Spontaneous Resolution of Catheter-Induced Iatrogenic Vein Graft Dissection: First Case Report and Review of Literature

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Abstract

An 86-year-old female patient with a history of bypass surgery was admitted for recurrent dyspnea and chest pain on exertion. She underwent coronary angiography showing high-grade saphenous vein graft stenosis supplying the right coronary. During an attempt to perform percutaneous intervention, saphenous vein graft to the right coronary artery was injured by the guide catheter resulting in dissection with thrombus formation but with normal distal blood flow. The procedure was abandoned since she had normal distal flow and also due to a fear of causing extension of the dissection with further manipulation. The patient was hemodynamically stable and free of chest pain after 30 minutes of observation. The patient was later discharged home on medical treatment. After 1 month, a repeat angiography showed patent saphenous vein graft to the right coronary artery with healed dissection. This is the first case report of spontaneous recovery of iatrogenic saphenous vein graft dissection. This case is followed by the review of literature.

Keywords

- ▶ dissection
- ▶ saphenous vein graft
- ▶ angiography
- ▶ cardiac catheterization
- ▶ coronary intervention

There are known case reports that coronary dissection occurring during cardiac catheterization or intervention of native coronaries in hemodynamically stable patient can completely heal. However, this observation has never been reported in a vein graft. We are presenting a case of iatrogenic vein graft dissection with complete recovery seen on a subsequent angiography.

Case Presentation

An 86-year-old female patient was admitted with complaints of dyspnea and chest pain on exertion. Her symptoms were recurrent and occurred upon walking for even half a block. Her medical history included long-standing hypertension, a 50 pack year smoking, hyperlipidemia, hypothyroidism, transient ischemic attack, and coronary artery disease. She had undergone coronary artery bypass grafting (CABG) for triple vessel disease with left internal mammary artery

(LIMA) to left anterior descending artery (LAD), saphenous vein graft (SVG) to right coronary artery (RCA) and to left circumflex artery (LCX) in the year 1995. Her home medications included aspirin, metoprolol, pravastatin, irbesartan, nifedipine, and levothyroxine. The clinical examination was unremarkable other than noticeable CABG scar in the midline. Admission electrocardiogram (ECG) showed normal sinus rhythm, left ventricular hypertrophy by voltage, presence of ST depression in leads I and aVL. Her cardiac enzymes were negative.

The left ventriculography revealed near-normal left ventricular function with estimated ejection fraction of 50%. Left main coronary artery had 99% distal lesion with diffuse LAD disease and occluded RCA. The LIMA to LAD and SVG to LCX were patent. A high-grade lesion was found in the RCA vein graft. An attempt to engage the vein graft with a Judkins R4 guide catheter resulted in the injury to the vein graft with dissection and thrombus formation without any changes in

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Fig. 1 Dissection of the saphenous vein graft occurring during guide catheter engagement.

TIMI 3 flow (► **Fig. 1**). Further attempts were thought to be too risky at this point in view of a possibility of extending the false lumen and causing abrupt closure of the vessel. A decision was made to observe the patient clinically and opt for medical management. The patient remained hemodynamically stable and chest pain free. She was transferred to cardiac intensive care unit. Subsequent ECGs showed multiple atrial premature complexes along with the previous changes but no ST elevation in any territory. Cardiac enzymes remained negative. She was discharged in a stable condition from the hospital with a plan for repeat coronary angiography at a later date.

Approximately 5 weeks later, she was readmitted to the hospital with history of sudden onset sweating, right arm numbness, and palpitations. ECG showed similar changes as seen in the first admission. Cardiac enzymes were elevated with troponin-I maximum of 2.74 ng/mL. A diagnosis of non-ST elevation myocardial infarction was made. The patient was started on heparin infusion and was sent to the catheterization laboratory. The catheterization revealed 80% occlusion of the proximal ramus intermedius lesion (RI). The SVG to RCA dissection which occurred on the previous admission was found to be healed (► **Fig. 2**). Successful percutaneous coronary intervention (PCI) of the ostial RI was performed without any complications. The patient did well with no further episodes of chest pain after a follow-up of 1 year.

Discussion

Aortocoronary vein graft injury is a rare but potentially life-threatening complication of coronary angiography. Different types of injuries result from different mechanisms and a proper understanding may serve to avoid this complication. More importantly, some cases can be managed conservatively without the need for stent placement. The first aortocoronary SVG implantation in a human by Garrett et al¹ and the subsequent pioneering work of Favalaro² in the era of surgical

Fig. 2 Healed dissection seen on repeat angiography 5 weeks later.

revascularization were important contributions for the treatment of ischemic heart disease. This major advance in surgical practice afforded an effective treatment for intractable angina and also a means of markedly improving long-term prognosis in certain patient subgroups.^{3,4} Ironically, with the demonstration of the dramatic benefits obtainable by saphenous vein grafting came recognition of the ultimately palliative nature of the operation due to the accelerated atherosclerosis that develops within the grafted saphenous vein conduits. Further revascularization, either redo bypass surgery or percutaneous intervention, is required in approximately 4% of patients by 5 years, 19% patients by 10 years, and 31% of patients by 12 years after initial bypass surgery.⁵

Both surgical and percutaneous forms of repeat revascularization have considerable limitations. As compared with the initial surgery, risk of perioperative morbidity and mortality escalates, and clinical benefits diminish.⁶ Percutaneous treatments for vein graft disease continue to be hindered, despite recent adjunctive therapies, by a high periprocedural morbidity resulting from distal embolization of atherothrombotic debris⁷ and possible vascular injuries. Any insertion of material into the coronary arteries can cause vascular injury at any time during the procedure, from the first insertion of the guide wire until the end of the angioplasty procedure. Complications include coronary artery dissection, intramural hematoma, coronary artery perforation, and occlusion of branch vessels. Certain clinical situations carry a particularly high risk of perforation. Female gender, older age, tortuous and calcified arteries, bifurcations and angulated lesions, small vessels, acute or chronic occlusions, SVGs, or mammary anastomoses are often reported as being the factors that contribute to a higher risk of coronary perforation. Stiff guide wires used for total chronic occlusion, excessive inflation pressure applied especially in extremely calcified lesions, oversized wires and stents, and usage of X-sizer atherectomy device in small, angulated, or calcified lesions increase the risk of vascular injury. Last but not the least, operator

experience and the volume of the procedures in the hospital are also important determinants of complications. Dissection and abrupt closure is a major complication of PCI without intentional stenting. Dissections are found in up to 50% of patients immediately after PCI. The occurrence of dissection cannot be predicted by preintervention analysis of lesion morphology or plaque composition by intracoronary ultrasound.⁸

Intimal tears or dissections following PCI have been arbitrarily divided into six types (A–F).^{9,10} Type A lesions are seen as luminal haziness, type B as linear dissection, and type C as extraluminal contrast staining. The presence of spiral dissection is classified as type D and dissection with reduced flow as type E. When there is dissection and total occlusion, the vessel is said to have type F dissection. Most cases of acute closure occur within minutes of the final balloon inflation, but subacute closure occurs up to hours later in 0.5 to 1.0% of the cases, typically as the heparin anticoagulation wears off.^{9,11} Stents can reverse abrupt closure in more than 90% of the cases. However, some patients still require bypass surgery after stenting because of persistent coronary occlusion or dissection resulting in ischemia. Follow-up PCI of mild dissections as early as 6 weeks after the procedure often demonstrates complete healing in the native coronaries, although occasional late localized aneurysm formation has been described.^{12,13} In contrast, larger dissections (C–F) are associated with an increased risk of progression to total occlusion (abrupt closure) of the treated arterial segment.^{11,12} The increased risk of abrupt closure and myocardial infarction associated with a large dissection has led to the routine use of stenting for any dissection. However, a stent may not be necessary for a non-occlusive dissection^{14,15} as in our case. This was illustrated in a study that compared the outcome of 45 patients with 49 nonocclusive dissections (types A–D) and good distal flow (TIMI grade 3) who did not receive a stent and 60 patients with a dissection who underwent stenting.¹⁴ At 6 months, there were no clinical adverse events in the unstented group, although the restenosis rate was lower than those who underwent stenting (12 vs. 25%). However, successful conservative treatment of a vein graft dissection with resolution has not been reported previously. Our case is the first case showing that the vein graft dissection could be treated conservatively, if TIMI 3 flow is present in a stable patient.

Conflict of Interest

None.

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