Infectious Aortitis

A Case-Based Review of Diagnostic Clues and Consequences

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SUMMARY

Aortitis is broadly divided into infectious and non-infectious etiologies, each with distinct treatment implications. We present the case of a patient who sustained a type A aortic dissection during urgent coronary angiography for acute coronary syndrome. Clinical findings and events during the procedure raised suspicion for an underlying vascular disorder; however, the diagnosis of staphylococcal aortitis was not made until pathological examination of resected tissue. Clues to the diagnosis of infectious aortitis noted throughout the patient’s clinical course are detailed as are potential consequences of diagnostic delays and treatment decisions, underscoring the difficulties in recognizing and managing the condition. In addition, we describe a previously unreported complication of cardiac catheterization in the setting of an infectious aortopathy. (Int Heart J 2016; 57: 645-648)

Key words: Aortic dissection, Coronary angiography, Iatrogenic disease

Difficulties in diagnosing aortitis are compounded by the importance of differentiating between its infectious and non-infectious etiologies given their markedly divergent treatment implications. We describe a patient who underwent urgent coronary angiography that was complicated by iatrogenic type A aortic dissection (iAAD) requiring surgical intervention. Staphylococcus aureus aortitis was diagnosed on pathological examination. We emphasize the need for a high index of suspicion to accurately diagnose infectious aortitis (IA), review pitfalls in its recognition and management, and describe its previously unreported association and postulated contribution to iAAD during coronary angiography.

CASE REPORT

An 83-year-old woman presented to hospital with polyarthralgia, muscle stiffness, and generalized weakness following a fall. She denied fevers or rigors, chest discomfort, or jaw claudication. Her medical history included stable angina with previous coronary angioplasty, hypertension, dyslipidemia, and hypothyroidism. Her exam was notable for bradycardia at 52 bpm, an oxygen saturation of 93% on 2 L/minute of oxygen via nasal cannula, mild jugular venous distention with unre-
Figure 1. Iatrogenic aortic dissection and right coronary artery (RCA) rupture. **A-E:** Second injection of the RCA during angiography showing extravascular extravasation of contrast along the aortic wall and a new ostial lesion. First injection (not shown) had not revealed a culprit lesion or damping of the arterial waveform. **F:** Computed tomography (CT) demonstrating significant narrowing of the RCA origin and a contained coronary rupture. Residual puddling of contrast from coronary angiography was noted to extend laterally, inferiorly, and posteriorly to the ascending aorta and to be contiguous with a moderate-sized pericardial effusion. **G:** Repeat CT imaging 7 days post-catheterization revealing an increase in size of the RCA ostial pseudoaneurysm to 2.1 × 1.0 cm. **H:** Three-dimensional CT reconstruction of the RCA depicting the contained rupture. Gray arrowhead: RCA; white arrowhead: RCA pseudoaneurysm; asterisk: aorta.

Figure 2. Pathological specimens of aorta. **A:** Gross specimen of resected aortic tissue with area of necrosis and suppuration on luminal surface. **B:** Lack of underlying cystic medial degeneration or other recognizable lesion (Movat’s stain, 10×). **C:** Gram stain at zone of acute septic arteritis demonstrating necrotizing infection with pus and abundant Gram positive cocci (40×). **D:** Hematoxylin phloxine saffron stain at the zone of purulent aortitis demonstrating abundant neutrophils (40×).
ostium, prompting surgical intervention (Figure 1G-H). Her markedly elevated inflammatory markers, persistent polyarthralgia, subdeltoid bursitis, negative rheumatoid factor and anti-cyclic citrullinated peptide antibody, and suspected aortopathy had raised suspicion for an underlying inflammatory disorder, particularly giant cell arteritis (GCA). Though this diagnosis was not confirmed, given her tenuous clinical status, she was empirically initiated on oral prednisone for this possibility.

Intraoperatively perforation of the right coronary sinus of Valsalva immediately adjacent to the RCA ostium with injury to the latter were seen. Aortic root remodeling with replacement of the right coronary sinus of Valsalva using a Dacron patch and bypass of her RCA with a saphenous vein graft were performed. Post-operatively the patient developed a fever and blood cultures grew MSSA. Pathologic examination of aortic tissue showed a pseudoaneurysm with a focus of suppuration, fibrosis, and abundant Gram positive cocci in clusters with no significant underlying medial degeneration. Sterile propagation of the dissection was confirmed through areas lacking any recognizable lesion (Figure 2). Her corticosteroid therapy was rapidly tapered and cloxacinil was initiated.

Over the subsequent 2 weeks she developed progressive dyspnea and a pulsus paradoxus of 20 mmHg. Echocardiography and CT imaging revealed an expanding loculated fluid collection surrounding the aortic graft site that communicated with a large loculated pericardial collection. Pericardiocentesis confirmed infected pericardial fluid. Rifampin was added to her antibiotic therapy and repeat surgical intervention considered; however, she opted for palliation and died soon afterward. Her family declined an autopsy.

### DISCUSSION

Promptly confirming the diagnosis of IA and differentiating it from non-infectious aortic pathologies can be challenging given the varied and often non-specific presentation of patients with this disease. However, a combination of clinical, laboratory, radiologic, and/or echocardiographic findings may be helpful. Baseline patient characteristics, including male sex, older age, diabetes, risk factors for bacteremia, and known pre-existing aortic abnormalities (eg. atherosclerotic disease, cystic medial necrosis, aneurysms, congenital vascular abnormalities, or prostheses) may predispose to this condition. An important exception to this profile is thoracic IA in patients with infective endocarditis. In this setting, IA occurs in similar proportions among men and women and has a much younger mean age of diagnosis.

Clinical signs and symptoms may or may not be suggestive of IA, particularly in cases without concomitant aortic aneurysm or dissection. Fever and thoracic or back pain may be present, but are neither specific nor particularly sensitive. Laboratory findings may similarly be of limited value with the notable exception of bacteremia, which should prompt a search for an infectious source when detected, but which may not be present as in our case. *S. aureus* in the urine, as was detected in our patient, has been reported as being suggestive of concomitant bacteremia with the same pathogen, but this association is contested. Elevated inflammatory markers may raise concerns for an inflammatory process, but do not distinguish between infectious and non-infectious etiologies even if leukocytosis or neutrophilia are observed. Emerging diagnostic tools such as broad-range 16S ribosomal ribonucleic acid (rRNA) gene polymerase chain reaction (PCR) and procalcitonin may play increasingly important roles in making this distinction in select cases.

Imaging studies are often instrumental in cases of IA either by supporting the diagnosis or ruling out alternate processes. Suggestive findings on CT imaging include aortic mural thickening, periaortic soft tissue nodularity or fluid accumulation, saccular aneurysms (particularly if evolving rapidly), and gas in the aortic wall; however, their absence does not rule out this diagnosis. Data stemming mainly from cases of non-infectious aortitis have suggested a role for magnetic resonance angiography (MRA) though its availability may be a limiting factor in certain centers. Similarly, positron emission tomography (PET) has been successfully used, particularly when combined with CT or MRA and in cases of GCA. Echocardiography can also be a valuable modality to assess for signs of infective endocarditis and to visualize the aortic root and ascending thoracic aorta in particular.

Our patient was elderly and had a history of atherosclerotic disease, but was female and afebrile. Furthermore, her inflammatory markers were markedly elevated and she had leukocytosis with neutrophilia, but this was in the context of a diagnosis of community-acquired pneumonia and soon afterwards a urinary tract infection, both of which were at first felt to satisfactorily explain her laboratory abnormalities. Given that aortitis was not suspected initially, CT imaging of the thorax was not performed until after the iAAD occurred. However, at that time, aside from the dissection and pericardial collection, only atherosclerotic disease and small lymph nodes consistent with being reactive in nature were noted in the thorax. Multiple subsequent CT scans also did not reveal abnormalities that clearly suggested aortic wall inflammation despite its clear diagnosis during surgery and on pathology. As well, the patient had several echocardiographic studies pre-operatively, which reported mild thickening of the aortic root, but no evidence of infective endocarditis. Therefore, though signs and symptoms consistent with IA existed in our case and are appreciated in retrospect, they were subtle or easily explained by alternate processes. Molecular diagnostic techniques or procalcitonin were not used nor were PET or MRA. Though the latter imaging modalities have been reportedly used to diagnose and assess treatment response in IA, their potential value in our patient’s case remains speculative.

Mechanistically, in our patient’s case, it is likely that IA was due to hematogenous seeding of the aorta during bacteremia from a distant focus of infection, probably from the lower respiratory tract. The events observed during coronary angiography and the pathological findings suggest that the iAAD was triggered by unintentional impingement of the catheter on a vulnerable area of infected and necrotizing tissue. To our knowledge IA predisposing to iAAD during cardiac catheterization has not been described, including in contemporary registries that comprise up to 14 years of data or include up to 50 large European referral centers. One case of “aortitis” in a single-center series of 37 patients with iAAD following cardiac surgery or catheterization has been reported, but whether the aortitis was infectious or which cardiac procedure the patient underwent is unclear.
Importantly, early in the patient’s clinical course consideration was given primarily to a non-infectious vasculitis (particularly GCA)\(^1\) – a suspicion that led to the decision to initiate corticosteroid therapy, which may have been detrimental in the setting of an undiagnosed vascular infection. Caution regarding analogous misdiagnoses and empiric management decisions has been advised by others in the setting of complicated abdominal aortic aneurysms,\(^5\) underscoring the importance of promptly confirming the etiology of suspected or diagnosed arteritis.

**Conclusion:** Infectious aortitis is a rare disease that can be easily misdiagnosed as a more common non-infectious vascular condition or altogether missed with potentially fatal consequences. Greater awareness of this disease, its often non-specific signs and symptoms, the importance of promptly distinguishing it from its non-infectious counterparts, and the limited specificity of diagnostic tests may help clinicians to avoid pitfalls in its recognition and treatment.

**REFERENCES**