

Idiopathic digital clubbing

John Shneerson¹

Author details can be found
at the end of this article

Correspondence to:

John Shneerson;
john@shneerson.org.uk

Sir,

The interesting case report by Awadallah and Dubrey (<https://doi.org/10.12968/hmed.2019.0417>) continues the search for the ‘elusive’ unifying mechanism of digital clubbing. One feature that is always present is the increase in vascular connective tissue at the tip of the digits presumably, as the authors state, as a result of increased levels of platelet-derived growth factor and vascular endothelial growth factor. Beyond this it is more complex. Blood in the systemic circulation is shunted across the lungs in most of the causes of clubbing and bypasses their normal filtering function. Exactly how this relates to the actions of platelet-derived growth factor and vascular endothelial growth factor remains unknown.

The authors mention hypertrophic osteoarthropathy as a cause of clubbing but this is a distinct condition in which, although clubbing is often gross, the periosteal and joint changes predominate. These regress after vagotomy, indicating that a neural mechanism, probably afferent, is essential (Shneerson, 1981). Hypertrophic osteoarthropathy differs from idiopathic clubbing described in this case, which is likely to be the result of a genetic hypersensitivity to platelet-derived growth factor and vascular endothelial growth factor in the microcirculation in the digits.

Author details

¹Royal Papworth Hospital, Cambridge, UK

Reference

Shneerson JM. Digital clubbing and hypertrophic osteoarthropathy: The underlying mechanisms. *Br J Dis Chest*. 1981;75(2):113–131. [https://doi.org/10.1016/0007-0971\(81\)90043-7](https://doi.org/10.1016/0007-0971(81)90043-7)

How to cite this article:

Shneerson J. Idiopathic digital clubbing. *Br J Hosp Med*. 2020. <https://doi.org/10.12968/hmed.2020.0687>