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Case Report

Coronary artery stenting in acute coronary syndrome associated with giant cell arteritis

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ABSTRACT

Coronary vasculitis is a rare but devastating complication of giant cell arteritis, otherwise known as temporal arteritis. Originally named for its propensity to attack the superficial temporal arteries, it is now recognized that it commonly involves a number of medium and large arteries throughout the body. Here we describe two cases of giant cell arteritis affecting the coronary arteries, one discovered at post-mortem and one which was successfully treated with immunosuppressive therapy and drug-eluting coronary stents.

<**Learning objective:** Giant cell arteritis is a large vessel vasculitis which commonly affects the superficial temporal, subclavian arteries, and aorta but in rare cases can involve the coronary arteries. In addition to standard corticosteroid therapy, drug-eluting stents may be useful in treating this rare but serious disease. Clinicians should both consider this condition in patients with atypical features of coronary artery disease and recognize drug-eluting stents as a potential effective therapy.>

Introduction

Giant cell arteritis (GCA), also known as temporal arteritis, is a medium to large vessel vasculitis affecting people over the age of 50 years. While it classically has a predilection for the temporal arteries, computed tomographic (CT) angiography has demonstrated involvement of other large arteries (particularly aorta, brachiocephalic trunk, carotid arteries, subclavian arteries, and femoral arteries) in up to two thirds of patients [1].

Coronary arteritis has classically been associated with medium and small vessel vasculitis such as Kawasaki disease and is a potential and known complication of Takayasu arteritis. However, there are a few reports of coronary arteritis caused by GCA, most of which were in the context of fatal myocardial infarctions, with autopsy evidence of coronary GCA [2]. GCA involving the coronary arteries has been treated with bare metal stents [3]. Stenting has also been used successfully in GCA affecting the subclavian, axillary, brachial, renal [4], and vertebral arteries [5].

Here we describe two cases of critical coronary artery stenosis in acute GCA, one which was only discovered at post-mortem, and one which is the first case reported to have been effectively revascularized with drug-eluting stents.

Case A

An elderly woman had a sudden death in the community which was referred to the coroner. Upon post-mortem, the aorta had ruptured, resulting in hemopericardium and cardiac tamponade. The aorta, coronary arteries, and pulmonary arteries demonstrated granulomatous inflammatory skip lesions and giant cells, consistent with GCA (Fig. 1a).

Case B

A 76-year-old woman presented to her ophthalmologist in November 2015 with a 3-week history of transient left-sided vision loss associated with headache, jaw claudication, weight loss, and

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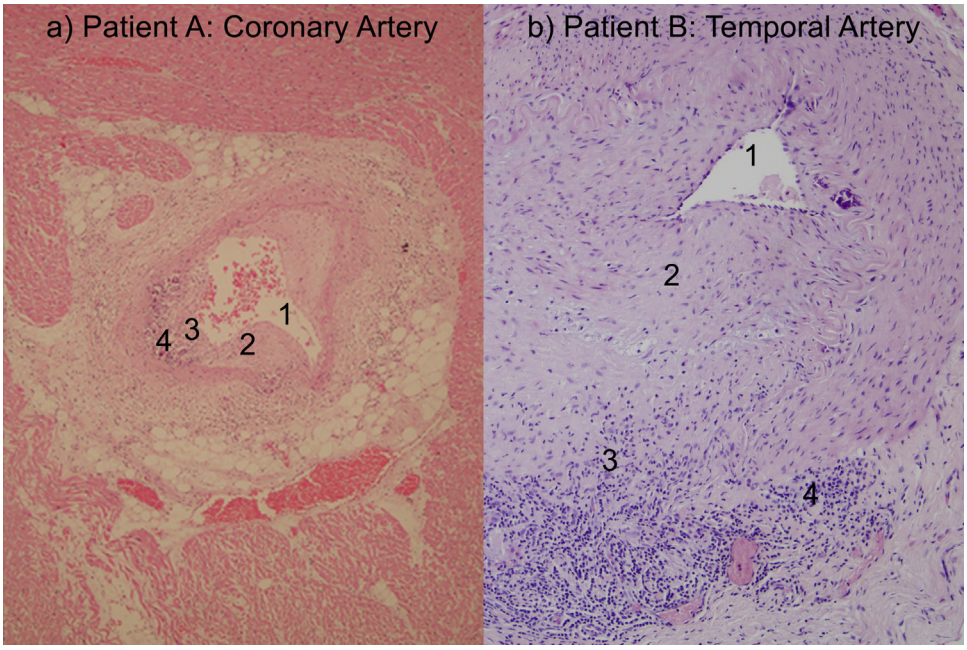

Fig. 1. Pathology of Patient A's coronary artery (a) and Patient B's temporal artery (b).
(1) Partial lumen occlusion.
(2) Intimal fibroplasia.
(3) Disrupted internal elastic lamina.
(4) Histocytes and lymphocytes.

neck stiffness. She had a past medical history of hyperlipidemia, multinodular goiter, vitiligo, intermittent supraventricular tachycardia, and minor non-obstructive coronary artery disease, which had been diagnosed on an angiogram in 2013 when she had presented with atypical chest pain. She denied a history of stroke, hypertension, diabetes, or smoking but reported limited mobility due to bilateral lower limb claudication type pain which she had been experiencing for the past few months at a distance of 200 m. Medications prior to presentation were aspirin 100 mg daily, sotalol 40 mg twice daily, and rosuvastatin 5 mg daily. Examination that day was suspicious for left retinal artery embolus and she was referred for urgent cardiology review for suspected atrial fibrillation. At this stage, electrocardiogram (ECG) showed normal sinus rhythm with no evidence of ischemia or previous infarction and transthoracic echocardiogram (TTE) was normal. Erythrocyte sedimentation rate and C-reactive protein were elevated to 59 mm/h (normal range 1–35 mm/h) and 58.9 mg/L (normal range 0.0–5.0 mg/L), respectively and she was commenced on oral prednisone 50 mg daily for possible GCA. Over the subsequent two days, she experienced intermittent chest and neck pain which was attributed to gastro-oesophageal reflux in the context of prednisone use.

She was admitted to hospital two days after initial cardiology review for a transesophageal echocardiogram looking for an intracardiac source of embolus. Unexpectedly, this showed marked anterior left ventricular wall hypokinesis, consistent with recent ischemia. There were corresponding new deep anterior Q waves on ECG (Fig. 2a). Urgent coronary angiography showed 90% stenoses of the proximal left anterior descending (LAD) and large caliber intermediate arteries, 70% stenosis of the mid right coronary artery, and 30% stenosis of the left main artery. The appearances were suggestive of widespread vasospasm but were unresponsive to intra-arterial glyceryl trinitrate. This was in stark comparison to an angiogram performed 2 years previously, where she had only 30% stenosis of the mid-LAD and mild irregularity of the remaining vessels. Troponin T was elevated at 353 ng/L (normal range <14 ng/L). Given the rapid progression of disease in the absence of other risk factors for cardiovascular disease, the possibility of superimposed coronary vasculitis was raised.

Due to the complexity of the coronary anatomy, initial resolution of symptoms on medical therapy, confounding comorbidities and potential for requiring surgical intervention, the decision was made to defer an attempt for percutaneous revascularization, pending further discussion. She was commenced on medical therapy for presumed missed ST-elevation myocardial infarction (STEMI) with aspirin, clopidogrel, and a heparin infusion and pulsed with intravenous methylprednisolone 1 g daily for 3 days. Further examination revealed palpable strong femoral pulses with absent popliteal and pedal pulses on both lower limbs, a left axillary bruit, a blood pressure gradient of 14 mmHg between arms, delayed radial pulse on the left, and bilateral tender temporal arteries.

The following evening, she developed acute chest discomfort and ECG showed ST elevation in the anterior leads (Fig. 2b). She underwent primary percutaneous coronary intervention (PCI) with drug-eluting stents to the ostial LAD (3.0 × 20 mm Synergy, Boston Scientific, Marlborough, MA, USA), the proximal intermediate (2.75 × 8 mm Xience Xpedition, Abbott Vascular, Santa Clara, CA, USA), and the ostial circumflex (3.25 × 8 mm Xience Xpedition) (Fig. 3) arteries. A tirofiban infusion was commenced and an intra-aortic balloon pump inserted for peri-procedural hemodynamic support.

Post-procedural TTE 4 days later showed complete normalization of left ventricular function. She was transitioned to 60 mg daily of oral prednisone and 10 mg weekly of oral methotrexate. Inflammatory markers normalized, ECG changes resolved (Fig. 2c) she had no further chest pain, and subsequent ophthalmology examination was normal.

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