



Revisión

Inflammatory abdominal aortic aneurysm: a review

Aneurisma aórtico abdominal inflamatorio: revisión del tema

Álvaro Morales Palma¹, Alejandro Campos Sáenz², Cristián Siqués Urzúa¹, Alejandro Campos Gutiérrez^{2,3}, Francisco Moraga², Daniel Gutiérrez Véliz^{1,3}

¹Hospital de Urgencia Asistencia Pública. Santiago, Chile. ²Hospital Clínico de la Universidad de Chile. Santiago, Chile. ³Clínica Las Condes. Santiago, Chile

Abstract

Inflammatory abdominal aortic aneurysm (AAAI) is a rare variant of abdominal aortic aneurysms. It is characterized by dense periaortic fibrosis and thickening of the aortic wall, associated with macroscopic and histological characteristics of chronic inflammation. This chronic inflammation has several pathways at the systemic and molecular level and various theories have been studied ranging from an autoimmune etiology to that related to some infections. AAAI has some different characteristics from its non-inflammatory simile, among the most important is the frequency of involvement of nearby structures such as the ureters and duodenum, which places AAAI as a surgical challenge.

Surgical alternatives include open and endovascular surgery, also associated with systemic immunosuppressive treatment in most cases. This review aims to analyze pathophysiological, diagnostic and treatment aspects of this pathology.

Resumen

El aneurisma aórtico abdominal inflamatorio (AAAI) es una variante poco frecuente de los aneurismas aórticos abdominales. Se caracteriza por poseer una densa fibrosis periaórtica y un engrosamiento de la pared aórtica, asociado a características macroscópicas e histológicas de inflamación crónica. Esta inflamación crónica posee diversas vías a nivel sistémico y molecular y se han estudiado diversas teorías, que van desde una etiología autoinmune hasta la relacionada con algunas infecciones. El AAAI tiene algunas características distintas de su símil no inflamatorio. Entre las más importantes se encuentra la frecuencia con la que afecta a estructuras cercanas, como los uréteres y el duodeno, lo que sitúa al AAAI como un desafío quirúrgico.

Las alternativas de resolución quirúrgica incluyen cirugía abierta y endovascular, asociando además un tratamiento sistémico inmunosupresor en la mayoría de los casos. Esta revisión tiene como objetivo analizar aspectos fisiopatológicos, diagnósticos, de tratamiento y seguimiento de esta patología.

Keywords:

Inflammatory aortic aneurysm. Aortic aneurysm. Aortic disease.

Palabras clave:

Aneurisma aórtico inflamatorio. Aneurisma aórtico. Patología aórtica.

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Correspondence:

Daniel Gutiérrez Véliz. Hospital de Urgencia Asistencia Pública. Curicó 345. 8330145 Santiago. Región Metropolitana, Chile
e-mail: dguty20@gmail.com

INTRODUCTION AND OBJECTIVES

Inflammatory abdominal aortic aneurysms (IAAA) are a variant of abdominal aortic aneurysms (AAA) characterized by dense periaortic fibrosis, thickening of the aortic wall, and macroscopic and histologic features of nonspecific chronic inflammation. These inflammatory findings are often indistinguishable from other causes of aortitis; therefore, diagnosis is not based solely on the inflammatory state. Their incidence is approximately 3-10 % of all AAAs. They occur more frequently in men, with a mean age at presentation of 65 ± 3 years, which is 10 years younger than for noninflammatory AAA (1).

The first description of the term “inflammatory aneurysm” was made in 1972 (2). Subsequently, the Mayo Clinic triad was described, characterized by “aneurysmal wall thickening, extensive perianeurysmal fibrosis, and retroperitoneal fibrosis with adhesions to adjacent organs” (3). The inflammatory process may extend to neighboring organs such as the ureters, duodenum, and, less frequently, the small intestine, inferior vena cava, and sigmoid colon.

The objective of this review is to analyze the main clinical and pathophysiologic aspects related to diagnosis and to provide guidance on the study and management of this disease (Table I).

ETIOLOGY AND PATHOPHYSIOLOGY

IAAAs are part of a group of disorders collectively classified as chronic periaortitis, which also includes idiopathic retroperitoneal fibrosis (IRF) and idiopathic periaortitis (4). However, the Society for Cardiovascular Pathology consensus defined aortitis and periaortitis as inflammatory conditions of the aorta and periaortic tissue that cannot be attributed solely to atherosclerosis (5).

Pathologically, IAAAs resemble noninflammatory AAAs (N-AAA) in showing atherosclerotic changes in the intima, marked thinning of the media with elastin degeneration, and loss of smooth muscle cells. However, the adventitia in IAAAs is markedly thickened (1-4 cm), with extensive fibrosis and chronic inflammatory infiltrate including plasma cells, histiocytes, lymphoid follicles, and granulomas.⁵ Although some inflammatory changes can also be observed in N-AAAs, they are not as pronounced as in IAAAs (6).

The etiology of IAAA remains controversial. Historically, multiple theories have been proposed, ranging from chronic blood leakage producing inflammation to lymphatic drainage abnormalities (7-10). These theories have largely been abandoned, with current research focused on the inflammatory response, both against elements of the atherosclerotic plaque and

Table I. Summary of the Most Relevant Clinical Aspects of Inflammatory Abdominal Aortic Aneurysm

Definition	AAA with dense periaortic fibrosis and mural thickening associated with histologic features of chronic inflammation
Incidence rate	3-10 % of all AAAs
Diagnostic workup	– CCTA of the entire aorta is the study of choice – PET-CT supports decisions regarding immunosuppressive therapy (assessing metabolic activity of inflammation) – CRP/ESR: unreliable for differentiating the inflammatory component
Indication for surgery	– Same size criteria as for noninflammatory AAA (> 5.0-5.5 cm) – Selected cases with symptoms refractory to medical therapy
Surgical approach	– Endovascular repair (EVAR) as first choice – Open repair: for anatomies unfavorable for EVAR
Follow-up and postoperative management	– Most patients require continued immunosuppressive therapy – Serial CCTA $\pm$ PET-CT; guideline intervals not clearly defined
Postoperative outcomes	– 1-year mortality: 14 % after open repair, 2-3 % after EVAR – Graft-related complications: 20 % EVAR, 9 % open repair

in association with systemic inflammatory diseases (eg, lupus, rheumatoid arthritis, or giant cell arteritis), which is currently the most widely accepted theory (11). Although the specific antigen responsible for initiating the response has not yet been identified, many authors have focused on elastin-derived peptides (EDPs)—extracellular matrix degradation products—as the initiators and propagators of the inflammatory response in IAAA (12). Among the best-known risk factors is active smoking. Historical series reported active smoking in 77-100 % of patients with IAAA; however, more recent series have not demonstrated significant differences in smoking prevalence between IAAA and N-AAA. Nonetheless, there is consensus that smoking plays an important role in the development of the disease by producing systemic inflammation, arteritis, metalloproteinase dysregulation, and exacerbating inflammation in genetically predisposed individuals (11-14). A family history of AAA has been described in 17 % of IAAA vs 1.7 % of N-AAA, suggesting an important genetic predisposition to the disease, especially in the context of systemic vasculitides such as giant cell arteritis or IgG4-related systemic arteritis (present in up to 50 % of IAAA cases) (11,15-18).

Less widely accepted theories include associations between IAAA and infectious agents such as *Chlamydia pneumoniae* and cytomegalovirus (18).

DIAGNOSIS

A total of 84 % of IAAAs are symptomatic, in contrast with N-AAAs, which are symptomatic in only 9 % (18,19). Symptoms include abdominal, lumbar, or flank pain. Unlike N-AAA, pain in IAAA is not clearly associated with rupture risk and may instead be attributable to the inflammatory process. Nitecki described a clinical presentation of back pain, pulsatile abdominal mass, and elevated erythrocyte sedimentation rate (ESR) as highly suggestive of IAAA, which may also be associated with significant weight loss, anorexia, weakness, and fever (20).

When adjacent organs are affected, clinical signs may be directly related to the involved organ, including intestinal obstruction, hydronephrosis, or inferior vena cava syndrome, among other factors.

Inflammatory parameters are often elevated but not universally, and remain nonspecific.¹

Imaging modalities are essential for diagnosis. Ultrasound may show aortic wall thickening and periaortic fibrosis, which can appear as a hypoechoic sleeve. Another important finding is the presence or absence of hydronephrosis. A limitation of this modality is the inability to distinguish mural thrombus from the aneurysm wall.

The current reference standard for diagnosis is coronary computed tomography angiography (CCTA), which demonstrates the typical 4-layer appearance of inflammatory aneurysm: central lumen, hypodense thrombus, calcified aortic wall, and periaortic fibrosis that enhances with contrast (mantle sign), the latter being pathognomonic (Fig. 1). Moreover, the CCTA provides information about the extent of the inflammatory process and the involvement of adjacent organs such as the ureters, small intestine, and inferior vena cava. IAAAs are located in the infrarenal abdominal aorta in 33-100 % of patients; however, chronic inflammatory changes may also be identified in the thoracic aorta in 2-10 % of patients (11).

Magnetic resonance imaging is indicated in patients with contraindications to iodinated contrast but offers no significant advantage over CCTA except for avoiding radiation exposure.

Positron emission tomography-computed tomography (PET-CT) is useful in identifying tissue with high metabolic activity, allowing differentiation between retroperitoneal fibrosis related or unrelated to cancer, but it does not allow differential diagnosis between IAAA and infectious abdominal aortic disease (4). PET-CT has also been reported to support decisions on corticosteroid therapy; greater metabolic activity suggests higher likelihood of response (21).

Differential diagnosis with infectious aneurysm is based on 2 criteria:

1. Morphologic criteria: CCTA shows fusiform morphology in IAAA, while infectious aneurysms are generally saccular and may demonstrate gas bubbles within the perianeurysmal inflammatory tissue.
2. Laboratory criteria: IAAA shows elevated inflammatory parameters such as immunoglobulins and interleukins; in contrast, infected aneurysms often show marked elevations in C-reactive protein (CRP) and procalcitonin.

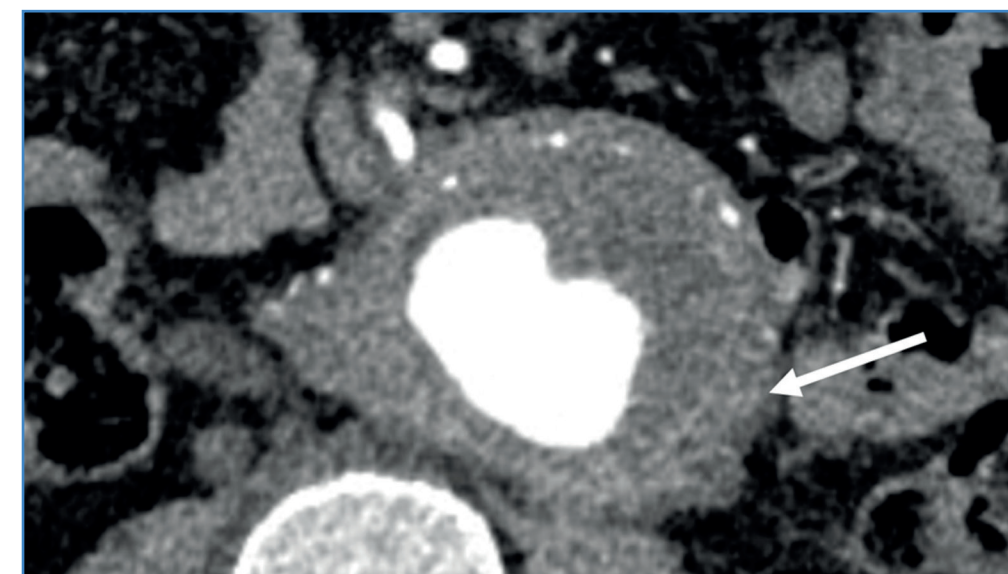


Figure 1. CCTA of an IAAA. The typical 4-layer appearance is shown: central lumen, hypodense thrombus, calcified aortic wall, and periaortic fibrosis (arrow) with contrast enhancement (mantle sign).

In recent years, the relationship between immunoglobulin G4 (IgG4) and IRF has attracted increasing interest. IgG4-related disease was first described in association with the pancreas (autoimmune pancreatitis) and has since been extended to other organs and systems. In 2008, Kasashima et al described a new clinicopathologic entity within IAAA—IgG4-related IAAA—which may account for up to 57 % of IAAA cases.¹⁷ These are characterized by significantly elevated serum IgG4 concentrations, which may directly influence treatment decisions given the high corticosteroid sensitivity. Differentiating IgG4-related IAAA is important, as they tend to be less painful and carry a lower rupture risk than non-IgG4-related IAAA. Pathologically, they are characterized by greater adventitial thickening and increased infiltration of IgG4-positive plasma cells, and may also be associated with elevated IgE and antinuclear antibodies.

MANAGEMENT OF INFLAMMATORY ABDOMINAL AORTIC ANEURYSM

The management of IAAA is complex and requires a multidisciplinary approach. Options include medical management and open or endovascular surgical repair. Although the risk of rupture is generally lower than in N-AAA, surgical indications regarding aneurysm diameter and growth rate are extrapolated from N-AAA and reflected in the most recent SVS and ESVS

guidelines (19,21). However, the evidence remains limited and of low quality.

MEDICAL MANAGEMENT

The goal of medical management is to halt progression of the periaortic fibroinflammatory reaction, reverse compression of the ureters and other adjacent organs, improve systemic symptoms, and prevent relapse. In patients with symptomatic retroperitoneal fibrosis associated with an aneurysm that does not meet surgical criteria, exclusive medical therapy may be offered. Classic treatment consists of glucocorticoids. Although there is no consensus on dose or duration, several retrospective studies have shown clinical improvement and reduction of the periaortic fibrotic mantle.²² As general guidance, the ESVS recommends initiating prednisone (or equivalent) at 30-80 mg/d until disease control is achieved, followed by gradual tapering to a target of 5-10 mg/d after 1 year (19).

Other options include immunosuppressive agents such as methotrexate, cyclophosphamide, and azathioprine,²³ although results to date are modest and derive from small retrospective series rather than randomized controlled trials. Their principal role is in refractory cases or as adjuncts/substitutes to corticosteroid therapy to reduce required dose and adverse effects (19,24).

More recently, tamoxifen has been proposed as a useful adjunct to corticosteroid therapy.¹⁹ Rituximab

has shown benefit in IgG4-related disease; however, that study included only 2 patients with aortitis/periaortitis and assessed remission of disease activity and IgG4 levels rather than aneurysm size and extent of periaortic fibrosis (25).

Response is assessed with serial imaging, demonstrating reduction of the periaortic fibrotic mantle, which may take months.¹² Reported response rates range from 67 % to 87 %.^{12,24} Recurrence rates are high, ranging from 24 % to 63 % (26,27). Recent literature shows that IgG4-related IAAA with renal involvement carries greater risk of progression to end-stage renal disease and higher rates of ureteral interventions (28,29). Conversely, several authors attribute the lower rupture risk described in IAAA to the periaortic inflammatory reaction and aortic wall thickening; theoretically, corticosteroid therapy could weaken the wall and thereby increase rupture risk (1).

SURGICAL MANAGEMENT

The reported rupture risk of IAAA is low (< 5 %) (3). However, diameter-based indications for repair are similar to those for noninflammatory AAA.¹⁹ Elective surgery may be performed after a prolonged course of corticosteroids—generally 3–6 months of prednisone—preferably in cases with periaortic organ involvement, with ureteral pigtail catheters or hemodialysis as needed.³⁰ Rarely, surgery may be indicated in symptomatic cases refractory to medical therapy to control the inflammatory process.

OPEN SURGERY

LOpen repair was the standard treatment for IAAA until recent years, when experience with endovascular aneurysm repair (EVAR) began to be reported. Mortality with open IAAA repair ranges from 6 % to 11 % and exceeds historical mortality for noninflammatory AAA,³¹ mainly because the operative field is hostile due to periaortic fibrosis, leading to higher postoperative complication rates. A hostile field increases the risk of injury to adjacent structures (duodenum, ureter, left renal vein, inferior vena cava) and may even precipitate postoperative pancreatitis (11).

Intraoperatively, IAAA appears whitish and shiny (Fig. 2) and is often adherent to the duodenum, left renal vein, inferior vena cava, and ureters. Extensive dissection of periaortic tissues is discouraged to avoid iatrogenic injury.

Approach options include retroperitoneal (RP) and transperitoneal (TP) routes (Fig. 2). Several authors advocate a modified TP approach with limited dissection to reduce risk to adjacent organs and to obtain better proximal and distal control of the IAAA away from the most thickened wall segments (32,33). This technique may require suprarenal aortic control in up to 40 % of cases (34), as well as reconstruction to the external iliac or femoral arteries. Most patients require preoperative ureteral pigtails to relieve hydronephrosis and/or to identify the ureters intraoperatively.

However, other series emphasize that the RP approach affords superior exposure and proximal neck control by enabling posterior aortic access, where fibrosis and inflammation are often less pronounced. RP repair has been associated with fewer pulmonary, cardiac, infectious complications, and hematomas, although not consistently across studies. Technical challenges include difficult exposure and control of the right renal artery and right common iliac artery—amenable to balloon occlusion—as well as challenging dissection around structures encased in fibrosis (eg, ureters, duodenum) (33,35,36).

Comparative studies of the 2 approaches exist but are older and often include a predominance of degenerative aneurysms.³⁷ A more recent review shows TP is the most widely used approach (87–100 %), achieving 87 % technical success with 30-day mortality ranging from 0 % to 32 % (11). Mortality increased linearly with aneurysm diameter and leukocyte count and tended to be higher in symptomatic cases.³⁸ After open repair, regression of periaortic inflammation and fibrosis has been observed in up to 86 %, and associated hydronephrosis regresses in up to 80 %; however, complete resolution may take years (34).

Given the heterogeneity and controversy in the evidence, surgeon and team experience is critical, keeping in mind that repairing an IAAA differs from repairing a noninflammatory AAA. As a general guide, most surgeons prefer the TP approach because dissection is easier and the risk of injury to adjacent struc-



Figure 2. Open approach to an IAAA. Whitish retroperitoneal fibrosis is shown (arrow).

tures is lower (1,11). In our experience, we recommend TP for juxtarenal or infrarenal IAAA with preoperative ureteral catheterization. If greater exposure is needed, the left renal vein can be ligated as close as possible to its junction with the inferior vena cava to preserve collateral venous drainage.

ENDOVASCULAR SURGERY

Multiple studies have described EVAR as a safe alternative for IAAA repair (39–41). EVAR has been associated with lower 30-day mortality (2–4 %) (42) and lower morbidity vs open repair (42). However, de novo fibrosis after EVAR has been reported, and hydronephrosis may persist or progress despite endovascular treatment (42,43). Close follow-up is therefore essential, and immunosuppressive therapy should be considered to control periaortic inflammation (42).

A systematic review comparing open surgery and EVAR for IAAA showed higher rates of hydronephrosis regression after open repair vs EVAR, with no differences in 30-day mortality or reduction of periaortic inflammation (42). However, 1-year mortality favored EVAR. Prosthetic limb occlusion occurred in up to 20 % of EVAR cases due to extrinsic compression from fibrosis (19,42).

In the specific population with IgG4-related IAAA, an analysis of outcomes after EVAR or open surgery showed that EVAR-treated patients had higher rates of persistent symptoms, increased periaortic fibrosis,

higher IgG4 levels, and greater aneurysm sac enlargement during follow-up (44).

The most recent ESVS guideline recommends an endovascular-first approach over open repair, weighing lower perioperative morbidity and mortality against potential risks of persistent hydronephrosis or fibrosis, which can be managed with double-J ureteral stents or immunosuppression, respectively (19).

FOLLOW-UP

Postoperative immunosuppressive therapy is required in most patients, particularly those with IgG4-related IAAA and persistent symptoms. Acute-phase reactants (CRP or ESR) can support follow-up assessment but are insufficient alone, as they do not always reflect metabolic disease activity and often normalize early in follow-up.¹⁹ Accordingly, imaging-based surveillance is indispensable. CCTA is the most established modality to monitor regression of the inflammatory–fibrotic process (15,45,46). PET-CT now enables more precise measurement of metabolic activity in response to medical therapy, allowing initiation, adjustment, or cessation of corticosteroids. After open repair, regression of periaortic inflammation and fibrosis has been reported in up to 86 %, though it may take years (19). Graft-related complications occur in 9 % (19). At 1 year, the 1-year all-cause mortality rate is 14 % after open repair and 2 % after EVAR

($p = 0.01$) (42). Despite medical or surgical treatment, the inflammatory/fibrotic process may persist lifelong, as may the risk of hydronephrosis; therefore, lifelong surveillance with imaging by a multidisciplinary team is essential (19).

CONCLUSIONS

Important differences exist between IAAA and N-AAA. The former is less common, occurs in younger patients—typically in the sixth decade—and is frequently symptomatic.

The rupture risk of IAAA appears lower; however, diameter-based indications for surgery (> 55 mm) generally follow those for N-AAA. A favorable response to corticosteroids has been observed; thus, all patients should receive them initially and postoperatively, especially those undergoing EVAR. Open repair of IAAA is technically challenging and may be associated with higher perioperative morbidity and mortality but with lower rates of persistent fibrosis and subsequent hydronephrosis. EVAR, while not free of follow-up issues, carries lower perioperative morbidity and mortality and is recommended over open repair in current guidelines. Posttreatment follow-up—medical or surgical—should be lifelong under a multidisciplinary team.

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