Unusual Presentation of a Rare Coronary Disease with Unexpected Complications: Giants with Mass Effect

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ABSTRACT

Coronary artery aneurysms (CAAs) are very rare findings in patients undergoing CT coronary angiography (CTCA) for suspected ischemic heart disease (IHD). CAAs involving more than one coronary artery are even rarer. We, herein, report a case of incidental finding of giant CAAs (GCAAs) that affected three major coronary arteries with mass effect on the adjacent structures. A 56-year male patient presented with chest pain, shortness of breath on exertion, weakness, and history of pre-syncopal or syncopal episodes. Transthoracic echocardiography (TTE) was performed which showed a suspicious mass behind left atrium. CTCA revealed multiple GCAAs involving left anterior descending artery (LAD), left circumflex artery (LCX), and its obtuse marginal (OM) branch and right coronary artery (RCA). Some of these aneurysms were partially thrombosed while others were almost completely thrombosed. There was evidence of moderate pericardial effusion also. Multiple giant thrombosed aneurysms of more than one coronary artery are very rare. To prevent potential complications, the only recommended treatment is surgical intervention.

Key Words: *Coronary artery aneurysms, Angina, Syncopal episodes, Giant coronary artery aneurysms*.

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INTRODUCTION

Coronary artery aneurysms (CAAs) are, by definition, "coronary arterial dilatations which exceed the diameter of normal adjacent segments or the diameter of the patient's largest coronary vessel by 1.5 times or 150%."[1] Although there is no agreed-upon definition, a scientific statement from the American Heart Association on Kawasaki disease proposed that CAAs with a diameter >8 mm could be called large CAAs or giant CAAs (GCAAs). Other authors suggested considering CAAs as ''giant'' if the diameter was >20 mm.[1]

The right coronary artery (RCA) is frequently affected, with the left anterior descending (LAD) artery being the next most commonly involved, and this pattern has shown a higher incidence in males.[2,3]

This case report aimed to present an atypical manifestation of a rare coronary artery disease along with unforeseen complications.

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CASE REPORT

A 56-year male patient presented to the cardiology outpatient department with angina pectoris, weakness, and shortness of breath on exertion. On further questioning, he reported pre-syncopal and syncopal episodes. His comorbidities included treated hypertension and a history of haemorrhoids with intermittent per-rectal bleeding. There was no history of recent infections, cardiac disease or any cardiac interventional procedure, and use of recreational drugs.

On examination, there were no marfanoid or connective tissue disorder features.

His blood tests showed anaemia (haemoglobin, 9.8 g/dL and MCV=77.6 fL) with normal renal and liver function tests. His C-reactive protein was raised (65.2 mg/L).

On 12 leads ECG, T-wave inversions were noted in lateral leads (I, aVL) (Figure 1).

Transthoracic echocardiography (TTE) was performed which showed normal-sized cardiac chambers, preserved left ventricular function with no regional wall motion abnormalities, and no significant valvular disease. There was an incidental finding of a suspicious echogenic mass adjacent to the left atrium in the atrioventricular groove measuring about 4×4.7 cm, which was causing compression of the left atrium. A thin rim of pericardial effusion was also noted (Figure 2).

To further delineate this suspicious cardiac mass, a multislice, prospective, ECG-gated CT coronary angiography (CTCA) with

contrast was performed. CTCA revealed normal origin of left coronary artery (LCA) from left coronary cusp. Left main coronary artery was normal. There were giant aneurysms in the proximal LAD and its proximal first diagonal branch, which were partially thrombosed. The distal LAD and distal first diagonal branch were normal. The largest aneurysm was that of the proximal LAD artery which measured 4.8×3.3 cm (Figure 3 A,B). These thrombosed aneurysms were causing mass effects on the pulmonary trunk with compression and displacement towards the right side.

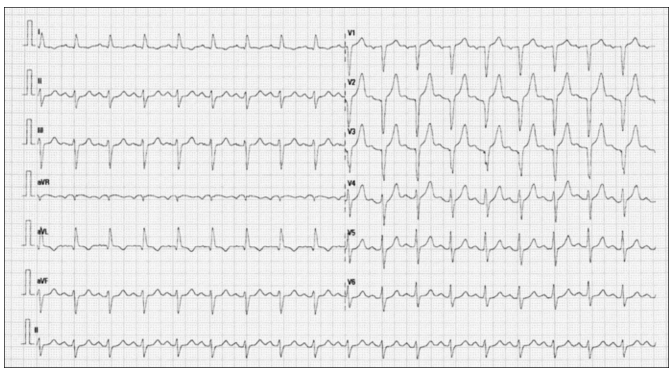

Figure 1: 12 leads ECG showing lateral T-wave inversions (leads I, aVL).

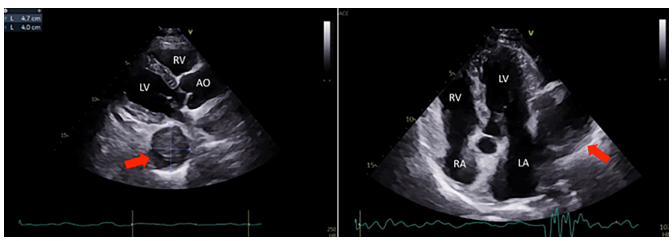

Figure 2: A suspicious echogenic mass (red arrows) adjacent to the left atrium in the atrioventricular groove which was causing compression of the left atrium.
AO: Aorta; LV: Left ventricle; LA: Left atrium; RV: Right ventricle; RA: Right atrium.

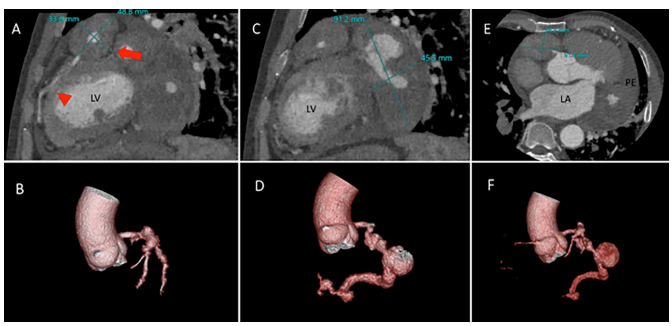

Figure 3: (A) Proximal LAD aneurysm measuring 4.8x3.3cm (arrow). Normal distal LAD (arrowhead). (B) 3D image of Aortic root and LAD. (C) Large aneurysm of obtuse marginal branch of left circumflex artery (arrow). (D) 3D image of LCX artery. (E) Completely thrombosed aneurysm of RCA measuring 5.1x3.3cm. (F) 3D image of aortic root and coronary vessels with almost completely thrombosed RCA (arrow).
LA: Left atrium; PE: Pericardial effusion; LV: Left ventricle.

There was only a small patent portion of the left circumflex artery visualised and the rest of left circumflex artery showed multiple large partially thrombosed aneurysms. The OM branch of the left circumflex artery was grossly dilated with a partially thrombosed aneurysm measuring about 9.1×4.5 cm, which was compressing and indenting the left atrium (Figure 3 C,D). A few other small aneurysms were also noted at the level of distal OM branches.

The RCA was non-dominant, arising from the right coronary cusp. There was an almost completely thrombosed aneurysm of the RCA near its origin with only a streak of contrast identified in the residual lumen in the proximal part and no contrast opacification in the distal part of the aneurysm (Figure 3 E,F). The posterior descending artery in the region of posterior interventricular groove was also dilated.

There was an evidence of moderate pericardial effusion (Figure 3 E). There was normal caliber of the visualised aortic root, ascending aorta, and descending thoracic aorta with no evidence of any aneurysmal dilatation. There was no associated mediastinal lymphadenopathy.

After discussing the case with the cardiologists and cardiac surgical team, the decision was to continue for medical management of the coronary artery disease and GCAAs and refer to the rheumatologist for a further investigation of any underlying vasculitis.

DISCUSSION

CAAs, which were first pathologically described by Morgagni in 1761 and clinically reported by Bourgon in 1812, are rare but their incidence is increasing due to the use of coronary angiography. The incidence of CAAs is 1.5-5% while that of GCAAs is 0.02%.[1,2]

The main aetiological cause of coronary artery aneurysm in adults is atherosclerosis.[3] Other aetiologies include congenital heart diseases, arteritis (e.g. Takayasu arteritis), connective tissue diseases (Marfan syndrome, Ehlers-Danlos syndrome, fibromuscular dysplasia, neurofibromatosis, etc.), vasculitis (lupus, polyarteritis nodosa, Behcet's disease, rheumatoid arthritis, ankylosing spondylitis, scleroderma), infections (HIV, bacterial, mycobacterial, syphilis, Lyme disease, mycotic aneurysm, septic emboli), drugs (cocaine, amphetamine, protease inhibitors), percutaneous interventions (PCIs) (drug-eluting stents (DES) implantation, high inflation pressure, balloon, rotablator), chest trauma, tumours, and cardiac lymphomas.[1,2]

In recent years, there has been a rise in the occurrence of CAAs associated with stent placement. This increase can be attributed to the utilisation of DES, which consists of immunosuppressants and chemotherapeutic substances that hinder immune response to affected tissue and cell proliferation. The polymer that holds the drug has the potential to trigger a hypersensitivity reaction, vessel wall inflammation, and weakening, ultimately leading to dilatation.[4]

The majority of individuals with CAAs do not experience any symptoms, but they might manifest with chest pain, heart attack, or sudden cardiac death. There is a possibility of various complications arising from CAAs, including thrombosis, embolisation, the development of abnormal connec-

tions with adjacent structures (fistulas), rupture of aneurysm, accumulation of blood in the pericardium (hemo-pericardium), cardiac tamponade, mass effect on neighbouring structures, infective endocarditis, or congestive cardiac failure.[4-6]

The choice of treatment for CAAs depends on factors such as the presence of symptoms, underlying causes, or associated abnormalities. The available treatment options encompass medical management, deployment of stent, or surgical intervention.[5,7] Surgical intervention is typically pursued for the management of GCAAs to prevent potential complications. On the other hand, conservative management is generally employed for small CAAs.[8,9]

Concurrent GCAAs involving more than one coronary artery are rare. These GCAAs cannot only present with chest pain but, as in this case, with compression effects on the surrounding structures (left atrium, pulmonary trunk). They can also present with atypical symptoms and presentations. Early diagnosis and surgical intervention are imperative to avoid life-threatening complications.

PATIENT'S CONSENT:
Written consent was obtained from the patient.

COMPETING INTEREST:
The authors declared no competing interest.

AUTHORS' CONTRIBUTION:
KR, MW, NK: Contribution in the design of the study, data and image collection, writing and drafting of the manuscript, critical review of the draft, referencing and making the final version of the draft.

All authors approved the final version to be published and are accountable for all aspects of this work.

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