germ layers. Therefore, the diagnosis of sacrococcygeal teratoma was not possible.

A meningomyelocele, one of the differential diagnoses of sacrococcygeal masses, should also be considered here. It is usually covered by a membrane or is partialy covered by the skin. A posterior sacral meningomyelocele may be differentiated from sacrococcygeal teratoma by the change in tension occurring with crying, bulging of the fontanelle when manual pressure is applied over the mass, its more cephalad position, the spinal bony defect and the lack of attachment to the coccyx. Anterior sacral meningomyeloceles present as a presacral mass and are easily demonstrated by computerised tomography with myelogram or magnetic resonance imaging. Furthermore, in meningomyelocele, there are usually neurological defects in the legs. This baby girl did not show a weakness in the lower extremities. None of the other features were present in this baby girl.

A lipoma of cauda equina or filum terminale is easily recognisable histologically and it may be associated with a sacral spinal defect and a capillary haemangioma in the overlying skin. The possibility of cutaneous sinuses like pilonoidal sinuses can be easily excluded in this case, as the overlying skin did not show orifices of sinus tracts. Angiomas, perirectal abscesses and chordomas are uncommon and can be differentiated easily with histology. Therefore, the most likely diagnosis in this case is a hamartoma consisting heterotopic brain tissue in the sacrococcygeal region.

In neonates, the alpha fetoprotein serum levels are highly elevated (term neonates: 41,687 ng/ml, preterm neonates: 158,125 ng/ml) [4,5]. The alpha fetoprotein level of this baby at birth was within the normal range and it cannot be defined as a teratoma. Also yolk sac elements which produce the alpha fetoprotein in teratomas were not present in this tumour . These features further support the absence of a teratoma in this case. In most infants, the alpha fetoprotein levels decrease to normal adult levels within the first 10 months [4,5]. The repeated level six months after the surgery in this patient was normal.

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Amyloidosis associated with HIV infection

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Introduction

HIV/AIDS is a multi system disease, often associated with a variety of chronic inflammatory and infective diseases of varying aetiology. Amyloidosis is a disorder of protein metabolism characterised by extracellular deposition of pathological insoluble fibrillar proteins in organs and tissues [1]. We report a patient with amyloidosis associated with AIDS.

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Case report

A 48-years old married heterosexual woman who presented with shortness of breath had an ejection fraction of 35-40% with global left ventricular hypokinesia without jerky movements in the myocardium. She denied a history of angina. Her ECG, cardiac enzymes and thyroid functions were normal.

She complained of loss of appetite and loss of weight. However her clinical examination was normal. Fasting blood sugar (FBS) was 106 mg/dl. Serum creatinine was normal. Urine full report (UFR) revealed trace protein with no evidence of urinary tract infections. Liver functions were normal other than a reversal of the albumin globulin ratio.

The patient was found to have persistently elevated ESR. Anti nuclear antibodies, Rheumatoid factor, Cytoplasmic Anti-Neutrophil Cytoplasmic Antibodies (c-ANCA) and Perinuclear Anti-Neutrophil Cytoplasmic Antibodies (p-ANCA) were negative. Serum protein electrophoresis showed polyclonal gammopathy with elevated $\alpha 1$, $\alpha 2$, β and γ globulins with compensatory reduction in albumin. Serum calcium was normal. The blood picture showed moderate to severe rouleaux formation suggestive of anemia of chronic disease. Bone marrow showed reactive marrow with granulocyte hyperplasia, which excluded multiple myloma. Mantoux test was negative. Three samples of sputum for acid fast bacilli and bone marrow for tuberculous PCR were negative.

Two months later she presented with bilateral carpal tunnel syndrome which was confirmed by a nerve conduction study. She continued to loose weight and ESR remained elevated. Tumor markers like CEA and CA125 were normal. Serum creatinine was marginally high. Ultrasound scan of the abdomen revealed a small gall bladder with a thick wall and early renal parenchymal disease. As she complained of dyspeptic symptoms, upper gastrointestinal endoscopy was done which showed oesophageal candidiasis.

Because she had heart failure of unknown aetiology, bilateral carpal tunnel syndrome and early renal parenchymal disease the diagnosis of amyloidosis was considered. Rectal biopsy was performed. An amorphous pink substance was seen in vessel walls staining for Congo red. Apple green birefringence was seen under polarised light after Congo red staining. Rectal amyloidosis was diagnosed. The patient did not have a family history of amyloidosis.

The possibility of HIV was considered due to persistent loss of weight, loss of appetite, elevated ESR and oesophageal candidiasis. HIV screen was positive, confirmed by the Western blot test. Hepatitis B and C screening were negative. She was started on anti-retroviral therapy.

Discussion

This is a case report of rectal amyloidosis in an HIV-infected patient. There are four main types of amyloidosis. Primary amyloidosis (AL) is characterised by deposition of amyloid fibrils composed of variable portions of the monoclonal light chain. AA amyloidosis, occurs most frequently as a complication of chronic infection or chronic inflammatory disease. The third type is haemodialysis-related and the final type is familial amyloidotic polyneuropathy [1,2]. Amyloidosis can involve a single organ or multiple organs. The gastrointestinal tract is a common site of amyloid deposition, occurring in 70% of cases [2,3]. Biopsies of affected organs are often performed to confirm the diagnosis. Radiological and angiographic diagnostic studies are insensitive [1]. The characteristic histological finding is an amorphous material with apple green birefringence under polarised light after Congo red staining [1,2]. In our patient, the absence of clonal dominance of amyloid light chains, absence of haemodialysis a history of, negative family history, make AL type, heamodialysis related type and familial type of amyloidosis less likely. Our patient is likely to have secondary AA amyloidosis [4]. Our patient had cardiac involvement which is rarely seen in secondary AA type amyloidosis [4]. Treatment for AA amyloidosis consists of treatment of the cause. Hence, the patient is receiving anti-retroviral therapy.

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