Article published online: 2021-07-27

Calcification of the submandibular gland in a patient with chickenpox

Cetin Celenk, Peruze Celenk[1]

Department of Radiology, Faculty of Medicine, [1]Department of Oral and Maxillofacial Radiology, Faculty of Dentistry, Ondokuz Mayis University, Samsun, Turkey

Correspondence: Prof. Cetin Celenk, Department of Radiology, Faculty of Medicine, Ondokuz Mayis University, Kurupelit 55139, Samsun, Turkey. E-mail: ccelenk@hotmail.com

Abstract

The pneumonia virus of chickenpox is now known to cause scattered calcified foci in the lungs, however to our knowledge, recent literature has not discussed calcification in the salivary glands. A 15-year-old boy consulted the department of radiology because of a swelling on the right side of the submandibular area. Radiological assessment included an ultrasonography and computerized tomography scan of the neck area, which demonstrated intraparenchymal amorph calcification, with approximately 13 mm diameter in the right submandibular gland. General condition and oral intake was good without distress in the patient, and hence he was discharged on the seventh day of follow-up treatment.

Key words: Calcification; chickenpox; computerized tomography; submandibular gland; ultrasonography

Introduction

Calcifications may be found in routine and normal radiographs of the major salivary glands. Plain radiography is the simplest, oldest, and cheapest technique for studying the salivary glands.[1] Conventional sialography, ultrasonography (USG), and computed tomography (CT) can detect sialolithiasis with a high degree of sensitivity.[2]

Sialolithiasis accounts for more than 50% of the diseases of the large salivary glands, and is hence the most common cause of acute and chronic infections; however, it remains idiopathic in many instances.[3,4]

To our knowledge, recent literature has not yet reported the calcification of the salivary glands in chickenpox. In patients with chickenpox and those who present with radiological findings of calcification in the major salivary glands, close observation is mandatory for better control of recurrent sialadenitis and early recognition of gland mass.

The purpose of this case report is to present the USG, CT, and clinical features related to calcifications within the submandibular gland of patients with chickenpox.

Case Report

A 15-year-old boy was referred to our hospital because of a 1-year history of swelling in the right submandibular region. He gave a history of pruritic vesicular rash, chest pain, and general malaise 1 year ago. The patient was diagnosed with chickenpox 1 year before the present admission. His medical history was positive for dermatomyositis, diabetes mellitus, and hyperlipidemia, for which he has been on medication

Access this article online	
Quick Response Code:	Website: www.ijri.org
	DOI: 10.4103/0971-3026.202954

Cite this article as: Celenk C, Celenk P. Calcification of the submandibular gland in a patient with chickenpox. Indian J Radiol Imaging 2017;27:49-51.

for the past 7 years. Family history was noncontributory. Initial treatment included antipyretic and antipruritic drugs.

An examination of the right submandibular region revealed that it was swollen and well-circumscribed; a firm mass was palpable. Minimal local tenderness and warmth were noted. His jaw movement was slightly restricted with pain. Intraoral examination was normal. His laboratory findings were the following: White blood cell count, erythrocyte sedimentation rate, C-reactive protein level, and lymphocytes were 10400/mcL, 51 mm/h, 41 mg/L, and 3400/mcL, respectively.

The gray scale ultrasonography showed enlargement, inflamed parenchymal, and calcification in the right submandibular gland [Figure 1]. In addition, the CT scan used for the differential diagnosis showed enlargement, inflammation, and calcification in the gland [Figure 2].

The patient was discharged and given supportive analgesics and advised to ingest fluids and use warm compresses.

Discussion

Plain radiography is the simplest, oldest, and cheapest technique for studying the salivary glands. It is useful in detecting ductal calculi, calcifications (as in hemangioma and lymph nodes), and adjacent osseous lesions.[1] Conventional sialography, USG, and CT can detect sialolithiasis with a high degree of sensitivity.[2] USG is, however, inferior to CT in differentiating a solitary large ductal calculus from a cluster of small calculi. CT is the mainstay of imaging in sialolithiasis.[1] Noncontrast CT may be enough in cases of sialolithiasis. However, ductal system is not optimally evaluated by any of these techniques. These studies are often performed after intravenous injection of the contrast media for better delineation of the anatomy and extent of lesion.[1]

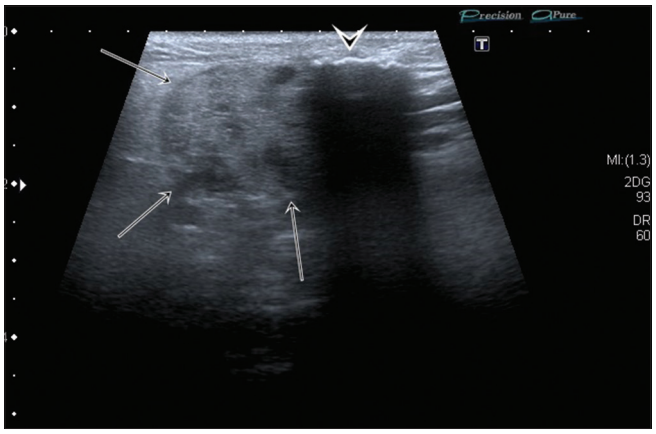

Figure 1: USG image showing acoustic shadow behind hyperechoic calcification (arrowhead) in the inflamed parenchyma of the right submandibular gland (arrows), which appears hypoechoic and inhomogeneous

Calcification, a phenomenon often regarded by pathologists as little more than evidence of cell death, is becoming recognized to be important in the dynamics of a variety of diseases, from which millions of people suffer in all ages.[5]

There are thought to be a series of stages that lead to the formation of calculi in salivary glands. Initially, factors such as abnormalities in calcium metabolism, dehydration, reduced salivary flow rate, altered acidity (pH) of saliva caused by oropharyngeal infections, and altered solubility of crystalloids, which lead to precipitation of mineral salts, are involved.[6] Obstructive salivary gland disease, or obstructive sialadenitis, may also occur due to fibromucinous plugs, duct stenosis, foreign bodies, anatomic variations, or malformations of the duct system. Sialoliths occur in two-thirds of cases of chronic sialadenitis, although obstructive sialadenitis is often a consequence of sialolithiasis.[3,7]

Chickenpox or varicella is an acute infectious disease of childhood caused by varicella-zoster virus (VZV) and belonging to the family Herpesviridae (herpes simplex, varicella-zoster, cytomegalovirus, and Epstein-Barr virus). The primary infection presents as fever and exanthematous rash but can affect almost any organ of the body. The disease is usually acute and self-limited, however, can occasionally lead to complications such as encephalitis, pneumonia, and secondary bacterial infections. However, in the neonatal period and in immunocompromised patients, rare and serious complications may be encountered.[8]

There is a long association between viral infections and diseases of the salivary gland. In general, viral infections of the salivary gland may result in reduced saliva production and/or swelling.[9] However, there is not information available regarding the subclinical salivary gland involvement in the course of VZV infection. It can only be assumed that, were they looked for, more cases of covert salivary gland involvement might be found.

The herpes group of viruses may cause focal cytopathic effects in either the airway or alveoli. Late sequelae of VZV infection consist of multiple 1–2-mm-diameter calcified nodules.[10,11] The herpes group of viruses may cause focal

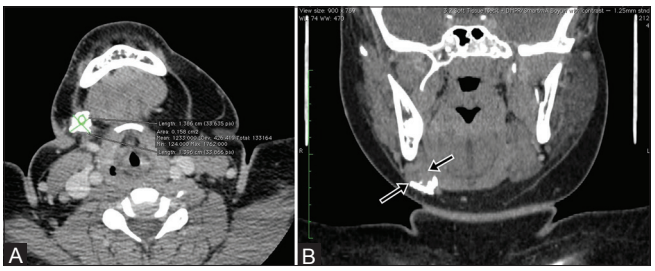

Figure 2 (A and B): Contrast-enhanced CT scan of the neck area (A axial, B coronal slices) showing enlargement, mild increased density, medioinferior amorphous calcification with 13 mm diameters, and 1233 HU density in the right submandibular gland

cytopathic effects in submandibular gland parenchyma. After that, there also occurs edema, congestion, and endothelial damage in small vessels, along with focal hemorrhagic necrosis, mononuclear infiltration, and fibrinous exudates in the gland parenchyma. Late sequelae of VZV infection may consist of multiple 1–2-mm-diameter calcified nodules. Consequently, chickenpox disease may cause calcification in the submandibular gland through the same mechanism in the lungs.

We thought that submandibular gland calcification may also occur by the same pathophysiological mechanisms caused by chickenpox sialadenitis. Through the bloodstream, virutic sialadenitis caused by acute varicella and consisting of endothelial damage in small blood vessels, along with focal hemorrhagic necrosis, results in mononuclear infiltration, fibrinous exudates with macrophages in the gland, and calcifications.

Conclusion

Here, we first presented a discussion of an unusual diagnosis submandibular gland calcification in the chickenpox disease. A description and images are presented, followed by the diagnosis and an explanation of how the diagnosis was determined.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

References

1. Rastogi R, Bhargava S, Mallarajapatna GJ, Singh SK. Pictorial essay: Salivary gland imaging. Indian J Radiol Imaging 2012;22:325-33.
2. Marchal F, Dulguerov P. Sialolithiasis management: The state of the art. Arch Otolaryngol Head Neck Surg 2003;129:951-6.
3. Zenk J, Benzel W, Iro H. New modalities in the management of human sialolithiasis. Minim Invasive Ther 1994;3:275-84.
4. Mimura M, Tanaka N, Ichinose S, Kimijima Y, Amagasa T. Possible etiology of calculi formation in salivary glands: Biophysical analysis of calculus. Med Mol Morphol 2005;38:189-95.
5. Ciftcioglu N, McKay DS. Pathological calcification and replicating calcifying-nanoparticles: General approach and correlation. Pediatr Res 2010;67:490-9.
6. Capaccio P, Torretta S, Ottavian F, Sambataro G, Pignataro L. Modern management of obstructive salivary diseases. Acta Otorhinolaryngol Ital 2007;27:161-72.
7. Rice DH. Advances in diagnosis and management of salivary gland diseases. West J Med 1984;140:238-49.
8. Feldman S, Hughes WT, Daniel CB. Varicella in children with cancer: Seventy-seven cases. Pediatrics 1975;56:388-97.
9. Jeffers L, Webster-Cyriaque JY. Viruses and salivary gland disease (SGD): Lesions from HIV SGD. Adv Dent Res 2011;23:79-83.
10. Nash G, Foley FD. Herpetic infection of the middle and lower respiratory tract. Am J Clin Pathol 1970;54:857-63.
11. Mohsen AH, McKendrick M. Varicella pneumonia in adults. Eur Respir J 2003;21:886-91.