

A VIEW OF ATHEROSCLEROTIC AND NON-ATHEROSCLEROTIC ABDOMINAL AORTIC ANEURYSMS

UMA VISÃO DOS ANEURISMAS DA AÓRTICA ABDOMINAL ATHEROSCLERÓTICOS E NÃO
ATHEROSCLERÓTICOS

Editor: Anderson Barbosa Baptista

Publicado: janeiro/dezembro 2025.

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DOI: [10.20873/RPT2026fluxocontinuo21762](https://doi.org/10.20873/RPT2026fluxocontinuo21762)

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RESUMO:

O aneurisma de aorta abdominal (AAA) é o tipo mais frequente de aneurisma, apresentando prevalência significativa na população idosa. Há hoje uma razoável soma de evidências de que um dos componentes fundamentais para que o aneurisma se desenvolva é a destruição das lâminas e fibras elásticas. Diferentemente do que ocorre na aorta ascendente, na maior parte dos aneurismas de aorta abdominal essa elastólise decorre da infiltração por células inflamatórias mononucleares, que são fontes de enzimas que agem sobre o componente elastogênico, tais como elastase, metaloproteinases, serinoproteinases e catepsinas. Assim, o tipo de AAA mais comum é o de origem aterosclerótica – a porção abdominal é a que parte da aorta que apresenta mais aterosclerose; no entanto, apenas uma minoria das pessoas com aterosclerose grave da aorta desenvolvem aneurismas – são as que apresentam componente inflamatório importante. Além dos ateroscleróticos, há os aneurismas de etiologia variada que também apresentam processo inflamatório e consequente destruição da parede aórtica, sendo que cerca de metade desses casos está relacionado à presença de IgG4. Esse tipo específico de aneurisma apresenta aspecto fisiopatológico peculiar, além de diferenças que vão desde clínicas até epidemiológicas quando comparado ao aneurisma clássico aterosclerótico. Dessa maneira, partindo do pressuposto que esses aneurismas são poucos estudados, o objetivo desse estudo foi ampliar o conhecimento sobre diversos tipos de AAA, enfatizando suas diferenças quanto à fisiopatologia e os aspectos clínicos, o que poderia otimizar o diagnóstico diferencial dos casos de AAA na clínica médica.

PALAVRAS-CHAVE: Doença Relacionada a Imunoglobulina G4; Aneurisma da Aorta Abdominal; Doença Esclerosante Relacionada a IgG4.

ABSTRACT:

The abdominal aortic aneurysm (AAA) is the most common type of aneurysm, with a significant prevalence in the elderly population. Currently, there is a reasonable amount of evidence that one of the fundamental components for an aneurysm to develop is the destruction of elastic sheets and fibers. Unlike what occurs in the ascending aorta, in most abdominal aortic aneurysms, this elastolysis results from infiltration by mononuclear inflammatory cells, which are sources of enzymes that act on the elastogenic component, such as elastase, metalloproteinases, serine proteinases and cathepsins. Thus, the most frequent type of AAA is that of an atherosclerotic origin – the abdominal portion of the aorta is the most affected by atherosclerosis; however, only a minority of people with severe aortic atherosclerosis develop aneurysms – those whose aorta bears a significant inflammatory component. Besides the atherosclerotic type, other aneurysms are also affected by substantial inflammation, approximately half of these related to the presence of IgG4. This specific type of aneurysm has a peculiar pathophysiological aspect and different clinical and epidemiological characteristics when compared to classic atherosclerotic aneurysm. Therefore, assuming that these aneurysms are rarely studied, the aim of this review was to elucidate the distinct types of AAA, emphasizing their differences in terms of pathophysiology and clinical aspects, which could optimize the differential diagnosis and treatment of AAA in the medical practice.

KEYWORDS: Immunoglobulin G4-Related Disease; Abdominal aortic aneurysm; IgG4-related sclerosing disease.

INTRODUCTION

The aortic aneurysm is characterized by dilation of the arterial wall, involving all 3 layers (intima, media and adventitia), with an increase of 50% or more of the normal diameter of the aorta or a diameter greater than 30mm^{1,3}. Abdominal aortic aneurysm (AAA) is a progressive and multifactorial vascular disease located between the aortic hiatus of the diaphragm and the bifurcation of the iliac arteries⁴. Several pathophysiological mechanisms are involved, with emphasis on the inflammatory process, oxidative and biomechanical stress, in addition to pronounced proteolysis of the aortic wall. These mechanisms lead to structural damage mainly due to destruction of the extracellular matrix (ECM), via marked elastosis and subsequent collagen deposition, and apoptosis of smooth muscle cells (SMC) resulting in arterial dilation^{2,5}.

The main type of AAA are those of atherosclerotic origin, which represent the majority of cases; but there are also AAAs of other etiologies, including infections, drugs, autoimmune diseases and lymphatic obstruction, which are denominated inflammatory abdominal aortic aneurysms (IAAA)⁶. In both AAAs, plasma cells play numerous roles in the pathophysiological process, mainly in IAAAs, receiving more attention recently due to new discoveries involving the action of these cells and their products, especially immunoglobulins IgA, IgM and IgG, in the development of an inflammatory cascade involving several chemical mediators leading to progressive destruction of the aortic wall².

Among IAAAs, there is a specific type in which there is intense infiltration of IgG4-producing plasma cells, known as IgG4-related IAAA.

The aim of this review was to assess the distinct types of AAA, with a greater focus on the pathophysiology and clinical aspects.

METHODS

This systematic review has as its purpose the clarification of the following question: "What is the importance of IgG4-related inflammatory abdominal aortic aneurysm in clinical practice?". All images included in this article were made using *BioRender software*.

Inclusion criteria

Original and review articles published since the first article about this theme (published in 1982) until 2024 were included, all of which were related to atherosclerotic or inflammatory abdominal aortic aneurysm, focusing in the studies about the last one subject.

Exclusion criteria

Articles not related to AAA or IgG-related inflammatory abdominal aortic aneurysm, case reports, errata, news articles, book chapters, letters, comments, editorials, duplicates, perspective, not peer-reviewed, and published in a non-English language were not included.

Search strategy and study selection

Pubmed and ScienceDirect were used. Free terms were used on both platforms. All articles since the discovery of the “Inflammatory IgG4-related aneurysm” in 1972 were gathered in May 2024 using the following search strategies: “Abdominal Aortic Aneurysm”; “Atherosclerotic Aneurysm”; “Inflammatory Abdominal Aneurysm”. All citations were managed using Zotero in order to eliminate duplicates and organize the article selection steps consistent with inclusion and exclusion criteria. Then, applying the criterias cited above 25 articles were selected for the results.

RESULTS

Table 1. Selected studies and information gathered

Authors	Title	Results
Kasashima et al.	A new clinicopathological entity of IgG4-related inflammatory abdominal aortic aneurysm	Differences between atherosclerotic AAA and IAAA
Keisler et al.	Abdominal Aortic Aneurysm	AAA definition
Prucha et al.	Abdominal aortic aneurysm as an IgG4-related disease	IgG4-related sclerosing disease properties
Aggarwal et al.	Abdominal aortic aneurysm: A comprehensive review	AAA definition
Yuan et al.	Abdominal Aortic Aneurysm: Roles of Inflammatory Cells	Atherosclerotic AAA pathophysiology
Anagnostakos et al.	Abdominal aortic aneurysms	AAA management
Lattanzi et al.	Abdominal aortic aneurysms: pathophysiology and clinical issues	Atherosclerotic AAA pathophysiology
Kasashima et al.	Adventitial matrix metalloproteinase production and distribution of immunoglobulin G4-related abdominal aortic aneurysms	IgG4-related inflammatory abdominal aortic aneurysm pathophysiology
Matsumoto et	Anatomy and Physiology for the	AAA anatomy and physiology

al.	Abdominal Aortic Aneurysm Repair	
Zhang et al.	B lymphocytes in abdominal aortic aneurysms	IgG4-related inflammatory abdominal aortic aneurysm pathophysiology
Ocana et al.	Characterisation of T and B lymphocytes infiltrating abdominal aortic aneurysms	Atherosclerotic AAA pathophysiology
Tanios et al.	CXCR4: A Potential Marker for Inflammatory Activity in Abdominal Aortic	Atherosclerotic AAA pathophysiology
Pérez-García et al.	IgG4-aortitis among thoracic aortic aneurysms	IgG4-related inflammatory abdominal aortic aneurysm pathophysiology
Hourai et al.	IgG4-positive Plasma Cells in Inflammatory	IgG4-related inflammatory abdominal aortic aneurysm pathophysiology
	IgG4-related cardiovascular disease.	IgG4-related inflammatory abdominal aortic aneurysm pathophysiology
Kasashima et al.	IgG4-related inflammatory abdominal aortic aneurysm	IgG4-related inflammatory abdominal aortic aneurysm clinical manifestations
Kasashima et al.	Inflammatory Abdominal Aortic Aneurysm: Close Relationship to IgG4-related Periaortitis	IgG4-related inflammatory abdominal aortic aneurysm histological findings
Tang et al.	Inflammatory Abdominal Aortic Aneurysms	IAA etiologies
Walker et al.	Inflammatory Aneurysms Of The Abdominal Aorta	Discovery of a non-atherosclerotic aneurysm
Raparia et al.	Inflammatory aortic aneurysm: possible manifestation of IgG4-related sclerosing disease	IAA etiologies
Furusho et al.	Involvement of B Cells, Immunoglobulins, and Syk in the	Atherosclerotic AAA pathophysiology

	Pathogenesis of Abdominal Aortic Aneurysm	
Xu et al.	Multidisciplinary diagnosis and management of inflammatory aortic aneurysms	IgG4-related inflammatory abdominal aortic aneurysm management
Ishizaka et al.	Multifocal fibrosclerosis and IgG4-related disease	Relation between AAAs and IgG4-related disease
Zen et al.	Retroperitoneal and aortic manifestations	Relation between AAAs and IgG4-related disease
Kasashima et al.	Upregulated interleukins (IL-6, IL-10, and IL-13)	IgG4-related inflammatory abdominal aortic aneurysm

Source: author

The atherosclerotic abdominal aortic aneurysm

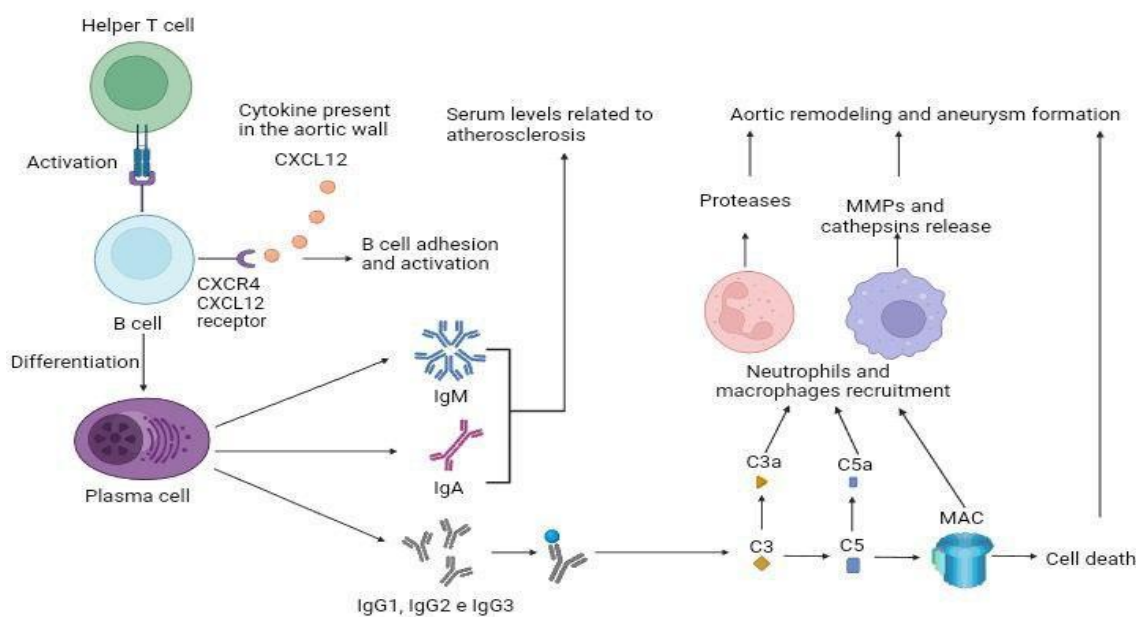
The pathophysiology of AAAs of atherosclerotic origin is related to an inflammatory microenvironment that involves the activation of several immune cells, both the innate immune response (neutrophils and macrophages) and adaptive immunity (T cells and plasma cells)². Plasma cells are mainly found in the adventitial layer of AAAs - although the limits between the layers are often lost due to the loss of the elastic layer that separates them - and are present in numerous mechanisms in the development of AAA, such as production of cytokines, immunoglobulins (IgA, IgM and IgG), complement activation, T cell regulation and antigen presentation to T cells^{2,7,8}. The role of IgA and IgM antibodies in atherosclerotic AAA has not yet been elucidated, but it is known that their serum levels are positively correlated with the degree of atherosclerosis⁸. On the other hand, it is known in relation to IgG that there is activation of the complement system via the classical pathway (IgG1, IgG2 and IgG3) with assembly of the membrane attack complex (CAM) and formation of chemoattractants, such as C3b and C5b, that lure neutrophils and macrophages, resulting in remarkable structural damage to the aortic wall⁸. Studies using mice and humans corroborated this idea by identifying significant amounts of IgG on aneurysm, those being responsible for chronic inflammation and local damage, as well as induction of the release of MMP-9 and IL-6 by macrophages and T cells, which intensified the destruction of the middle layer of the aorta^{8,9}.

Furthermore, plasma cells may exacerbate adventitial and middle layer remodeling by activating matrix metalloproteinases (MMPs), cathepsins, proteases and SMC

apoptosis². The presence of Syk tyrosine kinase was also observed in atherosclerotic aortic tissue of mice. This protein is important for the activation and functioning of immunoglobulins, which indicates that Syk is associated with the pathophysiology of atherosclerotic AAA⁹.

Moreover, infiltrative B cells in human atherosclerotic AAA samples express CXCR4, which is a chemokine receptor responsible for the adhesion, differentiation, recruitment and activation of B and T lymphocytes at the site of inflammation. This receptor binds to CXCL12, which is a cytokine found in high levels in aortic walls, especially in the adventitial layer, thus hypothesizing the contribution of the interaction of CXCR4 and CXCL12 in the development of atherosclerotic AAA¹⁰ as shown in the figure 1.

Figure 1. Atherosclerotic aneurysm pathophysiology. The B cell is triggered by the helper T cell, inciting cell differentiation and IgA, IgM and IgG immunoglobulin secretion. IgG activates the complement system, setting off a cascade of recruitment and activation of neutrophils and macrophages, which release inflammatory mediators forming the AAA. This effect is amplified by the cytokine CXCL12, located mainly in the adventitial layer of the aorta, through binding to the B cell receptor CXCR4.



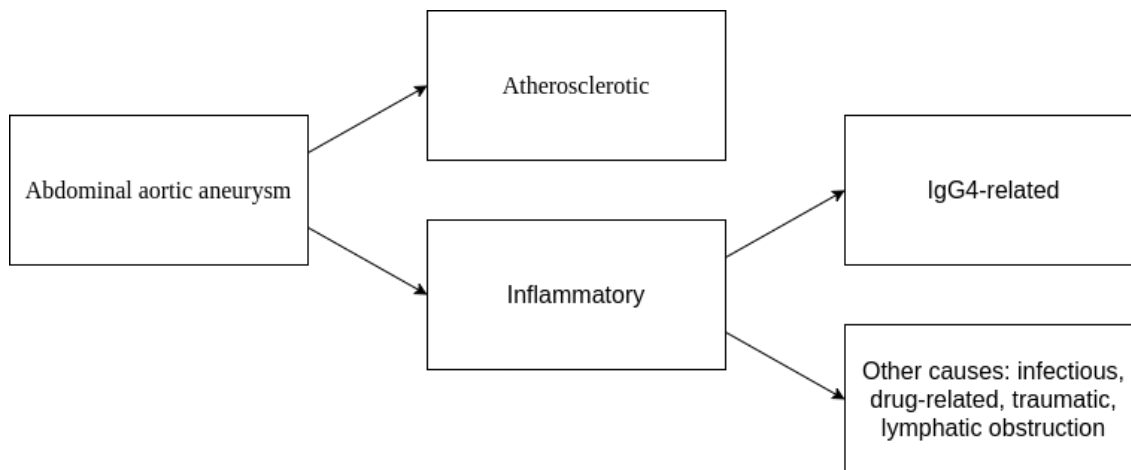
Source: author

The inflammatory abdominal aortic aneurysm

IAAA is classified as an aneurysm of inflammatory origin and has many distinct etiologies, from chronic infections and drugs such as methysergide, analgesics, antihypertensives, to aortic trauma, lymphatic obstruction, autoimmune diseases and

infiltration of IgG4-producing plasma cells^{11,14}. Flowchart 1 demonstrates these divisions in the AAA classification.

Flowchart 1. AAA classification.



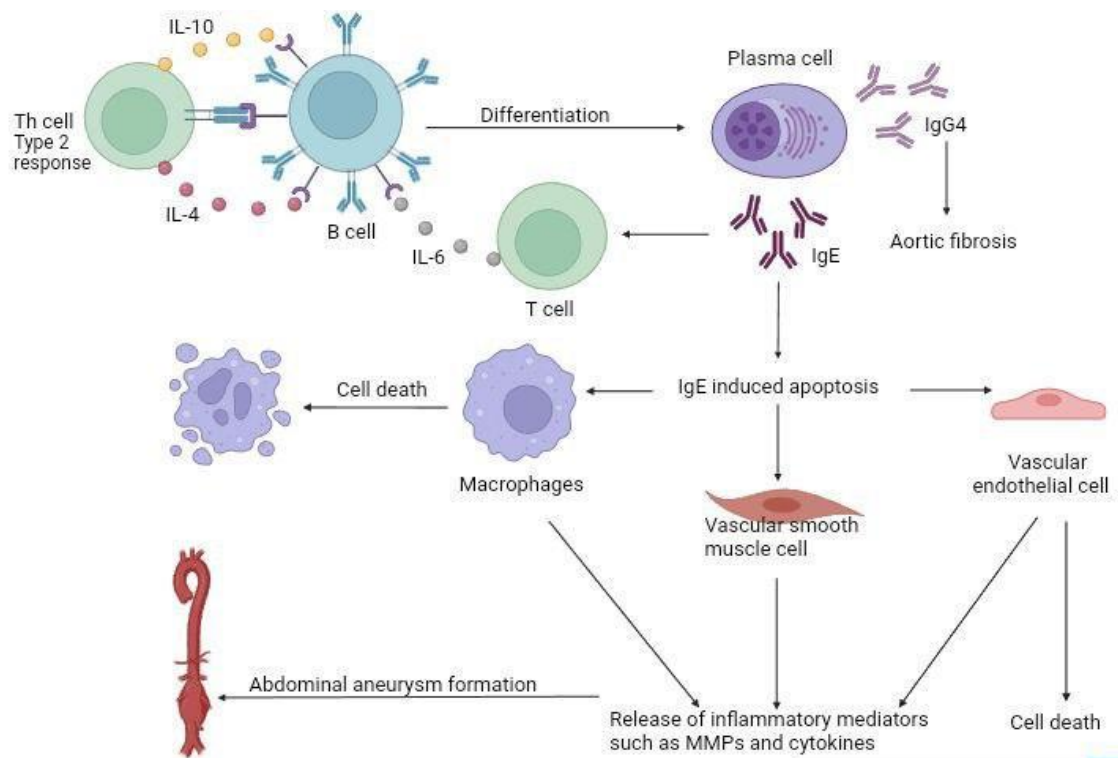
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IgG4-related inflammatory abdominal aortic aneurysm

The IgG4-related IAAA accounts for approximately half (40%) of cases of inflammatory aneurysms¹⁵. This type of aneurysm begins with an inflammatory process of the adventitia known as non-infectious aortitis, which targets the middle layer of the aorta, causing destruction of the elastic lamellar fibers¹⁵⁻¹⁸. It is believed that the immune response consists of Th2 activation, with the production of IL-4 and IL-10, which act by inducing the formation and expansion of IgG4 and IgE from plasma cells. This intense production of IgE incites T cells to synthesize IL-6, which induces more differentiation of plasma cells amplifying the secretion of IgG4. Studies have shown positive correlations between serum levels of IL-6 and thickening of the adventitia, which represents the degree of activity and fibroplasia of this type of aneurysm. Therefore, high serum levels of IgG4 are associated with aortic fibrosclerosis and retroperitoneal and perianeurysmal fibrous adhesion^{18,19}.

IgE antibodies also play another relevant role in the pathophysiology of IAAA. They act by stimulating macrophages, vascular smooth muscle cells (VSMC) and endothelial cells to undergo apoptosis and release their inflammatory mediators, such as cathepsins and MMP, which cause damage and promote remodeling of the aortic wall^{6,8}, as illustrated by figure 2.

Figure 2. Pathophysiology of IgG4-related IAAA. Activation of the Th2 profile response stimulates plasma cell differentiation and the production of IgE and IgG4. IgE, in addition to stimulating the synthesis of more IgG4, induces apoptosis of macrophages, vascular smooth muscle cells and vascular endothelial cells. These cells release inflammatory mediators that lead to aortic remodeling and AAA formation. IgG4 is related to aortic fibrosis.



Source: author

Another pertinent property of IgG4-related IAAA is the pattern of MMP expression in the adventitia accountable for aortic frailty and fibrosis. It is known that MMP-9-producing cells are directly related to IgG4-producing plasma cells, aneurysm diameter and adventitia thickening. Furthermore, the number of cells producing MMP-2 and MMP-9 present in the adventitia is higher in this type of aneurysm, compared to others. Therefore, it was suggested that serum levels of MMP-9 should be considered as predictors of the activity and progression of IgG4-related IAAA²⁰.

Differences between atherosclerotic AAA and IgG4-related IAAA in the clinical practice. Clinical manifestations

When comparing clinical manifestations, IgG4-related IAAA presents more frequently with low-grade fever, abdominal pain and back pain compared to atherosclerotic AAA,

especially in younger individuals. Additionally, IgG4-related IAAA can be associated with vasculitis involving other areas of vasculature besides the aorta and presenting symptoms specific to the affected vessel^{6,12,21}. Markers of systemic inflammation such as erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and leukocytosis are also more prominent in IgG4-related IAAA^{6,21}. Histologically, IgG4-related IAAA differs from atherosclerotic AAA by presenting active chronic inflammation with a harsher infiltration of plasma cells. Moreover, extensive fibrous adhesions occur with a greater frequency in adjacent tissues, retroperitoneal and perianeurysmal tissue being noteworthy examples, which is an unusual finding in atherosclerotic AAAs¹³. In the specific case of aneurysms due to IgG4-associated aortitis, the fibrosis often has a coiled pattern.

Management

In terms of therapy, particularly in IgG4-related IAAA with an extensive periaortic fibrosis, the use of corticosteroids is regarded as a promising medical treatment, nevertheless, it is still considered dubious, in view of the fact that at the same time the therapy would be able to reduce inflammation and consequent adhesion of the aortic and periaortic tissues, it can also contribute to weakening the vessel wall, hence increasing the risk of rupture²². Furthermore, the use of immunosuppressive therapy is often necessary in the postoperative period in patients with IgG4-related IAAA due to the systemic symptoms of this type of aneurysm²³. Regarding the surgical approach for IgG4-related IAAA, studies favor an open approach, because of the necessity for intervention on the extra-aortic tissue and its manifestations (fibrosis and adhesions)^{20,23}. Conversely, in the atherosclerotic AAA an endovascular approach is preferred, except when an unfavorable anatomy is identified, for instance, due to the presence of thrombi or occlusions²⁴. Lastly, there is evidence that IgG4-related IAAA is linked to a greater number of deaths after aneurysm repair surgery^{22,25}.

CONCLUSION

Even though both are inflammatory in nature, atherosclerotic AAA differs from IAAA in terms of etiology, histological findings and clinical manifestations. This differentiation is important clinically due to the repercussions on other organs, especially due to the extra-aortic manifestations in IgG4-related IAAA and due to the different approach in the treatment of both aneurysms. Atherosclerotic AAA is well characterized in the literature, and its pathophysiology is associated with atherosclerosis, which insidiously causes mechanical and structural changes in the aortic wall, which leads to weakening and the formation of aneurysms.

The majority of cases of IAAA are classified as IgG4-related, in which the immune response involves the activation of the Th2 profile, more commonly seen in allergic diseases. In this subtype of IAAA there is intense infiltration of IgG4-producing plasma cells associated with aortic fibrosclerosis and fibrous adhesion to adjacent tissues such as the retroperitoneum.

Given the limitations of the literature on this topic, it is important to highlight the need for future research to achieve greater depth and wider applicability in clinical practice.

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