Simoons ML. Platelet glycoprotein IIb/IIIa inhibitors in acute coronary syndromes: a meta-analysis of all major randomised clinical trials. *Lancet* 2002;**359**:189–198.
15. Lim MJ, Eagle KA, Gore JM, Anderson FA Jr, Dabbous OH, Mehta RH, Granger CB, Fox KA, Spencer FA, Goldberg RJ. Treating patients with acute coronary syndromes with aggressive antiplatelet therapy (from the Global Registry of Acute Coronary Events). *Am J Cardiol* 2005;**96**:917–921.
16. Alexander KP, Chen AY, Newby LK, Schwartz JB, Redberg RF, Hochman JS, Roe MT, Gibler WB, Ohman EM, Peterson ED. Sex differences in major bleeding with glycoprotein IIb/IIIa inhibitors: results from the CRUSADE (Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation of the ACC/AHA guidelines) initiative. *Circulation* 2006; **114**:1380–1387.
17. Rao SV, O'Grady K, Pieper KS, Granger CB, Newby LK, Van de Werf F, Mahaffey KW, Califf RM, Harrington RA. Impact of bleeding severity on clinical outcomes among patients with acute coronary syndromes. *Am J Cardiol* 2005; **96**:1200–1206.
18. Manoukian SV, Feit F, Mehran R, Voeltz MD, Ebrahimi R, Hamon M, Dangas GD, Lincoff AM, White HD, Moses JW, King SB 3rd, Ohman EM, Stone GW. Impact of major bleeding on 30-day mortality and clinical outcomes in patients with acute coronary syndromes: an analysis from the ACUITY Trial. *J Am Coll Cardiol* 2007; **49**:1362–1368.
19. Eikelboom JW, Mehta SR, Anand SS, Xie C, Fox KA, Yusuf S. Adverse impact of bleeding on prognosis in patients with acute coronary syndromes. *Circulation* 2006;**114**:774–782.

CARDIOVASCULAR FLASHLIGHTS

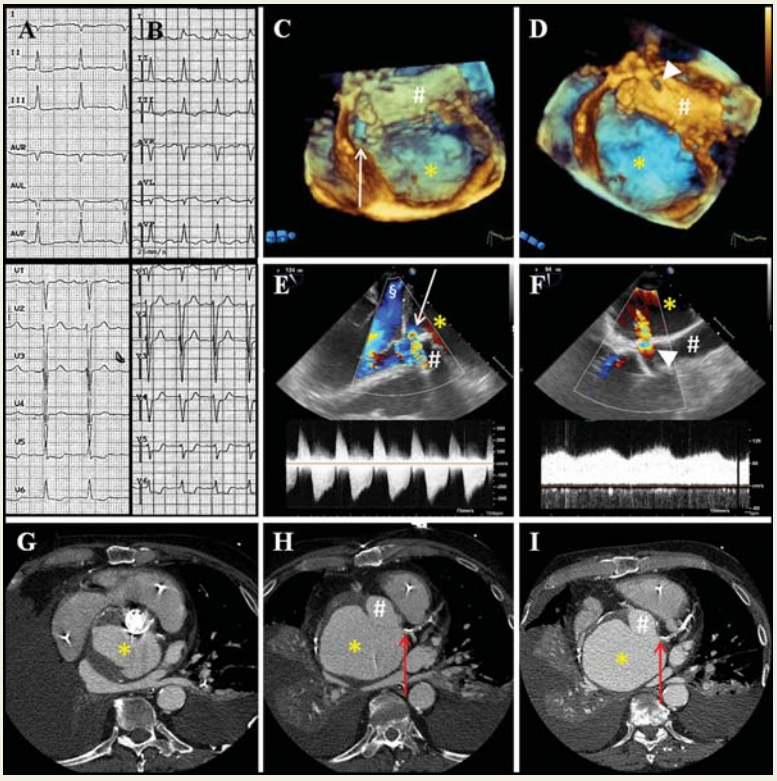
doi:10.1093/eurheartj/ehr257
Online publish-ahead-of-print 30 July 2011

Aortic pseudo-aneurysm caused by complete dehiscence of the left coronary artery 7 years after a composite mechanical-valved conduit aortic root replacement (Bentall operation)

Pierre Monney*, Cyril Pellaton, Salah Dine Qanadli, and Xavier Jeanrenaud

Service de Cardiologie, Centre Hospitalier Universitaire Vaudois-CHUV, Rue du Bugnon 46, CH-1011 Lausanne, Switzerland

* Corresponding author. Tel: +41 213140010, Fax: +41 213140055, Email: pierre.monney@chuv.ch



A 65-year-old man was admitted with rapidly progressive dyspnoea. He had been operated on for a type A aortic dissection 11 years earlier (supracommissural replacement), and 4 years later underwent a Bentall operation for dehiscence of the proximal graft anastomosis and severe aortic regurgitation.

On admission, high jugular venous pressure and pulmonary oedema were noted. Acute respiratory distress with cyanosis developed in the supine position and the patient was intubated. Dynamic electrocardiographic changes with ST-elevation (I–aVL) and ST-depression (V4–V6) occurred when the patient was moved from the supine (*Panel A*) to the left lateral position (*Panel B*). A three-dimensional transoesophageal echocardiogram demonstrated a 9 × 8 cm large aortic pseudo-aneurysm (*), posterior to the composite graft (#), compressing the left atrium (§), and communicating with the left ventricular outflow tract through a large fistula (arrow) with bidirectional flow (*Panels C* and *E*). There was a 5 × 9 mm orifice (arrowhead) on the left side of the aortic graft communicating to the pseudo-aneurysm with dense continuous flow, suggesting dehiscence of the left coronary artery (*Panels D* and *F*). A computed tomographic scan (*Panels G–I*) showed the proximal end of the left coronary artery (red arrow) originating from the pseudo-aneurysm, 10 mm apart from the composite graft. Emergency surgery was performed and intraoperative findings confirmed the dehiscence of the left coronary artery with the main stem floating freely in the pseudo-aneurysmal cavity. Inflammatory markers and blood cultures were negative.

Complete coronary artery dehiscence is an exceptional cause of pseudo-aneurysm after a Bentall operation; it resulted in extensive myocardial ischaemia, the myocardial perfusion being exclusively dependent on intra-aneurysmal pressure.