

CASE REPORT

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Persistent low back pain reveals a massive non-ruptured aortic abdominal aneurysm: A case report

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ABSTRACT

A 62-year-old man presented with an insidious low back pain that radiated to both knees. A pulsatile abdominal mass was detected on physical examination. Imaging study identified a pararenal aortic abdominal aneurysm measuring 20 cm long and with a caliber of 10 cm. The aneurysm was surgically repaired. The patient survived the procedure and has shown a positive recovery.

Keywords: Aortic aneurysm, Endovascular repair, Low back pain

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INTRODUCTION

Low back pain (LBP) is the fifth cause of medical consultation in the United States [1]. Despite its relation to spinal degenerative injuries, there are other less common but serious conditions that mimic this clinical presentation, namely vascular disorders such as aortic abdominal aneurysm (AAA). It is usually defined as abdominal aorta diameter greater than 3.0 cm and presents with low back pain in up to 50% of patients [2].

In our daily practice, it is important not only to recognize AAA-associated risk factors but also to thoroughly screen for non-musculoskeletal signs and symptoms to improve diagnostic accuracy and patients' outcome.

CASE REPORT

A 62-year-old retired man came to the emergency department complaining of steady low back pain that had been radiating through both legs to the knee for three weeks. Pain was rated 7 out of 10 on a visual analogic pain scale. He denied fever, muscular fatigue, intermittent claudication and lower-limb paresthesia. He didn't have genitourinary or gastrointestinal symptoms. He reported no known trauma that might have caused back injury. During this period, he had been seen by his general practitioner who prescribed him metamizole 575 mg bid but the pain persisted. The patient had previous history of smoking, hypertension and ischemic heart disease with two previous myocardial infarctions (1 and 7 years ago). On physical examination, he showed normal vital signs, apyrexia and normal cardiopulmonary auscultation. On neurological examination, no motor or sensitive dysfunction was observed and Lasègue's sign was absent on both lower limbs. Abdominal examination revealed a strong pulsatile mass on light palpation of the periumbilical region without local inflammatory signs or evidence of peritoneal irritation.

Blood tests showed an elevation of serum urea (92 mg/dl) and creatinine (1.87 mg/dl), but no leukocytosis nor C reactive protein elevation. Abdominal ultrasound

was conducted and detected an AAA measuring 9.6 cm of diameter, with an intramural thrombus about 4.4 cm thick. Liver, biliary tract and pancreas exploration was unremarkable. Renal ultrasound was performed, considering the evidence of renal impairment, and showed a reduced size of the right kidney associated with low corticomedullary differentiation. Given the diagnosis of symptomatic AAA, the patient was submitted to a CT angiography scan which showed a pararenal AAA with a maximum diameter of 10 cm and 20 cm long, presenting a circumferential intramural thrombus about 5.7 cm thick causing right renal artery stenosis. There was good vascular permeability of celiac trunk, superior mesenteric artery and left renal artery (Figure 1).

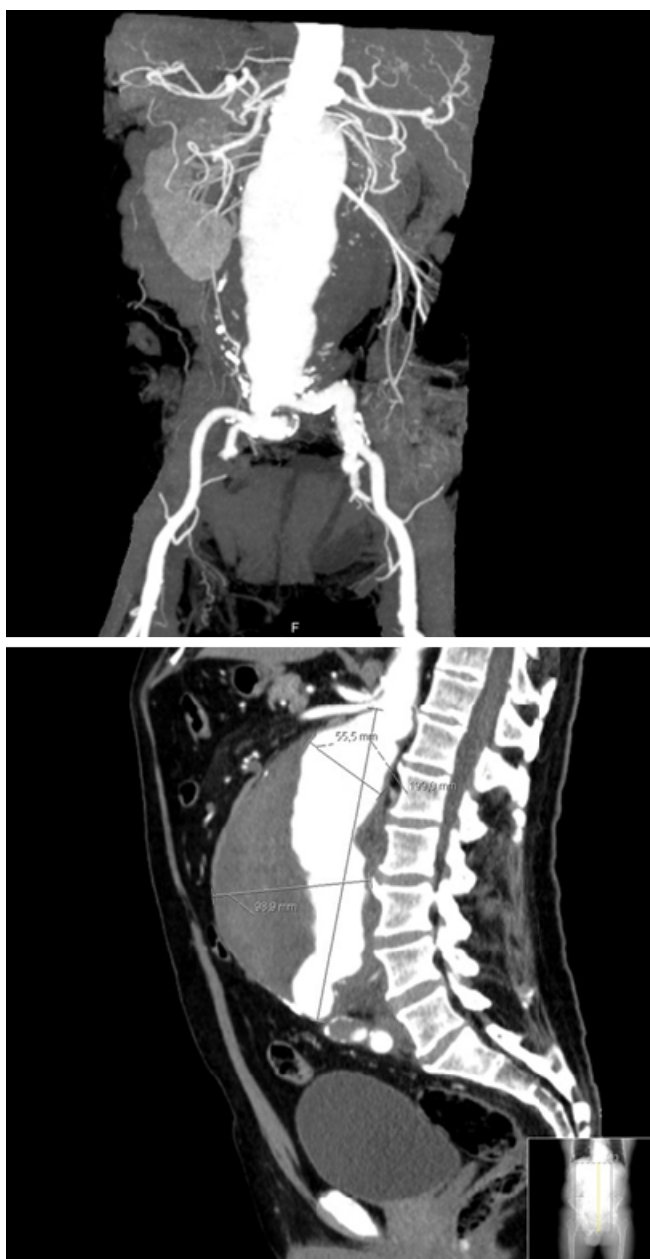


Figure 1: CT imaging showing a pararenal aortic abdominal aneurysm with a maximum diameter of 10 cm and 20 cm long, presenting a circumferential intramural thrombus about 5.7 cm thick and causing right renal artery stenosis.

After completing imagiological study, the patient was seen by the vascular team, who decided to perform endovascular repair of the aneurysm, a decision that was based on patient's choice and justified by the anatomic features of the AAA. The patient survived the surgery and was discharged after three weeks. He presented no post-operative vascular complications. During this period, he underwent lifestyle modifications and control of his cardiovascular risk factors, such as hypertension. After one year of follow-up, the patient had no more episodes of low back pain.

DISCUSSION

In the United States, population-based studies in adults older than 50 years have found that the prevalence of AAA is 3.9% to 7.2% in men and 1.0% to 1.3% in women, with an overall incidence of 7 per 1000 in those in their mid-60s. The US Preventive Services Task Force (USPSTF) published updated guidelines on the matter in 2014, concluding that there is moderate net benefit of one-time ultrasound screening for AAA in men 65 to 75 years of age with a history of smoking [3].

The Society of Vascular Surgery also recommends a one-time screening for AAA, but extends it to all men older than 65 years or as early as 55 years in men and women with a family history of AAA. Portugal estimated prevalence of AAA stands between 2–3% in screening pilot studies [4]. There is still an active debate regarding a viability of a Portuguese National AAA Screening Program [5].

An aortic dilation is considered aneurysmal if there is an increase in diameter over 50% than expected for the same aortic segment in unaffected individuals of the same age and sex. AAAs are usually categorized in terms of etiology, morphology, size and location. This vascular disorder occurs in 3 to 9% of men aged >50 years and up to 28% of first-degree relatives of patients with AAAs may be similarly affected [6].

The infrarenal aorta is involved in > 80% of cases, but up to 10% of AAAs could also affect the pararenal or visceral aorta. The pathogenesis of AAAs is based on a multifactorial environment, based on the interaction between atherosclerotic disease and genetic, hemodynamic or even immunologic factors. Smoking is the only modifiable factor associated with AAA expansion, particularly if there is a minimum consumption of more than 100 cigarettes in a lifetime [7].

Other AAA-related risk factors include age (most patients are 65 years or older), sex (5 to 10 times more common in men than in women) and previous history of stroke, coronary arterial disease (with/without previous history of myocardial infarction), hypertension and hyperlipidaemia [8, 9].

When it comes to clinical manifestations, AAAs are often asymptomatic and diagnosed as an incidentaloma [10]. AAAs represent an infrequent cause of low back

pain which may worsen over time in case of aneurysm expansion or impending rupture [11, 12].

The natural history of AAA is gradual expansion over a period of years and eventual rupture. The rate of expansion of AAA is not always linear and consistent; some patients may have stable AAA size for many years, followed by a sudden increase within a short period [13].

The pain related to a ruptured AAA is classically of sudden onset accompanied by hypotension. The mural thrombi associated with AAA may lead to distal arterial thromboembolism, eventually producing acute lower limb ischemia (the first sign in 2–5% of patients) and livedo reticularis [13].

An enlarging aneurysm may produce local mass effects due to compression of adjacent structures [13]. About 30% of asymptomatic AAAs are found on routine consultation. Curiously, there are some reports of AAAs diagnosed on chiropractic patients [14, 15]. During abdominal palpation, an AAA is revealed as a pulsatile, expansive mass at or above the umbilicus. The presence of a bruit may indicate aortic or visceral arterial atherosclerotic disease, or rarely an aortocaval fistula (machinery murmur). The accuracy of physical examination often depends on aneurysm size and body mass index [16]. Despite such limitations, some authors still recommend abdominal palpation and auscultation when there is suspicion of a non-mechanical cause for low back pain or history of back pain non-responsive to anti-inflammatory agents [17].

Acute AAA rupture is one of the most dramatic emergencies in medicine. Only approximately 50% of patients with ruptured AAA reach the hospital alive; of those who reach the hospital, up to 50% do not survive [18]. In general, endovascular AAA repair is reserved for asymptomatic aneurysms at least 5.0 to 5.5 cm in diameter [18, 19]. The selection of patients should not only contemplate the estimate risk of rupture (based on the size of the aneurysm, wall thickness, intraluminal thrombus thickness, and peak wall stress) but also other parameters such as life expectancy and operative morbidity and mortality, which include coronary artery disease (leading cause of early and late mortality after AAA repair), chronic kidney disease, chronic obstructive pulmonary disease and diabetes mellitus [20].

This complexity of this vascular disorder is well demonstrated in this case report and justifies its clinical interest. In fact, CT scan imaging detected a massive pararenal AAA (which is an infrequent location, present in up to 10% of cases) with no signs of rupture in hemodynamically stable patient with a history of an insidious low back pain and without thromboembolic complications.

The patient's AAA described in this case report fits the highest cardiovascular profile due to the previous history of smoking and ischemic heart disease and it stands out for being a rare documented example of a late diagnosed AAA with a high risk of rupture submitted to successful emergency surgical treatment. This case report also

highlights the importance of clinicians' awareness on the differential diagnosis of low back pain.

CONCLUSION

Aortic abdominal aneurysm represents a silent and fatal clinical condition affecting older adults with history of smoking and cardiovascular disease. It should be actively considered in the group of differential diagnosis in every male patient older than 50 years with low back pain. In case of suspicion, patient should be referred for advanced imaging investigation.

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Author Contributions

Jorge Vaz Lourenço – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Ricardo Marques – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Daniela Marado – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Adriano Rodrigues – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

None.

Consent Statement

Written informed consent was obtained from the patient for publication of this case report.

Conflict of Interest

Authors declare no conflict of interest.

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