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Case Study

Severe coronary disease in an adult considered at low cardiovascular disease risk with a healthy lifestyle

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Right coronary artery (RCA)

Abstract: Lipoprotein(a) [Lp(a)] is a lipoprotein subclass well-known among the lipid community to accelerate atherosclerosis and promote thrombosis through incompletely understood mechanism. We report a case of a young man with a healthy lifestyle and no major coronary or vascular risk factors who presented to the emergency department with an acute coronary syndrome and was ultimately found to have severe coronary artery disease. A diagnostic workup revealed elevated Lp(a). He was treated with consequent reduction in Lp(a) concentration. This case highlights the need to better understand atypical lipoproteins, how they relate to cardiovascular disease, the implications for screening family members, and the need to standardize patient management guidelines for the purpose of mortality risk reduction.

An imbalance in circulating atypical lipoproteins such as lipoprotein(a) [Lp(a)] often goes undetected until patients experience acute coronary events, usually at a young age. Firm guidelines for diagnosing and managing elevated Lp(a) are presently lacking.[1,2] We present a young man with a healthy lifestyle and no coronary risk factors with advanced coronary disease who presented with an acute coronary event, likely secondary to elevated Lp(a). We describe the diagnosis and management of elevated Lp(a) as well as highlight confounding issues in laboratory interpretation and pharmacologic management. This will aid clinicians who serve patients or family members with elevated Lp(a).

Case report

The patient is a 37-year-old Chinese American man with no significant past medical history and no family history of premature cardiovascular disease, hypertension, smoking or secondary smoke exposure, or diabetes who is a body builder, exercising 5 days per week. He began to feel nonradiating substernal chest pressure while exercising in the gym. The chest pressure did not remit with rest, emergency medical personnel were called, and the patient was taken to the emergency department. The patient's initial electrocardiogram (ECG; Fig. 1) showed clear ST-segment elevation in the inferior leads, indicative of acute transmural myocardial ischemia that progressed to an infarction pattern by the time he was transferred from the outside hospital to our institution. Before arrival, he was given 300 mg of clopidogrel by mouth, 325 mg of aspirin by

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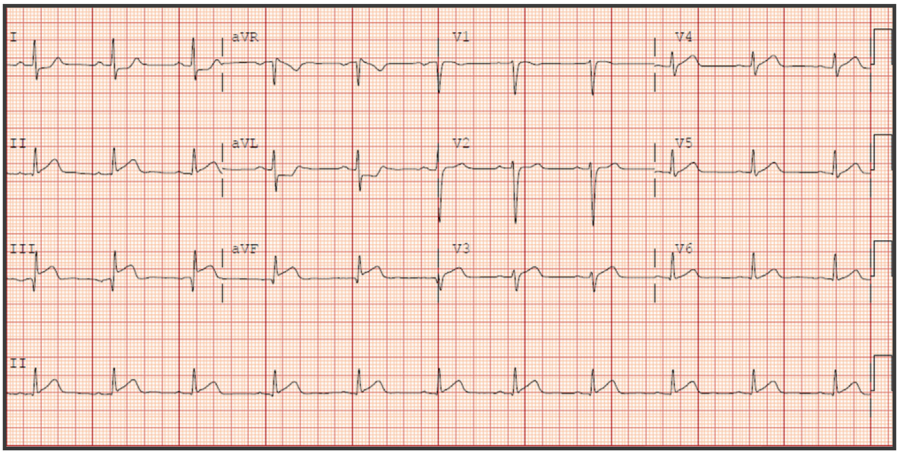

Figure 1 A 12-lead electrocardiogram taken at the time of arrival while the patient was experiencing chest pain is shown. Clear ST-segment elevation is seen in the inferior leads, suggestive of acute transmural myocardial ischemia, with reciprocal ST-segment depression in the lateral leads.

mouth, and 5000 U of intravenous heparin. On arrival, the patient's vital signs were as follows: temperature of 36.6°C, manual blood pressure measurement in the dominant arm while supine of 144/89 mm Hg, heart rate of 77 beats per minute, and respirations of 15 per minute. On the day of the examination, his body mass index (calculated as weight divided by height; kg/m^2) was 25.1. He appeared to be in mild distress. His lungs were clear to auscultation without audible rales. His cardiac examination was normal with a regular S_1 and S_2 without gallop, murmur, or rub. His measured jugular venous pressure was 8 cm H_2O at 45 degrees. His abdomen was nontender and nondistended, and his extremities were well perfused with 2^+ pulses. His chest x-ray showed a normal cardiomediastinal silhouette, without significant pulmonary interstitial markings or edema. His initial cardiac troponin T was <0.01 ng/mL and peaked at 5.68 ng/mL 7 hours later. His other laboratory data were unremarkable and within the reference range for our laboratory. His coronary angiogram showed severe 2-vessel coronary artery disease (CAD) with 70% stenosis of the left anterior descending coronary artery (Fig. 2A, arrowhead) and 95% occlusion of the right coronary artery with a distal thrombus (Fig. 2B, arrowhead). The left circumflex artery had only mild, diffuse disease with a maximum lumen narrowing of 20%. The left ventriculogram showed an estimated ejection fraction of 45% to 50% with a hypokinetic inferior left ventricle wall. According to the patient's ECG and coronary angiogram data, the right coronary artery was likely the culprit lesion for the patient's acute presentation, and he received a percutaneous intervention with a drug-eluting stent to that region with resolution of his chest pain and acute ECG changes. The patient recovered well, was discharged in a stable state, and completed outpatient cardiac rehabilitation.

Discussion

The patient's 10-year risk of cardiovascular events according to the Framingham risk stratification algorithm at the time of presentation was just 2%. The patient demographics, including his Asian ancestry, and objective data, including blood pressure, lipid profile, body mass index, as

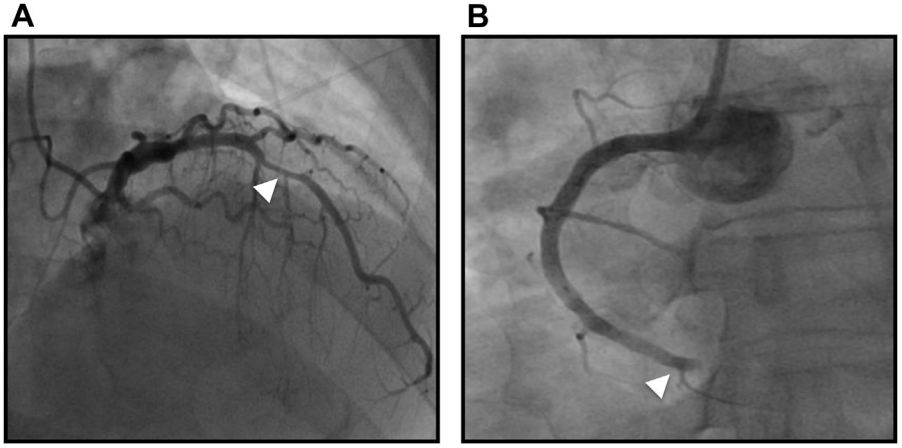

Figure 2 Coronary angiographic images. (A) Imaging of the left coronary system shows severe stenosis of the left anterior descending coronary artery (arrowhead). (B) Imaging of the right coronary system shows a thrombus in the distal right coronary artery (arrowhead) with impaired filling.