



Primary Parotid Tuberculosis, An Atypical Parotid Tumour

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Abstract

Tuberculous parotitis, a rare form of extrapulmonary tuberculosis, is an important consideration in the differential diagnosis of parotid tumors. We present the case of a 35-year-old woman with ganglionic tuberculous parotitis, emphasizing the importance of early detection to initiate antituberculous therapy and prevent complications.

Keywords Parotid Tuberculosis · Extrapulmonary Tuberculosis · Salivary Gland Diseases · Fine-Needle Aspiration Cytology · Antitubercular Agents

Introduction

Primary parotid tuberculosis (PPT) is an uncommon extrapulmonary manifestation, accounting for 0.04–0.4% of salivary gland lesions [1]. It typically presents as a solitary, painless swelling and is frequently misdiagnosed as a neoplasm or infection, especially in non-endemic regions with limited clinical awareness [1, 2]. Diagnosis relies on clinical evaluation, imaging, and microbiological confirmation, with fine needle aspiration (FNA) often revealing granulomatous inflammation [1]. Definitive diagnosis requires isolation of *Mycobacterium tuberculosis* via culture or PCR. Treatment consists of standard anti-tuberculosis therapy (ATT), reserving surgery for complications. Early recognition is essential for optimal outcomes [2]. This case underscores the need to include PPT in the differential diagnosis of parotid lesions, particularly when the initial presentation is a parotid mass followed by satellite adenopathy.

Case Report

We describe the case of a 35-year-old Bolivian woman who arrived in Spain six months prior and subsequently sought medical attention. In November 2024, she presented with a three-month history of small, fluctuating tumors in the left parotid region, which had gradually increased in size. In addition, over the past two weeks, she had noticed the development of new lymphadenopathies localized in the jugular and spinal regions. She denied experiencing any pain, inflammation, or systemic symptoms; however, she reported a significant family history of pulmonary tuberculosis, with both her mother and brother having received treatment previously. The patient's medical history was otherwise unremarkable, aside from the fact that she was continuing to breastfeed her two-year-old son.

On physical examination, a firm, non-tender retromandibular swelling was palpable, and four cervical lymph nodes were detected without overt signs of inflammation. The initial evaluation was supplemented by an ultrasound examination, which identified three hypoechoic lesions measuring between 8 and 15 mm, each demonstrating both hilar and peripheral vascularization. This finding raised the suspicion of an underlying infectious process. Subsequently, maxillofacial MRI (Fig. 1) was performed, revealing a well-circumscribed, rounded intra-parotid lesion exhibiting contrast enhancement along with cystic-necrotic areas on T1-weighted sequences and marked diffusion restriction; notably, T2-weighted images further illustrated hyperintensity within the lesion. Additionally, lymphadenopathies in

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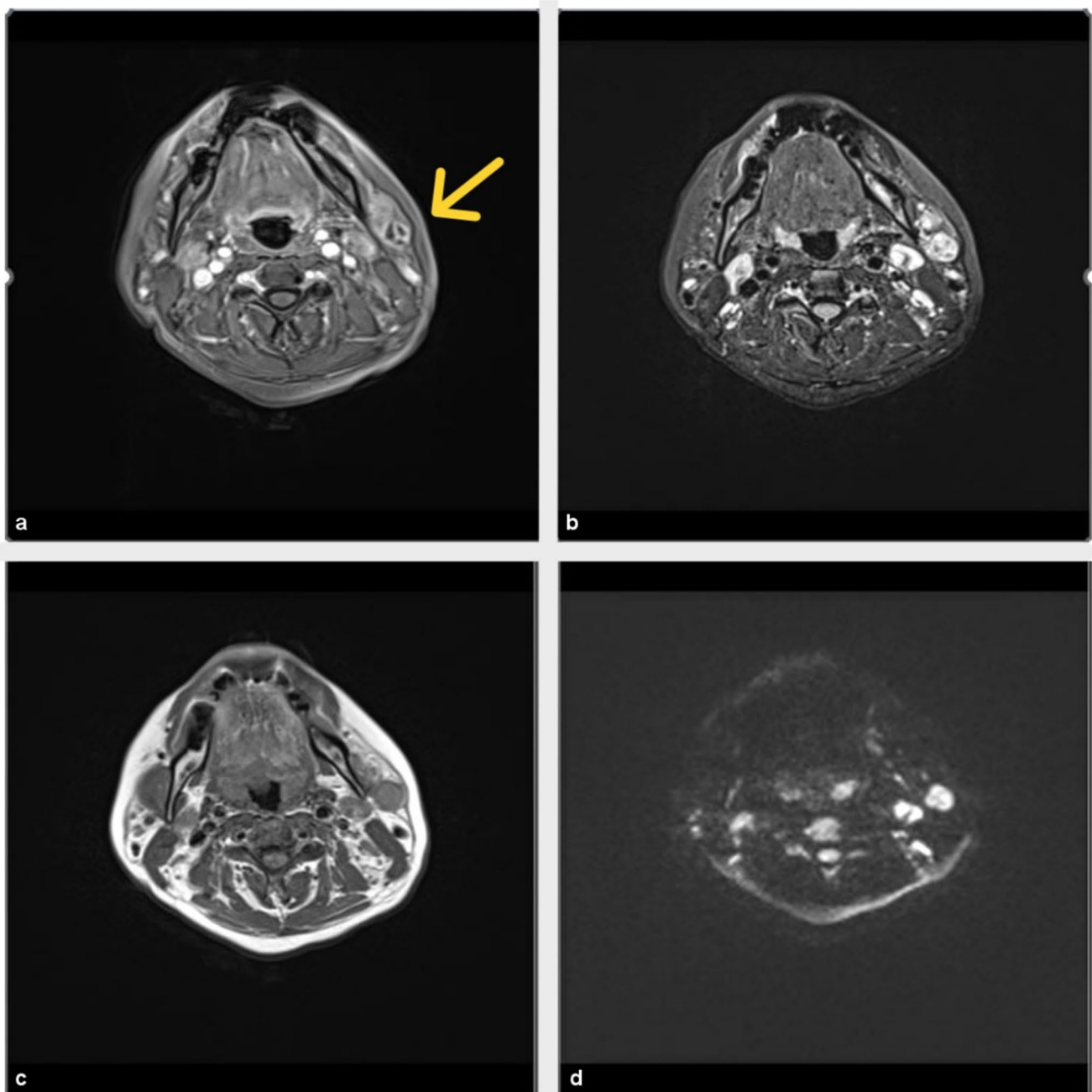


Fig. 1 Maxillofacial MRI image showing rounded intraparotid lesion with enhancement and cystic-necrotic content in T1 fat-sat (**a**), STIR (**b**) and standard T1 (**c**) sequences. Marked restriction is also observed in diffusion sequence (**d**)

the IB and IIA regions displayed imaging characteristics consistent with an infectious etiology.

Fine needle aspiration (FNA) was conducted (Fig. 2) and revealed non-necrotizing granulomatous adenitis characterized by a predominance of polymorphic lymphoid cells—specifically T lymphocytes (CD3+), B lymphocytes (PAX5+), and histiocytes (CD68+)—without malignant cells. Further confirmatory tests, including Ziehl-Neelsen staining, the Quantiferon test, and culture

in Löwenstein-Jensen medium, were performed, and the patient was referred to the Infectious Diseases Department. Three weeks later, while remaining clinically stable, both the culture and Quantiferon test confirmed the diagnosis of primary tuberculous adenitis with nodal involvement. She was advised to cease breastfeeding, and after assessment of her family environment, standard anti-tuberculosis therapy (ATT) was initiated.

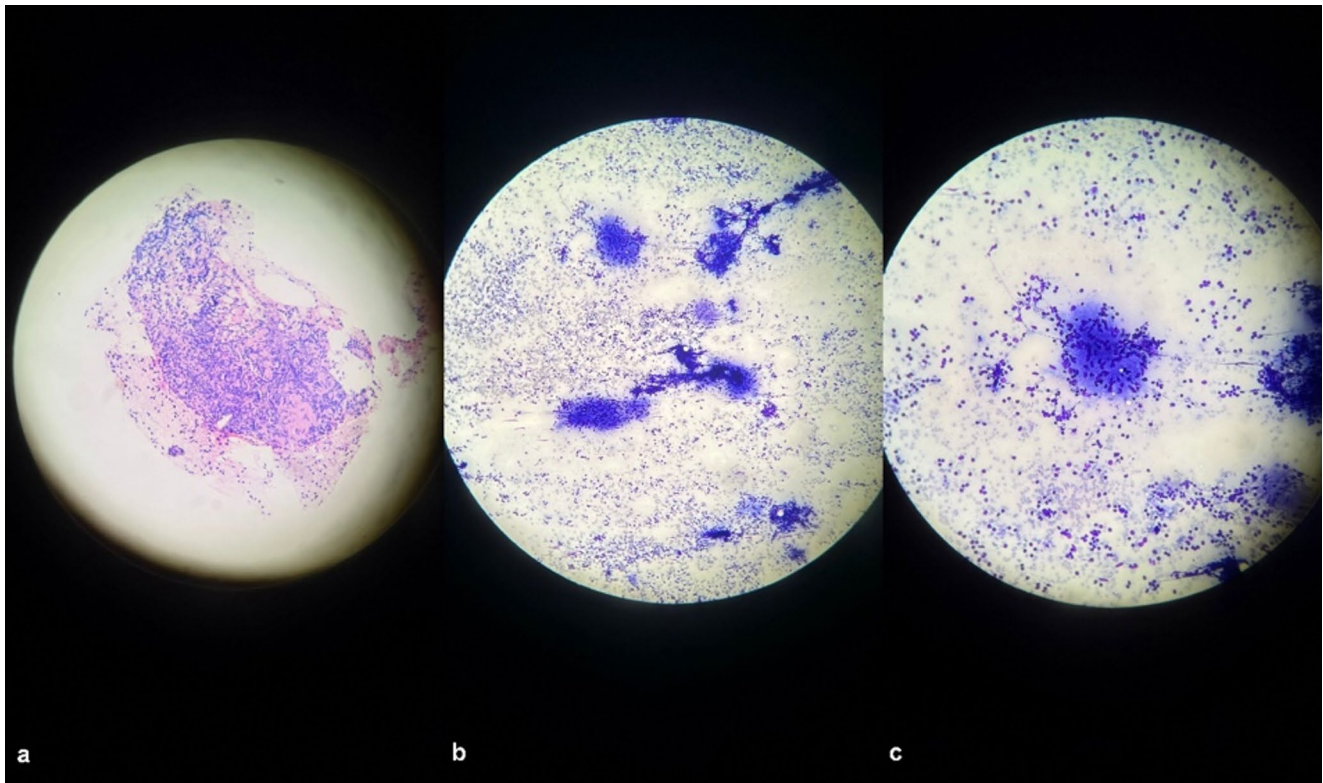


Fig. 2 Tissue staining image obtained by FNA, stained with H-E (**a**). Numerous epithelioid histiocytes and a crown of inflammatory cells, mainly lymphocytes, are visualized. The following two images show two enlargements of the sample stained with DIF-QUICK (**b** and **c**)

During the first two weeks of treatment, the patient experienced nausea and vomiting, which improved progressively. At her one-month follow-up visit, a significant reduction of the parotid swelling was noted and the cervical adenopathies had resolved. Based on her favorable clinical and laboratory response, it was decided to schedule another clinical-analytical control one month later before transitioning to a regimen consisting of two drugs.

Discussion

Tuberculosis is a systemic disease with a variable clinical spectrum. Although primarily affecting the lungs, extrapulmonary manifestations occur in up to 5.4% of cases [3]. Among these, lymphatic involvement is the most common (30–50%), whereas salivary gland infection is exceedingly rare [4]. Parotid tuberculosis, the most common salivary gland involvement, remains of uncertain incidence. This case emphasizes the necessity of considering this diagnosis, particularly in individuals from endemic regions or with risk factors [5].

From an otolaryngological perspective, PPT is a diagnostic challenge due to its similarity to neoplastic and inflammatory conditions. Typically, it presents as a painless,

fluctuating parotid swelling—often the sole symptom—with only 25% of cases showing an abnormal chest X-ray [6]. Differential diagnosis includes salivary neoplasms such as pleomorphic adenoma and low-grade malignancies [4–6]. Histopathology via FNA is crucial, usually revealing necrotizing granulomatous adenitis with granulomas (caseating or non-caseating) infiltrating glandular tissue from adjacent lymph nodes [6]. In our patient, parotid involvement preceded lymphadenopathy, an atypical course. Immunohistochemistry typically demonstrates an inflammatory infiltrate dominated by histiocytes (CD68+), multinucleated giant cells, and T-lymphocytes (CD3+) [5]; notably, caseous necrosis may be absent, as observed here.

Additional diagnostic techniques—PCR for *M. tuberculosis*, the Mantoux test, or culture in Löwenstein-Jensen medium—enhance sensitivity but should complement histopathology [6–8]. Imaging, including ultrasound and MRI, offers supportive but non-definitive data [5, 6]. A multidisciplinary approach, involving otolaryngologists, radiologists, and infectious disease specialists, is essential for accurate diagnosis and management.

Despite its rarity, primary parotid tuberculosis carries an excellent prognosis with prompt standard ATT. Most cases, including ours, exhibit rapid improvement after treatment initiation [9]. In the absence of specific clinical trials,

management follows general tuberculosis guidelines (Isoniazid+Rifampicin+Pyrazinamide+Ethambutol for two months, then Isoniazid+Rifampicin for four months) [10].

This case reinforces the importance of including parotid tuberculosis in the differential diagnosis of parotid lesions to prevent misdiagnosis, avoid unnecessary surgery, and ensure timely, effective treatment for optimal patient outcomes. Increased awareness and research are vital to enhance diagnostic accuracy and treatment strategies for this rare but clinically significant condition.

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Declarations

Ethical Approval Ethical approval was waived by the local Ethics Committee of Hospital Universitario y Politécnico La Fe in view of the retrospective nature of the study and all the procedures being performed were part of the routine care.

Consent to Participant The participant has consented to the submission of the case report to the journal and the publication of the images in Figs. 1 and 2.

Conflict of Interest The authors declare that they have no conflict of interest.

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