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Short Communication

Inflammatory Atherosclerotic Abdominal Aortic Aneurysms

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Walker., *et al*. first introduced the term inflammatory aneurysm [1]. Several reports describing ureteral involvement by the peri aneurysmal fibrosis had already appeared.

The Society for Cardiovascular Pathology describes 4 types of abdominal aneurysm: atherosclerotic (degenerative), inflammatory, non-infectious aortitis (including giant cell aortitis, IG4 related aortis) and suppurative.

Inflammatory aneurysms are associated with severe atherosclerosis [2-4]. The term Inflammatory Atherosclerotic Abdominal Aneurysms (IAAA) should be used, to introduce a clear distinction between Inflammatory IAAA and other forms of aortitis and periaortitis, which can result also in aneurysmal degeneration. IAAA shows severe atherosclerosis and excessive degree of adventitial inflammation, consisting mainly on lymphocytes and plasma cells. The thickness of the aortic wall should exceed 4 mm to make a diagnosis of IAAA. The main differential diagnosis is periaortitis. The presence in the adventia of histological findings not typical for atherosclerosis (like granulomata, extensive IG4+), combined with no significant or mild atherosclerosis, favors the diagnosis of periaortitis [5].

Table 1, 2 describe the current classification of aortic diseases [5].

Diagnosis	Characteristics of Inflammation	Common Examples
Atherosclerosis	Mild inflammation	Macrophages in the intima-Lymphocytes in the intima and adventitia –Scattered plasma cells in the adventitia
Atherosclerosis with excessive inflammation	Severe inflammation	- IAAA: Features of atherosclerosis with excessive adventitial inflammation. - Atherosclerosis with excessive neutrophilic inflammation.
Aortitis/Periaortitis	Severe inflammation with no evidence of atherosclerosis	-Granulomata/giant cells; Lymphoplasmacytic component

Table 1: *Major diagnostic classes of inflammatory aortic disease (*).*

**Modified from Stone., et al [5].*

Inflammatory Pattern	Examples
Granulomata/Giant Cells	Giant cell arteritis Granulomatosis with polyangiitis Rheumatoid arthritis Takayasu arteritis Sarcoidosis Eosinophilic granulomatosis with polyangiitis
Lymphoplasmacytic Pattern	IG4-Related Disease Lupus
Mixed inflammatory pattern without Granulomata	Cogan Syndrome Behçet Disease Relapsing Polychondritis

Table 2: *Inflammatory patterns in not infectious aortitis and periaortitis (*).*

**Modified from Stone., et al [5].*

We can hypothesize that in the majority of the patients with IAAA, the aneurysm is an atherosclerotic-degenerative pathology, in which the abnormal inflammatory reaction is related to sudden compression of the lymphatic and venous system close to the aneurysmal wall The possibility of inflammatory changes is related to a specific reaction of the patient, to a sudden increase in size of the aneurysm (and a consequent inability of the venous and lymphatic network to compensate), or to a genetically determined inability of the lymphatic-venous system to compensate the compression related to the enlarging aneurysm.

Several reports have described patients with simple atherosclerotic aneurysm which suddenly showed the CT changes related to an IAAA [3-8]. In all patients there was a sudden increase in size of the aneurysm.

IAAA are more common in the abdominal aorta, and rare in other arterial segments. In patients with proven aortitis, the thoracic aorta and other arterial segments are often involved, simultaneously or not with the abdominal aorta [9,10].

Anatomic studies show a close correlation between the lymphatic network and the distribution of inflammation in the IAAA. The lymphatic network is very close to the lateral and anterior wall of the aorta, and it is almost absent in the posterior wall (Figure 1). The inflammatory changes in IAAA are related to the lateral and anterior wall, and almost disappear at the origin of the renal arteries.

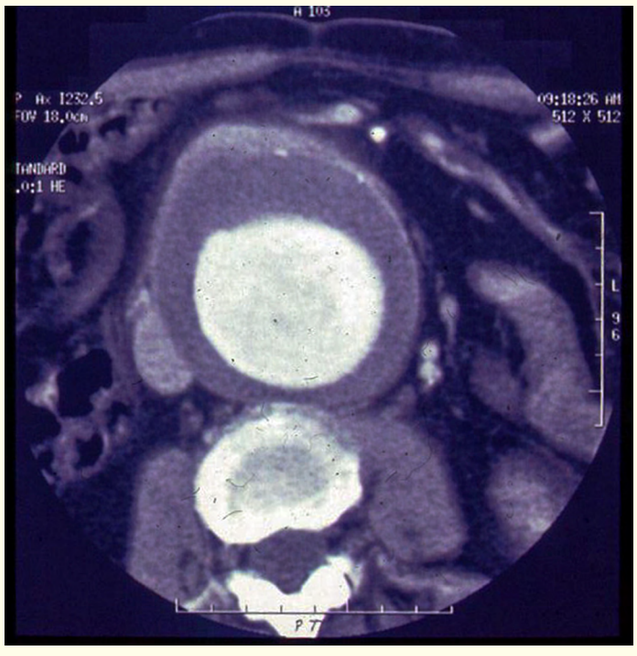

Figure 1: *Common CT appearance of an Inflammatory Atherosclerotic Abdominal Aortic Aneurysm. The peri aneurysmal fibrosis is more evident in the anterior and lateral wall. It is minimal in the posterior wall.*

Free intraperitoneal rupture in IAAA is less common than in simple atherosclerotic-generative aneurysms. The majority of the ruptures in IAAA are contained. It is possible that the sudden compression of the lymphatic vessels by the contained rupture induces inflammatory changes.

Several reviews have shown the possibility of regression of the peri-aneurysmal fibrosis after open and endovascular surgery [11-13]. In both types of procedure the aneurysmal wall is left in place, partially in open surgery and in toto in endovascular surgery. If the inflammation is secondary to an immunological reaction, we should expect the inflammatory changes to persist.

After endovascular surgery, perianeurysmal fibrosis disappears or partially regresses in more than 70% of the patients [11-16]. In these patients there is a close correlation between shrinkage of the aneurysm and regression of inflammatory changes, even when anti-inflammatory therapy is not given [17,18]. Regression of inflammatory changes, including partial or total regression of ureteral entrapment, is less common in patients without hematological and clinical symptoms of inflammation and in patients with larger aneurysms. It is probable that in case of long standing inflammation, with fibrosis, the regression of perianeurysmal reaction is minimal and ureteral entrapment, if present, rarely regresses, despite a simultaneous aggressive cortisone therapy. Thus, regression of inflammation is more common in case of recent-onset inflammation with higher prevalence of cellular component, that in case of old-standing.

Clinical Implications

Anti-inflammatory therapy, alone, can reduce the inflammation and resolve acute symptoms, but there is a risk of free rupture, and long term steroid therapy has major side effects and requires an exhaustive follow-up.

Short term steroid therapy, before and after surgery, can represent a valid option to reduce acute inflammation, to allow an easier operative act, and to prevent post-implantation syndrome. The possibility of regression of the inflammation and of ureteral entrapment is higher in patients with smaller IAAA and in patients with recent onset of the inflammatory process, when inflammatory cells predominate and there has been no time for fibrosis to develop. This indicate the appropriateness for an aggressive surgical approach in patients with IAAA, even for smaller aneurysms, before fibrosis occurs. In combination to surgery, endovascular or open, anti-inflammatory therapy can help to facilitate anatomical and clinical regression of inflammation, especially when the perianeurysmal inflammation is recent and it is composed mainly by inflammatory cells.

Open surgery determines a faster regression of the inflammation because of the more rapid resolution of the compression, but brings higher operative risks. Endovascular surgery carries less operative risks, and it is better tolerated and accepted by the patient. In case of endovascular surgery every effort should be made to prevent endo leaks, to have a faster shrinkage of the aneurysm, to relieve compression.

Controversies exist about the use of ureteral stents. In patients with acute inflammation and obstructive uropathy, the use of stents is appropriate to allow urine outflow. In patients with chronic fibrotic inflammation, the use of a stent could be complication prone and less useful, considering that the kidney has a chronically reduced function, and it will take a long time for a possible ureteral entrapment resolution.

Endovascular surgery, leaving the aneurysmal wall in situ, seems not appropriate for inflammatory aneurysms developing as aortitis in the general context of an immunological disease. The content of the aneurysmal wall, in this scenario, is probably a driver for an inflammatory reaction, and endovascular surgery do not allow to remove this stimulus. In patients with inflammatory aneurysm secondary to autoimmune aortitis/periaortitis open surgery seems a better option, as reported by Kasashima., *et al*. for patients with IG4-related aneurysm [19]. However, it is very important to define the type of aortitis. For example, good results have been reported for endovascular repair in patients with Bechet's disease, in whom open surgery is difficult and complication prone for the weakness and friability of the aortic wall tissue.

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