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Parotid Sialolithiasis without Classical Clinical or Imaging Features: A Case Report

Revathi Krishna, Rezhat Abbas, Priya Kumar *, Jeyaseelan Augustine, Zainab Chaudhary Aadithya Basavaraj Urs

Department of Oral & Maxillofacial Pathology and Microbiology, Maulana Azad Institute of Dental Sciences, BSZ Mard, New Delhi, India.

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**Corresponding author:*
Priya Kumar

Department of Oral & Maxillofacial Pathology and Microbiology, Maulana Azad Institute of Dental Sciences, BSZ Mard, New Delhi, India.

Tel: +98-21-849033747
Fax: +98-21-84903747
Email: drpri_kumar@yahoo.com

ABSTRACT

Sialolithiasis of the parotid gland is an uncommon occurrence, accounting for only 10–20% of all salivary calculi. This report describes an unusual case of parotid sialolithiasis in a 42-year-old female who presented with a soft, tender, and ill-defined swelling on the left side of the face persisting for one year. Clinical examination showed no notable extraoral or intraoral swelling. Magnetic resonance imaging revealed an intensely enhancing lesion anterior to the left masseter region, suggestive of a soft tissue neoplasm such as lipoma, fibroma, or hemangioma. Histopathological examination aided in the identification of sialolith within the excretory duct, accompanied by extensive destruction of the serous acini by chronic lymphoplasmacytic infiltrate, aiding in the diagnosis. This case underscores the diagnostic challenge posed by parotid sialoliths lacking classical symptoms and the absence of its detection in diagnostic imaging.

Keywords: Sialolithiasis; Parotid; Magnetic resonance imaging.

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Case Report

A 42-year-old female patient visited our hospital with the complaint of swelling on the left side of the face for one year. No swelling was evident on extraoral or intraoral inspection (Figure 1A, B). Palpation of the left side of the face revealed a 2 x 1.5 cm well-circumscribed, soft, tender swelling below the left zygomatic arch. On magnetic resonance imaging (MRI), an intensely enhancing space-occupying lesion anterior to the left masseter was evident, of size 22x18x24 mm (Figure 1C). A provisional diagnosis of lipoma was considered with the differential diagnosis of fibroma, hemangioma, and soft tissue pilomatricoma. Fine needle aspiration cytology of the lesion demonstrated blood mixed with reactive lymphoid tissue. The lesion was excised and sent for histopathological examination (Figure 1D). The whole slide image of the slide (Figure 2, 10x) demonstrated tissue infiltrated by dense collection of chronic inflammatory cells with focal areas of germinal center formation and surrounded by adipocytes resembling the structure of a lymph node with deranged architecture. However, on higher magnification (Figure 3A-C), numerous ducts were evident. Intercalated, striated and excretory ducts were identified with areas of ductal hyperplasia. Serous salivary gland acini, devoid of their normal lobular architecture, were observed at a focus. A provisional histopathological diagnosis of lymphadenoma was considered. Deeper sections of the tissue revealed irregular basophilic calcifications within an excretory ductal lumen. The surrounding inflammatory infiltrate was a mixture of lymphocytes and plasma cells. The identification of calcified mass within the duct and mixed chronic inflammatory cell infiltrate were the major clues changing the diagnosis of the case to sialolithiasis with chronic sialadenitis.

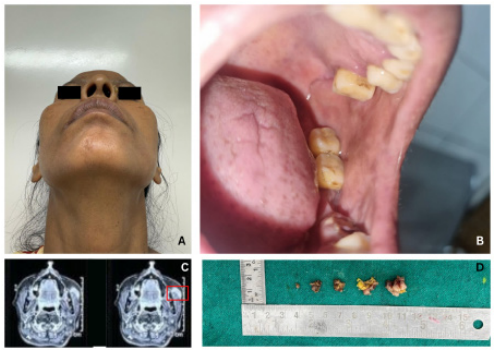

Figure 1. A, B: Extra-oral and intra-oral clinical photograph does not reveal any gross swelling; C: MRI shows a 22x18x24 mm space-occupying lesion anterior to the left masseter; D: Macroscopy of excision tissue shows multiple soft tissue bits.

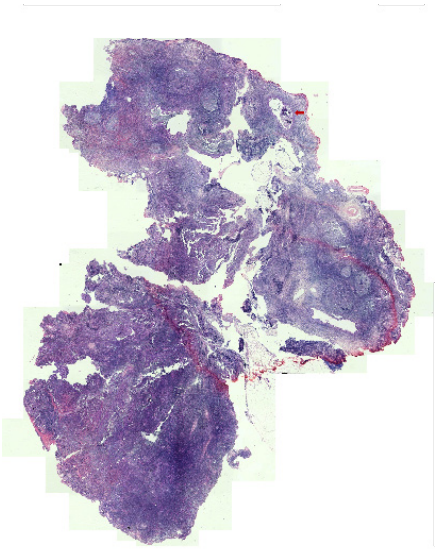

Figure 2. Whole slide image (10x) shows tissue infiltrated by dense chronic inflammatory cell infiltrate with focal areas of germinal center formation. Intercalated, striated and excretory ducts are noted within the connective tissue with a focal excretory duct showing diffuse basophilic calcifications (red arrow).

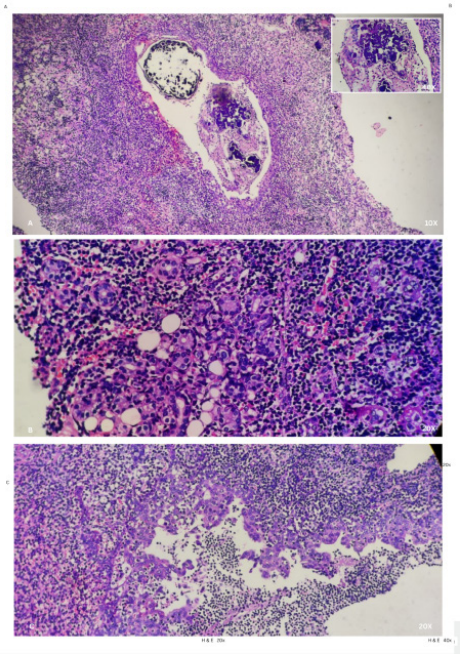

Figure 3. A: Hematoxylin and eosin (H and E) stained section (10x) shows diffuse basophilic calcifications within an excretory duct lined by pseudostratified columnar epithelium; B: Numerous serous acini and intercalated ducts are noted in intense chronic inflammatory cell infiltrate composed chiefly of lymphocytes and plasma cells. Note the effacement of lobular architecture of salivary gland; C: Ductal hyperplasia is seen with inflammatory microabscess within the ductal epithelium. Surrounding connective tissue is densely infiltrated by chronic inflammatory cell infiltrate composed chiefly of lymphocytes and plasma cells with areas of extravasated RBCs.

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Discussion

Sialoliths are calcified concretions that commonly cause salivary duct obstruction. Scanning electron microscopic and X-ray diffraction studies describe them as oolitic aggregates, having a central core surrounded by concentric rings of organic and inorganic materials [1]. (Barrueco 2025). Sialoliths, though common in the submandibular gland, are uncommon in the parotid gland and account for 10-20% of such cases, largely owing to distinct anatomical and physiological factors. The submandibular gland's Wharton's duct is longer, follows an uphill trajectory, and its saliva is more viscous with higher pH and calcium concentration. These features foster mineralization and stasis, making sialoliths more frequent and larger in this gland. In contrast, parotid saliva is less viscous, more acidic, and contains less calcium, resulting in fewer and generally smaller, rounder stones [2]. (Kraij 2014). The described case highlights the diagnostic complexities that can arise with parotid gland sialolithiasis, especially when atypical features such as deep location, absence of overt swelling, and ambiguous imaging findings lead to consideration of alternative diagnoses. The clinical course of subtle, soft, and deeply situated swelling, in the absence of obvious extraoral or intraoral changes, underscores the variability in the clinical manifestation of parotid sialolithiasis. While classical sialolithiasis presents with intermittent swelling and discomfort associated with meals, this patient's presentation was indolent, with symptoms presenting as a soft tissue neoplasm such as lipoma, or as vascular and fibroblastic lesions, as reflected in the initialclinical differential diagnoses [3]. (Antoniadis D 1989).

Radiological examination in this case, via MRI, highlighted a well-circumscribed, enhancing lesion anterior to the masseter but did not conclusively demonstrate calcifications. The use of MRI in the detection of calcifications or hard tissue lesions is less appreciated. The clinical presentation of the case, producing a soft, tender swelling in the region of masseter without the characteristic symptoms of sialolithiasis, led the clinician to perform an MRI for the present case. The extent of calcifications within the duct was minimal, thus exempting its identification by MRI. This emphasizes that not all sialoliths, particularly in the parotid gland, are readily detected by conventional imaging, with a significant proportion lacking radiopacity on plain radiographs and even advanced modalities. Histopathology played a pivotal role: initial low-power assessment suggested a lymphoid neoplasm, but high-power magnification revealed chronic sialadenitis with ductal hyperplasia, disrupted glandular architecture, and, most critically, ductal calcification. This finding clarified the true etiology as sialolithiasis with associated chronic inflammation, overturning initial suspicions of lymphadenoma or other neoplastic pathology. The chronicity of inflammation and predominance of lymphoid tissue are consistent with chronic sialadenitis secondary to long-standing ductal obstruction by sialolithiasis. The formation of lymphoid aggregates and ductal hyperplasia reflects persistent antigenic stimulation and ductal irritation. Chronic obstruction results in stasis, predisposing to secondary infection and tissue remodeling. The "retrograde theory" of sialolith formation, where debris or foreign material becomes a nidus for calcification, may be applicable in such cases with prolonged evolution [4]. (Marchal F et al. 2001).

Conclusion

This case exemplifies the importance of correlating clinical, radiologic, and particularly histopathological findings to avoid misdiagnosis and to recognize the less common presentations of parotid sialolithiasis. Early and accurate identification is essential to prevent irreversible glandular damage and recurrent infectious or inflammatory episodes.

Conflict of Interest

There is no conflict of interest to declare.

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