Case Letter

Delayed intervention in a giant coronary bypass saphenous graft aneurysm: a case report

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The incidence of saphenous vein graft aneurysms (SVGAs) after coronary artery bypass grafting (CABG) is approximately 0.07%;[1] however, the true incidence is likely underreported because of their frequent incidental discovery.[2] Due to its rarity, knowledge mainly comes from case reports and small case series, though some decision algorithms have been proposed in systematic reviews. These algorithms have evolved over time as the body of accumulated knowledge has grown. Initial reports established that there was no survival advantage with early surgical treatment versus a conservative approach.[1] Eighteen additional years of observational work revealed that there is a nearly linear relationship between SVGA size and the risk of complications. Most of these events occur in SVGAs over 40 mm in diameter, regardless of the clinical presentation.[3] Thus, the proposed treatment of choice (watchful waiting, surgical or percutaneous) depends on the patient and aneurysm characteristics. However, a noninterventional approach should be discouraged and limited to cases of an incidental finding of a true small aneurysm in which a percutaneous intervention is not feasible and the surgical risk is considered excessive.[3] We describe the case of an asymptomatic patient with a giant, growing, SVGA, incidentally discovered 10 years earlier and managed conservatively, with uncommon imaging documentation of its progression over time.

We report the case of a 73-year-old man with a history of previous smoking, dyslipidaemia, type 2 diabetes mellitus, atrial fibrillation, peripheral arterial disease and chronic obstructive pulmonary disease. At the age of 46, due to triple-vessel coronary artery disease, he underwent triple CABG of the left internal mammary artery to left descending artery and saphenous vein grafts (SVGs) from the aorta to obtuse marginal artery (OMA) and the posterior descending artery.

Seventeen years after CABG, the patient was incidentally diagnosed with a SVGA through a chest X-ray (CXR) (Figure 1A), which was further confirmed by a computed tomography coronary angiography (CTCA) scan (Figure 1B). It was a pseudo-aneurism, related to the SVG-OMA bypass, and had a maximal diameter of 66 mm and minimal contrast filling due to extensive thrombosis in its cavity. An invasive coronary angiogram was performed, revealing SVG patency, with no filled aneurysm-like structure, confirming the aneurysm was occluded and excluded from the coronary circulation. Given the asymptomatic status of the patient and extensive thrombosis, a conservative approach was chosen, focusing on antithrombotic therapy with warfarin due to atrial fibrillation, anti-anginal treatment with carvedilol and amlodipine, and cardiovascular risk management, including diabetes control with metformin and dyslipidemia management with simvastatin, along with close follow-up. The follow-up CXR and CTCA two years later revealed that the SVGA had grown to 75 mm (4.5 mm/year) (Supplementary Figure 1). However, the Heart Team maintained the management plan due to the patient's asymptomatic status and lack of mechanical complications. Another radiological reevaluation four years later revealed that the aneurysm had grown to 80 mm (1.25 mm/year, imaging finding is not displayed here). Given the stable clinical condition, the watchful waiting approach was continued.

Four years later, at the age of 73 years, the patient was admitted to the emergency department with

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sudden chest pain and dyspnea. On admission, he was tachypnoeic, unable to tolerate supine position, with 95% oxygen saturation on 2 L/min of supplemental oxygen. Blood pressure was 98/70 mmHg (1 mmHg=0.133 kPa), heart rate 80 beats/min. Pulmonary auscultation showed an absent vesicular murmur in the lower left hemithorax and crepitations in the right. Mild bilateral ankle oedema was present. Arterial blood gas analysis showed a normal pH of 7.37, balanced gas levels, and normal lactate. Blood tests revealed normal haemoglobin and leukocyte counts, with a mildly elevated C-reactive protein (CRP) levels (1.3 mg/dL; normal < 0.5 mg/dL). N-terminal pro B-type natriuretic peptide was elevated (4,958 pg/mL, normal <125 pg/mL), as was high-sensitivity troponin T (44 ng/L, normal <14 ng/L), though no significant variation was noted. The electrocardiograph (ECG) showed sinus rhythm with inverted T waves in leads DI and aVL, similar to the previous findings. The CXR revealed a towel-like opacity in the lower two-thirds of the left hemithorax, hiding the known SVGA mass (Figure 2A). A transthoracic echocardiogram revealed normal left ventricular size and function and a heterogeneous mass in close relation to the left ventricular lateral wall. The CTCA showed the pseudo-aneurysm increased to 94 mm (3.5 mm/year) without contrast filling, compressing surrounding structures, including the left pulmonary veins, left pulmonary artery branch, and part of the left lung (Figure 2B-D). A pleural effusion suggestive of hemothorax caused near-complete collapse of the ipsilateral lung and no active bleeding was found. The Heart Team determined that the actual clinical scenario was caused by the mechanical effect of the aneurysm in the affected structures. His age and comorbidities posed a high surgical risk. The extensive aneurysm thrombosis, lacking a discernible neck on angiography, ruled out percutaneous closure, which would not resolve the compressive symptoms. Thus, it was decided that surgery should be performed to repair the pseudo-aneurysm.

During surgery, the giant pseudo-aneurysm was found to push the heart anteriorly and to the right. After dissection of the SVGA, dark blood and clots were aspirated, decompressing the mass. Upon opening the left pleura, a hemothorax rapidly developed, which was impossible to stop, leading to refractory hypovolemic shock and death. However, it was possible to isolate the mediastinum and there was no bleeding from the aneurysmal sac during surgery.

SVGAs are rare and highly heterogeneous late complications of CABG, with an average diagnosis time of 13 years post-surgery.[3,4] Aneurysms are classified as true (involving all three vessel wall layers) or pseudo-aneurysms (involving disruption of at least one layer).[3] In terms of the presentation of SVGAs, 27.4% of cases are incidental findings, half of the patients show cardiovascular symptoms, and a few cases involve life-threatening conditions.[3-5] The average size of SVGAs reported in the literature was 61.8 mm.[3] Aneurysms are widely known to grow over time at an unknown rate.[4,6] There is a nearly linear correlation between SVGA size and adverse outcomes, with a marked increase in events for aneurysms larger than 40 mm.[3] These size threshold should be of greater concern, particularly in the case of pseudo-aneurysms, which have more fragile walls, making them prone to rapid enlargement and an increased risk of rupture.[3]

Currently, there are three types of management:

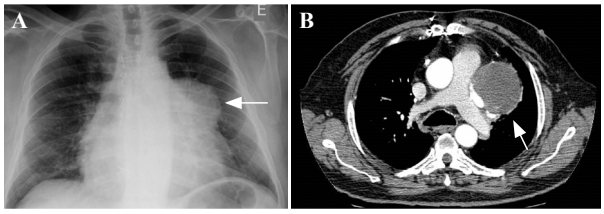

Figure 1. Saphenous vein graft aneurysms (SVGA) incidental finding. A: initial chest X-ray with the incidental finding of a mass (arrow indicated) silhouetting the heart; B: initial computed tomography coronary angiography showing a pseudo-aneurysm (arrow indicated) of the saphenous vein grafts from the aorta to obtuse marginal artery (SVG-OMA) bypass with a maximum diameter of 66 mm and extensive thrombosis, as there is minimal contrast within its lumen.

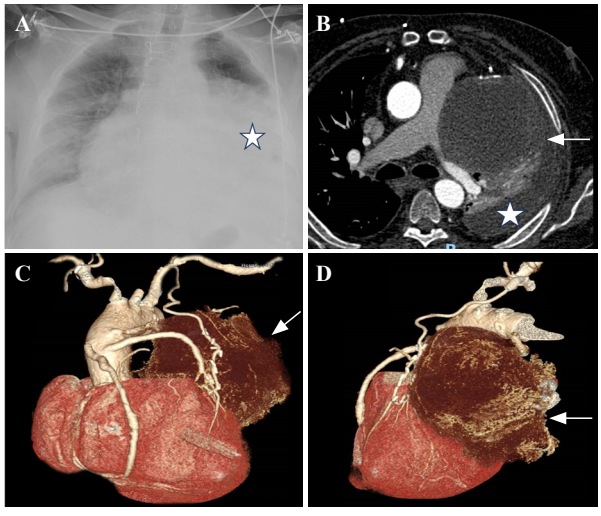

Figure 2. Saphenous vein graft aneurysms (SVGA) ten-year post-diagnosis imaging evaluation. A: chest X-ray 10 years after Figure 1A depicting pleural effusion in the lower two-thirds of the left hemithorax (star indicated), obscuring the mass; B: computed tomography coronary angiography (CTCA) performed 10 years after Figure 1B showing an increase in the pseudo-aneurysm size to 94 mm (arrow indicated), and suspected left hemothorax; C and D: CTCA 3D multiplanar reconstruction demonstrating a giant SVGA (arrow indicated) in close relation with lateral and posterior left ventricular walls.

conservative medical treatment (risk factor modification and surveillance), surgical (aneurysmal resection or ligation) and percutaneous treatment (coil embolization, Amplatzer vascular occlusion, or covered stent placement).[3]

The first case series reviewed 13 patients and concluded that early surgical intervention offered no survival advantage over a conservative approach.[1] Another case series of 16 surgically treated patients showed 83% survival at five years, supporting prompt repair for symptomatic patients.[5] In 2012, Ramirez et al[4] performed the first systematic review of 209 published cases. The most common strategy was surgical repair (58.4%), followed by conservative management (20.1%). The conservative strategy had the highest 30-day mortality rate (23.8%), followed by surgery (13.9%).

Cumulative knowledge from case reports, case series, and systematic reviews, along with advances in imaging and percutaneous treatments, has led to an updated review and a decision-making algorithm for managing this complex condition, based on 342 cases.[3] This review revealed a decline in surgical approaches, though it remains the preferred treatment in 52.6% of patients. Percutaneous intervention ranked second, performed in 23.3% of cases, whereas a conservative approach was used in 19% of patients, mainly for high-risk surgical patients. With advances in the interventional techniques, this review suggests that a conservative approach should be discouraged in patients suitable for treatment. The proposed algorithm prioritizes clinical presentation, followed by aneurysm type, graft patency, myocardial viability, and surgical risk. Non-interventional management should be reserved for cases where percutaneous intervention is unfeasible, and where the degree of surgical risk is high. However, this approach could be cautiously considered for asymptomatic, small true SVGA (under 40 mm), with a follow-up every 3 months.[3]

Situations such as the one we had, with an asymptomatic, incidentally found large aneurysm without mechanical complications, are underrepresented in the literature, making optimal treatment timing and approach more challenging. Recent research, unavailable during the initial presentation, indicates that surgery would have been the best option due to the large SVGA size (66 mm), its pseudo-aneurysm type, and unsuitability for a percutaneous approach. Earlier surgical referral could have allowed repair before symptoms developed, when the surgical risk was also lower. However, through a watchful waiting strategy, we were able to document changes in size and symptoms over time using various imaging methods. This allowed us to demonstrate the absence of a stable growth rate and the inability to predict changes in size over time. This case underscores the established concept that aneurysms grow at an unpredictable and inconsistent rate.

In summary, our case highlights that a non-interventional approach should be discouraged in patients eligible for treatment before the aneurysm reaches a larger, potentially life-threatening size. Therefore, for aneurysms larger than 40 mm, an interventional approach should be pursued regardless of symptoms or complications.

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