Letters to the Editor

hypothyroidism was kept in mind. Since there were no subcondral fractures and other epiphses i.e. of knee joints were also involved, Perthe's was ruled out. Perthe's is a rare finding in females. The bony age of the child and the biochemistry were normal, ruling out hypothyroidism.

Dysplasia epiphysialis multiplex occurs due to the failure of the epiphses to ossify normally. The radiological picture consists of delayed appearance of the epiphses and irregular and flattened epiphses [5]. Barrington Ward first described dysplasia epiphysialis multiplex in 1912 [2]. Fairbank gave the present term of DEM. Multiple epiphsal dysplasias are more commonly caused by dominant mutations in COMP (EDM1, McKausick 132400) and Col 9A2 genes [1].

The epiphses commonly affected are those of the hips, knees, shoulders, and ankles and less frequently the elbows and the wrists. Irregularities of the vertebral column are also reported but less frequently. As growth continues varying degree of deformity may persist. The epiphses of the long bones are usually normal. The metaphyseal ends may widen due to pressure effect. The hip joints are always affected. Gross fragmentation of the femoral heads with irregular acetabular roofs may be seen. Flattening of the condyles with obliteration of the intercondylar notch may be seen at the knee joint. At the ankles, lateral wedging of the lower tibial epiphses and corresponding deformity of the talus may occur. Congenital talipes equinovarus may sometimes be present [1]. In shoulder joints, the proximal humeral epiphses tend to be flattened and rotated. The tubular bones of the hands are short and stubby. The vertebral column is affected in about 26% of patients [5] and the changes may resemble those seen in Schuermann's disease. Our patient showed most of the radiological features of DEM, but the hands and spines were not affected. The spine is usually affected in the later ages i.e. after 14-15 years of age.

The disease is said to be of familial and hereditary nature by most of the authors. However in the case of our patient, no other family member was affected [1]. The disease is self-limiting however secondary osteoarthritic changes may occur in adult patients. The patients remain dwarves to variable degrees.

REFERENCES
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Splenic Calcification in Primary Hemochromatosis Mimicking Hydatid Cyst

Sir,

Primary hemochromatosis is caused due to the presence of excess of iron deposits in the reticuloendothelial system [1]. In primary hemochromatosis, CT demonstrates diffuse increase in liver parenchymal attenuation with normal splenic attenuation. This does not happen in an advanced stage [1]. We report peripheral splenic (eggshell) and rounded intrasplenic calcification mimicking calcified hydatid cyst in primary hemochromatosis.

A thirty-seven-years-old man presented with the classic triad of primary hemochromatosis; cirrhosis, diabetes mellitus, and hyperpigmentation of one-year's duration. Examination showed hepatosplenomegaly. Investigations revealed serum ferritin level of 1000 ng/ml, serum calcium of 8.1 mg%, and phosphorus of 3.1 mg%. An abdominal radiograph showed faint peripheral and intra-splenic calcifications. Ultrasound revealed moderate splenomegaly and increased peripheral echogenicity with a central rounded lesion that showed acoustic shadowing. On CT scan, there was increased hepatic parenchymal attenuation value (an average of 95 HU) with prominent hepatic and portal veins as low-density structures on plain scans (Fig.1). There was peripheral splenic (eggshell), and rounded intrasplenic calcifications (Fig.1). Fine needle aspiration cytology of the splenic lesion excluded the possibility of hydatid cyst or tumor. Immunological tests for hydatid were normal.

The differentials for peripheral and intrasplenic calcifications are few; echinococcal cysts, infection-pyogenic or tuberculous abscess, infarction, splenic artery aneurysm, dermoid, epidermoid, simple cyst, and
splenic artery aneurysm, splenic cyst, dermoid, epidermoid cyst and old hematoma may show peripheral or curvilinear calcification [2, 3].

Multiple small rounded or ovoid calcified nodules distributed throughout the spleen may represent healed calcified tuberculous nodules or histoplasmosis. The size, number and shape may be clues to their diagnosis [2]. If punctate calcifications are larger and more than six, they are said to be due to previous histoplasmosis [2]. This is not true when seen in a patient from a region endemic for tuberculosis [3]. Multiple calcified granulomata and chronic abscesses of the spleen can be demonstrated in chronic brucellosis. Unlike the lesions in histoplasmosis and tuberculosis, the lesions in chronic brucellosis tend to be suppurating even in the presence of calcifications. The calcified nodules in chronic brucellosis are larger (1-3 cm) and consist of a flocculent calcified centre in a radiolucent area that is surrounded by a laminated calcified rim [3]. In Pneumocystis carinii infection, the formation of splenic calcification is associated with calcification in kidneys and lymph nodes [2]. The splenic infarct calcification is usually triangular or wedge shaped with a broad capsular base [3]. Case reports of splenic calcifications in other diseases have been described in literature [1-4]. The exact mechanism of unique peripheral splenic (eggshell) and a rounded intra splenic calcification in primary hemochromatosis is not known.

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