

Case Study

MAGNETIC RESONANCE IMAGING OF INTRAMASSETER HEMANGIOMA. EVALUATION OF TWO CASES

D. Mazza¹, M. Di Girolamo², L. Baggi¹ and F. Cecchetti¹

¹Department of Social Dentistry and Gnathological Rehabilitation, National Institute for Health, Migration and Poverty (NIHMP), Roma, Italy;

²Università of Rome Tor Vergata – Department of Biomedicine and Prevention

Correspondence to:

Dr Dario Mazza, DDS

Department of Social Dentistry and Gnathological Rehabilitation,

National Institute for Health,

Migration and Poverty (NIHMP),

Roma, Italy

e-mail: francesco.cecchetti@inmp.it

ABSTRACT

This article aims to evaluate the Magnetic Resonance Imaging (MRI) semeiotic aspect of an intramuscular hemangioma in the masseter of two patients and review the literature regarding the diagnostic possibilities of MR imaging in interpreting the characteristics of benign and malignant types of this intramuscular lesion in order to ensure a correct diagnosis and treatment. Two patients, aged 29 and 25 years, underwent an MRI examination using a 1.5 Tesla superconducting magnet (Siemens, Erlangen, Germany), with a dedicated surface coil, in one case before and after administering a paramagnetic contrast agent (gadolinium). In both cases, MRI showed the intramuscular haemangioma both with and without the use of the paramagnetic contrast agent. MR images, taken before and after administering the paramagnetic contrast agent, provide important information about the type of intramuscular lesion analyzed, by which it is possible to differentiate a benign mass from a malignant one.

KEYWORDS: *Magnetic Resonance Imaging, MRI, hemangioma, Masseter muscle*

INTRODUCTION

Currently, every benign vascular pathology is referred to as a hemangioma. However, there are vascular pathologies with different evolutions; some have a tendency to regress after the physiological skeletal growth period, while others retain a certain capacity for local infiltrative growth with a tendency to relapse. Vascular and skin lesions were studied by Mulliken et al. in 1982, who provided a classification based on growth data and endothelial characteristics, dividing them into hemangiomas and vascular malformations (1).

Hemangioma is a benign vascular tumor with a particularly rapid growth tendency during the neonatal period, followed by a slow involution phase (2). During the growth of the neoformation, endothelial hyperplasia with multiple laminations of the basement membrane is evident, followed by fibrotic involution and a reduction in the cellular component during the involutionary period.

Vascular malformations are present at birth, grow during the development of the individual, and do not undergo involution; on the contrary, they can increase in size following trauma or hormonal stimuli (2). The head and neck are the regions where vascular malformations occur most frequently, followed by the trunk and limbs. Intramuscular hemangiomas (IMHs) are rare, benign vascular tissue tumors, occurring in approximately 0.8% of all hemangiomas (3-

Received: 12 February 2025

Accepted: 22 March 2025

Copyright © by LAB srl 2025

This publication and/or article is for individual use only and may not be further reproduced without written permission from the copyright holder. Unauthorized reproduction may result in financial and other penalties. Disclosure: All authors report no conflicts of interest relevant to this article.

5). Their recognition is important for the clinician because the location and the evolutionary characteristics might suggest erroneous interpretations of malignancy.

The definitive diagnosis is established only by histological examination. Less than 15% of IMHs occur at a level of the head and neck, where the masseter muscle appears to be the most frequent site of origin, followed by the trapezius and sternocleidomastoid muscles (6-12). Depending on the size of the blood vessels that compose them, there are three categories of hemangiomas: the capillary type, the cavernous type, and the mixed type (3, 4, 6, 12-15). A palpable mass is present in 98% of cases (3, 4, 16); some IMH can pulsate, and it is possible to perceive a noise or a tremble (3).

These lesions are typically studied using ultrasound (US), magnetic resonance imaging (MRI), or computed tomography (CT). Radiological examinations are crucial for evaluating the signs of benignity or malignancy of the lesion, as well as its vascularization and enhancement. The use of diagnostic methods that do not involve ionizing radiation is strongly recommended by the European Dental Radiology guidelines (17). The US is useful for evaluating the presence of IMH in superficial sites. A color-doppler ultrasound provides morphological and vascular information without the risk of ionizing radiation. If an intraosseous or intramuscular hemangioma is suspected, a contrast-enhanced MRI would be the imaging modality of choice and is considered superior to a CT scan (18).

In the maxillofacial region, MRI is the gold standard for the study of temporomandibular disorders (TMD) (19) but is also highly recommended for vascular pathologies because it permits vascularization without a contrast agent, while CT exam with intravenous contrast allows you to evaluate the enhancement.

Peripheral enhancement without progression throughout the mass suggests a lesion that is probably not primarily a vascular entity. Peripheral enhancement progressing to the center (low flow) is typical of venous malformation. In contrast, rapid enhancement throughout the mass characterizes an arterial malformation and, if accompanied by flow voids, an arteriovenous malformation (20). In cases of sudden lesion growth, uncontrollable pain, major functional disorders, necrosis of the overlying skin tissue, thrombocytopenia, or facial deformity, surgical therapy is recommended (9, 21).

The removal of IMH must include the resection of a significant portion of the surrounding muscle tissue, as the infiltrative capacity along the muscle bundles is the primary cause of any relapses (22, 23).

CLINICAL CASES

For the MRI examination, a 1.5 Tesla superconducting magnet and a dedicated surface coil were used. Multiplanar Spin-Echo (SE), Turbo-Spin-Echo (TSE), and Turbo Inversion Recovery Magnitude (TIRM) scans, T1 and T2-weighted sequences have been carried out. To evaluate the enhancement, in one case, 0.2 cc/kg of an intravenous gadolinium-based contrast agent (GBCA) was administered to the patient, and images were acquired using the fat suppression (FS) technique.

Case one

A 25-year-old female presented with swelling in the right masseteric site of taut-elastic consistency without signs of pulsation. The swelling was mobile on superficial levels but not dissociable from the muscle tissue. It was neither painful nor tender but exhibited slight tenderness. There were no signs of ongoing inflammation or local sweating.

The swelling had developed slowly in 3-4 years, with rapid growth in the last 6 months, at the site of a previous intervention of surgical removal of a muscular hernia of the masseter, interpreted clinically as relapse. The swelling deformed the features of the face, especially during chewing, since the contraction of the masseter determined its clear protrusion.

The MRI showed an oval formation (about 3.5 x 2.7 cm), homogeneous and slightly hypointense in T1 compared to the masseter signal; slightly uneven and hyperintense on T2 and homogeneous e markedly hyperintense in T2-weighted TIRM sequences, with slightly lobulated margins, which de-square muscle tissue without infiltrate it; the cortex of mandible appears only slightly thickened compared to the contralateral, but not eroded (Fig. 1).

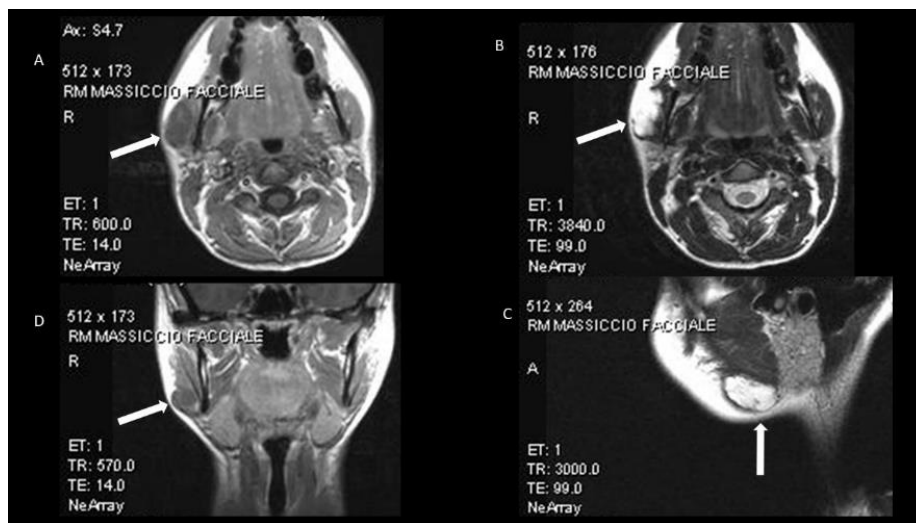


Fig. 1. TSE T1 W image on the axial plane (A), TSE T2 W image on the axial plane (B), TSE T2 W image on the sagittal plane (C), TSE T1 W image on the coronal plane (D). The white arrows indicate the presence of a hemangioma within the masseter muscle.

Case two

A 29-year-old woman with facial asymmetry caused by a swelling that had appeared about 18 months earlier and whose limits were not well demarcated. The patient did not report any painful symptoms, nor were they spontaneous or provoked. An MRI was performed. An oval lesion (about 4 x 1.5cm) with mixed structure non-homogeneous on T1 and non-homogeneous and hyperintense on T2, with slightly lobulated margins, was shown. Compared to the previous case, the lesion presented a greater number of fibrous septa inside and a minor homogeneity of hyperintensity in T2-weighted sequences.

Furthermore, within the lesion, some areas appeared hypointense in both T1 and T2, attributable to phleboliths or thrombosis. After administration of GBCA, the lesion showed the characteristic enhancement of hemangioma (Fig. 2).

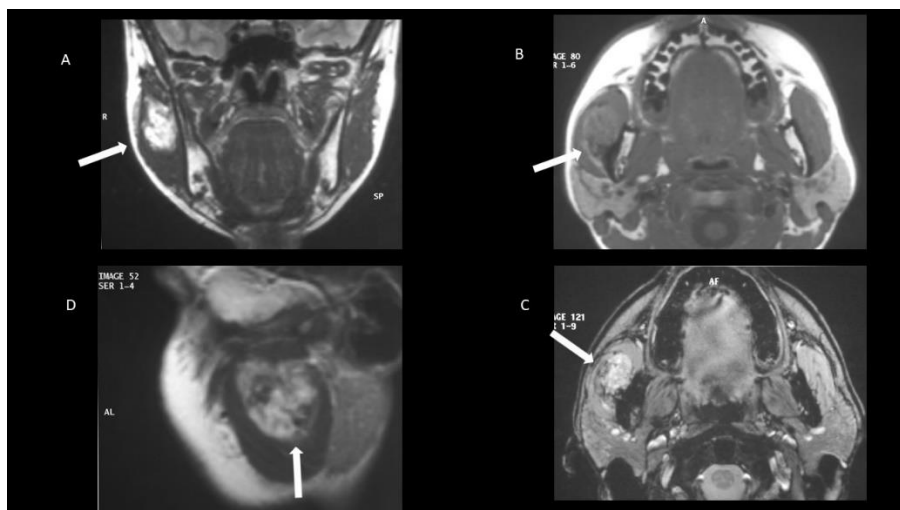


Fig. 2. TSE T2 W image on the coronal plane (A), TSE T1 W image on the axial plane (B), TSE T1 FS W image after GBCA on the axial plane (C), TSE T2 W image on the sagittal plane. The white arrows indicate the presence of a hemangioma within the masseter muscle.

DISCUSSION

Differences between benign and malignant lesions in early MRI studies to evaluate the nature of a mass inside soft tissue were considered insignificant by several authors (24-28).

The impossibility of characterizing the nature of such a mass based on the appearance of the margins and the intensity and homogeneity of the signal was highlighted by Kransford et al. (29). In the majority of the lesions they studied, the signal intensity was similar to that of muscle in the T1-weighted images and equal to or higher than the fat signal in the T2-weighted ones.

On the contrary, there are studies that demonstrate the validity of MRI in differentiating lesions within the soft tissue (30) and in distinguishing between benign and malignant tumors based on the characteristics of the signal obtained (31-33), especially with diffusion-weighted sequences, which enable the characterization of laterocervical lymph nodes (34).

In particular, in the study by Berquist et al. (31), the nature of 95 soft tissue lesions was defined. According to certain parameters considered by them, such as signal homogeneity and the characteristics of the margins, a sensitivity of 94% was achieved. From these results, it can be deduced that, in the majority of cases, a benign lesion would show well-defined margins, homogeneous signal intensity, and no invasion of bone or neuro-vascular structures. On the contrary, a malignant one would appear with irregular margins, nonhomogeneous signal intensity, and a tendency to invade bone and neurovascular structures.

In a study by Teo et al. (35), the presence of small hypointense areas in the context of a non-homogeneous, predominantly hyperintense area is reported as characteristic of a hemangioma, which is attributed to fibro-adipose septa or small clots formed within the vessels. Three radiological signs such as the lobulated shape, the strong enhancement after introduction of GBCA in T1 weighed sequences, and the presence of hypointense areas in T2 weighted sequences, would be sufficient conditions to avoid a biopsy, as they are indicators of a strong probability of a haemangiomatous lesion.

In another study conducted in 2000 by Kern et al. (36), a substantial semilogical similarity between hemangiomas and venous vascular malformations is highlighted. However, it was simple to differentiate an IMH from a lymphatic vessel malformation thanks to the absence of enhancement of the latter. MRI makes it easy and less invasive to diagnose IMH and can be considered the gold standard due to the absence of ionizing radiation. Additionally, with MRI angiography sequences, it is possible to highlight the afferent vascular branches of the lesion (37) even without the use of GBCA. However, CT appears to be more sensitive not only in detecting the presence of endosseous lesions and malformations (38) but also in showing calcifications in the soft tissue context.

Calcifications from phlebolithiasis are characteristic of hemangiomatous lesions, and the presence of this finding in CT, therefore, is highly indicative of IMH (2, 39-42). Such calcifications are typically laminated, with a radiopaque center and a spherical appearance (2, 41); they result from thrombotic formations within vessels, characterized by calcium, phosphate, and apatite deposits that subsequently organize into crystals. In MRI, such calcific findings appear as areas of flow void signal, and they are not easily identifiable in the signal area relating to the examined tissue.

Hyperintensity in T2-weighted sequences indicates the presence of free water in the context of stagnant blood within large vessels, while a low signal suggests the presence of fibro-adipose septa arranged between the vessels. In T1 weighted sequences, the adipose tissue shows a high signal intensity in the context of a hypointense blood area, which later to intravenous GBCA appears hyperintense and contrasts very well in the context of the muscle, which is visible as an area of medium intensity both in T1 and T2 weighted sequences, that easy visualize the IMH and its extension and infiltration in the surrounding tissue.

CONCLUSIONS

MRI can highlight certain morphological and signal features, such as late and persistent contrast enhancement, a lobulated form that does not infiltrate the surrounding tissue, and the presence of phleboliths, which allow for differentiation of these lesions from other malignant pathologies, including squamous cell carcinoma, adenoid cystic carcinoma, and malignant lymphoma. Furthermore, MRI angiographic sequences enable optimal visualization of the lesion and its surrounding vascular branches.

REFERENCES

1. Mulliken JB, Glowacki J. Hemangiomas and vascular malformations in infants and children: a classification based on endothelial characteristics. *Plast Reconstr Surg*. 1982;69(3):412-22. doi: 10.1097/00006534-198203000-00002.
2. Hessel AC, Vora N, Kountakis SE, Chang CY. Vascular lesion of the masseter presenting with phlebolith. *Otolaryngol Head Neck Surg*. 1999;120(4):545-8.
3. Wild AT, Raab P, Krauspe R. Hemangioma of skeletal muscle. *Arch Orthop Trauma Surg*. 2000;120(3-4):139-43. doi: 10.1007/pl00013761.
4. Allen PW, Enzinger FM. Hemangioma of skeletal muscle. An analysis of 89 cases. *Cancer*. 1972;29(1):8-22. doi: 10.1002/1097-0142(197201)29:1<8::aid-cnrcr2820290103>3.0.co;2-a.
5. Günther K, Naumann T, Puhl W. Das infiltrierend wachsende intramuskuläre Hämangiom [Infiltrating intramuscular hemangioma]. *Klin Padiatr*. 1994 Jan-Feb;206(1):59-61. German. doi: 10.1055/s-2008-1046583.
6. Ichimura K, Nibu K, Tanaka T. Essentials of surgical treatment for intramasseteric hemangioma. *Eur Arch Otorhinolaryngol*. 1995;252(3):125-9. doi: 10.1007/BF00178096.
7. Scott JE. Haemangiomas in skeletal muscle. *Br J Surg*. 1957;44(187):496-501. doi: 10.1002/bjs.18004418713.
8. Welsh D, Hengerer AS. The diagnosis and treatment of intramuscular hemangiomas of the masseter muscle. *Am J Otolaryngol*. 1980;1(2):186-90. doi: 10.1016/s0196-0709(80)80014-7.
9. Demir Z, Oktem F, Celebioğlu S. Rare case of intramasseteric cavernous hemangioma in a three-year-old boy: a diagnostic dilemma. *Ann Otol Rhinol Laryngol*. 2004;113(6):455-8. doi: 10.1177/000348940411300607.
10. Odabasi AO, Metin KK, Mutlu C, Başak S, Erpek G. Intramuscular hemangioma of the masseter muscle. *Eur Arch Otorhinolaryngol*. 1999;256(7):366-9. doi: 10.1007/s004050050165.
11. Broniatowski M. Intramuscular hemangiomas of the masseter and sternomastoid muscles. *Ear Nose Throat J*. 1993;72(4):303-5.
12. Heckl S, Aschoff A, Kunze S. Cavernous hemangioma of the temporal muscle. *Neurosurg Rev*. 2002;25(1-2):63-65; discussion 66-7. doi: 10.1007/s101430100181.
13. Beham A, Fletcher CD. Intramuscular angioma: a clinicopathological analysis of 74 cases. *Histopathology*. 1991;18(1):53-9. doi: 10.1111/j.1365-2559.1991.tb00814.x.
14. Cohen EK, Kressel HY, Perosio T, Burk DL Jr, Dalinka MK, Kanal E, Schiebler ML, Fallon MD. MR imaging of soft-tissue hemangiomas: correlation with pathologic findings. *AJR Am J Roentgenol*. 1988 ;150(5):1079-81. doi: 10.2214/ajr.150.5.1079.
15. Jones KG. Cavernous hemangioma of striated muscle; a review of the literature and a report of four cases. *J Bone Joint Surg Am*. 1953;35-A(3):717-28.
16. Morris SJ, Adams H. Case report: paediatric intramuscular haemangiomas--don't overlook the phlebolith! *Br J Radiol*. 1995;68(806):208-11. doi: 10.1259/0007-1285-68-806-208.
17. European Guidelines on Radiation Protection in Dental Radiology – the Safe Use of Radiographs in Dental Practice. *Publications Office*; 2004.
18. Lyssy LA, Puckett Y. Oral Hemangiomas. StatPearls Publishing; 2024.
19. Barchetti F, Stagnitti A, Glorioso M, Al Ansari N, Barchetti G, Pranno N, Montechiarelo S, Pasqualitto E, Sartori A, Marini A, Gigli S, Mazza D, Buonocore V, Marini M. Static and dynamic MR imaging in the evaluation of temporomandibular disorders. *Eur Rev Med Pharmacol Sci*. 2014;18(20):2983-7.
20. Gold L, Nazarian LN, Johar AS, Rao VM. Characterization of maxillofacial soft tissue vascular anomalies by ultrasound and color Doppler imaging: an adjuvant to computed tomography and magnetic resonance imaging. *J Oral Maxillofac Surg*. 2003;61(1):19-31. doi: 10.1053/joms.2003.50003.
21. Avci G, Yim S, Misirlioğlu A, Aköz T, Kartal LK. Intramasseteric hemangioma. *Plast Reconstr Surg*. 2002 ;109(5):1748-50. doi: 10.1097/00006534-200204150-00055.
22. Addante RR, Donovan MG. Right facial mass. *J Oral Maxillofac Surg*. 1994;52(10):1061-5. doi: 10.1016/0278-2391(94)90178-3.
23. Buetow PC, Kransdorf MJ, Moser RP Jr, Jelinek JS, Berrey BH. Radiologic appearance of intramuscular hemangioma with emphasis on MR imaging. *AJR Am J Roentgenol*. 1990;154(3):563-7. doi: 10.2214/ajr.154.3.2154914.
24. Sundaram M, McGuire MH, Herbold DR. Magnetic resonance imaging of soft tissue masses: an evaluation of fifty-three histologically proven tumors. *Magn Reson Imaging*. 1988;6(3):237-48. doi: 10.1016/0730-725x(88)90397-9.
25. Dooms GC, Hricak H, Sollitto RA, Higgins CB. Lipomatous tumors and tumors with fatty component: MR imaging potential and comparison of MR and CT results. *Radiology*. 1985;157(2):479-83. doi: 10.1148/radiology.157.2.4048459.
26. Petasnick JP, Turner DA, Charters JR, Gitelis S, Zacharias CE. Soft-tissue masses of the locomotor system: comparison of MR imaging with CT. *Radiology*. 1986;160(1):125-33. doi: 10.1148/radiology.160.1.3715023.
27. Sundaram M, McGuire MH, Schajowicz F. Soft-tissue masses: histologic basis for decreased signal (short T2) on T2-weighted MR images. *AJR Am J Roentgenol*. 1987;148(6):1247-50. doi: 10.2214/ajr.148.6.1247.

28. Totty WG, Murphy WA, Lee JK. Soft-tissue tumors: *MR imaging. Radiology.* 1986;160(1):135-41. doi: 10.1148/radiology.160.1.3715024.
29. Kransdorf MJ, Jelinek JS, Moser RP Jr, Utz JA, Brower AC, Hudson TM, Berrey BH. Soft-tissue masses: diagnosis using MR imaging. *AJR Am J Roentgenol.* 1989;153(3):541-7. doi: 10.2214/ajr.153.3.541.
30. Mazza D, Taffon C, Scarpato P, Barchetti F, Agrillo A. Atypical localization and atypical magnetic resonance imaging findings of a paraganglioma at the mouth mucosa. *J Craniofac Surg.* 2010;21(2):400-2. doi: 10.1097/SCS.0b013e3181cfa613.
31. Berquist TH, Ehman RL, King BF, Hodgman CG, Ilstrup DM. Value of MR imaging in differentiating benign from malignant soft-tissue masses: study of 95 lesions. *AJR Am J Roentgenol.* 1990;155(6):1251-5. doi: 10.2214/ajr.155.6.2122675.
32. Berquist TH. Magnetic resonance imaging of musculoskeletal neoplasms. *Clin Orthop Relat Res.* 1989;(244):101-18.
33. Hermann G, Abdelwahab IF, Miller TT, Klein MJ, Lewis MM. Tumour and tumour-like conditions of the soft tissue: magnetic resonance imaging features differentiating benign from malignant masses. *Br J Radiol.* 1992;65(769):14-20. doi: 10.1259/0007-1285-65-769-14.
34. Perrone A, Guerri P, Izzo L, D'Angeli I, Sassi S, Mele LL, Marini M, Mazza D, Marini M. Diffusion-weighted MRI in cervical lymph nodes: differentiation between benign and malignant lesions. *Eur J Radiol.* 2011;77(2):281-6. doi: 10.1016/j.ejrad.2009.07.039. Epub 2009 Aug 28.
35. Teo EL, Strouse PJ, Hernandez RJ. MR imaging differentiation of soft-tissue hemangiomas from malignant soft-tissue masses. *AJR Am J Roentgenol.* 2000;174(6):1623-8. doi: 10.2214/ajr.174.6.1741623.
36. Kern S, Niemeyer C, Darge K, Merz C, Laubenberger J, Uhl M. Differentiation of vascular birthmarks by MR imaging. An investigation of hemangiomas, venous and lymphatic malformations. *Acta Radiol.* 2000 ;41(5):453-7. doi: 10.1080/028418500127345677.
37. Cortese A, Letizia N, Gargiulo M, Bergaminelli F, Sica GS. Angiomi del distretto maxillo-facciale: studio clinico con RM ed angio-RM [Angiomas of the maxillofacial area: a clinical study with MR and angio-MR]. *Minerva Stomatol.* 1996;45(9):415-9.
38. Mazza D, Ferraris L, Galluccio G, Cavallini C, Silvestri A. The role of MRI and CT in diagnosis and treatment planning of cherubism: a 13-year follow-up case report. *Eur J Paediatr Dent.* 2013 Mar;14(1):73-6.
39. Smith JF, Drake J, Sollee N. Massive oral hemangioma with phlebolithiasis. *Oral Surg Oral Med Oral Pathol.* 1966;21(1):83-8. doi: 10.1016/0030-4220(66)90018-1.
40. Sano K, Ogawa A, Inokuchi T, Takahashi H, Hisatsune K. Buccal hemangioma with phleboliths. Report of two cases. *Oral Surg Oral Med Oral Pathol.* 1988;65(2):151-6. doi: 10.1016/0030-4220(88)90156-9.
41. Dempsey EF, Murley RS. Vascular malformations simulating salivary disease. *Br J Plast Surg.* 1970 ;23(1):77-84. doi: 10.1016/s0007-1226(70)80015-7.
42. O'Riordan B. Phleboliths and salivary calculi. *Br J Oral Surg.* 1974;12(2):119-31. doi: 10.1016/0007-117x(74)90120-6.