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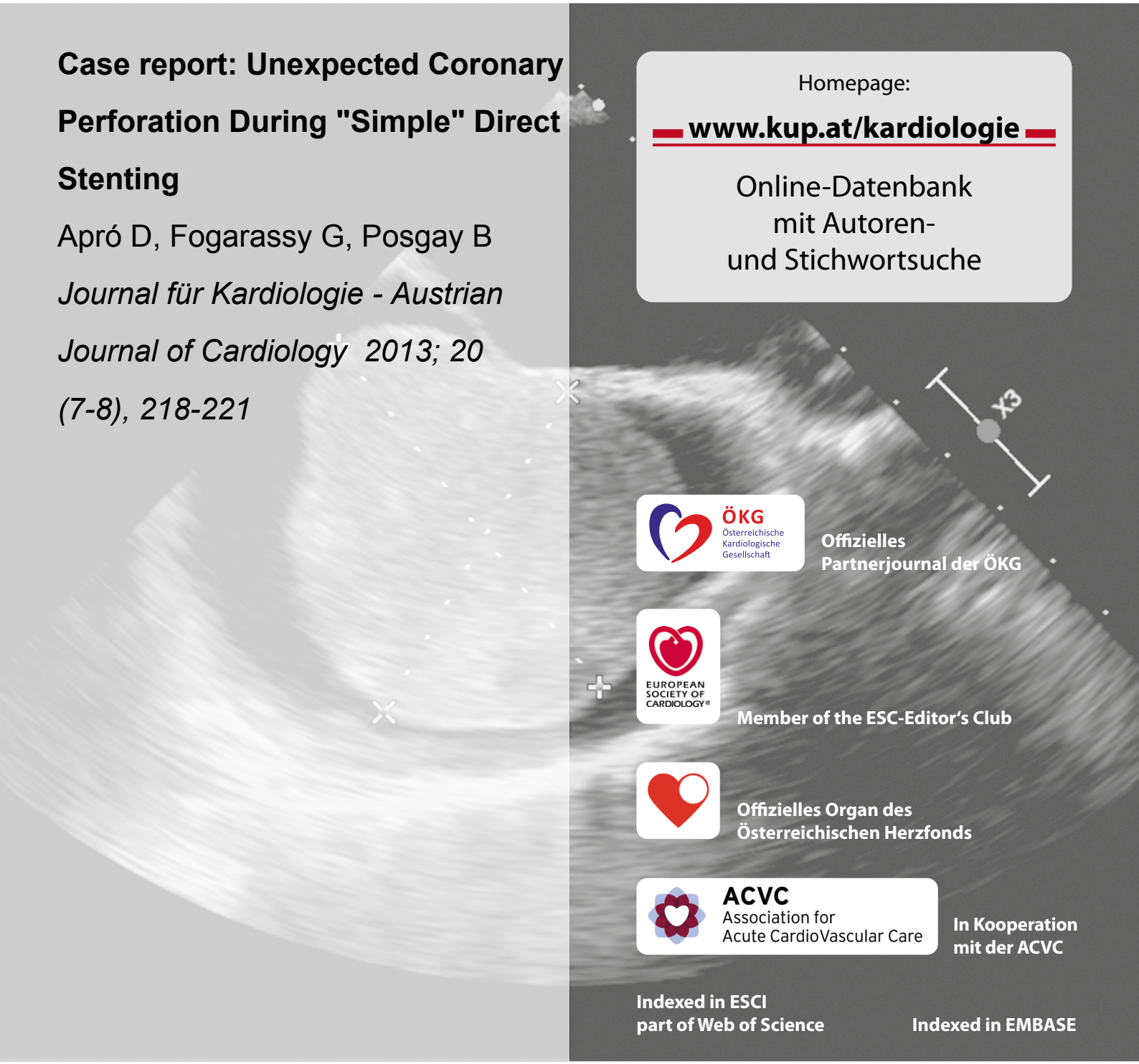

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Unexpected Coronary Perforation During "Simple" Direct Stenting

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Summary

Coronary artery perforation is a rare, but life-threatening complication of percutaneous coronary interventions (PCI). The occurrence of perforation increased with newer interventional devices and techniques like rotablation, excimer laser coronary angioplasty, routine high pressure balloon dilatation or chronic total occlusion interventions. We describe a case of unexpected Ellis grade 3 perforation following "routine" direct stenting of mid left anterior descending coronary artery (LAD) stenosis. The perforation was successfully sealed by a polytetrafluoroethylene-covered stent graft. In addition to the availability of covered stents, it is essential to be familiar with various skills necessary for successful management of these complications.

Introduction

Coronary artery perforation during percutaneous transluminal coronary angioplasty occurs very rarely, the incidence varies from 0.2 % to 0.5 % [1–3]. Historically it has been associated with a high rate of major adverse outcomes [3, 4], such as pericardial tamponade 17 % [5], myocardial infarction or death (9 %) [5]. A surgical approach to treat the perforation was necessary in 37–63 % of cases [4]. The incidence and the severity of perforations are reported to increase with debulking devices like directional coronary atherectomy (DCA), excimer laser coronary angioplasty (ELCA), rotablator [6–8], or evolving techniques of chronic total occlusion (CTO) interventions. Ellis grade 3 perforation [3] occurs approximately 25–35 % of cases [3, 9]. Of grade 3 perforations 40 % develop tamponade, 60 % require emergency CABG and 44 % die during index hospitalization [7]. Occasionally perforation can even be associated with simple stenting [7], therefore every interventional laboratory must be prepared to handle this complication. We describe a case of unexpected Ellis grade 3 perforation following "routine" direct stenting of mid LAD stenosis. The perforation was successfully sealed by a polytetrafluoroethylene-covered stent graft.

Case Report

A 59-year-old Caucasian male who had undergone non-diagnostic exercise test (non significant ST depression in leads V3-6) 3 weeks earlier, was admitted to our hospital. He had been having moderately severe effort angina for several months. In his past history a 6 year hypertension and long-standing bronchitis were reported. His blood pressure was 120/70 mmHg, pulse 60/min, laboratory tests were within normal limits with serum LDL cholesterol of 3,6 mmol/l. Physical examination revealed a patient with normal weight, normal heart sounds, without any remarkable physical findings. A 12-lead electrocardiogram showed normal sinus rhythm with non-specific ST-T changes in the anterior leads. Baseline echocardiography: aorta 23 mm; left atrium: 32 mm; left ventricle diastolic diameter: 42 mm; interventricular septum diastole: 13 mm; posterior wall diastole: 11 mm; ejection fraction: 58 %. There was no wall motion abnormality and the valves were functioning well.

Figure 1: Baseline angiogram showed a long, approximately 60 % left anterior descending artery (LAD) stenosis.

Baseline angiogram showed a long, approximately 60 % left anterior descending artery (LAD) stenosis (Fig. 1). Minimal calcification was seen in the proximal LAD and moderate wall contour irregularity in all segments. The curves of LAD were suspicious of intra-myocardial course but bridging was not seen even after intra coronary nitroglycerin (100 µg). The circumflex and right coronary arteries were practically free of disease.

Given his symptoms, we proceeded with coronary pressure measurement and calculation of fractional flow reserve (FFRmyo). A 6 French VL 3,5 guiding catheter with side holes (Boston Scientific, Natick, MA) was advanced into the left coronary ostium, and 7000 IU of heparin was administered. For distal coronary pressure measurements, the 0.014-in pressure wire (Pressure Wire; RADI Medical Systems, Uppsala, Sweden) was advanced distally through the LAD stenosis, and a repeated bolus injection of 100 µg nitroglycerine was administered. Steady-state maximum hyperemia was induced by the intravenous infusion of adenosine (150 µg/kg/min)

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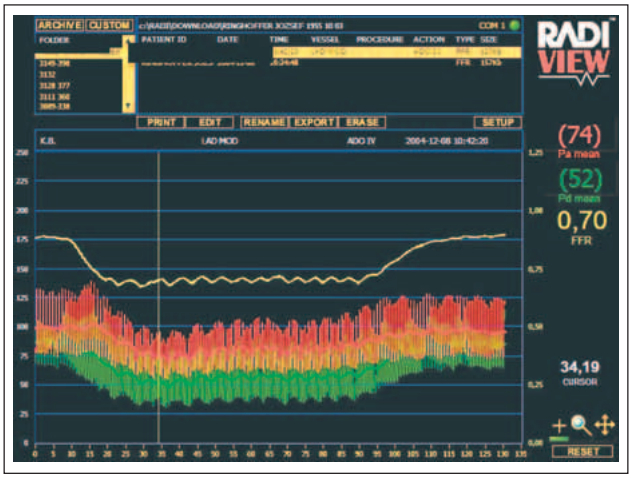

Figure 2: In this case the determined FFRmyo was = 0.70, therefore the stenosis was considered significant and angioplasty to the LAD with direct stenting was decided.

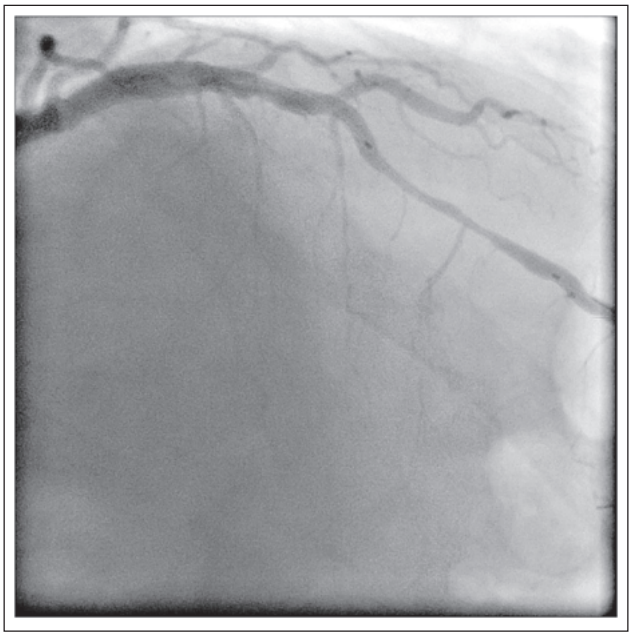

Figure 3: A 3.5 × 32 mm Liberte Stent (Boston Scientific, Natick, MA) was deployed at 12 atm.

through the femoral venous sheath. The aortic pressure (Pa) was recorded through the guiding catheter, while the distal coronary pressure (Pd_{LAD}) was measured by use of pressure wire. FFRmyo was determined as the ratio of the mean distal (transstenotic) LAD pressure divided by the mean aortic pressure (Pa) during hyperemia: $FFRmyo_{LAD} = Pd_{LAD}/Pa$. FFRmyo was calculated 15 min after a non-diagnostic angiographic result had been obtained. In this case the determined FFRmyo was 0.70 (Fig. 2), therefore on the basis of observations in earlier studies, the stenosis was considered significant and angioplasty to the LAD with direct stenting was decided.

The wire was changed and the LAD stenosis was crossed with a 0.014 IQ marker wire (Boston Scientific, Natick, MA). A 3,5 × 32 mm Liberte stent (Boston Scientific, Natick, MA) was deployed at 12 atm (Fig. 3), which resulted in severe chest pain and pressure drop. Angiography confirmed the presence of Ellis grade 3 coronary perforation at the proximal

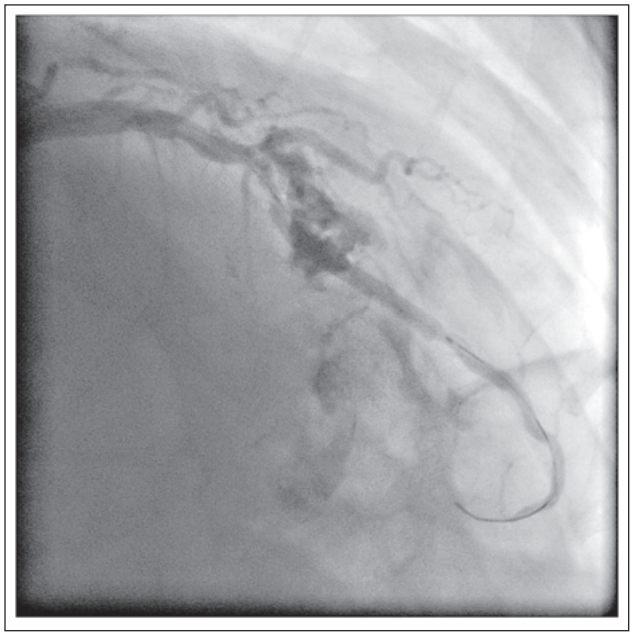

Figure 4: Angiography confirmed the presence of Ellis grade 3 coronary perforation at the proximal part of the stent with free flow of contrast into the pericardial space.

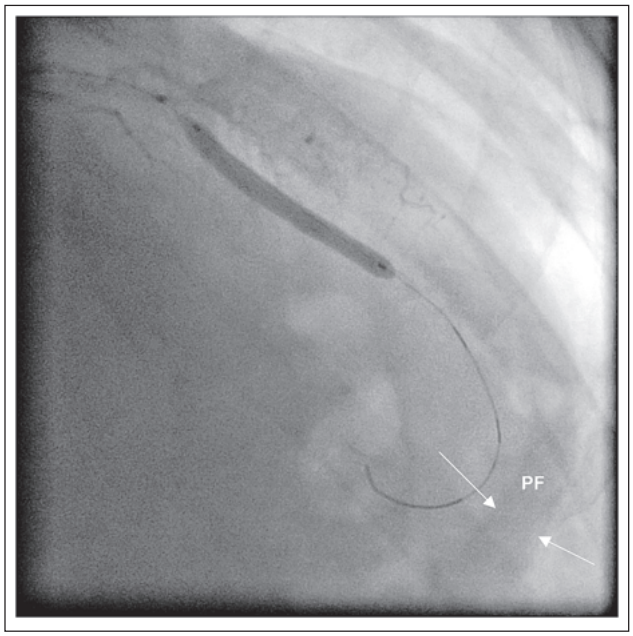

Figure 5: Considerable amount of pericardial fluid (PF) became apparent (between arrows).

part of the stent with free flow of contrast into the pericardial space (Fig. 4). The stent's balloon was reinflated immediately to 5 atm and the perforation was sealed temporarily, which restored hemodynamic stability. Considerable amount of pericardial fluid became apparent (between arrows Fig. 5). We have seen no chance of sealing the large perforation with a perfusion balloon, so heparin was reversed with 20 mg of intravenous protamine sulphate and the use of a coronary stent graft was decided. A 3,5 × 19 mm JOSTENT Graftmaster (Abbott Vascular, Santa Clara, California) was deployed at 12 atm over the perforation site (Fig. 6). Test injections revealed no further extravasation, the flow into the pericardial space abolished (Fig. 7). The patient remained hemodynami-

Figure 6: A 3,5 × 19 mm JOSTENT GraftMaster (Abbott Vascular, Santa Clara, California) was deployed at 12 atm over the perforation site.

Figure 7: Test injections revealed no further extravasation, the flow into the pericardial space abolished.

cally stable thereafter. Echocardiogram revealed 11–16 mm pericardial fluid. After urgent consultation with cardiothoracic surgeons, transport to a heart surgery (nearest 110 km) was decided. On the 4th day exudative pericarditis developed and pericardiocenthesis became necessary. The patient remained stable during the remaining hospital stay. There were no significant ECG changes and maximum CPK level was 186 U/l. He was discharged on aspirin 300 mg, and clopidogrel 2 × 75 mg. At 6-month follow-up, he had no effort angina and the treadmill test was negative.

Discussion

Coronary artery perforation is an infrequent, but dreaded complication, which occurs in 0.2–0.5 % during PCI [1–3]. It can be associated with adverse clinical outcome, such as pericardial tamponade, myocardial infarction, need for emergency coronary artery bypass surgery (CABG) or death. There are several factors that predispose to coronary perforation, such as excessive vessel tortousity, calcification, small vessel diameter, CTO, high pressure balloon dilatation, or use of an oversize balloon. Stiffer hydrophilic wires can also cause Ellis type 1 or type 2 perforation, but generally wire-related perforations have benign course [9]. The classical treatment of the perforation is the prolonged balloon inflation at the site of the extravasation and reversal of the anticoagulation with protamin [3]. The administration of protamin was reported to be safe and not to predispose to stent thrombosis, but the reversal of heparin after a complex PCI remained controversial [10]. Deployment of a conventional stent at the site of perforation may be effective, but rarely it can make perforations worse by expanding the vessel [9, 10]. In type 3 perforation the classical nonsurgical management often fails. The surgical management includes urgent repair or ligation, and grafting of the related artery as well as pericardial drainage. However, this intervention has an overall mortality rate up to 20 % [10].

At the end of the 1990ies covered stent grafts as a new method for perforations appeared. In the beginning autologous veins were surgically harvested, prepared and mounted on a conventional stent to cover it [11], but this approach is logistically impossible in an emergency situation. In contrast, the implantation of the polytetrafluoroethylene (PTFE)-covered stent grafts is much easier and faster, and does not require special skills. A PTFE-covered stent consists of two conventional stents and a thin polytetrafluoroethylene membrane in between. Therefore these stents are more rigid than other normal stents, and without adequate guiding catheter support they may be difficult to deliver [4]. A randomized study is not feasible to analyse the effectiveness of the covered stent in severe coronary perforations. Briguori et al. [4] reported lower rates of tamponade and need for emergency surgical intervention in patients in whom conventional prolonged balloon inflation therapy failed and who were treated with PTFE stent. However, this study compared the findings with a historical cohort before the availability of covered stents. At present 91–93 % of cases can be sealed successfully with the implantation of PTFE-covered stents [1, 4].

PTFE-covered stents in various clinical settings showed a subacute stent thrombosis rate of 5.7 %, which is higher than that of normal stents. The angiographic restenosis rate is also relatively high (32 %), mainly localized at the stent edge [12]. As indicated by angioscopic and optical coherence tomography (OCT) observations [13], the endothelialisation of these stents is delayed and restenotic lesions may also contain thrombus, similarly to drug eluting stents. There is no consensus on the duration of antiplatelet and anticoagulant therapies after PTFE-covered stent implantation [13]. Long-term data are not yet available to assess post-discharge thrombosis, restenosis and vessel reocclusion rate. The present case proves that coronary rupture can take place in simple direct stenting cases as well. Since coronary perforation is a potentially fatal complication, familiarity with steps to manage this complica-

tion: speed to obstruct the affected part, protamin and the use of stent graft in Ellis grade 3 perforations is essential. Transport to heart surgery may be recommended even in stable condition.

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