Adventitial *Vasa Vasorum*: A Potential Therapeutic Target But Yet a Long Way to Go

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To the Editor: I reviewed the article *Quantification of adventitial vasa vasorum (VV) vascularization in double-injury restenotic arteries* by Meng *et al*.[1] with great interest, which described their experiment about adventitial VV and postangioplasty restenosis. Percutaneous transluminal angioplasty is now widely used for the treatment of atherosclerotic lesions in various arteries. Restenosis is a major concern following angioplasty, and yet with no good solutions. Previous experimental studies suggested that there is an association between VV and atherosclerotic plaque formation and intimal hyperplasia. As occlusion of VV correlates well with neointima formation, but inhibition of VV neovascularization correlates with reduced plaque growth,[2] VV may play several different roles in different phases of atherosclerosis or postangioplasty restenosis. The causative mechanism is still unclear.

Meng *et al*.[1] described the postangioplasty change of VV in an animal experiment, and reported that double-injured arteries had a greater number of VV, increased luminal surface, an elevation in the intima/media ratio, an accumulation of macrophages and smooth muscle cells in the intima, but decreased VV density, as compared to sham or single-injured arteries. While a previous study showed similar results, but increased VV density after angioplasty,[3] the differences in VV density may be due to the different stages of restenosis as the authors argued. In the Meng's study, low VV density was observed at an early phase of restenosis just 4 weeks after angioplasty, as indicated by a lack of severe intimal hyperplasia, which is not long enough for a complete remodeling of the injured area. Lower VV density with increased sheer VV number implies that intimal growth surpassed the VV neovascularization. Low VV density correlates with hypoxia, oxidative stress and microinflammation,[2] thus may lead to intimal growth and further VV neovascularization. On the other hand, it is also possible that the low VV density is caused by intimal hyperplasia, and further VV neovascularization is induced by hypoxia and oxidative stress. VV neovascularization can not only serve as a compensatory mechanism for the delivery of more oxygen and nutrients to the injured vessel walls, but also function as a conduit for macrophages or inflammatory factor infiltration that promotes restenosis. Anyhow, all we have now are all associational evidence, the full mechanism and the real causative role of VV in different phases of atherosclerosis or restenosis is not yet known. Meng *et al*. provided interesting new evidence on VV function, suggesting that modifying VV growth could provide therapeutic effects. However, before we consider using VV as a therapeutic target, further mechanism studies on the dynamic change of VV and other factors after angioplasty are needed.

References

1. Ye M, Zhang BG, Zhang L, Xie H, Zhang H. Quantification of adventitial *vasa vasorum* vascularization in double-injury restenotic arteries. Chin Med J 2015;128:2090-6.
2. Gössl M, Versari D, Lerman LO, Chade AR, Beighley PE, Erbel R, *et al.* Low vasa vasorum densities correlate with inflammation and subintimal thickening: Potential role in location – Determination of atherogenesis. Atherosclerosis 2009;206:362-8.
3. Kwon HM, Sangiorgi G, Ritman EL, Lerman A, McKenna C, Virmani R, *et al.* Adventitial vasa vasorum in balloon-injured coronary arteries: Visualization and quantitation by a microscopic three-dimensional computed tomography technique. J Am Coll Cardiol 1998;32:2072-9.

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Access this article online

Quick Response Code:

Website: www.cmj.org

DOI: 10.4103/0366-6999.161404

Received: 22-03-2015 **Edited by:** Yuan-Yuan Ji
How to cite this article: Hua SR, Liu CW, Liu B. Adventitial *Vasa Vasorum*: A Potential Therapeutic Target But Yet a Long Way to Go. Chin Med J 2015;128:2130.

Source of Support: Nil. **Conflict of Interest:** None declared.