

25. Bhattacharya R, Senbanerjee S, Lin Z, Mir S, Hamik A, Wang P, Mukherjee P, Mukhopadhyay D, Jain MK. Inhibition of VPF/VEGF-mediated angiogenesis by the Kruppel-like factor KLF2. *J Biol Chem* 2005;**280**:28848–28851.
26. Kawanami D, Mahabeleshwar GH, Lin Z, Atkins GB, Hamik A, Halder SM, Maemura K, LaManna JC, Jain MK. Kruppel-like factor 2 inhibits hypoxia-inducible factor 1{alpha} expression and function in the endothelium. *J Biol Chem* 2009;**284**: 20522–20530.
27. Urbich C, Dimmeler S. Endothelial progenitor cells: characterization and role in vascular biology. *Circ Res* 2004;**95**:343–353.
28. Kumar A, Lin Z, Senbanerjee S, Jain MK. Tumor necrosis factor alpha-mediated reduction of KLF2 is due to inhibition of MEF2 by NF- κ B and histone deacetylases. *Mol Cell Biol* 2005;**25**:5893–5903.
29. Kucharczak J, Simmons MJ, Fan Y, Gelinas C. To be, or not to be: NF- κ B is the answer—role of Rel/NF- κ B in the regulation of apoptosis. *Oncogene* 2003;**22**:8961–8982.
30. Das H, Kumar A, Lin Z, Patino WD, Hwang PM, Feinberg MW, Majumder PK, Jain MK. Kruppel-like factor 2 (KLF2) regulates proinflammatory activation of monocytes. *Proc Natl Acad Sci USA* 2006;**103**:6653–6658.

CARDIOVASCULAR FLASHLIGHT

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Subtotal aortic occlusion due to dehiscence of a supracoronary graft 10 years after surgery for acute type A dissection

Maximilian Y. Emmert^{1*}, Markus J. Wilhelm¹, Christian Felix², and Volkmar Falk¹

¹Clinic for Cardiovascular Surgery, University Hospital Zurich, Raemi Street 100, 8091 Zurich, Switzerland and ²Institute for Anesthesiology, University Hospital Zurich, Zurich, Switzerland

* Corresponding author. Tel: +41 442553298, Fax: +41 442554446, Email: maximilian.emmert@usz.ch

A 55-year-old male patient was referred with a 1-month history of dyspnoea NYHA III and chest pain, 10 years after aortic replacement due to acute type A dissection. Angiography displayed an abnormal configuration of the supracoronary graft (Panel A, arrow), and invasive blood pressure monitoring revealed a peak-to-peak gradient of 180 mmHg confirming a severe stenosis in the ascending aorta. The computed tomography (CT) scan revealed proximal anastomotic dehiscence with a severe subtotal aortic stenosis (Panel B). A blind lumen of the supracoronary graft was identified (Panel B, white arrow), and perfusion appeared to be maintained only by a small residual lumen (Panel B, black arrow). The patient was scheduled for urgent redo aortic surgery. Cardiopulmonary bypass was initiated via the right subclavian artery and the right femoral vein, before re-sternotomy was performed. The ascending aorta, aortic arch, and descending aorta were explored in circulatory arrest with antegrade cerebral perfusion. The graft was found to be detached at the proximal anastomosis, involving approximately half of the circumference (Panels C and D, white arrow). As a result, the graft protruded into the lumen (Panels C and D, white arrow), building a blind sac and occluding the ascending aorta almost completely, with only a small lumen remaining (Panels B and D, black arrow). The aortic root, ascending aorta, and aortic arch were replaced by a conduit (Panel E). A re-entry was found in the proximal descending aorta (Panel F, arrow) which required additional graft replacement of the descending aorta. This case highlights that a regular follow-up including CT scan is mandatory after acute aortic surgery.

