

Case Report

Diagnostic Approach in Symptomatic or Asymptomatic Aneurismal Disease of the Coronary Arteries: A Case Report and Five-Year Angiographic Follow Up

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Coronary artery aneurysms are rare anomalies that are usually incidental findings in coronary angiography. We present the case of a 64-year-old male patient with a recent episode of epigastric pain, nausea and vomiting, accompanied by electrocardiographic alterations and positive troponin I. As the symptoms persisted, the patient was referred for coronary angiography, during which a significant degree of aneurysmal dilatation was found in all three coronary vessels along their length. Oral administration of anticoagulants was decided upon. After five years of follow up, a new coronary angiogram was recorded, which showed no aggravation of the aneurysmal dilatations, while the patient was free of symptoms.

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Aneurysms of coronary vessels are rare and are defined as dilatations of coronary vessels, diffused or saccular. Their diameter exceeds by more than twice the diameter of adjacent physiologic segments of the coronary vessel (in contrast with ectasias, whose diameter is 1.5 to 2 times larger).¹⁻³ They are accidental angiographic findings in up to 4.9% of cases (CASS study) and incidental necropsy findings in about 1.4%.⁴ They are characterized as saccular when their transverse diameter is bigger than their axial length and diffuse when they present oblong dilatations parallel to the vessel's axis.⁵ They were first described in the material of a necropsy by Morgagni in 1761.⁶ The involvement of the coronary arteries varies in different studies, but appears to be 25-68% for the left anterior descending coronary artery, 18-21% for the circumflex, 9-54% for the right coronary artery, and

finally, 5% for the branch. Aneurysms in the artery of the sinoatrial and atrioventricular node have very rarely been described.⁷⁻⁹ Coronary aneurysms occasionally coexist with other malformations of the heart, such as coronary arteriovenous communications, supravalvular aortic stenosis, aneurysm of the left ventricle, and hypertrophic cardiomyopathy.

Case presentation

A 64-year-old male, with no history of heart disease, was admitted to the cardiology department of our hospital for investigation of a recent episode of epigastric pain, nausea, vomiting, accompanied by electrocardiographic alterations with positive troponin I, elevated creatine kinase (CK), CK MB isoenzyme (CKMB), N-terminal pro-brain natriuretic peptide (NT-proBNP) and C-reactive protein (CRP).

His medical history included β -thalassemia (minor), right groin surgery, performed four years ago, arterial hypertension, and hyperlipidemia. The patient was also a casual tobacco user. His family's medical history included his father's death due to coronary disease, and his brother's aortocoronary bypass, as he suffered from the same disease. The remaining physical and laboratory examinations were unremarkable. The ECG on admission revealed sinus rhythm at 70 beats/min, $+45^\circ$ QRS, and q waves in leads II, III and aVF. Echocardiography showed an ejection fraction of 45%, with hypokinesia in the inferior and posterior left ventricular wall. No valvular abnormalities were observed, but left ventricular diastolic dysfunction, $E < A$, was recorded using tissue Doppler imaging.

As the patient's symptoms persisted, coronary angiography was performed, during which a significant degree of aneurismal dilatation was found in all three coronary vessels along their length (Figure 1). The patient subsequently underwent gastroscopy, which was negative. Oral administration of anticoagulants was decided upon, and after five years of follow up coronary angiography showed no aggravation of the aneurismal dilatations (Figure 2), while the patient was free of symptoms.

Discussion

Coronary aneurysms have been correlated in a causative manner with various disease states, including the following:

- Atherosclerotic coronary disease (50%), which

causes destruction and remodeling of the *tunica media* of the vessel wall.^{2,10}

- Familial anomalies, such as fibromuscular dysplasia (20-30%).
- Inflammatory disease states, such as Kawasaki disease, mycotic-syphilitic aneurysms, *polyarteritis nodosa*, Takayasu disease, Epstein-Barr virus, chronic infection (10-20%).
- Post-traumatic (following angiography or angioplasty).
- Connective tissue disease, such as systemic *lupus erythematosus*, rheumatoid arthritis, scleroderma.^{11,12}
- Diseases of collagen structure, such as Ehlers-Danlos syndrome, Marfan syndrome, neurofibromatosis type 1.¹³
- Other, for example Osler-Weber-Rendu syndrome, hyperhomocysteinemia.^{14,15}

Their pathogenesis varies, depending on regional distribution and the age of the patient. They are most often ascribed to Kawasaki disease in the far east, but to atherosclerosis in North America. Familial aneurysms are common at ages below 33 years, while atherosclerotic aneurysms more often occur at ages above 56 years.^{8,16}

Coronary aneurysms may progress asymptotically. Their discovery may be an incidental finding (angiographic or even necropsy), or they may cause complications.^{17,18} They may cause symptoms compatible with coronary disease (angina), or present with a clinical symptomatology similar to that of dissecting aortic aneurysm.¹⁹ Clinical examination may rarely show systolic or diastolic murmur. Their main complications include rupture and thrombus develop-

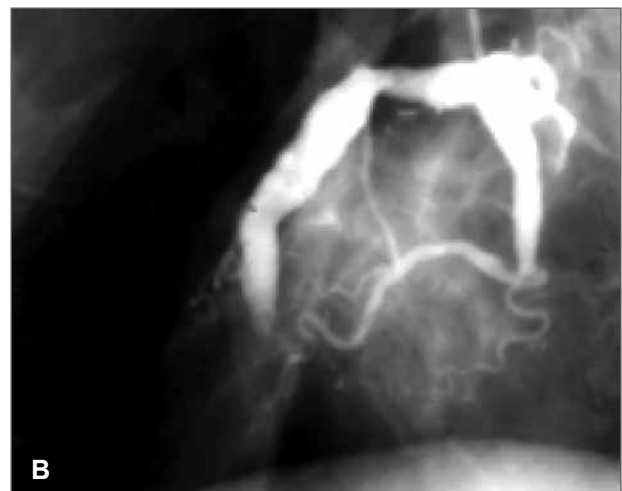


Figure 1. Coronary angiograms showing aneurysms of (A) the right coronary and (B) the left main, left anterior descending, and circumflex arteries on the patient's admission to our department.

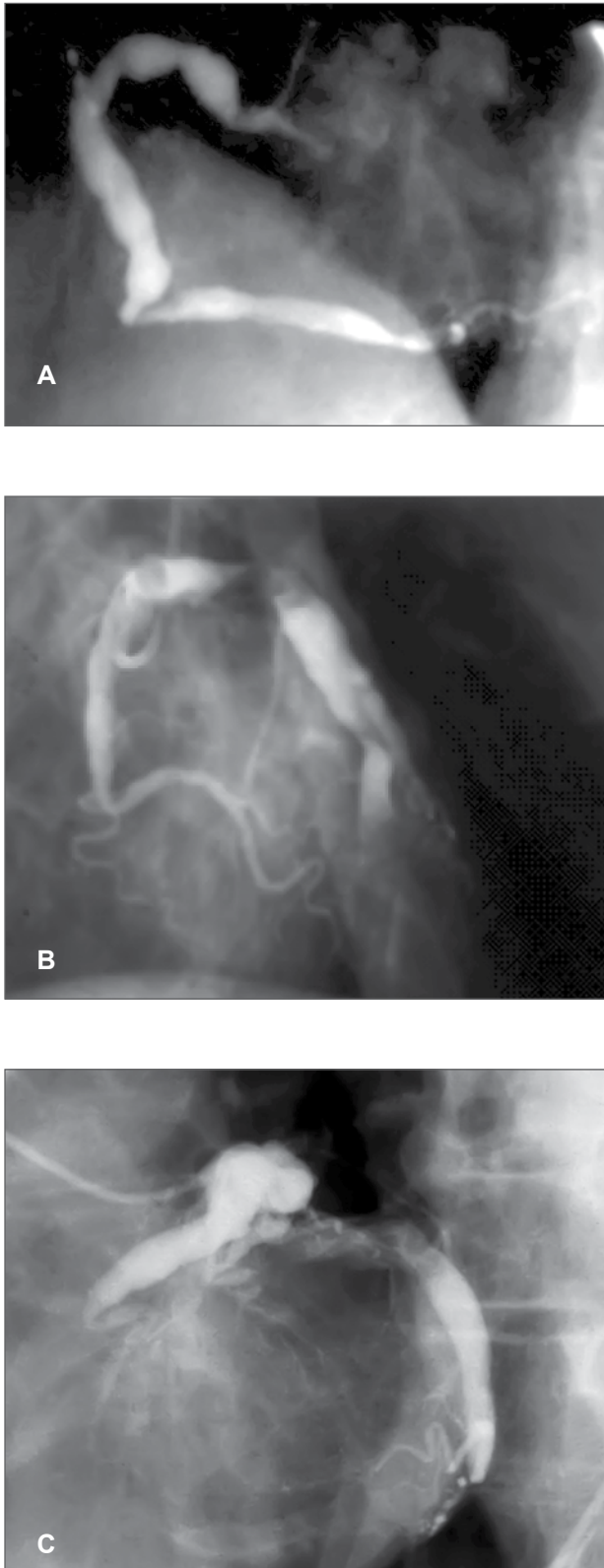


Figure 2. The angiograms at five-year follow up showed no aggravation of the aneurysms in (A) the right coronary and (B,C) the left main, left anterior descending, and circumflex arteries.

ment in their lumen (due to the low blood flow velocity inside the aneurysm) and consequently peripheral emboli that lead to acute myocardial infarction. Both complications may lead to sudden death.²⁰⁻²³

The gold standard for the diagnosis of coronary aneurysms is coronary angiography, but information about the size, localization, and behavior can be acquired using chest X-ray, magnetic resonance imaging of the chest and heart, transthoracic and transesophageal echocardiography, or intravascular ultrasound (IVUS).^{16,17,19,24-25}

The therapeutic strategy for coronary aneurysms is still unclear and the approach should be individualized. In patients who have aneurysms of coronary arteries without coexisting coronary disease, antiplatelet agents are administered together with oral anticoagulants for prevention of thromboembolic complications. These aneurysms may also be treated using angioplasty with stent deployment, mainly in those patients who have no coexisting coronary disease that would necessitate aortocoronary bypass. The stents that have been used in such cases are either invested with saphenous vein tissue, or covered with polytetrafluoroethylene (PTFE).²⁶ The drawbacks associated with the use of PTFE stents are the possibility of obstruction of adjacent arterial sectors that originate from the wall of the aneurysm, incomplete convergence of the aneurysm with persistence of flow within its lumen, and the risk of in-stent restenosis.²⁷⁻³⁰

For coronary aneurysms with coexisting coronary disease, surgical management is recommended,³¹ particularly when the coronary disease is serious or angina pectoris persists despite complete pharmaceutical treatment.^{20,32,33} Furthermore, many investigators propose surgical treatment for saccular aneurysms that show increased risk of thrombosis or rupture, despite the fact that so far there are insufficient studies to support this point of view.³⁴⁻³⁶ Surgical treatment involves ligation and aortocoronary bypass, or dissection and end-to-end anastomosis.^{17,37}

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