Primary Torsion of the Greater Omentum

Primary or idiopathic torsion of the greater omentum (TGO) is an uncommon cause of acute abdominal pain in children. A 12-year-old boy presented with abdominal pain for 24 hours, anorexia, nausea and low-grade fever. The pain was localized in the right lower quadrant of the abdomen. Physical examination revealed abdominal tenderness and muscle guarding in the right abdomen. The leukocyte count was normal with a left shift (WBC: 7800/mm^3, neutrophils 92%). The hematocrit was 37.4% and platelets count 2,26,000/μL. Ultrasonographic imaging was obtained but the findings were non-specific (small amount of fluid in the space of Douglas).

We suspected acute appendicitis, so emergency laparotomy was performed by Lanz incision, during which the presence of free intra-abdominal serosanguinous fluid was noted. The appendix had a normal appearance. Torsion was observed in the greater omentum, with necrosis of its distal part. This part was excised. Appendectomy was also performed. Pathology revealed an acute hemorrhagic infarct and fat necrosis. The boy recovered without any complications.

Primary omental torsion was first reported by Eitel in 1899 , and since then, fewer than 250 cases have been reported, mostly in adults. It has been estimated that 0.05-0.1% of children undergoing laparotomy for suspected appendicitis have primary omental torsion(1,3). TGO is a rare cause of abdominal pain in children and it has no distinguishing features to separate it from other causes of surgical abdomen, especially from acute appendicitis. It usually affects children older than 3 years of age, due to the increase in omental fat deposition as the child grows(4).

Torsion may be primary or secondary. In primary torsion, a mobile segment of omentum rotates around a proximal fixed point in the absence of any associated intra-abdominal pathology. If the mass is not excised it becomes atrophic and fibrotic and, on rare occasions, the pedicle may even auto-amputate, leading to automatic clinical regression(3). Secondary torsion occurs in the presence of lesions such as cysts, hernias, tumors and adhesions or congenital attachments and is believed to be more common than primary TGO(5).

The presentation of primary TGO seems to be less acute compared to other causes of surgical abdomen. The diagnosis is usually made at laparotomy or by laparoscopy. Treatment consists of resection of the affected portion of omentum. Appendectomy is performed at the same time, as it minimizes postoperative confusion and future diagnostic dilemmas. No postoperative recurrences are reported.

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Treatment Dilemma in Osteopetrorickets

A 1½-year-old boy who is followed up in our Nutrition and Growth Clinic was diagnosed to have co-existent osteopetrosis and rickets. At the time of diagnosis, he was seven months old and had anthropometric parameters below the 3rd centile of the NCHS reference standards except head circumference, which was between the 25th and the 50th centile. Clinical examination revealed pallor, hepato-splenomegaly, early optic atrophy and features of rickets like caput quadratum, wide open anterior and posterior, fontanels, Harrison's sulcus, rachitic rosaries and widening of wrists and ankles. Laboratory data showed hemoglobin 7.5 g/dL, total leukocyte count of 11,200/cumm, polymorphs 52%, lymphocytes 30% and eosinophils 18%, platelet count 1 lakh/cumm and leuko-erythroblastic blood picture with mild thrombocytopenia. He had normal thyroid function and normal karyotyping. The serum calcium (7.2 mg/dL) and phosphorus (1.4 mg/dL) levels were low and alkaline phosphatase (2005 IU/L) was markedly raised. Serum sodium, potassium, bicarbonate and urine pH were normal and associated renal tubular acidosis was excluded. Urine was negative for mucopolysaccharides and aminoaciduria. The liver and renal function tests were within the normal range. Skeletal survey revealed features of osteopetrosis, rickets and pathological fractures with 'bone in bone' appearance, increase in medullary density, defective metaphyseal remodeling with fraying and early cupping and widening of 'physis', the growth plate (*Fig.1*). US$ showed hepatosplenomegaly and CT scan head showed osteosclerosis and diffuse brain atrophy.

Osteopetrorickets is a rare association of osteopetrosis with rickets(1,2). In osteopetrosis, there is excess calcium reserve and hypophosphatemia(3), but when it is complicated by rickets there is an insufficient Ca:P product to mineralize the growing bone. In coexistent osteopetrosis and rickets, 99% of the calcium is shut off in the bone and is not

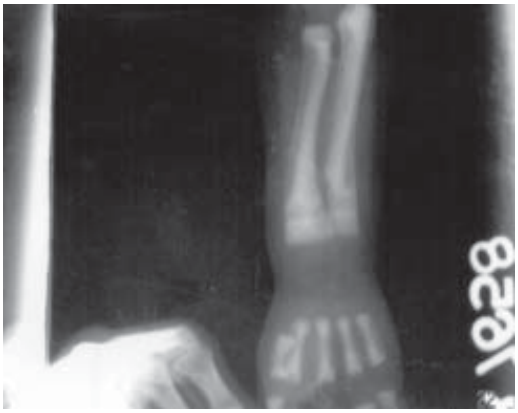

Fig. 1. X-ray of left forearm and hand showing osteosclerosis, 'bone in bone' appearance of shaft, fracture both bones, fraying and early cupping of metaphysis and widening of 'physis', the growth plate and absence of carpal bones.