combined with the nibbling effect of the head of the worm can lead to perforation of the normally impenetrable bowel wall[8].
Intraperitoneal tumoral ascariasis results when the perforation is sealed spontaneously. This situation may be self limiting without recourse to emergency surgery. Destruction of the larvae and the adult worm is usual, but eggs are resistant and result in a specific granulomatous reaction. The presence of viable ascaris eggs suggest that they were deposited on site prior to female adult worm destruction[9].
Ascaris perforation may cause acute diffuse peritonitis, usual symptoms in these patients are abdominal pain and vomiting. Abdominal pain is present in almost all cases with vomiting found in 80% cases and fever in 16% cases. 70% cases usually have typical signs of peritoneal irritation, including tenderness, rebound pain and guarding[10].
It is suggested that signs of infestation with ascariasis lumbricoides make a diagnosis of intestinal perforation more likely in a patient with an acute abdomen. Ascaris has a propensity to migrate from its usual habitat, duodenum, to other areas. It is because of tendency of the adult worm to migrate that even a single worm can cause serious sequelae. Wandering worms may move to any organ of the gastrointestinal system including liver, biliary tract, gall bladder, pancreatic duct, appendix, or to the peritoneal cavity. They may come out of the anus, mouth or nose. The worm may move to the peritoneal cavity through intestinal ulceration or may itself perforate the intestine. The female worm lays eggs which produces a granulomatous inflammation, and itself dies leading to a large abscess, which presents as a tumour like mass in the abdomen, peritonitis associated has a high morbidity and mortality. Surgical intervention is the treatment of choice.

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Case Report

Radiological Features in Actinomycosis of Paranasal Sinus region and Base of Skull with Oro-antral fistula.

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Abstract*: Actinomycosis of paranasal sinus region occurs rarely. It's a clinical and diagnostic dilemma associated with significant morbidity. If untreated it can spread to the base of skull and lead to fistula formation. Computed tomography scan (CT) can reveal the type and extent of disease but the definitive diagnosis is by demonstration of actinomyces on histopathology. The authors report a rare case of actinomycosis of the paranasal sinus region spreading to the base of skull with formation of oroantral fistula; CT scan findings, differential diagnosis and review of literature, has been discussed.* ***Key words:*** *Actinomycosis, paranasal sinus, oroantral, fistula*

INTRODUCTION

Actinomycosis is caused by actinomyces israelii, a commensal bacteria harboring human oral cavity usually around teeth and tonsillar crypts. Its pathologic potential is minimal in normal individuals but is enhanced by trauma resulting in disruption of mucous membranes[1,2]. Out of the three forms; cervicofacial, thoracic and abdominal, the cervicofacial type occurring in form of soft tissue abscess and draining cervical fistulae is the commonest. Sinonasal, laryngeal and pharyngeal disease due to actinomycosis is rarely encountered[1,3]. Actinomycosis involving the base of skull has not been described in the medical literature.

CASE REPORT

A forty years old female with complaint of foul breath and purulent discharge in the oral cavity since 3-4 months visited our department for computed tomography (CT) scan of the nasal and paranasal sinus region. Patient gave the history of tooth extraction in the molar region of maxilla on left side 8-9 months back. Local examination of oral cavity revealed periodontal disease, multiple carious tooth and presence of communication between the oral & nasal cavity on left side with purulent discharge. No evidence of any external abnormality was noted in the facial region. Laboratory examination was unremarkable. Chest radiograph and ultrasonography of abdomen

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was unremarkable.

CT scan of the paranasal sinus region (Figures 1a & 1b) revealed variable degree of osseous destruction & resorption involving left antrum except the posterolateral wall; nasal septum; left lateral nasal wall; maxilla including its alveolar process, hard palate, body and greater & lesser wings of sphenoid with predominant involvement on the left side. Additionally noted was partial destruction of petrous apex on left side, oroantral fistula on left side and multiple sequestra especially on the left side. There was partial destruction of osteomeatal unit complex on right side as well. Mucosal thickening was noted in the bilateral inferior turbinate and right antrum along with partial opacification of the ethmoid air sinus complex on both sides. There was no evidence of any obvious mass or sclerosis / reactive bone formation except in the posterolateral wall of left antrum.

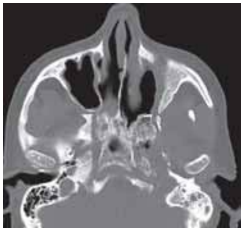

Figure 1a: *Transaxial CT image shows osteolytic destruction of medial and anterior wall of left antrum, nasal septum, sphenoid bone and petrous apex on left side along with opacification of mastoid air cells of left side*

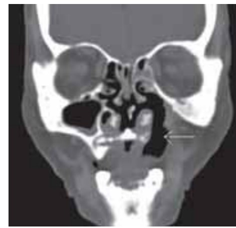

Figure 1b: *Transcoronal CT image shows osteolytic destruction of medial and lateral wall of left antrum, nasal septum and hard palate with formation of oroantral fistula on left side (arrow) along with partial opacification of ethmoid sinuses and mucosal thickening in the right antrum*

Based on the clinicoradiologic findings, the diagnosis of low grade / granulomatous chronic osteomyelitis involving nasal cavity, paranasal sinuses, hard palate and base of skull along with formation of oroantral fistula on left side was suggested. Biopsy of bone was suggested for further work up.

Histopathology of the biopsied bone fragment revealed areas of necrosed bone and degeneration of connective tissue stroma with chronic inflammatory cell infiltrate (figure 2). In addition, colonies of microorganisms with branching filaments were also noted. Modified acid fast staining ruled out Nocardia. Hence, the histopathological diagnosis of chronic osteomyelitis with actinomycosis as an etiologic agent was made.

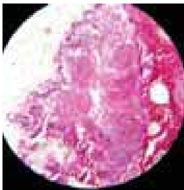

Figure 2: *H & E stained slide (high power) shows presence of colonies of microorganisms with branching filaments (sulfur granules).*

DISCUSSION

Actinomycosis is an uncommon chronic suppurative infection caused by actinomyces that are normal commensals of human oral cavity and pharyngeal region[1]. In this era of antibiotics, they rarely assume parasitic and pathologic role resulting in significant morbidity. Poor oral hygiene, oral disease and oral trauma are the predisposing factors for actinomycosis[4,5]. The disease is commoner in males and in middle aged and elderly, but uncommon in children, probably reflecting the higher incidence of periodontal and dental disease in higher age groups[4,6]. The disease is mainly seen in the tropics including Asia and Africa[6].

Actinomyces are slow growing, gram positive, anaerobic, non-acid fast, filamentous bacilli producing typical yellowish sulfur granules. They spread locally rather than by hematogenous or lymphatic route[5].

Cervicofacial region involvement is commonest. Ingestion of organisms results in abdominal involvement, while tracheobronchial aspiration results in thoracic involvement. Rarely, pelvic involvement may be associated with use of IUCD[4].

Irrespective of the region of involvement, the commonest feature is formation of phlegmon with increasing induration and fibrosis and resultant decrease in vascularity over several weeks followed by formation of discharging sinuses. But acute form with rapid suppuration is also seen[1]. The commonest site is cheeks, angle of jaw or submandibular region. The infection may spread contiguously to the adjacent areas disrupting fascial planes. Oral infection may further involve pharynx, larynx, salivary glands, tonsils and paranasal sinuses. Involvement of paranasal sinuses is rare and only few cases have been reported[1]. Involvement may occur in the form of pseudotumors causing sinus opacification; mucosal thickening and destruction of bony walls especially the medial wall. Destruction of posterior wall of antrum favors a malignant pathology[1]. Presence of hyperdense hyphae and calcification seen characteristically in fungal infection is absent in actinomycosis. Rarely actinomycosis can appear as an enhancing mass on CT scan making it difficult to differentiate from masses[7]. There may be formation of fistulas[4].

Primary actinomycosis of bone is rare, as seen in our case and is the result of adjacent soft tissue involvement. Unlike our case, where maxilla was involved, involvement of mandible is commoner by four times[4]. Osteolytic destruction ranging from minimal rarefaction or periosteal reaction to extensive osteomyelitis with sclerosis and periosteal reaction is the predominant radiologic manifestation. Sometimes, the appearance of osseous tumor may be simulated[4]. Besides, there may be formation of interosseous sinus tracts. In our case, extensive and contiguous osteolysis of bones of face & base of skull with formation of multiple sequestra was a predominant radiologic finding along with oroantral fistula and absence of any significant sclerosis or periosteal reaction except in posterolateral wall of left antrum, making it probably first instance of its type in medical literature.

Diagnosis of actinomycosis is based on the demonstration of sulphur granules in exudates, culture or histopathology[1]. Sulfur granules are, in fact aggregates of filaments of bacteria.

Radical excision of the sinus tracts, wherever possible along with heavy and prolonged doses (3-12 months) of penicillin is the treatment of choice[1,4]. Oroantral fistula can be closed successively by buccal flap advancement.

To summarize, primary actinomycosis of bone involving paranasal sinuses and base of skull is rare and is best evaluated by computed tomography which not only determines the extent of involvement but also helps to narrow the differential diagnosis and in deciding the site of biopsy. Actinomycosis should be suspected if there is extensive osseous destruction especially involving the medial wall and preserved posterior wall with absence of hyperdense hyphae.

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