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Editorial

REVERSION OF DIABETES

JK Panda[1], SK Dhar[2]

Reversing diabetes is a term that usually refers to a significant long-term improvement in insulin sensitivity in people with type 2 diabetes.

People with type 2 diabetes are able to get their HbA1c below 6% without taking diabetes medication are said to have reversed or resolved their diabetes. This also known as putting diabetes into remission. Loss of body weight can be particularly beneficial in helping to reverse the progression of diabetes. With time and dedicatio, type 2 diabetes can be reversed and the results can be very rewarding.

UNDERSTANDING HOW DIABETES PROGRESSES

The most common cause of type 2 diabetes is obesity-related, which generally follows a vicious cycle pattern:

- Diet high in calories -particularly if high in refined carbohydrates.
- Insulin levels in the bloodstream rise to cope with the high- and quick-acting carb intake.
- Weight is gained around the belly (central or truncal obesity).
- Consistently high insulin levels lead to the body's cells becoming resistant to insulin and commonly lead to weight gain.
- High insulin levels also increase weight gain.
- Insulin resistance leads to an increase in blood sugar levels, particularly after meals.
- The pancreas produces more insulin to cope with rising blood sugar levels
- High sugar levels lead to feelings of lethargy and high insulin levels lead to increased hunger.
- Hunger often leads to overeating and lethargy, with less physical activity being taken.
- Overeating, less activity and high insulin levels all lead to further weight gain and more insulin resistance.
- Consistently high demand on the pancreas to produce extra insulin leads to damage of the pancreas' insulin-producing beta cells.
- Beta cell damage results in the body struggling to produce enough insuli, and steeper rises in blood sugar levels leads to more recognisable symptoms of diabetes, symptoms of diabetes, such as thirst and a frequent need to urinate

BREAKING THE PROGRESSIVE CYCLE OF TYPE 2 DIABETES

To reverse diabetes, we need to be able to break this cycle by taking the strain off your insulin-producing Beta cells.

Research indicates that effective ways to reverse diabetes include:

- Low-carbohydrate diets
- Very low calorie diets
- Exercise
- Bariatric surgery

LOW-CARBOHYDRATE DIETS

Low-carbohydrate diets are known for lowering the amount of insulin the body needs to produce, resulting in less insulin resistance.

A study published in 2014 by the Second University of Naples showed that a low-carbohydrate Mediterranean diet was able to achieve significant rates of remission in people with type 2 diabetes. After one year of following the diet, 15% of participants achieved remission and, after six years, 5% had achieved remission on the diet – a stunning achievement.

By comparision, low-fat diets were not as effective in the study. After one year, 4% of participants on a low-fat diet had achieved remission and, after six years, 0% of participants had achieved remission.

Besides regular exercise and metabolic surgery also causes the so called reversion.

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Research Article

HOW TO CALCULATE BED OCCUPANCY RATE ?

MK Mohapatra

Patients are admitted to the hospital for curative care. The principal objectives of the curative care are to alleviate the symptoms, reduce the severity, protect against any complication and finally to cure an illness or injury. The admission of number of patients to a hospital is usually expressed as Bed Occupancy Rate (BOR). It is usually calculated as:

BOR= number of beds occupied (bed days) for curative care × 100/Number of available beds × 365 days[1]. Occupancy is a commonly used measure. However, there is inconsistency in its measurement[2].

Occupancy less than 80% is uneconomical and 100% means overutilization. 80 to 85% BOR is ideal for good quality of patient care. 15-20% beds are kept emergency, maternity care, intensive care.

But in hospitals of Odisha there is a surge of inpatient admission.Further in our state Medical College Hospitals there are multiple units and almost one unit is responsible for admission of patients on a day. In that situation the formula mentioned above is not suitable for assessing the BOR. Further to procure the new beds, medicines, and other requirements in daily basis may be required. During inspection of MCI also there is a requirement to mention BOR. The calculation of BOR is simple calculation pf percentage. Therefore, I have derived the following formulas to calculate BOR in daily, weekly, monthly, and annual basis for better analysis.

Formula-1 :

BOR in a Day in a Unit(Medicine Department) =***Number of patients admitted × 100/ number of Beds in the unit.***

Example: In 2nd unit from 9AM to 9 AM (24 hours) 98 patients (58 males+40 Females)were admitted against 60 beds (30 male + 25 Female). BOR= 98×100/ 55=178.2%.

You can also calculate BOR for male and Female wards separately.

BOR for male ward= 58×100/30=193.3%.

BOR for female ward= 40×100/25=160.0%.

Formula-2 :

BOR in a Day ofthe Hospital = Total ***Number of patients admitted to the Hospital (All Departments in a day e.g. 24 hours) × 100/ Total number of Beds in the hospital.***

Example: 9AM to 9 AM (24 hours) 625 patientswere admitted against 500 beds of the hospital.

BOR for hospital for a day= 625×100/ 500=125.0%

Formula-3 :

Bed Occupancy in a week of Medicine Department = ***TotalNumber of patients admitted in a week to Medicine Dept. × 100/ Total number of Beds in the Medicine Dept.***

Example: 9AM to 9 AM (24 hours) 525 patients were admitted against 330 beds of Medicine Dept.

BOR in a week of Medicine Dept.= 525 ×100/ 330=159.09%

Formula-4 :

Bed Occupancy in a week of Hospital = ***TotalNumber of patients admitted in a week to Hospital × 100/ Total number of Beds in theHospital***

Similarly one can calculate Bed Occupancy in a month and a year.

Formula-5

Rate is the number in a day (24 Hours) and can be calculated from total Bed occupancy divided by by the number of days. If you want to calculate the rate from the Formula 1 and 2 you must divide by 7. When

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you calculate from a month, divide by 30 days and when you derive it from a year you divide by 365 days. The formula given below has been derived from yearly turn over hence divided by 365 days.

BOR In Medicine= ***TotalNumber of patients admitted in a year to Medicine Dept. × 100/ Total number of Beds in the Medicine Dept.×365 days.***

Formula-6:

BOR In Hospital= ***TotalNumber of patients admitted in a year to Hospital × 100/ Total number of Beds in the Hospital×365 days***

For the calculation of BOR mid night bed census has been carried out in the hospital. For example, the BOR for Monday is based on the bed census taken at 0000 hours Tues day. As our outdoor starts from 9 AM and admission to an unit continues to next day 9 AM, one can take total number of patients admitted 9 to 9 as 24 hours to calculate BOR.

What is the ideal hospital occupancy rate?

Anybody will intuitively opine that 100% full in any commercial venture. For example, if the stadium is full then sport is a grand success for the organisers. Unlike the stadium or flight ticket the 100% occupancy of a hospital is not desirable neither financially nor for quality service. If the BOR is low (<50.0%) then many staffs will sit idle without work causing loss of money. If the occupancy is too high 100% or more, then there will be excess workload that can hurt morale and quality of service to the patients. Further, that will increase the turn away rate decreasing the duration of hospital stay affecting the recovery of patients and reputation of the hospital. Therefore, the most ideal BOR is 85%[3].

Another way to compare BOR is to convert it into turn-away. Patient turnaway is the inability to provide a needed inpatient service due to lack of beds. Queuing for beds results in cancelled operations, placing of patients on floor of the hospital. There is a possibility of hospital errors, cross infection, and patient deaths[4].

i.e. the percentage of time that a

Other Indices: Apart from BOR there are health care indices. Those are

i)Average length of stay (ALOS): This refers to the average number of days that a patient stays in a hospital. It is calculated using the following formula:

ALOS=(Inpatient days)/Admissions

ii) Bed-Turn over Rate (BTR): The turnover ratio is a measure of productivity of the hospital beds. It is the average number of patients cared for a bed during a given period.

Hospital BTR = Number of discharges (including deaths) in a given time / number of beds in the hospital during that time period.

It is an important measure of hospital utilization index[5].

By using thesesimpleformulas, we can make studies related to hospital indices of our Medical College Hospital for requirement of beds, faculty, and other resources.

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Original Article

STUDY OF CORONARY RISK FACTORS IN TYPE 2 DIABETES MALE PATIENTS HAVING ERECTILE DYSFUNCTION (ED)

S. Dutta

ABSTRACT:

Background

As per WHO 80 million people will be diabetic in India by 2030. India for a long time has sported the title of 'Diabetes Capital of the World', but because of the embarrassment that would arise due to the true levels of ED, a lot of erectile dysfunction cases are not recorded and subsequently diagnosed having cardiovascular disease . ED is a well known fore runner/ Clinical marker of CVD.

Aim

Erectile dysfunction (ED) is a common disorder of aging male and about 50% of the ED sufferers are type 2 diabetic men in India. As ED is strongly correlated with cardiovascular diseases, we explored how many patients with ED aged 40 to 69 years will develop cardiovascular disease in our location and, rationalize if and which preventive measures are available to reduce cardiovascular risks in this population.

Method

79 type 2 adult male patients were included and were comprehensively evaluated. All patients underwent ED Screening by International index of erectile function questionnaire and Focused physical examination for ED. Framingham risk functions were used to determine the 4 to 12 year coronary heart disease risk. The results were extrapolated to the Indian adult male population.

Results

In the age group 40 to 49 years and no significant difference was detected in coronary heart disease risk between patients with and without Erectile Dysfunction. In the age group 50 to 59 years patients with diagnosed ED showed a significantly increased risk to develop coronary heart disease.

Discussion:

Screening on cardiovascular risk factors and taking preventive measures is recommended in Diabetic men with ED. Diabetic Men having ED aged 50 to 59 years are especially prone to develop coronary artery disease and therefore should be aggressively investigated and manged in terms of Life Style Modification as well as pharmacointervention.(2)

Conclusion:

Aggressive management of above risk factors will not only improve erectile dysfunction but also prevent development of more serious complications such as CHD and stroke

1. Framingham Risk Assessment Scale should include ED as a potential risk factor (for Male) and all ED patients should be aggressively searched for underlying CHD.

2. Use of PDE5 Inhibitor may be cardioprotective in diabetics with ED

Reference:

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Original Article

PROGRESSION OF DIABETIC RETINOPATHY IN PREGNANCY

D. Hota[1], S. Swain[2], J.K. Panda[3]

Abstract :

Purpose : The objective of this study is to assess the rapidity of progression of retinopathy in pregnant diabetic women during gestational period with correlation with glycemic control.

Materials & Methods :

This was a prospective observational study of patients admitted/ visited to the department of Medicine, Endocrinology, Ophthalmology & Obstetrics & Gynecology in a tertiary health care system in State of Odisha during January 2016 to December 2017.The study included total 50 pregnant with diabetes vs 50 non-pregnant diabetes for progression of retinopathy during their gestational period.

Results :

At the end of study, 32.5% of cases in the study group showed progression of retinopathy staging during pregnancy as compared to 14% in non-pregnant women. The glycosylated hemoglobin, duration of diabetes, current age were evaluated as risk factors for progression of diabetic retinopathy. **Conclusion-** Pregnancy, level of glycemia (HbA1c) & age are risk factors for acceleration of Retinopathy progression.

Keywords :

Pregnancy, Diabetic retinopathy, HbA1c

Introduction :

Diabetes mellitus is a disorder in which the level of blood glucose is persistently raised above the normal range.[1] DM is a chronic disorder with multi organ involvement having many microvascular and macrovascular complications.[2] Microvascular complications are caused by chronic hyperglycaemia, whereas macrovascular complications are caused by both chronic hyperglycaemia and the consequences of insulin resistance.[3] Diabetic Retinopathy (DR) is the most common microvascular complication of Diabetes Mellitus (DM). Pregnancy is a major risk factor for the progression of Retinopathy & is definitely associated with increased prevalence & severity of Retinopathy compared to non-pregnant diabetic women.[4] The risk factors for the progression of Retinopathy in pregnancy are severity of Retinopathy at conception, glycemic control, duration of diabetes, age of pregnant women, hypertension. Pregnancy itself found to be an independent risk factor.[5]

The objective of this study is to assess the rapidity of progression of retinopathy in pregnant diabetic women during gestational period with correlation with glycemic control.

Materials & Methods :

This study conducted as prospective observational study of patients admitted/visited to the Department of Medicine, Endocrinology, Ophthalmology & Obstetrics & Gynecology in a tertiary health care system during January 2016 to December 2017. The study includes total 50 pregnant with diabetes vs 50 non-pregnant diabetes for progression of Retinopathy during their gestational period.

Inclusion Criteria :

* Diabetic pregnant women of either 5 years or more duration

* Any type (type-1/type-2/other type of known diabetic women)

Exclusion Criteria :

* Other causes of Retinopathy/Prior retinal diseases

* Gestational diabetes

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[2]Associate Professor, Dept. of Ophthalmology.
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Study Design :

In this study minimum 50 numbers of pregnant diabetic of either 5 years or more duration are selected as case. These pregnant ladies are evaluated for glycemic status (FBS, PPBS, HbA1C), any Retinopathy changes by direct & Indirect Ophthalmoscopy, fundus photography as soon as pregnancy is confirmed by Urine pregnancy test.

Diagnosis & Staging of Retinopathy :

For retinopathy changes evaluation by direct & Indirect Ophthalmoscopy, fundus photography was done for any fresh changes in Retinopathy during each ANC. Depending upon changes/development of Retinopathy, patients are staged according to International Clinical Diabetic Retinopathy Disease Severity scale for DR. (No apparent retinopathy, mild NPDR, moderate NPDR, severe NPDR, PDR).

Observation :

TABLE-1 No. OF Cases with RETINOPATHY at Baseline

	DIABETIC PATIENT		TOTAL
	PREGNANT	NON-PREGNANT	
R^+	9(18%)	12(24%)	21
R^-	41(82%)	38(76%)	79
TOTAL	50	50	100

At baseline only **18%** have retinopathy in diabetic pregnant as compared to 24% of non-pregnant.

TABLE-2 No. of cases with RETINOPATHY AT LAST VISIT

	DIABETIC PATIENT		TOTAL
	PREGNANT	NON-PREGNANT	
R^+	22(55%)	15(30%)	37
R^-	18(45%)	35(70%)	53
TOTAL	40	50	90

At the end of pregnancy, 10 patients could not visit for follow up. At last visit 55% cases had retinopathy as compared to 30% in the control group. On comparison at the end point of study, progression of retinopathy in pregnant diabetic patient is much higher as compared to other.

R- (No retinopathy) R+ (retinopathy) D (lost to follow up)

At start of study 82% of pregnant women had no Retinopathy as compared to 76% of cases in non-pregnant group. At the end of study, 45% of pregnant women had no retinopathy but 55% have various stages of Retinopathy as compared to 30% in the control group.

TABLE N0.3. STATUS OF DR AT END POINT OF STUDY

		DIABETIC	
		PREGNANT	NONPREGNANT
STATUS OF DR	NO PREGRESS	27(67.5%)	47(94%)
	PROGRESS	13(32.5%)	3(6%)
Total		40	50

At the end point of study, 32.5% of the Cases in the study group showed progression of retinopathy staging during pregnancy as compared to 6% in non-pregnant women. So we concluded that pregnancy is a risk factor for acceleration of retinopathy progression.

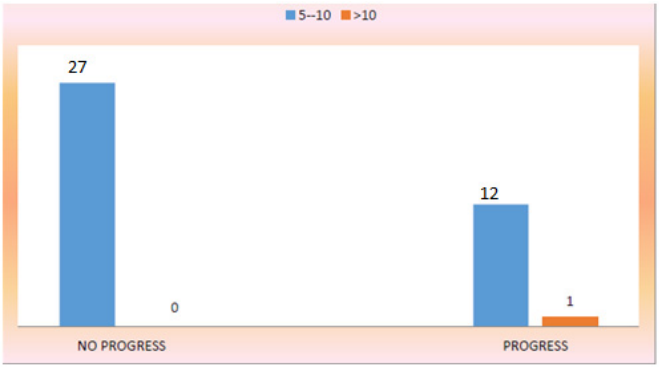
Table No.4 Risk Correlation between Retinopathy and HbA1C (Study Group)

		DIABETIC	
		PREGNANT	NONPREGNANT
STATUS OF DR	NO PREGRESS	27(67.5%)	47(94%)
	PROGRESS	13(32.5%)	3(6%)
Total		40	50

Among diabetic pregnant, patient having HbA1C <8 (80% show no progression of retinopathy). But HbA1C >8 (70% shows progression of retinopathy).so there is significant risk of progression in relation to long term glycemic control.

Graph 1 shows duration of DM Vs retinopathy progression.

Graph 1: Duration of DM Vs retinopathy progression.



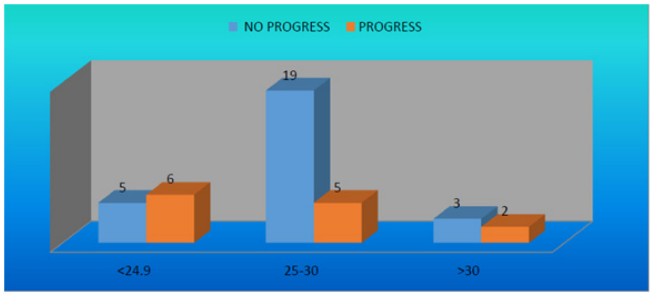
P value was 0.143. As the p value >0.05, so there is no significant difference in term of duration diabetes in both group, because of small sample size, very few pregnant cases more than 10 years.

TABLE NO.5. AGE VS RETINOAPTHY PROGRESSION(Study Group)

TABLE NO.5. AGE VS RETINOAPTHY PROGRESSION (Study Group)					
		AGE GROUP (In Year)			P VALUE
		20-24	25-30	>30	
STATUS OF DR	NO PROGRESS	2(66.7%)	16(66.7%)	9(39.1%)	0.032
	PROGRESS	0	3(12.5%)	10(43.4%)	

Among age group 25-30 years & more than 30 years progression of retinopathy occurs more frequently as age progresses (43.4% in age group> 30 years & 12.5% age group 25-30 years). As p <0.05, so more is age more likely there is progression of retinopathy during pregnancy.

Graph 2 shows BMI Vs retinopathy progression.



Graph 2: BMI Vs retinopathy progression.

P value was 0.109. As p>0.05, BMI & progression of retinopathy is non-significant.

Discussion :

All the above data collected as per study proforma & analysed by SPSS 21 using chi-squre test & unpaired t test to show significant P value.

At base line, 82% has no Retinopathy, 10% had mild Retinopathy & 8% had moderate degree. As the pregnancy proceeds more chance or progression depended upon base line DR & higher grade of Retinopathy. Sampson MJ et al study showed that Progression was more significant in women with moderate and severe forms of retinopathy compared to women with mild or no retinopathy at conception.[6] Maximum progression occurred in end or 2nd trimester or 3rd trimester. At end of study 30% had mild Retinopathy & 12% had moderate Retinopathy. 32.5% had progression of retinopathy staging during pregnancy as compared to 6% in non-pregnant diabetic women. PS Mallika et al although pregnancy does not have any long term effect on DR, progression of retinopathy changes occur in 50%-70% of cases.[7] Pregnant women having HbA1C <8, 20% showed progression of retinopathy, whereas patients having HbA1C >8, 70% showed progression of retinopathy. P <0.043 which significant. So there is strong correlation between glycemic control & retinopathy progression. Mathiesen ER et al stated that low incidence of severe progression of retinopathy during pregnancy observed by us in women reaching an average HbA1c of 5.6% from pregnancy week 20 is reassuring for continuing a strategy of strict metabolic control.[8] Duration of DM & progression of retinopathy in pregnant group for which P value is >0.05, non-significance. In Temple RC et al Progression of retinopathy was significantly increased in women with duration of diabetes 10-19 years compared with duration < 10 years (10% vs. 0%; P = 0.007) and in women with moderate to severe background retinopathy at booking (30% vs. 3.7%; P = 0.01).[9] This contradiction study by Temple Rc etal, because of small sample size, few pregnant women >10 years. Age group 25-30 & >30 years progression of retinopathy occur more frequently as the age progresses is 43.4% showed progression in >30 years as compared to 12.5% in 25-30 age group P< 0.05, significant. So more is age more likely there is progression of retinopathy during pregnancy. Kostraba JN et al showed that young age at onset is a protective factor in the long term or whether it only delays the onset of proliferative retinopathy.[10] BMI & progression of retinopathy is non-significant as P >0.05. BMI <24 & >30 there is progression of retinopathy but doesn't hold good in BMI group 25- 29.99. In Klein BE et al study group, the prevalence of being obese was 25.2%.[11]

Conclusion :

Diabetic retinopathy affects over one- third of all people with diabetes &sss the leading cause of vision loss in working age adults. Maximum changes

occurs during 24-30 weeks of gestation though accelerated progression may present through out pregnancy. There should be proper collaboration between physician, gynecologists & ophthalmologist regrading diabetic retinopathy management in pregnancy.

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Update Article

DIABETES AND TUBERCULOSIS

J.K. Panda[1], C. Patra[2]

INTRODUCTION:

Diabetes mellitus (DM) and Tuberculosis (TB) are important health problems globally where burden of diabetes is increasing day by day and tuberculosis is endemic in developing countries. The dual epidemic of TB and Diabetes is now one of the major health concern in the world. There is a sweet symbiosis between Diabetes and Tuberculosis which has been established by many researchers[1-3]. Diabetes being a immunocompromised state is an important risk factor for developing Tuberculosis. In some literatures the risk of getting tuberculosis of a diabetes patient is up to 3 fold[4] higher. Approximately 15% of TB patients are associated with Diabetes across the globe[5]. Tuberculosis also causes impaired glucose tolerance test which contributes to development of diabetes in future. Majority of diabetes and tuberculosis cases are remaining undiagnosed and treatment of the complicated diabetes and tuberculosis is also inadequate.

EPIDEMIOLOGY:

GLOBAL BURDEN OF DIABETES:

The global prevalence of diabetes among adults is 8.5% in 2014[6]. Diabetes prevalence is constantly rising in developing countries. Diabetes is a major cause of blindness, CKD, CVA, IHD and lower limb amputation. WHO estimates that diabetes was the seventh leading cause of death in 2016[7].

INDIAN SCENARIO OF DIABETES :

Indians show increased tendencies for developing insulin resistant as compared to rest of the world and develop DM at an early age[8]. Dietary changes, adoption of more sedentary lifestyles and urbanization are the factors suggested. There are an approximately 69 million people suffering from Diabetes Mellitus in India.

GLOBAL BURDEN OF TUBERCULOSIS:

More than 1.7 billion people *i.e.* 25% of the world population are infected with *M. tuberculosis*[9,10]. The global incidence of TB reached peak around 2003 and then it is declining slowly[10] since . According to the World Health Organization (WHO), in 2017, 10 million people fell ill with TB and 1.6 million died[10]

INDIAN SCENARIO OF TB:

India has a population of nearly 1.35 billion people *i.e.* 17.5% of the world population. India has approximately 2.8 million incident cases per annum with an incidence rate of 217 per 100, 000 per year in 2015. Treatment success for new and relapse TB cases documented was 74%[11] in 2014.

INCIDENCE AND PREVALENCE OF TUBERCULOSIS IN DIABETIC PATIENTS IN INDIA:

Recent studies published in BMC public health (2007) suggest that a DM is major factor for increased incidence of TB in India. 14.8% of pulmonary tuberculosis and 20.2% of smear-positive i.e. infectious tuberculosis may be directly attributed to diabetes.[2] In urban areas Diabetes is also responsible for increased incidence of smear positive tuberculosis *i.e.* 15.2% higher than that in rural areas.

According to recent studies in India 18.4% of people with pulmonary tuberculosis have diabetes and that in the smear-positive group diabetes prevalence is 23.5% . [12]

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IMPACT OF DIABETES ON THE PROGRESSION OF TUBERCULOSIS

Lifetime risk of developing active tubercular disease in a person infected with Mycobacterium is approx. 10%; 5% during the first two years and another 5% later in life. Recent studies have shown that patients with diabetes are at higher risk for the progression of the disease[3] as compared to non Diabetics.

CLINICAL FEATURES:

In diabetic patients the progression of tuberculosis is rapid and its course is aggressive. The spread and flair up of tuberculosis in diabetics depends on glycemic control of the patient.

In few studies clinical presentation of diabetics and non diabetics do not differ much. It was shown that extrapulmonary manifestations are more common in nondiabetics. The mean HbA1C level is higher in diabetes and TB patients as compared to non diabetes TB patients in some studies. TB diabetes patients are older (> 45yrs) age with male preponderance. They present with general ill-health like fever, weakness, apathy, cough, haemoptysis and chest pain. Active TB disease may present atypically with altered symptoms and signs in those with DM. Severity at presentation seems to be related to the degree of uncontrolled hyperglycaemia.

Pulmonary tuberculosis should be considered in diabetes patients presenting with general debilitating illness, fever, weight loss not explained by poor glycemic control alone and diabetes should be considered in a patient who show poor response to anti tubercular treatment.

DIAGNOSTIC WORK UP:

RADIOLOGICAL FEATURES:

The effects of DM on chest radiograph findings are inconsistent. "Atypical" radiological features are seen in these patients as evidenced by frequent lower lung fields involvement and multiple cavitations.

Some authors didn't find any differences in the chest x-ray patterns of pulmonary tuberculosis in diabetics and non-diabetic patients.[13] So it can be concluded that lower lobe involvement with cavitation should raise the possibility of co- existing diabetes in the patient with pulmonary TB.

SPUTUM CONVERSION RATE:

In DM developing sputum positive pulmonary TB is up to five times higher.[14] Sputum smear and culture conversion are important markers for the infectivity of TB and effectiveness of treatment.

A lower rate of sputum conversion at the end of 2 months of the intensive phase treatment is seen in patients with DM. So prolonging the intensive phase of treatment by 1 month in patients who remain smear positive at the end of 2 months, as in the WHO guidelines, may also be valid for diabetic patients.[15]

OUTCOME:

DM adversely affects TB treatment outcomes. The reasons are not completely understood but include the immunomodulatory effects, drug interactions, adverse effects from medications, decreased compliance , reduced bio-availability of the drugs etc. The evidence points to an almost doubling of the risk of death during TB treatment among those with DM .

DM also increases the risk of TB treatment failure and losses to follow-up. The risks of relapse and recurrent TB in those who have completed anti-TB treatment are also higher among those with DM[16] compared with those without DM. Some preliminary evidence suggests that improving glycaemic control can lead to better TB treatment outcomes and reduced risk of relapse and recurrence.

DRUG INTERACTION:

Concentration of anti tubercular drug particularly, rifampicin, is lower in diabetes patients[17]. This is probably associated with the severity of hyperglycemia. Rifampicin and isoniazid have hyperglycaemic effects. Rifampicin induces metabolism and decreases blood level of sulfonylureas, leading to hyperglycemia. It doesn't affect the metabolism of metformin or insulin. Pyrazinamide, also, may result in difficult control of diabetes.

DRUG RESISTANCE:

It is believed that patients with diabetes show a higher incidence of anti-tuberculosis drug resistance. In a study from New York School of Medicine showed that the relative risk of MDR-TB was to be 8.6 in the diabetic group compared to the control group. A greater suspicion for MDR-TB should be considered in diabetic patients as compared to nondiabetics.

Another recent study found insignificant difference in the incidence of MDR-TB in diabetic-TB population. Thus, the relation between diabetes and MDR-TB remains unclear. So further studies are needed especially in India in this context as India is the largest pool of both Diabetes and MDR-TB.

SCREENING:

Screening people with tuberculosis for diabetes mellitus

Routine screening of adult patients with active tuberculosis (TB) disease for diabetes mellitus (DM) should be carried out. The approach to screening should be standardized at the time of diagnosis and registration of TB. The first step is to ask TB patients whether they are diabetic or not, screen using a blood test. First, a single Random Blood Glucose (RBG) is performed and any patient whose plasma glucose e" 6.1 mmol/ l (e" 110 mg/dl) is at risk of DM and must have a second test. The second test is either a single Fasting Blood Glucose (FBG) or a single glycosylated haemoglobin (HbA1c) test. DM can be diagnosed if the HbA1c e" 6.5% (e" 48 mmol/l) or the fasting plasma glucose e" 7 mmol/l (e" 126 mg/ dl). Both these abnormal tests should be confirmed when anti-TB treatment is completed to avoid unnecessary, life-long labelling of the patient as having DM. The TB patient with already known DM must also have a single FBG or HbA1c test to assess glycaemic control.

Screening people with diabetes mellitus for tuberculosis

Even though diabetes mellitus (DM) increases the risk of tuberculosis (TB), the numbers of patients with new active TB disease that can be identified in DM clinics is relatively small. Screening for TB should therefore only be considered in TB endemic areas. Preference is given to people newly diagnosed with DM in whom a one-off active TB case finding screen at the time of registration of DM is recommended. The screening should be done by actively enquiring about symptomatology of TB and referring those with positive symptoms to the TB clinic for investigation. Given the association between DM and drug-resistant TB, Xpert MTB/RIF [CBNAAT for identifying Mycobacterium tuberculosis (**MTB**) DNA and resistance to rifampicin (**RIF**)], the diagnostic test is recommended. Persons with DM already in care should be educated about the risks, symptoms and signs of TB.

TREATMENT:

Management of tuberculosis in persons with diabetes mellitus

The standardised treatment for DM patients with drug-susceptible TB

Patients with new TB: The six-month rifampicin based regimen (2HRZE/4HR) is the one recommended for treatment of those with new drug-susceptible or presumed drugsusceptible TB. The initial intensive phase of 2 months consists of isoniazid, rifampicin, pyrazinamide and ethambutol. The continuation phase of 4 months uses just two drugs, usually isoniazid and rifampicin, preferably given by direct observation.

Patients with previously treated TB: On the basis of the drug susceptibility profile, the standard six-month rifampicin based regimen (2HRZE/4HR) can be repeated if no resistance is documented.

Patients with other types of TB: Some experts recommend 9-12 months treatment for tuberculous meningitis and 9 months treatment for TB of the bones and joints because of the difficulties of assessing treatment response. Adjuvant corticosteroid therapy is recommended for tuberculous meningitis and tuberculous pericarditis; more frequent monitoring of blood glucose will be needed in these circumstances because of the deleterious effects of steroids on glucose metabolism.

HIV- associated TB: Some recommend 9-12 months

treatment. More frequent monitoring of blood glucose will be needed if adjunctive corticosteroid therapy is given for the prevention or management of TB-associated IRIS (inflammatory immune reconstitution syndrome) in individuals with DM.

Dosages: The essential drugs and recommended dosages based on body weight are shown in Table-1.

Table 1: Recommended doses of first-line TB drugs for adults with DM

Drugs	*Abbreviation*	*Recommended daily doses*	
		Dose range in mg/kg	Max dose in mg
Isoniazid	H	5 (4-6)	300
Rifampicin	R	10 (8-12)	600
Pyrazinamide	Z	25 (20-30)	-
Ethambutol	E	15 (15-20)	-

Although, three times a week dosing is used in RNTCP (Revised National Tuberculosis Control Program) in the continuation phase, this may pose a risk for the development of acquired rifampicin resistance in patients with or without DM. It is thus recommended that dosing of drugs in both the intensive and continuation phases is DAILY.

For better compliance the daily dosage is usually standardised for three body weight bands –30-39 kg, 40-54 kg and more than 55 kg, as is done with the Global Drug Facility patient kits. (Table-2)

Table 2 : The number of FDC tablets to be given daily for adults on treatment according to weight bands and the contents of the tablets.

Month of Treatment	*Drug*	*Number of FDC tablets taken daily*		
		30-39kg	*40-55kg*	*>55 kg*
	RHZE (R150mg, H75mg, Z400 mg, E275 mg) combined tablets	2	3	4
3 – 6 Continuation Phase	RH (R150mg, H75mg) combined tablets	2	3	4

The drugs in the initial intensive phase and the continuation phase, whether in FDCs or provided in kit form, should be given under direct observation (DOT), where someone watches and observes the patient actually taking medication.

Monitoring of DM patients treated for Drug susceptible TB: The patient's weight should be monitored every month. Adverse effects from treatment need to be detected promptly and managed properly (Table 3). For sputum smear positive, culture positive or CBNAAT positive cases sputum smears are examined for acid-fast bacteria at 2 months, and again at 5 months and 6 months of therapy. For smear negative pulmonary TB cases monitoring with chest radiograph at 6 months is helpful otherwise it is not routinely recommended.

Table 3: The most common adverse effects from the four essential anti-TB drugs, their relationship to DM and the suggested management.

Drug	*Adverse effects*	*Considerations from DM*	*Management*
Isoniazid	Peripheral neuropathy Hepatitis	May be worsened by DM	Give pyridoxine Stop all medication
Rifampicin	Gastrointestinal Hepatitis Red urine	May be worsened by Metformin	Symptomatic therapy Stop all medication Reassure
Pyrazinamide	Arthralgia Hepatitis	Arthralgia and hepatic toxicity may be more common in DM	Aspirin or NSAID Stop all medication
Ethambutol	Retro-bulbar neuritis	May be worsened by DM retinopathy	Stop all medication

The treatment for DM patients with drug-resistant TB

Currently, the treatment of MDR-TB and XDR-TB is similar in patients with and without DM.

Longer MDR-TB regimens: The drugs have been regrouped in to three categories (Table 4). Group A drugs are to be prioritised; Group B drugs are to be added next; Group C drugs are to be included to complete the regimens and when agents from Groups A and B cannot be used. These regimens are usually designed to include at least five drugs. The longer MDR-TB regimens usually last 18-20 months.

Note that kanamycin and capreomycin are no longer recommended and generally use of injectable medicines is discouraged.

Table 4: Drugs recommended for the treatment of MDR-TB

Group	*Name of Drug*	*Abbreviation*
Group A:	Levofloxacin OR	Lfx
Include all three drugs	Moxifloxacin	Mfx
(unless	Bedaquiline	Bdq
they cannot be used)	Linezolid	Lzd
Group B:	Clofazamine	Cfz
Add both drugs	Cycloserine OR	Cs
(unless they cannot be used)	Terizidone	Trd
Group C:	Ethambutol	E
Add to complete the	Delamanid	Dlm
regimen	Pyrazinamide	Z
and when drugs from	Imipenem-cilastatin OR	Ipm-Cln
Groups	Meropenem	Mpm
A and B cannot be used	Amikacin	Am
	(OR Streptomycin)	(S)
	Ethionamide OR	Eto
	Prothionamide	Pto
	p-aminosalicylic acid	PAS

Shorter MDR-TB Regimen: MDR-TB who have not been treated with second-line drugs and in whom resistance to fluoroquinolones and second-line injectable agents has been excluded or is considered highly unlikely, a shorter MDR-TB regimen of 9-12 months may be used instead of a conventional, longer regimen.

Intensive phase 4-6 months: Am-Mfx-Pto(Eto)-Cfz-Z-E-H high-dose

Continuation phase 5 months: Mfx-Cfz-Z-E

Management of diabetes mellitus during tuberculosis treatment:

The management of DM during TB treatment is aimed at improving TB treatment outcomes and reducing DM-related complications. The key activities are optimizing glycaemic control (Table 5) (through dietary instructions and medication) and implementing measures to reduce the risk of cardiovascular disease. Metformin is the first choice oral glucose-lowering drug for TB patients. Sulphonylurea derivates can be used as add-ons or in patients who cannot use metformin althoughdrug-drug interactions with rifampicin limit their use. Insulin is effective in patients with severe hyperglycaemia. Cardiovascular risk assessment should be considered in TB-DM patients through counseling and prescription of anti-hypertensive, lipid-lowering and anti-platelet treatment with the aim of lowering early and long-term cardiovascular morbidity and mortality. Healthy life styles need to be promoted.

Table 5: Targets for glycaemic control during TB treatment

Measurement	*Target*
Fasting blood (capillary) glucose	< 10 mmol/l (< 180 mg/dl)
HbA1c	< 8%

CONCLUSION:

Diabetes is regarded as a major contributor towards increased tuberculosis incidence. In third world countries the diabetes epidemic may thus lead to a resurgence of tuberculosis. Coordinated action and formulation of active action plan is the primary need of this hour. It can be achieved through active case finding, treatment of latent tuberculosis and new research . Similarly, early diagnosis and adequate treatment DM may have a beneficial role on TB control. The sweet symbiosis between DM and TB pose a major threat globally and requires more attention and research.

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Case Report

ASKIN TUMOR : A RARE THORACOPULMONARY TUMOR

Rambabu Attoti[1], A. Malla[2], A. Dash[2], S.P. Sahu[2], S.K. Dhar[3], S. Kabi[3], S. Sahoo[4]

Abstract :

Askin tumor, a primitive neuroectodermal tumor of the thoracopulmonary region, is a rare tumor presenting in childhood and adolescents. We report a case of Askin tumor in 20yr male who presented to us just like a classical case of tubercular pleural effusion but investigations including immunohistochemistry confirmed the diagnosis of Askin tumor

Introduction :

Askintumor was defined by Askin and Rosai in 1979. Askin tumor and primitive neuroectodermal tumor(PNET) are considered as members of Ewing's family of tumors and when located to the thoracopulmonary region the are termed as Askin tumor. Its diagnosis requires multidisciplinary team. Given the rarity of this entity, no region has been validated. Here we present a case of Askin tumor in 20yr old healthy male when presenting complaints of only intermittent fever and non-productive cough.

Case report :

A twenty-year male college student presented in the out patient department with primary complaints of fever and cough for preceding two months. There was no history of chest pain, weight loss, loss of appetite or hemoptysis. Fever was intermittent, cough mostly non-productive. No history of significant past respiratory illness or contact with tuberculosis. He was able to carry out his normal day to day activities without any respiratory discomfort. He had been treated for upper respiratory tract infections with antibiotics, antipyretics and anti-allergic medications.

Upon presentation, he was not sick looking, no respiratory distress. General examination revealed normal BMI, no pallor, cyanosis, lymphadenopathy, clubbing or edema. Vitals revealed tachycardia(110/min), respiratory rate of 18/min, temperature 99.8^0F (axilla), blood pressure of 102/74mmhg.

Respiratory system examination revealed no shifting of trachea or mediastinum. No inter coastal fullness but diminished chest movements on left side. Percussion revealed stony dullness in left hemithorax, with absent breath sounds being the prominent findings on auscultation over left hemithorax. Other system examination was normal.

A provisional diagnosis of left sided pleural effusion, probably tubercular origin was made. And patient was admitted in medicine ward for further management.

Blood reports were normal except for HB 10.2g/dl, ESR 82mm/hr. Renal and liver functions are normal. HIV, HBsAg, HCV were negative. Chest X-ray revealed homogenous opacity in left hemithorax with obliteration of costophrenic angle [Figure 1].

During pleural tap, fluid was totally hemorrhagic, was sent for cytological, biochemical studies, also CB-NAAT. On repeated questioning the patient gave history of chest trauma (trauma history was not taken initially) in the form of road traffic accident in preceding 3 months. But there were no signs of abrasion, rib tenderness or splenic rupture. A second provisional diagnosis of left sided hemothorax(traumatic) was made and chest tube was placed. Additional blood tests to rule out any bleeding diathesis were sent and it revealed no abnormality. Pleural fluid study showed numerous RBC's, lymphocytes, elevated protein and decreased sugar levels. Pleural fluid ADA was within normal reference range and CB-NAAT was negative.

CECT thorax revealed large complex cystic heterogeneously enhancing pleural based mass in left hemithorax with moth eaten erosive destruction of left 8^{th} rib insinuating extra pleural soft tissue extension

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along intercostal space between 8th and 9th rib. Moderate left pleural effusion with compressive lower lobe atelectasis. Left fissural edema thickening with few pleuroparenchymal bands. CECT abdomen was normal.

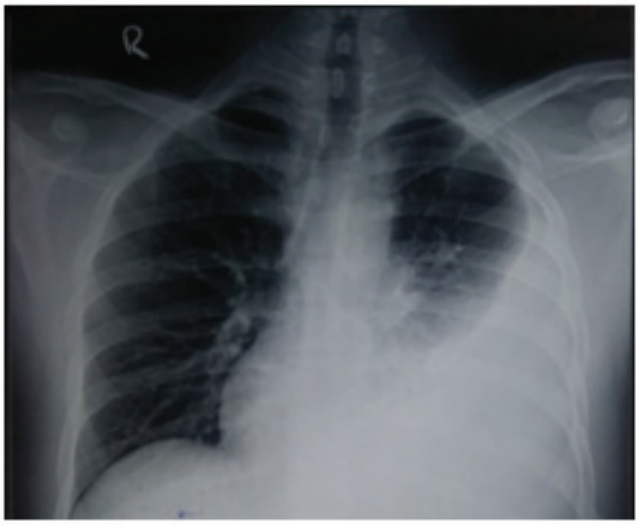
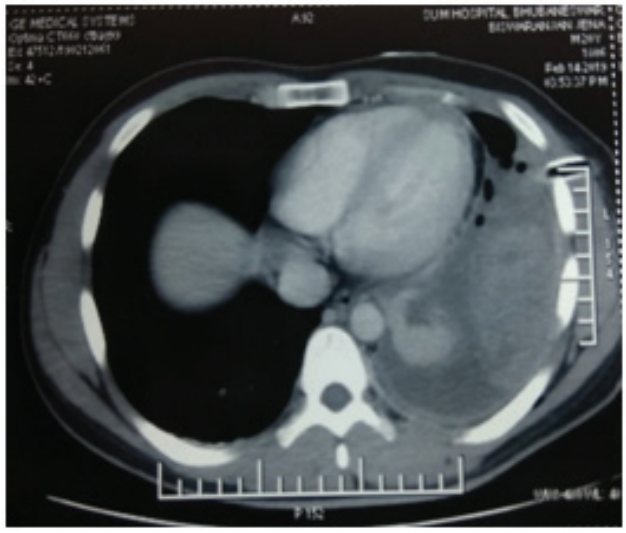
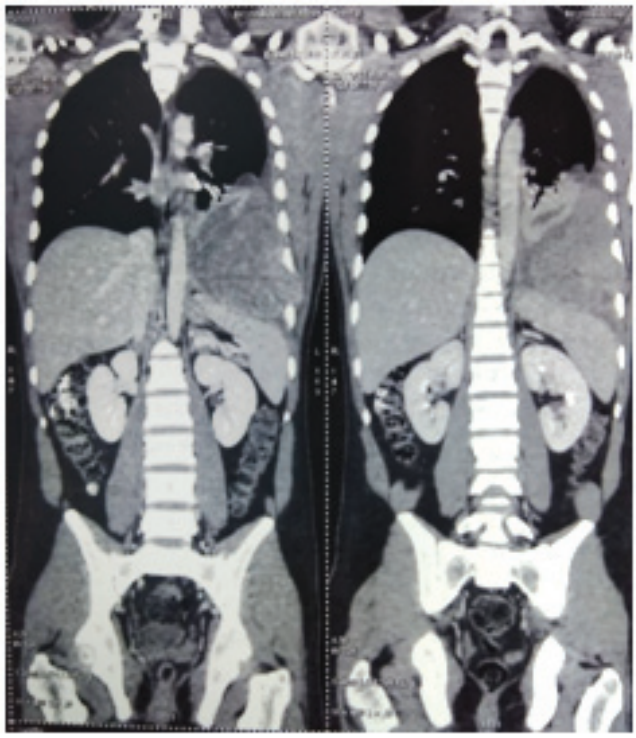


Figure 1: Chest X-Ray depicting a left hemithorax homogenous opacity



Trucut biopsy was taken from mass lesion and it showed malignant small blue round cell tumor with IHC s/o Ewing's sarcoma/PNET/Askin tumor.CD-99, FLI-1 and Synaptophysin +ve; MIB 1 index 40%. Tumor negative for LCA and Chromogranin.

Final diagnosis of Askin's tumor was made and patient was advised to undergo PETscan. Surgical excision and chemotherapy were planned.



PETscan revealed, findings are consistent with metabolically active large necrotic mass lesion in left thoracic cavity involving 8th rib likely sarcomatous neoplasm(?Ewing's sarcoma).Few enlarged metabolically active left axillary and internal mammary nodes likely metastatic. Few pulmonary nodules in both lungs-metastatic.

At the time of writing this case report, patient attendants have opted for other cancer hospital(outside state) to undergo further treatment.

Discussion :

Primitive neuroectodermal tumors(PNETs)- are a group of highly malignant tumors composed of small round cells of neuroectodermal origin that effects soft tissue and bone.

PNETs are classified into following three groups based on tissue of origin,

1. CNS (PNETs) – tumors derived from CNS
2. NEUROBLASTOMA – tumors derived from ANS
3. Peripheral primitive neuroectodermal tumors (pPNETs)– tumors derived from tissue outside the CNS and ANS.

pPNETs are also classified as part of the Ewing's familyof tumors (EFTs).

They both represent different manifestations of the same tumor and have similar genetic alterations. Ewing's sarcoma, however, is more common in bone while peripheral PNETs are more common insoft tissues.

The following tumors are classified as peripheral primitive neuroectodermal tumors

1. Ewing's sarcoma (osseous and extraosseous)
2. Malignant peripheral neuroectodermal tumors or peripheral neuroepithelioma of bone and soft tissue
3. Askins tumor (peripheral neuroepithelioma of the thoraco pulmonary region)
4. Other les common tumors (neuroectodermal tumor, ectomesenchymoma, peripheral medulloepithelioma)

Askins tumor was defined by Askin and Rosai in 1979. This is very rare tumor of childhood and adolescents and usually presents with common respiratory symptoms. Typically, Askin's tumor develops as solitary mass, rarely involving most of the hemithorax or as multiple masses in the thoraco pulmonary region (thoracic wall, lung, mediastinum orpericardium). Also named as 'extraskeletal'Ewing's sarcoma or soft tissue Ewing's sarcoma arising from the soft tissue of the chest wall, shows neural differentiation that can be demonstrated by immunohistochemical and ultrastructural methods. Similar to Ewing's sarcoma and PNETs, these tumors show positivity for neural markers, such as neuron specific **enolase** and also neuroendocrine markers, such as **chromogranin** and **synaptophysin**. These are also positive for **MIC – 2** gene which produces **CD99** and a cell membrane like protein **P30/32** which are highly sensitive but not specific.

Askins tumors are invasive and prone to involve bone (ribs and scapula), invading the retroperitoneal space, and spreading to lymph nodes, adrenals and liver. Median age for these being 14.5 yrs. Pain is the only main symptom in 60% of the cases. Radiological characteristics ranging from a unilateral chest wall mass to pleural fluid, invasion to adjacent lung parenchyma, pulmonary nodules and sometimes lymphadenopathy. The diagnosis of Askin's tumor rests on cytopathological investigation and immunohistochemical tests.

Treatment in Askin tumor consist of radical surgery, neo adjuvant or adjuvant chemotherapy & radiotherapy. As local recurrences after resection & metastasis are frequently seen in Askin tumor, it has a poor prognosis & short survival. The most common recurrence sites are the skeleton, sympathetic chain & the original site. Indications of poor prognosis include advanced age, metastatic disease, extra osseous primary tumor & recurrence.

Various chemotherapy regimens have been used that include VAC (vincristine, actinomycin D& cyclophosphamide), VACA (vincristine, actinomycin D, cyclophosphamide & Adriamycin) & VAC alternating IE (ifsomide & etoposide). Average survival has been reported to be 8 months after the diagnosis.

To conclude,Askin tumor should be considered as an etiological possibility in a small cell tumor of the thoraco pulmonary region, especially in the young age group. Patient with such tumors should be treated surgically, with wide local excision whenever possible. Combination chemotherapy should be considered in patients with inoperable disease.

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Case Report

DECOMPENSATED CIRRHOSIS OF LIVER IN SICKLE CELL DISEASE : A CASE REPORT

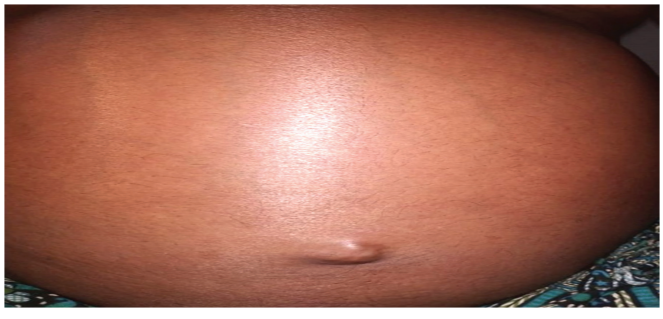
Binod Manthan[1], BK Kullu[2], PC Karua[2]

Introduction :

Hepatobiliary complications of sickle cell disease are relatively rare but well recognised in literature. Although cirrhosis has been reported in sickle cell disease,however no effective therapeutic approaches have been recognised either to prevent or treat this condition.

Case Report :

A 24 year-old-male with sickle cell disease presented with abdominal distension, bilateral lower limb swelling for 2months and blood vomiting for 2days.on examination vital signs were within normal limit but pallor, icterus, bilateral pitting pedal edema was noted. systemic examination revealed hepatosplenomegaly, signs of ascites and portal hypertension.there was no history of blood transfusion and alcohol intake.



Laboratory Investigations:

- HB-6.9g\dl, tpc-45,000, multiple sickle cell noted in peripheral smear, Hb E-ss pattern, HPLC-HbF-17.6%, HbA0-17.1%, HbA2-2.9%, HbS-60.3%.
- Protein-7.3g\dl, albumin-2.5g\dl, LFT-TB-8.2, DB-6.7, AST-104 IU\L, ALT-94 IU\L, ALP29IU\L, PT-23sec(T), 15 sec(C), INR-1.93
- USG of abdomen showed moderate ascites,hepatomegaly(162mm) with an altered echopattern, thickened gall bladder with sludge, dilated portal vein(14.2mm), splenomegaly, spleen is heterogenous with multiple calcifications.
- Ascitic fluid analysis revealed a SAAG 1.5,Upper GI endoscopy-grade 2 bleeding varices.
- Markers for chronic hepatitis B and C are negative and serum ferritin-54ng\ml, considering risk of bleeding liver biopsy was dicided against.
- RFT, ECG, ECHO are normal.
- Patient was treated with blood transfusion,folic acid supplimentation, salt restriction, spironolactone, betablocker. A considerable clinical improvement with reduction in body weight and edema was noted on follow up after 1 month of presentaion.

Discussion :

Various aetiologies for cirrhosis in sickle cell disease include:

1. hypoxic injury due to sickling
2. viral hepatitis due to repeated transfusion
3. gall stone due to hemolysis
4. iron overload due to repeated transfusion and absence of chelating factors
5. chronic alcohol intake.

After ruling out iron overload,chronic alcohol intake and viral hepatitis it was concludes that our patients liver disease due to SCD per se.

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Conclusion :

Evaluation of chronic liver disease should be carried out routinely in all patients with SCD.early identification and prompt intervention prevent or reduce the comlications thus reduce the overall morbidity and mortality associated with SCD.

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Case Report

THYROTOXIC PERIODIC PARALYSIS: A RARE ENTITY…

Sonalika Behera[1], Biswojit Behera[1], A. Kar[2], B. Pattnaik[3], J.K. Panda[4]

Introduction :

Periodic paralysis is an uncommon manifestation of thyrotoxicosis.The patients may not present with typicsal findings of thyrotoxicosis.Delayed diagnosis can kill the ot.So early diagnosis & early intervention can save lives…

Case Report :

A 38yr old female lady presented to our institute SCB MCH, CUTTACK

With sudden onset of weakness of bilateral lower limbs for one day .She noticed when she was getting up from bed in early morning.Muscle cramp is associated with it.NO history offever,vomiting, loss of consciousness ,bladder bowel involvement.Pt is a known case of thyrotoxicosis for 1yr& on regular treatment.No history of Gastrointestinal disorders,diureticuse.Family history was not relevant.

On exam Pt is conscious ,well oriented to time ,place &person.BMI 19 kg/m2 ,PR102/mnt BP 130/ 78 mm of Hg.

CNS-HMF normal, no cranial nerve deficit,motor hypotonia in bl limbs,diminished reflexes(b/lknee,b/l ankle,b/l plantar) ,2/5 power in both LL limbs,no involuntary movements. Sensory,Autonomic nervous system normal,Absent cerebellar ,meningeal signs.

CVS,RESP.,GI system was normal.

Investigations :

CBC Normal,s.k+ 2.5,Na+ 146 meq/l.RBS -118 mg/dl.,Thyroid function test showed FT3 14 microgram/ dl, FT4 29microgram/dl, TSH <0.01 Miu/l suggestive of thyrotoxicosis.Urine electrolytes tests were done to rule out to test for renal tubular acidosis.CXR ,USG abdomen were normal.ECG showed sinus tachycardia.Thus thyrotoxicosis was established as cause of recurrent paralysis in this pt.

Patient was treated with 120 meq of oral potassium over 48 hrs& She got recovered with this medication.No paralysis on discharge.She was discharged with carbimazole 10 mg TDS ,propranolol 40 mg BD for thyrotoxicosis.Hers.k+ was 3.6meq/l .

DISCUSSION:

Periodic palsy in thyrotoxicosis is ver rare .It occurs in age of 20-40 yrs of age.It affects mostly skeletal muscles ,rarely smooth muscles.Respiratory muscles may be affected. A typical attack of this palsy lasts from few hours to days.It occurs in morning or evening time.Asians& Hispanics are more commonly affected & basic defect may be genetically determined.M:F ratio 77:20.Precipitating factors are strenuous exercise,excesscarbohydrate rich ,infection,trauma,coldexposure,emotionalstress,drugs like insulin,epinephrine ..

Cause has been studied.Mostly genetics plays a major role.Association of TPP with HLA DRw8 gene in japaneese pts,A2BW22,AW19B17 genes in chineese pts have been seen.OthernHLA subtypes B5,BW46,genetic mutations KCNE3 also have been studied.Compared with other tissues in body ,human renal tissue contains a higher concentration of messanger RNA for thyroid receptors alpha 1,alpha 2,beta.As the thyroid hormone levels increase ,the hormone response in renal tissue changes ,causes increase in fractional excretion of potassium in various studies.

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Definitive treatment of thyrotoxicosis periodic paralysis consists of management of thyrotoxicosis by medical,surgical,radio active iodine therapy.Acute attack managed by potassium supplements. No potassium supplements in between attacks because in that period s.potassium is normal. Pt got Counselled about precipitating factors.

In this pt proper history was very important to diagnose the disease…

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Case Report

DOWN SYNDROME PRESENTING AS ATRIAL SEPTAL DEFECT

D. K. Bhoi[1], B. Behera[1], H. Satpathy[1], A. Sahoo[2], A. Kar[3], B. Pattnaik[4], J.K. Panda[5]

Abstract:

Down syndrome is a genetic disorder caused when abnormal cell division results in extra full or partial copy of chromosome 21. Down syndrome varies in severity among individuals, causing lifelong intellectual disability and developmental delay. It also commonly causes other medical abnormalities including heart, musculoskeletal, gastrointestinal system and hematological system.

We report a case of Down syndrome of 22years presenting with OS-ASD with Bilateral pneumonia.

Abbreviation:

OS-ASD-Ostium secundum

ALL-Acute lymphoblastic leukemia

AML- Acute myeloid leukemia

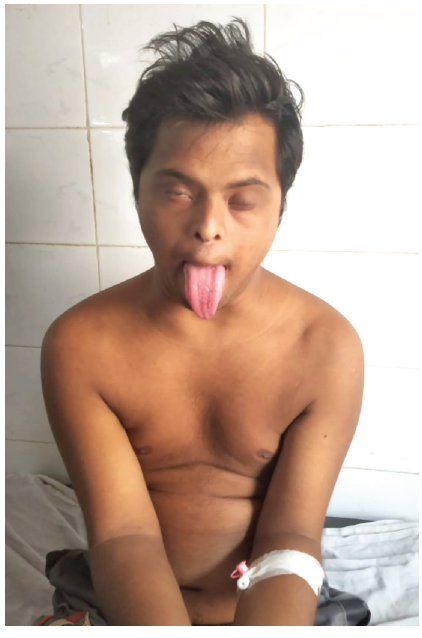
TLPS-Transient lymphoproliferative syndrome

Introduction:

Down syndrome is one of the most common chromosomal abnormalities occur one in one thousand birth children, Trisomy 21 found in 94% cases rest 5% are due to translocation. The heart is affected most frequently along with neuro psychiatric illness and also eye, skin, ligament may be affected. Fifty percent children have risk of congenital heart disease, 40-60% for endocardial cushion defect.

Case Report:

A 22 years old male presented to medicine OPD, SCB Medical College, Cuttack with complaint of fever, cough and breathlessness for 9days. General examination revealed mild pallor, no-icterus, clubbing cyanosis. Lymphadenopathy and edema.



Physical examination finding shows :

1. Stunted growth
2. Short hands
3. Short neck
4. Narrow roof of mouth
5. Flat head
6. Protruding tongue

Hight-148cm, Wt-45kg, Pulse-80/min,regular, BP-110/70mmhg Resp-22/min temp-100°F examination of respiratory system-There is decreased breath sound and fine crepls bilateral infrascapular areas.

Examination of cardiovascular system first heart sound normal and splitting of sound heart sound which

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is fixed in nature. Examination of central nervous system-There is intellectual disability of IQ-60 and no abnormality found. Abnormal examination related no abnormality found.

Abdominal examination-revealed no abnormality.

Laboratory investigations were Hb-9.6%, TLC-6400cu/mm, DC: N-60%, L-36%, E-02%, ESR-12mm, FBS-100, PPBS-152, Urea-75, Creatinine-2.0mg/dl, serum bilirubin (T)-1.1mg/dl AST-110Iu/L ALP-241Iu/L Urine-N, TSH-4.79 Iu/ml FT3-0.29 ng/ml FT4-7.35 mg/dl

Sputum for A+B-negative

2D echo-large OS- ASD with left-Rt shunt

HRCT of thorax:

B/L lung field diffuse GGO with right lower lobe, left lower lobe patchy consolidation.

-Cardiomegaly with minimal pericardial effusion

-F/S/O-Pulmonary edema

-Genetic karyotyp.. was done at the age 7years-Trisomy 21.

With the above finding, the patient was diagnosed with down syndrome with OS-ASD and bilateral pneumonia. He was given course of antibiotic and improved.

Discussion:

People with Down syndrome can have verity of complications, some of which become more prominent as they get older. These complications heart defect, about half of the children with Down syndrome are born with some type of congenital heart defect, that can be life threatening and may require surgery. Gastrointestinal defects include abnormalities of intestine like atresia, GERD etc. and also ophthalmologic problem, hearing defect, Thyroid dysfunction, decrease physical growth i.e lineak growth is retarded … normal children. Patient of down syndrome may increase risk of lymphoproliferative disorders include ALL, AML, TLPS. The diagnosis of down syndrome require genetic…clinical feature, laboratory and pathologic finding.

Hence we report an usual case of a young boy presenting of Down syndrome presenting with breathlessness fever, on CVS examination and echocardiography suggesting a diagnosis of Down syndrome with OS ASD with B/L pneumonia.

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Case Report

AN UNUSUAL PRESENTATION OF LUNG CARCINOMA AS ON/OFF ALTERED SENSORIUM WITH NECK PAIN AS FIRST CLINICAL MANIFESTATION

Sonalika Behera[1], Biswojit Behera[1], A. Kar[2], B. Pattnaik[3], J.K. Panda[4]

Abstract :

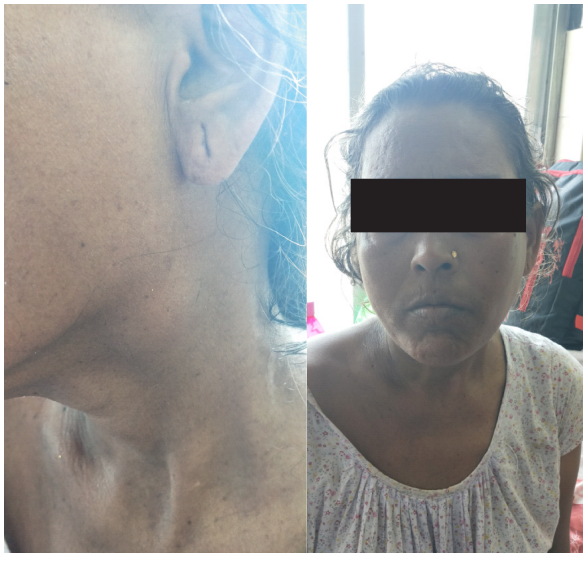
We report a case of lung squamous cell carcinoma metastasing to brain and left cervical lymphnodes.

Case Report :

A 46 yr hindu female was admitted to our hospital with a complaint of on/off altered sensorium and left side neck pain with intermittent vomiting for last 15days. She had no headache ,no convulsion, no shortness of breath since the illness began.

General Examination

• Patient is conscious and oriented, there is mild pallor with clubbing of grade-II in both hand with left posterior cervical lymphadenopathy of size 2 X 2 cm which was tender,firm in conscistancy with regular surface not attached to underlying structure and skin and temperature is not raised.



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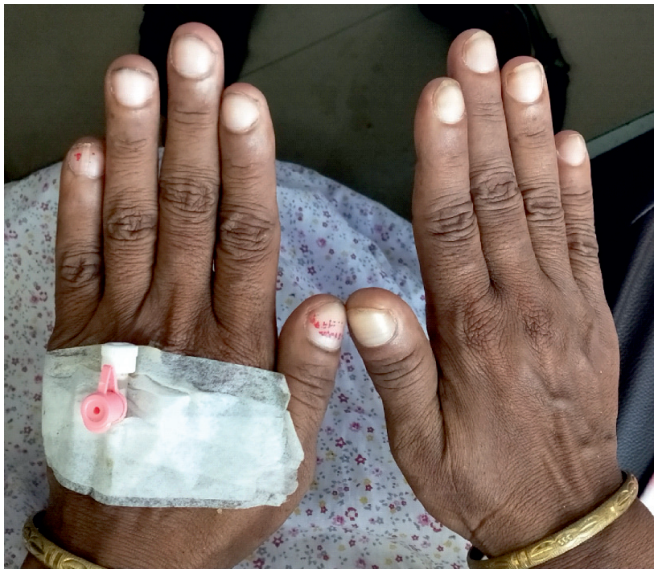


• INVESTIGATION-

Hb-8.6, TLC-11,000 , TPC-2.0, DC-N55,E03,L40,B0,M2

PCV-27,ESR-18

RBS-113, Ur-22,Cr-0.8,Na+=140,k+=3.3

LFT-Totol bil-0.4,direct bil.-0.1,AST-141,ALT-18,ALP-247

HIV,HBsAg,HCV - Negative

FNAC of Lt.post.Cervical lymphnode-

Metastatic squamous cell carcinomatous deposits.

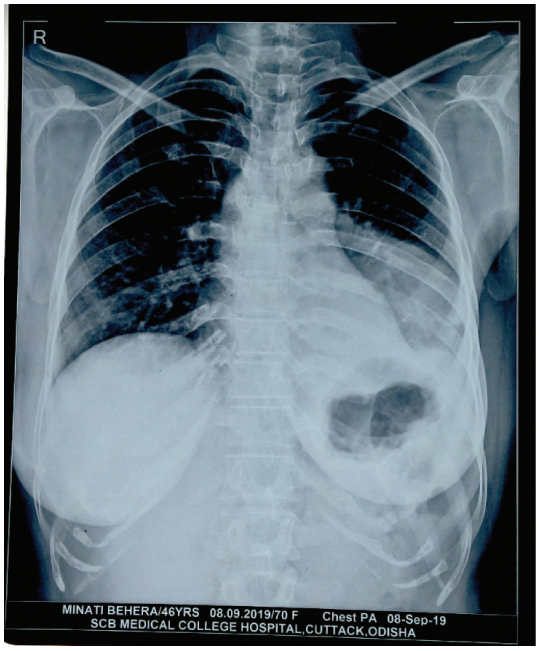
USG of Neck-

enlarged rounded markedly hypoechoic left IB,II,III cervical LNs largest 27x16 mm

USG of Abdomen and Pelvis- mild hepatomegaly otherwise normal.

CHEST X RAY-

left lower lobe homogenous consildation without air bronchogram

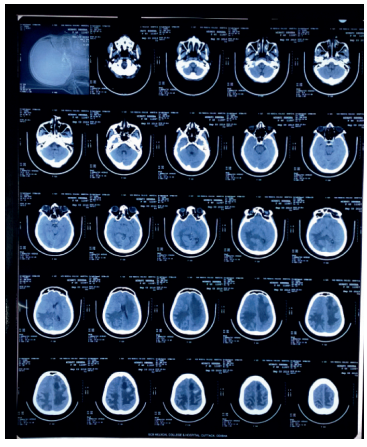


HRCT THORAX-

Heterogenous attenuation lesion in left lowerlobe lung with proximal consolidation and small cavitary areas.

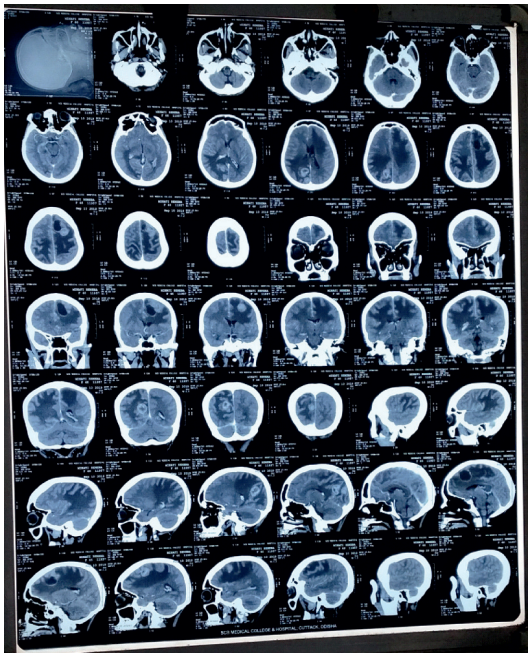
NCCT BRAIN-

irregular hypodense lesions in both frontal lobe and right temporal and occipital areas with hyperdensity in left frontal lobe area.



CECT BRAIN-

right frontal lobe ring enhancing lesion with surrounding edema with hyperdensity within and with adjacent cystic lesion right parietal and occipital lobe similar lesion suggestive of metastases.



DISCUSSION :

Lung cancer is the leading cause of cancer death in both male & female.35% of all lung cancers are assquamous cell cancer.5 yr survival rate is about 15%. 20-40% of adults with squamous cell carcinoma present with brain metastasis .Other common sites of metastasis are adrenal gland,bones,brain,liver,lymph nodes.Prognosis of mets to brain is very poor.Median survival of pts with untreated brain metastasis is reported to be 1-3 months.

Routes of metastasis are through blood, lymphatics, contiguous spread.

Treatment options include chemotherapy, radiation thearpy.

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Case Report

A CASE OF FUO WITH CERVICAL LYMPHADENOPATHY-DIAGNOSED AS KIKUCHI DISEASE

J. Niari[1], P. Jena[2], S. Behera[2], R .Mohanty[3]

ABSTRACT:

Kikuchi-Fujimoto disease, a benign condition of unknown cause characterised by fever and cervical lymphadenopathy . Here we report a case of FUO with cervical lymphadenopathy diagnosed to be a case of kikuchi disease.

INTRODUCTION:

Kikuchi histiocytic necrotizing lymphadenitis is more common in young women . Although initially described in Japan, it is more common in East Asia . The most common clinical presentation is low grade fever that persists for about one week, rarely one month and cervical lymphadenopathy in a previously well young women.

CASE REPORT:

A 24 year old female presented to medicine OPD, S.C.B. Medical College , Cuttack with complaint of high grade fever and bilateral neck swelling for last one month. There is no history of cough, night sweat, arthritis or rash.

During this period she had received treatment with various antibiotics at different clinics. Done thrice her FNAC of cervical lymph node had revealed reactive hyperplasia only.

General examination revealed moderate pallor, bilateral cervical lymphadenopathy {R>L}, 12 in number, largest of size 16x8 mm on right , firm , tender, discrete and mobile. She had no icterus, cyanosis, clubbing, edema. Pulse 104/min, regular; BP-110/70 mmHg; respiratory rate-16/min; temperature -103^0 F. Respiratory, cardiovascular and abdominal examination revealed no abnormality.

Laboratory investigations were:

Hb-7.1gm%, TLC-3800Cu/mm, DC: N-52%, L-46%, E-02%, ESR-100mm, MCV-65fl, RDW-CV-22.5, CPS-Anisocytosis, microcytosis, hypochromia. RBS-106 mg/dl; LFT and RFT with in normal limits; Sr Protein-3.6 gm/dl ,Sr Albumin-2.5 gm/dl; Urine-normal; HIV,HBsAg,HCVAg-Negative; Sr Ferritin-401 ng/ml; ANA Titre 1:100,speckled pattern; USG of neck-B/L multiple enlarged rounded, hypoechoic level IB,II,III,IV lymph node(?Tubercular). USG- abdomen &pelvis-Normal, Chest X-Ray & HRCT Thorax-Normal. FNAC Lymph Node- NECROTISING LYMPHADENITIS (KIKUCHI DISEASE). Lymph Node Biopsy-HISTIOCYTIC NECROTIZING LYMPHA DENOPATHY WITH PAUCITY OF POLYMORPHS (KIKUCHI DISEASE).

With the above findings, the patient was diagnosed as a case of Kikuchi Disease . She was given a course of NSAID and improved.

DISCUSSION:

Kikuchi disease, also called histiocytic necrotizing lymphadenitis or kikuchi-fujimoto disease, is an uncommon, idiopathic, generally self limited cause of lymphadenitis. The most common clinical manifestsation is cervical lymphadenopathy, with or with out systemic signs and symptoms. Clinically and histologically the disease can be mistaken for lymphoma or SLE. It almost always runs a benign course and resolves in

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several weeks to months. Disease recurrence is unusual and fatalities are rare.

The most common laboratory abnormalities are leukopenia, increased ESR, increased liver enzymes level. Lymph node biopsy finding shows necrosis, karyorrhectic debris and the presence of the typical cell types, namely crescentic histiocytes and plasmacytoid monocytes.

Kikuchi disease sometimes mimic SLE as both can present with lymphadenopathy and fever and the cutaneous findings seen in 30% of Kikuchi disease patients can resemble those seen in SLE. But in Kikuchi disease, antinuclear antibodies, Rheumatoid Factor are usually , although not always, negative. They can also have similar histopathologic appearances. Kikuchi disease is suggested by the absence or paucity of the hematoxylin bodies, plasma cells and neutrophils usually seen in SLE.

Treatment of kikuchi disease is generally supportive. NSAIDs may be used to alleviate lymphnode tenderness and fever. The use of corticosteroids has been recommended in severe extranodal or generalised kikuchi disease.

Here we report an unusual case of a young girl presenting with **high grade fever** for 1 month duration and bilateral tender cervical lymphadenopathy, as it has been seen that those who have high grade fever has longer duration of fever in Kikuchi disease. Our patient also had leucopenia and elevated ESR and characteristic lymphnode histopathological findings with paucity of polymorphs, nonspecific ANA Titre and excellent responce to NSAIDs , confirms the diagnosis of Kikuchi Disease and rules out SLE.

CONCLUSION:

Although Kikuchi Disease is rare, any patient presenting with FUO and cervical lymphadenopathy is to be look out for Kikuchi Disease after ruling out Lymphoma and SLE, as it is not unusual and has excellent prognosis.

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Current Concept

A STEP-BY-STEP APPROACH FOR THE BUSY CLINICIAN TO DIAGNOSE DISTAL SYMMETRICAL POLYNEUROPATHY

J.K. Panda[1], S. Nayak[2]

Introduction :

Neuropathic disorders can be broadly categorized as those that affect the nerve cell body or neuropathies & those affecting the peripheral process or peripheral neuropathies.[1] Peripheral neuropathies can be subdivided into myelinopathies & axonopathies. The peripheral numbers have different sensory, motor & autonomic fibres whose involvement leads to diverse symptoms, signs & electro-diagnostic features. Focusing on these features is useful in diagnosing Peripheral neuropathy.[2]

Epidemiology :

The overall prevalence of peripheral neuropathy is 2.4% which increased to 8% in individuals greater than 55 years. Study in Mayo clinic reported 76% etiologic diagnosis after thorough evaluation of 205 cases of Peripheral Neuropathy of which hereditary neuropathy constituted 42%, inflammatory demyelinating peripheral neuropathy (CIDP) is 21% & 13% with systemic diseases (e.g DM).[3] However in the Indian scenario DM appears to be the most common cause followed by alcoholism, vasculitis, Hansen's, systemic diseases & exposure to toxins.[4] Distal systemic polyneuropathy (DSPN) is the most common cause of neuropathy in diabetics occurring in up to 27-46% cases.[5] A logical approach is imperative in identifying an underlying etiology.

The Challenge :

Physicians face 3 district challenges when encountering a patient of Peripheral Neuropathy

1) How to effectively & efficiently screen an asymptomatic patient (in <2 minutes) for peripheral neuropathy.
2) Stratify patients with symptoms of neuropathy to discern those requiring super-specialty (Neurology) consultation.
3) Treatment of the underlying etiology.[3]

Most recommendations for screening in a busy clinic require light touch perception to a 10g Semmes-Weinstein monofilament, vibration testing with a 128 Hz tuning fork, superficial pain (pinprick) perception & testing of DTRs. Clinical history alone is an insufficient screen. Single modality testing like monofilament light touch & vibration may miss 25-50% diabetic neuropathy. Combination testing of vibration & 10g monofilament provides the most sensitive (90%) & specific (85-89%) screen for Diabetic PN (in less than 2 minutes).[6]

EVALUATING PATIENTS WITH CLINICAL SIGNS & SYMPTOMS OF PERIPHERAL NEUROPATHY

Important information can be obtained from carful history & examination. It is imperative to ask 5 key questions based on patient symptoms & signs.

1. Which systems are involved?

Whether symptoms & signs suggest pure motor, sensory, autonomic involvement or a combination of above. Sensory symptoms may be positive or negative. Positive include pins &needles, burning pain, band like sensation on feet, tingling & negative symptoms(numbness, loss of sensation) in hands and feet. Autonomic symptoms include postural hypotension,

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sphincter disturbance, impotence, constipation/diarrhea, dryness/excessive sweating.

2. Distribution of weakness

a. Whether distal or proximal or both simultaneously?

b. Focal & asymmetric or symmetric?

3. Nature of sensory involvement

a. Whether there is loss of sensation (numbness) or dysesthesias & pain. Neuropathic pain can be burning, dull and poorly localized (protopathic pain) C-nociceptor fibers or sharp lancinating (epicritic pain) □ ¡ □P Ô Aä fibers.

b. Patient with loss of proprioceptive loss of balance and in coordination especially in the dark.

4. Temporal Evolution

Onset, duration & evolution of symptoms & signs is important. Whether disease has an acute (days to 4weeks), subacute (4-8 weeks) or chronic (>8weeks) course? Whether monophasic, progressive or relapsing? It is also important to ask for preceding or concurrent infections, associated medical conditions, drug use, alcohol &dietary habits.

5. Hereditary Neuropathy?

Especially in chronic very slowly progressive distal weakness with foot deformities (e.g Pes cavus).[7]

DIAGNOSTICS CLUES FROM CLINICAL EXAMINATION

Once a neuropathy is suspected (from history & screening examination), a detailed history & clinical examination (motor power, sensation, reflexes and gait) allow the neuropathy to be categorized by clinical symptom distribution (length dependent or multifocal) or the clinical modality affected (motor, sensory, autonomic & combination of all).[8]

I. The most common clinical pattern of involvement is a length dependent peripheral neuropathy. This usually is symmetric& symptoms usually begin in the largest nerves at their terminals and ascend individually upto legs. Hand symptoms become evident when leg symptoms approach knee. Majority of peripheral neuropathies are length dependent.

II. Sensory & motor symptoms in a more diffuse symmetric pattern of involvement including both proximal & distal limbs suggest polyradiculoneuropathy. Reflexes are globally reduced/absent.

III. Multifocal neuropathies point to simultaneous or sequential damage of two or more contiguous nerves. Etiologies include amyloidosis, vasculitis, infections, inflammatory and immune-mediated cases.

IV. Small fiber and autonomic neuropathies manifest with features of dysautonomia like sweating, pupillary, cardiovascular, gastrointestinal and micturition disturbances. Referral to a Neurologist is required in polyradiculoneuropathies, multifocal neuropathies, small fiber, autonomic neuropathies apart from ganglionopathies/neuronopathies, entrapment neuropathies and plexopathies.

DIABETIC NEUROPATHY

Diabetic neuropathy is the most common cause of peripheral neuropathy seen in western societies with prevalence up to 30-66 % only 10-15 % patients are symptomatic whereas up to 60-70 % have underlying neuropathy at the time of diagnosis. 11-26 % are limited by associated neuropathic pain.[9]

Classification of Diabetic Neuropathies :

Generalized symmetric polyneuropathies

- Distal sensory or sensory motor polyneuropathy
- Small fiber neuropathy
- Autonomic neuropathy
- Large fiber sensory neuropathy

Focal and Asymmetric neuropathies

- Cranial neuropathy
- Thoracic neuropathy
- Limb neuropathy
- Proximal motor neuropathy

COMBINATIONS

- Polyradiculoneuropathy
- Diabetic neuropathic cachexia

TREATMENT ASSOCIATED

- Insulin neuritis
- Hyperglycemic Neuropathy[10]

DIABETIC NEUROPATHIES-TYPES

1. Distal symmetric sensory/Sensi-motor polyneuropathy (DSSN/DMSN)

This is the most common peripheral neuropathy occurring in diabetes is upto 50% of points at presentation. CIDP has similar findings as seen in DSPN but is more common in non-diabetes. A maximum of two abnormalities (from symptoms, signs, NCS, QSTs or AFTs) is required for diagnosis.[11]

2. Diabetic Autonomic Neuropathy

DAN is a widespread disorder that may manifest as dysfunction of one or more organ systems. DAN typically affects both parasympathetic and sympathetic part of the autonomic nervous system, so it results in cardiovascular, gastrointestinal, urination, sweating, pupils and metabolic disturbances and it manifests first in the vagus nerve which is the longest of the ANS nerves. The cardinal signs of DAN are orthostatic/postural hypotension, orthostatic bradycardia and orthostatic tachycardia, resting tachycardia, exercise intolerance; decreased hypoxia induced respiratory drive, impaired heart rate variability, heart rate unresponsiveness to respiration and abnormal blood pressure regulation.

DAN & more specifically CAN (Cardiovascular Autonomic Neuropathy) is linked to increased incidence of asymptomatic ischemia, myocardial infarction and decreased rate of survival. Age, duration of diabetes, obesity, smoking & poor glycemic controls are risk factors which increase the prevalence of DAN.[12]

DIAGNOSTIC PROCEDURES

The combination of history, examination, ancillary testing & serologic evaluation is important in reaching an etiology in 74-82 % cases.

Serologic evaluation/tests

The American Academy of Neurology has published practice parameters for the evaluation of mild to moderate, symmetric length dependent peripheral neuropathies. Highest yield include screen for diabetes, vitamin B12 and serum protein immune-fixation electrophoresis (SPIEP).[13]

Electrophysiology

Nerve conduction studies including motor, sensory conductor studies, F-wave & EMG (Electromyography).

Slowing of conduction velocity with prolongation of distal latency, temporal dispersion and conduction block in both sensory & motor nerve studies indicate demyelinating neuropathy. In axonal neuropathy, there may be reduced CMAP & fibrillations on EMG. The conduction velocity largely remains same or there may be mild slowing. SNAPs & sensory conduction velocities are reduced in both axonal & demyelinating neuropathies.[14]

Quantitative Sensory Testing

The Semmes-Weinstein monofilament examination (SWME) is a low cost noninvasive rapid (< 2 minutes) test used for clinical testing of pressure sense. The use of 5.07/10 g SWME is sensitive and highly specific, data shows biothesiometry and finer (1 g) monofilaments may improve detection rates. Neurotip is used for superficial pain detection. Vibration is performed by using a 128 Hz Rydel-Seiffer tuning fork. Great toe vibration and on-off/timed method of vibration is the usual testing method.[15]

Autonomic Function Tests

Bedside autonomic tests include blood pressure response to standing or vertical tilt (Normal fall, <20/10 mmHg), heart rate response to standing (increase, 11-90 beats/min; 30:15 ratio e" 1.04), isometric exercise (normal increase in diastolic blood pressure, 15 mmHg), heart rate variation with respiration (normal, e"15 beats/min, inspiratory expiratory ratio 1.2), Valsalva ratio (Ne"1.4), cold pressure test (after 1 min systolic blood pressure, 15-20 mmHg/diastolic 10-15 mmHg), sweat test, axon reflex (piloerection, sweating), pupillary & Schirmer's test (15mm after 5min).[16]

Autonomic function tests can be evaluated by SSR (Sympathetic Skin Response) & QSART (Quantitative Sudomotor Axon Reflex Test).

Nerve Biopsy

Sensory nerve biopsy is a confirmatory test for etiologies like vasculitis, demyelination & infections. It should be performed where electron microscopy, teased fiber techniques and immunohistochemistry are available. Combined nerve muscle study increases diagnostic yield in selected cases.

Corneal confocal microscopy

It is a new sensitive non-invasive examination, which may be used to detect early small fiber nerve damage in patients with small nerve fiber neuropathy. Corneal nerve parameters are assessed and it allows quantification of corneal nerve morphology. Cornea nerve damage correlates with the severity of pain and autonomic neuropathy.[17]

Skin biopsy

This is done nowadays for diagnosis of small fiber neuropathy. Loss of intra epidermal nerve density increases the risk of developing neuropathic pain. Skin biopsy /Intra epidermal nerve fiber density (IENFD) has a diagnostic yield of 88.4% compared to clinical examination (54.6%) & quantitative sensory testing methods (46.9%).[18]

Laser-Doppler-imager (LDI) flare technique

LDI flare is a novel method for detecting early small fiber changes in symptomatic DSPN.

Neuropathic pain assessment

Various neuropathic pain questionnaires have been developed and validated over years. The Leeds assessments of Neuropathic symptoms and signs (LANSS) scale (5 symptoms and 2 signs), Neuropathic pain questionnaire (NPQ) – 12 questions and Neuropathic pain symptoms Inventory (NPSI) – 12 questions, NSS (Neurologic symptoms score) – 17 questions, DNS (Diabetic Neuropathy score) – 4 questions are some of the most commonly used scores.

Michigan Neuropathy Screening Instrument (MNSI) which consists of a 15 point self-administered questionnaire is one of the most comprehensive where a score e”7 is considered abnormal. NDS is commonly used in research studies to stratify DPN severity as none (0 to 2), mild (3 to 5), moderate (6 to 8) and severe (9 to 10).[19-20]

Treatment of symptomatic polyneuropathy

Management depends on 3 principles of approaches

1. Treatment of the etiology
2. Treatment of Neuropathic pain
3. Preventive therapy

1. In diabetic patients, the risk of DPN & DAN can be reduced with improved glycemic control, improved lipid & blood pressure indices and avoidance of smoking and alcohol consumption already recommended for prevention of microvascular complications (EURODIAB II).[21]
2. Symptomatic treatment of painful diabetic Neuropathy

a. Tricyclic antidepressants (Amitryptiline & Nortryptiline)

b. Antiepileptic drugs (Gabapentin and Pregabalin) were the first approved drugs for the effective treatment of Neuropathic pain.

c. Selective Serotonin reuptake inhibitors like Duloxetine, Venlafaxine and Milnacipran were the next approved drugs in the line of treatment. Duloxetine at 60mg daily is effective in treating painful DPN in the short form with a risk ratio for 50% pain reduction at 12 weeks of 1.65 (95% CI: 1.34 – 2.03) and NNT of 6 (95% CI: 5 – 10).

d. Other agents tried are Opioids like tramadol, trapentadol, benzodiazepines and local lidocaine & capsaicin cream. Out of these only 3 – 4 drugs have received FDA approval (Gabapentin, Pregabalin, Duloxetine & Trapentadol).[22]

Treatment of DAN

Intensive diabetes therapy, multifactorial cardiovascular risk education and lifestyle intervention are recommended in patients with CAN. Resting tachycardia may be treated with â- blockers. Symptomatic orthostatic hypotension may be improved

by non-pharmacological measures like compression stockings and by midodrine and/or fludrocortisone. Pro-kinetic drugs, including metoclopramide, erythromycin and domperidone are the mainstay of the treatment in Diabetic gastroparesis. The treatment of ED is based on phosphodiesterase-5-inhibitors.[23]

Preventive Treatment

Aldose reductase inhibitors not only improved impaired conduction velocity but also provided symptomatic relief & improved clinical outcome. Epalrestat, tolrestat and zenarestat are examples.

á-Lipoic acid is also a potent antioxidant in experimental models and is reported to reduced diabetic micro-vascular and macro-vascular complications in animal models. A total of 4 trials (ALADIN I, ALADIN III, SYDNEY and NATHAN II) showed that treatment with á-Lipoic acid (600 mg/day) over 3 weeks is safe and significantly improves both positive neuropathic symptoms and neuropathic deficits to a clinically meaningful degree in diabetic patients. Methyl-cobalamin has also shown to have positive neurotropic effects.[24]

RECOMMENDATIONS

Revised EFNS guidelines confirm TCAs (25-150 mg/day), gabapentin (1200-3600 mg/day) and pregabalin (150-600 mg/day) as the first line for various neuropathic pain conditions. SNRI (duloxetine 60-120 mg/day, venlafaxine 150-225 mg/day) are first line in painful diabetic polyneuropathies based on their most established efficacy. Strong opioids (Tramadol/ Trapentadol) at (200-400 mg/day) and capsaicin cream are used as 2nd/3rd line treatment in neuropathic pain. [25]

Combination Therapy

COMBO-DN study has proved that combination of duloxetine and pregabalin/gabapentin is superior to each drug in maximum recommended dose.

Balanced Approach

The clinician has to be a balance between (A) Pragmatist – who aim at minimal possible investigation to solve the clinical problems and (B) Completist – who aim to eliminate every possibility by complete evaluation. It is important to have a balanced view and follow clinical & investigative clues for a likely diagnosis.[8]

Conclusion :

Peripheral neuropathy is commonly encountered in primary care settings. Most common etiologies are diabetes, vitamin B_{12} deficiency and vasculitis. Most common are length dependent sensory predominant symmetric polyneuropathies. A rational approach and precise questions, clinical evaluation and investigations will help in reaching a correct diagnosis in time constrained clinic settings.

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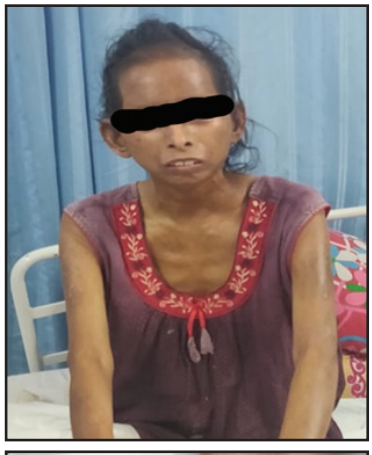
Pictorial CME

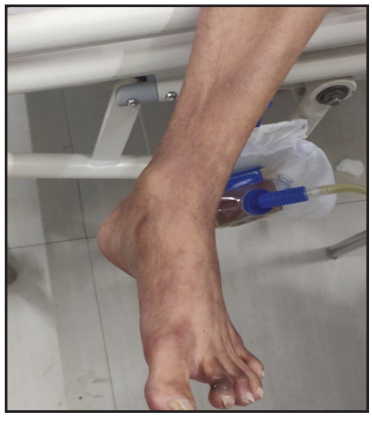
A CASE OF CREST SYNDROME

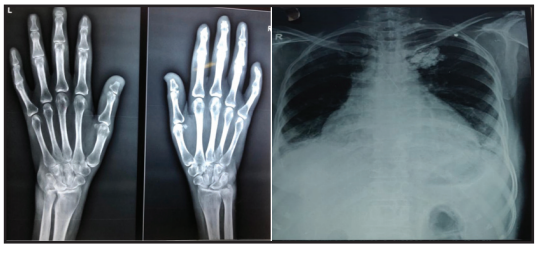
S.K. Dhar[1], P. Patro[2], C. Das[1], G. Naidu[3]

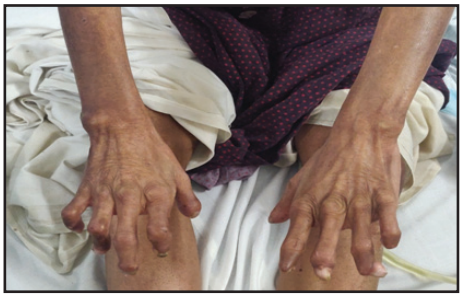
Case Vignet:

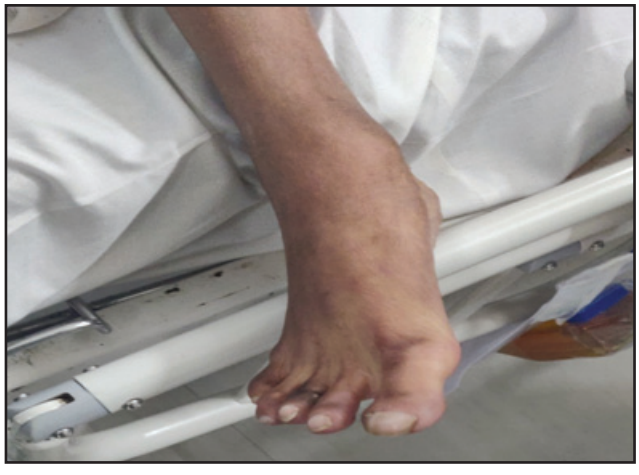
- A 55years female came with complaints of shortness of breath, skin tightening pain in fingers with discoloration of figure on cold exposure, difficulty in swallowing solid food, and dyspepsia
- O/E
- Blood pressure- 150/70 mmg, Pulse rate 88/min.
- Head to toe examination
- Shiny tight skin, telengiectasia, microstomia, acro-osteonecrolysis, blackish discoloration of 3 mtp on right foot,2 mtp left foot .
- Investigation showed hemoglobulin of 7.1 mg/dl, peripheral smear showed normochromic normocytic anemia, ESR -35 mm/1hr, creatine - 0.7 mg/dl,ANA profile – scl 70 positive.HRCT throx showed NSIP pattern of ILD in bilatral lower lobe, 2DECHO showed no PAH.











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