【Outstanding Academic Research Meeting II-3】

Novel Mechanisms of Neointimal Hyperplasia in Arteriovenous Fistulae for Hemodialysis Access

透析血管內膜增生的新機制

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Hemodialysis vascular access failure is a major problem for end stage renal disease (ESRD) patients. Arteriovenous fistulas (AVFs) have lower thrombosis rates but high dysfunction rates due to neointimal hyperplasia and venous stenosis. Percutaneous transluminal angioplasty (PTA) can temporarily treat stenotic lesions but restenosis rates are 36-62% at 6 months. Rapid cell proliferation drives neointimal hyperplasia (NH) leading to stenosis/restenosis. Degradation of the internal elastic lamina, potentially due to flow induced elastinolytic enzyme expression, may also promote NH. Marked inflammation in the vascular wall post-AVF thrombosis likely contributes to the high failure rate after thrombectomy. Repeated AVF puncture for dialysis causes vascular dilation but not necessarily stenosis. Disturbed blood flow at the AVF juxta-anastomotic area induces endothelial-mesenchymal transition (EndMT) via the osteopontin/CD44 pathway, which also dives NH and stenosis in AVF. Targeting this pathway could help prevent AVF dysfunction. In essence, disturbed blood flow activates cellular pathways that lead to vascular access failure in hemodialysis AVFs, highlighting potential therapeutic targets.