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BEGINNER

CASE REPORT: CLINICAL CASE

Ventricular Fibrillation Due to Aortocoronary Vein Graft Spasm During Angiography

Case Report and Literature Review

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ABSTRACT

A 69-year-old man underwent coronary angiography 7 years after coronary artery bypass. Saphenous vein graft spasm was observed during contrast injection, resulting in ventricular fibrillation. Angiography 6 years later showed graft patency. Vein graft spasm after coronary artery bypass grafting is rarely described. Further investigation is needed regarding incidence, mechanism, and clinical outcomes. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2021;3:388–91) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 69-year-old man with coronary artery disease and history of coronary artery bypass grafting (CABG) was evaluated via cardiac catheterization for unstable angina.

Seven years previously, he presented with dyspnea on exertion and a positive exercise stress test. Coronary angiography showed significant stenoses of the mid-left anterior descending artery (LAD), the ostium of the first diagonal branch, and a large second marginal branch. He underwent uncomplicated CABG with left internal mammary artery graft to the LAD and saphenous vein grafts (SVGs) to diagonal and marginal branches.

LEARNING OBJECTIVES

- To highlight the potential for spasm of aorto-coronary vein grafts during diagnostic angiography.
- To understand the mechanism for spasm of coronary bypass grafts.
- To discuss opportunities for investigation regarding contemporary incidence and outcomes of vein graft spasm.

PAST MEDICAL HISTORY

The patient's medical history was also significant for ischemic cardiomyopathy with a left ventricular ejection fraction of 45% by echocardiography, hypertension, hyperlipidemia, peripheral vascular disease, type 2 diabetes, and obstructive sleep apnea.

INVESTIGATIONS

On examination, the patient was in no distress with normal neurological status. He had regular cardiac rhythm with a soft systolic murmur heard best at the left upper sternal border and clear lung sounds. There

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was mild lower extremity edema and no jugular venous distension. Peripheral pulses were normal. Laboratory testing for circulating troponin I was negative, and serum levels of creatine kinase-muscle brain were within normal range. He had normal serum concentrations of creatinine and liver enzymes.

The patient was referred for coronary angiography, which revealed severe two-vessel disease of native coronary arteries and patent grafts to the LAD and marginal branch. During the first contrast injection into the vein graft to the diagonal branch, 99% mid-graft occlusion was observed (**Figure 1A**, Video 1). He immediately developed bradycardia followed by ventricular fibrillation cardiac arrest. He underwent successful resuscitation with chest compressions and epinephrine.

DIFFERENTIAL DIAGNOSIS

A guide catheter was advanced into the vein graft and a 0.014-inch coronary guidewire was placed across the lesion in preparation for emergency percutaneous coronary intervention. However, on the second contrast injection of this graft, complete return of vein graft patency was noted, a finding that was confirmed on repeat imaging with wire removed (**Figures 1B and 1C**, Videos 2 and 3). This sequence of events was believed to represent aortocoronary vein graft spasm provoked by contrast injection.

MANAGEMENT

The patient was started on calcium-channel blocker therapy and discharged 3 days after catheterization. He was re-admitted 1 week later for recurrent angina, which resolved with an increased dose of amlodipine.

DISCUSSION

Coronary arterial spasm is a well-described cause of angina and, rarely, acute coronary syndrome. Vasospasm is mediated by a combination of factors: mechanical disruption, central and autonomic neural impulses, direct stimulation by α-adrenoceptor agonists, and by molecular mediators, most notably thromboxane A_2 released by platelets. Healthy endothelium maintains balanced vascular tone via release of nitric oxide and prostacyclin. States of endothelial dysfunction impair release of vasodilatory substances and lead to unopposed vasoconstrictor effects of thromboxane.

Unlike with native and grafted arteries, aortocoronary vein grafts, per conventional wisdom, do not experience spasm beyond the peri-operative period. During harvesting of saphenous veins, perivascular tissue containing autonomic nervous system plexi is removed, eliminating neural input for smooth muscle constriction. Furthermore, early histological studies showed inflammatory infiltration and intimal proliferation in the first several months after surgery, leading to replacement of viable smooth muscle cells with collagen and fibroblasts. The final product is described as a rigid, noncompliant conduit without physiological smooth muscle responses (1).

Our experience and prior case reports suggest that exceptions exist. In 1981, Victor et al. (2) described a man evaluated for unstable angina 2 months after CABG who experienced spasm of SVG during angiography that resolved with sublingual nitroglycerin. A series of similar case reports dating 3 months to 3 years after CABG followed. In 2 cases, patients were diagnosed with vasospastic angina of native coronary arteries before CABG and experienced subsequent spasm of vein grafts to the same target vessels (3,4). In the only case series published to date, Kafka et al. (5) reported SVG spasm events in 24 patients, with data extracted from review of 1,264 consecutive CABG cases (with routine surveillance post-CABG angiography) in the 1970s and 1980s. Only 4 of the patients had anginal symptoms before catheterization. Nearly all events occurred within the first year after surgery, although 2 occurred >5 years' post-surgery. More than one-half of these cases involved difficult engagement of the graft, and standard procedure in this study involved 4 contrast injections (for 4 imaging planes) per graft. Importantly, 3 patients in this series experienced ventricular fibrillation and 3 had persistent graft occlusion at the end of the study, 2 of whom experienced clinical myocardial infarction in the affected region.

There have been few reported cases of vein graft spasm during angiography in recent years, following the introduction of safer low-osmolality contrast media and an expanded selection of specialty catheters for difficult-to-engage vessels. Among the 3 recent reports, all patients presented 1 to 3 years after CABG with rest angina or positive stress test findings, and they experienced vein graft spasm during catheter engagement or passage of a guidewire for a separate lesion. All events resolved with intravenous or intracoronary vasodilator therapy with subsequent angiographic demonstration of full graft patency (6–8). It is intriguing that in our case, spasm resolved without vasodilators, which were not given because the occlusion was initially interpreted as a stenotic lesion. In 2 of 3 patients, intensification of nitrate

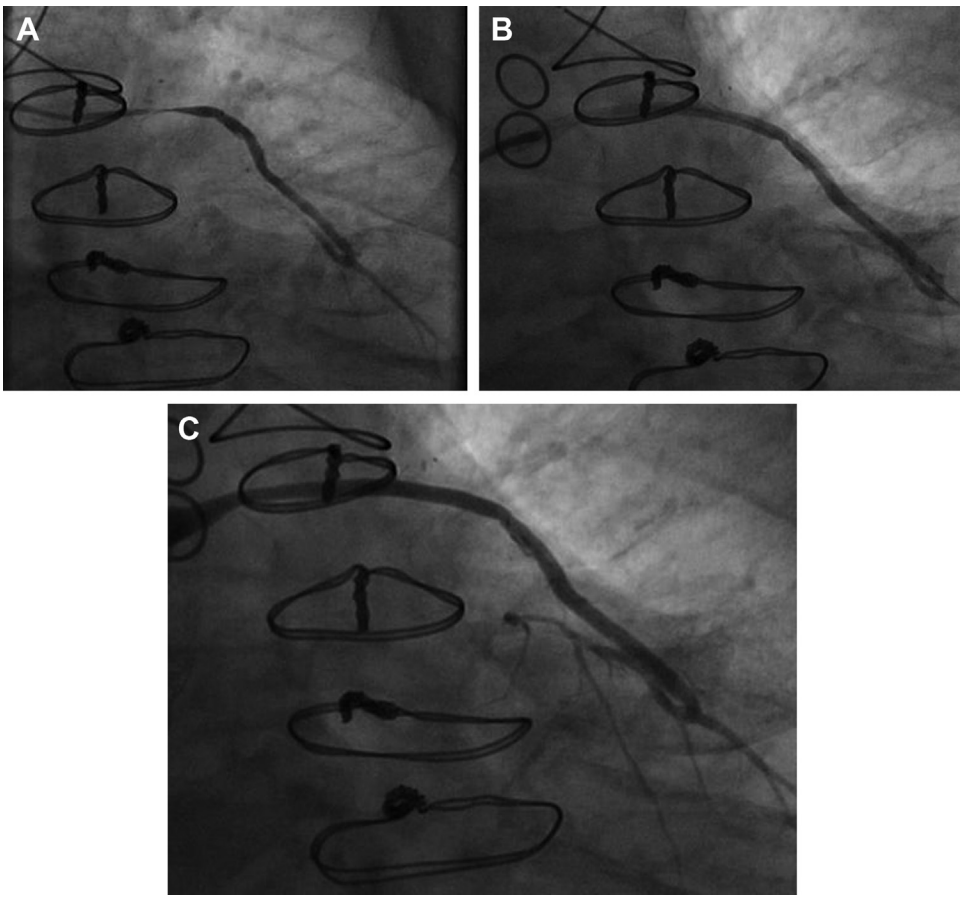
ABBREVIATIONS AND ACRONYMS

CABG = coronary artery bypass grafting

LAD = left anterior descending artery

SVG = saphenous vein graft

FIGURE 1 Dynamic Spasm of Saphenous Vein Graft to Diagonal Branch



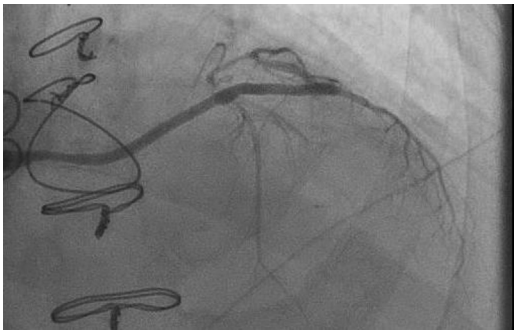
Coronary angiography in right anterior oblique 25° caudal 12° position. **(A)** Angiogram of vein graft to the diagonal demonstrates 99% mid-vessel occlusion. **(B)** After placement of coronary guidewire, spontaneous resolution of spasm is observed. **(C)** After removal of coronary guidewire, graft patency persists.

and/or calcium-channel blocker therapy led to long-term symptom resolution (6,7), as it did in our patient. At 7 years' post-surgery, our case represents the latest described occurrence of aortocoronary vein graft spasm and the only contemporary report of vein graft spasm leading to ventricular fibrillation.

The mechanism of spasm is suggested by in vitro studies. At physiological arterial blood pressure, vein grafts are at near-maximal distension and are insensitive to the dilating effects of nitrates (9–11). However, a preserved vasoconstriction response to ergonovine, norepinephrine, and serotonin has been reported (9,11), with a more pronounced response at lower vascular pressure (e.g., with reduction in perfusion pressure from 90 mm Hg to 50 mm Hg) (11). This finding indicates that relative hypotension and sympathetic stimulus create an environment conducive to spasm. Furthermore, harvested saphenous veins subjected to vasoconstricting agents respond favorably to reversal of vasoconstriction with nitric oxide and calcium-channel blockers (10). Finally, the "no-touch" method of vein graft harvesting, in which the peri-vascular tissue is left in place, is used by some in an effort to improve long-term vein graft patency and may facilitate neural stimulus for spasm.

Taken together, clinical experience and laboratory studies suggest that in an environment of high adrenergic tone, systemic hypotension, platelet activation, and endothelial dysfunction, spasm of aortocoronary vein grafts is possible years after surgery and may produce clinical ischemic syndromes. In many prior cases, including the one described here, clinical symptoms of angina preceding coronary

FIGURE 2 Saphenous Vein Graft Patency 6 Years After Spasm Event



Angiogram of vein graft to the diagonal in right anterior oblique 13° cranial 35° position shows patency without angiographically significant stenosis

angiography improved or resolved with long-term vasodilator therapy (2,6,7), suggesting that vein graft spasm in these patients may represent a clinical event triggered during routine activity rather than simply an adverse effect of catheterization. Undetected spasm may be a mechanism for recurrent angina after CABG in the absence of angiographically significant graft disease. In addition, it is possible that spasm accompanied by endothelial disruption with thrombus formation is one mechanism for irreversible vein graft occlusion in the years after CABG. Investigation into multifactorial mechanism for spasm in the setting of modern surgical techniques and current medical therapy may elucidate strategies to prevent occult events and preserve long-term vein graft patency.

FOLLOW-UP

Six years later, the patient was referred for coronary angiography to evaluate progressive systolic heart failure. Coronary angiography revealed unchanged severe native vessel disease and patency of all 3 grafts (**Figure 2**, Video 4).

CONCLUSIONS

Aortocoronary vein graft spasm is a rare but clinically relevant entity that may occur years after CABG. Long-term vasodilator therapy seems useful to prevent the recurrence of vein graft spasm events. Future study is recommended to better characterize correlation between angiographic spasm events during cardiac catheterization and clinical angina syndromes or permanent vein graft occlusion.

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KEY WORDS coronary angiography, coronary artery bypass, percutaneous coronary intervention, ventricular fibrillation

APPENDIX For supplemental videos, please see the online version of this article.