



Review article

Oral varix: a review

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Oral varix: a review

Background: Ageing produces several changes on the oral cavity, and oral varix (OV) is among the most common, and they are related with some medical diseases; however, this association is not clear.

Objective: The aim of this article is to offer a review of OV, regarding aetiology, clinical and histological features, associated factors, treatment and its clinical significance.

Conclusion: Except for a higher incidence of OV in elder individuals, there is limited evidence that supports its relationship with medical conditions such as cardiovascular diseases or portal hypertension. Also, there is no consensus regarding its pathogenesis, but the hemodynamic theory embodies the most comprehensive approach. The high prevalence in elderly people stresses the need for regular oral examination, but more detailed studies regarding OV in relation to systemic diseases are needed.

Keywords: oral varicosities, review, epidemiology, treatment.

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Introduction

In recent years, there has been great advances in the understanding of ageing biology¹. The increase elderly population is real challenge and requires that the dental practitioner is fully aware of prevailing oral changes in this particular age group in order to handle them properly². Oral lesions are strongly age related, as there is an increase in severity and prevalence with increased age³. On the oral mucosa, the ageing process induces a number of gradually and cumulative changes, and among them, Oral Varix (OV) represents a regular finding⁴. An oral venous varix, or varicosity, is common type of acquired vascular malformation⁵. More than a pathology, varix is regarded as a physiological process, usually referred as *conditions* – variation of normal⁶ (WHO), described as common and not hazardous to oral health⁷. This emphasises the need that the general practitioner knows and understands the oral conditions that a senior citizen to properly recognise them.

Varicosities are acquired benign lesions of a vein, artery or lymphatic vessels abnormally dilated and tortuous^{3,8–10}, but within oral cavity typically is only used in reference to venous lesions. In these lesions is usual that a progressive vascular ectasia

with an increase in the diameter, expanding because of hypertrophy, unlike haemangioma that grows by means of hyperplasia¹¹. Grinspan¹² describes them as a tortuous and elevated vascular ectasia.

Varices of the ventral surface of the tongue represents a common oral finding¹³. *Caviar tongue* is a widely used name that has been given to them, given its typical feature of multiple, round little masses of purplish blue colour^{14,15}. However, it has been given several denominations, including phlebectasia linguae^{16,17}, caviar tongue^{14,15}, spots or lesions¹⁸, lingual^{19–22} and sublingual varicosities^{3,23–25}. OV is believed to be a developmental anomaly and is often discovered incidentally during routine oral examination²⁶. Its prevalence increases with age, being described as typical finding in elderly people, resulting of structural alterations, usually deemed insignificant^{16,25,27–29}. However, OV is mentioned in association with several medical diseases³⁰. Glossodynia^{12,30,31} or haemorrhage as a consequence of trauma on varices is rare^{27,28}. The aim of this article is to provide a comprehensive review of OV.

Epidemiology

Varicosities are rare in infants³², however, common in adults³³ and tend to affect both genders

similarly^{23,34}. Nevertheless, a survey with a meaningful sample by Ettinger and Manderson³ ($n = 1751$) states that OV occurs earlier and shows a small tendency in males over all age groups, in contraposition with varix lesions of lower limbs, which are highly more common in women³⁵.

In the oral cavity, ventral aspect and borders of the tongue are by far the most affected localisation and usually they involved the lingual ranine veins^{27,30,31}. Lower lip mucosa is mentioned as the next in occurrence^{16,23,28}, and here, varices are believed to be caused by chronic sun exposure³⁶. Other authors referred floor of the mouth, particularly next to the *ostium* of sublingual glands as the second preferred place^{3,9,22,25,31,37,38}. OV also could be found in other mucosal surfaces, such as buccal mucosa^{3,9,29,31,38}, labial commissures⁹, soft and even hard palate¹⁶.

Clinical features

OV presents predominantly as irregular, blue/purple lesions³⁹, usually multiple with a bilateral lineal distribution from the posterior part to the apex of the tongue¹². Venous varicosities may be seen as round, superficial and tortuous lesions grouped within an area, sometimes following vascular branches (Figs 1–3)^{12,30,31}.

In a normal condition, collateral vessels of the sublingual vein should be of <2.7 mm⁴⁰. Early in its development, OV is of 1–2 mm, but its diameter could reach more than 5 mm, although its height seldom exceeds 2–3 mm^{34,38}. Its colour ranges from a red from an intense blue-purple tonality, changing in relation of deepness and



Figure 1 Typical varicose veins of ventral surfaces of the tongue and floor of mouth, the asymmetry of the lesions should be noted.



Figure 2 Lateral tongue varices, resembling the so-called *caviar tongue*.



Figure 3 Buccal mucosa phlebectasias, along with melanotic macules.

grade of ecstasy of the lesion⁴¹; more superficial lesions are purple, whereas deeper ones could be seen more bluish¹¹. On palpation, lesions may be soft, depressible and usually painless^{27,30}. With diascopy, OV shows blanching, which proves its vascular origin³¹. This is especially useful to establish differential diagnosis with *melanotic or purpuric lesions*, which do not change coloration under pressure, like Osler's syndrome (hereditary haemorrhagic telangiectasia)^{30,38}.

Likewise, it should be distinguished from vascular tumours (commonly called *haemangiomas*), which are congenital malformations that arise at early ages. Also, lingual varices could be confused with lymphangioma, Kaposi's sarcoma, melanoma or other conditions like blue rubber bleb nevus syndrome, but most of these conditions can be differentiated by a thorough history and detailed clinical evaluation⁴².

Vascular Malformations (VM) are congenital lesions that represent errors in morphogenesis, are present at birth and grow proportionately with the child⁴³. Its clinical appearance may resemble OV, but usually the time of onset is enough to safely distinguish between them. However, when the lesion is first seen in adult age, it should be stressed that OV may be located in labial or buccal mucosa, but always compromises ventral surface of the tongue. So, to make a proper differential diagnostic is useful to duly note multiple vascular lesions *including* tongue and other localisations. Besides, oral VM may present combinations of capillary, arterial, venous and lymphatic components⁴³.

Varicosities become progressively more numerous and prominent with age, so they are more likely to be diagnosed mainly on elder individuals^{34,36}. Tortuous and enlarged veins have an ominous look when patients notice them by the first time, and some may suffer cancerophobia¹². If OV is traumatised they may produce minor haemorrhage, although this is an unusual situation^{5,30}. Failure to blanch under pressure could indicate the presence of a thrombus, situation that is not uncommon in long-lasting OV^{5,9,21,25,27,44}. This confers them a firm texture, becoming palpable. Occasionally, small calcifications (phleboliths) arise within the thrombus and could be radiographically identified⁴⁵. Most of the oral varices are usually asymptomatic, so they can be easily missed on clinical examination^{46,47}. However, *Grinspan* states that occasionally, OV could be found in relation to glossodynia^{12,30,31}.

Histopathology

Microscopically, OVs resemble cavernous haemangiomas⁴⁸. They are morphologically composed by one to three extensive and tortuous blood vessels lined by a flat mature endothelium²⁴ lacking a muscular coat⁴⁸, together with scarce connective tissue without angioblastic activity, and no signs of inflammatory alterations^{16,18}. If thrombosis takes place, the vascular lumen contains concentric layers of erythrocytes and platelets, which are called lines of Zahn⁴⁹. In such cases, an intraluminal thrombus develops and may undergo organisation and canalisation by granulation tissue³⁶.

Interestingly, an histological study of sublingual varices found increasing age was associated with an increase in size and number of large vessels (diameter over 240 µm) and also an increase in the amount of fat and elastic tissue in the submucosa²⁴. Oral varix has showed negative staining to

the human erythrocyte-type glucose transporter protein (GLUT-1), which is a discriminant diagnostic method specific of haemangioma¹⁰. Nonetheless, It should be noted that most varicosities are diagnosed clinically and therefore not submitted to biopsy³⁹.

Treatment

In general, OV usually needs no treatment except for reassurance regarding its benign nature^{23,34,37,42}. However, OV localised on sites prone to trauma (e.g. lips or buccal mucosa), or when they are cosmetically objectionable, treatment should be considered. Few modalities of management of OV are described in the literature, and the main types are surgery and intralesional injection of sclerosant agents⁵⁰. Sclerotherapy is an effective, conservative and safe technique for the treatment of OV, and monoethanolamine oleate has been widely used in their management^{8,46}. Others sclerosing agents used are 5% sodium morrhuate, sodium psylliate, quinine urethane, 1% polidocanol, sodium tetradecyl sulphate, ethanol⁵¹ and hypertonic saline^{23,46,52}. The mechanism of action is based on the damage of the endothelium, causing a protein denaturalisation, which induces endothelial damage, thrombus formation, sclerosis, immediate vascular occlusion, consequent inflammation and the subsequent associated fibrosis^{51,53}. Nonetheless, this technique may require multiple sessions and is often associated with slight post-operative discomfort^{8,54}. Surgery can be used exclusively or more often associated with sclerotherapy in lesions that do not show complete resolution. In addition, cryosurgical, electrosurgical, steroid therapy, embolisation and laser therapy has been also used in the management of venous lesions^{48,53,55,56}. Coagulation using laser is based on the high capacity of absorption of light by haemoglobin, which in conjunction with the heat released by the absorption of laser light during the tissue penetration, produces a selective coagulation of the affected blood vessels⁵³. This technique is associated with low complication rates and acceptable post-operative results, but requires a trained operator.

Although there is certain agreement regarding OV multifactorial aetiology, it does not have an only accepted cause, and known associations are detailed in Table 1.

Ageing. A major conundrum in ageing research is attempting to distinguish between gradual effects

Table 1 OV described associations.

Ageing
Venous insufficiency (particularly in relation to lower limb varices)
Portal hypertension
Other associations: chronic vitamin C deficiency, phlebectasias of jejunum and scrotum, superior vena cava syndrome and chronic hepatitis C

of disease states versus true effects of ageing free of pathologic changes⁵⁷. However, there is a broad consensus in the high incidence of oral varicose lesions on elder people^{3,9,12,14–18,20,22–25,27–31,34,36–38,41,44,46,48,58–61}. Percentages given by various authors go between 16.2 and 80% (see Table 2). A recent study shows that in the age group of 40–49, oral varices are found in only 10%, increasing to 72% in the ≥70 years group, suggesting that its prevalence increases as age increases⁶². *Rappaport y Shiffman*¹⁶ pointed that the loss of supportive connective tissue associated with ageing leads to venous dilatation. It has been suggested that when OV is observed prior to the fifth decade, may be an indication of premature ageing^{9,22}.

Venous insufficiency. It has been mentioned that individuals with history of varicose venous on legs may also show similar lesions on tongue^{12,18,27,28,31,37,63,64}. They are caused by a chronic venous insufficiency of the lower limbs, and usually, a family history of varicose veins is found^{65,66}. Although both lingual and leg varices incidence increases with age, *Ettinger y Manderson*³ states that a person with varicose veins of the legs is likely to have lingual varicosities, but the reverse is not necessarily true.

Some authors propose that in an avalvular venous system, such as the tongue, cough could cause an recurrent increase in venous pressure, and this might be an significant factor in varix

aetiology^{14,18}. This could be correlated with anatomical descriptions that state that venous vessels without valves are found mainly in sites where circulation is against gravity⁶⁷, and the brachiocephalic trunk is an avalvular venous system⁶⁸. It should be noted that on lower limbs, variceal formation is caused by sustained elevations on venous pressure, vascular wall weakness or both, which produces vascular dilatation and valvular insufficiency; as a matter of fact, this last one could occur *before* the varix⁶⁹.

Cardiovascular diseases. It has been mentioned OV in relation to cardiopathies^{12,37,63}, as chronic elevations of right-heart pressure may predispose to variceal formation⁷⁰. Specific disorders referred are right-heart and congestive heart failure³⁰ and mitral heart disease¹⁷. Additionally, OV has been linked with cardiopulmonary diseases^{3,12,22} and intense cyanotic emphysemas^{9,17}. This refers to process known as *cor pulmonale* (heart disease of pulmonary origin⁶⁹), which could be produced for a pulmonary emphysema, and one of its main consequences is a pure right-heart failure.

Even so, some authors denied this associations^{23,34} and two independent studies could not demonstrate it either^{3,22}. Hedström and Bergh⁶² found varices were significantly associated with cardiovascular diseases (*p-value* 0.021, OR 2.7), also mentioning another cardiovascular diseases (such as hypertension and myocardial infarction).

Portal hypertension. Sublingual varix has been observed on patients with portal hypertension^{27,28}. It has been proposed that vascular dilatation and tortuousness could be caused by bloodstream increase through a collateral circulatory path to elude the liver obstruction. This could cause oesophageal varices, which could be associated with OV¹². Portal hypertension (PH) secondary to cirrhosis of the liver is the main

Table 2 OV frequency (%).

Authors	Sample size	OV incidence (%)	Age range
Ettinger and Manderson (1974) ³	1751	68.2	7–99
Kaplan (1990) ⁶⁰	298	41.1	Over 50 years
Kignel (1997) ²⁹	Data not available	80	Over 50 years
Mosqueda Taylor <i>et al.</i> (1998) ⁴	100	58	Over 50 years
Kovac-Kovacic & Skaleric (2000) ⁸⁵	1609	16.2	17–75 years
Jainkittivong <i>et al.</i> (2002) ⁸⁶	500	59.6	Over 60 years
Hedström and Bergh (2010) ⁶²	281	35	Over 40 years
Rabiei <i>et al.</i> (2010) ⁸⁴	216	22.7	Over 65 years
Mozafari <i>et al.</i> (2012) ⁸⁷	237	42	Over 60 years

factor leading to the formation of portosystemic collaterals, but the formation of tongue varices is seldom referred associated in association with PH⁷¹. Although oesophageal varices are common manifestations of advanced chronic liver disease causing PH^{69,72,73}, only a few cases of tongue varices in relation with PH have been reported^{70,71,74}, and some authors denied this theory^{23,25}.

*Southman and Ettinger*²⁴ proposed a hemodynamic mechanism for OV development, indicating that an increased arteriovenous blood flow could transmit arterial pressures – much more higher than venous – to the venous part of the circulation, with vein dilatation and secondary morphologic changes in their walls. The negative staining of oral varix to GLUT-1 is coherent with the hypothesis that this lesions result from structural alterations¹⁰.

In addition, OV has been mentioned in relation to chronic vitamin C deficiency^{58,59,75}, phlebectasias of jejunum and scrotum^{16,76}, superior vena cava syndrome^{37,42} and chronic hepatitis C⁷⁷; however, there is no clear evidence to support this associations.

Discussion

As we mentioned, mouth mucosal phlebectasias are found so regularly in older individuals that they have been bestowed with little clinical significance. Its presence is noticed and even could be mentioned to the patient, but its finding is seldom recorded in the medical history. Also, the lower level of awareness of the subjects with tongue lesions may explain the fact that only few of them requested consultation²⁶, mainly because most of the subjects with OV reported no symptoms or were not aware of its existence, they did not seek treatment.

The wide-ranging diversity concerning the epidemiological data of tongue diseases can be explained by the multiple character of sampling, diagnostic criteria and other methods used in different types of examinations^{78,79}. However, it is relevant to consider that in most of the prevalence studies on aged population, no distinction between pathological and non-pathological conditions are performed, and this could induce bias, because some of them included oral varicosities and some did not. Other epidemiological studies directly exclude non-pathological or developmental conditions such as OV, suggesting that its prevalence could be different⁸⁰. For example, Triantos⁸¹ excluded OV because of their high fre-

quency with ageing; Espinoza *et al.*⁸² excludes only sublingual varix, but not varicose veins on other locations. Also, it is noteworthy that there are few studies with demographic data on OV⁴¹. Overall, epidemiological studies show great variation in prevalence and distribution, which might be enhanced by proper classification schemes⁸³.

Standard clinical criteria of oral lesions are needed to avoid variability in epidemiological studies²⁶. With the exception of *Kleinman*²², most of the studies did not offer a well defined and clear concept of how to establish a varix diagnosis. Besides, extension (degree of development) or localisations other than tongue are not addressed, for example, partial or total involvement of ventral surface. Lack of specification about used criteria makes difficult to compare results between studies⁶², which emphasises the need for a proper definition, including minimally localisation and symmetry. Most studies focus only in the presence of sublingual varices without any mention to other locations. Hedström & Bergh⁶² classified sublingual varices in two grades (none or few visible *vs.* medium/severe), which can be a source of bias, and only refers to lesions on the ventral surface of the tongue. Likewise, in studies with large samples, it is not clear which cardiac or pulmonary disease is associated, which hinders conclusions on the possible connection between OV and cardiopulmonary diseases⁶².

Although venous ingurgitation is a well-known cardinal sign of right-sided heart failure, it has been not described together with OV lesions on medical textbooks. Jassar *et al.*⁷⁴ reported a case of base tongue varices in patient with portal hypertension secondary to liver cirrhosis. They mention that in medical literature, there is no recognised anastomosis between lingual venous drainage and portal circulation. Booton and Jacob⁷⁰ described a case of chronic obstructive pulmonary disease with haemoptysis due to tongue base varices, stressing that chronic elevations of right-heart pressure may predispose to variceal formation.

In light of the aforesaid, currently, there is not conclusive data about a potential relationship between OV and some medical conditions mentioned. It is important to stress that there is only general consensus regarding its higher incidence in old ages, but not so with its development mechanism. Therefore, more detailed studies are needed to explain OV association with systemic diseases. The high prevalence of OV in elderly

people stresses the need for regular oral examination of these rapidly expanding age groups⁸⁴. Early diagnosis of medical conditions could be improved by the possibility that through a simple oral examination a proper patient referral could be made. So it is of outmost significance that the dental practitioner knows OV lesions and its possible implications.

References

1. **Partridge L.** The new biology of ageing. *Philos Trans R Soc Lond B Biol Sci* 2010; **365**: 147–54.
2. **Ettinger RL.** The unique oral health needs of an aging population. *Dent Clin North Am* 1997; **41**: 633–49.
3. **Ettinger RL, Manderson RD.** A clinical study of sublingual varices. *Oral Surg Oral Med Oral Pathol* 1974; **38**: 540–5.
4. **Mosqueda Taylor A, Díaz Franco M, Velázquez Alva M, Irigoyen Camacho M, Caballero Sandoval S, Sida Martínez E.** Prevalencia de alteraciones de la mucosa bucal en el adulto mayor. Estudio en dos grupos del sur de la ciudad de México. *Temas Selectos de Investigación Clínica* 1998; **4**: 39–50.
5. **Regezi JA, Sciubba JJ, Jordan RCK.** *Oral Pathology: Clinical Pathologic Correlations*. St. Louis, MO: Saunders, 2003.
6. **Kramer IR, Pindborg JJ, Bezroukov V, Infirri JS.** Guide to epidemiology and diagnosis of oral mucosal diseases and conditions. World Health Organization. *Community Dent Oral Epidemiol* 1980; **8**: 1–26.
7. **Ferreira RC, de Magalhães CS, Moreira AN.** Oral mucosal alterations among the institutionalized elderly in Brazil. *Braz Oral Res* 2010; **24**: 296–302.
8. **Gomes CC, Gomez RS, Do Carmo MA, Castro WH, Gala-García A, Mesquita RA.** Mucosal varicosities: case report treated with monoethanolamine oleate. *Med Oral Patol Oral Cir Bucal* 2006; **11**: E44–6.
9. **Shafer WG, Hine MK, Levy BM, Tomich CE.** *Tratado de patología bucal*. México, D.F.: Nueva Editorial Interamericana, 1986.
10. **Johann ACBR, Salla JT, Gomez RS, de Aguiar MCF, Gontijo B, Mesquita RA.** GLUT-1 in oral benign vascular lesions. *Oral Dis* 2007; **13**: 51–5.
11. **Redondo P.** Classification of vascular anomalies (tumours and malformations). Clinical characteristics and natural history. *An Sist Sanit Navar* 2004; **27** (Suppl 1): 9–25.
12. **Grinspan D.** *Enfermedades de la boca: semiología, patología, clínica y terapéutica de la mucosa bucal*. Buenos Aires: Mundi, 1970.
13. **Pemberton MN.** Sublingual varices are not unusual. *BMJ* 2006; **333**: 202.
14. **Da Costa SM, Cremer G.** Kaviarahnliche Korner unter der Zunge. *Dermatol Wochenschr* 1930; **91**: 1206–9.
15. **Kocsard E, Ofner F, D'Abrera VS.** The histopathology of caviar tongue. Ageing changes of the undersurface of the tongue. *Dermatologica* 1970; **140**: 318–22.
16. **Rappaport I, Shiffman MA.** The significance of oral Angiomas. *Oral Surg Oral Med Oral Pathol* 1964; **17**: 263–70.
17. **Schaffer J.** Clinical pathology of the tongue. *Oral Surg Oral Med Oral Pathol* 1951; **4**: 1287–316.
18. **Bean WB.** *Vascular Spiders and Related Lesions of the Skin*. Springfield, IL: Thomas, 1958.
19. **Burket LW, Lynch MA, Brightman VJ, Greenberg MS.** *Burket's Oral Medicine: Diagnosis and Treatment*, 8th edn. Philadelphia: Lippincott, 1984.
20. **Colby RA, Kerr DA, Robinson HBG.** *Color Atlas of Oral Pathology*. Philadelphia: Lippincott, 1961.
21. **Miller AS, Pullon PA.** Change in lingual varicosities. *Gen Dent* 1985; **33**: 526–8.
22. **Kleinman HZ.** Lingual varicosities. *Oral Surg Oral Med Oral Pathol* 1967; **23**: 546–8.
23. **Bhaskar SN.** Oral lesions in the aged population. A survey of 785 cases. *Geriatrics* 1968; **23**: 137–49.
24. **Southam JC, Ettinger RL.** A histologic study of sublingual varices. *Oral Surg Oral Med Oral Pathol* 1974; **38**: 879–86.
25. **Adler I.** *Atlas fotográfico de estomatología*. Weber Ferro SRL, 1998. Available at: http://www.cfnavarra.es/salud/anales/textos/suple27_1.html (last accessed 22 August 2013).
26. **Darwazeh AMG, Almelaih AA.** Tongue lesions in a Jordanian population. Prevalence, symptoms, subject's knowledge and treatment provided. *Med Oral Patol Oral Cir Bucal* 2011; **16**: e745–9.
27. **Borghelli R.** Temas de Patología Bucal Clínica: con Nociones de Epidemiología Bucal. In: Borghelli R ed. *Temas de Patología Bucal Clínica: con Nociones de Epidemiología Bucal*, 1st edn. Buenos Aires: Mundi, 1979:837–47.
28. **McCarthy PL, Shklar G.** *Diseases of the Oral Mucosa*. Philadelphia: Lea & Febiger, 1980.
29. **Kignel S.** *Diagnóstico bucal*. São Paulo: Robe, 1997.
30. **Grinspan D.** *Enfermedades de la boca: Semiología, patología, clínica y terapéutica de la Mucosa bucal*. Tomo IV. Buenos Aires: Mundi, 1982.
31. **Grinspan D.** *Enfermedades de la boca: Semiología, patología, clínica y terapéutica de la Mucosa bucal*. Tomo III. Buenos Aires: Mundi, 1976.
32. **Ugar-Cankal D, Denizci S, Hocaoglu T.** Prevalence of tongue lesions among Turkish schoolchildren. *Saudi Med J* 2005; **26**: 1962–7.
33. **Nevalainen MJ, Närhi TO, Ainaimo A.** Oral mucosal lesions and oral hygiene habits in the home-living elderly. *J Oral Rehabil* 1997; **24**: 332–7.
34. **Kerr A, Phelan J.** Benign Lesions of the Oral Cavity. In: Greenberg MS, Glick M, Ship JA eds. *Burket's Oral Medicine*, 11th edn. Hamilton, ON: BC Decker Inc, 2008:129–52.
35. **Carpentier PH, Maricq HR, Biro C, Ponçot-Makinen CO, Franco A.** Prevalence, risk factors, and clinical patterns of chronic venous disorders of lower limbs: a population-based study in France. *J Vasc Surg* 2004; **40**: 650–9.
36. **Eversole LR.** *Clinical Outline of Oral Pathology: Diagnosis and Treatment*. Shelton, CT: People's Medical Pub. House-USA, 2011.
37. **Martínez AB, Llanes Menéndez F.** *Medicina bucal*. Madrid: Ediciones Avances Médico-Dentales, 1996.

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38. **Strassburg M, Knolle G.** *Diseases of the Oral Mucosa: A Colour Atlas*, 2nd Revised edn. USA: Quintessence Publishing Co Inc., 1993.
39. **Corrêa L, Frigerio MLMA, Sousa SCOM, Novelli MD.** Oral lesions in elderly population: a biopsy survey using 2250 histopathological records. *Gerodontology* 2006; **23**: 48–54.
40. **Chiu CC, Lan CY, Chang YH.** Objective assessment of blood stasis using computerized inspection of sublingual veins. *Comput Methods Programs Biomed* 2002; **69**: 1–12.
41. **Corrêa PH, Nunes LCC, Johann ACBR, de Aguiar MCF, Gomez RS, Mesquita RA.** Prevalence of oral hemangioma, vascular malformation and varix in a Brazilian population. *Braz Oral Res* 2007; **21**: 40–5.
42. **Viswanath V, Nair S, Chavan N, Torsekar R.** Caviar tongue. *Indian J Dermatol Venereol Leprol* 2011; **77**: 78–9.
43. **Levy C, Mandel L.** Sclerotherapy of intraoral hemangioma. *N Y State Dent J* 2012; **78**: 19–21.
44. **Assi R, Kessler HP, Clark CL.** Oral and maxillofacial pathology case of the month. Varix with phlebolith. *Tex Dent J* 2012; **129**: 684–5, 712–3.
45. **Eversole LR.** *Patología bucal, diagnóstico y tratamiento*. Buenos Aires: Médica-Panamericana, 1983.
46. **Johann ACBR, Aguiar MCF, do Carmo MAV, Gomez RS, Castro WH, Mesquita RA.** Sclerotherapy of benign oral vascular lesion with ethanolamine oleate: an open clinical trial with 30 lesions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2005; **100**: 579–84.
47. **Nasir AGD, McBride G.** Minerva picture case. *BMJ* 2006; **333**: 104.
48. **Eversole LR.** Pigmented Lesions of the Oral Mucosa. In: Greenberg MS, Glick M eds. *Burket's Oral Medicine Diagnosis & Treatment*, 10th edn. Ontario: BC Decker Inc, 2003, 126–36.
49. **Weathers DR, Fine RM.** Thrombosed varix of oral cavity. *Arch Dermatol* 1971; **104**: 427–30.
50. **Mariano FV, Vargas PA, Della Coletta R, Lopes MA.** Sclerotherapy followed by surgery for the treatment of oral hemangioma: a report of two cases. *Gen Dent* 2011; **59**: e121–5.
51. **Talens Ferrando A, Ferrer Mengual S, González-Cruz Soler A, Martínez Sanjuán V, Poveda Roda R, Sanchis Bielsa JM et al.** Alcohol sclerotherapy to treat vascular malformations in the oral cavity. *Radio-logia* 2012. doi: 10.1016/j.rx.2011.10.009.
52. **Schwartz L, Maxwell H.** Sclerotherapy for lower limb telangiectasias. *Cochrane Database Syst Rev* 2011; **12**: CD008826.
53. **Alvarez-Camino J-C, España-Tost A-J, Gay-Escoda C.** Endoluminal sclerosis with diode laser in the treatment of orofacial venous malformations. *Med Oral Patol Oral Cir Bucal* 2013; **18**: e486–90.
54. **Das BK, Hoque S.** Treatment of venous malformations with ethanolamine oleate. *Asian J Surg* 2008; **31**: 220–4.
55. **Bekhor PS.** Long-pulsed Nd:YAG laser treatment of venous lakes: report of a series of 34 cases. *Dermatol Surg* 2006; **32**: 1151–4.
56. **Tal H, Gorsky M.** Cryosurgical treatment of a buccal varix. *J Oral Med* 1987; **42**: 63–5.
57. **Blumenthal HT.** The aging-disease dichotomy: true or false? *J Gerontol A Biol Sci Med Sci* 2003; **58**: 138–45.
58. **Taylor G.** Diet for elderly women. *Lancet* 1966; **1**: 805.
59. **Andrews J, Letcher M, Brook M.** Vitamin C supplementation in the elderly: a 17-month trial in an old persons' home. *Br Med J* 1969; **2**: 416–8.
60. **Kaplan I, Moskona D.** A clinical survey of oral soft tissue lesions in institutionalized geriatric patients in Israel. *Gerodontology* 1990; **9**: 59–62.
61. **Pinzón Tofiño ME, Gaitán Cepeda LA.** Aging and the oral cavity. *Pract Odontol* 1989; **10**: 33–6.
62. **Hedström L, Bergh H.** Sublingual varices in relation to smoking and cardiovascular diseases. *Br J Oral Maxillofac Surg* 2010; **48**: 136–8.
63. **Blanco Carrión A BMA, Llanez Menéndez F.** Capítulo 12: Patología Lingual. In: Bascones Martínez A ed. *Tratado de Odontología*. Tomo III, 2 edn. Madrid: Avances Médico-Dentales, 1998: 619–20.
64. **Ceballos Salobreña A.** *Medicina bucal práctica*. España: Danú Editorial, 2000.
65. **Jawien A.** The influence of environmental factors in chronic venous insufficiency. *Angiology* 2003; **54** (Suppl 1): S19–31.
66. **Sancini A, Tomei G, Schifano MP, Nardone N, Andreozzi G, Scimitto L et al.** Phlebopathies and occupation. *Ann Ig* 2012; **24**: 131–44.
67. **Testut L.** *Tratado de anatomía topográfica: con aplicaciones medicop quirúrgicas*. Tomo 2, 8th edn. Barcelona: Salvat, 1984.
68. **Figun ME, Garino RR.** *Anatomía odontológica funcional y aplicada*, 2nd edn. Buenos Aires: El Ateneo, 1994.
69. **Robbins SL, Kumar V, Cotran RS.** *Robbins and Cotran Pathologic Basis of Disease*. Philadelphia, PA: Saunders/Elsevier, 2010.
70. **Booton R, Jacob BK.** Varicosities of the valliculae: an unusual cause of hemoptysis? *Chest* 2002; **121**: 291–2.
71. **Castiglione U, Curcio M, Salvaggio S, Vancheri F.** Hemoptysis from dorsal tongue base varices secondary to portal hypertension. *Recenti Prog Med* 2001; **92**: 756.
72. **Cecil RL, Goldman L, Ausiello DA.** *Cecil: Tratado de Medicina Interna*. Barcelona: Elsevier España, 2009.
73. **Cheung RC, Cooper S, Keeffe EB.** Endoscopic gastrointestinal manifestations of liver disease. *Gastrointest Endosc Clin N Am* 2001; **11**: 15–44.
74. **Jassar P, Jaramillo M, Nunez DA.** Base of tongue varices associated with portal hypertension. *Postgrad Med J* 2000; **76**: 576–7.
75. **Eddy TP, Taylor GF.** Sublingual varicosities and vitamin C in elderly vegetarians. *Age Ageing* 1977; **6**: 6–13.
76. **James WD, Berger TG, Elston DM.** Disorders of mucous membranes. In: James WD, Berger TG, Elston DM eds. *Andrews' Diseases of the Skin; Clinical Dermatology*, 10th edn. Philadelphia: W.B. Saunders Co, 2006: 803.
77. **de Mattos Camargo Grossmann S, Teixeira R, de Aguiar MCF, de Moura MDG, do Carmo MAV.** Oral mucosal conditions in chronic hepatitis C Brazilian patients: a cross-sectional study. *J Public Health Dent* 2009; **69**: 168–75.
78. **Mathew AL, Pai KM, Sholapurkar AA, Vengal M.** The prevalence of oral mucosal lesions in patients visiting a dental school in Southern India. *Indian J Dent Res* 2008; **19**: 99–103.
79. **Dombi C, Czeglédy A.** Incidence of tongue diseases based on epidemiologic studies (review of the literature). *Fogorv Sz* 1992; **85**: 335–41.
80. **Mujica V, Rivera H, Carrero M.** Prevalence of oral soft tissue lesions in an elderly venezuelan population. *Med Oral Patol Oral Cir Bucal* 2008; **13**: 270.
81. **Triantos D.** Intra-oral findings and general health conditions among institutionalized and non-institutionalized elderly in Greece. *J Oral Pathol Med* 2005; **34**: 577–82.

82. **Espinoza I, Rojas R, Aranda W, Gamonal J.** Prevalence of oral mucosal lesions in elderly people in Santiago, Chile. *J Oral Pathol Med* 2003; **32**: 571–5.
83. **Splieth CH, Sümrig W, Bessel F, John U, Kocher T.** Prevalence of oral mucosal lesions in a representative population. *Quintessence Int* 2007; **38**: 23–9.
84. **Rabiei M, Kasemnezhad E, Masoudi rad H, Shakiba M, Pourkay H.** Prevalence of oral and dental disorders in institutionalised elderly people in Rasht, Iran. *Gerodontology* 2010; **27**: 174–7.
85. **Kovac-Kovacic M, Skaleric U.** The prevalence of oral mucosal lesions in a population in Ljubljana, Slovenia. *J Oral Pathol Med* 2000; **29**: 331–5.
86. **Jainkittivong A, Aneksuk V, Langlais RP.** Oral mucosal conditions in elderly dental patients. *Oral Dis* 2002; **8**: 218–23.
87. **Mozafari PM, Dalirsani Z, Delavarian Z, Amirchaghmaghi M, Shakeri MT, Esfandyari A et al.** Prevalence of oral mucosal lesions in institutionalized elderly people in Mashhad, Northeast Iran. *Gerodontology* 2012; **29**: e930–4.

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