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Case Report

PRIMARY NASAL TUBERCULOSIS PRESENTING AS SEPTAL PERFORATION: A CASE REPORT

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ABSTRACT
Second half of last century witnessed a decline in the incidence of upper respiratory tract tuberculosis, thanks to the advent of highly effective chemotherapeutic agents. Primary nasal tuberculosis is an extremely rare condition in modern antibiotic era. Most of the cases of nasal tuberculosis are attributed to contagious, hematogenous or lymphatic spread from a primary site (most often lungs). However, nose may sometimes be primarily affected by tuberculosis once it's self protective mechanisms are lost. We are reporting the case of a 10 years old boy who presented in the department of TB & Chest Diseases S. N. Medical College Agra with nasal obstruction, crusting, recurrent episodes of epistaxis and disfiguration of nose. Diagnosis of nasal tuberculosis was made on the basis of CT scan findings and histopathological examination. An excellent response was noted after commencing anti tubercular treatment.

Keywords: *Nasal Tuberculosis, Septal Perforation, Extra Pulmonary Tuberculosis*

INTRODUCTION
Giovanni Morgagni first described nasal tuberculosis in 1761. His descriptions were actually based upon autopsy findings of a young man (Waldman *et al.,* 1981). In 1852 Clarke presented the first case of primary tuberculosis of upper respiratory tract and nose to pathological society of London. Tuberculosis has now emerged as the most important communicable disease in the world and particularly in developing countries like India. Advent of HIV infection has substantially boosted the incidence of tuberculosis. However, primary nasal tuberculosis is still a rarity even in HIV positive patients. This can be easily explained by the ciliary action of nasal mucosa, the bactericidal action of nasal secretion and the protective mechanisms of nasal vibrissae which combindly serve to guard the nasal mucosa against any infection (Kadambari *et al.,* 2002).

CASES
A 10 years old boy belonging to rural area of Mathura district, Uttar Pradesh, presented in department of TB & Chest Diseases, S. N. Medical College, Agra with chief complaints of nasal obstruction, crusting, recurrent nasal bleeding episodes for last 8 months and progressive disfiguration of nose (Figure 1) for last 4 months.
He had no history of headache, excessive sneezing, ear discharge, any throat complaint or any visual disturbance. There was no history of dyspnea, chronic cough, hemoptysis or loss of weight. History of BCG vaccination was present.
Patient had consulted many general practitioners in previous few months and he had been repeatedly and ineffectively put on broad spectrum antibiotics, antihistaminic and topical applications. On clinical examination, patient was afebrile and his general condition was fair. His pulse rate was 82 per minute, respiratory rate 17 per minute, blood pressure was 112/74 mm of Hg and SpO_2 was 99%. Local examination of nose revealed destruction in antero-inferior aspect of cartilaginous part of nasal septum. Bony nasal septum appeared intact.
No other significant findings were noted on general, physical and systemic examination of patient including no evidence of any lymphadenopathy.

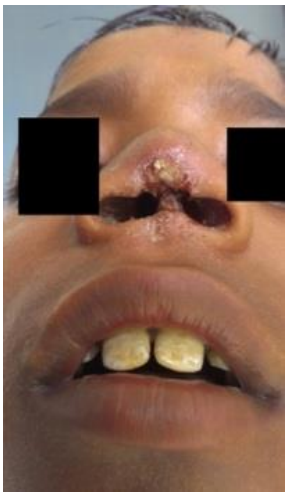


Figure 1: Showing nasal septal destruction

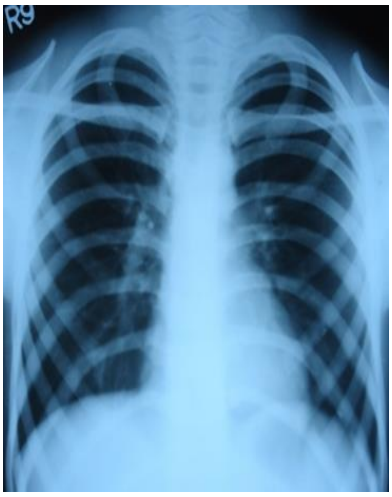


Figure 2: Chest Xray PA view showing no obvious pleuro-parenchymal disease

Patient's routine investigations including CBC, LFT, KFT, RBS, and urine analysis were with in normal limits, except for ESR which was 22 mm in first hour. Patient was HIV 1 & 2 nonreactive and VDRL seronegative. The tuberculin skin test revealed an induration diameter of 20 mm after 72 hours. No pleuro-parrenchymal disease was detected on patient's Chest X-Ray PA view (Figure 2).
Patient's sputum was subjected to staining and culture and it was found to be negative for acid fast bacilli.

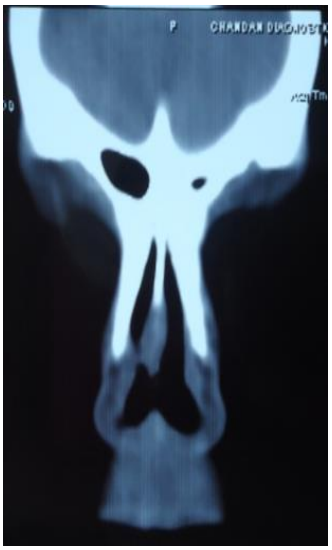


Figure 3: Coronal and axial scan showing destruction in anterio-inferior aspect of nasal septum (cartilaginous part) with soft tissue thickening in tip of nose

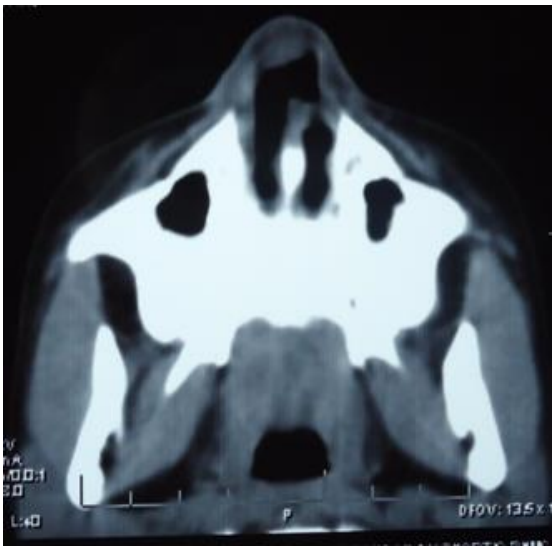
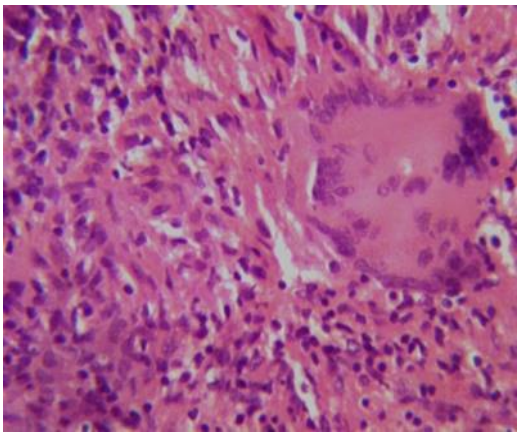


Figure 4: Coronal and axial scan showing destruction in anterio-inferior aspect of nasal septum (cartilaginous part) with soft tissue thickening in tip of nose

Helical axial and coronal computerized tomographic scans of PNS were obtained, which showed destruction in antero-inferior aspect of cartilaginous part of nasal septum with soft tissue thickening in tip of nose (Figure 3&4), likely suggestive of tubercular etiology. Absence of bony involvement was confirmed on CT scan. Intranasal biopsy was taken from the margins of septal perforation and sent for histopathological examination. Histopathology showed granulomas along with Langhans type multinucleated giant cells and mixed inflammation (Figure 5&6).



Fgure 5: Picture shows langhans type giant cell along with epithiloid cells and lymphocytes (H&E stain, 400X)

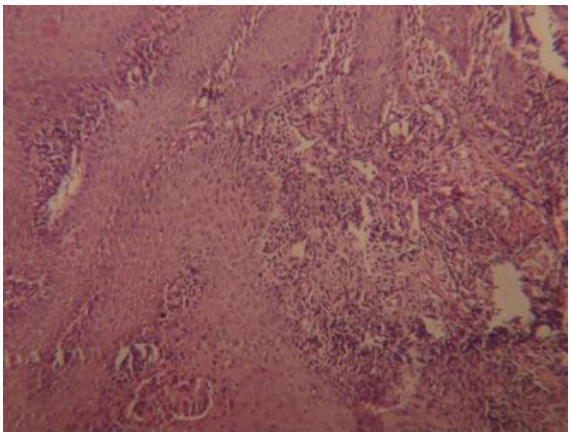


Figure 6: Picture shows hyperplaastic squamous epithelium with underlying chronic inflammation (H&E stain, 100X)

Nasal scrapings were sent for AFB culture and were subsequently found to be negative for AFB. Fungal smear and culture were negative. ANCA (anti neutrophlic cytoplasmic antibody) was also negative. In the view of above mentioned investigations a presumptive diagnosis of ulcerative form of primary nasal tuberculosis was made and anti tubercular treatment was planned.

Patient was put on a 2 months course of rifampicin (10 mg/kg) , isoniazid (5 mg/kg), ethambutol (15mg/kg) and pyrazina Figure 3: Coronal and axial scan showing destruction in anterio-inferior aspect of nasal septum (cartilaginous part) with soft tissue thickening in tip of nose.mide (25mg/kg) followed by a continuation therapy of rifampicin (10mg/kg) and isoniazid (5mg/kg) for next 4 months. Patient progressively improved during the treatment (Figure 7) and became totally asymptomatic after a total 6 months of anti-tuberculosis treatment, however the patient's attendant refused for any surgical correction of residual cosmetic abnormality.

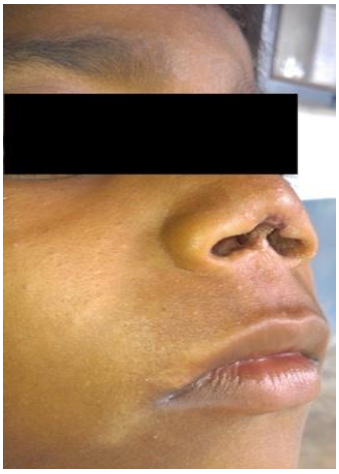


Figure 7: Same patient -1 month after continuing anti tubercular treatment showing considerable improvement

DISCUSSION

In more than 75% cases, tubercular involvement of nose is a manifestation of generalized disease (Waldman *et al.,* 1981). Most of the reported cases till date have occurred in females and elderly (Friedmann, 1971). The disease is very rarely infectious and bacterial counts in lesions are typically much lower than a cavitatory lesion of pulmonary tuberculosis (Goguen and Karmody, 1995), this is the reason

why most of the times these lesions do not show a direct evidence of Acid Fast Bacilli in smear or culture. Nasal tuberculosis can occur either by hematogenous or lymphatic route from a distant site or rarely by direct inoculation of acid fast bacilli. Most common site of inoculation of bacilli is mucocutaneous junction of nasal septum because this is frequently exposed to trauma in persons who have the habit of nose picking.

Three forms of nasal tuberculosis have been recognized. Nodular and ulcerative forms are seen in nose while 3rd form sinus granuloma is seen in paranasal sinuses (Bahadur and Thakar, 2008). Nodular form presents with reddish brown papules or nodules also called as apple jelly nodules. Ulcerative form usually involves cartilaginous part of nasal septum or inferior turbinate and presents with crusting, obstruction and recurrent epistaxis. The lesion may progress to perforation of nasal septum as it occurred in this case. A differentiating feature from syphilis is lack of bony septum involvement in nasal tuberculosis.

In all forms, nasal obstruction is the most common presenting symptom which may be the result of formation of foul smelling crusts. Local spread of disease may also occur through mucocutaneous junction to involve the skin of nose and face. Lacrimal sac involvement can also occur if the disease spreads through lacrimal duct. When internal nose is involved, it may lead to atrophic rhinitis.

Differential diagnosis of nasal tuberculosis include wegener's granulomatosis, sarcoidosis, leprosy, fungal disease, discoid LE, pseudolymphoma, tertiary syphilis, lupoid leishmaniasis and chronic pyodermas (Brown *et al.*, 1982).

The definitive diagnosis of nasal tuberculosis can be made by isolation of Mycobacterium tuberculosis from culture. However a negative AFB culture can not exclude the diagnosis of tuberculosis considering a much lower bacterial load in these sort of lesions as compared to that in a cavitatory pulmonary tuberculosis (Goguen and Karmody, 1995). A combination of histopathological findings (caseation, well defined granulomas), favorable CT findings, a successful therapeutic trial with ATT and failure to detect any other tubercular lesion in body can very well establish the diagnosis of primary nasal tuberculosis. A chest X-ray PA view should always be ordered to look for any primary disease irrespective of absence of any chest complaint. A negative tuberculin test makes the diagnosis of tuberculosis less likely in immunocompetent patients. HIV status of patient must be clearly established because atypical presentation of tuberculosis is more common in HIV infected people. Laboratory work up should also include VDRL, CBC, ESR, LFT, KFT, RBS and urinalysis.

Treatment should consist of an intensive phase of 2 months with at least 4 drugs (rifampicin, isoniazid, ethambutol and streptomycin) which should be followed by a continuation phase of 4 months with rifampicin and isoniazid.

Conclusion

In conclusion, it is worthwhile that clinician should remain cognizant for the rare but possible tubercular involvement of head and neck area, as prompt diagnosis and early commencement of treatment can not only save the patient from any permanent disability but also it can save the patient from any cosmetic abnormality.

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