

Opinion

Recent advances in pathophysiology and management of subglottic Hemangioma

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Subglottic hemangioma is the most common vascular tumor of the larynx of pediatrics; in contrast, it is relatively uncommon, accounting for an estimated 1.5% of congenital laryngeal anomalies [1].

It has similar feature to tumors because it grows independently in the first few months of life, causing symptoms of the air way obstruction. In contrast to tumours it involutes after the first year. Studies suggest that angiotensin converting enzyme and angiotensin II (ATII) receptor are expressed on immature capillaries of proliferating hemangiomas. Increased renin levels observed during the first year of life cause increased conversion of angiotensin to angiotensin I, resulting in increased ATII and proliferation in hemangiomas [2].

True hemangiomas have an erythrocyte-type glucose transporter (GLUT1) that is expressed in the endothelia of blood-tissue barriers. [3], this marker has been validated for the identification of hemangiomas of the airway [4] so that final and definitive diagnosis is mad after lasar or surgical excision of resistant cases. Such new advances may have an essential role to play in management of the hemangioma. For example, Léauté-Labrèze et al. concluded that involution of hemangiomas of infancy may be accelerated with the administration of propranolol [5]. Such great observation has been confirmed by other investigator who concluded that at doses of 2 to3 mg/kg/day used to treat cardiac complications of their hemangiomas, two children experienced marked and rapid involution of their hemangiomas[6]. This finding can be easily explained by above mentioned findings of recent histopathological examination; decreased renin activity induced by betablockers results in decreased ATII and accelerated involution in hemangioma tissue. On the ground of these recent advances, Propranolol dosed at 2–3 mg/kg/day has replaced high-dose steroid therapy as first line pharmacotherapy for airway hemangiomas [7]. Future also can be changed by the availability of new data about factor that induce and inhibit endothelia growth in hemangioma. It was founded that during the proliferative phase of hemangioma many factors are also unregulated including vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), tumor necrosis factor-a (TNF-a), and interleukin-8 (IL-8) [8]. On the other hand there are many factors that increase during the involution phase of such pathology such as tissue inhibitor metalloproteinase (TIMP-1), transforming growth factorb (TGF-b), interferon-a, and platelet factor-4 [9].

Because Interferon is an antiproliferative agent that inhibits angiogenesis by lowering the concentration of angiogenic factors [10], Interferon alpha-2A has been used with success in treating hemangiomas, but should only be considered when traditional modalities fail owing to its complications [11]. So that, from my point of view

it will be of crucial importance to induce more researches that focus on leukotriens and inflammatory mediators to find the strongest inhibitor of growth of hemangioma, this will allow otolaryngologist to avoid surgical approaches, which carry high risk of subglottic stenosis, for grade 1 and some of grade 2 laryngeal hemangioma.

References

1. Holinger PH, Brown WT. Congenital webs, cysts, laryngoceles and other anomalies of the larynx. *Ann Otol Rhinol Laryngol.* 1967; 76: 744-752. **Ref.:** <https://goo.gl/yp7SnN>
2. Itinteang T, Brasch HD, Day DJ, Tan ST. Renin-angiotensin system in hemangioma. Presented at the 18th International Workshop on Vascular Anomalies. 2010.
3. North PE, Waner M, Mizeracki A, Mihm MC Jr. GLUT1: a newly discovered immunohistochemical marker for juvenile hemangiomas. *Hum Pathol.* 2000; 31: 11-22. **Ref.:** <https://goo.gl/byVezL>
4. Badi AN, Kerschner JE, North PE, Drolet BA, Messner A, et al. Histopathologic and immunophenotypic profile of subglottic hemangioma: multicenter study. *Int J Pediatr Otorhinolaryngol.* 2009; 73: 1187-1191. **Ref.:** <https://goo.gl/xc6q3w>
5. Léauté-Labrèze C, Dumas de la Roque E, Hubiche T, Boralevi F, Thambo JB, et al. Propranolol for severe hemangiomas of infancy. *N Engl J Med.* 2008; 358: 2649-2651. **Ref.:** <https://goo.gl/Y4ajtw>
6. Tan ST, Itinteang T, Leadbitter P. Low-dose propranolol for infantile haemangioma. *J Plast Reconstr Aesthet Surg.* 2011; 64: 292-299. **Ref.:** <https://goo.gl/BfKfCN>
7. Denoyelle F, Garabédian EN. Propranolol may become first-line treatment in obstructive subglottic infantile hemangiomas. *Otolaryngol Head Neck Surg.* 2010; 142: 463-464. **Ref.:** <https://goo.gl/HrQ69a>
8. Kråling BM, Razon MJ, Boon LM, Zurakowski D, Seachord C, et al. E-selectin is present in proliferating endothelial cells in human hemangiomas. *Am J Pathol.* 1996; 148: 1181-1191. **Ref.:** <https://goo.gl/tD3fdW>
9. Enjolras O, Mulliken JB. Vascular tumors and vascular malformations (new issues). *Adv Dermatol.* 1997; 13: 375-423. **Ref.:** <https://goo.gl/uP4KeL>
10. Ohlms LA, Jones DT, McGill TJ, Healy GB. Interferon alfa-2a therapy for airway hemangiomas. *Ann Otol Rhinol Laryngol.* 1994; 103: 1-8. **Ref.:** <https://goo.gl/wFzDX4>
11. Ezekowitz RAB, Mulliken JB, Folkman J. Interferon alfa-2a therapy for life-threatening hemangiomas of infancy. *N Engl J Med.* 1992; 326: 1456-1463. **Ref.:** <https://goo.gl/bFmKt2>