

Vanishing Venous Coronary Artery Bypass Grafts after Sepsis

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The dehiscence of saphenous vein grafts (SVGs) is a rare, often fatal, complication of coronary artery bypass grafting (CABG). We present the case of a 57-year-old man who underwent hemiarch graft interposition and CABG for a Stanford type A aortic dissection. Five months after discharge, the patient developed streptococcal sepsis caused by a hemodialysis catheter. Complete rupture of the proximal anastomoses of the saphenous veins and containment by the obliterated pericardial cavity was observed 25 months after the initial operation. The patient was successfully treated surgically. This report describes a patient who developed potentially fatal dehiscence of SVGs secondary to infection and outlines preventive and management strategies for this complication.

Key words: 1. Saphenous vein graft
2. Infection
3. Graft rupture

Case report

A 57-year-old man was transferred to emergency department of Ajou University Hospital for treatment of Stanford type A aortic dissection. The patient had chronic kidney disease (serum creatinine, 1.8 mg/dL) and mild cognitive impairment (Mini-Mental State Examination score, 16) due to previous tuberculous meningitis. The initial electrocardiogram showed inferior myocardial infarction, complete atrioventricular block, and a temporary pacemaker that was inserted before transfer to our hospital. Emergency surgery was performed, and the operative findings showed intimal tearing at the proximal ascending aorta with occlusion of both coronary ostia by the flap. We performed hemiarch graft interposition with a Hemashield 22-mm graft and coronary artery bypass grafting (CABG) to the left anterior descending (LAD) ar-

tery and right coronary artery with saphenous vein grafts (SVGs). The patient was treated with hemodialysis during the early postoperative period, but was discharged on the 100th postoperative day without renal replacement.

Three months after discharge, the patient was admitted to the nephrology division to manage acute renal failure on preexisting chronic renal failure (serum creatinine, 4.3 mg/dL). A permanent hemodialysis catheter was inserted and hemodialysis was performed for 3 days. The patient's renal function improved gradually, and he was therefore discharged on the 11th day of hospitalization with a permanent hemodialysis catheter (serum creatinine, 2.1 mg/dL). Two months after discharge (8 months after first operation), the patient was admitted to the infectious diseases division to treat hemodialysis catheter-related sepsis. The patient's white blood cell (WBC)

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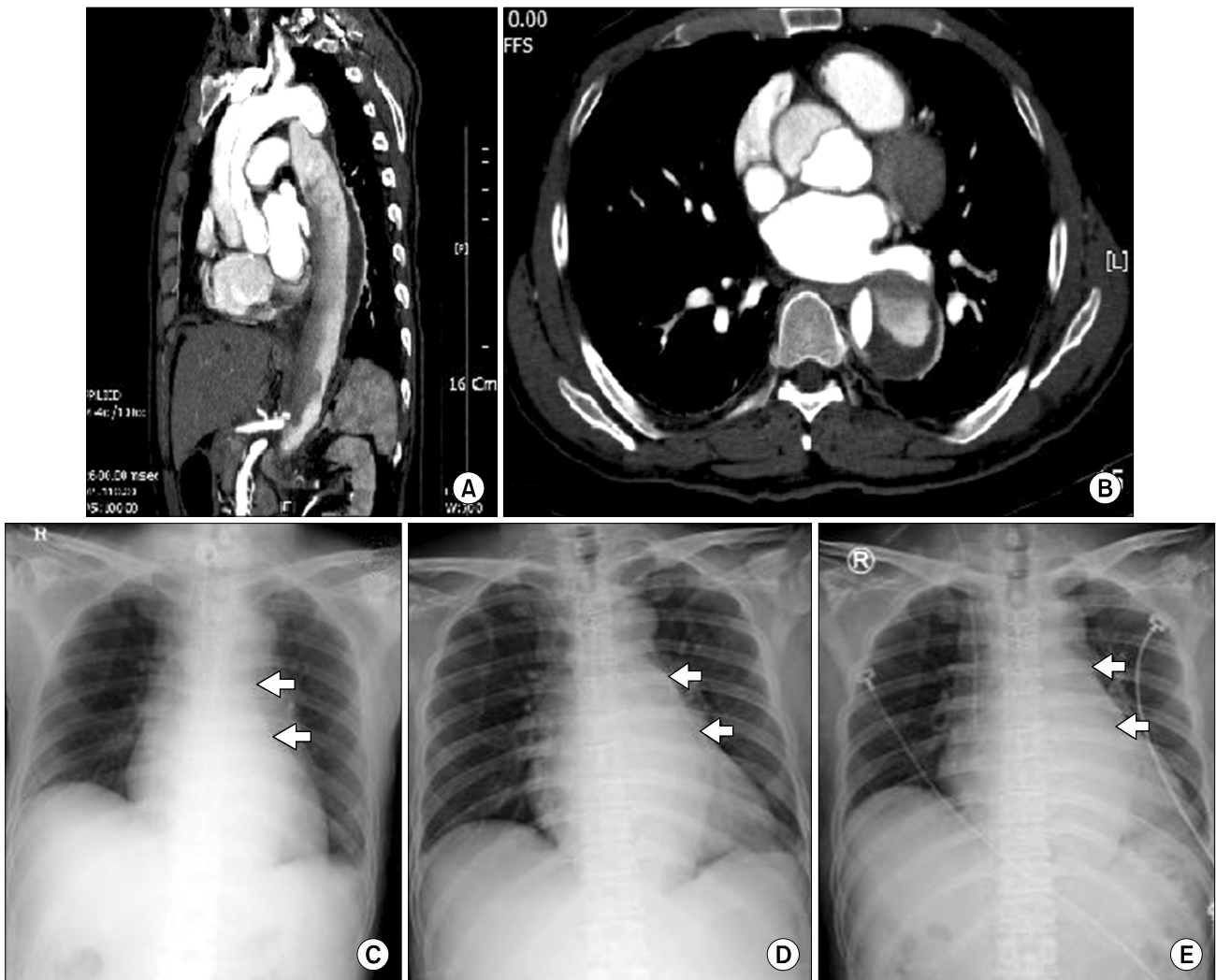


Fig. 1. (A) Preoperative computed tomography revealed a Stanford type A aortic dissection (sagittal plane). (B) Preoperative computed tomography revealed a Stanford type A aortic dissection (axial plane). (C) Chest X-ray after hemiarch graft interposition and coronary artery bypass grafting surgery. (D) Apparent mediastinal widening on the chest X-ray (arrow) during outpatient follow-up. (E) Chest X-ray before re-operation.

count, percentage of neutrophils, and C-reactive protein (CRP) level were 7,900/ μ L, 93.2%, and 26.02 mg/dL. The causative organism was *Streptococcus pyogenes*. The permanent hemodialysis catheter was removed and the patient was treated successfully with systemic antibiotics for 3 weeks.

Twelve months after that admission (21 months after first operation), the patient revisited our emergency department complaining of a 3-day history of general weakness and fatigue. The mediastinum was wider than had been observed in a previous study (Fig. 1), and contrast-enhanced chest computed tomography (CT) was performed (Fig. 2A). This re-

vealed a 5×4×5-cm hematoma alongside the ascending aorta graft. Contrast enhancement of the hematoma suggested rupture of the artificial graft, and we performed aortography to obtain a precise diagnosis. This showed the extravasation of contrast medium from the proximal portion of the ascending aorta and rapid flow of contrast medium in the pseudo-lumen alongside the ascending aorta (Fig. 2B). We performed an emergency explorative sternotomy under the impression of a rupture of the artificial graft.

The upper portion of the pericardium was bulging and resembled a pseudoaneurysm sac. Before opening the bulging pericardial sac, we started cardiopulmo-

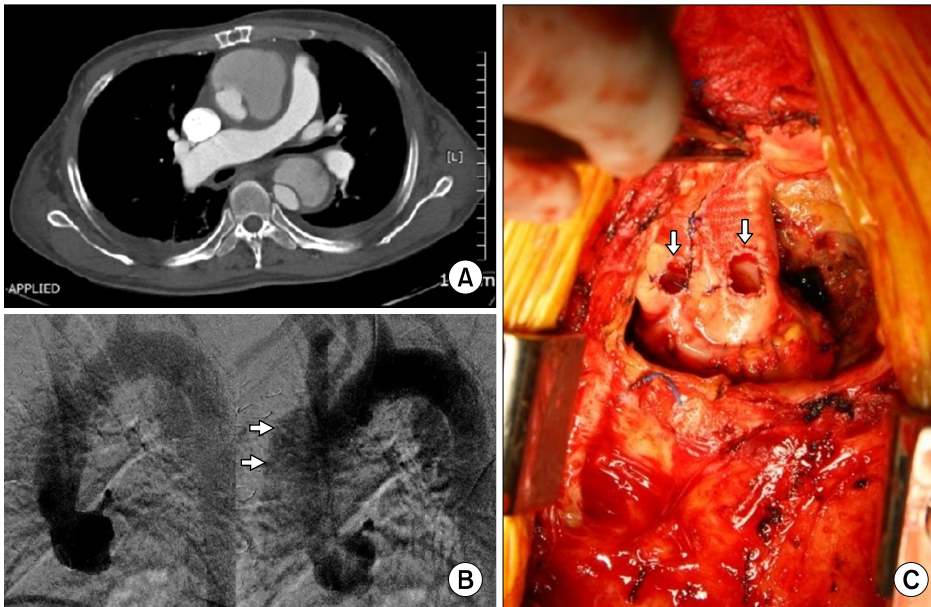


Fig. 2. (A) Preoperative computed tomography revealed a ruptured artificial ascending aorta. (B) Extravasation of the contrast medium from the ascending aortic graft (mid-segment) and rapid flow of the contrast medium in the pseudo-lumen alongside the ascending aorta. (C) Proximal anastomosis site of the saphenous vein grafts to the ascending aortic graft (arrows).

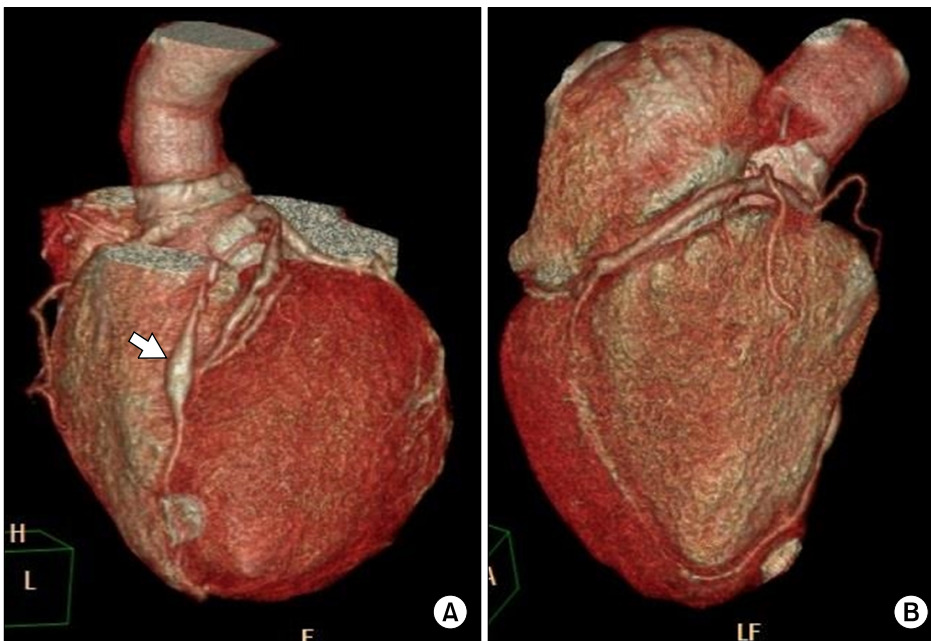


Fig. 3. (A) Obstruction of the SVGs from the ascending aorta to the distal LAD artery with 7.6 mm of ectasia at the distal SVG (arrow) and diffuse mild-to-moderate stenosis (up to 58%) at the proximal and middle LAD. (B) Obstruction of the SVG from the ascending aorta to the RCA with a 5.2 mm aneurysm at the distal RCA and discrete mild stenosis at the proximal RCA (34%). SVG, saphenous vein graft; LAD, left anterior descending; RCA, right coronary artery.

nary bypass and lowered the patient's temperature to 28°C. After opening the pericardial sac under total circulatory arrest, two oval defects were seen in the proximal portion of the artificial graft. The inner surface of the sac was smooth and clean, with no evidence of pus or current infection. The defects in the artificial graft were the sites of the proximal anastomoses of the SVGs from the previous CABG, but no visible SVGs were present in the operative field (Fig.

2C). The flow of the native coronary arteries was intact in preoperative aortography, and no newly developed regional wall motion abnormality was found on intraoperative transesophageal echocardiography. We resected the original artificial graft and interposed a new artificial graft. No evidence of coronary artery malperfusion was found while weaning the patient from cardiopulmonary bypass or during the postoperative period. Coronary artery multidetector

CT (MDCT) performed postoperatively revealed obstructed SVGs and ectatic changes of the distal portion of the SVG to the LAD artery (Fig. 3). The systolic function of the left ventricle and the flow in the native coronary artery were intact. The patient was discharged on the 42nd postoperative day.

Discussion

We are not certain of the exact cause of the SVGs dehiscence from the ascending aorta graft in this case. There are two possible causes: infection and aneurysmal changes in the saphenous vein and subsequent rupture.

The dehiscence of SVGs at the proximal anastomosis site is a rare complication of CABG that is usually fatal. In some cases, the blood is contained within the obliterated pericardial cavity [1,2]. Fatal dehiscence of proximal anastomosis can occur due to graft infection. Douglas et al. [3] reported a case with dehiscence of the proximal aorta graft anastomosis secondary to staphylococcal mediastinitis, pericarditis, and phlebitis, and a mycotic aneurysm was found in the mid-portion of the graft. Microscopically, a dense, partially organizing polymorphonuclear infiltrate was found covering the surface of the SVGs and heart [3]. The rupture of the SVGs into the dense pericardial cavity might also have been contained by mediastinal adhesions from the previous operations [1,2].

The first report of aneurysmal dilatation of aortocoronary SVGs was published in 1975, and the reported incidence is up to 14% of vein grafts 6–12 years after CABG [4,5]. The time interval between the operation and the detection of aneurysmal changes in the SVGs has been reported for range from 11 days to 21 years [4,5]. Various pathophysiologies have been proposed, but the mechanism of aneurysmal dilatation of aortocoronary SVGs is still unclear, although several causative factors have been suggested [4,5].

Saphenous vein graft aneurysms (SVGAs) have different pathophysiologies according to the timing of occurrence. Early SVGAs (appearing within several months after surgery) are generally related to graft infections, undetected varicosities, and some operative factors (e.g., technical factors related to graft harvesting and anastomosis) [1,5,6]. Late SVGAs (appearing more than 5 years after surgery) are thought to oc-

cur by atherosclerotic degeneration of the saphenous vein as the main cause, especially in patients with hyperlipidemia [4,5]. These atherosclerotic changes, as well as graft endothelial dysfunctions with changes in the medial smooth muscle cells around the valve sites, play a key role in late aneurysm development [5,6]. SVGAs are usually found incidentally on imaging as a simple mediastinal mass or an enlarging pulsatile mass [4,5]. In cases of rupture, fistula formation with surrounding structures and hemodynamic instability caused by compression of the adjacent cardiac and vascular structures are possible [1,5].

Our patient was admitted for sepsis related to a hemodialysis catheter 8 months after evidence of dehiscence or aneurysmal dilatation of the grafts. However, serial chest X-rays showed a subsequent gradual widening of the mediastinum, although this finding was neglected by several doctors who provided care to the patient during OPD follow up after first operation (Fig. 1D). The WBC count, percentage of neutrophils, and CRP were 11,300/ μ L, 83.1%, and 9.93 mg/dL, respectively, a week after a chest X-ray showing mediastinal widening was obtained from the patient. When the patient came to our emergency room, the WBC count, percentage of neutrophils, and CRP levels had decreased to 6,400/ μ L, 78.2%, and 0.46 mg/dL, respectively. From these data, we deduced that the SVGs were dissolved by a previous infection and that the unruptured sac was formed by a chronic infection.

We performed a second operation under cardiopulmonary bypass with femoral artery cannulation and hypothermic (28°C) total circulatory arrest to identify exact ruptured site. On operation table, we found no remnant of saphenous vein graft and there was no bleeding from surrounding tissue. A smooth-surfaced capsule-like space was found around the proximal ascending aorta graft, and 2 holes for the previous saphenous vein anastomoses were seen with no remnant tissue, except suture material. The infection seemed to have dissolved the saphenous veins. We resected the previous graft, including the coronary anastomosis sites and interposed a new graft. We initially attempted primary repair, but there was a risk of aortic stenosis. Therefore, we performed a segmental replacement with a new graft (22 mm, same size) to prevent aortic stenosis. We did not perform

CABG for the second operation because no evidence of myocardial ischemia was present in the preoperative evaluations and no newly developed regional wall motion abnormalities were observed on intraoperative transesophageal echocardiography. Postoperative coronary artery MDCT showed intact native coronary arterial flow (Fig. 3). The patient's postoperative course was uneventful, with no evidence of infection, including mediastinitis.

We report the very rare complication of saphenous vein graft dehiscence from an artificial ascending aorta after CABG with ascending aorta replacement. The cause of the dehiscence was likely infection caused by hemodialysis catheter-related sepsis. To detect such complications earlier, we must have a greater level of suspicion regarding abnormal findings on imaging or laboratory examinations and also perform regular echocardiography or CT imaging.

Conflict of interest

No potential conflict of interest relevant to this article was reported.

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