Case Report

TUBERCULOUS OSTEOMYELITIS OF THE BONE FLAP FOLLOWING CRANIOTOMY

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Summary: A patient of tuberculous osteomyelitis of the bone flap following craniotomy for acute subdural hemorrhage which was treated at Surat Municipal Institute of Medical Education & Research (SMIMER) from June 2010 has been reported. This report emphasizes the fact that while treating osteomyelitis of bone flap following craniotomy, possibility of tuberculosis should be considered, especially in our country. Treatment wise, the disease responded readily to routine anti-tubercular chemotherapy. ***[Indian J Tuberc 2011;58: 129-131]***

***Key words*:** Tuberculous osteomyelitis, Skull, Craniotomy

INTRODUCTION

Tuberculous osteomyelitis of the skull is a rare manifestation of extra-pulmonary disease[1,2]. Skeletal tuberculosis accounts for 1-3 % of all cases of tuberculosis[3] and calvarial involvement is seen only in 0.2-1.3 % of patients with skeletal TB[1].

CASE REPORT

A 20-year-old male patient with alleged history of road accident was admitted in unconscious state with history of vomiting and nasal bleeding on 28th March 2010. On examination, vitals were normal with GC Score E1V1M5. On neurological examination, there was swelling of scalp on left side and hemiparesis on right side.

Routine haematological & biochemical investigations were normal. The chest X-ray was also normal. Computerized Tomography (CT) of brain revealed acute Subdural Hemorrhage (SDH) in left fronto-temporoparietal region with significant mass effect midline shift (Figure 1). The patient was immediately subjected to left fronto-temporoparietal craniotomy and evacuation of acute SDH. The post-operative period was uneventful with no neurological deficit and patient improved well.

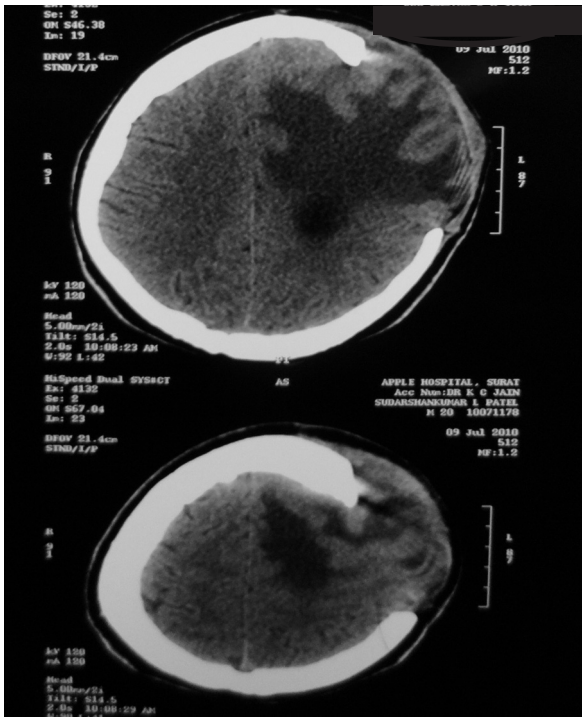

Figure 1: Post head-injury left parietal craniotomy status with scalp edema. Mild subdural haemorrhage along with falx-cerebri and tentorium cerebelli is observed

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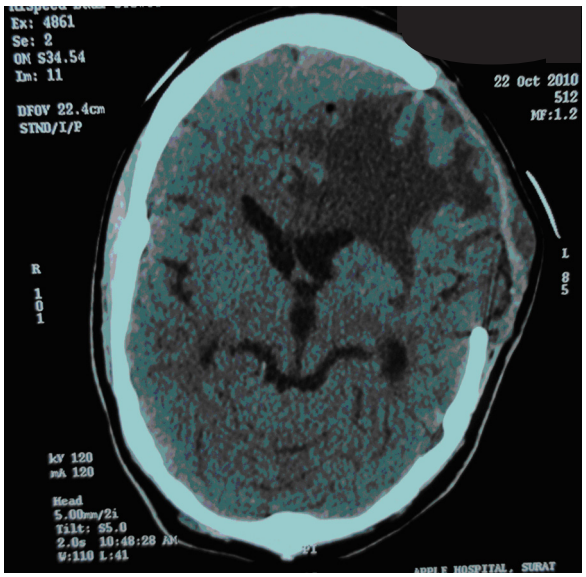

Figure 2: Development of Tuberculous Osteomyelitis in the bone flap and nereby followed by removal of the bone flap

On 3rd June 2010, approximately two months after surgery, though the patient improved with routine treatment, there was purulent discharge from the operative wound which did not subside with regular dressings and antibiotics (cefaperazone-sulbactum and netilimicin). The antibiotic therapy was based upon the culture and sensitivity testing in which Klebsiella species was reported. On examination, a sinus discharging pus was present in the surgical wound. The neurological examination was normal except diplopia.

The swelling over the local area continuously increased; hence the patient was readmitted on 19th June 2010 for debridement of the wound. During debridement, a portion of bone flap was also removed. The debrided material was sent for histopathological examination and the pus was sent for routine, fungal and AFB culture and sensitivity testing. The biopsy report was suggestive of tuberculous inflammation. The routine and fungal cultures showed no growth. The smears and culture were positive for AFB, sensitive to all the primary line of anti-tuberculous drugs. Repeat CT scan of brain was done on 28th June 2010 and showed large parenchymal hypodense area with edema in left fronto-parietal region with scalp edema and mild bony destruction with thinning of superior border of left squamous temporal bone (possibility of infective aetiology likely was reported). The patient was started on anti-tuberculous treatment as per RNTCP guidelines from the same day.

On follow up, two weeks later, in view of persisting pus discharge, another CT scan of brain was done which showed marked reduction in scalp edema and hypodense collection with mild reduction of parenchymal edema. Routine culture and sensitivity of the pus was repeated and was reported to be sterile for pyogenic organism.

Six weeks later, on 19th September 2010, the patient was readmitted with history of one episode of generalized convulsion, nausea and vomiting. During course of hospitalization, patient was treated conservatively to which the patient responded satisfactorily (Figure 2).

As on 30th November 2010, the anti-tuberculous therapy was being continued and on local examination of the wound, discharge of pus was absent with the healing of the wound almost complete. Overal, the patient was doing well, except for the history of occasional convulsions along with mild diplopia for which he was being treated conservatively.

DISCUSSION

Tuberculosis of the skull is a rare entity with occurrence of 1 in 10,000 cases of tuberculosis[3]. The majority of these cases occur at an early age with three-fourth of the patients below 20 years of age and 50% being less than 10 years of age[4]. There is no sex predilection and both sexes are almost equally affected.

In common with tuberculosis of bones and joints, lesions in the skull are almost never primary, unless there has been direct inoculation of the bone by a penetrating injury; in almost all cases, a primary lesion elsewhere in the body most commonly in the lung can be shown. Skull lesions are seen more

commonly in the fronto-parietal region than in occipito-temporal region, the ratio being five to one[5].

The tuberculous focus in the skull starts in the diploe and may erode either one or both tables of the skull, giving a clear punched out appearance on skull x ray. When the response to the infection is good, the lesion develops slowly. Wide extension of tuberculous granulation tissue through the diploe is prevented by the proliferation of and encircling layer of concentrically placed fibroblasts and if the process is not arrested, extension then takes place through either tables. If the outer table is destroyed, a fluctuating swelling of scalp develops and subsequently the skin breaks down with the formation of sinus, discharging tubercular pus. When the tissue response is poor, the infection spreads more rapidly through the dipole. The sutures form no barrier to the advance, and perforation of either tables may occur at several points. An extensive area of destruction occurs before a sinus or a fluctuating swelling appears. If the process is rapid, sequestration may occur which can take the form of so called bone sand in the punched out lesions. The dura matter forms an excellent protective barrier to the spread of the brain and meninges.

Two possible routes for origin of the infection have been suggested. Trauma has been suggested as playing a role in the genesis of this disease. Trauma by increasing the vascularity may help in localizing the lesion, to a particular part of the skull. Paucity of early symptoms is a major feature of this condition. Appearance of a fluctuating swelling of the scalp is usually the first evidence of the disease.

Treatment-wise, the disease responds very well to the usual anti-tubercular chemotherapy in the early stages. In later stages, with sequestration of bone and extensive caseation, surgical removal of all diseased tissues is essential.

CONCLUSION

It is important to consider tuberculosis as a cause of post-operative osteyomyelitis, as its treatment is quite distinct from pyogenic osteomyelitis which is the commonest cause of post-operative osteomyelitis. Hence, all bone flaps which are removed for suspected osteomyelitis should be sent for histopathological examination and for AFB and fungal cultures, in addition to routine cultures.

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