Case Reports

535. A Case of Syphilis with Pseudolymphoma - The Story of a Clinician’s Triumph

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Clinical presentation: Thirty year old male patient, seropositive for HIV, presented with reddish skin lesions on abdomen, and legs with intermittent high grade fever for 2 months. He had occasional dry cough with mucoid expectoration and mild headache.

On admission: He had generalized lymphadenopathy, multiple red papulonodular lesions over chest, abdomen, neck, back, palms, and soles and coarse crepitations in lung bases.

Investigations: ESR - 84 mm, CBP-Normal, Biochemistry: RFT-Normal, LFT-Normal, Serum positive for HIV (2 samples), VDRL and TPHA, Cultures-Negative (Bone Marrow, Blood), Chest x-ray normal man strongly positive, USG abdomen-hepatomegaly, Bone marrow-no evidence of infiltration, sputum AFB-Negative on 4 occasions, CT head-normal, CSF : Pressure - 84 mm, Glucose-43 (PL-101), Protein-125 Cultures-Neg for Bacteria, Fungi, CSF VDRL and TPHA-Positive, CD4 count-150 (absolute), 8.4 Dermatology consultation was done, clinically they were also of the same opinion as to the rash, skin was taken by them.

Treatment and Course of stay: Inj. Crystalline penicillin given in a dose for secondary syphilis with CNS, PCP prophylaxis and INH prophylaxis started, Psychiatric evaluation and counselling given.

Final Skin Biopsy Report: Immature lymphoid cells suggestive of lymphoma, Immunohistochemistry shows CD45 (RO) positivity for T cells.

We continued treatment with penicillin in spite of the biopsy report and the rash subsided along with other symptoms.


Discussion: The prevalence of syphilis is common in patients with HIV. The papulonodular form of syphilis is also very well described. Syphilis, especially the nodular variety of secondary syphilis can present histologically similar to lymphoma. This patient probably had a similar problem (pseudolymphoma). The patient did not have any recurrence of skin lesions and later HAART therapy was started for him.

536. Quadriaparesis in A Quinine Resistant Plasmodium falciparum Malaria

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A 38 years old female from Moreh, a town near Myanmar presented as loose motion, marked dehydration and fever with chills and rigors of three days duration. As Plasmodium falciparum (PF) was positive she was treated with injection quinine infusion for three days as a case of Algid Malaria. She got deteriorated and hence referred to Regional Institute of Medical Sciences, Imphal.

On hospitalization, she was icteric, severely dehydrated, temperature 104°F, hepatosplenomegaly with features of shock. Peripheral smear still showed heavy parasitemia with PF and hence started treatment with injection Arteether αβ 80 mg Iµ twice daily along with supportive measures including 5 units of blood transfusion. On second day she became unconscious but gained consciousness on 5th day with flaccid quadriaparesis. PF was negative on fifth day of injection Arteether. Investigation revealed - Hb 8.4 gms%, Platelets - 20,000/cumm, Normal CSF with Pre-renal uraemia, MRI brain and cervical spine was also found to be normal.

Quine Resistant Algid Malaria presenting as Cerebral Malaria, Quadriaparesis and Hepatitis is rarely seen.

540. Dengue Haemorrhagic Fever Causing Myositis, Acute Inflammatory Demyelinating Polyneuropathy and Myocarditis

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Mr. P 20 yr old male presented with complaints of high grade fever with chills and rigors for 6 days associated with severe headaches myalgias and bodyaches during dengue epidemic. For last one day patient was having pain and weakness in all the four limbs. Examination revealed stable vitals with a positive tourniquet test. There was tenderness in all muscle groups with decreased tone in all four limbs. All four limbs had weakness proximal > distal. Deep tendon reflexes were absent in bilateral ankles, supinators, left triceps and knees. Rest of the CNS and systemic examination was within normal limits. Routine investigations revealed thrombocytopenia (plt 21000/cc) with PCV 44, AST-500, ALT-375. Dengue serology was positive for both IgG and IgM. CPK-T was 40260 on day 1 Progressively platelet count improved to > 1.5 lakh/cc day 5. NCV done on day 5 was s/o demyelinating polyneuropathy involving LL>UL, motor>sensory. EMG revealed myopathic pattern. CXR showed cardiomegaly. ECG showed increased PR interval and occ ventricular ectopics. ECHO revealed moderate diastolic dysfunction. CSF done on day 5 was WNL and HIV by ELISA nonreactive. Patient was then started on oral steroids. By day 18 the power and DTR in all four limbs had recovered to normal and CPK-T had reduced to 113. Repeat CXR showed reversal of cardiomegaly, ECG showed normal sinus rhythm.

This was a rare presentation of Dengue with myositis, neuropahty and myocarditis. Isolated case reports of dengue with myositis or with neuropathy are known. Myocarditis is also reported. But to the best of our knowledge no case of dengue has been reported with myositis, neuropathy as well as myocarditis has been reported.
**Classification systems.**

Recognizing disease associations assume significance in excluding patients with mental subnormality and epilepsy as part of HSMN-Epilepsy can be part of a few metabolic neuropathies which were not the known metabolic neuropathies was normal. Electrophysiological studies revealed an axonal neuropathy Schizophrenia and the youngest girl had Cryptogenic Epilepsy. Physical examination was done. The three children were mentally bound state by the second decade. A detailed neurological and the diseased had normal developmental milestones with features of neuropathy. The second child, a girl was normal. All describe a family of the so-called HSMN-plus syndrome.

We exhibited a modified regime of dexamethasone (1.5 mg/kg stat followed by eight doses of 0.5 mg/kg, given every 6 h) to 3 patients of rapidly deteriorating, culture positive, complicated enteric fever (2 cases of enteric encephalopathy and one case with rapidly progressive ARDS) with gratifying results. Steroids thus appear to be an important adjuvant therapy in selected high risk, rapidly progressive cases of enteric fever.

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**Hereditary Motor Sensory Neuropathy Plus - Report of a Family**

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Introduction: A few diseases have been described in association with the hereditary motor sensory neuropathies. We describe a family of the so-called HSMN-plus syndrome.

Case Reports: A family of third degree consanguineous parentage with three diseased children presented with clinical features of neuropathy. The second child, a girl was normal. All the diseased had normal developmental mile stones with disability starting in the first decade progressing to a wheel chair bound state by the second decade. A detailed neurological and physical examination was done. The three children were mentally subnormal. The eldest girl was diagnosed to have Catatonic Schizophrenia and the youngest girl had Cryptogenic Epilepsy. Electrophysiological studies revealed an axonal neuropathy confirmed by nerve biopsy. The metabolic workup done to rule out the known metabolic neuropathies was normal.

Discussion: The disease associations observed in the family namely Mental sub normally, Catatonic Schizophrenia and Epilepsy can be part of a few metabolic neuropathies which were excluded by appropriate investigations. There are case reports of patients with mental sub normality and epilepsy as part of HSMN-plus syndromes but Schizophrenia is a non-described entity. Recognizing disease associations assume significance in excluding metabolic diseases, in genetic linkage studies and in the disease classification systems.

**HCC in an Asymptomatic HBV Carrier with HIV Co-Infection**

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HCC is a known complication of HBV related chronic liver diseases in about 70% of the cases. But its association with underlying HBV carrier state without evidence of chronic liver disease is rarely seen. HIV co infection in this clinical entity is rarely reported and hence this case report.

A 28-year male presented with C/o pain abdomen right upper quadrant and fever of 45 days duration. Pain continuous dull aching relieved partially with nsaids. Fever low grade continuous not associated with chills or rigors. No history of jaundice or history suggestive of chronic liver disease. No history of blood transfusion. On examination patient febrile with pallor. Pulse 95/min regular, BP120/80 mmHg, R rate 24/min, No cyanosis, clubbing, icterus or pedal edema. Investigations: Hb% 9.4, TLC 7,800, ESR 60/mm/1 hr, PT/APTT - Normal, T.Bil 0.7, C.Bil 0.2, T. Proteins 7.4, Alb 4.7, SCOT 36, SGPT 30, ALP 200, UGI Endoscopy: No varices (Normal), CXR normal, HIV-1 Western Blot Positive, CD4 count: 440 cells, HBsAg +ve, Anti HBe +ve, HBe Ag-ve, Anti HCV -ve, USG and CT abdomen: Heterogeneous mass lesion 11.2 x 8.3 cm in left lobe of liver with vascularity, minimal ascites. FNAC mass lesion s/o HCC. IHC report: AFP positivity in tumour cells. Serum AFP levels: 3050 ng/ml. Patient on treatment with HAART and chemotherapeutic agents.

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**Intraductal Papillary Mucinous Tumour (IPMT): An Unusual Cause of Acute Pancreatitis**

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A 70 year old man presented with a 12 hour history severe epigastric pain and markedly elevated serum amylase and lipase. A diagnosis of acute pancreatitis was made and the patient responded promptly to conservative management. An abdominal ultrasound was negative for gallstones. CT scan of the abdomen showed a dilated main pancreatic duct with minimal evidence of acute pancreatitis. Upper endoscopy with endoscopic ultrasound (EUS) showed a gaping papilla with mucinous material extruding through it. EUS revealed a markedly dilated main pancreatic duct that was ectatic and had echogenic debris suggestive of mucin. These features were consistent with a diagnosis of intraductal papillary mucinous tumour (IPMT). Endoscopic retrograde cholangiopancreatography (ERCP) with intraductal ultrasound showed dilatation of the main pancreatic duct and a cystic area at the neck of the pancreas. He underwent a distal pancreatectomy and histopathology showed an intraductal papillary mucinous tumour with moderate dysplasia.

IPMT should be considered in the differential diagnosis of pancreatitis like pain with or without elevation of pancreatic enzymes, recurrent pancreatitis, and incidentally found pancreatic duct dilatation or cystic lesions of the pancreas. Characteristic features on CT, MRI, esophagogastroduodenoscopy, ERCP and EUS should lead to the diagnosis. The tumour is low growing, but often has a multifocal origin and a potential for malignant degeneration. Management is surgical resection, but only for patients who are otherwise in good health.

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**Gastrointestinal Stromal Tumour**

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A 49 years old farmer, Mr. G presented with complaints of multiple episodes of melena associated with lower abdominal pain since 2 days. He had similar episodes in October 2002. Required blood transfusions and gastroscopy at that time was reported to be normal. Examination revealed tachycardia, hyperdynamic pulse and moderate pallor. Systemic examination was normal. The patient was stabilised with fluids and blood transfusions.
Investigations revealed moderate anaemia, normal hepatic and renal function tests, electrolytes and bleeding parameters. Gastroscopy was normal.

Since he continued to have melena and haematemesis. He was treated with somatostatin, pantoprazole, colloid and crystalloid infusions. Barium meal follow through and enteroscopy were considered.

Enteroscopy was done which revealed and fleshy mass arising from antimesenteric border 20 cm from duodenojejunal flexure with ulcerated bleeding surface.

Patient then underwent surgery during which mass and enlarged lymph nodes in drainage area were resected. Biopsy was reported as gastrointestinal stromal tumour (GIST).

Gastrointestinal stromal tumours (GIST) are rare neoplasms, constituting 3% of all gastrointestinal tumours. They often present with recurrent GI bleed.

This case is being presented to highlight that such tumours should be considered as differential diagnosis for both haematemesis and melena of obscure origin.

552. Amoebic Colitis Masquerading As Inflammatory Bowel Disease

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43 yr old male presented with painless bloody diarrhea of 10-12 episodes/day for the past 2 1/2 yrs. It was watery, non foul smelling and admixed with blood. There was no history of fever, but had severe anorexia and weight loss of 28 kgs over 2 yrs.

Examination revealed the patient to be poorly nourished (BMI-14) with pallor, postural hypotension and oral candidiasis. Systemic examination was unremarkable.

Investigations showed anemia, leucocytosis, severe hypoproteinemia, hypokalemia and hyponatremia. There was both iron and vitamin B12 deficiency. Stool occult blood was positive. Thyroid function test was normal.

He was treated intravenous fluids and electrolyte abnormalities were corrected. A gastroenterology consultation was sought and sigmoidoscopy done revealed multiple colonic ulcers with intervening normal mucosa. A provisional diagnosis of Crohns disease was made pending colonic biopsy report.

Colonic histopathology was diagnostic of severe amoebic colitis. ELISA for Entamoeba histolytica antibody was positive. Patient was started on combination of metronidazole and diloxanide furato and patient markedly improved.

A follow up colonoscopy showed significant improvement. On follow up patient is asymptomatic and has regained his original weight.

555. Alkaptonuria

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Alkaptonuria is a disorder of tyrosine metabolism where deficiency of enzyme homogentisic acid in urine and excessive deposition of it in connective tissue. We described the disorder in a 14 years old boy born of consanguineous parents admitted with history of passage of normal colour urine turning black on staining which would stain clothes and ground. No such history was present in family pedigree. The patient was of average built and his general physical and systemic examinations were normal. There was no evidence of pigmentation of sclerae and ears, arthritis or degenerative joint disease. The diagnosis was established by few laboratory tests. A purple-black colour of urine was produced upon addition of ferric chloride, Benedict’s reagent gave brown colour, and black colour was produced by addition of silver nitrate solution. This is a very rare disorder, and to our knowledge, this is the first case reported from Jammu and Kashmir.

558. Idiopathic Lipoid Pneumonia

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Lipoid pneumonia is rare in adults. It can be either endogenous or exogenous. We report a 33 years male who was referred to us from Dahran, Nepal. He was having cough with minimal expectoration and progressively increasing dyspnea since 1 year. He was investigated locally which revealed reticular shadows on CXR, reticulo fibrous shadows mainly in lower zones on HRCT and BAL was negative for AFB and fungi. He was diagnosed to be suffering from sarcoidosis and was treated with prednisolone 30 mg/day for six months. However he failed to respond. Seven year ago he had been treated with anti-tubercular drugs for three years. On examination he was normal except for bilateral fine crepitations in infrascapular areas on both sides. His investigations revealed mild airway obstruction on PFT. Sputum was repeatedly negative for AFB and TBLB was inconclusive. HRCT revealed bilateral reticulonodular shadows and bronchiectasis in lower zones in both lungs. An open lung biopsy was undertaken which revealed lipoid pneumonia. As there was no history of nasal instillation of any oils, it was diagnosed to be idiopathic. He was sent back to Nepal where he is under follow-up and is doing well till present report. To the best of our knowledge idiopathic lipoid pneumonia in adults has not been reported so far from India.
appetite or loss of weight. He had a past history of pulmonary tuberculosis and left lung hydatid disease, which was operated. He had a past history of smoking and alcohol intake. On examination, he was moderately built and well nourished; there was no pallor, clubbing or lymphadenopathy; the vital data were within normal limits. Respiratory system evaluation revealed bronchial breathing and wheeze in the left inrACLAVicular, mammary and axillary areas, alongwith features of left lower lobe collapse.

On evaluation, the chest radiograph showed features of left lower lobe collapse, while the contrast-enhanced CT scan of the chest revealed, in addition, a cavity in the left lower lobe. The patient was treated conservatively at first. However, there was a recurrence of massive hemoptysis. Thus, he was taken up for bronchial artery embolization, during the course of which a tortuous feeder from the left 5th intercostal artery was discovered and embolized. Following the procedure there was a significant reduction in the quantity of hemoptysis. Soon, however, hemoptysis recurred and the digital subtraction angiography (DSA) was repeated to identify new feeder vessels. During this session a new feeder was observed arising from the 6th left intercostal artery, which was similarly embolized. Following this there were no further episodes of hemoptysis.

The above case illustrates how massive hemoptysis, not responding to medical therapy, can be successfully managed with therapeutic bronchial artery embolization, thereby avoiding a major surgical intervention.

563. An Atypical Presentation of Panacinar Emphysema

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We present a case of intractable breathlessness in a 33 year old male, non-smoker in whom the HRCT revealed bullous and emphysematous changes of the left upper lobe of lung. In view of a young smoker with COPD, alpha-1-antitrypsin levels were assayed, which was found to be very low. The atypical feature in our case was the bullous presentation and unilateral upper lobe involvement contrary to the bilateral lower lobe disease mentioned so far in literature.

564. A Patient with ‘Unresolved Pneumonia’

R Bhat, A Nawaz, M Anoop
KMC, Mangalore.

Mr. S 30 years, presented with left sided chest pain after his morning walk on a cold day. Chest X-ray showed ‘pneumonia’ with a paracardiac ‘consolidation’. When the shadow failed to disappear despite antibiotic treatment, he was referred as having ‘unresolved pneumonia’. CT scan chest revealed the ‘shadow’ to be fat-mediastinal lipomatosis. A similar shadow was found on right side and also posteriorly.

Mediastinal lipomatosis is condition where large amounts of mature adipose tissue gets deposited in the mediastinum and can masquerade as mediastinal widening or ‘Pneumonia’. Fat being fluid at body temperature does not produce pressure symptoms.

The literature review revealed that rest of the cases where associated with Cushing’s syndrome, corticosteroid therapy or obesity. Our patient was overweight (BMI 29), but did not have any of these.

His blood lipid profile was normal. Being a harmless condition he was sent home with reassurance.

565. Nitrofurantoin Induced Interstitial Lung Disease

M Shetty, M Rao, SP Kumar, NM Rao, YSN Raju, N Chandra Nizam’s Institute of Medical Sciences, Hyderabad.

A 55 year female diabetic hypertensive and case of recurrent UTI on prophylactic nitrofurantoin 100 mg OD c/o dry cough, dyspnea since 6 months insidious onset progressive, no PND or orthopnea, no other significant history O/E PR-90/min BP - 130/80 RR - 28/min Respiratory System : Bilateral basal fine end inspiratory crepts, CVS-S1+S2+ESM at PA, P2 loud P/A: soft, no organomegaly. CNS-NAD. Investigations revealed CBP-Normal, ESR 55 mm/1st hr, ECG normal, Collagen profile-negative.

HRCT Chest : patchy changes of parenchymal fibrosis, b/l patchy ground glass opacities with areas of interlobar septal thickening, subpleural areas of parenchymal fibrosis no pleural effusion vascular structures normal.

Diagnosed as a case of drug induced ILD, Nitrofurantoin was stopped and steroid were started. On oral steroids patient improved. We are reporting it as a case of Nitrofurantoin induced interstitial lung disease.

566. Uncommon Complications of Common Poisoning Agents : Two Case Studies

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Case 1 : 25 year old female presented to our emergency department with history of consumption of FURODAN-3-G, a carbamate used as agricultural pesticide. She presented with features of pulmonary oedema. With treatment she improved clinically and by the second week she developed neuropsychiatric manifestations like irritability, restlessness, bizarre movements of extremities and involuntary bowel and bladder movements.

With treatment she improved clinically and later she came to followup and examination at time showed that she had developed contractures of the upper and lower limbs because of the bizarre posturing and she regained her control over the bladder and bowel.

Her routine investigations including EEG, CSF study were normal. MRI brain done showed atrophy and hyperintense lesion and hypothalamus and basal ganglia.

Case 2 : 21 year old male was admitted in our ward with history of suicidal ingestion of CHLORPYRIPHOS. He was treated symptomatically as no antidotes were available. On the 2nd day he developed jaundice and his LFT done showed increase in both bilirubin and enzymes. By the next day he began to develop oliguria and urea and creatinine reports 186 mg% and 12.4 mg% respectively. He was sent for emergency dialysis and he died on the dialysis table because of sudden cardiac arrest. CHLORPYRIPHOS is known to produce hepatopathy but in our case the patient developed both renal and hepatic failure. The viscera has been sent for histopathological examination and the report is awaited.

Conclusion : The agents mentioned FURDAN-3-G and CHLORPYRIPHOS are commonly used as suicidal agents in our country. We present the two cases as the complications by the agents - atrophy of both hypothalamus and basal ganglia producing neuropsychiatric manifestations by FURDAN-3-G and renal failure by CHLORPHYLIPHOS are rarely reported in literature.
567. Pulmonary Thromboembolism with Cavitating Pulmonary Infarction: A Rare Presentation

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Pulmonary thromboembolism with pulmonary infarction is a common occurrence in any acute care units. The infarction presenting as a cavitating lesion confusing the picture is a very rare occurrence and there have been only two cases reports in world literature.

29 yrs female presented with c/o breathlessness of 2 months duration slowly progressing to become grade 4, cough since 2 months continuous, mucoid sputum, no hemoptysis, fever, low grade, no chills or rigors, no other history suggestive of Tuberculosis. History of recurrent abortions 6 in a matter of 3 years. H/o DVT Right leg on actrom 2 mg. On examination patient febrile, dyspneic, Pulse 130/min, BP 120/80, Resp 30/min, no cyanosis, clubbing, pedal edema, jaundice or signs of congenital heart disease. On chest X-ray - opacification along right lung hilum and right lower zone, soft tissue swelling along right femoral vein. Doppler study of lower limbs - DVT Right external iliac, femoral, popliteal vessels. Initial started on support therapy, Antibiotics, anticoagulation, antituberculous therapy, and antihypertensives. Since no improvement CT scan chest done which showed pulmonary infarction, which has cavitates. Diagnosed as APLA syndrome with DVT, PTE and cavitating pulmonary infarction. Treated with anticoagulation and supportive therapy patient recovered and was discharged.

572. Glycogen Storage Disease - A Rare Presentation

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A 55 years old female presented with complaints of perspiration, palpitation, uneasiness followed by loss of consciousness. She had a significant history of skipping dinner the previous day. She promptly responded to intravenous glucose administration. She had a history of similar episodes suggestive of hypoglycemia precipitated by fasting and relieved by consuming sugar in some form for the past 2 years. She was nonalcoholic, not on oral hypoglycemic agents or other drugs. Alimentary system examination revealed moderate splenomegaly with signs of free fluid. Liver biopsy revealed distended hepatocytes with glycogen deposition (strongly PAS positive) and minimal fibrosis. Liver function tests showed conjugated hyperbilirubinemia.

Investigations revealed Hemoglobin 7.0 gm%, TLC 10,400/CC ESR 146, RBS 95 mgm%. PT 19.7 INR 1.8, APTT 63.9, D-dimer positive, ACA IgG 200 IU. Doppler study of lower limbs - DVT Right external iliac, femoral, popliteal vessels. Initial started on support therapy, Antibiotics, anticoagulation, antituberculous therapy, and antihypertensives. Since no improvement CT scan chest done which showed pulmonary infarction, which has cavitates. Diagnosed as APLA syndrome with DVT, PTE and cavitating pulmonary infarction. Treated with anticoagulation and supportive therapy patient recovered and was discharged.

573. Familial Hypercholesterolemia Type-IIa with Supravalvular Aortic Stenosis

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A 12 years old female presented with complaints of swelling over knees, behind both elbows, behind both ankle, over sacrum, over back of fingers and over left eyelid for three years which yellowish in colour. Patient’s sister also had similar swelling behind left elbow. On examination swellings were multiple, yellowish in colour, and about 1x1 cm. in size. Tendon Xanthoma, Tubercous Xanthoma, Xantheleshma over left eyelid was present. Bilateral Carotid bruit was present. Systemic examination did not reveal any abnormality. On investigation lipid profile showed S. Cholesterol : 655 mg%, S. LDL : 590 mg%, S. HDL : 36 mg%, S. TG : 144 mg%, S. VLDL : 29 mg%. ECG : suggestive of left ventricular hypertrophy. Carotid Doppler was normal. Echocardiography was suggestive of discrete supravalvular aortic stenosis with peak gradient of 28 mm Hg.

Conclusion: A rare case of familial hypercholesterolemia type-IIa with supravalvular aortic stenosis.

581. Foetus Kick-Starting Paroxysmal Hypertension

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Pheochromocytoma is a rare catecholamine-secreting tumour derived from chromaffin cells. Tumours arising outside the adrenal gland are termed extra-adrenal pheochromocytomas or paragangliomas. The trigger for catecholamine release is unclear, but multiple mechanisms have been postulated, including changes in tumour blood flow, direct pressure, and by manipulation during or after pregnancy. A 19 year old female was detected to be hypertensive during her first pregnancy at 7th month of gestation. Labour was induced and she delivered a healthy baby. Following delivery she was normotensive and all antihypertensive was withdrawn. She presented during the 6th month of gestation of her second pregnancy with high BP. As her BP was uncontrolled on antihypertensive drugs she was evaluated for secondary causes of hypertension. Her 24 hour urinary VMA and catecholamine was high. CT abdomen showed a brilliantly enhancing mass lesion in preaortic area at level of bifurcation of aorta. She was diagnosed to have extra-adrenal pheochromocytoma on the basis of history, laboratory findings and imaging studies. Pregnancy was terminated. She was on methyldopa, alpha-blockers, and calcium channel blockers for control of her high BP. With these drugs her BP was under control and she is being planned to be taken up for surgery. The highlight of this case is the fact that the patient was detected to be hypertensive after the 6th month of gestation during both pregnancy. We pressure that the growing fetus and the increased foetal movement which starts around the 6th month of gestation might have kick started the paroxysmal episodes of hypertension in this case.

583. A Case Report of Beta Thalassemia Intermedia

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A 24 year old male, presented with jaundice and generalized weakness with abdominal distention since 20 years.


Investigations: Hemoglobin 7.7 g/dl, peripheral smear showing microcytic hypochromic anemia, anisopoikilocytosis and numerous target cells. Reticulocyte count - 11% with red cell production index of 2.8, indicating hemolytic anemia. Total bilirubin - 8.7 mg% with 7.8 mg% of indirect thus indicating unconjugated hyperbilirubinemia.

Hemoglobin electrophoresis indicated: Hb A 42.8%, Hb F - 53.6%, Hb A2 - 3.6%. In his family, father, sister and paternal uncle were found to have thalassemia trait.

The above investigations revealed thalassemia intermedia with double heterozygous state.
584. Vertebral Hemangiomas in Pregnancy

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Hemangiomas of vertebral column, occurring in up to 10% of population but are rarely symptomatic. The hemangiomas may involve any portion of the spinal column but occur most often in thoracic spine. Vertebral hemangiomas can occur at any level. Mostly only one vertebra is involved. Sometimes 2-5 vertebral levels may be involved. More than 5 vertebral hemangiomas are rare. Our patient had 6 vertebral hemangiomas.

The enlargement of vertebral hemangiomas in pregnancy has been described on a few occasions previously. The three mechanisms which may explain compression of the spinal cord during pregnancy from vertebral hemangiomas are as follows: (A) Increased venous pressure due to obstruction of the inferior vena cava by enlarge uterus. (B) Hormonal effect on the endothelium. (C) Blood volume increase.

Reported here is a case of 35 years old female who presented in the third trimester of third pregnancy with first episode of subacute flaccid pure motor progressive paraparesis with marked radicular symptoms in lumbosacral areas remaining stable after delivery. On investigation, MRI showed 6 vertebral haemangiomas involving D6, D7, D9, D10, L1 and L3. The haemangiomas involving L1 compressed the conus. After delivery she was given steroids for 1 month and then gradually tapered. The haemangiomas appeared to have regressed significantly with steroid therapy. MRI confirmed the regression and there was significant improvement neurologically. She remained admitted for 1 1/2 month. On discharge, she was advised to avoid future pregnancy. Even after two months of stopping the steroids she is symptom-free and following up with us.

Progressive paraparesis in the third trimester of pregnancy may have hormone dependent tumour as a cause and may necessitate watchful, conservative treatment as the first line approach. Other invasive measures adopted in treatment of haemangiomas such as CT guided percutaneous alcohol injections, embolisation or laminectomy can be avoided.

586. Pure Red Cell Aplasia with Diabetes Mellitus

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Pure red cell aplasia (PRCA) in adults is usually an acquired disease. It may be idiopathic associated with some medical disorders like lymphoma, thymomas, solid tumours, immunological disorders like SLE and rheumatoid arthritis, certain viral infections and drugs. The association of the disorder with diabetes mellitus has not been reported. We describe the occurrence of this disease in a 32 years old male admitted with history of easy fatiguability and exertional dyspnea. There was no such history in other family members, nor was history suggestive of any preceding viral infection or drug intake. Examination revealed patient severely anemic with normal systemic examination. On investigating, the patient proved to be diabetic with fasting blood glucose of 176, 208, 231 mg/dL and postprandial 346, 405 and 386 mg/dL on different days. His diabetic was controlled with fasting blood glucose of 176, 208, 231 mg/dL and postprandial 346, 405 and 386 mg/dL on different days. His hemoglobin was 3.01 g/dL, leucocyte count 8.3 x 10^9/µL, platelets 1.82 x 10^11/µL, and peripheral blood film showed hypochromic microcytic red blood cells. His RBC count was 1.28 x 10^12/dL, MCV was 54.7 fl (normal 80-100), MCH 25.1 pg (normal 27-31) and MCHC 25.4 g/dL (normal 30-35). Reticulocyte count was 0.2%. Bone marrow aspiration picture was suggestive of pure red cell aplasia. There was no evidence of thymoma on chest radiograph and computed tomography, and profile for immunological diseases was negative. Patient was initially given a therapeutic trial of erythropoietin with no response. Then, he was put on prednisolone 30 mg/day for one week and 20 mg/day for next two weeks alongwith antithymocyte globulin. Within next two weeks the patient showed marked increased in hemoglobin and other red cell indices. He is under our regular follow up for future course.

589. Acute Pancreatitis As The Initial Manifestation of Hereditary Spherocytosis

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Introduction : Hereditary spherocytosis is a hematologic disorder leading to pigmented gall stones. Pancreatitis as initial manifestation of hereditary spherocytosis is rarely seen.

Case history : A middle aged female was presented with history of acute abdominal pain with vomiting. On examination she was anemic, icteric with tenderness in epigastric region and splenomegaly was present. Her investigations revealed - Hb 7.2 g%, TLC - 3000/cu mm, MCV - 109 fl. platelet count - 14000/ cu mm, MCHC - 33.5 g/dL, serum amylase - 933 iu/l, serum bilirubin - 3.3 mg% (indirect - 2.1 mg%), SGOT - 50 iu/l, SGPT - 17 iu/l, S. Alkaline phosphate - 206 iu/l. USG abdomen showed multiple gall bladder stones, spleen of 19 cm size and hypoechogenicity of pancreas. CT abdomen revealed haziness of anterior margin of pancreatic body with adjacent peripancreatic fat streaking. Patient was diagnosed as a case of acute pancreatitis and managed conservatively. In view of anaemia, splenomegaly and gall stones possibility of hemolytic anemia was kept. On further investigations peripheral smear showed normochromic, normocytic RBC’s with anisocytosis; macrocytosis, polychromatophils and spherocytes were also present. Reticulocyte count was 6%. LDH was increased (332 iu/l). G6PD and Coomb’s test were negative. Osmotic fragility test revealed increased in hematocrit to 0.4. Hence the diagnosis of hereditary spherocytosis was made. Later, on elective cholecystectomy multiple pigmented gall stones were found confirming hereditary spherocytosis as cause of gall stones leading to acute pancreatitis.

Conclusion : Hereditary spherocytosis may remain asymptomatic till late age and may present as acute pancreatitis.

592. Granulomatous Interstitial Nephritis (GIN) as an Unusual Cause of Nephrotic Syndrome

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A young female presented with 1 1/2 months h/o generalized edema decreased urinary output, dyspnea on exertion of 1 week duration, h/o low grade fever for a week before the onset of generalized edema. Patient was not on any medication in the past. Examination revealed generalized edema, pitting edema. There was no lymphadenopathy, BP 120/90, systemic examination was unremarkable. Investigations revealed : Hb - 9 gm%, TLC 15,000, DLC P74L35E2, ESR 130, Urea 122 mg%, Creatinine 2.4 mg%, Uric acid - 8 mg%, SGPT, SGOT and ALP - normal, T. Protein 4.3 gm% (A - 2.3 gm%, G - 2.0 gm%), Ca ++ 8.1, P04 - 3.2, HDL-C - 21 mg%, LDL-C - 380 mg%, VLDL - 55 mg%, TG - 262 mg%, 24 hour urinary protein - 5.49 gm. Urine mic. - protein - 3+, RBC - 5/ hpf, pus cells 4-5/hpf, cast - neg. ASO - normal, anti GBM Ab - neg., coagulation profile (PT and PTTK) were deranged, chest x-ray - normal, USG-Abd. - s/o - medico renal disease. After correction of coagulation profile, kidney biopsy was done which revealed necrotizing granulomatous interstitial nephritis with normal glomeruli and vessels. Usually, GIN presents with ARF and presentation as nephrotic syndrome is very rare.

Diagnosis and details of management will be discussed.
Membranous Nephropathy Superimposed with Anti-glomerular-Basement-Membrane Glomerulonephritis

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Introduction: Simultaneous Membranous glomerulonephritis as well as anti-GBM disease has been previously described in literature as rare presentations. Evolution membranous nephropathy in to Anti-GBM nephritis has been rarely described in literature.

Care report: A 54-year-old female presented with history of pedal edema of 20 days duration. On evaluation she was found to have nephrotic range of proteinuria, hypoalbuminemia, Hypercholesterolemia with serum creatinine was 1 mg/dl. ANA, DsDNA, HBsAg, HCV and HIV were negative. Renal biopsy was consistent with Membranous glomerulonephritis patient was on ACE inhibitors and doing well.

Later she was admitted with progressive renal failure, oliguria and uremic symptoms and had significant pallor and pedal edema. On evaluation revealed, HB - 5.5 gm/dl, Urine showed 2+ proteins, granular casts, Plenty of RBC and 8-10 WBC/PHF.

Blood uera 134 mg/dL, Creatinine 11.6 mg/dl, Sodium 122 mEq/L, Anti-GBM antibodies were positive. Other serological investigations for ANA, ANCA, HBsAg, HCV and HIV were negative treated with plasmapheresis. Renal biopsy : Light microscopy (12 glomeruli) showed diffuse thickening of basement membrane. The lumens of capillaries were patient. There were extensive cellular crescents seen more than 50% of glomeruli.

Immunofluorescence showed linear ribbon like deposits of IgG along the capillaries. The diagnosis of membranous glomerulonephritis with anti-GBM transformation was made.

Conclusion: Transformation of human membranous nephropathy a condition assumed to be produced by immune complexes in to an anti-glomerular-basement-membrane-glomerulonephritis distinctly unusual.

Secondary Antiphospholipid Syndrome Presenting As Chronic Renal Failure

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We are reporting an interesting case where a 34 year old female, known hypertensive for past 10 years presented to us with decreased urinary output for 2-3 days and 1 episode of generalized tonic clonic seizure prior to presentation. There was no associated fever, headache, vomiting or weakness of any limb. She had no DM, CAD or chronic illness. Further probing revealed 1 episode of hemiparesis 6 years back, from which she recovered in 1-2 months. She was not investigated for cause of CVA at that time and no records were available. There was no h/o recurrent abortion but delivery of a premature malformed fetus 8-9 years back was present. In her family, her sister had SLE and expired due to CVA but delivery of a premature malformed fetus 8-9 years back was present. There was no history of recurrent abortion but delivery of a premature malformed fetus 8-9 years back was present.

In her family, her sister had SLE and expired due to CVA but delivery of a premature malformed fetus 8-9 years back was present. In her family, her sister had SLE and expired due to CVA but delivery of a premature malformed fetus 8-9 years back was present.

Relevant investigations revealed dimorphic anemia (HB = 6.6 gm%), azotemia (BUN : 55 mg/dl; creatinine 10.8 mg/dl), proteinuria and pus cells with RBC casts in the urine. Special examination was unremarkable.

We report a case of a 24-year-old male presenting on the third day following snakebite envenomation. Patient developed breathlessness, sudden deterioration in the sensorium, presence of gross edema, swelling, petechiae and subcutaneous bleeding with cyanosis in left arm, was drowsy and spontaneously moving all four limbs. Clotting time of more than 15 mins detected and a total of 40 vials of anti-snake-venom (ASV) given over 12 hours. Renal parameters were S. urea 126 mg/dl; S. creatinine 2.3 mg/dl. Started on IV fluids. Urine output adequately maintained.

Conclusions: 1. ARF after multiple wasp stings is not necessarily due to hemolysis or rhabdomyolysis. It may result from direct toxic effect or from hypersensitivity reaction to wasp venom. 2. ARF due to multiple wasp stings can lead to a fatal outcome in children.
in our case a neurological hemorrhagic abnormality, a subarachnoid hemorrhage, with a non-hemorrhagic cerebral infarction. Once bitten by a snake, a wide spectrum of clinical manifestations may result. The emphasis for treatment should be placed on early and adequate medical management.

598. Toxic Methemoglobinemia

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Methemoglobin is an altered state of hemoglobin in which the ferric (Fe³⁺) iron of the heme is oxidized to the ferrous (Fe²⁺) state. The patient with increased concentrations of methemoglobin has a functional anemia, to the extent that the circulating methemoglobin-containing molecules are unable to carry oxygen and deliver it to the tissues.

Case Study: A 35 yr old male presented to casualty with impairment of consciousness and bluish discoloration of extremities and tongue. He had been working in a dyes and chemicals factory. One of the containers was found to be leaking on that day. If he did not have any complaints before going to work, but after working in the factory for about an hour he complained of generalized weakness, choking sensation in chest and throat and shortly developed cyanosis and headache. Following it he had vomiting twice and then his consciousness level started dropping when he was immediately shifted to the hospital. On examination he had tachycardia (pulse rate = 128/min), tachypnea (RR=28/min), slate grey cyanosis. His blood pressure and temperature were normal. He was stuporous. No other abnormalities were found on neurological, cardiovascular, respiratory and alimentary system examination. ECG and Chest X-ray were unremarkable. ABG was suggestive of mild metabolic acidosis and PaO₂ of 92%. Pulse oximetry showed SaO₂ of 75%. The blood collected for routine examination was brownish red in colour. CBC and other routine biochemical tests were normal. Patient was not responding to continuous oxygen. Based on occupational history and clinical picture a provisional diagnosis of toxic methemoglobinemia was kept and 1 mg/kg of methylene blue was given intravenously to which the patient responded in a short time.

In India, the chemical industry is growing rapidly and is highly concentrated in the western part, manufacturing dyes and dye intermediates. Workers in these units are at high risk of developing acute methemoglobinemia due to multiple reasons viz. lack of awareness, non-availability of protective devices and poor work practices.

599. Paraplegia Due to Organophosphorus Poisoning

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Organophosphorus (OP) poisoning accounts for the overwhelming majority of self poisoning in our country. Delayed neuropathy is very uncommon with the present generation of OP insecticides available commercially. We studied 300 patients over a period of 3 yrs. and found only one pt. with delayed neuropathy.

PB aged 23 yrs. presented to us on 19/5/04 with C/o weakness of both lower limbs and urge incontinence of urine for 5 days. His urge incontinence passed off over the next week but the weakness of the lower limbs persisted. He gave a H/O ingestion of OP (Noorani) 505 - containing chlorpyrophos 50% and cypermethrin 5%) about 6 weeks ago which was treated with PAM + Atropine. On examination - pt. was conscious and cooperative. Higher functions, cranial nerves and upper limbs were all normal. In the lower limbs there was wasting of extensor digitorum brevis (both sides). Power was 2-3/5 in the proximal group and 0/5 in the distal limb muscles of the lower limbs. Tone was increased in the proximal group and reduced in the distal. Plantar was equivocal on both sides. No sensory deficit was detected. Abdominal and cremasteric reflexes were lost. Ankle jerks were normal but knee jerks were brisk bilaterally.

Investigation showed normal blood sugar, urea, creatinine, Hb-10.5 gm% and ESR - 5 mm in the 1st hr. NCV studies showed in both lower limbs sensory motor neuropathy (axonopathy) in advanced stage.

Pt. made a gradual recovery and was discharged in ambulant condition after 3 weeks.

Delayed neuromyelopathy is very uncommon in OP poisoning in our experience. Preferential affection of the lower limbs completely sparing the upper limbs is peculiar in this case.

600. Hypoxic Encephalopathy - A Rare Complication of Aluminium Phosphide (Celphos) Poisoning

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Aluminium phosphide (Celphos) is a pesticide commonly used to preserve wheat and is a common cause of accidental poisoning. It usually presents with gastrointestinal upset and shock followed by adult respiratory distress syndrome and central nervous system manifestations. We present a case report of a young female who consumed 1/2 tab of celphos with suicidal intent and subsequently developed hypoxic encephalopathy, which forms the subject of this communication.

A previously healthy 24 years female was admitted at Hamidia Hospital Bhopal with the history if ingestion of 1/2 fresh tab of celphos (3 gm). Within 10-15 mins, she developed persistent vomiting and was hospitalized after 2 hours of ingestion. On examination she was drowsy, restless cyanosed with cold extremities. Heart rate was 130/min with absent peripheral pulses, respiratory rate was 40/min, oral temp was 96°F, BP was not recordable and O₂ saturation by pulse oximetry was 84%. Gastric lavage with KMnO₄ was done and charcoal tab were left in stomach. Oxygenation and resuscitative measures for shock were started using intravenous fluids, hydrocortisone, dopamine infusion and ceftriaxone. Subsequently ECG was done which showed ST-T changes suggestive of myocarditis. Patient responded to therapy and after 6 hrs of intensive care, her BP rose to 90/60 mmHg and cyanosis improved but her conscious level deteriorated and she became stuporose. Next morning her BP recovered to normal, cyanosis improved, respiratory rate 20/min but she was comatose with no neurological localizing signs except for extensor plantar response. CT Scan head was done after 2 days, which showed infarcts in multiple territories suggestive of hypoxic ischaemic encephalopathy. Patient was managed accordingly with IV mannitol, corticosteroids and O₂. But patient showed no signs of improvement in her conscious status and after 20 days she developed multiple bed sores and septicaemia. She was treated accordingly, but 10 days later she collapsed.

In Celphos poisoning, patient usually preserves consciousness till end and rarely it leads to acute hypoxic encephalopathy or coma and possible explanation for this manifestation is acute circulatory failure due to myocarditis.

601. Seizures and Acute Renal Failure Due to Herbal Aphrodisiac Containing Mercury

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A 23 year young male was admitted with acute onset of generalized tonic-clonic seizures for the first time in his life. After which he had fall and suffered injuries to palate, tongue and upper lip; aspirated blood and he required tracheostomy.
602. Scorpion Sting with Delayed Acute Renal Failure (On 6th Day)

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A rare case of a 30 yrs female who presented with vomiting and loose motions, decreased urine output and increasing swelling over the body of 2 days duration and severe breathlessness since morning.

There was history of her having had a scorpion sting over right middle finger 6 days prior to the onset of first symptom and on questioning admitted to have had intense pain in arm and leg and reddish coloured urine for 2 days after the sting.

Clinical examination revealed raised Jugal Venous Pressure, facial puffiness, pedal oedema and crepitations over the chest. Investigations revealed metabolic acidosis, microscopic hematuria, Blood urea - 110 mg%, Creatinine of 7.8 mg% and X-ray showed bilateral pulmonary oedema features. USG abdomen showed bilateral renal loss of corticomedullary differentiation.

Patient was diagnosed as acute renal shutdown with likely haemoglobinuria and haemolysis (initially). After two sessions of haemodialysis, antibiotics and steroids, patient recovered rapidly. We didn’t find such delayed presentation of acute renal failure (after 6 days) of Scorpion sting in an adult in literature.

603. Rhabdomyolysis - An Uncommon Complication of Organophosphorus Poisoning

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Government Medical College, Mysore.

A thirty five year old man is brought to the emergency department with suspected poisoning with Parathon. At the time of admission, he was conscious, Pulse 67 pm, BP 120/70 mmHg, pupils 2 mm bilaterally, breath smell of kerosene. Cardiovascular and respiratory systems, abdomen were normal on examination. Serum Pseudocholinesterase was 1800 U/L. ABG was normal. Gastric lavage was given, and he was started on pralidoxime and atropine intravenously in titrated doses, along with other supportive measures. He developed respiratory paralysis on the second day of admission. He was intubated and was managed with mechanical ventilator. He was conscious, stable and ABCGs were normal till the day 7. He developed oliguria and urine became dark in colour. Urine examination showed no RBCs, but dip-stick positive for heme. His Creatine Phosphokinase was > 2,00,000 U, LDH 700 U/L. His BUN and Serum Creatinine became raised, and he developed uremic encephalopathy on day 10, for which he under went daily Hemodialysis for 3 days, after which he became conscious.

He required tracheostomy on the day 13. He was weaned from ventilator on day 16. He underwent 10 more cycles of hemodialysis over the next 2 weeks for persistent azotemia. He had weakness of both deltoids, hip flexors, Right quadriceps and Right ankle dorsiflexors. He was put on physiotherapy. He recovered from azotemia in over 3-4 weeks. Muscle biopsy from quadriceps confirmed rhabdomyolysis. CPK enzyme was 200 U/L at time of discharge.

608. A Rare Case of Myocardial Tuberculosis

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Ms I, a 20 yr old female presented to MOPD with complaints of swelling of feet and palpitations for one week along with low to moderate grade fever, anorexia and weight loss for six months. On examination, patient was pale, had tachycardia, raised JVP and pedal edema. Abdominal examination revealed an enlarged and tender liver. Rest of the examination including CVS examination was within normal limits. Investigations revealed Hemoglobin of 8 gm% and ESR of 55 mm in 1st hour. LFT, KFT and blood sugar were normal. Serial blood and urine cultures were sterile.

Mantoux was 18 x 14 mm. ECG showed sinus tachycardia and CXR showed mild cardiomegaly. Bone marrow examination did not show presence of granulomas or AFB. CRP was positive ASO titres were < 200. CPK-MB was 20 u/L. RA factor, ANA and LE cell phenomenon was negative. Echocardiography was done which showed multiple masses in the right atrium. One mass was seen in the left atrial appendage.

Mild pericardial effusion was present. MRI chest was done which showed diffuse nodular thickenings in pericardium, which were eroding into the myocardium causing loss of differentiating planes between endo-myo-pericardial sites. Mild pericardial effusion with constrictive pericarditis and matted mediastinal lymphadenopathy were suggestive of a tubercular etiology. The patient has been put on antitubercular therapy along with steroids. Patient is on regular follow up, is afebrile and has regained her weight and appetite.

The incidence of myocardial TB is < 0.3% in all TB patients in post mortem biopsy. Single or multiple tuberculomas are present maximally in the right atrium. They may erode into myocardium causing thrombosis, dissemination, SVC obstruction, arrhythmias and sudden cardiac death. Resolution of myocardial lesions may take months. Antemortem diagnosis of endomyocardial tuberculosis is rare.

612. Insulin Induced Aggravation of Cardiac Failure : A Rare Side Effect of Insulin

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Generalized edema due to water retention is a very rare complication of insulin therapy. Though the exact mechanism of this phenomenon is not known-anti-natriuresis, increased vascular permeability might be the mechanisms. It is especially common in the patients with hepatic dysfunction, hypoalbuminemia and high level of insulin resistance. We are reporting a case of ‘insulin edema’ - where the insulin was the causative factor in the aggravation of the cardiac failure.

Case : 38 year old male, known case of type 2 DM with hypertension since 4 years, was admitted with complaints of breathlessness, generalized swelling since 2 months. Due to poor glycemic controls, the patient was started on insulin treatment (Human mixtard) 3 months back. Patient felt worse after the insulin therapy and he himself omitted it. At that time we ignored patient’s statement and considered him non-compliant. He was a smoker and consumed alcohol on regular basis.

On examination pulse was 120/min, BP was 160/90 mm Hg, and he had anasarca, bibasal crepitations. Rest of the clinical
examination revealed no significant abnormality. His CXR showed cardiomegaly, bilateral pleural effusions. Pleural fluid was transudate. FBS was 190 mg% and PPBS was 240 mg%. Since last 15 days he was not taking any treatment for diabetes. 2D ECHO showed dilated cardiomyopathy with LVEF of 30%. CT abdomen showed fatty liver. He had low albumin (2.2 mg%), rest of LFT were normal. RFT were completely normal.

He was started on antifailure treatment and glimepiride. He improved significantly as far as his cardiac failure was concerned over 7 days. For better sugar control again he was started on insulin (Human mixtard). The patients was very reluctant for insulin as he believed that the edema would worsen with insulin. At that time we had no reason to consider patient’s argument. After 3-4 days of insulin, we noticed the peripheral edema was increasing. At 10th day patient had frank episode of pulmonary edema. He was treated successfully for the pulmonary edema.

At this point we reviewed the literature for the possibility of insulin as a cause for aggravation of cardiac failure. We found few references of such intriguing phenomenon.

We stopped insulin and observed the patient for 2 weeks indoor. He received antifailure treatment and metformin this time. By now he was again free of failure symptoms - had significant reduction in the edema. We gave him the “Insulin challenge” to prove our hypothesis of insulin edema. After restarting of the insulin (this time regular/soluble insulin) we did appreciate that patient’s peripheral edema started increasing significantly.

After this it was confirmed that insulin was the aggravating factor in patient’s cardiac failure (which otherwise was due to dilated cardiomyopathy). We stopped insulin and gave him glimepiride and metformin. At 1 month follow up, the patient was free from failure symptoms.

Because of an observant patient, we learned about a rare side effect of insulin. We emphasize the importance of patient’s observation about their disease-treatment.

613. HIV Cardiomyopathy Presenting As Acute Dyspnoa

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Cardiomyopathy in HIV is a known complication without past history of opportunistic infections. Cardiomyopathy can result from HIV infection per se or as a first time manifestation of opportunistic viral myocarditis. 2D Echo may show reduced ejection fraction with global hypokinesia. Management is with ACE inhibitors and anti failure line of treatment.

Presented here is a case of a 23 year married female with acute onset dyspnoea grade IV of one day duration. There was no history of pedal edema and prior cardiac or pulmonary illness.

On examination patient had tachycardia, was hypotensive and tachypnoeic with bilateral basal crepitations. ECG features were suggestive of myocarditis. The patient was treated with inotropic support comprising of dobutamine. Subsequently patient improved.

On 2D Echo, she had ejection fraction of 30% with global left ventricular hypokinesia. Her Elisa for HIV was positive. Other viral markers such as toxoplasma, EB virus and CMV for causing viral myocarditis were negative.

Uniqueness of the case lies in the fact that in a young woman, where CHF may have rheumatic/nutritional at the background, HIV Cardiomyopathy should be looked into, in the current times.

614. Cerebellar Infarction in HIV Infected Patients

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Introduction: Approximately 30-50% of patients suffering from acquired immunodeficiency syndrome also suffer from neurological complications. These include opportunistic infections neoplasms of the CNS as well as AIDS dementia complex. Stroke due to infectious vasculitis, embolism or anteriopathy may also occur in a significant number of patients. While cerebral infarction is not uncommon in HIV infected patients, there is paucity of reports of cerebellar infarction. We report two cases of cerebellar infarction in HIV infected patients because of its rarity.

Case 1: A 4 years old male, a known IVDU presented with giddiness and unsteady gait since 15 days. He had weight loss, low grade fever, frequent loose motion in the last 3 months. On examination, he had oral thrush, BP 110/70 mmHg. He had scanning speech. Cranial nerves were intact. Nystagmus was absent. Fundoscopy was normal. Tone and strength of muscles were diminished on the right limbs (power grade 3/5). DTR were normal in upper limbs and diminished in lower limbs. Plantar response was downgoing on both sides. Tests of coordination were impaired on the right side. Gait was reeling in type. Sensory examination was normal. There was no carotid or vertebral bruit.

Other systems were normal.

HIV antibody test was positive. CD4 count 348 cells/cu mm. VDRL test, CMV, HSV and toxoplasma antibodies were negative. Mantoux test, mycobacterial antibody test were negative. S. lipid profile and homocysteine level were normal. CSF : Normal pressure, protein 90 mg%, sugar 48 mg%, total cells 30/cu mm., polymorphs 10% lymphocytes 90%. India ink preparation for cryptococcus was negative. CT scan and MRI of brain showed infarction on the anterior inferior cerebellum.

Case 2: A 45 years old male, again an IVDU with history of multiple sexual partners were admitted with difficulty in speech and unsteady gait for 8 days. There was a history of mild headache, low grade fever and weight loss since 3 months. He too had oral candidiasis. CNS examination revealed scanning speech. Nystagmus was present. Ocular fundi were normal. Cranial nerves were intact. There was hypotonia of all limbs. Gait was ataxic. Sensory system was intact. There was no sign of meningial irritation. Other systems were normal.

Investigation: Total lymphocyte count 1100/cu mm. S. bilirubin 1.5 mg%. VDRL test was negative. HCV antibody was positive. HBSAg was negative. HIV antibody test was positive. CD4 count 286/cu mm. Here again, antibody test for CMV, HSV and toxoplasma were negative. Coagulation and lipid profile were normal. CSF : Normal pressure, protein 90 mg%, sugar 48 mg%, total cells 26/cu mm., polymorphs 8%, lymphocytes 92%. India ink preparation for cryptococcus was negative. CT scan of brain showed haemorrhagic infarction on both side of the cerebellum predominantly on the left side. On post GAT, DTPA, MRI the lesion showed peripheral contrast enhanced rim with low signal focal lesion within it suggestive of haemorrhagic cerebellar infarction.

Conclusion: In both the cases, clinical, laboratory and neuroimaging evaluation did not reveal any specific identifiable etiology. HIV arteriopathy may perhaps explain the pathogenesis in our cases. Cerebellar infarction can occur in HIV infected patients.
617. An Unusual Presentation of Antiphospholipid Syndrome

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A known case of 30 years female - Systemic lupus erythematosus (SLE) of five years duration came with malignant hypertension, headache, vomiting, fever and recurrent seizure of three days duration. She became unconscious with status epilepticus on the next day. On fifth day she became conscious with flaccid quadriaparesis. There is history of one miscarriage at the age of 12 weeks.

The confirmatory laboratory values are positive dsDNA (40 IU/ml), moderately high antiphospholipid (33.46 U/ml), anticardiolipin antibodies (IgG = 22.39 U/ml, IgM = 21.12 U/ml) and thrombosis of left internal jugular and transverse sinus on MRI venography.

Malignant hypertension, status epilepticus and quadriaparesis are rare presentations of secondary APS.

618. A Rare Case Report of Adult Onset Still’s Disease (Wissler-Fanconi Syndrome) Presenting as PUO

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Twenty two years male came with high grade fever with chills and Rt knee joint pain since 1 week. O/E - mild pallor and fever. Rest clinically normal. Blood routine- Normal, except raised ESR, RBC normocytic normochromic to microcytic hypochromic; malaria by QBC and smear - negative; Culture-no growth; chest X-ray - normal; USG abdomen - mild hepato-megaly (homogenous); mantoux, widal - negative; ASO - negative empirically given chloroquine, Ciprofloxacin, Ceftriaxone. High grade fever persisting, minimal knee joint swelling, bilateral elbow joint pain, also muscle pain. CPK - normal; RA factor, ANA negative, Blood culture for endocarditis - no growth; ECHO - normal study, HIV - negative; Brucella agg. - negative. Fever, joint pain persisting, muscle pain reduced. Empirically started on ATT, NSAID for pain relief. Rpt. USG: minimal Rt. sided joint pain persisting, muscle pain reduced. Empirically started on Inj. Amphotericin B and after one month there was marginal improvement in power though because of spastic gait he remained wheelchair bound. Clinically there was no further progress of the lesion. Repeat imaging could not be done as he was lost to follow up.

619. A Case of Pulmonary - Vertebral Aspergillosis with Myelopathy

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Fifty six years non diabetic male patient presented with sudden onset of an electric shock like sensation radiating from the neck to the back and weakness and numbness of both lower limbs following about of sneezing. There was no history of root pains or sphincter incontinence, band like sensation, fever, loss of weight or chronic cough. He gave past history of pulmonary tuberculosis 10 years ago. On examination the patient was moderately built and normotensive. There was no pallor, lymphadenopathy, or neurocutaneous markers. Neurological examination revealed increased tone in both lower limbs, exaggerated knee and ankle jerks, bilateral extensor plantar reflexes, spastic gait and a sensory level at D4 spinal segment. Magnetic resonance imaging of the dorsal spine revealed a low signal intensity mass involving the right lung apex extending into right upper dorsal foramina producing moderate cord compression. He was started on ATT and steroids empirically and he underwent a laminectomy and dual biopsy of D1-D2 vertebrae. The per operative findings were a bluish yellow area of dural thickening seen over D2 level, adherent to hypertrophied longitudinal ligament. Histopathology of the dual biopsy revealed fibrocartillogenous tissue with dense inflammatory infiltrate composed of lymphocytes plasma cells and histiocytes. Some areas showed ball like filamentous structures which were sepatate and PAS positive. He was started on Inj. Amphotericin B and after one month there was marginal improvement in power though because of spastic gait he remained wheelchair bound. Clinically there was no further progress of the lesion. Repeat imaging could not be done as he was lost to follow up.

620. Neuromyotonia

S Iyer, H Kagdi, NC Mehta, RV Dosi, NV Yajnik, MR Shah, V Desai
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A 30 years male patient presented with complaint of weakness in left upper limb or one year, gradual onset, progressive, proximal more than distal, associated with complaint of thinning of limb followed by similar complaints in right upper limb six months later.

No other positive history. No significant past, family or personal history. On examination vitals were normal. CNS examination showed hypotonia and grade 3 power in both upper limbs with fasciculation and myokalma with absent biceps and left supinator reflexes with exaggerated rest of the reflexes. Rest of the systemic examination was normal.

Routine investigations were normal. EMG and NCV studies showed Neuromyotonia.

622. Progressive External Ophthalmoplegia

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Medical College, Vadodara.

A 49 year male patient presented with complaint of progressive dropping of bilateral eyelids since age of five years with no complaint of diurnal variation of weakness of eyelids or no other complaint suggestive of other cranial nerve involvement.

Family history was significant for similar complaint in patient’s mother, brother and patient’s daughter. On examination vitals were there normal.

Ptosis was present with weakness of levator palpebrae superioris, superior rectus, inferior oblique muscles. Routine investigations were normal. EMG was suggestive of myopathic potentials.

623. Persistent Headache - An Interesting Case

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Lady Hardinge Medical College, New Delhi.

A 34 years male with bilateral pulmonary tuberculosis, already on ATT, presented to us with severe diffuse headache unresponsive to maximum doses of analgesics. On examination, he had terminal neck rigidity due to which a lumbar puncture was done to rule out tubercular meningitis. It revealed a protein = 425 mg%, sugar 32 mg%, TLC = 80/mm$^3$ and DLC-N8L92.
Patient was put on 5 drugs ATT, IV steroids and decongestive therapy which led to symptomatic improvement. CECT Brain revealed a normal study. After 3-5 days patient again presented with headache, vomiting and h/o seizure like activity. Repeat LP revealed - protein = 144 mg%, sugar = 58 mg%, TLC = 16/mm3 and DLC-N2L98. ZN stain, AFB stain and India ink staining were negative. Antiepileptic drugs were added to the treatment regimen. Patients hematological, biochemical profile as well as systemic examination was normal. Fundus examination was also normal. On the 21st day of the second admission, the patient developed left sided sixth and seventh cranial nerve palsy. There was no other focal neurological deficit. In view of these fresh findings and persistent headache, MRI scan was done which showed bilateral symmetrical non enhancing basal ganglionic hyperintensities s/o ischemic or demyelinating etiologies. In presence of pulmonary tuberculosis, meningitis, focal CNS signs and demyelinating lesions on MRI, a possibility of HIV was kept which was subsequently proved by a positive serology. Interesting points in this case are to be discussed.

626. Pneumocephalus - Mystery Unravelled

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Traumatic CSF rhinorrhea is a common clinical entity but non-traumatic CSF rhinorrhea is very rare. Pneumocephalus is a consistent finding in all cases of CSF. Rhinorrhea. It may be a clue to CSF rhinorrhea in patients who come with watery nasal discharge from nose/recurrent meningitis. Localization of the site of leak is important to carry out surgical/endoscopic repair. Extremely rarely granulomatous infiltration of nasal mucosa may result in CSF rhinorrhea. Very few cases of tuberculosis of nasal mucosa causing CSF rhinorrhea are reported in world literature.

A 51 years female presented with h/o fever, headache and watery nasal discharge since two days. On examination, patient had signs of meningial irritation. CT showed pneumocephalus. CSF analysis was suggestive of acute pyogenic meningitis and pneumococcus was isolated. She was treated with Inj. Ceftriaxone. After recovery from acute infection, endoscopic repair of defect was done. Biopsy of nasal mucosa showed tuberculosis. Patient was treated with 4 drug ATT. She is currently asymptomatic since 8 months.

631. Rasmussen’s Encephalitis

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Institute of Internal Medicine, Madras Medical College, Chennai - 600 039.

Introduction: We are reporting a case of Epilepsia Partialis Continua in a 14 years boy due to possible Rasmussen’s encephalitis.

Material and Methods: A 14 years boy presented to us with right-sided focal, partial seizures without aura, sensory accompaniments or loss of consciousness. These seizures have been present since early childhood and have increased in frequency during the past 6 months. Since 3 days the seizures are continuous unabated and are right-sided focal, motor partial seizures; the child developed right-sided motor hemiplegia. This was of subacute onset following an episode of fever with seizure. The hemiplegia was initially progressive.

Antental, birth, delivery, milestone acquisition were normal. Language-normal; Examination revealed. Right-sided epilepsy partialis continua. Right sided spastic hemiplegia, arms involved more than legs.

Investigations: Neuropsychological assessment: On stanford intelligence scale Mental age is 6 years EEG showed left frontal temporal epileptogenic activity.

Viral studies: Titres for HSV and Coxsackie virus were negative; CT showed atrophy of the left hemisphere.

Discussion: This case seemed to answer the criterial needed to diagnose Rasmussen’s syndrome viz. 1) Development of epilepsia partialis continua, 2) Slowly progressive hemiparesis and mental retardation, 3) Progressive atrophy of the affected hemisphere.

Conclusion and Message: Rasmussen’s syndrome is focal encephalitis that usually develops following a viral infection. The exact mechanism is not known. Phenytoin, Carbamezepine clobazam caused partial improvement.

632. Becker’s Muscular Dystrophy in an Indian Family

R Dewan, VV Mittal, S Kumar, R Anand, S Zachariah, P Mehta, D Manocha, K Kumar
Maulana Azad Medical College, New Delhi.

Mr. R, 17 years male and his brother R, 13 years, presented with complaints of insidious onset gradually progressive weakness in all 4 limbs for 7 yr and 2 yr respectively. History was s/o progressive proximal weakness. H/O loss of bulk in all limbs except calves. H/O poor scholastic performance was present. No H/O tightness/looseness, fasciculation involuntary movement. The youngest brother Mr. R, and 2 sisters were asymptomatic. One of the maternal uncles of the patient also had similar problem and was bedridden since the age of 23 yr. On examination vitals were normal. BMI was decreased in both patients. Kyphoscoliosis, macroglossia and contractures of Achilles tendon were present. Chest, CVS and P/A was WNL. In CNS exam there was presence of poor social interaction and poor short-term memory. Bulb was reduced in all muscles but calves were hypertrophied. Generalized hypotonia was present. Power was reduced in all four limbs with absent deep tendon reflexes, B/L planters flexor. Mr. R, 7000 in R2 ECG was s/o increased R+S voltage in right precordial leads in R1 and R2. ECHO done was normal. EMG of B/L Deltoid and Quadriceps revealed myopathic pattern, NCV of all 4 limbs was normal. Muscle biopsy in R3 was s/o myopathy. Special stains on biopsy tissue showed patchy Dystrophin I and III with intact Dystrophin II, s/o Becker’s muscular dystrophy. R3 was found to have proximal muscle weakness and positive Gower’s sign with raised CPK-T levels (12490). Both the sisters were found to be normal on exam and investigation.

This is a classic case of a family of Becker’s where all three brothers and the maternal uncle had the disease. Becker’s muscular dystrophy is genetically determined progressive degenerative myopathies. Abnormal or reduced levels of Dystrophin characterize it. It’s incidence is 1/6150 male births.

633. A Rare Case of Wernicke’s Encephalopathy Induced by Hyperemesis Gravidarum

M Krishnakumar, KP Gireesh Kumar, Bindu K Nair
Amrita Institute of Medical Sciences and Research Centre, Elamakkara PO, Kochi - 682 026.

A 27 years female patient in the 16th week of third pregnancy presented with persistent vomiting of 3 month’s duration and altered sensorium of 2 days duration. Prior to the admission, she was treated symptomatically in a peripheral hospital with intravenous glucose infusion. On examination, patient was disoriented, pupils were reacting sluggishly to light with evidence of horizontal nystagmus. There was paucity of movements on the right side with plantar response bilaterally extensor.

Her blood picture, blood sugar and renal functions tests were normal. Serum aminotransferases and prothrombin time were elevated. MRI scan of the brain showed hyperintense lesions in the periaqueductal, periventricular and cerebellar grey matter.
areas. The patient was administered intravenous thiamine after which there was improvement of level of consciousness and focal deficits.

During the stay in the hospital patient developed bilateral pneumonia which was treated with antibiotics. Since she had persistent tachycardia, mild hypertension and her thyroid function tests were suggestive of hyperthyroidism - regimen for thyrotoxicosis was given. She also underwent induction of labour and expulsion of conceptus since ultrasound showed absent cardiac activity for the foetus. She responded to therapy with IV thiamine, antibiotics and antithyroid medications.

Hyperemesis Gravidarum is a rare etiological factor for Wernicke’s Encephalopathy. A review of literature revealed 21 cases of Wernicke’s Encephalopathy induced by hyperemesis gravidarum in the past 30 years. Only 50% of these pregnancies resulted in the birth of a normal infant. Untreated Wernicke’s Encephalopathy is always fatal. The mortality in treated cases is 10%. Concomitant infection or presence of liver failure make the cause of death unclear. Characteristic findings on magnetic resonance imaging in Wernicke’s Encephalopathy enables early confirmation of the diagnosis and hence it is the investigation of choice.

634. Mobius Syndrome

MV Kaneria, Akbar, R Bhole, J Hrishikesh, M Pardesi, S Pednekar
TN Medical College and BYL Nair Ch. Hospital, Mumbai.

A 65 years male presented with fever, headache and vomiting of 5 days duration. There was no history of HT/DM or IHD. There was no other positive history. On examination, he was drowsy but arousable. Neck rigidity and bilateral ptosis were present. Plantars were bilaterally extensor and there was no focal neurological deficit. He was haemodynamically stable. All other system were unremarkable on examination. CSF study was suggestive of pyogenic meningitis. All biochemical investigations and chest radiograph were within normal limits. He was treated with meningitic doses of Ceftriaxone and cerebral dehydrants. It was noticed that both his sons who were attending to him also had bilateral ptosis and absent wrinkling of the forehead on upward gaze. On enquiry, they said that this was present since childhood. The family was diagnosed as having Mobius Syndrome.

Discussion: Mobius syndrome, a rare genetic disorder characterized by facial paralysis, is caused by the absence or underdevelopment of the 6th and 7th cranial nerves. These nerves control eye movements and facial expression. As children get older, lack of facial expression and inability to smile become the dominant visible symptoms.

There is no cure for this syndrome. With proper care and treatment, many individuals have normal life expectancy, in spite of the severe impairments that characterize this disorder.

636. A Case Report of Parkinson’s Disease with Catatonia Following Viral Encephalitis

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St. John’s Medical College Hospital, John Nagar, Bangalore, Karnataka - 5600 034.

Introduction: Encephalitis of Von Economo is a well known cause of Parkinson’s disease.

Mr. M, a 21 years man from Bangalore presented with a history of fever, and headache of 3 days duration. Clinical examination revealed the presence of meningeal signs. Initial CSF study showed 30 cells with lymphocyte predominance and normal protein. CSF for Antimycobacterial and HSV antibodies was negative. The patient was treated symptomatically for viral encephalitis.

On the 5th day of hospital stay, patient developed extrapyramidal features such as mask like facies, postural imbalance, hypophonia, rigidity and bradyphrenia. The patient also had catatonic features like waxy flexibility. An MRI brain done showed bilateral hyperintensities in the substantia nigra. The patient was treated with syndopa and improved gradually. Postencephalitic Parkinson’s with catatonia has been described very rarely in literature.

638. Non-Islet Cell Tumor Presenting as Hypoglycemia

K Selva Kumary, P Alagia Nambi
Sri Gokulam Hospital, Salem - 636 004.

Mr. Azzizudin, 70 years gentleman, known diabetic, hypertensive, non alcoholic, non smoker, thin built, who was revived in our emergency room (Sri Gokulam Hospital, Salem) by administering 100 ml of 25% Dextrose as he was brought in a state of altered sensorium. When he and his family members were probed they recollected that it was a recurrent event. The reason for hypoglycemia was also direct (i.e.) the commonest cause of hypoglypemia wherein anti diabetic medication was taken regularly as ever while food was avoided, only when interrogated patient could correlate that his inability to have his regular food was because of anorexia, nausea, severe abdominal pain radiating to the back. General examination and systemic examination were not contributory. Investigation: Complete haemogram was normal excepting the HB 8.8 gms%. Liver function tests: - Normal X-ray chest PA view - Elevation of left hemi diaphragm. USG Abdomen: Pancreas - Normal in size and echo pattern. There is 4.4 x 3.5 cms solild mass in the region/tail of pancreas and is seen engulfing the celiac axis. Gall stones seen. CT abdomen: Soft tissue mass superior to the neck of the pancreas and anterior to the crus of the diaphragm -? Lymph node mass (or) exophytic mass arising from the neck of the pancreas and cholelithiasis seen.

Though he was on oral food in limited quantities he needed 10% Dextrose intravenously continuously to maintain his casual plasma glucose of 120 mg/dl. As huge malignant mass may be the single reason for his persistent hypoglycemia as well as for his other symptoms Laparatomy and proceed was done, per operatively surgeon could find huge non resectable retroperitoneal mass which on histopathological examination revealed Mucin producing adenocarcinoma, infiltrating fibromuscular connective tissue. Therefore he was referred for Oncologist’s care.

Discussion: It is proved beyond doubt that non-islet cell tumours induce persistent hypoglycemia. The reason being that there is a humoral mediator known as insulin like growth factor IGF I and II which is a substance that exhibits high degree of homology to proinsulin. Yet another mechanism is that tumor itself utilizes more glucose. The utility of measuring of IGF II in the circulation in the diagnosis of tumour induced hypoglycemia is not established. Even when the mass is inoperable debulking of the tumor mass (or) partial excision would help in alleviating hypoglycemia.

Conclusion: It is presented to create awareness among physicians and stress the importance that hypoglycemia cannot be neglected as an isolated sign but it has to be considered as a warning signal. It needs careful analysis which will help him in arriving at the correct cause; as earlier recognition alone would prevent morbidity, mortality and improve long term survival of the patient with hidden malignancy.
with testosterone depot injections resulted in improvement of suggested features of polyostotic fibrous dysplasia. Treatment MRI scan. Trans-sphenoidal surgery was done and 2x4x3.5 cm with optic chiasmal compression was visualized on levels (92 mcg/L at 120 mins). A macroadenoma measuring hormone-glucose tolerance test failed to suppress growth hormone (>100 mIu/ml) and low testosterone levels (33.1 ng/dl). A growth poorly developed. Investigations showed elevated prolactin levels with secondary optic atrophy. Secondary sexual characters were dysplasia 9 years back and had recurred. Clinical examination in right maxillary region which was operated revealing fibrous a growth hormone producing macroadenoma. He had a swelling dysfunction. This is a case report of an 18 years old body who fibrous dysplasia, cafe au lait pigmentation and varying endocrine problems.

**As G Sanjay, RS Gupta, MM Gupta, S Garg Government Medical College, Patiala, Punjab.**

**Introduction :** TPP is an uncommon entity characterized by muscular weakness of lower more than upper limbs, proximal more than distal. Failure to recognize the entity may lead to missed diagnosed and may result in recurrences, arrhythmias, respiratory failure and death.

**Summary :** A 30 years female arrived at our hospital in emergency department with H/o weakness of all the four limbs. All the process started when patient had fever for 5 days and had vomiting for 1 day, after which patient started having weakness. Patient could not standup from lying down position without support and later on after 6 hours had complete weakness of all the four limbs. No history of bladder involvement. History of weight loss was positive.

**Examination :** GPE was unremarkable except tachycardia.

Local examination showed weakness of extremities with legs affected more than arms. Deep tendon reflexes were depressed with plantars bilaterally flexor. Sensory system normal. Cranial nerves normal.

Routine investigation were normal ECG showed tachycardia with ST ↓ in inf and lat leads. S potassium was 2.8 mEq/L.

**Treatment :** Patient was put on potassium replacement therapy. Patient’s potassium improved in two days to 3.8 mEq/L but her weakness did not improve and tachycardia was persisting. Then thyroid function test were done.

T3-2.61 ng/ml, T4-14.86 mcg/dl, TSH - 0.21 mu/ml.

She was put on anti-thyroid drugs (Carbimazole), betablockers (Propranolol), in addition to potassium replacement.

Patient’s weakness improved in the following 2 days and within a weak the recovery of her paralysis was complete and she was discharged in a satisfactory condition with anti-thyroid drugs, propranolol and oral potassium.

**McCune Albright Syndrome with Acromegaly**

**S Das, JM Idiculla, V Ayyar, B Ganapathi**

St. John’s Medical College Hospital, Bangalore, Karnataka.

McCune Albright syndrome is characterized by polyostotic fibrous dysplasia, cafe au lait pigmentation and varying endocrine dysfunction. This is a case report of an 18 years old body who had right maxillary fibrous dysplasia, acromegalic features and a growth hormone producing macroadenoma. He had a swelling in right maxillary region which was operated revealing fibrous dysplasia 9 years back and had recurred. Clinical examination revealed features of gigantism. He had bitemporal hemianopia with secondary optic atrophy. Secondary sexual characters were poorly developed. Investigations showed elevated prolactin levels (>100 mhu/ml) and low testosterone levels (33.1 ng/dl). A growth hormone-glucose tolerance test failed to suppress growth hormone levels (92 mcg/L at 120 mins). A macroadenoma measuring 2x4x3.5 cm with optic chiasmal compression was visualized on MRI scan. Trans-sphenoidal surgery was done and histopathology confirmed pituitary macroadenoma. Bone scan suggested features of polyostotic fibrous dysplasia. Treatment with testosterone depot injections resulted in improvement of secondary sexual characters and his height remained static.

A 55 years woman was symptomatic with 4-week history of sweet taste to all foods. The taste between meals was normal. There was no associated nausea, vomiting, or altered sensorium. The dysgeusia progressed over next 2 weeks and unpleasant sweet taste was associated with every food item patient ate. There was no history of cough, hoarseness, weight loss, respiratory symptoms, or altered smell. She was a smoker but had stopped smoking for last 6 weeks due to sickening sweet taste. Clinically general examination, pulse rate, and blood pressure were normal. There was no dehydration. The respiratory rate was 20 per minute. Respiratory system examination revealed features of left pleural effusion. Other systems were clinically normal. The routine haemogram, urinalysis, serum bilirubin, liver enzymes, serum creatinine, blood urea nitrogen, plasma glucose, thyroid function tests, serum cortisol, serum potassium were normal. The serum sodium levels were 118 mmol/L. The urinary osmolality was 356 mmol/Kg with serum hypo-osmolality (238 mmol/kg). This was compatible with a diagnosis of SIADH. Chest radiograph revealed multiple nodular infiltration in right lung and left sided pleural, and pericardial effusion. Fine needle aspiration of lung nodules revealed a diagnosis of small cell carcinoma of lung with pericardial and pleural involvement. The sweet taste subsided over a week on water restriction as her serum sodium concentration increased to 132 mmol/L. She was advised combination chemotherapy and radiotherapy, which she could not afford. The dysgeusia did not recur again. She died six months later. In this patient of small cell carcinoma of lung the presenting symptom was dysgeusia caused by hyponatraemia due to SIADH.

**Case Report :** A 68 years woman had chest pain and her ECG showed lateral wall ischemia. Her CPK-MB was high and serum cholesterol was 280 mg%. She was started on antiplatelets and atorvastatin 10 mg/day. Five days later she was readmitted with two days history of severe muscle pain. She had severe muscle tenderness. CPK total was 16,800 IU/ml. Haemogram, sugar, urea and creatinine were normal. Atorvastatin was stopped. Over next few days CPK total came down to 6000 IU/ml. Though partly relieved of pain, her persistent elevated CPK made us to evaluate for hypothyroidism. Her TSH was > 60 IU/ml. She was put on thyroxin and over next few days she improved clinically and her CPK returned to normal.

**Conclusion :** Myxoedema should be considered in people with hypercholesterolaemia before starting statins, because thyroxine will usually resolve the problem, as in our case. Hypothyroidism might potentiate the toxic myopathy induced by statins. In our case, however, the muscle toxicity was not reversed completely by stopping atorvastatin. Only after starting thyroxine did the raised plasma enzyme activities fall towards normal values. Atorvastatin might have exacerbated myxoedema myopathy in our case.

**Challenge**

**R Bommanan, P Dhanalakshmi, R Sudha, N Balamurugan Sri Gokulam Hospital, Salem.**

**Background :** With the increasing use of statins, toxic myopathy related to these drugs is becoming apparent. Hypercholesterolaemia is a common and may be the sole presentation of myxoedema. Likewise myopathy, which may be clinical or sub clinical can occur in myxoedema. We report on a patient with myxoedema who presented with hypercholesterolaemia and developed apparent muscle toxicity after taking atorvastatin.

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A 27 years male patient presented at medicine outpatient department with history of palpitation and headache since one year; and diminished vision since one month. There was no significant past or family history. He was a known smoker and not a known alcoholic.

On Examination Pulse: 102 per minute BP: RUL- 230/170 mmHg, LUL - 230/170 mmHg, RLL - 240/174 mmHg. Standing for 3 minutes BP: 200/146 mmHg, there was a postural drop.


Routine investigations were normal. ECG showed LVH with strain, Chest X-ray was normal, USG abdomen-hypoechogenic mass seen in right suprarenal area (pheochromocytoma), CT scan abdomen - well defined hypodense mass lesion measuring 6.4 x 6.2 cms in right supra renal region. Areas of hypodense region are seen within this mass suggestive of necrosis (pheochromocytoma). Cranial CT scan - normal, VMA level (24 hrs urine)- positive.

Patient was diagnosed to have Pheochromocytoma.

Pheochromocytomas are rare, fewer than 1% of hypertensive patients harbour a chromaffin tumour as the cause of increased BP. Most of the patients present with headache (80) and palpitation (64): blurring of vision being a rare symptom (11).

645. Hand-Schuller-Christian Disease Presenting as Diabetes Insipidus

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Hand-Schuller-Christian disease is an infiltrative disorder characterised by monoclonal Langerhans cell proliferation with multifocal involvement. We report a case of Hand-Schuller-Christian-disease with features of Diabetes insipidus.

A four years child presented with recurrent fever and maculo papular rashes over the body. Biopsy of cervical lymph node revealed features suggestive of Langerhans cell histiocytosis. Bone marrow study was normal. She improved with chemotherapy. Later, she presented with two month’s history of polyuria and polydipsia. Upon further investigations, she was found to have central diabetes insipidua which responded to treatment with desmopressin. MRI of the sella was done which revealed absence of posterior pituitary bright spot which is suggestive of infiltration of the structure in this condition.

Diabetes insipidus has been noted to be present in 5-20% of patients with Langerhans cell histiocytosis. Central diabetes insipidus with similar MRI picture was later detected in this patient’s twin sister who is currently under our evaluation. This may indicate a genetic basis for this condition.

646. A Case of Hypothyroidism with Organification Defect

R Thomas, R Balasubramanian, PK Shenoy, HS Kiran
Kasturba Medical College, Manipal.

Fifty nine years male patient, who underwent laser excision for glottic malignancy (Squamous cell carcinoma) three months back. During follow up was detected to have multinodular thyroid swelling. Thyroid function test revealed hypothyroidism. Thyroid scan showed multinodular goitre with a possibility of organification defect which was confirmed by “Perchlorate discharge test”. FNAC of thyroid gland showed features suggestive of florid lymphytic thyroiditis.

“Perchlorate discharge/wash out test - to identify congenital or acquired organification defect i.e. thyroid can trap iodide but cannot organify it. In thyroid organification defect, a significant percentage of labelled iodide is discharged from the thyroid upon administration of perchlorate, indicating a defect in converting accumulated iodide to organically bound iodine. Organification defect is usually seen in congenital hypothyroidism. Very few cases have been reported in which organification defect is acquired. In this case, it is due to lymphocytic thyroiditis.
pathological examination revealed gross and microscopically bilateral findings.

706. IgM Nephropathy - 3 Cases of Varied Clinical Presentation

D Kirpalani, R Prabhu, M Mathew, N Bhaskarananda, M Nagaraj
Kasturba Medical College, Manipal.

Mesangial IgM deposition on Immunofluorescence (IF) staining, in glomeruli from patients of glomerular disease, in absence of other Immunoglobulins, is occasionally seen on renal biopsies. Whether this is a clear cut disease entity or a variation of the complex consisting of minimal change disease, mesangioproliferative glomerulonephritis and focal segmental glomerulosclerosis, is not yet clearly understood. We report 3 cases of mesangial IgM deposition and present their clinical findings below-

Case 1 : Forty three yr M presented with nephrotic syndrome (NS). Urine sediment showed absence of red cells/red cell casts. Twenty four hr urine protein was 1740 mg/ S. creat 1.2 mg/dl, S. protein 4.1 g/dl, S. albumin 1.8 g/dl and S. cholesterol 224 mg/dl. Patient responded to Prednisolone and Ramipril in conventional doses. He had complete remission in 3 months and is relapse free to date (6 months follow up).

Case 2 : Two yr F first presented with acute glomerulonephritis (RBCs in urine, hypertension, azotemia and proteinuria). Urea and creat returned to normal, proteinuria and hypertension disappeared. Two mo later she developed steroid responsive NS. Two steroid responsive relapses later, kidney biopsy (KBx) was done which revealed mesangial proliferative GN with IgM deposits in mesangium. Patient was started on Cyclophosphamide 2.5 mg/kg for 12 wks. This had no effect on relapse rate and she continues to relapse every 3-6 months and is steroid responsive.

Case 3 : Thirty five yr M presented with hematuria and uremia, 2.8 gms proteinuria, normal sized kidneys and S. creat of 17.7 mg/dl. Patient required dialysis. KBx done-Light microscopy consistent with Chronic GN approaching ESRD. On IF microscopy-predominant IgM deposits seen.

Conclusion : IgM nephropathy exists as an entity similar to IgA nephropathy with variable clinical presentation of acute nephritis, nephrotic syndrome or ESRD and variable histopathological features but consistently showing IgM deposition in mesangium.

A Case of Bronchoalveolarcarcinoma in a Young Woman

MM Puri
LRS Institute of Tuberculosis and Respiratory Diseases, New Delhi.

Bronchoalveolar carcinoma is an infrequent malignant lung tumor, especially in patients younger than 50 years. Diagnosis is difficult and usually late because its clinical and radiological features are similar to other lung diseases. We report a case of multinodular diffused and bilateral bronchoalveolar carcinoma in a young woman.

*Adjudged Best Papers and got an award of Rs. 1000/- each from Chairman Scientific Committee, Diamond APICON 2005.