Clinical picture

Adult Kawasaki disease

A 44-year-old male presented with 1 h history of substernal, persistent pressure-like chest pain. The pain had no radiation and started while he was watching TV. He denied associated nausea, vomiting, palpitations or shortness of breath. He has history of hypertension controlled with irbesartan. He never smoked or drank alcohol. Exam showed normal vital signs; cardiovascular examination showed regular rate and rhythm, no gallops, murmurs or pericardial rub, had symmetric and bounding distal pulses. Electrocardiogram showed normal sinus rhythm with no ST or T wave changes, and with normal intervals. Chest X-ray was normal. Laboratory studies showed elevated cardiac enzymes: CK 662 U/L (reference range: 30–220 U/L) and CK MB 8.1% (reference range: 0.0–4.0%), troponin T 2.28 ng/ml (reference range: 0.00–0.10 ng/ml). He underwent an emergent left heart catheterization (LHC) for non-ST-elevation myocardial infarction (NSTEMI). Surprisingly, he had diffuse triple coronary artery ectasia with no evidence of focal stenosis or thrombosis (Figure 1). No intervention was done since there was no coronary stenosis. Echocardiography showed preserved left ventricular function with ejection fraction of 60% and no evidence of valvular disease. Erythrocyte sedimentation rate (ESR) was 3 mm/h. He denied childhood history of vasculitis—including Kawasaki’s—or any autoimmune disease. Based on the LHC findings and work up, a diagnosis of adult Kawasaki’s disease was made in the absence of significant cardiac risk factors. The patient was started on aspirin, clopidogrel and enoxaparin with bridging to coumadin on discharge.

Coronary artery ectasia is defined as an arterial segment dilatation at least 1.5 times that of an adjacent normal coronary artery. Most cases of reported adult Kawasaki’s disease diagnosis are based on findings of diffuse coronary artery ectasia and patients do not report or recall a childhood illness compatible with Kawasaki’s disease. Coronary artery ectasia usually occurs in patients with significant cardiac risk factors and usually affects only one or two coronary vessels. In this case, the presence of triple coronary artery ectasia with absence of significant cardiac risk factors prompted the diagnosis of adult Kawasaki disease.

The aim of management of coronary artery ectasia is preventing formation of thrombus within these vessels. A paucity of evidence evaluating treatment of coronary artery ectasia is available; however, recommendations are to start patients on an antiplatelet agent such as aspirin and long-term anticoagulation with warfarin to prevent thrombus formation. Coronary spasm has been hypothesized as the etiology of angina in patients with coronary artery ectasia and calcium-channel-blockers have been recommended for their antispasmodic properties.

Kawasaki disease is predominately a vasculitis disease of childhood; ~20–25% of patients develop coronary artery aneurysms. Increased awareness
that the condition can occur in adults is important, as well as that a history of Kawasaki’s disease in childhood is not required for diagnosis.

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References