

[F-PO1572] Cellular Infiltration and Proliferation in Arteriovenous Fistula Failure

Mahesh Krishnamoorthy, Michelle Kurian, Yang Wang, Rao Khan, Timmy Lee, Mahmoud El-Khatib, Rupak Banerjee, Rino Munda, Lois Arend, Prabir Roy-Chaudhury Cincinnati Dialysis Access Program, University of Cincinnati, Cincinnati, OH

Early and late arteriovenous fistula (AVF) failure as a result of venous segment stenosis is a growing clinical problem. Despite the magnitude of the clinical problem the exact pathogenesis of AVF failure remains unclear and this could be the reason for the current lack of effective therapies. The aim of this study was to describe the pattern of macrophage infiltration and cellular proliferation in stenotic venous segment tissue from dialysis patients with AVF failure.

Venous segment tissue was obtained from 17 patients with AV fistula stenosis at the time of revision surgery. Formalin fixed paraffin embedded specimens were assessed for luminal stenosis using standard morphometric techniques (Image J). Macrophage infiltration (PGM-1;1:200) and cellular proliferation (PCNA; 1:500) were analyzed using a standard streptavidin biotin immunohistochemical technique. The endothelium, intima-media and adventitia of each specimen was assessed separately using a semi-quantitative 0-4+ scoring scale (0 = 0-10% of total nucleated cells in that region; 1+ = 11-25%, 2+ = 26-50%, 3+ = 51-75%, 4+ = 76-100%). An unpaired t test was used to identify differences between staining patterns.

Cellular infiltration and proliferation scores were maximal within the intima-media (Macrophage = 1.9+/-0.7; PCNA = 1.7+/-0.2), followed by the adventitia (macrophage = 0.72+/-0.2; PCNA = 1.1+/-0.2) and endothelium (macrophage = 0.1+/-0.1; PCNA = 0.9+/-0.3; note the minimal macrophage infiltration at this site). Cellular proliferation scores were significantly higher as compared to macrophage infiltration scores within the endothelium (p=0.02) and intima-media (p=0.04) but not the adventitia.

Our results describe suggest that there is a differential pattern of macrophage infiltration and cellular proliferation within different layers of the stenotic venous segment. Preferential targeting of the adventitia and intima-media with anti-proliferative/anti-macrophage therapies could play a role in reducing future AVF stenoses.

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Poster Session: Dialysis: Hemodialysis/Vascular Access: Fistulas and Grafts (10:00 AM-12:00 PM) Poster Board Number: F-PO1572

Location: Exhibit Halls A/B/C

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