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SECTION EDITOR: C. DOUGLAS PHILLIPS, MD

Radiology Quiz Case 1

Artemis Christoforidou, MD; Athanasios Kyrgidis, MD; Konstantinos Markou, MD, PHD; Stefanos Triaridis, MD, PHD; AHEPA University Hospital, Thessaloniki, Greece

A PREVIOUSLY HEALTHY 63-YEAR-OLD WOMAN presented with a 2-year history of gradual, painless enlargement of the right parotid gland. She denied fever, weight loss, or malaise. There was no indication of cough or shortness of breath. There was no complaint of trismus. Physical examination revealed a palpable, mobile mass in the right parotid region. The mass was nontender and nonfluctuant, without obvious inflammatory changes in the overlying skin. There was no evidence of facial nerve paralysis. The findings of examination of the oral cavity, including the Stensen duct orifice, were normal. The results of routine laboratory investigations and radiography of the chest were normal. The patient was admitted for further investigation and treatment of a suspected tumor of the parotid gland. A contrast-enhanced computed tomographic scan of the neck demonstrated a 2.8-cm cystic or necrotic mass confined to the superficial right parotid gland (**Figure 1** and **Figure 2**). The lesion had a relatively well-defined, smooth margin with an enhancing wall of variable thickness. Posteriorly, the wall demonstrated focal thickening and nodular enhancement (Figure 2, arrow). No calcifications were present. There was no extension to the deep lobe, the masticator space, or the overlying skin. The left parotid gland was normal. Mild nonspecific bilateral cervical adenopathy was also identified.

The patient underwent a right superficial parotidectomy with preservation of the facial nerve branches and excision dissection of the upper jugular lymph nodes on the right side. The specimen, which consisted of the superficial lobe of the parotid gland with the lesion and several lymph nodes, was sent for histologic examination.

What is your diagnosis?

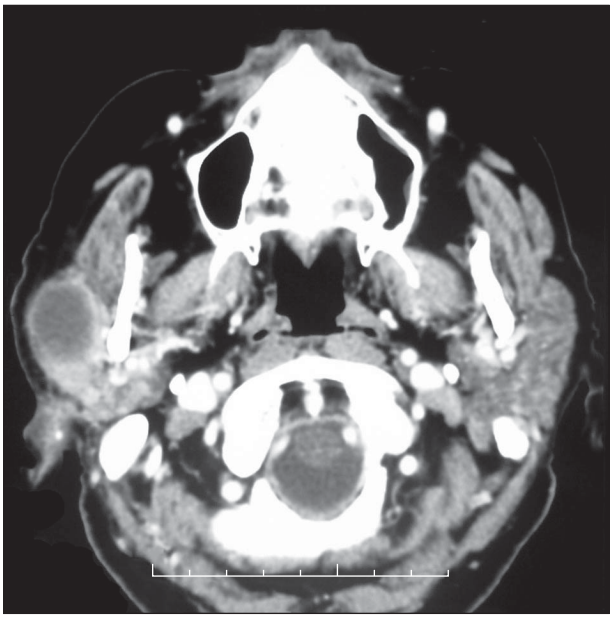

Figure 1.

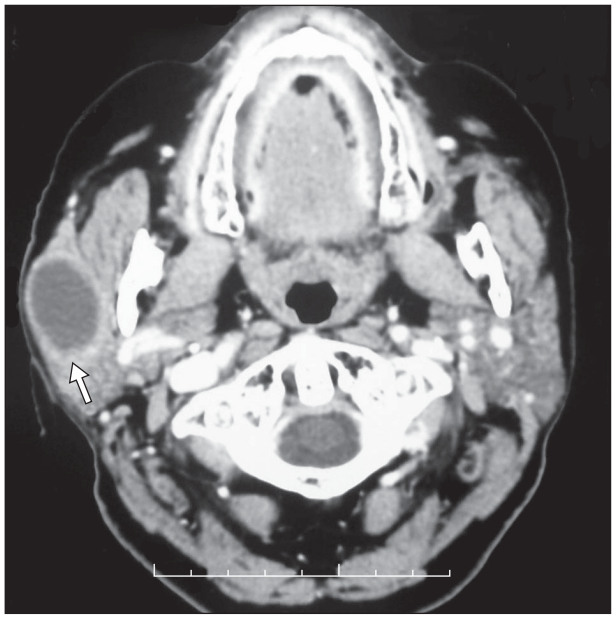

Figure 2.

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Radiology Quiz Case 1: Diagnosis

Diagnosis: Tuberculosis of the right parotid gland

Histologic examination of the specimen revealed multiple granulomas with areas of central necrosis and infiltration by epithelioid cells and numerous Langhans giant cells. The lymph nodes also showed multiple caseating granulomas. Periodic acid–Schiff and Ziehl-Nielsen stains were negative for organisms. The results of a tuberculin skin test were positive, although no history of exposure to tuberculosis was reported. The patient was referred to a pulmonary physician for further treatment and received antituberculous therapy for a year. Five years after surgery, she remained well and asymptomatic.

Tuberculosis of the major salivary glands was first described as an isolated pathologic entity in 1894.[1] Tuberculosis can infect virtually any organ. It is classifed into chronic specific infections caused by *Mycobacterium tuberculosis* and, in rare cases, *Mycobacterium bovis*.[2] It is usually acquired by inhalation of airborne droplets from the cough of a patient with active lung disease or through consumption of contaminated nonpasteurized milk.

The incidence of tuberculosis dramatically decreased over the last century, making its clinical manifestation in the head and neck region extremely rare[3]: however, it increased again in recent years, partly as a result of the ongoing worldwide epidemic of AIDS.[4] Tuberculosis of the salivary gland is rare, even where the disease is endemic, as saliva is reported to have some inhibitory effect on mycobacteria.[1] The most probable routes of infection are considered to be hematogenous and lymphogenous spread as well as retrograde infection through the salivary duct. Most commonly, the lesion has a localized form and is confined to the intraglandular and periglandular lymph nodes in the major glands. In its diffuse form, invasion of the parenchyma is either primary or secondary owing to spread from the nodes themselves.[4] Facial nerve paralysis, pain, and trismus may be present, but they are more indicative of a malignant process.[5] The skin over the lesion is often very thin and shows a bluish discoloration and occasionally fistulization. Therefore, excision of the affected skin is essential.[6]

When affected by the disease, the lymph nodes are initially nontender, either isolated or gathered at groups, with no signs of acute inflammation. At later stages, they fuse and become tender and confluent; they also form a cold abscess and become painful and sensitive on palpation.[1] Our patient did not present with any of the aforementioned typical signs of tuberculosis.

Diagnosis of tuberculosis is considered to be difficult because there are no specific clinical, radiologic, or biologic signs of the disease.[5] The isolation and identification of *M tuberculosis* from a diagnostic specimen are essential for the definitive diagnosis.[2] A thorough examination should be performed, and the patient should be explicitly asked about any history of contact with persons with active tuberculosis. Other investigations should include chest radiography and skin testing. Needle biopsy usually reveals only cellular debris and nonspecific signs of inflammation, whereas little further information is provided by other diagnostic studies, such as sialography of the parotid gland and computed tomography.[7] Often, only gland excision can confirm the diagnosis of parotid gland tuberculosis.

The radiologic features of tuberculous parotitis are not diagnostic. Computed tomograms may show central necrosis of the affected lymph nodes, inflammatory soft-tissue thickening, or even a cystic lesion, as in this case.

Cysts of the parotid gland account for 2% to 5% of all parotid gland lesions.[8] Clinically, it is usually impossible to differentiate between a cyst and other lesions of the parotid gland. Benign cysts of the parotid gland may be congenital or acquired or occasionally arise from surrounding structures. Congenital lesions include branchial cleft cysts, lymphoepithelial lesions, and sebaceous cysts and lymphadenomas. The acquired lesions present after trauma or obstruction of the parotid duct as a result of infection, calculi, or neoplasms; they are also associated with benign lymphoepithelial disease, malignant tumors, tuberculosis, and parasitic infections.[9] Neoplasms with a cystic component vary widely, ranging from entirely benign cystadenomas to cystadenocarcinomas that are capable of metastasis. Other neoplasms that may present with a cystic component include pleomorphic adenoma, Warthin tumor, intraductal papilloma, mucoepidermoid carcinoma, acinic cell neoplasm, and mucinous adenocarcinoma.[10]

Prompt recognition and appropriate referral of tuberculous parotitis are important because a delay in administrating the correct treatment may result in destruction of the gland. Treatment of parotid gland tuberculosis may be medical or surgical, depending on the patient's condition. If treated, tuberculosis of the parotid gland responds well to traditional antituberculous therapy, resolving completely within 1 to 3 months with little or no residual effects. Medical management involves the use of isoniazid, rifampicin, and pyrazinamide. In the atypical disease, medical treatment is normally ineffective but may play a role after suboptimal surgery. The use of clarithromycin, azithromycin, and ethambutol,[4] as well as a combination of a macrolide with a quinolone,[2] has been suggested for this clinical condition. Parotidectomy with facial nerve preservation is the preferred technique when surgical treatment is required,[4] as described in our case. The anatomy of the parotid gland and the fibrotic response to infection complicate surgical treatment of this condition.

In conclusion, the clinician must bear in mind that any atypical ulcer or mass in the head and neck may be of tuberculous origin. A confirmed preoperative diagnosis, followed by effective antituberculous treatment, can lead to resolution of the primary lesion and possibly prevent the need for surgery.[3]

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