Primary Tuberculosis of Mandible

A 9-year-old male child presented with a gradually increasing swelling of left cheek for 3 months associated with pain for initial few days. There was no history of fever, anorexia, any discharge from swelling, difficulty in chewing or swallowing. Child had received multiple courses of oral antimicrobials. Family history revealed possible contact with tuberculosis. Child had received BCG vaccine in infancy. Anthropometry, general physical and systemic examinations were unremarkable. On local examination, the swelling was diffuse, nontender, and hard in consistency with smooth surface. There was no restriction of movements at temporomandibular joint. There was significant submandibular lymphadenopathy.

Routine laboratory tests were grossly unremarkable. Radiograph of mandible revealed expansile osteolytic lesion involving angle of left mandible while the chest *X*-ray done to look for any evidence of primary focus, was normal. Gastric aspirates for acid-fast bacilli (AFB) and tuberculin skin test were negative. Non-contrast CT scan of mandible (*Fig.1*) revealed destruction of inner as well as outer wall of the angle of left mandible due to expansile osteolytic mass lesion. Osteoblastic activity was also seen as new bone formation around the lytic lesion. Breach in periosteum with extension of expansile lesion into surrounding soft tissue was also noted. CT findings were reported as malignant bone tumor. Fine needle aspiration smear from the swelling showed necrotizing granulomatous inflammation consistent with tuberculosis but Ziehl Neelsen staining for AFB was negative. Standard antitubercular therapy for bone tuberculosis was started. At 2 weeks follow-up some reduction in size of swelling was noticed and swelling completely disappeared at 2 months follow-up.

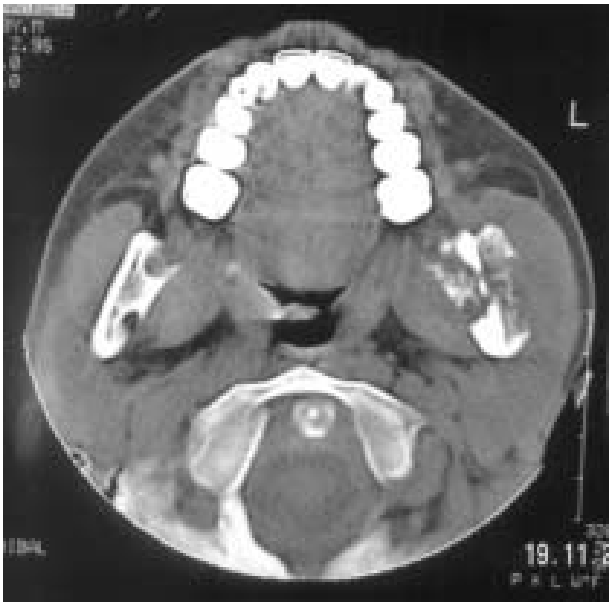

Fig. 1. CT scan of mandible taken prior to start of therapy.

Bone tuberculosis is a relatively uncommon form of extrapulmonary tuberculosis seen in approximately 1% of children with tuberculosis(1). It more frequently seen in children as compared to adults because epiphyseal region of the bones is highly vascularized in infants and young children. Most reported cases of mandibular tuberculosis were secondary to tuberculous focus elsewhere in the body. To best of our knowledge, only 4 cases of primary mandibular tuberculosis have been reported(2-4). Three routes of infection to the mandibular bone are postulated. One is direct transfer of infected material through a carious tooth, a post extraction socket or mucosal wound. Other two are direct extension from local soft tissue lesion to the underlying bone and hematogenous route. In our patient, a slight wound in the oral cavity or gingivitis might be the entry site, though there was no history of extraction of carious tooth or trauma. A similar route of entry had been postulated in another reported case(4).

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Acute Renal Impairment after Oral Ibuprofen for Medical Closure of Patent Ductus Arteriosus

Patent ductus arteriosus (PDA) is a common occurance in preterm infants with significant morbidities. Indomethacin, has been widely used to treat hemodynamically significant PDA. However, this agent can cause adverse reactions such as reduced renal, mesenteric, and cerebral perfusion leading to transient or permanent renal dysfunction, necrotizing enterocolitis, gastrointestinal hemorrhage, and intraventricular hemorrhage or periventricular leukomalacia(1). Research has shown that ibuprofen is as effective as indomethacin for achieving PDA closure with minimal adverse effects(2-3). However, before giving treatment an accurate echocardiography should rule out ductus dependent cardiac malformations, because ibuprofen/indomethacin can be dangerous and highly fatal in these conditions. This paper reports a preterm infant who was treated with oral ibuprofen and developed acute transient renal failure.

Case Report

An 880 g female infant was born at 29 weeks' gestation. At birth, the baby showed signs of RDS, and she required intubation and administration of exogenous surfactant. On day 7 of life, hemodynamically significant PDA was diagnosed by echocardiography. A course of ibuprofen starting at a dose of 10 mg/kg and followed by two doses of 5 mg/kg at 24 h and 48 h later was ordered. On day 3 of treatment, the infant's renal function deteriorated: blood urea nitrogen, 92 mg/dL; creatinine, 2.0 mg/dL; creatinine clearance, 5.1 mL/min/1.73 m^2. Fluid retention (> 5% actual weight), oliguria (0.5 mL/kg per h) and hyponatremia (113 mEq/L) were detected. Ultrasonographic examination of the renal system revealed no abnormalities. Three days later, the baby recovered full renal function.

Discussion

Reports have shown that infants with PDA who receive intravenous ibuprofen exhibit a less pronounced rise in serum creatinine and are less likely to develop oliguria than those who receive intravenous indomethacin(2-3). In Cochrane database review, they conclude that indomethacin should remain the drug of choice for the treatment of a PDA, because ibuprofen may increase the risk for chronic lung disease, and pulmonary hypertension has been observed in three infants(3).

A recent work on newborn rabbits has revealed that intravenous ibuprofen causes a dose-dependent significant reductions in urine volume, glomerular filtration rate, and renal blood flow, together with an increase in renal vascular resistance and a decrease in urinary sodium excretion(4). Ibuprofen may also cause tubulointerstitial nephritis.

Currently, recommended dose regimen of ibuprofen is 10-5-5 mg/kg at 24-h intervals. A recent study suggested that higher dosing (20-10-10) of ibuprofen may result in higher rates of closure, however, with increased risk of oliguria and renal complications(5).

It is important to underline that premature babies with immature organ systems are more susceptible to pharmacological interactions and this baby was also receiving aminoglycosides. Concomitant drugs influencing renal function would have an additive effect in renal impairment.

In conclusion, it is important that pediatricians