

Rapid Diagnostics and Treatment of Early Complications after CABG Surgery: A Life Saver

Ioannis Thomas Passaloglou, MD, Anton Sabashnikov, MD, Mohamed Zeriouh, MD, Stefanie Reutter, MD, Javid Fatullayev, Yeong-Hoon Choi, MD,* Thorsten Wahlers, MD*

Department of Cardiothoracic Surgery, Heart Center of the University of Cologne, Cologne, Germany

ABSTRACT

Early graft failure after CABG surgery may lead to severe adverse events and death. Because the cause of the graft failure can vary, rapid diagnostic management is mandatory in order to address these complications appropriately. In the present 2 cases, patients who underwent CABG procedures showed typical electrocardiograms and serology of a perioperative myocardial ischemia shortly after surgery. In the first case, a rapidly performed coronary angiogram revealed a torqued right CABG, which was detorqued and, in order to avoid further torsion, fixated to the pericardium in a redo procedure. In the second case, the patient underwent a revascularization by means of percutaneous coronary intervention with stent implantation for severe stenosis due to a localized dissection of the vein graft, diagnosed on coronary angiogram. The further postoperative course of both patients was smooth and both could be discharged on day 8 and 11 after initial surgery, respectively.

INTRODUCTION

Coronary artery bypass grafting (CABG) remains the best choice for improving outcome in patients with IHD [Serruys 2012]. However, CABG can be associated with severe early postoperative complications, including early graft failure. Risk evaluation as well as immediate and correct decisions are mandatory in order to avoid further severe complications. Whereas early bypass occlusion is a known and well-studied complication after CABG, only a few cases of torsion of a bypass immediately after CABG surgery have been previously reported [Rerkpattanapipat 1999; Diarte 2001]. It was shown that graft torsion and kinking mainly occur in arterial grafts, such as internal mammary artery (IMA) grafts [Brenot 1988; Kim 2001; Sachdeva 2007]. Furthermore, recent evidence

suggests that skeletonized IMA grafts are particularly prone to torsions and kinking [Imamaki 2007]. However, in the published literature there is less evidence on the greater saphenous vein, the most common venous graft used for CABG procedures, which is usually harvested in a skeletonized fashion. For the following 2 cases, we present our experience with and rapid management of both early graft occlusion and graft torsion after CABG surgery.

CASE I

In this case, a 77-year-old patient with IHD underwent elective CABG surgery. A total of 3 CABG were performed through median sternotomy: left internal mammary artery (LIMA) to left anterior descending (LAD) artery and single saphenous venous grafts (SVG) to the marginal branch of the circumflex artery and to the right coronary artery (RCA). Intraoperatively, the patient did not require blood transfusions and the weaning off the cardiopulmonary bypass (CPB) was uneventful. Following surgery, the patient was transferred to the intensive care unit (ICU) on low support of nor-epinephrine (0.02 µg/kg per min).

Shortly after arriving at the ICU the patient developed repeated ventricular fibrillation requiring 2 short interventions of cardiopulmonary resuscitation. There was no evidence of cardiac ischemia in the first postoperative laboratory investigation (troponin T, 0.298 ng/L; creatine kinase [CK], 359 U/L; CK-MB, 40 U/L); however, the patient became increasingly hemodynamically unstable. During the hemodynamic breakdown the patient incrementally needed high-dose inotropic support (adrenalin, 0.5 µg/kg per min), and his central venous pressure rapidly increased up to 22 mmHg. Additionally, the patient developed severe, therapy-resistant tachyarrhythmia (treated with amiodarone, 60mg/h and xylocaine, 80 mg/h). Repeated blood samples revealed increased levels of CK of 1238 U/L, CK-MB of 142 U/L, and troponin T of 2.030 ng/L. Echocardiography was performed and showed significantly reduced right ventricular pump function. After immediate interdisciplinary consultation with a cardiologist, the patient was transferred to our institutional catheter laboratory for diagnostic catheterization. Surprisingly, the cardiologist did not find any occluded grafts during this procedure. However, the RCA bypass showed a conversion

Received August 28, 2013; accepted November 25, 2013.

Correspondence: Anton Sabashnikov, MD, University Hospital of Cologne Heart Center, Department of Cardiothoracic Surgery; +49 (221) 478-32452; fax: +49 (3212) 107-9682 (e-mail: anton.sabashnikov@uk-koeln.de).

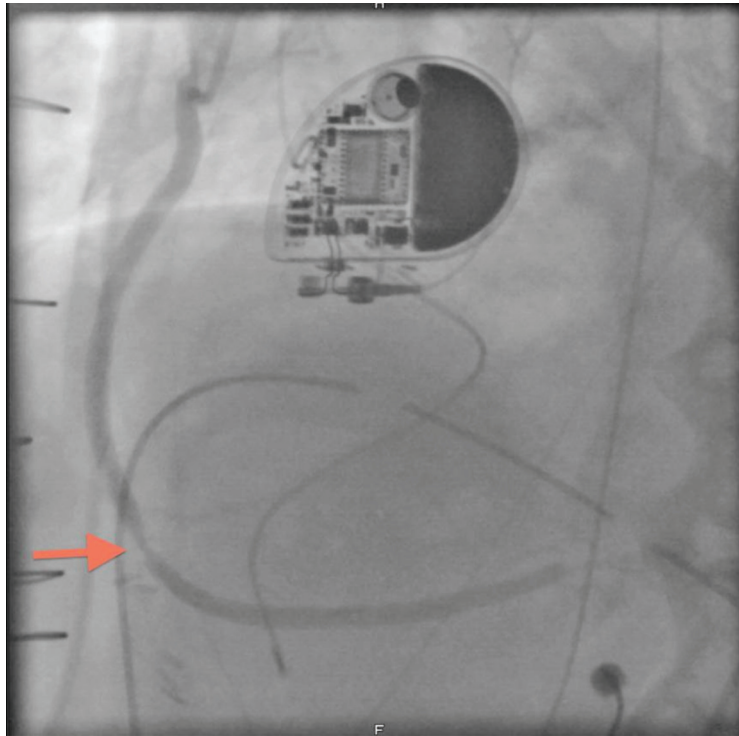


Figure 1. Postoperative emergency cardiac catheterization image: RCA graft during systolic muscle contraction. A graft narrowing can clearly be seen during every systolic contraction.

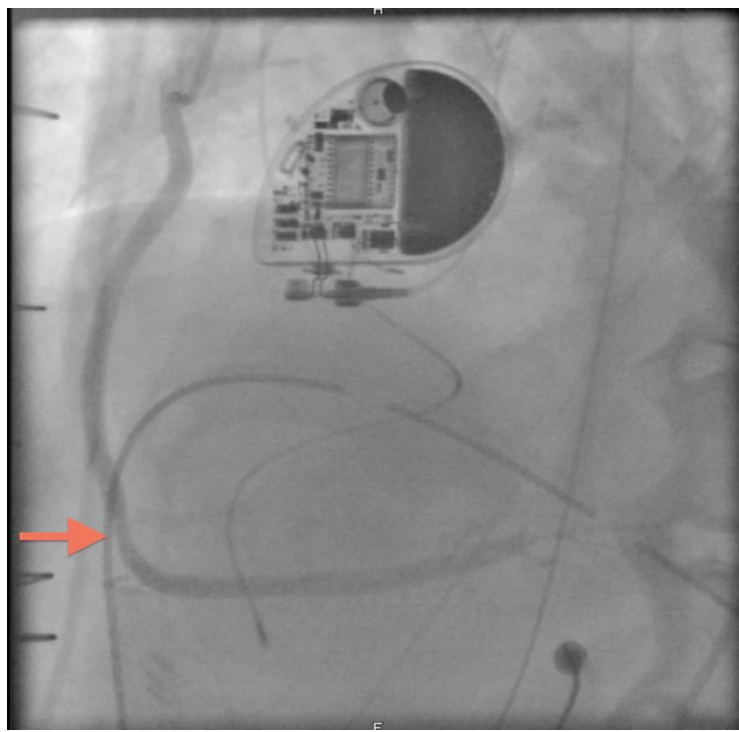


Figure 2. Postoperative emergency cardiac catheterization image: RCA bypass graft during diastolic cardiac muscle relaxation. The bypass is open and free of pathologic signs.

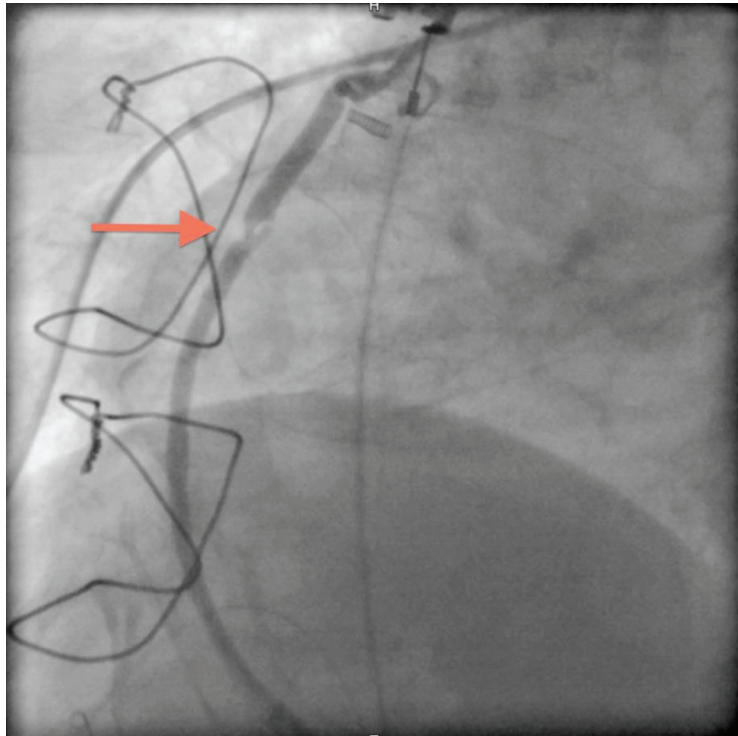


Figure 3. Postoperative emergency catheterization image: bypass graft stenosis at the proximal third of the venous graft anastomosed to the RCA.

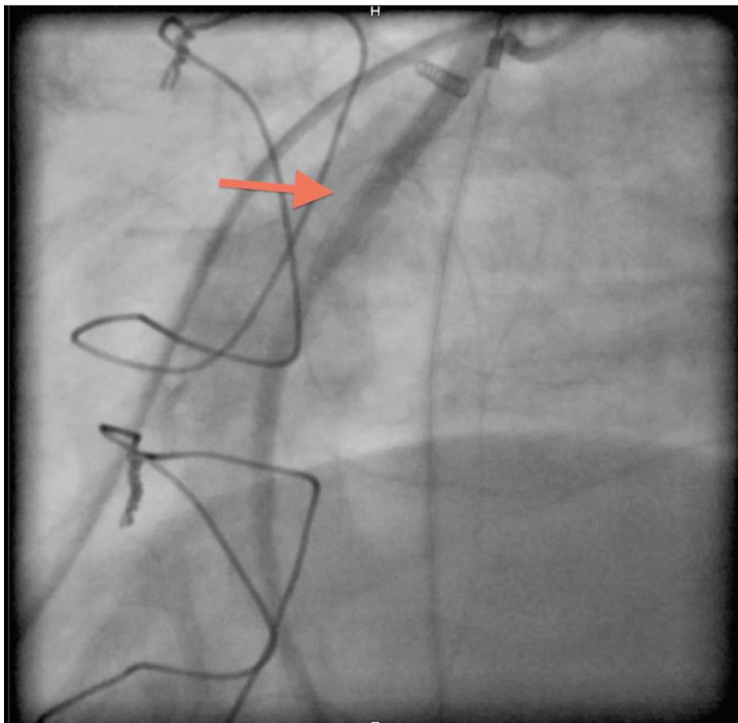


Figure 4. Catheterization image after stent implantation into the venous bypass graft, which is patent.

during each myocardial muscle relaxation (Figure 1) and a narrowing below the crux during every systolic myocardial contraction (Figure 2). Immediately after these findings, the patient was taken back to the operating theater and the RCA bypass graft was detorqued and shortened. Postoperatively, the arrhythmia discontinued, the central venous pressure normalized, and the patient remained hemodynamically stable. On the following day the patient was successfully extubated and rapidly weaned off inotropic support. After successful mobilization he was discharged from the hospital on the eighth postoperative day.

CASE 2

A 70-year-old patient with IHD underwent elective CABG surgery. The LIMA was anastomosed to the LAD and an SVG was connected to the RCA. During the weaning off the CPB, no pathologic electrocardiogram (ECG) signs appeared and the entire operation was unremarkable. Postoperatively, the patient was transferred to the ICU on norepinephrine (0.03 µg/kg/min) support. On the morning of postoperative day one, the patient was weaned off all inotropics and extubated, and shortly after this he was transferred to the general cardiac surgery ward. On day 2 after surgery, the patient showed intermittent ventricular tachycardia and was transferred back to the ICU. No ischemic ECG signs were seen, and there was no evidence of cardiac ischemia in the laboratory analysis (troponin T, 0.174 ng/L; CK, 183 U/L; CK-MB, 6 U/L). The ECG performed was unremarkable. A treatment attempt with intravenous antiarrhythmic drugs (metoprolol, 5 mg intravenous; amiodarone 60 mg/h; xylocaine 70 mg/h) as well as atrial overstimulation with an external pacemaker did not result in any improvement.

After an interdisciplinary exchange, a cardiac catheterization was performed which showed a local dissection of the vein graft, resulting in a severe stenosis at the proximal third of the SVG anastomosed to the RCA (Figure 3). Immediate angioplasty with primary stenting was performed using a 3.5 × 12 mm Resolute Integrity zotarolimus-eluting CoCr coronary stent (Medtronic, Minneapolis, MN, USA).

Postinterventionally, intermittent ventricular tachycardia persisted, which was converted into a stable normofrequent atrial fibrillation after 1 ampulla ajmalin (50 mg) was given as a bolus intravenously. On postinterventional day 2 the patient was transferred back to the normal cardiac surgery ward. After mobilization and further treatment with oral β -blockers and electrolyte substitution, on day 11 postoperatively the patient was discharged from the cardiac surgery service into cardiac rehabilitation under normal sinus rhythm.

COMMENTS

Graft kinking of SVG and LIMA have been previously reported in the literature [Hutchins 1977], but ischemia based on these complications has been described as a rare phenomenon [Rerkpattanapipat 1999; Diarte 2001]. Therefore, in the event of postoperative graft kinking, immediate intervention

is not always necessary if there are no symptoms of angina or ischemic ECG changes [Imamaki 2007]. Previous studies have shown that early postoperative graft abnormalities usually disappear in the following late angiographies [Ivascau 2005; Izumi 2005].

In cases in which intervention is inevitable, a rapid and precise diagnostic determination is imperative to find out the cause of ischemia and provide adequate treatment. Simple graft occlusions can be addressed by percutaneous coronary intervention, which has a success rate of approximately 80% [Dimas 1991; Sketch 1992]. However, dissections or restenosis of the graft remain significant risk factors. In case of graft torsion or kinking, a redo procedures and bypass fixation should be considered, if this technique is applicable. Unfortunately, the success rate of this procedure remains the merit of further investigations.

However, the reasons for graft kinking and torsion after surgery are still not clearly understood. Previous observations have indicated that the design and anatomical position of the bypass grafts may lead to such phenomena [Imamaki 2007]. Additionally, histological changes and the use of harmonic scalpels are factors that should be taken into consideration.

Graft kinking and torsion may be avoided by appropriate fixation of grafts, which can be undertaken during surgery if there are doubts about the predetermined graft design. Sutures along the cardiac surface have proven to be a cheap and effective method of keeping the appropriate anatomical graft position [Izumi 2005]. However, it may be difficult to fixate a skeletonized graft with sutures because there is only a small amount of connecting tissue around the graft. Additionally, fixation between the vessel adventitia and cardiac surface should be done with caution because of the present risk of graft damage. In difficult cases in which fixation with sutures is not possible, fibrin glue can be applied as an alternative [Imamaki 2007].

The 2 cases described above illustrate that not only occlusion but also kinking or torsion of bypass grafts in early postoperative phases should be considered as a differential diagnoses of chest pain or persistent arrhythmias in patients who have undergone CABG surgery. Furthermore, a rapid coronary angiogram can play a crucial role in adequate diagnostics of such rare complications and helps in improving postoperative mortality in this extreme patient group.

REFERENCES

- Brenot P, Mousseaux E, Relland J, Gaux JC. 1988. Kinking of internal mammary grafts: Report of two cases and surgical correction. *Cathet Cardiovasc Diagn* 14:172-4.
- Diarte JA, Salazar JJ, Placer LJ. 2001. Saphenous vein graft kinking: angiographic image. *Revista Espanola de Cardiologia* 54:997-8. In Spanish.
- Dimas AP, Arora RR, Whitlow PL, et al. 1991. Percutaneous transluminal angioplasty involving internal mammary artery grafts. *Am Heart J* 122:423-9.
- Hutchins GM, Bulkley BH. 1977. Mechanisms of occlusion of saphenous vein--coronary artery "jump" grafts. *J Thorac Cardiovasc Sur* 73:660-7.
- Imamaki M, Sakurai M, Shimura H, Ishida A, Fujita H, Miyazaki M.

2007. Pitfalls of skeletonized internal thoracic artery: comparison of graft kinking between skeletonized and pedicled grafts based on postoperative angiography findings. *J Card Surg* 22:195-8.

Ivascau C, Buklas D, Massetti M, et al. 2005. Can an early peri-anastomotic lita stenosis be reversible? *Ann Thorac Surg* 79:348-51.

Izumi C, Hayashi H, Ueda Y, et al. 2005. Late regression of left internal thoracic artery graft stenosis at the anastomotic site without intervention therapy. *J Thorac Cardiovasc Surg* 130:1661-7.

Kim WS, Lee YT, Choi JH, et al. 2007. Kinking of internal thoracic artery graft without symptoms: should it be revised? *Heart Surg Forum* 10:E372-5

Rerkpattanapipat P, Ghassemi R, Ledley GS, et al. 1999. Use of stents to

treat kinks causing obstruction in a left internal mammary artery graft. *Catheter Cardiovasc Interv* 46:223-6.

Sachdeva R, Sarkar K, Sureddi RK. 2007. Myocardial ischemia due to kinking of left internal mammary artery graft with the expiratory phase of respiration. *J Invasive Cardiol* 19:241-2.

Serruys PW, Farooq V, Vranckx P, et al. 2012. A global risk approach to identify patients with left main or 3-vessel disease who could safely and efficaciously be treated with percutaneous coronary intervention: the SYNTAX Trial at 3 years. *JACC Cardiovasc Interv* 5:606-17.

Sketch MH Jr, Quigley PJ, Perez JA, et al. 1992. Angiographic follow-up after internal mammary artery graft angioplasty. *Am J Cardiol* 70:401-3.