms of acute kidney injury following PPD ingestion. First, PPD per se is toxic to the kidney due to its aromatic structure leading to acute tubular necrosis. Second, rhabdomyolysis is a recognized cause of renal injury.[5]

Early and adequate fluid resuscitation can prevent acute kidney injury. Otherwise, it could lead to renal failure necessitating the need for haemodialysis. Initial fluid management in adult patients is administered as per Parkland's formula. 50% of the calculated total dose is administered in the first 8 hours and the other 50% over the next 16 hours. Simultaneously, clinical parameters (raised jugular venous pressure, crepitations in the lung fields) should be checked to prevent pulmonary oedema. Also, observe the renal output.

CONCLUSION

In the case of PPD poisoning, immediate protection of airway from life-threatening laryngeal oedema and fluid therapy to overcome rhabdomyolysis is the mainstay of treatment. Where acute renal failure occurs, hemodialysis may be helpful.

REFERENCES

1. Pattnaik, R. B., Satpathy, S. K., Mishra, S. K., Dutt, A. K., & Mohapatra, D. (2001). Self injection of organophosphorous insecticide. *The Journal of the Association of Physicians of India*, *49*, 770.
2. Senthilkumaran, S., Jena, N. N., & Thirumalaikolundusubramanian, P. (2019). Super vasmol poisoning: dangers of darker shade. *Indian Journal of Critical Care Medicine: Peer-reviewed, Official Publication of Indian Society of Critical Care Medicine*, *23*(Suppl 4), S287.
3. Gude, D., Bansal, D. P., Ambegaonkar, R., & Prajapati, J. (2012). Paraphenylenediamine: Blackening more than just hair. *Journal of research in medical sciences: the official journal of Isfahan University of Medical Sciences*, *17*(6), 584.
4. Tainter, M. L., & Hanzlik, P. J. (1924). The mechanism of edema production by paraphenylenediamine. *Journal of Pharmacology and Experimental Therapeutics*, *24*(3), 179-211.
5. Sampathkumar, K., Sooraj, Y., Ajeshkumar, R., Mahaldar, A. R., & Muthiah, R. (2007). Case Report-Rhabdomyolysis due to hair dye poisoning: An emerging threat. Indian Journal of critical care medicine, 11(4), 212-214.

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Case Report

WILSON'S DISEASE IN SIBLINGS-A CASE REPORT

Putul Bara[1], Rina Mohanty[2], C R Khatua[2]

ABSTRACT

Introduction: Wilson's disease (WD) is a disorder of copper metabolism leading to the accumulation of this metal in different organs. Hepatic manifestations tend to occur in the first decade and neurological symptoms in the third decade. Neurological manifestation are said to worsen with chelation therapy.

Case report: We present two cases of Wilson's disease, who are siblings, and had different atypical manifestations of the disease.

Patient 1-A 18 year Hindu female presented with complaining of slurring of speech, abnormal movement of limb for 2 years which was gradually progressive and inability to walk for 2 months, progressive in nature and unable to walk for 2 months. She had hepatomegaly. On evaluation she was diagnosed to have Wilson's disease. She is now on treatment with copper chelating agent.

Patient 2-The younger sister of above patient a 17 year Hindu female presented with amenorrhoea, easy fatigability, swelling of abdomen since 1 year and joint pain in bilateral knee and ankle for 3 months . She had huge splenomegaly. After evaluation she was also diagnosed to have Wilson's disease.

Conclusion: These cases also serve as a reminder not to dismiss this disease as a rare theoretical possibility but to suspect it in a case of liver cirrhosis of unknown etiology or when the initial presentations are atypical. Siblings of Wilson's disease should be evaluated for the same even if they do not have symptoms. Delayed recognition of the disease or stopping therapy can lead to a progression of the disease. The patients had many unusual features which are being reported for future references by researchers and practioners.

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INTRODUCTION:

Wilson's disease is a rare inherited disorder of copper metabolism with deposition of copper in the liver, brain and other tissues with an incidence of one in 30, 000. [1] Initially there is deposition of the metal in the liver followed by its release into circulation and thereafter chronic accumulation in the brain and other extra-hepatic tissues. Liver cirrhosis occurs early. In the nervous system basal ganglia and midbrain are affected most frequently. According to a German study the patients who presented in first decade show predominantly hepatic manifestations while the patients with neurological symptoms presented during the third decade [2].Untreated Wilson's disease has a progressive course and may be fatal. The disease tends to be underdiagnosed and timely diagnosis remains a challenge.

CASE REPORT

Patient 1-Our patient was 18 year old Hindu female who presented in the out- patient department with history of slurring of speech, abnormal movement of limbs since 2 years which was gradual in onset, progressing in nature and subsided during sleep. She was unable to walk for last 2 months .Routine investigation revealed microcytic normochromic anaemia with leucopenia. Liver function tests and renal function tests were within normal limits. MRI of brain shows bilateral symmetrical hyperintense areas in lentiform nuclei, thalami and in brain stem .Ultrasonography of abdomen showed chronic hepatic parenchymal disease with splenomegaly. On slit lamp examination she was found to have bilateral Kayser-Fleischer ring. Further biochemical investigation revealed serum ceruloplasmin was found to be 9.75 mg/dl (normal range 20-60 mg/dl) and 24 hour urinary copper was found to be 390.63 mcg/day (normal range 3-50 mcg/dl).

Patient is now on copper chelating agent and on regular follow up.

Patients 2-This patient ,siblings of patients 1 was 17 year old hindu female who presented in the out -patient department with history of amenorrhoea ,swelling of abdomen since 1 year and bilateral knee and ankle joint pain for last 3 months. On examination patient had huge splenomegaly . After routine examination complete blood count showed pancytopenia with study of bone marrow showing erythroid hyperplasia with normal pattern of maturation suggestive of hypersplenism. Hepatitis B and C were negative.After taking family history patient was sent for the slit lamp examination and found to have bilateral Kayser-Fleischer ring and ultrasonography of abdomen showed chronic hepatic parenchyma disease with huge splenomegaly with endoscopic features of grade II esophageal varices. On further investigation 24 hour urinary copper is 223.73 ug/day (3-50 mcg/day). MRI of brain showed no significant abnormality.Patient is now on regular follow up.

DISCUSSION

The patients with Wilson's disease usually present with liver cirrhosis and followed byneurologic manifestation but in our case 1st patient presented with neurological symptoms and subsequent investigation revealed cirrhosis of liver whereas in 2nd patient presented with a features of hypersplenism , which was diagnosed to be due to cirrhosis of liver and Kayser- Fleischer ring but no neurological manifestation.

Paradoxically the neurological manifestation are said to become worse with penicillamine. This is attributed to mobilization of copper from liver with elevation in unbound copper which produce worsening of neurologic symptoms. In different studies the initial neurological deterioration was observed in 30-70% of patients following penicillamine therapy[5, 6.]Though penicillamine is the chelating agent advised for treatment of Wilson's disease, this was refuted by certain other reports[7.] The tremor showed slight improvement in our case with penicillamine and anticholinergic treatment however longer duration of treatment may be required for complete remission of symptom. It is considered that Wilson's disease occurs in siblings (25%) and the offspring (0.5%) and also occurs in the previous generation (0.5%) [8].

CONCLUSION

We present, two siblings, who had Wilson's disease with varied atypical and rare manifestations. As Wilson's disease is a rare disease the diagnosis is likely to be missed. There should be a high index of suspicion in all cases of liver cirrhosis with no clear-cut etiology or an isolated neurologic symptoms such as tremor. It is also important to warn patients not to stop therapy.

REFERENCES

1. PatilM ,Sheth KA, Krishnamurty AC et –al. A review and current perspective on Wilson disease. J ClinExpHepatol. 2013;3:321-36.
2. Merle U,SchaeferM,FerenciP,Stremmel W. Clinical presentation,diagnosis and long term outcome of wilson's disease :a cohort study.Gut.2007;115-20.
3. Taksande et al. MR Imaging of the Wilsons's Disease-A case report. www.jkscience.org 2009;11:150-151J.
4. Ferenci P Czlonkowska AMerie U Ferenc S Gromadzka G Yurdaydin C et al. Late onset Wilson's disease. Gastroenterology 2007;132:1294-8.
5. JayanteeKalita, Kumar Vijay et al. Worsening of Wilson Disease following Pencillamine Therapy EurNeurol 2014;71:126-131.
6. Medici V, Trevisan CP, D'Inca R, Barollo M, Zancan L, Fagiuoli S, et al: Diagnosis and management of Wilson's disease:results of a single centre experience. J ClinGastroenterol 2006;40:936-941.
7. Lange J: Long-term treatment of Wilson's disease with D- pencillamine. Report on 20 cases. Dtsch Med Wochenschr 1967;92: 1657-1662.
8. Brunet AS, Marotte S, Guillaud O, et al. Familial screening in Wilson's disease: think at the previous generation. J Hepatol 2012;1394-5[PubMed] [Google Scholar].

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Case Report

A RARE CASE OF IMMUNE THROMBOCYTOPENIA PURPURA SECONDARY TO SUBCLINICAL HASHIMOTO'S THYROIDITIS

Devasi Manoharlal B[1], Ambika Mohanty[2], Lalatendu Mohanty[2], Annam Chaitanya Teja[1.]

INTRODUCTION

Immune Thrombocytopenia Purpura(ITP) is the mostly observed as isolated thrombocytopenia in normal people. ITP may rarely be associated with other autoimmune disorders like autoimmune thyroid disorders, which may indicate more complex defect in immune system[1]. Primary ITP usually responds well to steroids and IV immunoglobulins. However, ITP may be tough task to treat when associated with thyroid autoimmune disorders. In such cases, treating the underlying thyroid disorder may substantially improve platelet count and can either cause remission of disease or improve response to standard ITP therapy. We report a case of 35-year-old male, who came with complaints of swelling in neck and was diagnosed with subclinical Hashimoto's thyroiditis associated with ITP.

CASE REPORT

A 35-year-old male presented to our out- patient department with complaints of swelling in neck for two months. It was not associated with pain or dysphagia. The patient denied any history of fever, chills, sore throat, body pain, skin rashes, bleeding or drug intake. On examination, vitals were stable, xerosis seen over both upper and lower limbs. Neck examination revealed a non tender diffuse swelling over midline of size around 7cm x 3cm which moves with deglutition. Systemic examination was unremarkable. On investigation complete blood count showed platelet count of 8000/ µLwith normal WBC and hemoglobin, TSH was 19.62 µIU/mL with normal free T4 and T3 and positive anti-TPO antibodies (600 IU/mL). Other causes of thrombocytopenia were ruled out. As platelet counts were critically low, it was considered a medical emergency, so he was immediately given platelet transfusion. After starting levothyroxine and steroids, platelet count gradually increased to 1 lakh/µL within 7 days, he was discharged with amaintenance dose of 40 mg of prednisolone[2] and tapered off over 1month.

CONCLUSION

Association of ITP with Hashimoto's thyroiditis has been documented in few reports and studies[3], but subclinical Hashimoto's thyroiditis as the cause of secondary ITP is a very rare phenomenon. Treatment of Hashimoto's thyroiditis with levothyroxine may either induce remission or enhance response of standard ITP therapy.

REFERENCES

1. Tahir H, Sheraz F, Sagi J, Daruwalla V. Immune thrombocytopenia (ITP)secondary to subclinical hashimoto's thyroiditis: role of levothyroxine in improving the clinical outcome of ITP. Journal of investigative medicine high impact case reports. 2016 Apr 28;4(2):2324709616647085.
2. Cohen R, Garcia CA, Mena D, Castellanos M. Case Review:Idiopathic Thrombocytopenic Purpura. Journal of Medical Cases. 2012 Mar 19;3(2):130-4.
3. Biró E, Szekanecz Z, Dankó K, Kiss E, Szabó NA, Szûcs G, Zeher M, Bodolay E, Szegedi G, Bakó G, Czirják L. Association of systemic and thyroid autoimmune diseases.Clinical rheumatology. 2006 Mar 1;25 (2):240.

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Abstract

Abstract

HYPOCALCEMIA A POTENTIAL BIOCHEMICAL MARKER OF SEVERE DENGUE : A CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL IN EASTERN INDIA

Aishwarya Joshi , Nabakishor Sundaray, Subhashchandra Dash, Samir Sahu, Praveen Patil, Karanam Gowtham Naidu , Navin Sudhakaran

INRODUCTION:

Dengue fever is a major public health issue in the sub-tropical climate of eastern India. The first virologically proven epidemic of Dengue Fever occurred in Calcutta and East coast of India in 1963-1964. Calcium is an important serum electrolyte in cardiac and circulatory homeostasis. Hypocalcemia has been associated with the immune response and severity of infection. Several studies showed hypocalcaemia to be more prevalent in patients with dengue haemorrhagic fever (DHF) than in patients with Dengue fever [DF].

OBJECTIVE:

To establish and correlate the severity of Dengue with hypocalcemia.

METHODOLOGY:

A cross-sectional observational study was conducted at IMS and SUM hospital, a tertiary health care centre in eastern India at Bhubaneswar, Odisha over a duration of 18 months. 150 probable cases of dengue were admitted and confirmed by either IgM antibody or NS1 antigen detection. It was classified according to World Health Organization criteria and followed up with daily clinical progression and serum calcium level and other essential lab parameters. Data was entered in SPSS 20. Presence of hypocalcemia was compared in patients with DF and DHF.

RESULTS:

The sample size was 150. The mean age was 39.1 years, and the majority were males (n = 104, 69.3%). DHF was diagnosed in 47 patients (31.33%). Mean serum Ca2+ level of the study population was 8.71mg/dL (range 8.4 – 10.5). Mean serum Ca2+ was significantly higher in patients with dengue fever (DF) (8.937 mg/dL) than in those with DHF (8.16 mg/dL) (p 0.00). A significant difference was observed between mean serum calcium levels of DF and DHF. Prevalence of hypocalcemia in DHF and DF patients was 61.70% (n =29) and 31.06% (n = 32), respectively (p = 0.00).

CONCLUSION:

Serum Ca2+ levels had significant correlation with dengue severity. Serum Ca2+ levels were significantly lower, and hypocalcemia more prevalent, in patients with DHF than in patients with DF. This study potentiates the use of serum calcium as a biochemical marker for early detection of severe dengue infection.

KEYWORDS:

Biochemical marker, dengue, hypocalcemia, severe dengue.

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Abstract

A RARE CASE OF LUPUS MYOCARDITIS AS THE INITIAL PRESENTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS

Pavan Sai Kumar[1], Shubhransu Patro[2], Rabinarayan Rout[3], Pankaj Kumar Khora[3], Siddhartha Mishra[2]

INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a common autoimmune disease throughout the world. In India, the prevalence of SLE is 3.2 per 1,00,0000[1]. SLE has protean clinical manifestations that may differ dramatically from patient to patient. Lupus Myocarditis is dangerous potentially life-threatening and an uncommon presentation of systemic lupus erythematosus (SLE) with a prevalence of 8 to 25 per cent in past studies[2]. We report a case who presented to our hospital with myocarditis as an initial manifestation of systemic lupus erythematosus. The mainstays of treatment are corticosteroids, immunosuppressive agents, and anti-heart failure medications.

CASE PRESENTATION

An 18-year-old young male patient had a history of on and off a fever for last eight weeks, partially treated outside attended to the emergency department with complaints of shortness of breath on exertion associated with palpitations, swelling of both lower limbs and low-grade fever for the last ten days. On clinical examination, no abnormality found except tachycardia, bilateral pedal oedema and bilateral basal crepitations. Laboratory investigations revealed thrombocytopenia, raised acute phase reactants and elevated cardiac biomarkers. Immunological workup revealed positive for anti-double-stranded DNA antibodies (ds-DNA), SS-A/Ro antibodies, RNP/Sm antibodies and Ribosomal-P-protein antibodies and decreased C3 and C4 complement protein concentration. Based upon 2019 EULAR /ACR criteria for SLE, the patient diagnosed as a case of Lupus myocarditis.

DISCUSSION AND CONCLUSION

Myocarditis, an uncommon manifestation of SLE and potentially fatal, should be suspected with the following clinical features: tachycardia disproportionate to temperature, unexplained heart failure or cardiomegaly and electrocardiographic abnormalities[3]. Any patient without any known chronic disease attended with history of low grade of fever, dyspnoea, palpitations and swelling of lower limbs then myocarditis of a connective tissue disorder should think as a differential diagnosis. Early diagnosis and treatment of the disease can prevent complications and lead to a favourable prognosis with improved quality of life.

REFERENCES

1. Malaviya AN, Singh RR, Singh YN, Kapoor SK, Kumar A. Prevalence of systemic lupus erythematosus in India. Lupus. 1993 Apr;2(2):115-8. doi: 10.1177/096120339300200209. PMID: 8330032.
2. Apte M, McGwin G Jr, Vilá LM, Kaslow RA, Alarcón GS, Reveille JD; LUMINA Study Group. Associated factors and impact of myocarditis in patients with SLE from LUMINA, a multiethnic US cohort (LV). [corrected]. Rheumatology (Oxford). 2008 Mar;47(3):362-7. doi: 10.1093/rheumatology/kem371. Epub 2008 Feb 4. Erratum in: Rheumatology (Oxford).2008 May;47(5): 742. PMID: 18250089.
3. Miner JJ, Kim AH. Cardiac manifestations of systemic lupus erythematosus. Rheum Dis Clin North Am. 2014 Feb;40(1):51-60. doi: 10.1016/j.rdc.2013.10.003. PMID: 24268009.

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Abstract

RARE CASE OF SECONDARY HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS IN A CASE OF MILIARY TUBERCULOSIS

Poloor Naveen Kumar[1], Samir Sahoo[2], Arpita Das[3]

ABSTRACT

A 27yrs old male presented with fever, cough, loss of weight and appetite for three months. Based on symptoms, Contrast Enhanced Computed Tomography Thorax showed – multiple tiny centrilobular nodular opacities in bilateral lower lobe with mediastinal necrotic lymphadenopathy suggestive of miliary tuberculosis. Anti-tubercular therapy (ATT) was started based on clinical features and Computed Tomography findings. Oral steroids was administered later in the course of disease in view of worsening clinical features and persistent oxygen requirement. Patient continued to have fever even after 20 days of completion of ATT. Laboratory investigations showed of pancytopenia, high serum ferritin and triglyceride levels. Bone marrow biopsy showed increased reticuloendothelial activity and hemophagocytosis with granulomatous lesions. High dose steroids added to the existing treatment. General condition of the patient gradually improved and patient was discharged. Miliary Tuberculosis with secondary HLH is rare, but can be fatal, if not diagnosed and treated early.

KEYWORDS:

Hemophagocytic lymphohistiocytosis (HLH), miliary tuberculosis, Steroids

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Abstract

PREVALANCE OF CARDIOMYOPATHY IN TYPE 2 DIABETES MELLITUS PATIENTS ADMITTED TO MKCGMCH, BERHAMPUR

Jasobanta Behera, Jagannath Sarangi

INTRODUCTION:

Diabetes mellitus is an established risk factor for congestive heart failure. The Framingham Heart study has shown that the incidence of congestive cardiac failure in diabetic patients occurs irrespective of coronary artery disease or hypertension. In overt heart failure, diastolic dysfunction often co-exists with systolic dysfunction as a consequence of ischemic heart disease, but diastolic dysfunction is a frequent finding in type 2 diabetes mellitus without signs and symptoms of heart disease and is presumably due to diabetic cardiomyopathy. Left ventricular diastolic function (LVDF) is affected earlier than systolic function in the development of congestive cardiac failure.

Therefore left ventricular diastolic dysfunction may represent the first stage of diabetic cardiomyopathy, thus an early examination of left ventricular diastolic function may help to detect this condition in patients with diabetes, thereby allowing early intervention for a more favorable outcome.This study was done to understand the burden of left ventricular diastolic dysfunction (LVDD) in patients with newly diagnosed type 2 diabetes and to assess the risk factors for the development of diastolic dysfunction in such patients.

MATERIALS AND METHODS

The study was conducted in MKCG medical college hospital odisha from 2018 to 2020. Hundred patients of type 2 diabetes was included in the study after fulfilling inclusion and exclusion criteria. Along with vitals and routine investigation, ECG and echocardiography was done. Diabetic cardiomyopathy was graded into four grades of severity.

RESULTS

Among the study participants, 27 % of patients were found to be normal and 73% of patients had cardiomyopathy of various grades. Maximum patients were having grade 1 diastolic dysfunction i.e 54 %, followed by grade 2 diastolic dysfunction i.e 10 %. 9% of patients had systolic dysfunction with restrictive diastolic dysfunction, out of which 8 patients had heart failure with preserved ejection fraction and one had heart failure with reduced ejection.

CONCLUSION

In my study prevalence of cardiomyopathy was more in patients with uncontrolled diabetes. Hence all newly detected diabetes with or without risk factors like hypertension, obesity should be evaluated for with echocardiography for early detection of diastolic dysfunction and for further management to halt the progression of disease.

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Abstract

EVALUATION OF RENAL CHANGES IN SICKLE CELL DISEASE IN A REFERRAL INSTITUTE OF SOUTHERN ODISHA AT MKCG MCH, BERHAMPUR

Susanta Sekhar Behera[1], Bijaya Kumar Behera[2]

INTRODUCTION

Sickle cell disease is a hereditary genetic disease that is characterized by presence of abnormal crescent shaped RBC.Renal dysfunctions collectively known as sickle cell nephropathy (SCN) is a known complication of it.

OBJECTIVE

To study the renal profile in patients of sickle cell disease and to evaluate risk factors.

METHODS

It was a observational,cross-sectional study conducted in the department of general medicine on patients of sickle cell disease coming to medicine and nephrology department of MKCG medical college, berhampur,a referral institute of southern odisha involvng 82 ptients(50 SCT patients,32 SCA patients). Study period was january2019-december2020.CBC,Urine routine,microscopy,serum urea creatinine, USG tests were done to find out renal dysfunction.

RESULTS

Glomerular and tubular dysfunction was more in SCA(Hb SS) patients then SCT(Hb AS) patients and the abnormality was more in patients in crisis.In this study we found; in SCA Albuminuria was found in 78.12% ,hematuria in in 46.87%,cast and crystal in 28.12%, epithelial cell in 31.25%,hyposthenuria in 56.25%.In SCT Albuminuria was fouund in 38%,hematuria in in 16%,cast and crystal in 22%, epithelial cell in 12%,hyposthenuria in 24%.All above findings were found in more percentage of cases in crisis patients of both groups.In SCA 37.5% and in SCT 2% were found to have CKD.

CONCLUSION

Renal involvement in the form of glomerular and tubular dysfuction occur in sickle cell disease and more in crisis patients, leading to renal complication and end stage renal disease.so the patients should be taught about regular medical check up measures should be taken to prevent crisis.

KEYWORDS

SCD-sickle cell disease,SCA-sickle cell anemia,SCT-sickle cell trait,CKD-chronic kidney disease.

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Abstract

A STUDY OF RELATIONSHIP BETWEEN IRON DEFICIENCY AND THYROID FUNCTION IN FEMALES OF REPRODUCTIVE AGE GROUP OF SOUTHERN ODISHA

Anshuman Mishra[1], Diptimayee Tripathy[2], SN Jali[3], Chitta Ranjan Khatua[4]

INTRODUCTION

Iron deficiency occurs in all ages, but is especially common in women of child bearing age. Iron plays a key role in the synthesis of thyroid hormones. Serum ferritin, a measure of iron stores is the single best test to confirm iron deficiency. The effect of Thyroid disorders on women of reproductive age group is noteworthy. One of the most common thyroid disorder in this age group is hypothyroidism. Several heterogeneous studies had been done worldwide to show the relation between iron deficiency and thyroid function on different study groups. In our study females of reproductive age group(non-pregnant) have been taken as the study population. The reason being iron deficiency and thyroid disorders are more prevalent in non-pregnant females and very few studies have been done worldwide and none in India.

METHODS

A cross-sectional observational study was done on 50 non-pregnant women of reproductive age group. The study population was divided as per their serum ferritin values into Iron deficient and non-iron deficient groups. Then their TSH and FT4 were compared.

RESULTS

In our study, 68% were iron deficient, 20% had low FT4 and 26% had raised TSH. A positive correlation was obtained between FT4 and ferritin with correlation coefficient of 1.212 and P value < 0.005. Serum TSH levels and ferritin were inversely correlated with correlation coefficient of -0.335 and significant P value < 0.004.

CONCLUSION

We concluded from our study that the prevalence of iron deficiency was very high in non-pregnant Indian women and was associated with a higher serum TSH and lower FT4 levels. Thus, it is recommended that iron deficiency should be evaluated and treated to combat thyroid dysfunction.

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Abstract

EVALUATION OF CARDIOVASCULAR STATUS IN PATIENTS WITH SICKLE CELL DISEASE ADMITTED TO MKCG MEDICAL COLLEGE, ODISHA

Debabrata Rath[1], Umashankar Mishra[2]

INTRODUCTION

Sickle cell disease is a common hemoglobinopathy worldwide and also in India. Cardiovascular changes due to sickle cell disease are a major complication and contributing factor for significant number of mortality in sickle cell disease patients. Pulmonary hypertension (PHT) is a common complication in patients with sickle cell disease (SCD), with a reported prevalence of approximately 30% (Simons et al, 1988; Sutton et al, 1994; Ataga et al, 2004; Gladwin et al, 2004).

METHODS

A cross sectional hospital based study, conducted in MKCG Medical College involving 88 SCD (Sickle cell anemia and Sickle cell trait) patients admitted to Medicine and Cardiology ward during period of 2 yrs from January 2019-November 2020. Those having other hemoglobinopathy, renal diseases, valvular and congenital heart diseases, systemic diseases, lung diseases were excluded. Detail clinical examination including specific cardiovascular examination like x ray, ECG , Echocardiography were done.

RESULTS

It was found that pulmonary hypertension, Right ventricular hypertrophy and Left ventricular hypertrophy were most common cardiovascular manifestation in Sickle cell disease patients. The prevalence of RVE among the study participants was 20.5%, LVE was 13.6% and Pulmonary Hypertension it was 28.4%.

Pulmonary hypertension was most common among these findings and more prevalent among Sickle cell anemia patients. Those patients having episode of acute chest syndrome had more prevalence of pulmonary hypertension.

CONCLUSION

The study finds certain cardiovascular manifestations are due to pathologic process related to Sickle cell disease. Pulmonary hypertension is the most common manifestation among those. Hence early identification of these complications and prevention will help these patients to reduce their morbidity and mortality.

Keywords-PHT-Pulmonary Hypertension, SCD-Sickle cell Disease, RVE- Right Ventricular Enlargement, LVE- Left Ventricular Enlargement

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Abstract

A STUDY ON SERUM AMYLASE LEVEL IN PATIENTS OF ACUTE ORGANOPHOSPHOROUS POISONING IN SOUTHERN ODISHA

Dipak Kumar Nayak, Suvendu Sekhar Acharya

ABSTRACT

Acute organophosphorus poisoning produce clinical alteration in the serum amylase. Earlier plasma cholinesterase level was used to assess the severity of poisoning. Presently serum amylase is being recommended by some literature as a better indicator of severity.

The present study was undertaken to estimate serum amylase levels in acute organophosphate poisoning and to correlate serum amylase levels with the outcome of the patient.

This cross-sectional study was conducted at M.K.C.G Medical College, Brahmapur, Odisha India. A total of 80 patients with acute OP poisoning admitted to the hospital during the study period of 20 November 2018 to 20th November 2020 were included in the study. Biochemical evaluations which include serum amylase, blood glucose, urea, and creatinine were analyzed and correlated with outcome.

In acute OP poisoning patients, the s.amylase levels were significantly elevated at the time of admission (264±72U/L) and have shown a gradual remission with proper treatment. The mean s.amylase level in severely poisoned patients was (296±108) U/L. The clinical features are very well correlated with s.amylase levels. S.amylase in cases with pinpoint pupil, fasciculations, secretions, CNS depression, respiratory failure, and convulsions 182±108 U/L, 205±107 U/L, 172.1±106U/L, 204±131 U/L, 229±106 U/L, 318.6±93 U/L respectively. The overall mean value of s.amylase was significantly higher in non-survivors Vs survivors 218±144 Vs 115±91U/L, p=0.0038. Persistently elevated s.amylase level on day 2 has a significant relationship with the poor outcome. Hyperglycemia at admission also had a bad prognostic outcome whereas s.urea and creatinine had no such correlation with outcome.

Serum amylase levels may be considered as a marker of OP poisoning since it enables the early recognition of severity and to identify those at risk of developing the complications of OP poisoning.

Keywords: Organophosphorus poisoning, Serum amylase, Plasma cholinesterase, Serum creatinine, Blood glucose.

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Abstract

A CASE OF MULTIORGAN SYSTEM FAILURE DUE TOMULTIPLE BEE STINGS

Rajasekhar Gurrala[1], Lalatendu Mohanty[2], Ambika Mohanty[3], BRP Rao[4]

INTRODUCTION:

In a developing country like India, where we find one or two honeycombs at every corner; a bee sting is sometimes unavoidable.In most patients, bee stings cause local reactions and anaphylaxis but multiorgan system failureis an uncommon complication ofbee sting.[1]There is inadequate data for honey bee sting-induced multiorgan system failure in the world population.Though allergic reaction can be the result of a lone sting, prognosis is considered worse in the presence of multiple stings due to inoculation of greater volume of venom.[2]Bee venom contains peptides (mellitin, apamin), enzymes, amine and other proteins with mellitin & phospholipase A2 (PLA2) being the fatal components because of vasoactive and hemolytic properties.[3]Here, we report a case who presented with multiorgan system failure 3 days after he had sustained multiple bee stings.

CASE REPORT

A 48-year-old male, with no comorbidities presented to ED with a 2 day history of multiple episodes of vomiting, swelling of whole body with facial puffiness, oliguria after multiple honeybee stings.On physical examination, multiple bee sting bite markswere seen over his back, chest, neck and arms.Laboratory investigations revealed leucocytosis, thrombocytopenia, deranged renal parameters and liver function tests.ABG was suggestive of metabolic acidosis. The patient was treated with antihistamines, hydrocortisone, fluid infusion and hemodialysis. After few sessions of hemodialysis, urine output was 1.4 lit/day with improved kidney and liver parameters.

CONCLUSION

Treatment for bee sting should be given as soon as possible to avoid severe AKI because, prognosis of the patient in terms of AKI can be calculated by the time interval between the massive honey bee attack and the initiation of treatment.The mortality rate of patients who experience AKI has been reported to be as high as 25%.[1]

In patients presenting to emergency department with honey bee sting, a possibility of multiorgan system failure should always be kept in mind as it carries a high mortality. A high index of suspicion can lead to early diagnosis and treatment with successful outcomes.

ENDNOTES

1. Sharma M, Das HJ, Barman AK, Mahanta PJ. Acute kidney injury due to multiple wasp stings. Saudi Journal of Kidney Diseases and Transplantation. 2017 Jan 1;28(1):196.
2. Silva Junior GB, Vasconcelos Junior AG, Rocha AM, Vasconcelos VR, Barros Neto JD, Fujishima JS, Ferreira NB, Barros EJ, Daher ED. Acute kidney injury complicating bee stings–a review. Revista do Instituto de Medicina Tropical de Sã o Paulo. 2017;59.
3. Sathi S, Garg AK, Chand J, Singh MK, Saini VS. Severe Acute Kidney Injury after Multiple Honey Bee Stings. Int J Nephrol Kidney Fail. 2020;6(2).

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Abstract

ECHOCARDIOGRAPHIC ASSESSMENT OF HYPERTENSIVE CHANGES IN ELDERLY PATIENTS OF ISOLATED SYSTOLIC HYPERTENSION WITH SPECIAL REFERENCES TO CARDIOVASCULAR COMPLICATIONS.

Rakesh Mohanty[1], Bijaya Kumar Behera[2]

INTRODUCTION

ISH (Isolated systolic hypertension) is a rapidly growing concern in the elderly population with respect to development of significant morbidity and mortality such as myocardial infarction, heart failure, CVA, retinopathy etc. Most studies describe the effect of diastolic hypertension on heart and little is known about implications of ISH in the ageing population. It is a new public health concern and its management still remains a challenge to practicing physicians.

OBJECTIVE

To assess the Echocardiograhic changes and cardiovascular complications in patients with Isolated Systolic Hypertension.

METHODS

It was a cross sectional, observational study done at the department of medicine and department of cardiology, MKCG medical college and Hospital, Berhampur, Odisha, India. Seventy patients above the age of 65 years with systolic blood pressure $\geq$ 140 mm Hg and diastolic blood pressure <90 mm Hg , without any secondary causes of hypertension and shouldn't be on any antihypertensive drug therapy were selected for this study and echocardiography and electrocardiography studies were implemented for demonstrating the development of cardiovascular complications. Chi square test and Student T test were applied for analysis of result and significance of the study.

RESULTS

Out of 70 cases (41 male and 29 female) , 64% are symptomatic with palpitation being the major symptom, 40% (28 cases) developed retinopathy, 45.71% developed increased LVMI(left ventricular mass index)(p=0.04) , 18.57% had increased in LV volume (>90ml/m^2) .30% cases had RWMA (regional wall motion abnormalities), 27.1% had reduced ejection fraction(<56%), 52.66% showed LA enlargement in this study (p =0.048). In this study Doppler measurement of diastolic filling are significant in patients with ISH with higher peak atrial velocity (A wave) with 79.71$\pm$11.79) and a ratio of peak early to atrial velocity lower (0.82$\pm$0.29) as compared to study group without ISH.

Conclusion: This study demonstrates that ISH elderly patients have prevalence of concentric LVH similar to that of patients with combined systolic and diastolic hypertension. ISH is the most common cause of hypertension in the elderly. ISH associated risk factors has definite effect on the cardia in terms of LVH. It is to be treated promptly so as to reduce cardiovascular morbidity and mortality.

Keywords: Systolic, Hypertension, Left ventricular mass index, RWMA

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Abstract

COVID-19 PATINETS WITH DIABETES -A DREADFUL CURSE

A retrospective analysis of deceased covid-19 patients with Diabetes in a tertiary care hospital

Annam Chaitanya Teja[1], Ambika Prasad Mohanty[2], Devasi Manoharlal[2]

A novel corona virus, severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) (corona virus disease 2019 [COVID-19]) is now at global pandemic levels causing significant morbidity and mortality. Patients with severe COVID-19 can develop a systemic inflammatory response that can lead to lung injury and multisystem organ dysfunction. However, patients with diabetes are particularly vulnerable and more likely to get severe complications when infected with this virus and evidence in the COVID-19 pandemic shows that people with diabetes are associated with poor prognosis and diabetic people with associated risk factors are liable for increase risk of mortality.

AIM AND OBJECTIVES

To analyse the prevalence and risk factors among the deceased individuals with diabetes in a government designated COVID care tertiary center.

METHODS

This study includes 400 deceased COVID-19 patients in government designated COVID tertiary care Hospital from April, 2020. (COVID-19 positivity is considered either by RTPCR/Rapid antigen analysis of COVID-19)

RESULTS

Out of these 400 patients 72 (18 %) patients are diabetic. Out of these 72 diabetic patients 24(33.3%) patients had hypertension (among which 6 had associated cardiovascular disease) 16 (22.2%) patients had cardiovascular disease and 9(12.5%) patients had chronic kidney disease and 2 patients had COPD. 2 patients are received brought in dead to hospital.

Among these 72 patients the peak of mortality was observed in people aged 50-59 (33 patients).

CONCLUSION

People with diabetes with COVID-19 are at a greater risk of worse prognosis and mortality. Given the high worldwide prevalence of diabetes, these individuals represent a large vulnerable segment of the COVID-19 population. The poorer prognosis of people with diabetes is the likely consequence of the syndromic nature of the disease: hyperglycaemia, older age, comorbidities, and in particular hypertension, obesity, and cardiovascular disease all contribute to increase the risk in these individuals.

REFERENCES

1) COVID-19 in people with diabetes: understanding the reasons for worse outcomes;Matteo Apicella*, Maria Cristina Campopiano*, Michele Mantuano*, Laura Mazoni*, Alberto Coppelli, Stefano Del PratoLancet Diabetes Endocrinol 2020: 8; 782–92.doi.org/10.1016/S2213-8587(20)30238-2

2) Erener S. Diabetes, infection risk and COVID-19. Mol Metab. 2020;39:101044. doi:10.1016/j.molmet.2020.101044

3) Zaim S, Chong JH, Sankaranarayanan V, Harky A. COVID-19 and Multiorgan Response. Curr Probl Cardiol. 2020;45(8):100618. doi:10.1016/j.cpcardiol.2020.100618

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Abstract

A STUDY OF CORRELATION BETWEEN OESOPHAGEAL VARICES WITH PORTAL AND SPLENIC VENOUS DIAMETER RATIO AND GRADIENT IN PATIENTS WITH CIRRHOSIS OF LIVER IN SOUTHERN ODISHA.

Putul Bara[1], DM Tripathy[2], RMohanty[3], CR Khatua[4]

ABSTRACT

Esophagealvarices develop as a consequences of portal hypertension in patient with chronic liver disease and are present in approximately 50% of patient with cirrhosis of liver.

There is high prevalence of alcoholic liver disease with cirrhosis of liver in Southern Odisha. But there is lack of endoscopy facility in periphery setup,for which it is not possible that all patient with cirrhosis of liver could undergo screening for varices. Portal vein to splenic vein diameter ratio (portalvein size/ splenic vein size)and gradient in millimetre (portal vein size-splenic vein size) could be a valuable tools in predicting the presence of porto- systemic shunting .

USG of abdomen being a non –invasive procedure can be a useful alternative to endoscopy in patient with cirrhosis of liver and hence the study undertaken.

Total 74 patients taken into study out of which 64 patients have cirrhosis with varices and 10 patients have cirrhosis without varices.

The study shows mean value of portal vein and splenic vein diameter ratio and gradient are statistically significant.

So to assess presence of varices in cirrhosis of liver following are the recommendations:

- USG abdomen should be used as a non- invasive tool for predicting esophagealvarices.
- Splenic vein diameter and portal vein diameter needs to be assessed in all cases of CLD.
- Portal vein and splenic vein diameter ratio gradient to be calculated in all cases of cirrhosis to predict presence of oesophageal varices in centres where endoscopy facility is not available.

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Abstract

GBS IN ASSOCIATION WITH HASHIMOTO'S THYROIDITIS : A RARE CASE REPORT

Abinash Hota

ABSTRACT

Guillain-Barre' syndrome is an acute inflammatory polyradiculoneuropathy having an autoimmune origin. A number of case reports show its association with post-viral infections, post-vaccinations, medications and one or other autoimmune diseases as well. But its fair enough to say that association of GBS with Hashimoto's thyroiditis is a quite rare entity.

CASE STUDY

A 33 year Hindu male presented to ED with acute onset paraparesis progressive and ascending in nature affecting both upper limbs with bulbar affection gradually over 4 days which is associated with severe autonomic dysfunction. There was no prior history of fever or any respiratory and gastrointestinal infection. The csf analysis and electromyoneurography suggested an acute demyelinating polyneuropathy. Investigations for the viral aetiologies came negative. The atypical facies, coarse skin, forceful voice gave a suspicion of hypothyroid state which was investigated and found to be due to Hashimoto's thyroiditis.

TREATMENT& FOLLOW UP

Post diagnosis the motor component responded gradually to IVIG treatment with persistence of the autonomic dysfunction. Replenishment of the hypothyroid state with l-thyroxine and steroids to combat the autoantibodies has been done. After managing the autonomic dysfunction the patient has been discharged. On follow up the patient is doing quite well without any residual weakness.

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Abstract

STUDY ON SERUM URIC ACID IN CHRONIC HEART FAILURE AND ITS CORRELATION WITH NYHA CLASSIFICATION

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BACKGROUND

Heart Failure is responsible more than 20 million patients annually. Hyperuricemia is a constant finding in Chronic Heart Failure. Assessing serum uric acid may allow rapid and cost-effective determination of clinical prognosis in heart failure patients.

AIM AND OBJECTIVE

Serum uric acid level in chronic heart failure and it's correlation with the prognosis and its relevance with NYHA classification

METHODS

This case-control study was conducted in MKCG Medical College,Berhampur, Odisha , from 2018 to 2020.A total of 80 patients with chronic heart failure were included in the study and followed after 1month.40 age and sex matched controls were included. The prognostic markers of heart failure both before and after treatment were compared.

RESULTS

The mean serum uric acid was 6.615 mg/dl (control 3.71 mg/dl). The mean uric acid was higher in male (male=7.456 mg/dl, female=5.353 mg/dl).It was found that mean serum uric acid level was 9.570 in NYHA class IV, 6.702 in NYHA class III and 4.853 in NYHA class II. The NYHA class worsened if there was an increase in uric acid.Before treatment mean serum uric acid was 6.61mg/dl and after treatment it became 5.91 mg/dl. Mean uric acid was found to be 9.408 mg/dl in non survivors. There was significant difference in serum uric acid between the survivors and dead (p =<0.001).

CONCLUSION

Serum uric acid levels are higher in patients of heart failure. Uric acid is an important objective marker in prognosis of heart failure which can be done easily.Patients in higher NYHA Class of heart failure have higher serum uric acid levels, thus serum uric acid levels positively correlated with NYHA Class. serum uric acid level and NYHA class in combination is a good predictor of mortality of heart failure.

Keywords: serum uric acid, chronic heart failure, NYHA

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Abstract

STUDY OF SERUM FERRITIN & CRP LEVEL IN DENGUE FEVER AND THEIR CORRELATION WITH THROMBOCYTOPENIA

Sananda Kumar Sethi, U S Mishra

INTRODUCTION

The commonly used investigations to identify dengue virus infection are NS1,IgM,IgG by ELISA/ Rapid kit tests. A part form these investigations , serum ferritin and CRP levels are also high in dengue fever. This is observed that sr. ferritin & CRP are negatively correlates with total platelet count(TPC).

AIM :

1) To study serum ferritin and CRP level in dengue fever.

2) Correlation of serum ferritin and CRP with thrombocytopenia.

METHODS

This prospective study was done at department of medicine,MKCGMCH , Berhampur, Odisha, India. Total 100 cases of NS1 &/or IgM ELISA positive were included in this study. Routine blood investigations, serum ferritin and C-reactive protein levels were investigated for all patients. Patients were catagorize according to their clinical profile like uncomplicated dengue fever, dengue haemorrhagic fever and dengue shock syndrome. Chi square and student T test were applied for analysis of result and significance of the study.

RESULTS

The results were tabulated based on the observation of ferritin ,CRP,TPC count on day one & day five of admission. It is shown that ,there is a strong correlation between sr. ferritin , CRP and thromboctopenia, severity of dengue fever.

CONCLUSION

From the study it can be concluded that the serum ferritin & CRP levels of the patients can be taken as a biomarker for early prediction of sever dengue fever.

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Abstract

CORRELATION BETWEEN EARLY PREGNANCY BMI AND RISK OF GESTATIONAL DIABETES MELLITUS.

Bisweswar Rout (JR3)[1], Namita Mohanty[2]

INTRODUCTION

Until the mid-19th century diabetes was considered to be incompatible with successful pregnancy. Diabetes in pregnancy considered as a major risk factor for sessions maternal and fetal complications GDM is defined as any degree of glucose intolerance with onset or first recognition during pregnancy.

OBJECTIVE

To study the correlation between early pregnancy BMI and the risk of developing gestational diabetes mellitus.

METHODS

This is a prospective observational study conducted in pregnant women attending antenatal outpatient Department obstetrics and gynaecology, MKCHG MCH, Berhampur. In our study height weight and waist hip ratio of pregnant female measured upto 14 wks. BMI is calculated according to quetlet index (weight in kg/height in meter squared). Catergorization of pregnancy women according to ASIAN INDIAN BMI Guide lines. Screening is done by using WHO, CARPENTAR and COUSTON, IADSP, DIPSI Guidelines

RESULTS

A total of 250 women were studied. 104 women develop GDM. Concept of GDM was developed

In our study underweight women with BMI < 18.4 are 4 and none of them developed GDM. Women with normal BMI between (18.5 – 22.9) were 185 and in this population 36.2% developed GDM, 63.8% did not develop GDM. Overweight women with BMI between (23-24.9) were 35, out of which 21 (60%) developed GDM and 14 (40%) did not develop 80% obese women and 60% overweight women developed GDM and showed a significant correlation of 'P' = .0064.

CONCLUSIONS

Our study population showed, early pregnancy BMI has significant relation in developing future GDM and can be represented as a modifiable risk factor.

Key Words : GDM-Gestational Diabetes Mellitus BMI – Body Mass Index. IADSP The International Association of Diabetes and Pregancy study Group.

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Abstract

STUDY OF SERUM FERRITIN IN PATIENTS OF STROKE

Aswin Suresh[1], Namita Mohanty[2]

BACKGROUND

Stroke is the second commonest cause of death and fourth leading cause of disability worldwide. It is suggested that high serum ferritin levels on the first day of hospitalization for stroke are related to poor prognosis. Objective of this study is to estimate the concentration of serum ferritin and to correlate it with early neurological deterioration in patients of acute stroke.

METHODS

In this prospective observational study, a total of 100 patients who presented with stroke (ischemic or hemorrhagic) according to WHO criteria who met the inclusion criteria were enrolled. Serum ferritin estimation was done at the time of presentation within 48 hrs of the onset of stroke. Neurological assessment was done first on the day of admission and then again on day 6.

RESULTS

Out of the 100 patients in the study (64 males and 36 females),55 patients had poor outcome (MRS 3-6) and 45 patients had good outcome (MRS 1-2).Of the 55 patients with poor outcome 46 (83.6 %) had high serum ferritin values(>220ng/ml),whereas out of the 45 patients with good outcome ,only 7 patients(15.5%) had high serum ferritin value. Applying chi square test and p value(<0.001) to this outcome a positive correlation was found to be present between high serum ferritin values within 48 hrs of stroke, and poor prognosis within 6 days of stroke.

CONCLUSION

This study demonstrates that patients with elevated serum ferritin levels measured within 48 hrs of stroke had a poorer outcome when compared to patients with lower levels of ferritin, 6 days after the onset of stroke. Thus serum ferritin estimation which is a simple and cost effective investigation may be helpful in predicting future adverse neurovascular events and outcome.

Keywords: Stroke, Serum ferritin

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Abstract

EVALUATION OF PATIENTS WITH UPPER GASTRO-INTESTINAL BLEEDING COMING TO M.K.C.G. MEDICAL COLLEGE & HOSPITAL, BERHAMPUR

Asish Kumar Swain[1], Jagannath Sarangi[2]

INTRODUCTION

Upper GI bleeding is one of the most commonly seen clinically entity encompass many different scenario requiring more than 300,000 hospitalisation annually. The overall incidence of upper GI bleeding has been estimated to be 50-100 per 1,00,000 population per year with an annual hospitalisation rate of approximately 100 per 1,00,000 hospital admission. Bleeding from upper GI bleeding is approximately five times more common than bleeding from lower GI tract. Historically the most common cause of upper GI bleeding has been gastro-duodenal ulcer disease but now-a-days the trend is increasing towards other causes of upper GI bleeding due to CKD other causes of upper GI bleeding due to CKD, cirrhosis of liver etc. which are evaluated in this study. Clinically all these patients were presented with hematemesis and melena.

MATERIALS AND METHODS

The study was conducted in M.K.C.G. Medical College and Hospital, Berhampur, Odisha from 2018 to 2020. Hundred twenty five of upper GI bleeding patients were included in the study after fulfilling inclusion and exclusion criteria along with vitals and routine examination, upper GI endoscopy and ultrasonography.

RESULTS

Among the study participants, 46.4% of patients were found to be having peptic ulcer disease overall. Duodenal ulcer is the most common endoscopic findings overally and also among the acid peptic lesions, followed by Gastric ulcer, gastric erosion respectively. Varices accounts for 14.4% of cases. Melena is the most common clinical presentation.

CONCLUSION

In my study, prevalence of peptic ulcer disease was more in patients with upper GI bleeding. Hence, all patients with recurrent peptic ulcer disease and elderly should be screened for H. pylori infection work up and endoscopic biopsy.

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Abstract

DYSFUNCTIONING OF KIDNEY AS UTILITY OR FUTILITY TO DEFINE ACUTE ON CHRONIC LIVER FAILURE

Pawan Sharma[1], Ambika Prasad Mohanty[2], Dibyalochan Praharaj[3]

INTRODUCTION

Acute-on-chronic liver failure (ACLF) is acute deterioration of liver functioning in patients with chronic liver disease.

Widely accepted definition is:

European Association for Study of the Liver (EASL): an acute deterioration of pre-existing chronic liver disease, usually related to a precipitating event and associated with **increased mortality** at 12 weeks due to multisystem organ failure.[i]

Grade 1: Kidney failure or dysfunction.

Grade 2: two organ failure. Grade 3: Three or more organs failure.

"Is it necessary that patient has to die to define the illness ?"

"Presentation not necessarily to be liver failure ?"

"Kidney dysfunction is must part for acute failure of liver ?"

AIMS AND OBJECTIVES

To study the involvement of kidney dysfunction or failure in patients of ACLF and evaluate the necessity of kidney failure for defining ACLF.

MATERIALS AND METHODS

Single centre prospective observational study Sept. 2019 to Aug. 2020

CONCLUSION

Kidney dysfunctioning/ failure may or may not be the associated with acute liver insult in patients with chronic liver disease. So kidney failure is a sequelae of the disease, not the characteristic feature of ACLF. Kidney failure is not the utility to define ACLF.

REFERENCES

1. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, Durand F, Gustot T, Saliba F, Domenicali M, Gerbes A, Wendon J, Alessandria C, Laleman W, Zeuzem S, Trebicka J, Bernardi M, Arroyo V; CANONIC Study Investigators of the EASL-CLIF Consortium. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. Gastroenterology. 2013;144:1426–1437. doi:10.1053/j.gastro.2013.02.042
2. Sarin SK, Kedarisetty CK, Abbas Z, Amarapurkar D, Bihari C, Chan AC, et al. Acute-on-chronic liver failure: consensus recommendations of the Asian Pacific Association for the Study of the Liver (APASL) 2014. Hepatol Int [Internet]. 2014 Oct 26 [cited 2018 Jan 30];8(4):453–71

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Abstract

STUDY OF RENAL ADVERSE EFFECTS OF NSAIDS USED IN SPONDYLOARTHRITIS PATIENTS

Gautam Kumar Chaudhary[1], Chandan Das[2], J R Parida[3], Shree Dash[1]

INTRODUCTION:

Spondyloarthritis is a diverse group of arthritis feature by inflammation in the axial skeleton and enthesis.(1) Spondyloarthritis comprises of ankylosing spondylitis, reactive arthritis, arthritis, or spondylitis associated with Psoriasis, arthritis or spondylitis related to inflammatory bowel disease.(3) Spondyloarthritis may manifest with vague symptoms. Mostly present with back pain and stiffness. (3)The prototype of Spondyloarthritis is ankylosing spondylitis.

1.3 AIMS AND OBJECTIVES

AIMS:

To study the association of use of NSAIDs and early changes in kidney biomarker(cystatin-c) in spondyloarthritis patients.

OBJECTIVES:

1. To study early changes in kidney biomarker(cystatin-c) after the use of NSAIDs in Spondyloarthritis patients.
2. To study the relation of the duration of use of NSAIDs in Spondyloarthritis patients and the incidence of subclinical kidney injury by comparing serum creatinine with serum cystatin-c.

MATERIALS ANDMETHODS

A hospital based prospective observational study carried out over a period of one year in IMS and SUM hospital over 31 patients on spondyloarthritispatients.where level of serum creatinine and cystatin-c level calculated on baseline,four week and twelve week.

RESULTS

Patients using different type of NSAIDs there is no significant change in serum creatinine value ($p=0.546$).while significant change in serum cystatin-c level ($p<0.012$)over a period of twelve week

CONCLUSION

There is no significant change in serum creatinine value after intake of NSAIDs.while significant change in serum cystatin-c value which increase two or three fold higher than initial value and that is concluded that serum cystatin-c can be used as an early biomarker for subclinical kidney injury than serum creatinine.

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Abstract

PROGNOSTIC MARKERS OF SEVERITY IN SCRUB TYPHUS

Gayathri C., Prof. M.K. Mohapatra

INTRODUCTION

Scrub typhus, a mite borne tropical infection caused by the organism Orientia tsutsugamushi, has been prevalent in the world since third century especially in the endemic regions of tsutsugamushi triangle. The causative organism has been found to have diverse phenotype and genotype which is responsible for the wide clinical spectrum of the disease. The vector mites which require preferred environmental conditions and biotopes determines the geographical distribution of the infection. There has been a clear re-emergence of infection due to unplanned urbanization and deforestation leading to displacement of vectors. It is now the third most common cause of acute febrile illness in south east Asia and present as flu like illness simulating other common tropical infections like malaria, leptospirosis, chikungunya, dengue fever, etc. making early diagnosis and treatment difficult. The delay in treatment can cause fatal complications due to its widespread vasculitis like pathology. However, injection doxycycline has been found to be the drug of choice for effective treatment and prevention of systemic complication and mortality. Hence thorough knowledge on prognostic markers of severity helps in early diagnosis and treatment to prevent complications and mortality.

METHODOLOGY

The observational study was conducted in VIMSAR, Burla, during the period October 2018 to October 2020. The study population included 155 patients, who had presented with acute febrile illness and were diagnosed with scrub typhus infection by rapid IgM slide test (Immunochromatographic test) selected by non-probability convenience sampling. Detailed history taking, thorough physical examination and monitoring and laboratory investigations were done and recorded throughout the hospital stay. The data was analyzed by chi square tests and student t tests using using Microsoft Excel 2019 and SPSS 26.0.

RESULTS

Highest incidence was found during monsoon and post monsoon season, in the westernmost districts of Odisha where scrub regions and deep forest areas are prominent, which include Bargarh (36.8%) and Sambalpur (12.3%).

Among 155 patients, there was a male predominance with 55% males and 45% females. The young working population was more affected (26.5% in 31-40 years, 25.2% in 21-30 years). Age group 21-30 years was found to have good prognosis and older age groups > 51 years bad prognosis.

Regarding comorbidities, 23.2% had dyslipidemia, 11.6% had HTN, 11% had CKD and 9% had type 2 DM. Presence of hypertension (OR=3.996, 95% CI = 1.35-11.85, p=0.008) and type 2 diabetes mellitus (OR=3.68, 95% CI = 1.1-12.3, p=0.026) were found to be the significant prognostic markers of severity.

The most common clinical presentation was fever with a mean temperature of 100.630F and an average duration of 8.85 + 3.5 days. The most common associated symptom was headache (68.4%) and vomiting (67.7%). The symptoms with prognostic significance include vomiting (OR=2.3, 95% CI = 1.7-4.3, p=0.001), abdominal pain (OR=3.14, 95% CI = 1.6-6, p=0.001), decreased urine output (OR=18.65, 95% CI = 8.2-42.3, p<0.001), generalized swelling (OR=13.6, 95% CI= 6.2-29.2, p<0.001), jaundice (OR=11.8, 95% CI = 1.7-4.3, p<0.001), breathlessness (OR=4.1, 95% CI = 1.8-9.1, p<0.001) and altered sensorium (OR=7.81, 95% CI = 3.13-19.5,p<0.001).

On physical examination, the signs with prognostic significance were hypotension in 36.8% (OR=3.7, 95%

CI = 1.9-7.4, p<0.001), tachycardia in 23.2% (OR=2.2, 95% CI = 1- 4.7, p=0.037), tachypnoea in 34.9% (OR=4.5, 95% CI = 2.3-9.2, p<0.001) and desaturation in 29.7% (OR=4.8, 95% CI = 2.3-10.2. p<0.001), icterus in 36.1% (OR=10.34, 95% CI = 4.8-22.3, p<0.001), edema in 36.1% (OR=12.1, 95% CI = 5.5-26.7, p<0.001), ascites in 13.5% (OR=7.14, 95% CI = 2.3-22.4, p<0.001), tender hepatomegaly in 38.1% (OR=10.8, 95% CI = 5-23.2, p<0.001), splenomegaly in 13.5% (OR=3, 95% CI = 1.2-8, p=0.02) and neck stiffness in 14.2% (OR=2.6, 95% CI = 1-6.7, p=0.037). The eschars were seen in 29% cases with most common sites being abdomen (29%), back of trunk (13%), axilla, breast and chest (9%), neck and foot (7%), inguinal region and scrotal base (4%), shoulder, gluteal region and legs (2%). Lymph nodes draining the sites of eschar were enlarged in 16.2%. However, presence of eschar or lymph node enlargement did not have prognostic significance.

On analysis of CBC, the most common abnormality was found to be thrombocytopenia in 67.1% and microcytic hypochromic anemia in 63.86%. Prognostically significant results were lymphocytosis(p=0.017), leukocytosis > 8745 cells/micro L which is 77.6% sensitive and 54.5% specific (p<0.001) and neutrophilia > 5435 cells/micro L which is 71.6% sensitive and 55.7% specific (p<0.001) in predicting severity.

The biochemical abnormalities with prognostic significance were total bilirubin > 1.99 mg/dL with 68.7% sensitivity and 86.4% specificity (p<0.001), direct bilirubin > 1.43 mg/dL with 68.7% sensitivity and 87.5% specificity (p<0.001), liver enzymes AST (p<0.001), ALT (p<0.001) and ALP of value > 234.5 with 56.7% sensitivity and 88.6% specificity (p<0.001), hypoalbuminemia < 2.85 mg/dL with a sensitivity of 62.7% and specificity of 75% (p<0.001), serum urea > 49 mg/dL (p<0.001) with a sensitivity of 77.6% and 73.1% and specificity and serum creatinine > 1.85 mg/dL (p<0.001) with 76.1% sensitivity and 86.4% specificity in predicting severity.

CONCLUSION

Scrub typhus presents as acute febrile illness with a specific pattern of epidemiology, symptoms and signs. Clear understanding of prognostic markers of severity is necessary for a physician to suspect severe infection as early as possible. Scrub typhus is one of the most common treatable cause of acute febrile illness. Once the case is diagnosed, patient has to be monitored for the prognostic factors to predict the progression to severity. Delay in treatment leads to development of systemic complication due to the widespread vasculitis like pathology causing mortality. This can be prevented by early treatment with doxycycline or azithromycin.

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NOTES