www.jmscr.igmpublication.org Impact Factcor-1.1147
ISSN (e)-2347-176x

IGM Publication Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Vascular Lesions of Oral And Maxillofacial Region

Authors

Dr. Sri Varshini Poojitha Senapathi[1], Dr.Pradeep[2]

Saveetha Dental College and Hospital, Saveetha University, Chennai-77 India.

Email:*rithusena@gmail.com*

ABSTRACT:

Vascular lesions are the most common congenital and neonatal abnormalities.The occurrence of vascular lesion in oral and maxillofacial region causes aesthetic and functional problems . The review summarizes the current classification,recent terminology clinical features and advanced management technique of various vascular lesions.

KEYWORDS: *Haemangioma ,Capillary malformation ,Venous malformation ,Arterial malformation ,Lymphatic malformation.*

INTRODUCTION:

Vascular lesions are the most common congenital and neonatal abnormalities[1].Vascular lesions are derived from blood vessels and lymphatics with different clinical and histological features and treatment also varies[2].More than 50% of these vascular malformations are benign lesions located in the head and neck[3].Vascular lesions can be localized defects in vascular morphogenesis that is usually caused by dysfunction of embryogenesis and vasculogenesis regulator component[4].Various etiology that causes vascular lesion are trauma,infection,hormonal alterations and progressive increase with age[5].They usually occurs in children and young adults with a prevalence in head and neck areas this is true for venous type[6] .In oral cavity the lips ,tongue,cheek mucosa and palate are mostly affected[7] which leads to esthetic changes,pain,ulceration and

bleeding impaired speech ,dental symmetry and obstruction of upper airway[8].Vascular malformation is usuallya defect in capillary,arterial,venous and lymphatic[9].Clinical these lesions appear soft mass or stain that is compressible,non pulsating and has colouration from blue to bluish red[10].This review highlights the current classification ,clinical features,diagnosis and treatment of various vascular lesions in the head and neck region.

CLASSIFICATION:

First hostological classification of vascular lesionwas developed by Virchow and his student Wegener.They classified it as angiomas and lymphangiomas which were then characterized as simplex,cavernosum and racemosis.In 1976 the first international vascualar workshop was performed it was a multidisciplinary workshop.Foundation year of international society for vascular anomalies[11]. .Mulliken and Glowacky classified it based on biological classification of vascular anomalies encompaing ,physical finding, clinical behavior and cellular kinectics[4]. According to the classification approval in rome at the XI workshop on vascular anomalies in 1996[11].

Current classification of haemangioma and vascular malformations[12]

A. Haemangiomas
Superficial (capillary haemangiomaa)
Deep (cavernous haemangiomaa)
Compound (capillary cavernous haemangiomaa)
B. Vascular malformations
Simple lesions
Low-flow lesions
Capillary malformation (capillary haemangiomaa, port-wine staina)
Venous malformation (cavernous haemangiomaa)
Lymphatic malformation (lymphangiomaa, cystic hygromaa)
High-flow lesions
Arterial malformation
Combined lesions
Arteriovenous malformations
Lymphovenous malformations
Other combinations

HAEMANGIOMA:

Haemangioma are usually a single lesion in 80% and 20% of affected children develop multiple tumour[13].Most of the lesions are solitary in 80% of the cases[12]. .Haemangioma usually appear soon after birth and proliferate during the first year of life and involute during the childhood years[4].Superficial haemangioma arise from papillary dermis and present as bright red macular or papular masses previously called as strawberry heamangiomas[12].Deep haemangiomas are located within recticular dermis or subcutaneous tissue and appear as bluish or relatively colourless mass earlier called as cavernous heamangioma[12].Compund haemangiomas are also called as capillary cavernous haemangiomas which has both superficial and deep component[12].

TREATMENT :

Management of haemangiomas can be based on stage of lesion (proliferative or involutive phase) or

type of lesion (superficial,deep,compound) and management of residual deformity[14].Prednisolone is the first line of drug used in life threatening hemangiomas[12].

Proliferative phase:

Superficial haemangiomas can be treated with pulsed dye laser[15].Treatment should be stared at the started at the earliest sign of haemangioma and repeated for 4-6 weekly interval until the lesion is completely cured[16].During the later stages of proliferative haemangioma thickness is acquried that the yellow light laser cannot penetrate.Interstitial potassium-titanyl phosphate(KTP)laser and neodymium:yttrium-aluminum-granet(Nd:YAG)lasers have been used[17].Oral steroids can be given depending on the size and location of lesion as it will shrink the lesion[18].

Involutive phase:

Steroids are not effective against involutingHaemangiomas[12]. Superficial lesions with small and intermediate size vessels can be treated successfully with pulsed dye laser. Larger vessels can be treated with a copper bromide and Nd-YAG laser[19].Resection remains the of treatment for deep haemangiomas[12].In the case of compound lesions, the superficial component should first to treated with by laser photocoagulation and the deep component can subsequently be excised[19]. Intralesional Nd:YAGand argon lasers have been used to treat deep haemangiomas, it can cause substantial pigmentation and scars[20].

Telangiectasia:

The small vessels often present as a red blush and are treated with pulsed dye laser[21]. The medium and large vessels can be treated with copper bromide, pulsed KTP laser or Nd:YAG laser[22].

Fibrofatty tissue:

This is can treated by excision, but can be more vascular than expected. The use of a thermoscalpel and contact laser (Nd:YAG) should be considered[12]. Haemangiomas of the lip and nose almost always leave residual tissue and patients benefit from its careful remova[l23].

Epidermal atrophy :

Skin resurfacing with carbon dioxide laser and Erbium:YAG (Er:YAG) laser seems to be effective and a recent report on the treatment of atrophic scarring in partially or completely involuted haemangiomas in children, there was considerable improvement in almost all patients[24].

CAPILLARY(VENULAR)MALFORMATION

It was previously called port wine stain, are made up of postcapillary venules within the papillary and superficial reticular dermis[25]. They present initially as flat pink macules but they get darken and thicken with age, which gives a cobblestone appearance[12] . Capillary malformations may be

associated with Sturge–Weber and Klippel–Trenaunay syndromes[25].

TREATMENT:

The choice of treatment depends on the degree of diameter of the vessels[13]. Pulsed dye laser is now considered the treatment of choice for capillary malformations, using wavelengths of 577 nm and more recently 585 nm[26]. For vascular lesions the target chromophore is oxyhaemoglobin and selection of a laser wavelength that is preferentially absorbed by oxyhaemoglobin is crucial[27]. Lesions resistant to pulsed dye laser may respond to KTP, copper vapour, argon, and other new lasers[28]. The complications of scarring and hypopigmentation and/or hyperpigmentation are more common with copper and KTP lasers[29].

VENOUS MALFORMATION:

It is characterised by an abnormal collection of veins, which do not have any demonstrable mitotic activity in endothelial or pericyte cells and often lack a uniform smooth muscle layer[13]. The lesions are usually soft, compressible, and enlarge in size when venous pressure is increased[12]. Venous malformations can occasionally be completely intraosseous and the mandible is most commonly involved bone, although maxillary, nasal, and frontal lesions have also been reported[30].

TREATMENT:

The choice of treatment depends on the depth, extent, and anatomical location of the lesion[12]. The methods available are lasers, excision, sclerotherapy, and combinations[12]. Smaller lesions can be treated by excision or sclerotherapy[31]. Acquired adult venous malformations are common in small venous lakes in the lower lip and face and can be treated with laser photocoagulation or excision[32]. Cryotherapy can be sometimes beneficial in the treatment of small intraoral lesions[33]. Nd:YAGlasers form the promising treatment for a superficial lesion or the superficial component of a compound lesion, as the vessels are often large[34]. Larger venous malformations are often managed by a combination of sclerotherapy and excision, or sclerotherapy alone[12]. The commonly used agents are sodium tetradecyl sulphate, 100% ethanol, and Ethibloc[35]. Residual or recurrent lesions can be treated by excision or sclerotherapy[12].

ARTERIOVENOUS MALFORMATION:

Arteriovenous malformations are the least common of the vascular malformations[13].The mid-face, particularly the cheek and ear, are the most common sites[36]. The lesions are often deep, but those close to the surface may produce a palpable thrill or pulsation[12]. Although arteriovenous malformations are present at birth, clinical presentation is usually delayed and lesions may present in the second, third, or even fifth decade[37]. The pathogenesis remains speculative, and it is thought that the primary abnormality or nidus is a bed of dilated capillaries and that the hypertrophied arteries and dilated veins are secondary phenomena resulting from increased flow across the nidus. It is postulated that as the

precapillary sphincter regulates the flow of blood through the capillary bed, abnormalities in control of the sphincter are responsible for arteriovenous malformations and the age of presentation and speed of progression are dependent on the absolute or relative absence of control of the precapillary sphincter[13].

TREATMENT:

The options include embolisation, resection, and combinations[12]. Embolisation is useful for larger arteriovenous malformations and can be used as the sole treatment or as an adjunct. As a adjuvant, it is most commonly used before excision[38]. Because collateral blood supply becomes established quickly, operation should be within 24–48 h after embolisation[39]. Embolisation is used alone as a palliative procedure for unresectable lesions and those that have bled and are likely to do so again[40].

LYMPHATIC MALFORMATION:

Lymphatic malformations constitute a spectrum of disorders that present in various ways, often in childhood and adolescen[41]. Over 75% of lymphatic malformations are found in the neck and the clinical manifestation varies according to the extent and depth of the lesions as well as the extent of fibrous reaction around the[12]. They are subdivided into microcystic, macrocystic, and combined lesions depending on the size of the vesicles[13]. Cervical lesions are usually of the macrocystic variety (*cystic hygroma*) and those involving the floor of the mouth cheek, and tongue are more likely to be of the diffuse microcystic variety[12]. The complications include infection, bleeding, obstruction of the airway, disturbances of speech, and abnormal facial growth[12].

TREATMENT:

Lymphatic malformation are the most difficult to eradicate[12]. Microcystic lesions in particular are diffuse, do not respect tissues planes, and it is difficult to distinguish involved tissue from normal tissue[12]. Macrocystic lesions on the other hand are more localized and respect tissue planes and are more easily excised[12]. The options for treatment include laser ablation, excision, and sclerotherapy[12]. Carbon dioxide laser should be reserved for superficial mucosal lesions[12]. Excision is the preferred method of treatment and localized macrocystic lesions are most easily excised[42]. Postoperative lymphoedema can be a considerable problem, particularly after extirpation of lesions in the floor of the mouth, the resultant oedema and protrusion of the tongue taking up to a year to subside[43]. Sclerotherapy is a promising, but as yet not fully evaluated option[12]. It can be used either as definitive treatment, or for palliation when the morbidity of operation outweighs the benefits. Various agents have been used and recently OK 432 (lyophilised *Streptococcus pyogenes* treated with benzyl penicillin) has been found to be successful in treating macrocystic lesions, with the advantage of avoiding a scar[43].

CONCLUSION:

Vascular malformation are usually unclear to understand the history of the lesion.The

management of these lesion should be done correctly to avoid recurrence.

REFERENCE:

1)Smith JD. Vascular lesion of the head and neck. In: Pediatric Facial Plastic and Reconstructive Surgery. New York: Raven Press, 1993, cap. V, pp. 53, 78.

2) S. C. Nair, N. J. Spencer, K. P. Nayak, K. Balasubramaniam: Surgical management of vascular lesions of the head and neck: a review of 115 cases. Int. J. Oral Maxillofac. Surg. 2011; 40: 577–583,

3) Alesˇ VESNAVER1, David A. DOVSˇ AK. Treatment of vascular lesions in the head and neck using Nd:YAG laser. Journal of Cranio-Maxillofacial Surgery (2006) 34, 17–24.

4) Mulliken JB, Glowacki J: Hemangiomas and vascular malformations in infants and children: A classification based on endothelial cell characteristics. Plast Reconstr Surg 69:412, 1982,

5) Enjolras O, Mulliken JB: Vascular tumours and vascular malformations (new issues). Adv Dermatol 13:375, 1997.

6) Dubois J, Soulez G, Oliva V, et al: Soft-tissue venous malformations in adult patients: Imaging and therapeutic issues. Radio-Graphics 21:1519, 2001,

7) Buckmiller LM: Update on hemangiomas and vascular malformations. Curr Opin Otolaryngol Head Neck Surg 12:476, 2004,

8) Garcia EMD, Jiménez IF, Carrera MST, et al: Hemangiomas y malformaciones vasculares. Qué se puede hacer? Bol Pediatr 41:137, 2001,

9) Boon LM, Mulliken JB, Enjolras O, et al: Glomuvenous malformation (glomangioma) and venous malformation—Distinct clinicopathologic and genetic entities. Arch Dermatol 140:971,2004.

10) Garzon MC, Huang JT, Enjolras O, et al: Vascular malformations—Part. I. J Am Acad Dermatol 56:353, 2007,

11) Filippo Maria Tucci *, Giovanni Carlo De Vincentiis, Emanuela Sitzia, Loanna Giuzio, Marilena Trozzi, Sergio Bottero. Head and neck vascular anomalies in children. International Journal of Pediatric Otorhinolaryngology 73S (2009) S71–S76.

12) M. Ethunandan, Timothy K. Mellor. Haemangiomas and vascular malformations of the maxillofacial region—A review. British Journal of Oral and Maxillofacial Surgery 44 (2006) 263–272.

13) Waner M, Suen JY. *Hemangiomas and vascular malformations of the head and neck.* New York: Wiley-Liss; 1999.

14)Williams III EF, Stanislaw P, Dupree M, Mourtzikos K, Mihm M, Shannon L. Hemangiomas in infants and children: an algorithm for intervention. *Arch Facial Plast Surg* 2000;**2**:103–11.

15) Hohenleutner S, Badur-Ganter E, Landthaler M, Hohenleutner U. Long term results in the treatment of childhood hemangioma with flash lamppumped pulsed dye laser: an evaluation of 617 cases. *Lasers Surg Med* 2001;**28**:273–7.

16) Landthaler M, Hohenleutner U, El-Raheem TA. Laser treatment of childhood hemangiomas. *Br J Dermatol* 1995;**133**:275–81.

17) ClymerMA,Fortune DS, Reinisch L,ToriumiDM,Werkhaven JA, Ries WR. Interstitial

Nd:YAG photocoagulation for vascular malformations and hemangiomas in childhood. *Arch Otolaryngol Head Neck Surg* 1998;**124**:431–6.

18) Bennett ML, Fleischer AB, Chamlin SL, Frieden IJ. Oral corticosteroid use is effective for cutaneous hemangiomas: an evidence-based evaluation. *Arch Dermatol* 2001;**137**:1208–13.

19) Waner M, Suen JY, Dinehart S, Mallory S. Laser photocoagulation of superficial proliferating hemangioms. *J Dermatol Surg Oncol* 1994;**20**:1–4,

20) Burstein FD, Simms C, Cohen SR, Williams JK, Paschal M. Intralesional laser therapy of extensive hemangiomas in 100 consecutive pediatric patients. *Ann Plast Surg* 2000;**44**:188–94.

21) West TB, Alster TS. Comparison of the long pulse dye (590–595 nm) and KTP (532 nm) lasers in the treatment of facial and leg telangiectasias. *Dermatol Surg* 1998;**24**:221–6.

22) Cassuto D, Anacona D, Emanuelli G. Treatment of facial telengiectasias with diode-pumped Nd-YAG laser at 532 nm. *J Cutan Laser Ther* 2000;**2**:141–6.

23) Zide BM, Glat PM, Stile FL, Longaker MT. Vascular lip enlargement: hemangiomas-tenets of therapy. *Plast Reconstr Surg* 1997;**100**:1664–73.

24) Waner M. Laser resurfacing and the treatment of involuting hemangiomas. *Lasers Surg Med* 1996;**40**(Suppl. 8):219.

25) Mulliken JB, Young AE. *Vascular birthmarks. Hemangiomas and malformations*. Philadelphia: Saunders; 1988.

26) Namba Y, Mae O, Ao M. The treatment of port-wine stains with a dye laser: a study of 644 patients. *Scand J Plast Reconstr Surg Hand Surg* 2001;**35**:197–202,.

27) Anderson RR, Parrish JA. Selective photothermolysis: precise microsurgery by selective absorption of pulsed radiation. *Science* 1983;**220**:524–7,

.28) Lanigan SW. Port-wine stains unresponsive to pulsed dye laser: explanations and solutions. *Br J Dermatol* 1998;**139**:173–7.

29) Chowdhury MM, Harris S, Lanigan SW. Potassium titanyl phosphate laser treatment of resistant port-wine stains. *Br J Dermatol* 2001;**144**:814–7,

30) Kaban LB, Mulliken JB.Vascular anomalies of the maxillofacial region. *J Oral Maxillofac Surg* 1986;**44**:203–13.

31) Berenguer B, Burrows PE, Zurakowski D, Mulliken JB. Sclerotherapy of craniofacial venous malformations: complications and results. *Plast Reconstr Surg* 1999;**104**:1–11,

32) Landthaler M, Haina D, Waidelich W, Braun-Falco O. Laser therapy of venous lakes (Bean-Walsh) and telangiectasias. *Plast Reconstr Surg* 1984;**73**:78–83,

33) Gongloff RK. Treatment of intra oral hemangiomas with nitrous oxide cryosurgery. *Oral Surg Oral Med Oral Pathol* 1983;**56**:20–4.

34) Rebeiz E, April MM, Bohigian RK, Shapshay SM. Nd-YAG laser treatment of venous malformations of the head and neck: an update. *Otolaryngol Head Neck Surg* 1991;**105**:655–61.

35) Svendsen P,Wikholm G, Fogdestam I, Naredi S, Eden E. Instillation of alcohol into venous malformations of the head and neck. *Scand J Plast Reconstr Surg Hand Surg* 1994;**28**:279–84.

36) Kahout MP, Hansen M, Pribaz JJ, Mulliken JB. Arteriovenous malformations of the head and neck:

natural history and management. *Plast Reconstr Surg* 1998;**102**:643–54.

37) Jackson IT, Carreno R, Potparic Z, Hussain K. Hemangiomas, vascular malformations, and lymphovenous malformations: classification and methods of treatment. *Plast Reconstr Surg* 1993;**91**:1216–30.

38) Azzzolini A, Bertani A, Riberti C. Superselective embolisation and immediate surgical treatment: our present approach to treatment of vascular hemangiomas of the face. *Ann Plast Surg* 1982;**9**:42–60.

39) Burrows PE, Lasjaunias PL, Ter Brugge KG, Flodmark O. Urgent and emergent embolisation of lesions of the head and neck in children: indications and results. *Pediatrics* 1987;**80**:386–94.

40) Enjolras O, Logeart I, Gelbert F, Lemarchand-Venencie F, Reizine D, Guichard JP, et al. Arterivenous malformations: a study of 200 cases. *Ann Dermatol Venereol* 2000;**127**:17–22.

41) Coffin CM, Dehner LP. Vascular tumors in children and adolescents: A clinicopathological study of 228 tumors in 222 patients. *Pathol Annu* 1993;**28**:97–120.

42) Hancock B, StVilD, Luks F, Dilorenzo M, Blanchard H. Complications of lymphangiomas in children. *J Pediatr Surg* 1992;**27**:220–6.

43) Giguere CM, Bauman NM, Sata Y, Burke DK, Greinwald JH, Pransky S, et al. Treatment of lymphangiomas with OK-432 (Picibanil) sclerotherapy: a prospective multi-institutional trial. *Arch Otolaryngol Head Neck Surg* 2002;**128**:1137.