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TEACHING FILES (GRAND ROUNDS)

MAXILLARY SINUS TUBERCULOSIS WITH LOWER MOTOR NEURON FACIAL PALSY: A RARE PRESENTATION

Dhruv Gandhi, Ira Shah.
Department of Pediatric Infectious Diseases, BJ Wadia Hospital for Children, Mumbai, India.

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Clinical Problem:

A 5-year-old male was diagnosed to have Rosai-Dorfman disease in 2006 for which he received chemotherapy with vincristine, methotrexate, 6-mercaptopurine, cyclophosphamide, prednisolone and celecoxib. After treatment completion at 6 years of age, he had multiple recurrences of Rosai-Dorfman disease. In September 2024, at 23 years of age, he had fever, breathlessness, and productive cough for 3 days for which he was put on mechanical ventilation and underwent pleural effusion drainage at another centre. He was investigated and non-contrast computerized tomography (NCCT) chest showed multiple small nodules in the lungs and a right-sided pleural effusion. Blood cultures were negative. Bronchoalveolar lavage (BAL) Xpert MTB/Rif was positive for rifampicin-sensitive *Mycobacterium tuberculosis* (MTB). BAL Mycobacterium growth indicator tube culture was positive and phenotypic drug-susceptibility testing revealed sensitivity to isoniazid, rifampicin, pyrazinamide, and ethambutol. Other investigations are shown in Table 1. He was started on steroids and first-line antitubercular therapy (ATT) with isoniazid, rifampicin, pyrazinamide, and ethambutol. He was subsequently weaned off the ventilator and discharged in October 2024. However, he developed drug induced liver injury and was shifted to ethambutol, amikacin and moxifloxacin. In November 2024, he developed a recurrence of Rosai-Dorfman disease for which he received weekly vinblastine (10 mg/m^2/dose), and also developed a right-sided maxillary swelling. NCCT paranasal sinuses showed a 4.6x5.2x5.5 cm swelling in the right maxillary sinus, eroding into the side of the maxilla, and extending into the right ethmoid sinus, sphenopalatine foramen, and pterygopalatine fossa. There was no intracranial extension. Tissue biopsy Xpert MTB/Rif was positive for rifampicin-sensitive MTB complex. Bacterial and fungal cultures of the biopsied tissue were negative. The same ATT was continued. In December 2024, he developed right-sided lower motor neuron (LMN) type of facial palsy. He was subsequently referred to us for further management. Magnetic resonance imaging (MRI) brain in December 2024 showed a stable size of the maxillary sinus lesion (5x4.9 cm) and no brain parenchymal lesions. He started on prednisolone (1 mg/kg/day) and ATT was changed to linezolid, amikacin, ethambutol, and moxifloxacin. In January 2025, at the 1-month follow-up, his facial weakness had recovered and he was advised to taper and stop prednisolone.

How common is paranasal sinus tuberculosis (TB) and how to treat LMN facial palsy with paranasal sinus TB?

Discussion:

Sinonasal TB is a rare entity, even in TB endemic countries like India, and thus often flies under the radar of suspicion. Paranasal sinus involvement may rarely be primary, however, it more commonly occurs secondary to pulmonary TB, either through infected droplets or miliary or lymphatic spread.[1] The risk factors identified include immunosuppression, as in our patient, human immunodeficiency virus infection, and diabetes. The nasal cavity and paranasal sinuses are protected from developing TB by bactericidal secretions, ciliary functions, and mechanical filtering by the nasal vibrissae.[1,2] The most common paranasal sinus involved is the maxillary sinus, as in our patient, and the disease is commonly unilateral.[1] Paranasal sinus TB without the involvement of the nasal cavity, as in our patient, has been rarely reported in literature.[2] Females are more commonly affected and the clinical spectrum commonly includes non-specific symptoms such as nasal obstruction, recurrent epistaxis, rhinorrhoea, nasal crusting, hyposmia, and headache.[1,2] Patients may rarely present with facial swelling mimicking an odontogenic infection, with ear discharge, hearing loss, and tinnitus due to middle ear involvement, with cheek pain and numbness resembling trigeminal neuralgia, and proptosis and trismus resembling malignancy.[3,4,5] Confirmation of the diagnosis of sinonasal TB is often difficult. Nasal swabs and nasal secretions cannot be used due to their paucibacillary yield. Tissue specimens may show acid-fast bacilli but are not always confirmatory of the diagnosis. Histopathological examination may be required showing caseating granulomas with necrosis.[2] Molecular methods, as in our patient, can be used to diagnose TB on the tissue specimen with a high specificity. However, in many cases, neither a histological nor a microbiological confirmation is possible, resulting in the diagnosis being made on the basis of the absence of a clinical

Address for Correspondance: Dhruv Gandhi, 5B/13 Shyam Niwas, Breach Candy, Mumbai-400026, Maharashtra, India.
Email: dhruvgandhi2610@gmail.com

Table 1. Investigations of the patient.

Parameters	November 2024	December 2024	January 2025	Reference Ranges
Hemoglobin (gm/dL)	-	9.5	-	11.5-15.5
White blood cell count (cells/cumm)	-	3420	-	5000-13,000
Platelets (105 cells/cumm)	-	5.10	-	1.50-4.50
ALT (IU/L)	141	711	143	<41
AST (IU/L)	224	450	138	<41
Total bilirubin (mg/dL)	0.42	0.73	-	0.0-1.10
Direct bilirubin (mg/dL)	0.21	0.15	-	0.0-0.60
Serum total protein (gm/dL)	-	8.38	7.96	6.00-8.30
Serum albumin (gm/dL)	-	4.13	4.46	3.80-5.40

Note : ALT- Alanine aminotransferase, AST- Aspartate aminotransferase.

improvement to antibiotics, and the presence of a clinical response to ATT. Findings on CT or MRI are non-specific for the diagnosis of paranasal sinus TB, however, these modalities are primarily used to determine the size and extent to the disease.[2] LMN facial palsy has been reported in association with several forms of head and neck TB, however, it has not been previously reported with paranasal sinus TB. In our case, we ruled out intracranial and temporal bone tuberculomas as potential causes of LMN facial palsy on CT and MRI imaging. Additionally, imaging showed a tubercular lesion restricted to the maxillary sinus, thus direct facial nerve compression from the paranasal sinus lesion was ruled out. For Bell's palsy, steroids should be administered within 72 hours of paralysis onset at a dose of 1 mg/kg/day of prednisone for 5-7 days.[6] Other treatment options include antiviral medications and surgical decompression of the facial nerve. However, there is no current consensus on the benefit of antivirals in cases of Bell's palsy, and surgical options are not preferred in the acute stage and have similar outcomes with medical management.[6] Overall, Bell's palsy has an excellent prognosis with recovery within a few weeks to months. About 80% patients recover without treatment and the addition of steroids alone increases the recovery rate to around 90-97%.[6] The facial palsy in our patient was idiopathic, and thus we treated his Bell's palsy with steroids while concomitantly treating with ATT for his paranasal sinus TB.

Compliance with ethical standards
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Conflict of Interest: None

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