

Circulatory Failure and Lactic Acidosis Associated with Giant Saphenous Vein Graft Aneurysm

Rick Mooi¹, Carsten Arnoldussen² and Jos le Noble¹

- 1 Department of Intensive Care, VieCuri Medical Center, Tegelseweg, The Netherlands
- 2 Department of Radiology, VieCuri Medical Center, Tegelseweg, The Netherlands

Abstract

Context: Saphenous vein graft aneurysms may be a life-threatening condition. It may take 15-20 years to fully develop and is regarded as a relatively rare and late complication of coronary artery bypass grafting. Giant aneurysms may particularly cause mechanical complications due to neighboring structure compression. Simultaneous right atrial and ventricle compressions due to saphenous vein graft aneurysms as a cause of circulatory failure and lactic acidosis are rarely demonstrated in intensive care unit patients.

Case report: We hereby report a case of a 74-year-old patient who was diagnosed with circulatory failure that developed into acute cardiac failure with obstructive shock. The patient had undergone coronary bypass grafting 20 years previously. Computed tomography angiography revealed a right-sided 6.7 × 7.1 cm paracardial aneurysm, originating from the venous graft, compressing the right atrium and ventricle inflow tract.

Conclusions: We hypothesize that hemodynamic instability due to right ventricular inflow obstruction contributed to circulatory failure with consequently inadequate oxygen delivery and lactic acidosis. Despite immediate supportive treatment, the clinical condition deteriorated and a percutaneous intervention with a stent or surgical ligation with excision was not feasible thereafter. Saphenous vein graft aneurysms should be considered in patients with circulatory failure who have a history of coronary artery bypass grafting.

Keywords: Circulatory failure; Paracardial aneurysm; Saphenous vein graft; Lactic acidosis

Abbreviations: CABG: Coronary Artery Bypass Grafting; CTA: Computed Tomography Angiography; EVAR: Endovascular Abdominal Aneurysm Repair; ICU: Intensive Care Unit; SVGA: Saphenous Vein Graft Aneurysm

Corresponding author: Jos le Noble

 jlenoble@viecuri.nl

MD, PhD, Department of Intensive Care, VieCuri Medical Center, Tegelseweg 210, 5912 BL Venlo, The Netherlands.

Tel: +31 (0)77-320 55 55

Fax: +31 (0)77-320 6378

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Introduction

Saphenous vein graft aneurysm (SVGA) is usually a late complication of coronary artery bypass grafting. However, it is commonly unsuspected until any related symptoms appear. The spectrum of clinical presentation varies from asymptomatic to acute dyspnea, angina, and myocardial infarction or symptoms related to cardiac structure compression and death. Extra luminal compression of the right ventricle inflow tract can acutely or gradually occur, and the obstruction can be complete or partial [1].

SVGA, presenting with right-sided compression, as a cause of right ventricular tract inflow obstruction is very unusual, and a similar case has never been reported in literature.

Case Report

A 74-year-old patient with known ischemic cardiomyopathy and coronary artery bypass grafting (CABG; RCA MO1-RDP) 20 years prior was referred for coronary angiography. Past medical history revealed an endovascular abdominal aneurysm repair (EVAR) and right kidney nephrectomy. Transthoracic

echocardiogram 2 years before this admission showed no abnormalities.

The patient developed acute respiratory failure with mental changes and was admitted to the intensive care unit (ICU). The patient reported dyspnea for 3 days and chest discomfort on ICU admission. The vital signs showed that blood pressure (BP), pulse, respiration, oxygen saturation at room air, and temperature of 120/88 mmHg, irregular 130 beats/min, 30 cycles/min, 90%, and 36.7°C, respectively. Right- and left-sided cardiac failure was also found, which was evidenced by prominent jugular veins, congestive face, and peripheral cyanosis of the skin, with cold extremities and weak pulses.

Laboratory test results of the arterial blood sample showed that pH, pCO₂, pO₂, bicarbonate, CRP, BNP, and lactate levels of 7.11 (7.35-7.45), 3.4 kPa (4.7-6.4), 8.0 kPa (10.0-13.3), 16 mmol/L (22-29), 107 mg/L (<5), 3149 ng/L (<100), and 8.2 mmol/L (<1.3), respectively. Serum creatinine as an index of renal function and hemoglobin levels were 158 µmol/L (44-80) and 6.3 mmol/L (7.5-10), respectively.

The chest X-ray revealed an abnormally enlarged heart and pulmonary edema. Electrocardiogram revealed atrial fibrillation with a ventricular rate of 143/min, an intermediary heart axis, and normal atrioventricular junction conduction. Q-waves in leads II, III, and AVF, and ST-depression in V3-V6 were found.

The patient was suspected of having unstable angina and underwent emergent cardiac catheterization, which demonstrated no severe stenosis or culprit lesions, but a large paracardial mass on the right side of the heart. Consecutive bedside echocardiography confirmed the presence of a right-sided, paracardial mass (**Figure 1**). Systolic function parameters, including left ventricle ejection fraction, were decreased compared with those in the previous routine echocardiograms and showed no significant valvular heart disease. Color Doppler flow revealed the existence of blood flow inside the cardiac mass. Computed tomography angiography (CTA) was eventually performed, which demonstrated a right-sided 6.7 × 7.1 cm paracardial aneurysm, originating from the venous graft and compressing the right atrium and ventricle inflow tract (**Figure 2**).

Hypotension persisted despite aggressive initial fluid resuscitation, inotropic support, and supportive measures due to imminent obstructive shock. The patient's clinical condition deteriorated.

We decided to rapidly transfer the patient to a specialist cardiothoracic center for further evaluation and treatment. Unfortunately the patient died as a direct consequence of obstructive shock shortly afterwards. No consent for autopsy was obtained.

Discussion

CABG is the standard surgical procedure for the treatment of advanced coronary disease. Despite its success, the long-term outcome of coronary bypass surgery is strongly influenced by the condition of the vascular conduits, and its use is limited by poor long-term survival [1]. Late complications of vein graft

atherosclerosis and restenosis are well known, but the unique presentation of our patient with right atrial compression and right ventricle inflow tract compression causing obstructive shock has not been described in literature before.

Much of the current literature on SVGA indicates that patients remain often asymptomatic for a long time or even until death from another cause. This shows that SVGAs are often an incidental finding during regular follow-up or on routine chest radiography or CT of the thorax [1]. The time of SVGA presentation may vary greatly, but previous case studies noted a delay ranging between 7 days and 21 years after surgery. Most of the SVGAs develop 10-15 years after CABG. The difference is remarkable in the prevalence between men and women. A recent systemic

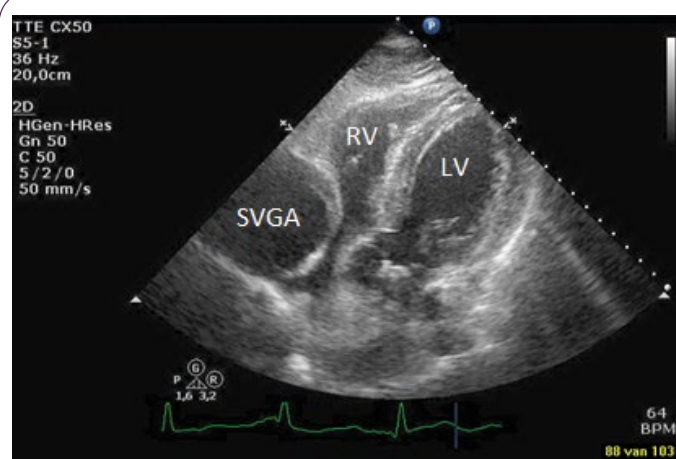


Figure 1 Bedside echocardiography with subcostal view that shows the SVGA compressing the right atrium and ventricle inflow tract.

RV: Right Ventricle; LV: Left Ventricle; SVGA: Saphenous Vein Graft Aneurysm



Figure 2 CT-angiography, confirming the mass to be SVGA compressing the right ventricle inflow tract.

LV: Left Ventricle; SVGA: Saphenous Vein Graft Aneurysm

literature review concluded that 87% of the SVGA cases were found in male patients [1]. Our patient fits these statistics; however, signs of mechanical complications during a patient's first presentation are rarely seen [2,3].

The data regarding the cause and development of aneurysms are limited because systematic studies are lacking. Small case studies indicate that trauma to the vessel during the harvesting, either accidental or using a wrong harvesting technique, may be one of the causes. Other risk factors for SVGA include atherosclerosis and atherosclerosis contributors, such as hyperlipidemia and hypertension. Pre-existent venous wall weakness and infections leading to proximal or distal anastomosis disruption may also be etiological factors [4,5].

The diagnostic approach for a patient with a suspected SVGA should include specific imaging of the aneurysm and obtaining information of the hemodynamic consequences and coronary blood flow. These imaging techniques include echocardiography, angio-computed tomography, and coronary angiography, and other techniques, such as magnetic resonance imaging and transesophageal echocardiography, could supply additional information [1,6]. Knowing that not all SVGAs can be properly imaged using diagnostic echocardiography is important; several studies confirmed misdiagnosing SVGAs when only echocardiography is used [7,8].

The preferred treatment of SVGA is surgery. Studies comparing surgical versus conservative treatment showed that patients who

underwent surgery had lower mortality rates [1,5,9]. Exclusion or resection of the aneurysm and revascularization of the coronary blood flow are the treatment goals.

Embolization or stenting of the aneurysms is another treatment option [10,11]. Unfortunately, our patient deteriorated rapidly to provide the surgical treatment he needed.

Our uncommon clinical images illustrate the features of symptomatic SVGA presenting right atrial compression and right ventricle inflow tract compression. Most SVGAs are incidentally found on routine check-ups; however, the prevalence is likely to increase with increasing trends in the use of chest CT imaging. Progression of asymptomatic SVGA may occur over the years. It only rarely leads to irreversible hemodynamic derangements as presented by the patient in this case report.

The learning points of our case indicate that SVGA is a late complication in patients with CABG history and can eventually cause hemodynamic derangements due to compression of important cardiac structures, such as the right atrium and ventricle inflow tract. Acute treatment may be required although this is an uncommon presentation.

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