# The Kidney's False Alarm: When a Suspected Renal Colic Reveals a Hidden Aortic Threat


## Abstract

We report the case of a 76-year-old patient without cardiovascular risk factors, who was admitted for acute abdominal and low back pain. Diagnostic imaging revealed an intramural aortic hematoma, a rare but life-threatening cardiovascular emergency. This case highlights the importance of rapid diagnosis and multidisciplinary management.



**Keywords: Intramural Aortic Hematoma (IAH); Aortic Dissection;Acute Aortic Syndromes**


## Introduction

Intramural aortic hematoma (IAH) represents a major component of the spectrum of acute aortic syndromes, standing alongside with classic aortic dissection, penetrating atherosclerotic ulcer, and aortic rupture. (1)

First described as a distinct pathological entity in the late 20th century, IAH is now recognized as a life-threatening condition characterized by hemorrhage within the aortic media without a detectable intimal tear. Its clinical significance has grown in recent decades as advances in imaging—particularly contrast-enhanced computed tomography—have allowed for more accurate differentiation between IAH and classic dissection, a distinction with important prognostic and therapeutic implications.

The prevalence of IAH varies across studies but is estimated to represent 10–30% of acute aortic syndromes, depending on the population and diagnostic criteria. Consistent with existing literature, hypertension remains the strongest and most frequently reported risk factor, present in over 80% of cases. Current data suggest that the natural history of IAH is dynamic, with possible progression to overt dissection, aneurysmal dilation, or rupture if not promptly recognized and adequately treated.

Consequently, early identification and risk stratification are essential, especially for Type A IAH, which carries a high mortality and typically mandates surgical intervention.(2) In contrast, Type B IAH is generally managed medically, though careful surveillance is crucial given the risk of complications.

Comparative studies have highlighted that although IAH shares clinical features with classic dissection—most notably abrupt chest or back pain—it often carries a distinct pathophysiological background, frequently associated with vasa vasorum rupture rather than intimal disruption. This unique mechanism may explain differences in radiologic appearance, clinical course, and therapeutic response. As modern management strategies evolve, IAH continues to occupy a critical yet nuanced position within the acute aortic syndromes, underscoring the need for heightened clinical awareness and multidisciplinary coordination.

# Case Presentation

**Patient presentation and clinical findings and diagnostic assessment:**

A 76-year-old patient**,** with no cardiovascular risk factors and no significant past medical history**,** except for regular follow-up for glaucoma for the past two years, was admitted to the emergency for acute low thoracic pain radiating posteriorly to the dorsal region. The pain occurred at rest**,** andwas described as constrictive**,** lasted approximately 30 minutes**,** and was accompanied by recurrent angina-like episodes**.**

The initial electrocardiogram was nonspecific, showing sinus tachycardia with features of ventricular hypertrophy

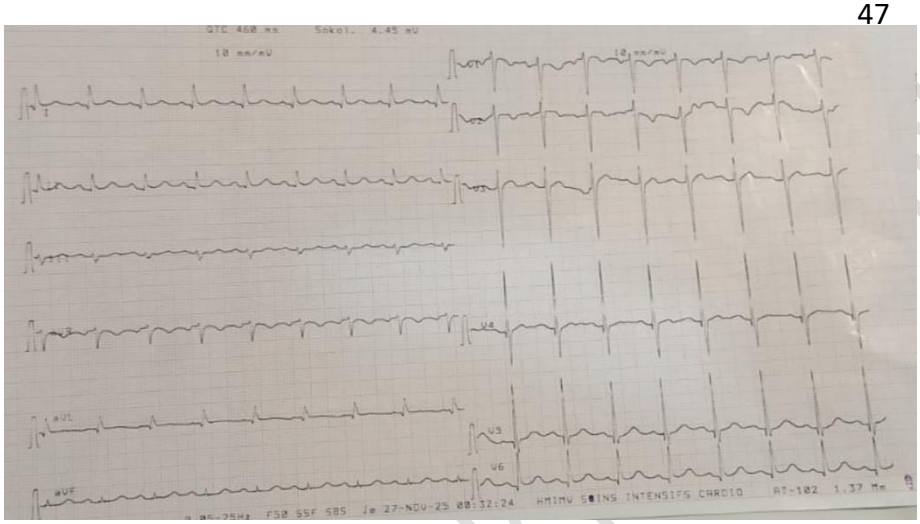


**Figure 1:** ECG performed on admission in the emergency department showed sinus tachycardia with evidence of left ventricular hypertrophy and associated secondary repolarization abnormalities

On arrival, the patient was conscious, fully oriented, and hemodynamically stable, with a GCS score of 15/15. He was in pain but remained eupneic and afebrile. Vital signs revealed sinus tachycardia at 99 bpm, an oxygen saturation of 98%, and markedly elevated blood pressure at 196/104 mmHg in both arms.

Cardiac auscultation revealed no murmurs or rales, and there were no signs of heart failure. Abdominal examination was unremarkable.

A bedside transthoracic echocardiogram (TTE) was performed, demonstrating preserved left and right ventricular systolic function, non-dilated atria, and normal filling pressures. A small pericardial effusion was noted, along with dilation of the thoracic aorta and mild aortic regurgitation.

Aorticmeasurements on TTE:

- Left ventricular outflow tract (LVOT): **22 mm**
- Sinus of Valsalva: **45 mm**
- Sinotubularjunction: **43 mm**
- Ascendingaorta: **48 mm**
- Aorticarch: **41 mm**
- Descendingthoracicaorta: **40 mm**
- Abdominal aorta: **37 mm**

Aorticregurgitationparameters:

- Regurgitant orifice area (ROA): **9 mm²**
- Regurgitant volume (RV): **22 mL**

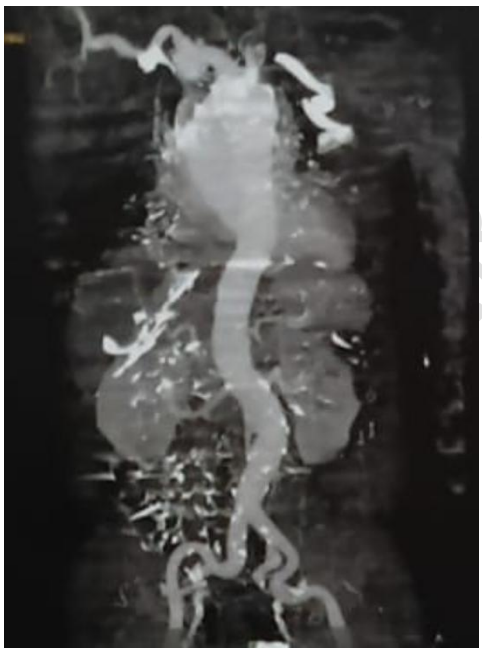
**Laboratory evaluation revealed a marked elevation in troponin levels (15× the upper reference limit), while renal and hematologic parameters remained within normal ranges. Lipid values were adequately controlled. Lactate dehydrogenase (LDH) was elevated at 340 U/L.** All relevant serologic tests were negative

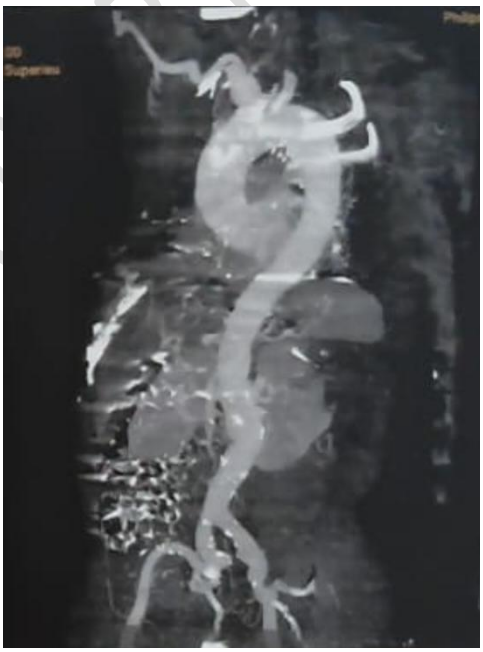
An urgent CT angiography demonstrated circumferential wall thickening of the abdominal aorta—sparing the visceral branches—with contiguous extension throughout the thoracic aorta, The overall imaging pattern was most suggestive of either an aortitis or an extensive aortic intramural hematoma.

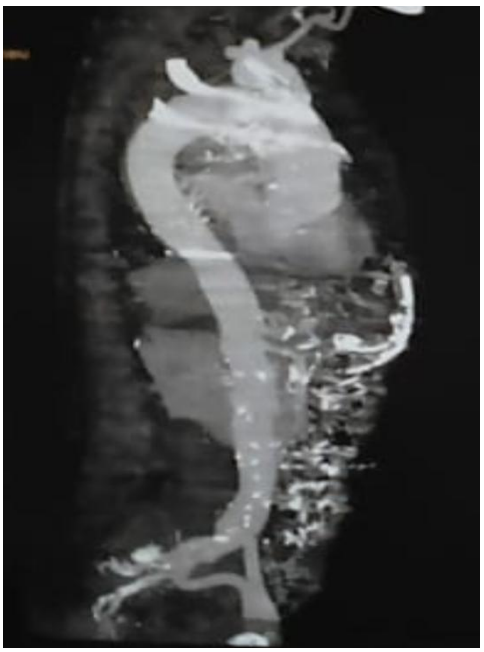
Aortic measurements on CTA:

- Ascending aorta measured 48 mm
- The aortic arch measured 36,5 mm
- The descending thoracic aorta measured36 mm

**Figure 3: SCANNER**







To differentiate between these two entities, an 18F-FDG PET scan was performed, which demonstrated an absence of pathological hypermetabolic uptake, thereby effectively ruling out aortitis..

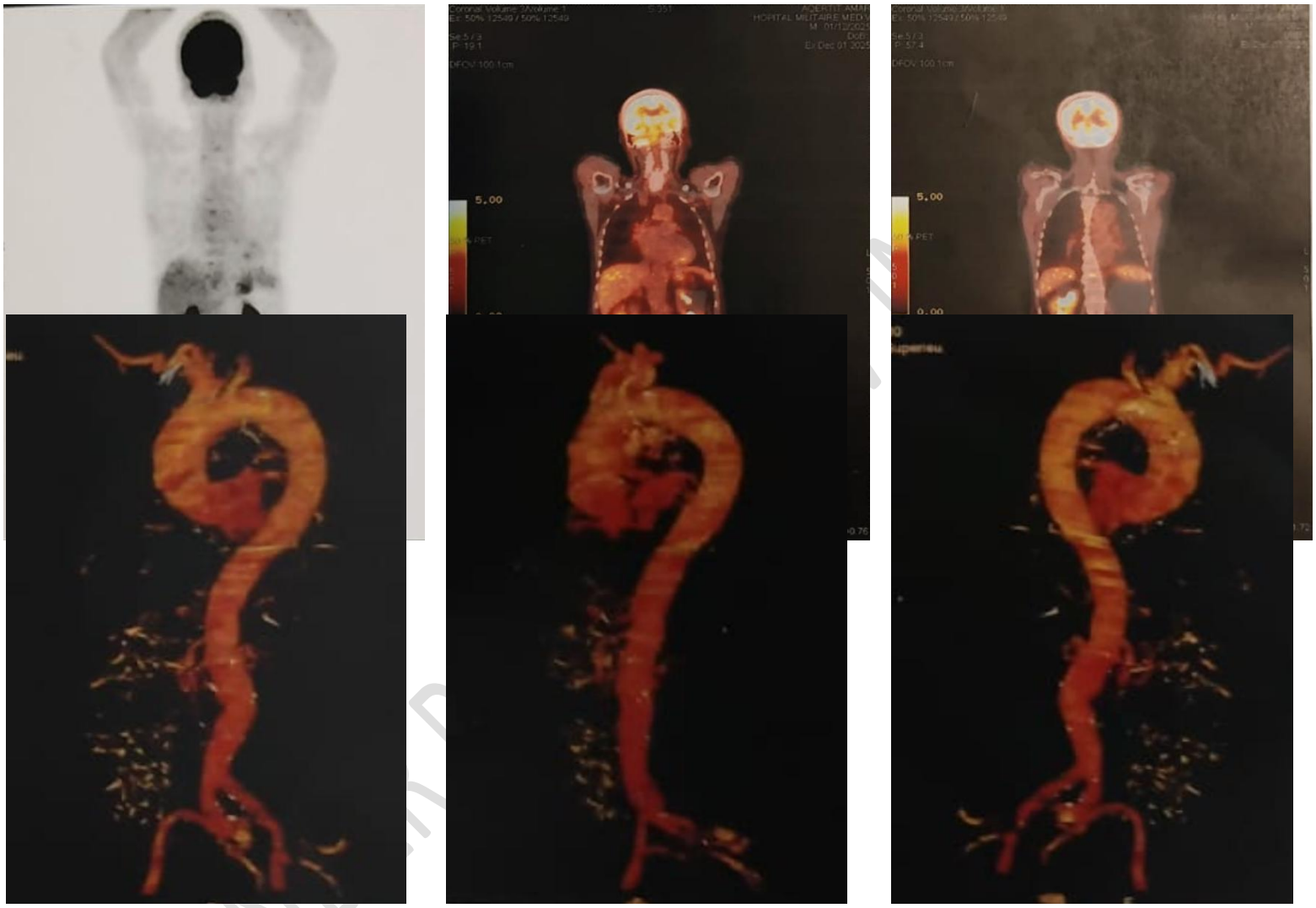



**Figure 4:** $^{18}$F-FDGPET SCAN

**Therapeutic decision:**

The therapeutic decision was conservative, with no intervention performed. Antihypertensive medical therapy was initiated, along with close monitoring of blood pressure.

# Discussion

Intramural aortic hematoma (IAH) is part of the group of conditions referred to as Acute Aortic Syndrome (AAS), which also includes classical aortic dissection and penetrating atherosclerotic ulcer. (3)Although less common than dissection (dissection accounts for ~ 85–

95% of AAS), IAH makes up a substantial portion of cases. Pathophysiologically, IAH is characterized by bleeding into the media of the aortic wall — often related to rupture of the vasa vasorum or ulcer-like atherosclerotic lesions — without a visible intimal tear. (3) This mechanism weakens the aortic wall and can predispose to aneurysm formation, progression to classical dissection, or even rupture. (4)

One of the main difficulties in IAH lies in differentiating it from other causes of aortic wall thickening — notably inflammatory aortitis (for example in IgG4-related aortitis). Indeed, inflammatory aortic disease may mimic IAH on imaging, presenting as circumferential wall thickening without an obvious intimal tear.(5)In this context, relying solely on morphological imaging may be insufficient.

In our case, to exclude aortitis we added a functional imaging modality: a PET scan. The absence of pathological hypermetabolic uptake along the aortic wall strongly argues against active inflammatory aortitis, and thus supports the diagnosis of IAH rather than a vascular inflammatory disease.

This diagnostic strategy — combining anatomical (CT / MRI) and functional (PET) imaging — is increasingly recognized as a robust approach when the differential includes IAH vs inflammatory aortitis. (5)

Unlike classical aortic dissection — which tends to follow a more predictable (though often severe) course — IAH displays a highly variable and dynamic natural history. It may:

- regress spontaneously,
- remain stable,
- evolve into classical dissection, or
- lead to aneurysm formation or rupture. (6)

In long-term follow-up studies, some IMH resolved completely (in a subset of patients), whereas others progressed to dissection or aneurysm. (6)For example, in one 6-year follow-up study with serial MRI, a notable proportion of IMH cases reabsorbed without aortic dilation, while others developed ulcer-like lesions or localized dissections. (6)2/17/2026 12:12:00 PM

Prognostic factors identified in the literature include maximal aortic diameter (e.g. > 50 mm associated with higher risk), hematoma thickness, presence of ulcer-like projections, and high blood pressure. (6)In contrast, patients with smaller aortic diameter (< 50 mm), thinner hematoma, and stable clinical course often show favorable outcomes with medical management. (7)

These data emphasize the unpredictable but potentially benign course of IAH in selected cases, underscoring the importance of individualized management and close surveillance.

Given the heterogeneity of IAH, the optimal management remains debated. For IAH involving the ascending aorta (type A), many authors historically recommend urgent surgical repair — analogously to classical type A dissection — due to the risk of life-threatening complications. (3)

However, accumulating evidence supports a more nuanced approach: in carefully selected, stable patients (small aortic diameter, limited hematoma thickness, no signs of imminent rupture), a conservative strategy (medical therapy + blood pressure control + close imaging follow-up) may be justified — sometimes with good long-term outcome.(8)

If surgical or endovascular treatment is chosen, current data indicate that outcomes after ascending aorta repair for IAH are comparable to those for classical dissection — but with a higher rate of postoperative pericardial effusion in some series. (9)For descending aorta (type B) IAH, evidence supports medical management similar to type B dissection, except when complications arise (growth, ulceration, dilation, rupture risk). (7)

Endovascular repair (TEVAR) is increasingly used in complicated IMH cases, although its role in the acute phase remains debated due to potential complications and lack of long-term data. (10)

2/17/2026 12:12:00 PMBecause IAH can evolve in multiple ways over time — regression, stability, progression — a structured follow-up plan is essential. In many series, close imaging during the first 6–12 months (CT or MRI) is recommended, followed by periodic surveillance if stable. (7)2/17/2026 12:12:00 PM

Hemodynamic management also plays a central role: strict blood pressure control, heart rate control (e.g. with beta-blockers), analgesia and monitoring are key measures to reduce stress on the aortic wall and minimize risk of expansion, progression, or rupture. (4)

Our patient is noteworthy in several respects:

- He lacks the common cardiovascular risk factors typically associated with aortic syndromes (e.g., chronic hypertension, atherosclerosis). This underlines that IAH can occur even in “non-classic” patients, and that clinicians must remain vigilant in the face of atypical presentations.
- The use of PET imaging to exclude aortitis demonstrates a thorough, multidisciplinary diagnostic approach. Given the overlap in imaging appearance between inflammatory aortitis and IAH, such a strategy strengthens diagnostic confidence.
- Nevertheless, even with a negative PET and initial stability, the unpredictable natural history of IAH necessitates long-term follow-up. The risk of late complications—including aneurysm formation, dissection, ulcer-like lesions, or rupture—always persists, and the absence of early complications does not preclude future risk.

Therefore, this case supports the concept of individualized management, combining clinical, morphological and functional data, and close surveillance rather than a one-size-fits-all approach.

# Conclusion

IAH must be considered among differential diagnoses in patients with acute chest, back, thoracic or lumbar pain, even in the absence of typical risk factors.Accurate diagnosis requires high-quality imaging; when differentiation from aortitis is needed, combining anatomical imaging (CT/MRI) with functional imaging (PET) may be very useful.Therapeutic strategy should be individualized, balancing the risks and benefits of conservative versus invasive approaches depending on anatomical features, clinical stability, age, comorbidities and risk of progression.In stable patients selected for conservative management, strict blood pressure control and a rigorous follow-up schedule with periodic imaging are mandatory, especially during the first year.

The reporting of such atypical cases provides valuable contributions to the existing literature, facilitates the ongoing refinement of diagnostic and therapeutic frameworks, and

advances a more individualized and nuanced approach to the management of intramural aortic hematoma.

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