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.1–3 Coronary involvement in systemic vasculitis may manifest with aneurysm formation, thrombosis, or coronary dissection, all of which could potentially lead to myocardial infarction.4 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.

Sources of Funding
Dr Mills was supported by a British Heart Foundation Intermediate Clinical Research Fellowship (FS/10/024/28266).
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).
Coronary Angiitis and Cardiac Arrest in Antineutrophil Cytoplasmic-Antibody Associated Systemic Vasculitis
Anoop S.V. Shah, Jehangir N. Din, John R. Payne, Neeraj Dhaun, Martin A. Denvir and Nicholas L. Mills

Circulation. 2011;123:e230-e231
doi: 10.1161/CIRCULATIONAHA.110.981936
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/123/6/e230

Data Supplement (unedited) at:
http://circ.ahajournals.org/content/suppl/2011/02/15/123.6.e230.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/