CASE REPORT

Anomalous left anterior descending artery: an uncommon cause of ST elevation myocardial infarction

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SUMMARY

A healthy 32-year-old Asian man presented in emergency with a history of severe chest pain. ECG showed normal sinus rhythm with ST elevations in the anterior chest leads. He was diagnosed with anterior wall ST segment elevation myocardial infarction and was thrombolysed with tenecteplase. The cardiac enzymes and troponin level were significantly high and echocardiography demonstrated akinetic anterior segment of left ventricle. Coronary angiography exhibited anomalous left anterior descending artery originating from right sinus of Valsalva without atherosclerotic lesion.

BACKGROUND

Anomalous coronary artery originating from opposite sinus (ACAOS) of Valsalva is a rare congenital condition.¹ Most patients with coronary anomalies are diagnosed incidentally on coronary angiography or at autopsy. The most common anomaly is separate origin of left anterior descending (LAD) artery and left circumflex (LCX) artery from left coronary sinus (LCS). Other common anomalies include origin of LCX artery from right coronary sinus (RCS) or right coronary artery (RCA) and RCA arising from LCS. The majority of coronary anomalies are benign while few of them manifest life-threatening symptoms including arrhythmias, syncope, myocardial infarction or sudden death.

CASE PRESENTATION

A 32-year-old man presented with a 6 h history of severe chest pain. The patient developed heaviness on the left side of the chest. It was radiating to his left arm and associated with sweating. He denied any history of vomiting, palpitation or shortness of breath. The patient denied any history of fever or flu-like symptoms in the recent past. There was no history of medical illness or family history of ischaemic heart disease. On physical examination, the vital signs, cardiovascular and chest examination were unremarkable.

INVESTIGATIONS

The patient's ECG showed normal sinus rhythm with ST elevation in anterior chest leads V1, V2 and V3, with mild ST segment depression in limb leads II, III and aVF (figure 1). The ST segment settled slightly after 90 min (figure 2).

Blood investigations included haemoglobin 15.3 g/dL, white cell count $22.9 \times 10^3/\mu\text{L}$ and platelets $370 \times 10^3/\mu\text{L}$. Cardiac markers on admission were serum troponin 0.04 ng/mL, creatine kinase (CK) 106 units/L and CK-MB fraction 25 units/L. All cardiac markers went high after 13 h; they were serum troponin 1.6 ng/mL, CK 1633 units/L and CK-MB 227 units/L. The patient's lipid levels were total cholesterol 234 mg/dL, triglyceride 144 mg/dL, high-density lipoprotein 29 mg/dL and low-density lipoprotein 187 mg/dL.

Other blood tests including cardiac Pro-brain natriuretic peptide, urea, creatinine and electrolytes were within normal range.

Echocardiography showed anterior wall akinesia causing impaired left ventricle systolic function with ejection fraction of 40%.

The patient underwent coronary angiography, which revealed normal left main stem and was bifurcating into LCX artery and first diagonal branch (figure 3). LDA artery was anomalous in origin from RCS. RCA was normal in origin (figure 4). All epicardial arteries were normal and did not show atherosclerotic lesion or dissection.

DIFFERENTIAL DIAGNOSIS

- ST elevation myocardial infarction
- Coronary artery dissection
- Prinzmetal angina
- Takotsubo cardiomyopathy
- Pericarditis
- Myocarditis

TREATMENT

On the basis of history and ECG findings, the patient was diagnosed as anterior wall ST elevation myocardial infarction. He was given a bolus dose of tablet aspirin 300 mg and clopidogrel 300 mg, and was thrombolysed with intravenous tenecteplase 35 mg. The patient received thrombolytic therapy because primary percutaneous coronary intervention (PCI) was not available. The chest pain was treated with 3 mg of intravenous morphine and a low dose of nitroglycerine infusion. The patient's condition improved and he was free of chest pain. The following day, coronary angiography was performed.

The antiplatelet tablets aspirin 100 mg and clopidogrel 75 mg both once daily were continued. Additionally, the patient received injection clexane 60 mg subcutaneously two times per day and tablet

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Figure 1 ECG showing ST elevation in anterior chest leads.

atorvastatin 80 mg at bedtime, tablet captopril 6.25 mg three times per day and tablet bisoprolol 2.5 mg once daily.

OUTCOME AND FOLLOW-UP

Post-angiography the patient remained stable and asymptomatic. He was discharged on single antiplatelet medication tablet aspirin 100 mg once daily since there was no atherosclerotic lesion found in his arteries. Other tablets prescribed were atorvastatin 80 mg and ramipril 2.5 mg once daily, and bisoprolol 2.5 mg.

The patient was advised to undergo computed coronary angiography to evaluate the course of LAD artery and to look for any possible surrounding structure causing compression of the artery.

DISCUSSION

ACAOS of Valsalva is a rare congenital condition. Krasuski *et al*¹ found 301 cases of ACAOS in a review of 210 700 catheterisations, which made an incidence of 0.14%. Most patients with coronary anomaly are asymptomatic and diagnosed incidentally on coronary angiography. Twenty per cent of such patients may develop chest pain, arrhythmias or even manifest fatal complications such as myocardial infarction or sudden death.² The exact mechanism of myocardial ischaemia is unclear. The possible reasons are narrow ostium of the artery, acute bend of the artery after its origin, or compression of the artery from adjacent structures during the course of the artery.

Bahram reviewed the coronary angiography of 6065 patients and reported that the incidence of coronary anomaly was 1.3%.

The most common anomaly was separate origin of LAD artery and LCX artery from LCS, which was 53.1%. The other most common anomalies were LCX artery arising from RCS or RCA, accounting for 21.5% and RCA arising from LCS, at 7.5%.³

Another review of 7400 patients by Harikrishnan found similar results of separate origin of LAD and LCX arteries from LCS being the most common anomaly. The other common anomalies were RCA arising from LCS and LCX originating from RCS.⁴ Other rare anomalies include single coronary artery, double RCA, LAD artery from RCS and all three coronaries originating separately from RCS.

An anomalous origin of left coronary artery from pulmonary artery is a rare and fatal condition. It is usually associated with myocardial infarction, congestive heart failure and sudden death.⁵ Origin of LCA from RCS or RCA with subsequent course of artery between aorta and pulmonary artery is associated with sudden death during or just after exercise in young individuals.⁶ Karim reported this condition in a 14-year-old boy who was diagnosed as anterolateral ST segment elevation myocardial infarction. The coronary arteriogram showed that LM coronary artery was originating from RCS and passing intramurally in the wall of aorta between aorta and the main pulmonary artery. The patient underwent surgical derofing to treat his condition.⁷

Selcoki *et al* reported a case of a 37-year-old patient diagnosed to have myocardial infarction. His angiography showed anomalous LCX arising from RCA and myocardial bridge of mid LAD artery.⁸ Zimarino published a case report of two

Figure 2 ECG at 90 min showing slight improvement in ST segment.

Figure 3 Coronary angiography showing left main coronary artery arising from left coronary sinus and bifurcating into left circumflex artery and diagonal branch.

patients who presented with inferior wall ST elevation myocardial infarction. Both were taken for primary PCI and anomalous origin of left coronary system from the opposite sinus was discovered. This emphasises the need for knowledge of coronary anomalies in patients undergoing primary PCI.⁹

In a case series by Karur 13 patients were found to have coronary anomalies among 2482 patients who underwent coronary angiography. Three patients among them presented with acute coronary syndrome and were found to have significant stenosis in their anomalous arteries, requiring PCI. Two patients had anomalous LCX artery and 1 had anomalous RCA.¹⁰

Yuksel cited a case of a 48-year-old woman who presented with a history of stable angina and palpitation. Her angiography exhibited all the three coronary arteries arising from RCS.¹¹

Figure 4 Coronary angiography showing normal origin of right coronary artery and anomalous origin of left anterior descending artery from right coronary sinus.

Similarly, Pourafkari *et al*¹² published a case report of single coronary artery in a 44-year-old man who presented with anterior wall myocardial infarction; his angiography showed RCA was arising from LCX and retrogradely, LCX followed the course of RCA up to the base of the heart.

In our case report, the patient presented with a short history of typical chest pain and ECG showed ST elevation in anterior chest leads with reciprocal changes in inferior leads highly suggestive of acute coronary syndrome. Myocarditis was an unlikely diagnosis since there was no history of fever or infection in the recent past. The patient's angiography displayed anomalous LAD artery originating from RCS, which manifested as anterior wall myocardial infarction. The artery was normal without stenotic lesion. He was treated initially with dual antiplatelets and after angiography he was put on single antiplatelet tablet aspirin only, along with atorvastatin, ramipril and bisoprolol. He was advised to undergo computed coronary angiography to further delineate the course of anomalous LAD artery.

Learning points

- ▶ Coronary atherosclerosis is the most common cause of acute coronary syndrome (ACS). However, coronary anomaly, which is very rare, can present itself as ACS.
- ▶ Prior knowledge of coronary anomalies is imperative when patients are considered for primary percutaneous coronary intervention (PPCI).
- ▶ Coronary anomaly should be included in the differential diagnosis of angina, arrhythmias or heart failure in a young patient.
- ▶ Coronary anomalies can cause sudden death in young people during or after exercise.

Competing interests None declared.

Patient consent Obtained.

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