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CASE REPORT
Cardiovascular imaging

Massive left ventricular pseudoaneurysm presenting as dysphagia: a case report

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Background Left ventricular pseudoaneurysm is a recognized, however, uncommon presentation of acute myocardial infarction in the current era. This is due to early reperfusion therapy for acute myocardial infarction. Left ventricular pseudoaneurysm after myocardial infarction can present in a variety of ways, including heart failure, chest pain, and dyspnoea.

Case summary We present a case of a 61-year-old male who presented with extremely atypical symptoms of dysphagia and weight loss due to a massive left ventricular pseudoaneurysm. Transthoracic echocardiogram and computed tomography revealed a large pseudoaneurysm causing mass effect on multiple gastrointestinal organs. Organic causes for dysphagia and weight loss were ruled out by gastroscopy. Surgical management was carried out but was ultimately unsuccessful.

Discussion Despite the heterogeneity in presentation for patients with left ventricular pseudoaneurysm, rapid diagnosis is important for management and prognosis. Diagnostic tools include transthoracic echocardiography, computed tomography, and cardiac magnetic resonance imaging. Management is usually surgical; however, there is some debate in the literature regarding conservative vs. surgical management for chronic pseudoaneurysms. More data are needed to determine optimal management strategies and prognosis for patients with left ventricular pseudoaneurysms.

Keywords Myocardial infarction • Pseudoaneurysm • Echocardiography • Computed tomography • Case report

ESC Curriculum 2.2 Echocardiography • 2.1 Imaging modalities

Learning points

- To recognize left ventricular pseudoaneurysm as an uncommon complication of acute myocardial infarction that has heterogenous clinical presentations.
- To highlight that dysphagia and weight loss is a possible presentation of left ventricular pseudoaneurysm due to mass effect.

Introduction

Ventricular pseudoaneurysm is an uncommon complication of acute myocardial infarction (MI), with an incidence of 0.23%.[1] A left ventricular pseudoaneurysm (LVP) is when a left ventricular wall rupture is contained by adherent pericardium, scar tissue, and/or thrombus.[2] This differs from a true aneurysm, as LVP is formed when intracardiac rupture is contained by the pericardium, thrombus, and/or pericardial adhesions without any myocardium. Although LVP can present in many ways, most of the time, cardiac symptoms are present. We present a case of LVP presenting with extremely atypical gastrointestinal symptoms.

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Timeline

Day 1	Patient presents to the emergency department with 8 months of dysphagia, intermittent vomiting, weight loss, and progressive shortness of breath.
Day 2	Transthoracic echocardiogram (TTE) reveals massive LVP. Computed tomography (CT) of chest/abdomen/pelvis shows large LVP causing mass effect, displacing the diaphragm inferiorly, and partly distorting the liver, gastric fundus, and oesophagus.
Day 3	Consultation with cardiology and cardiovascular surgery.
Day 5	Coronary angiogram performed, which demonstrates left circumflex artery (LCx) artery with mid-occlusion and small distal reconstitution and dominant right coronary artery (RCA) with mid-occlusion.
Day 6	Cardiovascular surgery and initiation of extracorporeal membrane oxygenation (ECMO).
Day 9	Patient unable to be weaned from ECMO due to continuing refractory biventricular dysfunction and cardiogenic shock with end-organ dysfunction. The patient passed away on post-op Day 3.

Case presentation

A 61-year-old male Caucasian presented to the emergency department with 8 months of progressive dysphagia (liquids and solids) with intermittent vomiting, 50 pounds of unintentional weight loss, and progressive shortness of breath on exertion. He also had intermittent chest heaviness for 1 month when his symptoms started. There were at least four episodes at rest, lasting several hours each time. There was no history of fevers, chills, or night sweats. There was no pre-syncope, syncope, or palpitations. There was no overt evidence of gastrointestinal bleeding. Medical history included gastroesophageal reflux disease, hypertension, and dyslipidaemia. His medications were rosuvastatin 20 mg PO daily, irbesartan 300 mg PO daily, and rabeprazole 20 mg PO daily.

His vital signs were normal at rest. Cardiovascular examination revealed normal first and second heart sounds, a soft grade 1/6 holosystolic murmur at the left lower sternal border and apex, and a left parasternal heave. Jugular venous pressure was normal and bibasilar crackles were noted.

Laboratory investigations revealed a normal white blood cell count, a normocytic anaemia with haemoglobin 101 g/L [reference range (RR) 140–180], mean corpuscular volume 92 fL (RR 80–92), and thrombocytopenia with platelets 109×10^9/L (RR 150–250). Electrolytes, urea, creatinine, liver function tests, lipase, and gamma-glutamyl transferase were normal. C-reactive protein was elevated (51 mg/L; RR 0–8). Serial high-sensitivity troponin T levels were elevated (112, 117, and 121 ng/L, RR <14). *N*-terminal pro-brain natriuretic peptide was significantly elevated (13 440 ng/L, RR <300). Electrocardiogram showed low voltages in the limb leads with inferior Q waves, borderline inferior ST elevations, and T-wave inversions (*Figure 1*). Chest radiograph showed

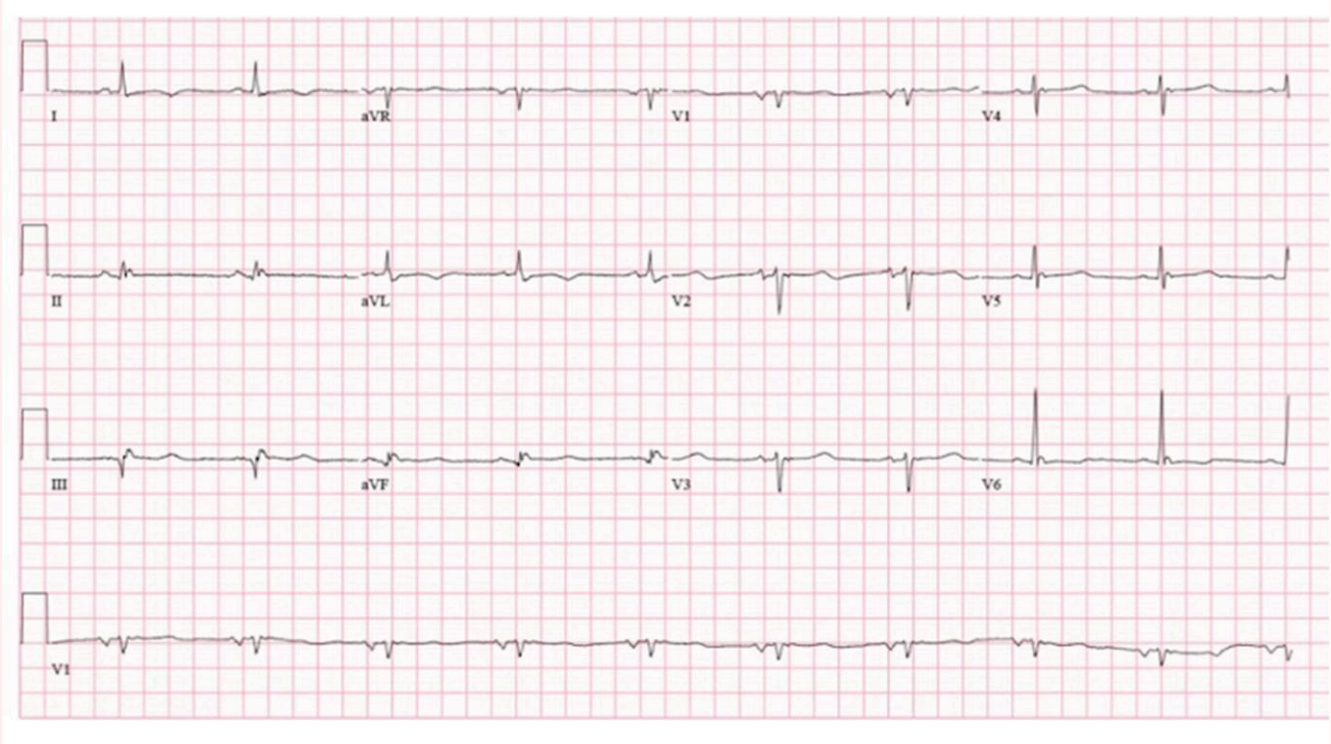

Figure 1 A 12-lead electrocardiogram showing low voltages in the limb leads with inferior Q waves, borderline inferior ST elevations, and T-wave inversions.

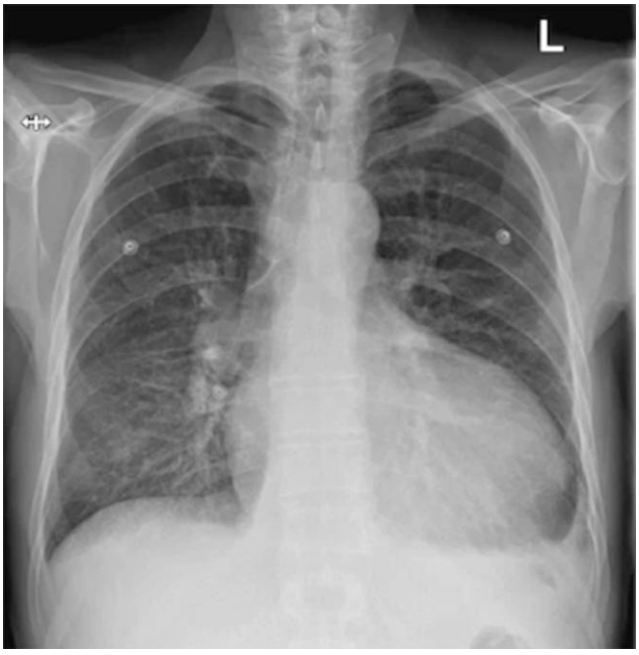

Figure 2 Chest radiograph (anteroposterior projection) demonstrating cardiomegaly, mild interstitial oedema, and small bilateral pleural effusions.

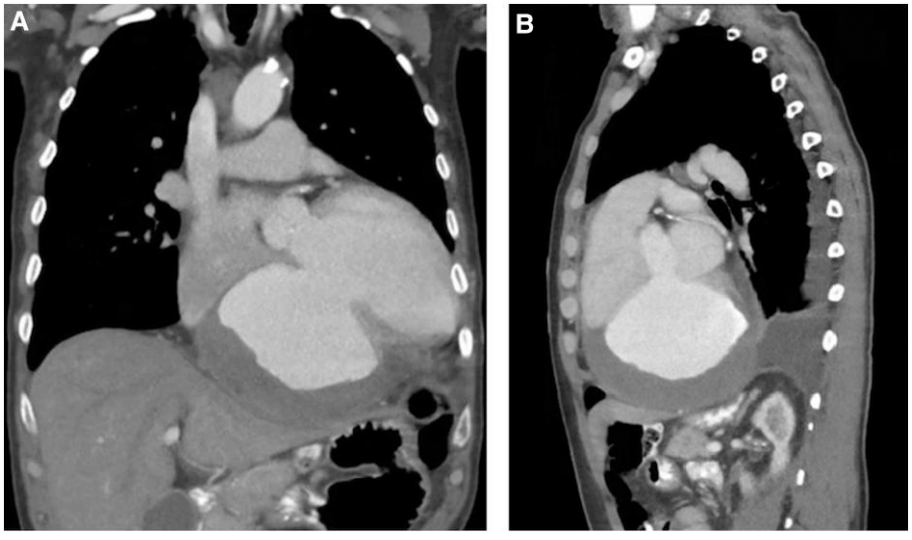

Figure 3 Computed tomography chest/abdomen/pelvis scan in coronal (*A*) and sagittal (*B*) planes showing large left ventricular pseudoaneurysm causing mass effect, displacing the diaphragm inferiorly, and partly distorting the liver, gastric fundus, and oesophagus.

cardiomegaly, mild interstitial oedema, and small bilateral pleural effusions (*Figure 2*). He was admitted to hospital and treated with intravenous diuretics for new onset heart failure felt to be due to a late presentation MI.

TTE revealed a moderately dilated LV with an end-diastolic diameter of 6.7 cm and moderately reduced LV systolic function with an ejection fraction of 33% and end-diastolic volume index of 84.8 mL/m^2. The inferior wall was akinetic and had a 3.5 cm basal inferior wall defect with a very large (~11 × 8 cm) LVP (see supplementary material online, *Video S1*). This was lined with 2.4 cm of echodense material suggestive of thrombus. The right ventricle was also moderately dilated with severely reduced systolic function. There was mild mitral regurgitation, mild tricuspid regurgitation, and severe pulmonary hypertension with a right ventricle systolic pressure of 60 mmHg.

The CT scan of the chest, abdomen, and pelvis was arranged to rule out concomitant malignancy. There was no malignancy, but it showed a

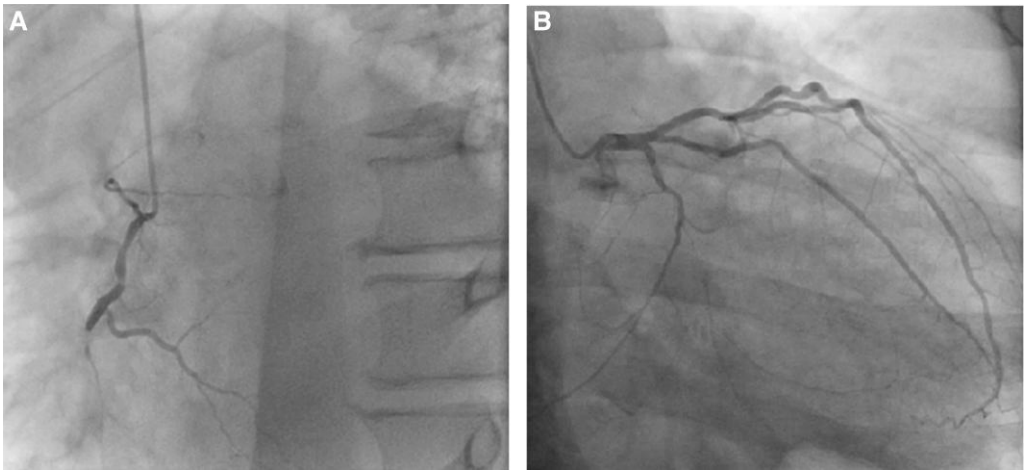

Figure 4 (*A*) Selective coronary angiography of the right coronary artery showing dominant right coronary artery with mid-third occlusion. (*B*) Selective coronary angiography of the left coronary system showing left circumflex artery with mid-occlusion and small distal reconstitution, and left main and left anterior descending with no significant obstructive coronary artery disease.

large LVP causing mass effect, displacing the diaphragm inferiorly, and partly distorting the liver, gastric fundus, and oesophagus (*Figure 3*). Upper endoscopy revealed no obvious intrinsic structural cause for dysphagia or anaemia. Coronary angiography demonstrated a dominant RCA with mid-third occlusion. The LCx also had mid-occlusion with small distal reconstitution. The left main, left anterior descending, and ramus branches had minor wall irregularities only (*Figure 4*).

In consultation with cardiovascular surgery, a decision was made for surgically management of the patient's LVP by performing LV pseudoaneurysm resection and endoventricular circular patch plasty of the inferior wall. Cardiac surgery was performed with institution of cardiopulmonary bypass and hypothermia. The inferior LV showed transmural infarction with ~1 L of clot near the inferior pericardium. The ventricular defect was ~3 cm at the basal inferior wall. A door-type patch was sutured to create a neo-inferior wall and the aneurysm sac was resected.

Unfortunately the patient was unable to be weaned from cardiopulmonary bypass after surgery. ECMO was initiated in the operating room. The patient could not be weaned from ECMO due to ongoing biventricular failure despite successful surgical repair. In the cardiovascular intensive care unit, he had continuing refractory biventricular dysfunction and cardiogenic shock with worsening lactic acidosis and end-organ dysfunction. He unfortunately passed away 3 days after surgery.

Discussion

LVP is uncommon in the modern era, and the incidence has decreased over the last 40 years primarily due to early reperfusion in acute MI. In one study, the rate of cardiac rupture in a 5-year period decreased from 6.2% in 1982% to 3.2% in 2006.[2] Acute MI accounts for over half of LVP cases (55%).[3] Other common causes are cardiac surgery (33%) and trauma (7%). Pseudoaneurysms have a higher rate of ventricular rupture when compared with true LV aneurysms. Thus, an urgent management plan is paramount once LVP is diagnosed.

The most common presentation in LVP is congestive heart failure (36%), chest pain (30%), and dyspnoea (25%).[4] Stroke from LV thrombus is also possible. However, patients can have non-specific clinical presentations or be asymptomatic and at times the diagnosis is made incidentally. The incidence of asymptomatic patients is varied in the literature, ranging from 10 to 48%.[3,5]

Dysphagia is an extremely uncommon symptom of LV pseudoaneurysm. McIlmoyle *et al.*[6] previously described this entity in a 60-year-old patient presenting with dysphagia and forty pound weight loss 4 months after acute posterolateral MI. The patient was managed medically due to prohibitive surgical risk. Autopsy revealed a massive (12 × 13 × 15 cm) pseudoaneurysm compressing the oesophagus. In our case, the patient had multiple symptoms which accounted for his presentation, with the predominant symptom being dysphagia. Intrinsic causes of dysphagia were ruled out by upper endoscopy and CT revealed an obstructive cause with mass effect on the oesophagus. Dysphagia due to LVP also accounted for our patient's 50 pound unintentional weight loss and vomiting.

Rapid diagnosis of LVP is important for management and prognosis.[7] Diagnostic imaging tools include left ventricular angiography, TTE, transoesophageal echocardiography, and cardiac magnetic resonance imaging (CMRI). LV angiography has been done historically; however, this is an invasive procedure which also carries risk of displacing the thrombus/fibrinous material in the LVP with contrast injection.[8]

TTE is the preliminary non-invasive imaging modality used for detecting LVP. A narrow neck, thin wall, and subsequently wide apex are the hallmark findings of LVP.[7] However, if there is poor endocardial visualization, cases of LVP can be missed.[4] CMRI is non-invasive imaging modality that is not limited by technical imaging factors. CMRI can identify normal and abnormal myocardium, thrombus, and characteristics of the pericardium. It also accurately localizes the site of the LVP with a sensitivity of 100% and a specificity of 83%.[9] In our case, there was excellent visualization of the LVP by TTE and CT scan and therefore CMRI was not pursued. Furthermore, CMRI would not have changed management since the size and potential risk of rupture of this LVP required surgical management as the optimal technique.

Management of acute LVP is usually surgical, since the annual risk of rupture is usually greater than the risk of surgery.[8] There is debate regarding surgical vs. conservative management for chronic LVPs. In one small study, nine patients with LVPs were managed conservatively due to prohibitive surgical risk with a mortality of 36% at 3.8 years with no deaths.[10] However, these patients had LVPs with small communicating orifices (mean 9.8 mm), suggesting conservative management could be considered in these cases. More data are needed to

determine optimal management strategies and prognosis for patients with LVPs.

Conclusion

LVPs are an uncommon complication of acute MI, especially in the era of timely revascularization. Dysphagia and weight loss is an extremely rare presentation of LVP. We describe a patient presenting with dysphagia, weight loss, and progressive shortness of breath due to a massive LVP causing mass effect on the oesophagus. A very high index of suspicion is needed to diagnose LVP presenting with non-specific signs and symptoms.

Lead author biography

Ashar Pirzada is currently a third year cardiology resident and chief cardiology resident at Dalhousie University in Halifax, NS, Canada. He received his undergraduate, masters, and medical degree at Memorial University in NL, Canada. He has a special interest in advanced echocardiography, heart failure, and medical education.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient and the patient's next of kin in line with COPE guidance.

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