Cardiovascular Surgery International

ISSN 2692-7969

Letter to Editor

Vascular Anomalies and Hemangiomas

Panneerselvan N[1] and Jaganathan R[2]*

[1]*Department of Genetics, University of Madras, Dr.ALM.PG.IBMS, Chennai, Tamil Nadu, India*

[2]*Department of Laboratory, University Kuala Lumpur Royal College of Medicine Perak, Malaysia*

Letter to Editor

Vascular anomalies are a spectrum of rare diseases classified as vascular tumors or malformation. Arteriovenous malformations such as capillary and venous make up the majority of vascular malformations; lymphatic malformation are likely to be involved. Vascular anomalies can cause many clinical problems such as disfigurement, chronic pain, recurrent infections, coagulopathies like hemorrhagic and thrombotic, organ dysfunction, and also death in few cases due via growth and expansion of anomalies [1].

Infantile hemangiomas are the most common benign vascular anomalies (vascular tumors) in infants compared with congential hemangiomas [2]. Individuals with vascular anomalies, often experience progressive clinical symptoms with worsening life quality. So it is essential to the observe reduction of size over time to stabilize and help the diagnosis. Unless there is no haemorrhage, these lesions do not enlarge into the tumor. Many types of infantile hemangiomas have been diagnosed till date. Among which, congenital hemangiomas is prevalent and difficult to diagnose earlier, which includes Rapidly Involuting Congenital Hemangiomas (RICH), Partial Involuting Congenital Hemangiomas (PICH) and Non- Involuting Congenital Hemangiomas (NICH) types [3]. Hemangiomas can ulcerate and bleed and can cause transient heart failure and mild coagulopathy in new borns. Currently, depending on the location of the lesions and whether they because functional impairment, the lesions may need to be removed surgically else treated with medicines.

Hemangiomas are associated with conditions related to placental hypoxia and multiple targets of hypoxia are demonstrated in proliferating hemangiomas such as VEGF-A, GLUT1, and IGF-2 [4]. Few other mutations are found to be involved in hemangiomas. Somatic activating mutations of GNAQ and GNA11 have been found to be associated with congenital hemangiomas [5]. Pathogenesis involves abnormal levels of many proangiogenic factors such as b-FGF, VEGF, TGF-beta and also matrix metalloproteinases (MMP-9). Recently, genetic errors in growth factor receptors have also been shown to affect development of hemangiomas [6].

Citation: Panneerselvan N, Jaganathan R. Vascular Anomalies and Hemangiomas. Cardiovasc Surg Int. 2020; 1(1): 1003.

Publisher Name: Medtext Publications LLC

Manuscript compiled: March 04th, 2020

***Corresponding author:** Navina Panneerselvan, Department of Genetics, University of Madras, Dr.ALM.PG.IBMS, Taramani, Chennai, Tamil Nadu, 600113, India, E-mail: navinapanneerselvan@yahoo.com

In the proliferative phase of infantile hemangiomas, Vascular Endothelial Growth Factor (VEGF) and basic Fibroblast Growth Factor (FGF) have shown increased expression. And also vascular endothelial growth factor expression has been up-regulated by adrenergic stimulation. Hemangiomas formation comprises of possibility of both germline risk factor mutations and somatic mutations in case of sporadic conditions, similar to that of venous malformations [7]. Vascular endothelial cells are responsible for the formation and rapid growth of infantile hemangiomas in a negatively controlled signalling VEGF pathway.

Most recent work identifies the role of signaling pathway for vascular endothelial cells via VEGF and extracellular matrix regulations, which acts as the basis for novel therapeutic strategies [8]. To the clinicians, hemangiomas can be difficult to diagnose and might be unfamiliar with these lesions, leading to treatment failure. Hence, researchers are necessary to assess the significance genetic and molecular cause for hemangiomas is highly recommended, as this may aid-in diagnosis and pathophysiology.

References

1. Steiner JE, Drolet BA. Classification of Vascular Anomalies: An Update. Semin Intervent Radiol. 2017;34(3):225-32.
2. PDQ Pediatric Treatment Editorial Board. Childhood Vascular Tumors Treatment (PDQ®): Health Professional Version. Source PDQ Cancer Information Summaries. Bethesda (MD): National Cancer Institute (US); 2002-2019.
3. Brás S, Mendes-Bastos P, Amaro C. Rapidly involuting congenital hemangioma. An Bras Dermatol. 2017;92(6):861-63.
4. Boye E, Olsen BR. Signaling mechanisms in infantile hemangioma. Curr Opin Hematol. 2009;16(3):202-8.
5. Ayturk UM, Couto JA, Hann S, Mulliken JB, Williams KL, Huang AY, et al. Somatic Activating Mutations in GNAQ and GNA11 Are Associated with Congenital Hemangioma. Am J Hum Genet. 2016;98(4):789-95.
6. Calicchio ML, Collins T, Kozakewich HP. Identification of signaling systems in proliferating and involuting phase infantile hemangiomas by genome-wide transcriptional profiling. Am J Pathol. 2009;174(5):1638-49.
7. Richter GT, Friedman AB. Hemangiomas and vascular malformations: current theory and management. Int J Pediatr. 2012.
8. Ames JA, Sykes JM. Current trends in medical management of infantile hemangiomas. Current opinion in otolaryngology & head and neck surgery. 2015;23(4):286-91.