A 58-year-old man was admitted with a 6-week history of fever, lethargy, arthralgia, and dyspnea on exertion. He had no prior illness or cardiovascular risk factors, and with the exception of a pyrexia, his physical examination was unremarkable. Investigations were performed, including blood and urine cultures and a connective tissue screen. He was subsequently discharged with a view to an early outpatient review when he collapsed at home with cardiac arrest.

The initial rhythm was ventricular fibrillation, which was successfully cardioverted. His postarrest electrocardiogram revealed transient right bundle-branch block and his plasma troponin I concentration was raised at 8.5 μg/L. His renal function was normal, but markers of inflammation were raised, with a C-reactive protein concentration of 134 mg/L and an erythrocyte sedimentation rate of 96 mm/h. His PR3-antineutrophil cytoplasmic antibody titer was strongly positive at 90 U/mL.

An echocardiogram revealed mild concentric left ventricular hypertrophy with normal left ventricular function and no regional wall motion abnormality. He underwent coronary angiography, which excluded obstructive atheromatous disease in the major epicardial vessels but identified pruning of the distal branch vessels, including the first and second marginals and the first diagonal branch, in keeping with a systemic vasculitic process (Figure 1; Movie I of the online-only Data Supplement). A gadolinium-contrast-enhanced cardiovascular magnetic resonance scan was performed (Figure 2); functional cines confirmed basal posterolateral hypokinesis, while late enhancement sequences following contrast demonstrated patchy hyperenhancement involving the subendocardium (25% to 75% transmurality) in all 3 coronary artery territories (Movie II of the online-only Data Supplement). A renal biopsy identified areas of focal and necrotizing glomerulonephritis with crescents (Figure 3), consistent with a diagnosis of antineutrophil-cytoplasmic-antibodies-associated small vessel vasculitis. He subsequently commenced prednisone and mycophenolate mofetil and experienced a marked and rapid improvement in both his symptoms and inflammatory markers.

Coronary angiitis leading to myocardial infarction is rare, and few cases have been reported where coronary angiography was performed. Coronary involvement in systemic vasculitis may manifest with aneurysm formation, thrombosis, or coronary dissection, all of which could potentially lead to myocardial infarction. The combination of multiple areas of late enhancement in different vascular territories on cardiovascular magnetic resonance imaging with normal epicardial vessels and pruning of the smaller coronary vessels confirm that vasculitis per se was responsible for this unusual case of myocardial infarction and cardiac arrest.

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Disclosures
None.

References


Figure 2. T2-weighted image of mid short-axis (A) demonstrating increased signal (brighter) of infero-postero-lateral subendocardium indicative of myocardial edema. Late enhancement images (B, 2-chamber, C, 4-chamber and D, mid short-axis) following gadolinium contrast administration using an inversion recovery sequence identifies patchy subendocardial hyper-enhancement (bright areas demonstrated by arrows) of myocardium in multiple coronary artery territories, consistent with infarction.

Figure 3. Histology specimen from renal biopsy identifying areas of interstitial inflammation and a vasculitis (A, ×200 original magnification; arrow shows vasculitis), a focal necrotizing lesion (B, ×400 original magnification), and a cellular crescent (C, ×400 original magnification).
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Anoop S.V. Shah, Jehangir N. Din, John R. Payne, Neeraj Dhaun, Martin A. Denvir and Nicholas L. Mills

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