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Original Research Article

# Fungal Parotid Abscess: Rare Entity But Not to Be Missed

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**Abstract**

**Background:** Parotid abscesses are uncommon infections of the major salivary glands, most often caused by bacterial pathogens such as Staphylococcus aureus and mixed anaerobes. Fungal parotid abscesses are exceedingly rare, with only a few reported cases in the literature. Immunocompromised states, including diabetes mellitus, predispose individuals to opportunistic fungal infections, often caused by Candida species. Early recognition of this entity is critical to avoid delayed treatment and potential life-threatening complications.

**Case Presentation:** The case report of a 70-year-old woman with long-standing diabetes mellitus who presented with a painful swelling of the left parotid region. Clinical examination revealed a tender, erythematous swelling without discharge and intact facial nerve function. Laboratory investigations demonstrated leukocytosis and raised C-reactive protein. Ultrasound showed a hypoechoic lesion suggestive of an abscess, and contrast-enhanced CT of the face and neck confirmed a peripherally enhancing lesion involving both superficial and deep lobes of the parotid. Surgical incision and drainage yielded approximately 15–20 ml of pus. Initial empirical antibiotic therapy failed to improve the condition. Gram stain revealed budding yeast cells, and culture grew Candida non-albicans, resistant to fluconazole and voriconazole but sensitive to Nystatin and Amphotericin. The patient was subsequently treated with intravenous Caspofungin and oral Nystatin, with marked improvement in inflammatory markers, wound healing, and full recovery at two weeks. Histopathology confirmed fungal elements.

**Conclusion:** This case underscores the importance of considering fungal etiology in parotid abscesses, particularly in elderly diabetic or immunocompromised patients unresponsive to antibiotics. Prompt imaging, microbiological diagnosis, and tailored antifungal therapy are essential to achieving favorable outcomes.

**Keywords:** Candida Non-Albicans, Caspofungin, Diabetes Mellitus, Fungal Infection, Nystatin, Parotid Abscess, Salivary Gland.

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## Introduction

The parotid glands are the largest of the salivary glands, located anteroinferior to the external ear, extending from the zygomatic arch to the angle of the mandible [1]. They play a crucial role in the production and secretion of saliva, aiding in digestion and maintaining oral health. Because of their anatomical position and the long course of Stensen's duct, the parotid glands are vulnerable to both inflammatory and infectious processes. Acute parotid infections are relatively common in elderly patients, postoperative individuals, or those with systemic comorbidities, particularly when salivary flow is reduced due to dehydration, ductal obstruction, or poor oral hygiene. The most frequently encountered pathogens in acute bacterial parotitis include Staphylococcus aureus, which remains the predominant causative organism, followed by anaerobic bacteria that may ascend through the ductal system [2]. In comparison, fungal infections of the parotid gland are exceedingly rare, and when they occur, they are often associated with immunocompromised states such as uncontrolled diabetes mellitus, HIV infection, or long-term steroid therapy [3]. Among fungi, Candida albicans is the most common species implicated in invasive infections; however, non-albicans Candida (NAC) species such as Candida tropicalis, Candida parapsilosis, Candida glabrata, and Candida auris are increasingly recognized as opportunistic pathogens, often demonstrating greater antifungal resistance and posing significant therapeutic challenges. The clinical recognition of fungal parotid abscess is difficult because its presentation often impersonators that of bacterial infections, with localized swelling, erythema, pain, and systemic

signs of sepsis [4]. In the absence of early suspicion and microbiological confirmation, fungal etiology may remain undiagnosed, leading to inappropriate treatment, poor clinical outcomes, and a higher risk of complications, including facial nerve palsy, extension into deep neck spaces, and systemic dissemination [5]. The rarity of this entity, combined with its potentially severe consequences if overlooked, underscores the importance of thorough diagnostic evaluation, including imaging and microbiological analysis, particularly in high-risk patient groups.

In this report, we present the case of a 70-year-old woman with diabetes mellitus who developed a parotid abscess caused by Candida non-albicans. The aim is to highlight the unusual occurrence of this infection, emphasize the role of radiological and microbiological assessment in its diagnosis, and discuss the clinical implications of early recognition and tailored antifungal management.

**Case Presentation**

**Patient History:** A 70-year-old woman presented to the Department of Medicine of a tertiary care center with complaints of painful swelling in the left parotid region for one week. She had a known history of type 2 diabetes mellitus and hypertension, both of which had been poorly controlled over the preceding years. She was subsequently referred to the Department of Otorhinolaryngology for further evaluation and management. The patient denied any history of trauma, dental procedures, or preceding upper respiratory tract infection. There was no prior history of recurrent parotid swelling, xerostomia, or known autoimmune disease.

**Clinical Findings:** On physical examination, a localized swelling measuring approximately 3 × 2 cm was noted just below the left ear lobule, situated in the retromandibular groove. The swelling displaced the ear lobule upwards, and the overlying skin appeared tense, shiny, and erythematous, suggesting an underlying suppurative process. Palpation revealed a tender, firm-to-fluctuant swelling with ill-defined margins. Intraoral examination revealed an adequate mouth opening with no evidence of discharge from the Stensen's duct orifice; there were no signs of oropharyngeal candidiasis. Neurological examination confirmed that the facial nerve was intact, with preserved motor function. Systemically, the patient was febrile and reported malaise.

**Laboratory Findings:** Routine hematological investigations showed leukocytosis, with a total white blood cell count of 20,110/µL, and a markedly elevated C-reactive protein (CRP) level of 197 mg/dL, suggestive of ongoing systemic inflammation. Renal and liver function tests were within normal limits.

**Imaging Findings:** Ultrasound of the neck revealed an irregular, ill-defined, heterogeneously hypoechoic lesion located in the left parotid region adjacent to the gland. The lesion demonstrated no internal vascularity and measured approximately 2.1 × 3.1 × 2.2 cm (volume: 7.1 cc), raising the suspicion of abscess formation.

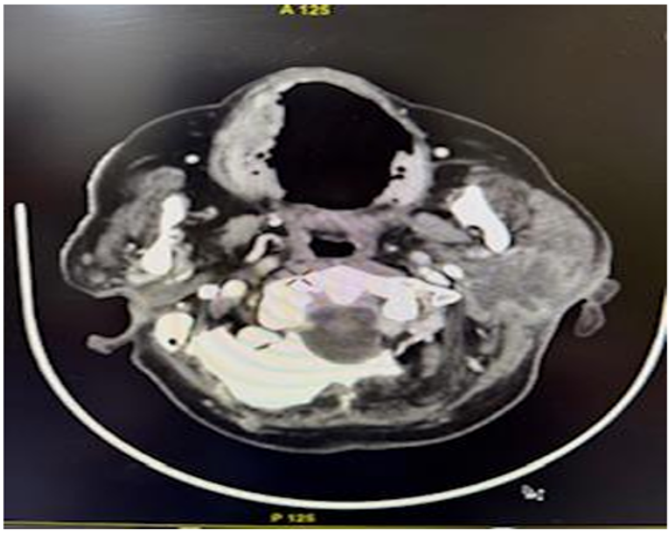

**Figure 1: Contrast CT Face and Neck Showing Left Parotid Abscess.**

For further evaluation, a contrast-enhanced computed tomography (CT) scan of the face and neck was performed. The scan demonstrated a large, irregular, peripherally enhancing hypodense lesion within the left parotid gland, involving both the superficial and deep lobes. The lesion measured approximately 37 mm (anteroposterior) × 33 mm (transverse) × 39.2 mm (craniocaudal). The features were highly suggestive of an abscess (Figure 1). No obvious extension into the deep neck spaces was realized, and the adjacent great vessels appeared uninvolved.

**Management:** Given the clinical and radiological findings, the patient was planned for surgical drainage. Under aseptic precautions, incision and drainage of the abscess was performed, yielding approximately 15–20 mL of thick purulent material. The pus was sent for Gram stain, bacterial culture and sensitivity, fungal culture, and acid-fast bacilli (AFB) smear. Postoperatively, the patient was started on broad-spectrum intravenous antibiotics empirically, while awaiting culture results. Gram staining of the pus revealed the presence of budding yeast cells, raising the suspicion of a fungal etiology. Initially, the patient was started on oral fluconazole. However, culture sensitivity testing later confirmed the presence of Candida non-albicans, which demonstrated resistance to fluconazole, voriconazole, and itraconazole, but sensitivity to Nystatin and Amphotericin B. Based on this report, fluconazole was discontinued and the antifungal regimen was revised to intravenous Caspofungin along with oral Nystatin suspension. Over the following days, the patient's inflammatory markers gradually improved, with a reduction in leukocyte count and normalization of CRP. Daily wound dressings were carried out until healthy granulation tissue was observed. The wound was subsequently closed and sutured. Following two weeks of antifungal therapy, the patient was afebrile, the swelling had resolved, and she was discharged in a stable condition with advice for follow-up.

**Histopathology:** Histopathological examination of the pus specimen confirmed the presence of fungal elements consistent with Candida species. Microscopy demonstrated budding yeast cells and pseudohyphae characteristic of invasive candidiasis (Figure 2).

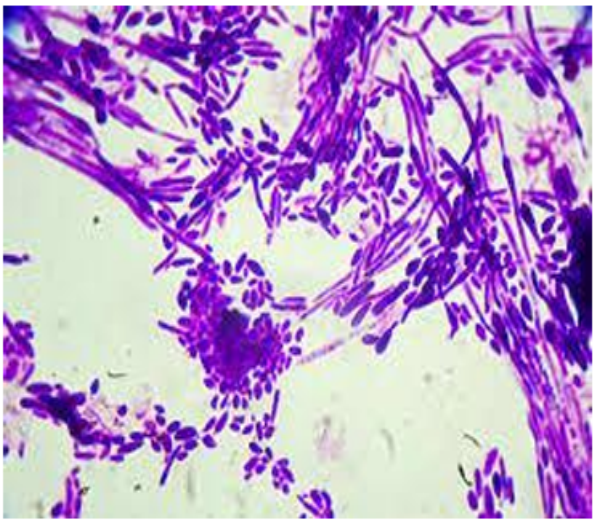

**Figure 2: Hpe S/O Candida Non Albicans**

**Consent:** Written informed consent was obtained from the patient for the publication of this case report, including the use of clinical and imaging data for academic and research purposes.

**Discussion**

**Epidemiology and Pathogenesis:** Fungal infections of the parotid gland are an exceptionally rare clinical entity, in contrast to bacterial parotitis, which is commonly encountered in routine otolaryngology practice. Candida species are commensals of the oral cavity and form an integral part of the normal oral flora [6] demonstrated that nearly 75% of healthy individuals carry Candida species asymptomatically, with Candida albicans being the predominant species, accounting for approximately 40% of isolates, followed by Candida parapsilosis and Candida tropicalis at around 15% each. While carriage is usually harmless, these organisms may become opportunistic pathogens under favorable conditions. Immunocompromised individuals, including patients with poorly controlled diabetes mellitus, malignancy, HIV infection, or those on immunosuppressive therapy, are at increased risk of invasive candidiasis. In diabetics, hyperglycemia impairs neutrophil function, reduces salivary flow, and creates an environment conducive to fungal proliferation [7]. Additional risk factors include xerostomia due to medications, poor oral hygiene, dehydration, and obstruction of Stensen's duct by stones or tumors. In such settings, Candida can ascend through the ductal system and invade the glandular parenchyma, leading to abscess formation.

**Review of Literature:** Reports of fungal parotid abscesses are scarce in medical literature, with only a handful of cases documented to date. [8] Described a Candida abscess of the parotid gland associated with facial nerve paralysis, underscoring

the potential severity of this condition. [9] Reported a case of Candida parotitis with abscess formation, while describing a Warthin's tumor representing a fungal abscess in an immunocompetent host. More recently, [10] documented a case of Candida glabrata parotid abscess, further expanding the spectrum of NAC implicated in parotid infections.

By contrast, bacterial abscesses of the parotid gland remain far more common, with Staphylococcus aureus being the most frequently isolated organism, followed by mixed anaerobes. These bacterial infections typically respond well to broad-spectrum antibiotics combined with drainage. However, fungal parotid abscesses pose a unique therapeutic challenge, as they are not only rare but also often resistant to first-line antifungal agents.

The rise in NAC infections has been particularly concerning. NAC species, including C. tropicalis, C. parapsilosis, C. glabrata, and C. auris, now account for a significant proportion of invasive candidiasis cases, collectively rivaling C. albicans in clinical importance.

These species are often more virulent in immunocompromised hosts and exhibit intrinsic or acquired resistance to commonly used azoles such as fluconazole, voriconazole, and itraconazole. This trend necessitates reliance on newer antifungal agents such as echinocandins (e.g., Caspofungin) or polyenes (e.g., Amphotericin B).

**Radiological Findings:** Imaging plays a pivotal role in the evaluation of parotid abscesses, both to confirm the diagnosis and to exclude important differentials. On ultrasound, bacterial and fungal abscesses both appear as hypoechoic or heterogeneously hypoechoic lesions with irregular margins, and thus cannot be reliably distinguished on sonography alone. However, ultrasound remains valuable as a first-line, non-invasive modality to detect fluid collections and guide aspiration.

Contrast-enhanced CT scans provide a more definitive assessment. In parotid abscesses, CT typically reveals a hypodense lesion with peripheral rim enhancement, often extending into both superficial and deep lobes, as in the patient. Importantly, CT scan delineate the size and extent of the abscess, its relationship with adjacent neurovascular structures, and potential spread into deep neck spaces. In addition, imaging is critical to differentiate abscesses from imitators such as neoplasms (benign and malignant salivary gland tumors), lymphadenitis, or obstructive sialolithiasis. While CT cannot specifically distinguish bacterial from fungal abscesses, its role in ruling out alternative pathologies and guiding surgical drainage remains indispensable.

**Microbiological Significance:** Given the non-specific clinical and radiological features, microbiological confirmation is essential in the diagnosis of fungal parotid abscess. Gram staining of aspirated pus can provide an early clue, as in this case, where budding yeast cells were identified. Culture remains the gold standard, allowing not only species identification but also antifungal susceptibility testing. This is of paramount importance, as NAC species frequently demonstrate resistance to azoles, necessitating a tailored therapeutic approach.

In patient, culture revealed Candida non-albicans resistant to fluconazole, voriconazole, and itraconazole, but sensitive to Nystatin and Amphotericin. This highlights the importance of performing sensitivity testing for all suspected fungal pathogens, rather than relying on empirical antifungal therapy alone.

**Management Principles:** The cornerstone of management in parotid abscesses, regardless of etiology, remains timely incision and drainage. This reduces the local infectious load, alleviates symptoms, and prevents further spread into adjacent spaces. Empirical broad-spectrum antibiotics are usually commenced initially, but in cases where fungal elements are suspected or identified, antifungal therapy must be promptly instituted.

In fungal abscesses, antifungal choice should be guided by sensitivity reports. In this case, failure of fluconazole necessitated a switch to intravenous Caspofungin, an echinocandin with strong fungicidal activity against NAC species, combined with oral Nystatin suspension for local control. Duration of antifungal therapy is typically two to three weeks, with close monitoring of clinical and laboratory parameters.

**Clinical Relevance:** Fungal parotid abscesses, though rare, can have significant clinical consequences if not recognized early. The proximity of the parotid gland to the facial nerve places the patient at risk of facial nerve palsy, as documented. Furthermore, untreated infections may extend into deep neck spaces, leading to mediastinitis, sepsis, and potentially fatal systemic candidiasis.

Diabetic patients, like the one presented here, are particularly vulnerable, and clinicians should maintain a high index of suspicion when abscesses fail to respond to standard antibacterial therapy. Early microbiological testing, guided imaging, and multidisciplinary management are essential in preventing complications and ensuring favorable outcomes.

**Novelty of the Case:** The present case is notable for several reasons. Firstly, fungal abscesses of the parotid gland are exceedingly rare, with very few cases reported in the literature worldwide.

Secondly, the causative organism in the case was Candida non-albicans, a group of pathogens increasingly recognized for their resistance to conventional antifungal agents. This necessitated escalation to advanced antifungals such as Caspofungin, underlining the therapeutic challenges posed by NAC infections.

Lastly, this case underscores the importance of integrating clinical, radiological, and microbiological findings in diagnosis and management. It adds to the growing body of evidence that NAC species are emerging pathogens in ENT practice and highlights the need for heightened awareness among clinicians to avoid delays in recognition and treatment.

**Differential Diagnosis:** The presentation of a parotid abscess, particularly with swelling, erythema, pain, and systemic features of infection, is not unique to fungal involvement. Several other conditions may impersonator its clinical and radiological appearance, and thus, differential diagnosis is essential to avoid mismanagement.

**Suppurative Bacterial Abscess:** The most common cause of acute parotid swelling is a bacterial abscess, typically due to Staphylococcus aureus or mixed anaerobes. Patients often present with acute pain, swelling, fever, and tenderness, and pus can sometimes be expressed from Stensen's duct. Radiologically, bacterial abscesses appear as rim-enhancing hypodense lesions on CT and hypoechoic collections on ultrasound, findings that overlap with fungal abscesses. However, bacterial abscesses usually respond well to broad-spectrum antibiotics and drainage. Lack of improvement with conventional therapy should raise suspicion for fungal etiology.

**Lymphadenitis:** Cervical lymphadenitis, particularly when suppurative, can closely resemble a parotid abscess. Tubercular lymphadenitis, common in endemic areas, can present as chronic swelling with intermittent abscess formation. Ultrasound typically shows hypoechoic nodes with central necrosis, while CT may reveal rim-enhancing lymph nodes. Distinguishing lymphadenitis from a parotid abscess relies on careful imaging to assess the epicenter of the lesion and its relationship to the parotid gland, combined with microbiological evaluation of aspirated material.

**Sjögren's Syndrome:** This autoimmune disorder of the salivary glands can impersonator infectious parotid swelling, particularly in its chronic phase when glandular enlargement and recurrent painful swellings are common. Unlike acute abscesses, Sjögren's syndrome is usually bilateral and associated with xerostomia, keratoconjunctivitis sicca, and positive autoantibodies such as anti-Ro and anti-La. Imaging may reveal multiple small cystic changes within the parotid gland, giving a "honeycomb" appearance rather than a discrete abscess cavity.

**Salivary Gland Neoplasms:** Both benign and malignant tumors of the parotid gland may impersonator the clinical features of an abscess. Benign lesions such as pleomorphic adenoma or Warthin's tumor can present as slow-growing, painless swellings but may occasionally undergo cystic degeneration or superinfection, leading to confusion with an abscess. Malignant tumors such as mucoepidermoid carcinoma or adenoid cystic carcinoma may also present as firm, irregular parotid masses. Unlike abscesses, neoplasms typically lack signs of acute infection (fever, elevated inflammatory markers) and are often associated with progressive facial nerve dysfunction. Contrast-enhanced CT and fine-needle aspiration cytology are useful in differentiating neoplastic from infectious etiologies.

**Sialolithiasis:** Obstruction of the salivary duct due to calculi can result in painful parotid swelling, especially during meals. Secondary bacterial infection may lead to abscess formation, representing fungal or bacterial parotitis. Ultrasound can identify ductal or intraparenchymal stones, and CT is highly sensitive for sialoliths greater than 2 mm. In pure obstructive disease, the swelling often fluctuates with salivary stimulation and lacks systemic signs of infection, which helps in distinguishing it from a fungal abscess.

Differentiating fungal parotid abscess from these conditions requires a combination of clinical evaluation, imaging, and microbiological testing. In the patient, the persistence of symptoms despite initial antibiotic therapy and the presence of yeast cells on Gram stain guided the diagnosis toward a fungal etiology.

## Conclusion

Fungal parotid abscess represents a rare but clinically significant entity that demands high clinical suspicion, especially in immunocompromised individuals such as diabetics. While bacterial pathogens remain the predominant cause of parotid infections, clinicians must be aware of the possibility of fungal involvement when patients fail to respond to conventional antibacterial therapy.

Diagnosis relies on a multimodal approach imaging modalities such as ultrasound and contrast-enhanced CT scans playing a vital role in localizing the abscess, determining its extent, and excluding important differential diagnoses, including neoplasms and sialolithiasis. However, imaging findings alone cannot differentiate between bacterial and fungal etiologies. Microbiological

evaluation through Gram stain, culture, and antifungal susceptibility testing is crucial for accurate identification and tailored therapy.

Management of fungal parotid abscess requires timely incision and drainage in combination with appropriate antifungal therapy guided by culture sensitivity. Non-albicans Candida species, as demonstrated in the case, are often resistant to commonly used azoles, necessitating the use of advanced antifungals such as echinocandins or polyenes. A treatment duration of two to three weeks is generally recommended, with close clinical monitoring.

Early recognition and prompt intervention are key to preventing complications such as facial nerve palsy, deep neck space infections, and systemic dissemination. This case highlights the importance of considering fungal parotid abscess in the differential diagnosis of parotid swellings, particularly in high-risk patients. It also adds to the growing body of literature emphasizing the emergence of non-albicans Candida as clinically relevant pathogens in otolaryngology. By combining radiological assessment, microbiological confirmation, and appropriate surgical and medical management, clinicians can significantly improve patient outcomes in this rare but potentially life-threatening condition.

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