CASE REPORT

Primary nasal tuberculosis complicated by mrsa co-colonization and septal perforation: A rare case report

Sonny Soebjanto, MD, ORLHNS, Shod Abdurrachman Dzulkarnain, MD, M.Biomed

Faculty of Medicine, Universitas Negeri Surabaya, Surabaya, East Java, Indonesia

SUMMARY

Primary nasal tuberculosis is an exceptionally rare extrapulmonary manifestation, usually arising after trauma or direct inoculation and presenting with chronic nasal obstruction, rhinorrhea, epistaxis, anosmia, or intranasal masses, frequently complicated by septal perforation. This report describes a 58-year-old woman with bilateral obstruction, anosmia, and rhinorrhea, in whom endoscopy revealed a friable nasal mass with anterior septal perforation. Pulmonary tuberculosis was excluded by negative sputum AFB, GeneXpert, and chest radiography. Nasal biopsy demonstrated caseating granulomas with Langhans giant cells, confirming primary nasal tuberculosis without pulmonary involvement. Concurrent MRSA colonization was identified. Management consisted of standard first-line anti-tuberculosis therapy with conservative septal care and targeted MRSA eradication. Follow-up showed improvement and clearance.

INTRODUCTION

Primary nasal tuberculosis (TB) is exceedingly rare, with fewer than a few dozen cases reported in the modern literature. The septum, particularly the cartilaginous portion, is a common site for TB involvement because of its vulnerability to trauma and infection. Caseous necrosis associated with TB granulomas can destroy cartilage and mucosa, leading to anterior septal perforation.[1] Primary nasal TB mimics fungal or malignant sinonasal disease but is distinguished by caseating granulomas and response to anti-TB therapy.[2] Nasal TB involving nasal septum perforation requires anti-TB treatment followed by surgical intervention, such as debridement or endoscopic sinus surgery.[3]

A further challenge in patients with nasal TB is the risk of secondary bacterial infections with multidrug-resistant bacteria. Resistant bacteria, such as methicillin-resistant *Staphylococcus aureus* (MRSA), have emerged as major global pathogens. MRSA infections are associated with higher morbidity and require targeted eradication therapy combining systemic and topical antimicrobials.[4]

CASE PRESENTATION

A 58-year-old woman presented with bilateral nasal obstruction for one month, accompanied by complete anosmia. She reported thick rhinorrhea that was initially white, occasionally turning green, and sometimes mixed with blood, along with a nasal voice and post-nasal drip. She denied recurrent epistaxis, foul nasal odor, facial pain, or headaches. Systemic symptoms such as cough, hemoptysis, fever, night sweats, weight loss, and anorexia were absent. No auditory or vestibular symptoms were noted, including otalgia, hearing loss, tinnitus, or otorrhea. There were no throat-related complaints such as dysphagia, odynophagia, foreign body sensation, or hoarseness. She had no history of diabetes, hypertension, or prior pulmonary TB. Family history was notable for malignancies (mother with a uterine tumor, sibling with a lymph node tumor). Social history revealed a neighbor with a chronic cough and a husband who smoked 2-3 packs of cigarettes daily. She reported no allergies.

On examination, her general condition was fair, and she was alert. Vital signs were within normal limits: blood pressure 130/80 mmHg, pulse 80/min, respiratory rate 20/min, and body temperature 36.8 °C. There were no signs of respiratory distress such as stridor or chest retractions. Cardiopulmonary and abdominal examinations were unremarkable. No systemic lymphadenopathy was initially detected; however, during the course of illness, a firm, mobile, non-tender 2 cm mass was palpated in the left supraclavicular region, later interpreted as lipofibroma versus chronic lymphadenitis on fine needle aspiration biopsy (FNAB).

Ear, nose, and throat (ENT) evaluation revealed normal ears, with intact tympanic membranes and preserved light reflexes. Rhinoscopy and subsequent endoscopic assessments consistently showed a friable mass covered with greenish crusts and an anterior septal perforation. Post-nasal drip was observed. The tonsils were small (T1-T1) without erythema, crypt debris, or detritus. The pharynx was not hyperemic and showed no granulations. Endoscopy confirmed persistent septal perforation without active bleeding or new mass formation.

The diagnosis of primary nasal TB was supported by the presence of bilateral nasal obstruction with a mass covered by greenish crusts, anterior septal perforation, and post-nasal drip on physical examination, all of which suggested a destructive granulomatous process localized to the nasal cavity. Laboratory evaluation showed mild anemia, neutrophilia, and monocytosis, with negative ANCA, HIV, and sputum AFB results, excluding systemic granulomatous diseases and pulmonary TB. Imaging revealed iso-dense

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Corresponding Author: Sonny Soebjanto
Email: sonnysoebjanto@unesa.ac.id

Table I: Evidence supporting diagnosis of primary nasal TB with MRSA co-colonization (no pulmonary involvement)

Category	Findings	Interpretation/Support
Physical Examination	- Nasal mass with greenish crusts - Anterior septal perforation - Post-nasal drip (+) - Later supraclavicular 2 cm mass (lipofibroma/lymphadenitis)	Suggests destructive granulomatous disease of the nasal cavity → consistent with nasal TB; perforation also seen in granulomatous diseases.[5]
Laboratory	- Hb 11.1 g/dL (mild anemia) → later normalized - Neutrophilia, monocytosis - ANCA negative (rules out Wegener's) - HIV negative - TB ICT, GeneXpert, AFB negative - MRSA swab positive initially, later eradicated	Histopathology confirmed TB despite negative sputum; consistent with localized nasal TB.[5] MRSA confirmed as secondary infection.[6]
Imaging	- CT: Iso-dense bilateral nasal lesions, septal deviation, suspected granuloma - Endoscopy: perforated nasal septum. - Chest radiograph: Normal repeatedly	Supports localized granulomatous nasal disease without pulmonary involvement.[5]
Histopathology	- Caseating granulomas with epithelioid cells and Langhans giant cells - Chronic granulomatous inflammation due to TB	Diagnostic hallmark of TB[1]; excludes malignancy.
Microbiology	- MRSA swab positive initially, later eradicated	Confirms secondary MRSA infection complicating TB.[6]

Table II: Diagnostic & therapeutic plan with objectives and reasoning

Objective/Condition	Plan	Reasoning
Confirm the etiology of the nasal lesion	Multiple biopsies (nasal cavity, septum) with histopathology	Gold standard for nasal TB[5]; rules out malignancy, midline granuloma, Wegener's, sarcoidosis.
Exclude systemic TB or mimics	Chest radiograph, sputum AFB, GeneXpert, ANCA, HIV test	Differentiate nasal TB from pulmonary TB or systemic granulomatous diseases.[5]
Assess the extent of the lesion	CT scan head & neck	Defines nasal/para-nasal involvement[5], excludes sinus extension, and guides surgical biopsy.
Evaluate lymph node mass	FNAB supraclavicular node, planned neck ultrasound	Rule out malignant spread or TB lymphadenitis[5]; FNAB showed a benign lesion.
Treat primary nasal TB	Anti-TB therapy Category I: 2HRZE/4HR; extended 9-12 months if persistent	Standard regimen for extrapulmonary TB[7]; extended duration in bone/cartilage involvement.
Manage septal perforation	Conservative (nasal irrigation, sprays)	Relieves symptoms, reduces crusts[5], and prevents secondary infection.
Eradicate MRSA coinfection	Oral cotrimoxazole, mupirocin nasal ointment, and chlorhexidine baths	Evidence-based eradication[4]; eradication confirmed.
Supportive therapy	Vitamin B6 with OAT, analgesics, and ciprofloxacin if needed	Prevents neuropathy, relieves symptoms, and treats superinfections.
Monitor treatment response	ENT & pulmonary follow-up, repeat swabs, imaging, blood monitoring	Ensure TB resolution, MRSA eradication, and detect complications early.

lesions in the nasal cavity with septal deviation but normal paranasal sinuses and chest radiographs, consistent with a localized extrapulmonary process. The initial lesion was not documented as the patient never noticed the perforation. Histopathology confirmed TB by demonstrating caseating granulomas with epithelioid cells and Langhans giant cells, ruling out malignancy. Additionally, microbiology identified MRSA colonization in the nasal cavity and throat, which was treated and eradicated, confirming a secondary infection complicating the underlying nasal TB.

The diagnostic and therapeutic approach in this case was aimed at confirming the etiology, excluding systemic involvement, and providing targeted management. Multiple biopsies of the nasal cavity and septum were performed to establish a definitive diagnosis. At the same time, chest radiographs, sputum AFB, GeneXpert, ANCA, and HIV testing ruled out pulmonary TB and systemic granulomatous diseases. CT scan defined the local extent of the disease, and FNAB of the supraclavicular lymph node excluded malignancy.

Once nasal TB was confirmed histologically, the patient was started on standard anti-TB therapy (Category I, 2HRZE/4HR), with possible extension to 9-12 months if granulomatous lesions persisted. Septal perforation was managed conservatively with saline irrigation and sprays to relieve symptoms, while secondary MRSA infection was eradicated with cotrimoxazole, mupirocin ointment, and chlorhexidine baths. Supportive treatment with vitamin B6, analgesics, and antibiotics for superinfections was added, and the patient was monitored closely with regular ENT and pulmonary follow-ups, repeat cultures, imaging, and laboratory tests to ensure resolution and prevent recurrence.

On follow-up, the patient continued anti-TB therapy (Category I, extended up to 9-12 months) with gradual clinical improvement. Repeated chest radiographs and

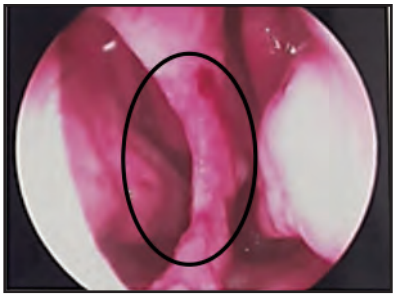

Fig. 1: An endoscopic image, presenting a perforated nasal septum due to necrosis

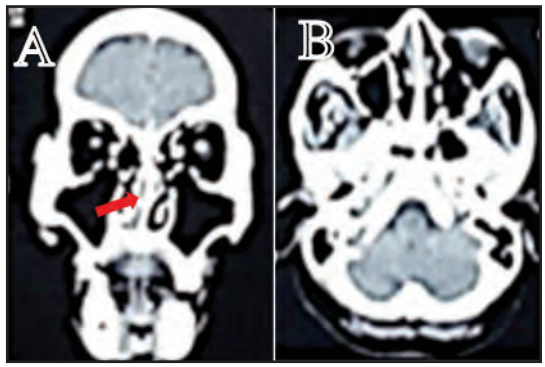

Fig. 2: A head CT-scan results, presenting a coronal(A)-axial(B) section, the red arrows highlight the rupture in the nasal septum

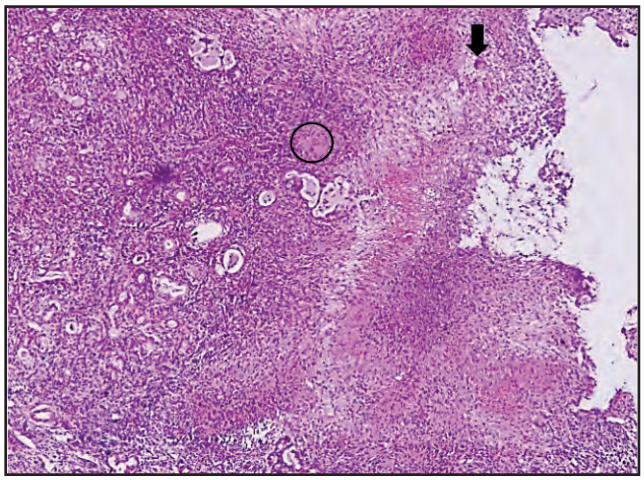

Fig. 3: In a histopathology evaluation, the circle indicates caseous necrosis, while the arrow indicates Langhans cells

sputum AFB remained normal, confirming no pulmonary involvement. MRSA colonization detected earlier was eradicated after cotrimoxazole, mupirocin, and chlorhexidine, with subsequent swabs turning negative. A supraclavicular lymph node mass was later found, but FNAB showed a benign process. The patient remained under regular ENT and pulmonary monitoring with stable progress.

DISCUSSION

Primary nasal TB is an extremely rare form of extrapulmonary TB. Since the first case was described in the 18th century, only a limited number of cases have been documented in the medical literature, with most reports highlighting its rarity and diagnostic difficulty. The disease can present either as a secondary manifestation of pulmonary TB or, more rarely, as a primary infection localized to the nasal cavity without pulmonary involvement.[5] Primary nasal TB is strongly associated with immunocompromised states, with sinonasal involvement typically arising from hematogenous or lymphatic spread.[8]

Reported cases span across all adult age groups, but several studies suggest a predominance in women, consistent with the present case. Although the nasal mucosa is generally resistant to *Mycobacterium tuberculosis* due to protective mechanisms such as mucociliary clearance and bactericidal nasal secretions, trauma or direct inoculation allows bacilli to penetrate the nasal mucosa and cause infection. Common presenting symptoms include chronic nasal obstruction, rhinorrhea, anosmia, and epistaxis, with anterior septal involvement being the most frequent site of pathology.[5,8]

In this patient, the combination of nasal obstruction, anosmia, a friable intranasal mass, and anterior septal perforation closely matched the typical features described in the literature. Nasal septal perforation is a complication of TB, as caseous necrosis associated with granulomatous inflammation destroys cartilage and mucosa of the upper respiratory tract.[9] The clinical significance of septal perforation extends beyond structural damage: it alters the intranasal environment, leading to recurrent crusting and creating a niche for bacterial colonization.[3] In this case, the perforation was further complicated by MRSA colonization, which underscores the importance of considering secondary infections in TB-affected nasal mucosa.[6]

The diagnostic process remains a challenge. Established modalities, chest radiography, sputum AFB smear, and GeneXpert, were consistently negative in this patient,

consistent with isolated nasal TB without pulmonary disease.[1] This diagnostic limitation necessitates reliance on histopathological examination of nasal tissue, which in this case revealed caseating granulomas with epithelioid cells and Langhans giant cells, confirming the diagnosis.[1,5] The granulomatous-hyperplastic cells are clinically important because their mass-forming and chronic inflammatory nature may mimic or predispose to malignant transformation, necessitating careful long-term surveillance.[5] However, histopathology has limitations due to sampling errors or low bacillary load; hence, repeat biopsies are required. These challenges highlight the need for innovative yet accessible diagnostic approaches for extrapulmonary TB. Treatment of nasal TB follows the same principle as pulmonary TB, employing the standard two-phase anti-TB regimen. In this case, the patient received Category I therapy consisting of an intensive phase with four drugs (isoniazid, rifampicin, pyrazinamide, ethambutol) followed by a continuation phase with two drugs (isoniazid, rifampicin). This regimen remains effective in eradicating bacilli and preventing relapse, although therapy may be extended to 9-12 months in cases with persistent granulomatous lesions or bony involvement.[7] Supportive local therapy, such as nasal saline irrigation, was beneficial for reducing crusting and improving hygiene.

Management of the secondary MRSA infection was essential for the patient's recovery. MRSA colonization of the nasal cavity was likely facilitated by mucosal disruption and environmental changes resulting from septal perforation. The eradication protocol, oral cotrimoxazole, topical mupirocin ointment, and chlorhexidine baths, proved effective, with subsequent swabs confirming clearance.[4]

CONCLUSION

This case illustrates the diagnostic complexity of primary nasal TB, particularly in the presence of negative conventional tests, and the value of histopathological confirmation. It also highlights the clinical importance of recognizing complications such as nasal septal perforation and secondary MRSA infection, both of which can significantly alter disease course and management. A comprehensive approach combining systemic anti-TB therapy, targeted eradication of resistant bacterial coinfection, and supportive nasal care allowed favorable clinical outcomes in this rare presentation.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

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