Inflammatory Abdominal Aortic Aneurysms

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Key words: abdominal aortic aneurysm, retroperitoneal fibrosis, aortitis, inflammatory aortic aneurysms

IMAJ 2005; 7: 385–387

In 1972, Walker et al. [1] first coined the term inflammatory abdominal aortic aneurysm to describe what they thought of as a distinct subset of AAA. The entity they described was an aneurysm characterized by "an unusually thick wall surrounded by extensive fibrous adhesions, involving adjoining tissues and structures." The reported incidence of inflammatory aortic aneurysms in the literature varies between 2.5% and 15% of all AAA [1–16]. The distinct clinicopathologic findings (symptomatology as described below, together with the triad of a thickened aneurysmal wall, extensive peri-aneurysmal and retroperitoneal fibrosis, with adhesions to adjacent abdominal organs) led to the view that inflammatory AAA was a separate clinical entity [1–8]. Recent evidence, however, suggests that the inflammatory AAA probably represents the extreme end of the spectrum of the inflammatory process found in all AAA, inclusive of the well-known atherosclerotic type.

Inflammatory AAA represent the extreme end of the inflammatory process found in all AAA

Etiology and pathogenesis

The etiology and pathogenesis of inflammatory AAA remain obscure [1,4,5,7,11]. Early reports advocated that the intense peri-aortic fibrosis was secondary to retroperitoneal leakage of blood from tiny subclinical perforations of the non-inflammatory AAA. However, the absence of hemosiderin-laden macrophages in the peri-aneurysmal tissue, expected to be found in the case of blood extravasation, has refuted this theory [1,4,5,11]. Although an infectious causation has been considered, bacterial cultures of the aneurysm wall have consistently been negative, as have serologic tests for syphilis [1,4,5,16]. Of interest in this regard is the finding by Tanaka and colleagues [17] that the herpes simplex virus or cytomegalovirus was more frequently present in the wall of aneurysms than in the normal aortic wall. Furthermore, these viruses were more prevalent in inflammatory than in non-inflammatory AAA. Current thinking on the pathogenesis of AAA has centered on a locally occurring immune response. Rose and Dent [18], in a landmark histologic study of 51 surgically repaired AAA, showed that an inflammatory reaction of varying intensity was present in the aneurysm wall of all specimens. Sterpetti et al. [19] described a gradual progression of inflammation from atherosclerotic to inflammatory AAA. Remarkably, the development of an inflammatory AAA from a non-inflammatory AAA, as shown by abdominal computed tomography over a 7 month period, was reported by Latifi and Heiken [20]. It is now accepted that AAA are characterized by chronic aortic wall inflammation, destructive remodeling of the extracellular matrix, and depletion of vascular smooth muscle cells. The inflammatory response involves immune system components such as macrophages, and T and B lymphocytes induce proteolytic activity mediated by cytokines. Thus, compared to normal aortic walls, increased expression of interleukins 6 and 8, tumor necrosis factor-alpha, matrix metalloproteinase and tissue type plasminogen activator have all been documented in the walls of AAA [10,21]. Of note, inflammatory AAA have a strong familial tendency compared

Although the rate of rupture of inflammatory AAA is less than that of the non-inflammatory type, the natural history is one of progressive enlargement and eventual rupture. Surgical repair is therefore indicated and should involve minimal dissection of adjoining structures

AAA = abdominal aortic aneurysm

with non-inflammatory AAA. This may imply an abnormality in the cellular or humoral responses of the immune system, possibly related to cytokine gene polymorphism [10,11,22]. In summary, the cause of both inflammatory and non-inflammatory AAA is probably multifactorial, including a combination of environmental, endothelial and genetic factors, but is in essence an immunologic process [2,6,8].

Symptomatology

Presenting symptoms are more commonly encountered in inflammatory AAA than in their non-inflammatory counterpart (65–90% vs. 8–18%, respectively). The leading symptoms are abdominal pain followed by back pain and a weight loss averaging approximately 11 kg. In fact, the triad of abdominal or back pain, weight loss, and elevated erythrocyte sedimentation rate in patients with AAA is highly suggestive of the inflammatory variant [6,8,11,14]. These aneurysms also tend to be larger than the atherosclerotic type [8,9,11,14,16]. Entrapment of the duodenum (100%) and ureters (53%) in the retroperitoneal fibrotic process is frequently observed [3,9]. Obstructive uropathy has previously been described as a presenting manifestation of inflammatory AAA and is found in about 21% of patients [1–3,9].

Diagnosis

On clinical examination, a pulsatile abdominal mass is present in 15–30% of patients. The imaging modality of choice for the preoperative diagnosis of an inflammatory AAA is abdominal CT. Characteristic findings on CT are a thickened calcified aortic wall surrounded by a low attenuation soft tissue density with relative sparing of the posterior wall [Figure 1]. This peri-aortic inflammatory mantle can be up to 1.5 cm thick and shows uniform enhancement with intravenous contrast agents. The full extent of the peri-aortic reaction and the secondary involvement of adjacent structures, in particular the duodenum and the ureters, are best defined by CT. High resolution ultrasound is also useful, although its sensitivity in establishing the correct diagnosis is 60% compared to 90% for CT. The distinct finding on ultrasound is a sonolucent halo encompassing the aneurysm. However, this halo is not always present. An important imaging clue is the fact that in inflammatory AAA the ureters are drawn medially, whereas in non-inflammatory AAA they are laterally displaced [3,4,9,11,14]. Angiography offers no information as to the presence, size or extent of the peri-aortic inflammatory process [1–15].

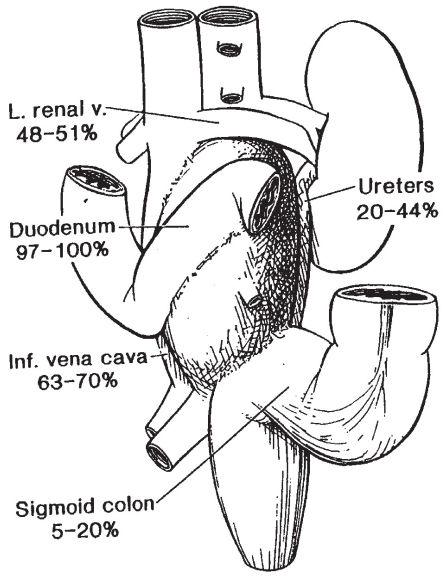

Figure 1. Inflammatory abdominal aortic aneurysm and frequency of associated adherent viscera.

Surgical management

Although inflammatory aneurysms rupture less frequently than aneurysms of the non-inflammatory type, their natural history is one of enlargement and eventual rupture [2,8,9]. Surgical repair is, therefore, the treatment of choice.

Abdominal CT is the imaging diagnostic modality of choice

Inflammatory AAA are easily recognizable at laparotomy by their characteristic white and glistening aneurysmal surface. They are usually confined to the infrarenal aorta extending to the common iliac arteries. The fourth portion of the duodenum and inferior vena cava are invariably incorporated in the fibrotic process. Other retroperitoneal structures that may be involved include the left renal, adrenal and gonadal veins, the small bowel or its mesentery, the colon and the pancreas [Figure 2]. The anterior and lateral wall thickness ranges from 1 to 3.5 cm, but is not uniform [Figure 3].

The optimal definitive treatment is graft inclusion [Figure 4]. State-of-the-art methodology advocates against dissection of surrounding structures. Employing the technique of lim-

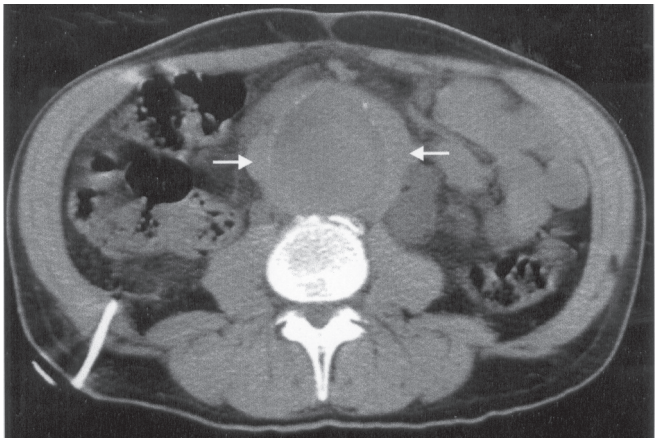

Figure 2. Large inflammatory abdominal aortic aneurysm clearly delineated by cross-sectional view on computed tomographic scan.

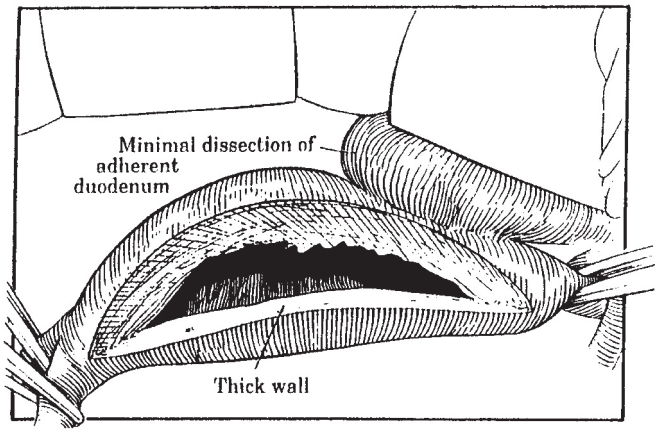

Figure 3. Anterior and lateral wall of abdominal inflammatory aneurysm; note thickness of the aneurysmal wall.

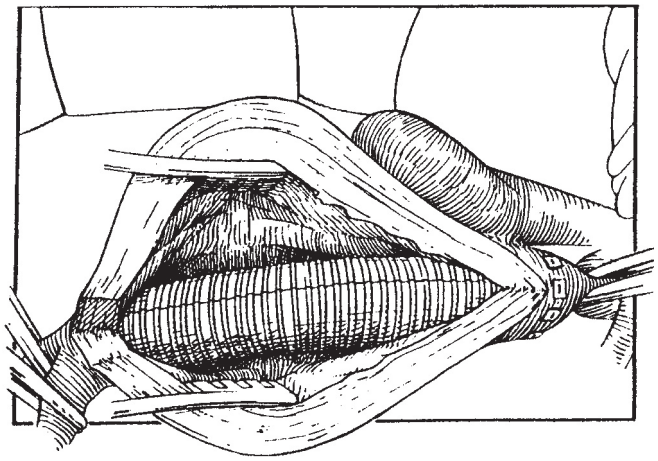

Figure 4. Resection and graft replacement of inflammatory abdominal aortic aneurysm.

ited dissection, surgical mortality rates (3–4%) match those of non-inflammatory aneurysms [2,4,6,8,14,15]. Preoperative ureteral catheterization may be helpful in the intraoperative identification of the ureters and prevention of ureteral injury [4,5]. Ureterolysis is unnecessary because the retroperitoneal inflammatory reaction tends to subside after repair of the aneurysm with spontaneous resolution of ureteral obstruction [4,5,15]. The new onset of ureteral obstruction, its recurrence or its progression following graft replacement, is extremely rare [1,15,16]. Following repair of the inflammatory AAA, clinical symptoms resolve and the erythrocyte sedimentation rate declines. Postoperative CT scans show gradual regression, either complete or partial, of the inflammatory mantle in up to 70% of cases [4,5,7,11,15,16]. Interestingly, no progression of the inflammatory reaction has been documented.

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