CLINICAL IMAGE

Aortitis as a cause of severe abdominal pain

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A 64-year-old man with hypertension was referred to the hospital because of sudden severe abdominal pain radiating from the lumbar region to the lower and upper abdomen over a period of 3 days. He had experienced night sweats for about 6 months but with no symptoms of infection. A physical examination revealed only diffused abdominal pain on palpation. Blood tests revealed increased levels of inflammatory markers (leukocyte count, 11.7 G/l; C-reactive protein [CRP] levels, 132 mg/l), D-dimer (1883 μ g/l), and α -1 globulins in protein electrophoresis (6.4%). Serum amylase and lipase levels as well as IgG4 levels were within the reference ranges. Dissecting aneurysm of the aorta was suspected and computed tomography (CT) angiography was performed. It showed concentric thickening of the wall of the thoracic and abdominal aorta with expansion of the thoracic aorta in the ascending section to 40 mm and also expansion of the final section of the abdominal aorta (FIGURE 1A-D). In view of fractures to vertebrae L₃ and Th₇, dualenergy X-ray absorptiometry was used to confirm osteoporosis (T-score, -3.5). Wide-spectrum antimicrobial treatment (cefuroxime and ciprofloxacin) was started, but CRP levels remained elevated. On the basis of negative microbiological cultures (urine and blood) and serological tests for hepatitis B and C, active infections from Mycoplasma pneumoniae, Chlamydia pneumoniae, Mycobacterium tuberculosis, Ureaplasma urealyticum, Treponema pallidum and Yersinia species were excluded.

The radiological presentation, elevated levels of inflammatory markers, and the lack of response to antibiotic therapy suggested large vessel vasculitis. High doses of corticosteroids (methylprednisolone in pulses and prednisone, 1 mg/kg body weight/d) and intravenous cyclophosphamide were initiated as well as bisphosphonates and vitamin D. After 5 months of treatment (a total cyclophosphamide dose of 5.8 g), the follow-up CT angiography revealed that the inflammation of the aortic wall had been almost totally reversed (FIGURE 1E-H). Azathioprine was administered to maintain remission.

Acute aortic syndrome (AAS) is a group of sudden states with a similar clinical picture. CT makes it possible to rapidly exclude any aortic pathologies that may mimic acute aortitis, such as aortic



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FIGURE 1 A–D – Computed tomography (CT) angiography of the aorta; A – CT scans showing concentric thickening of the wall of the aortic arch to 8 mm (arrow); B – wall of the thoracic aorta to 8 mm (arrow); C – reconstruction of the sagittal plane of the aorta shows wall thickening of the aortic arch and thoracic aorta to 8 mm and of the abdominal aorta to 4–5 mm (arrows), as well as osteoporotic fracture of vertebra L₃ (asterisk); D – reconstruction of the frontal plane shows wall thickening of the thoracic and abdominal aorta, mainly on the left side; E – CT angiography of the aorta after 5 months of immunosuppressive treatment; a CT scan performed at the level of the aortic arch shows regression after treatment of the aortic wall thickening (arrow); F – segmental thickening of the aortic wall to 3 mm is seen at the level of the thoracic aorta (arrow); G – reconstruction of the sagittal plane of the aorta shows thickening of the wall to 3 mm on the left side at a distance of approximately 11 mm (arrow); H – reconstruction of the frontal plane, in which almost complete correction of the thickening of the wall of the aorta is visible; I–J – in nonenhanced CT, the wall of the aorta has a density of 40 jH (arrow); after contrast administration, it increased to 64 jH (arrow).











dissection, intramural hematoma, and penetrating atherosclerotic ulcer. The absence of any typical hyperdensity in nonenhanced CT scans in our case enabled intramural hematoma to be excluded (FIGURE 11 and 1J). Similarly, the absence of intimal disruption or intimal flap allowed us to exclude both classic aortic dissection and penetrating aortic ulcer. Concentric uniform thickening of the aortic wall with contrast enhancement in our patient, along with clinical presentation and laboratory abnormalities, suggested aortitis, the pathological term for the presence of inflammatory









lesions in the aortic wall, regardless of their underlying cause. It may be infectious (secondary to tuberculosis, syphilis, and infection by other bacteria such as *Salmonella* or *Staphylococcus* or viral pathogens) or noninfectious (occurring in large-vessel vasculitides, spondyloarthropathies associated with HLA-B27, and connective tissue diseases).¹⁻³ Although most cases of aortitis are noninfectious, the possibility of an infectious cause must always be considered because treatment strategies for infectious and noninfectious aortitis differ widely. As there were no other signs of systemic vasculitis or other symptoms of connective tissue disease in our patient, isolated aortitis was diagnosed.

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