The Role of Transcortical Vessels in Diaphyseal Vascularity

Sir,

A recent article published in Nature Metabolism entitled, "A network of trans-cortical capillaries as a mainstay for blood circulation in long bones" by Anja Hasenberg (2019), is an excellent description of the bone-vascular network. The discovery of transcortical vessels (TCVs) in long bones has witnessed the enigma of bone vascularity.[1]

The long bone has a thick cortical shell in human with well-developed Haversian system. This system is absent in bone of children and at fracture sites leading to the soft or immature bone. The medullary and vascular networks in rodents or small mammals are in series, but in human, both vascular networks are parallel except in children and immature bone of fracture sites.[2] Throughout the cortex of the long bone, there are cortical capillary networks housed in small canals. The transcortical nourishment of bone takes place by cortical capillaries of Volkmann's canal, and longitudinal irrigation occurs through Haversian vessels. After the conversion of red-to-yellow marrow in the diaphysis, cortical supply shifts from the cortical capillaries of medullary circulation to periosteal vascular plexus.[3]

Several modern imaging technologies, e.g., light-sheet fluorescence microscopy and X-ray microscopy, revealed TCVs along the entire bone shaft that cross the cortical bone perpendicularly and form a direct connection between the endosteal and the periosteal circulation in compact bone. The authors have shown bleeding spot on the external surface of the human tibia and femoral neck and claimed them TCVs. They demonstrated that the source of bleeding after periosteal elevation was TCVs not from periosteal vascular bed or cortical capillaries.[1] These TCVs are the mainstay of vascularity of femoral neck in place of retincular vessels from the metaphyseal vascular ring. However, they did not discuss the difference between the retincular vessels and TCVs.

A normal synovial membrane, especially its superficial layers, is richly vascularized to meet the metabolic needs of the avascular cartilage. The authors depicted an increased density of TCVs in chronic inflammatory arthritis in a mice model. These TCVs lined by endothelial cells are sites for engagement of inflammatory cells. TCVs connect bone marrow to the synovium and play an active role in murine arthritis models similar to rheumatoid arthritis (RA).[1] The increased vascularity of the synovium is the indisputable sign of the invasive synovial tissue (joint pannus); it results from angiogenesis, the formation of new capillaries from immature vessels (now named as TCVs) under the influence of many chemokines, including the vascular endothelial growth factor.[4] However, in RA, the TCVs are redistributed and densely packed in the deeper layers of the synovium which worsen the hypoxia and flares anaerobic processes in the chronically inflamed synovium. The new TCV angiogenesis impairs the cartilage, adversely affecting its biomechanical properties.[5]

They reported the significance of the TCVs in angiogenesis, joint pannus, and bone remodeling, but more definitive studies on inflamed and damaged human bone and joint tissues are required to confirm these observations. Future work needs to address not only the dynamics of interaction osteoclasts and osteocytes with TCVs but also its bidirectional regulation of trafficking of cells, the vascular niche of hemopoietic cells, and insoluble mediators for bone remodeling.

Financial support and sponsorship

Nil.

Conflicts for interest

There are no conflicts for interest.

Adil Asghar

Department of Anatomy, AIIMS, Patna, Bihar, India

Address for correspondence:
Dr. Adil Asghar,
Department of Anatomy, AIIMS, Patna, Bihar, India.
E-mail: dradilasghar2009@gmail.com

References

1. Grüneboom A, Hawwari I, Weidner D, Culemann S, Müller S, Henneberg S, *et al*. A network of trans-cortical capillaries as mainstay for blood circulation in long bones. Nature Metabolism. 2019;1:236-50. [https://doi.org/10.1038/s42255-018-0016-5]
2. Cowin SC, Cardoso L. Blood and interstitial flow in the hierarchical pore space architecture of bone tissue. J Biomech 2015;48:842-54.
3. Gurevitch O, Slavin S, Feldman AG. Conversion of red bone marrow into yellow – Cause and mechanisms. Med Hypotheses 2007;69:531-6.

4. Walsch DA. Angiogenesis and arthritis. Rheumatology 1999;38:103-12.
5. Sudoł-Szopińska I, Kontny E, Maśliński W, Prochorec-Sobieszek M, Kwiatkowska B, Zaniewicz-Kaniewska K, *et al.* The pathogenesis of rheumatoid arthritis in radiological studies. Part I: Formation of inflammatory infiltrates within the synovial membrane. J Ultrason 2012;12:202-13.

Access this article online

Quick Response Code:

Website: www.ijoonline.com

DOI: 10.4103/ortho.IJOrtho_276_19

How to cite this article: Asghar A. The role of transcortical vessels in diaphyseal vascularity. Indian J Orthop 2019;53:785-6.