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Case Report

Coronary artery vasospasm and cardiogenic shock as the initial presentation for eosinophilic granulomatosis with polyangiitis

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ABSTRACT

A 68-year-old woman presented to our hospital with unstable angina and was admitted for further evaluation. While hospitalized, she developed persistent angina with hypotension along with ST-segment elevation in leads V_1–V_2 along with lead aVR elevation on 12-lead electrocardiogram. Coronary angiography revealed diffuse multi-vessel coronary vasospasm most notably in the left anterior descending artery (LAD). Due to incomplete resolution of vasospasm with intracoronary verapamil and nitroglycerin, along with hemodynamic compromise requiring an intra-aortic balloon pump, percutaneous coronary intervention (PCI) of the LAD was performed. Clinical workup revealed hypereosinophlia and elevated IgE; diagnosis of eosinophilic granulomatosis with polyangiitis was confirmed with evidence of radiographic migratory pulmonary infiltrates and airway obstruction on spirometry. The patient had recurrent angina after PCI but her symptoms resolved fully after a course of corticosteroids. We attribute her refractory vasospastic angina to previously undiagnosed small/medium-vessel vasculitis.

<Learning objective: We present a case of refractory coronary artery vasospasm in the setting of eosinophilic granulomatosis with polyangiitis (EGPA). Typically, calcium channel blockers and nitrates are used to treat hyperreactive narrowing of the coronary vasculature but we propose this case was refractory to standard treatment due to the underlying pathology of EGPA. Alternative causes of coronary vasospasm when standard therapies fail should be considered.>

Introduction

Patients with eosinophilic granulomatosis with polyangiitis (EGPA; formerly known as Churg-Strauss syndrome) are traditionally diagnosed with at least 4 of the following 6 criteria: severe asthma, fleeting pulmonary infiltrates on chest radiographs, eosinophilia, paranasal sinus abnormalities, eosinophilic infiltration on biopsy specimens, and neurologic manifestations, *e.g.* mononeuritis multiplex [1]. Presentation occurs in the 4th–5th decade of life with a predilection in women (7:1) [2]. Cardiac manifestations may include: acute pericarditis, restrictive or dilated cardiomyopathy, myocarditis, arrhythmias, and sudden cardiac death. Cardiac involvement portends a poorer prognosis in patients with EGPA. Herein, we present a patient admitted for dyspnea and angina and found to have refractory coronary vasospasm causing cardiogenic shock, unresponsive to calcium channel blockers and nitrates requiring percutaneous coronary intervention (PCI) to the left anterior descending artery (LAD), and successfully treated with corticosteroid treatment after clinical diagnosis of EGPA.

Case report

A 68-year-old Caucasian woman presented in July of 2015 with complaints of intermittent and sudden onset dyspnea with classical angina radiating to her back. She had associated symptoms of nausea and vomiting with each episode that was sudden in onset, along with bilateral arm numbness. Initially, while being evaluated for admission, she was asymptomatic but

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her symptoms recurred with development of acute hypoxic respiratory failure requiring supplemental oxygen and anterior chest discomfort not relieved by nitroglycerin. She developed hypotension and was admitted to the intensive care unit for suspected acute coronary syndrome.

Her past medical history was significant for late-onset chronic bronchitis/asthma with forced expiratory volume in 1 second (FEV1) of 2.53 L (47% of predicted), complicated with presumed pneumonia that led to acute hypoxic respiratory failure requiring rapid-sequence intubation and mechanical ventilation during a prior hospitalization with similar presenting symptoms. During her earlier admission, she was medically managed for a non-ST elevation myocardial infarction, type II, in the setting of presumed sepsis [3]. In addition, she had been evaluated for chronic rhinitis with identification of nasal polyposis.

On physical examination, she appeared acutely ill and in distress. Vital signs revealed tachycardia and tachypnea. She did not have an elevated jugular venous pulse while cardiac auscultation revealed normal heart sounds. Pulmonary examination was unremarkable. Extremities were unremarkable for edema or cyanosis. No signs of rash were noted and evidence for neurological deficits was not appreciated. Complete blood count was notable for a leukocytosis of 11,100/μL and eosinophilia of 22%. Arterial blood gas revealed respiratory acidosis. Initial cardiac biomarkers were normal. Cardiac enzyme analysis was repeated after 4 h and troponin I was elevated at 0.331 ng/mL. The initial electrocardiogram (ECG) tracing while the patient was asymptomatic revealed a normal sinus rhythm without concerning ST-segment or T-wave abnormalities (Fig. 1a). A subsequent ECG recorded while the patient was symptomatic and hypotensive

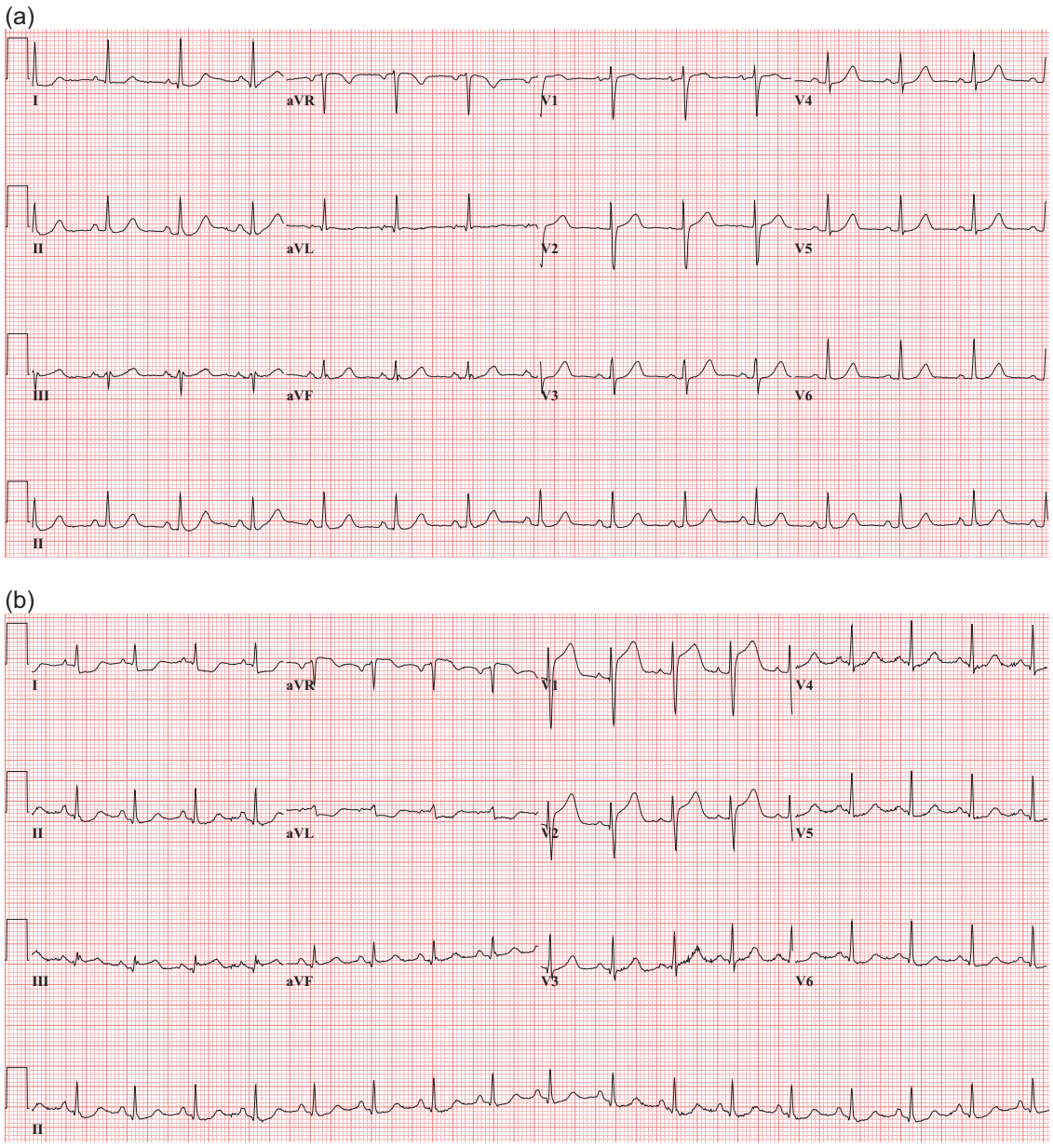

Fig. 1. (a) Initial electrocardiogram (ECG) with normal sinus rhythm and no underlying pathology. (b) Subsequent ECG with development of angina revealing ST-segment elevations in V_1, V_2, III, aVF, with reciprocal changes in I and aVL.