CASE REPORT

The accuracy of combined physical examination and ultrasonography for the detection of abdominal aorta aneurysm

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Abstract
Atherosclerosis is the most frequent cause in the appearance of an abdominal aorta aneurysm (AAA) and plays an important role in its development. Most AAA does not cause any symptoms, especially when talking about elderly patients, however, many of those aneurysms can be detected during physical examination. Their detection is very important because the natural evolution and the major reason in treating AAA is their tendency to rupture. We present the case of an adult man with a complex clinical pathology, but not related to the AAA. The diagnosis of the AAA has been suspicion through palpation, and the abdominal ultrasound exam confirmed it. This case is particularly interesting, as the AAA requires surgical intervention, while patient’s health status was poor. An essential issue is establishing the importance of the AAA screening, when there are no symptoms present. For now, there are not satisfactory studies to be used as a guide.

Keywords: abdominal aorta aneurysm, clinical examination, echography, treatment.

Introduction
The incidence of abdominal aorta aneurysm (AAA) has been remarkably increasing for the past decades and that seems not to be related only with the diagnostic tool’s improvements and the increasing life span. The scientists now conducting medical researches are very interested in identifying the factors responsible for the real increase in the incidence of AAA. Although atherosclerosis plays for sure an important role in AAA’s development, recent researches sustain the assumption that AAA might be mostly genetic, yet no genetic marker can be firmly associated with it [1, 2].

Other experimental evidences underline the cellular, inflammatory and hemodynamic mechanisms’ role in AAA’s growth [3, 4].

The presence of an abdominal aorta aneurysm can be a marker for multiple aortic aneurysms. This is the reason why Alcorn HG and Wolfson SK Jr recommend that, when finding an AAA, we should examine the entire aorta in order to see if there are others, too [5].

Most AAAs does not cause any symptoms, especially when talking about elderly patients, however, many of those aneurysms can be detected during physical examination. The always-improving medical field gives us a wide variety of imaging technique to find and measure them, observe their evolution and precisely define the aorta before surgery.

The major reason in treating AAA is their tendency to rupture. The most reliable predictor for the aneurysm’s growing rate is its initial size. Bigger aneurysms enlarge faster than the smaller ones, because of La Place’s law [5]. Moreover, faster growing is likely to predict a rupture, especially when the diameter of the aneurysm is 5 cm or greater [4, 5]. Right now, there is no clear statement about the correlation between the aneurysm’s minimum size and the surgery indication.

While all surgeons agree upon operating the aneurysms whose diameter is bigger than 6 cm, and most of them perform surgery on those bigger than 5 cm if they are associated with reasonable risks, there are not many who would treat a silent one with a less than 4 cm diameter. We expect the conclusions of multi-centric prospective trials now conducted, in order to identify the best moment for surgery, based upon their size [6]. Anyway, an AAA with a diameter bigger than 4 cm needs CT evaluation every three months to see if it is rapidly increasing in size (more than 0.5 cm/year) or if it is reaching a 5 cm diameter, both situations requiring surgical treatment [6].

As for the medical treatment, β-blocking therapy has the best effects in reducing both the expansion and rupture risks. A recent study on mice demonstrated that β-blocking effectiveness is not related to blood pressure and dp/dt decreasing, being rather due to the changes made in the aortic wall [4].
A human study recently reported that the average growing rate was slower in the patients who received β-blocking therapy compared with the patients who did not, this effect being more obviously when was treated a big aneurysms [9].

Patient, Methods and Results

A 64-year-old patient, retired, former worker in a medical institution, with a recently right nephrectomy was admitted to the “Filantropia” Municipal Hospital via Emergency Department with hypertensive crisis.

Medical history

The affection began 4–5 years ago, as the patient describes left calf pain then appearing, when rapidly walking for long distances. The symptoms were neglected. In time, the disease evolved slowly, the claudication being avoided by limiting the exercise.

In the last year, when walking distance was dramatically shorten (about 100 m until the claudication appeared) and the calf pain was annoying. Left thigh pain and numbness also appeared while walking. After a physical examination, a peripheral vasodilator and neurotrophic treatment was initiated with little improvements in increasing the walking distance without pain. We did also mention a recent right nephrectomy (three months before coming to our clinic) for pyonephrosis. At that moment, the patient was advised to consider lower limb revascularization procedures. The clinical evolution after nephrectomy was favorable, but usual blood tests at one month after revealed anemia. His family physician considered these findings related to the recently performed surgery. Therefore, the patient was treated with oral iron for another one month.

The following risk factors were identified: prolonged cigarette smoking (about 20–30 cigarettes/day for 40 years), dyslipidemia, age and sex. There was no previous family history of AAA.

The patient was referred to us by his family physician who found his blood pressure high (200/120 mmHg), though he did not have any symptoms and went to him for a periodical exam. At that time he was treated with a peripheral vasodilator drug – Pentoxyfilinum retard – 800 mg/day.

Clinical examination

Physical examination at the time of admission revealed: obesity (BMI = 28.9 kg/m²), android fat distribution, mild pallid skin, a post-nephrectomy scar in his left lumbar region, hair loss on toes and both calves and reduced skin temperature, especially in the left calf.

Cardiovascular examination: pulse 104/min., BP 200/110 mmHg (same for both upper limbs). Femoral and distal pulses present on the right side, but absent on the left, the color return time was prolonged (20 seconds), no superficial sensory impairments. There was a large pulsating mass, mild tender to palpation, just above the umbilicus.

The rest of the physical examination was normal.

Laboratory investigation

Laboratory findings: Hb 9.4 g/100 mL, Ht 30.7% with anisocytosis and hypochromia, normal renal function: serum creatinine 0.88 mg/100 mL, creatinine clearance 110 ml/min., serum K + 4.1 mg/100 mL, fasting plasma glucose 90 mg/100 mL.

Lipid profile revealed mixed dyslipidemia: hypertriglyceridemia 198 mg/dL, elevation of plasma LDL-cholesterol 196 mg/dL, total serum cholesterol 240 mg/dL, low plasma HDL-cholesterol 38 mg/dL.

C-reactive protein was present, but other acute phase reactants, such as fibrinogen and ESR, were normal. Serum renin was not tested. The rest of the laboratory examination was normal.

Twelve lead EKG at the admission showed: sinus tachycardia (ventricular rate = 104/min.), incomplete right bundle branch block.

Transthoracic echocardiography (useful in evaluating the effects of an old HBP on the heart) showed: normal sized heart chambers, concentrically left hypertrophy (interventricular septum = 13 mm, ppVS = 12 mm), normal ventricular systolic function (ventricular ejection fraction of 60%), left ventricular diastolic dysfunction – the delayed relaxation pattern, as seen in Doppler transmitral flow profile (E/A<1, TDE = 140 ms).

Abdominal echography revealed left kidney enlargement (long axis = 14.3 cm, cortical thickness = 2.2–2.8 cm) considered to be compensatory hypertrophy after nephrectomy, with no other abnormalities. The ultrasound also confirmed the clinical suspicion of AAA, revealing abdominal aorta’s enlargement with a diameter of 60–65 mm, without recalibration and with hyperechoic, asymmetrically thick walls (thickness 2.6 mm, lumen 9 mm). Moreover, we suspicion the presence of a thrombus in the aneurysmal lumen, as we so a hypoechoic mass, with right-posterior eccentric blood flow of 20 mm. The relationship with the renal and mesenteric arteries cannot be seen precisely, but it seems that the aneurysm includes them.

EchoDoppler revealed the external iliac artery and the left common femoral artery with diastolic forward flow that leading to the suspicion of left common iliac artery stenosis.

Left common iliac artery’s pulsation index was low (1.95), while right common iliac artery’s pulsation index was almost normal (3.85).

Abdominal CT confirmed the partly thrombosed aneurysm, his maximum diameter being 7.4/6.8 cm. The aneurysm had an eccentric lumen and was located between the left renal artery and the aortic bifurcation. It is worthy to note that there were atheroma plaques located at the origins of celiac trunk, superior mesenteric artery and left renal artery. CT also revealed a large hiatal hernia.

Thoracic CT revealed a minimum enlargement of the thoracic descending aorta, with a thin parietal thrombus (maximum thickness = 9 mm) located on the left side.
Diagnosis

Based on clinical, laboratory and imagistic findings, following diagnosis were formulated:

- Hypertension stage 3, with very high cardiovascular risk (ESC/ESH 2003);
- Infrarenal abdominal aorta aneurysm partly thrombosed;
- Aneurismal enlargement of the descending thoracic aorta;
- Peripheral arterial disease of the right lower limb – stage IIIB La Fontaine;
- Left common iliac artery stenosis;
- Ischemic heart disease;
- Incomplete right bundle branch block;
- Mixed dyslipidemia;
- Obesity;
- Mild anemia;
- Large hiatal hernia;
- Solitary left kidney, after nephrectomy for pyonephrosis.

We did not consider a priority high blood pressure’s etiological investigation, so we temporized it. We also did not extended our investigation in order to find out the anemia’s exact cause, though the most likely cause seems to be the chronic iron loss due to the large hiatal hernia.

Treatment

After evaluating both clinical and imagistic data, the indication was for surgery and the patient was transferred to a Cardiovascular Surgery Clinic.

Considering the rupture risk due to high blood pressure, until the transfer was made, we opted for a conservative treatment in order to decrease the blood pressure to normal high values, for the tissue pressure of the chronic ischemic lower limbs to be maintained. We also advised the patient to start a treatment for the risk factors’ long-term control, thus implying systemic atherosclerosis control, too [7, 8].

On admission, we gave him an ACE inhibitor – Enalapril – 1.5 mg/ml, 1 phial in slow intravenous injection, a ββ1-blocker – Carvedilol 6.25 mg/tb p.o. The next three days he was treated with Perindopril 8 mg/tb, 1 tb/day in the morning, Carvedilol 6.25 mg/tb, 2 tb/day (one every 12 hrs.), Atorvastatin 20 mg/tb, 1 tb in the evening, Aspirin 75 mg/tb, 1 tb/day together with an anti-ulcer drug – Omeprazol 20 mg/tb, 1 tb/day, Pentoxifilin retard 400 mg/tb, 2 tb/day, oral iron – one teaspoon three times a day. Serum creatinine measurements during this treatment revealed minimum increasing – from 0.88 mg/dL to 0.94 mg/dL. With these drugs, blood pressure decreased to a value of 130/85 mmHg, but the claudication remained [9].

Lifestyle changes are imperative, the patient being advised to change his diet – to reduce his daily sodium intakes, his total and saturated fat intakes, to reduce to minimum alcohol consumption and to increase fruit and vegetable consumption. He was advised to change his lifestyle – to lose excess weight, to avoid sedentary life, to regularly exercise, to remain non-smoker [9].

Aortography before surgery revealed no extension of the aneurysm above the renal arteries origin.

Coronarography revealed an atheroma plaque at LAD’s origin, considered to be insignificant (30% stenosis).

The AAA was first resected, then the aorta was ligated and sutured at the common iliac arteries’ origin. In the same session, lower limbs’ revascularization was performed using an aorto-biiliac by-pass with a B Braun 18/9 stent implanted to replace the enlarged artery, too, and the aorta was wrapped around the graft.

The evolution after surgery was favorable and the lower limbs’ circulation was reestablished.

Discussion

AAA could have been suspicioned through palpation, thus requiring an abdominal ultrasound in order for this suspicion to be confirmed [10, 11].

Because most AAAs are “silent”, not diagnosing them leads to:

- Severe aneurismal complications such as: rupture, acute aneurismal thrombosis, lower limbs embolism with signs of acute peripheral ischemia, compression caused complications (e.g. intestinal occlusion due to duodenum’s compression) and, also, infectious complications may occur if the AAA is ignored;
- Not diagnosing the atherosclerotic lesions simultaneously present, with all the consequences deriving from it.

Another significant aspect concerns the ecography’s importance in diagnosing AAA. In this particular case, ultrasonography revealed AAA’s presence, location and size; it also revealed AAA thrombosis and his relationship with visceral and renal arteries. Actually [12–14], Ct (a much more expensive exploration and a less available one, not mentioning that it uses ionization radiance and intravenous contrast) did not bring any new information regarding the AAA, but it revealed the descending thoracic aorta’s aneurismal enlargement and the atheroma plaques inside visceral and renal arteries. EchoDoppler revealed abnormal blood flow in the left common iliac artery and the arteriography performed before surgery confirmed left common iliac artery stenosis.

Although non-invasive diagnosis was identical with the invasive one, we consider the invasive exploration important to be done before surgery, as they reveal possible coronary arteries damage.

This case is particularly interesting, as the AAA requires surgical intervention, while patient’s health status was poor. On short-term we managed to control the symptoms and the patient was transferred to long-term treatment in a Cardiovascular Surgery Clinic [11].

The first problem concerned the surgery risk, thus coronarography was needed. This exploration itself may involve some risk as we are talking about a patient with AAA and descending thoracic aorta aneurismal enlargement (embolism or dissection risk), no matter the approach (femoral or brachial). In addition, the bleeding due to the puncture may increase the already present anemia.
Another therapy problem concerned \(\beta\)-blocking treatment. This is used in order to lower AAA’s expansion and rupture risks during blood pressure’s elevation, but it might worsen lower limbs’ ischemia as it decreases their tissue pressure. Carvedilol, as any \(\alpha_1\)-blocker, has a vasodilator action and minimizes the relative contraindications for \(\beta\)-blockers imposed by the peripheral vascular disease.

The antiplatelet medication is essential, considering the peripheral artery disease and the partly thrombosed AAA. Clopidogrel seems to be more efficient than aspirin in preventing acute thrombosis in patients with symptomatic peripheral disease, as a recent, highly significant study revealed [15, 16]. Clopidogrel’s high price made us choose aspirin, together with an anti-ulcer drug, after thoughtfully considerations regarding the advantages and the disadvantages of such a treatment, knowing that the patient had a large hiatal hernia, with no endoscopic exploration. To correct his dyslipidemia, we choose Atorvastatin as it confers endothelium protection and reduces atheroma plaques’ volume [14].

\section{Conclusions}

Because of the real increasing in AAA’s incidence, it is very important to associate this disorder with coronary atherosclerosis. Another important issue is establishing how important screening for AAA is, when there are no symptoms present. For now, there are not satisfactory studies to be used as a guide in this matter. A recent study reveals that clinical screening in men over 60-year-old is cost-efficient, but with little benefit, while ultrasound screening in men in the same age group has cost efficiency close to the margin of acceptability, with reduced benefits. Instead, most authors recommend ultrasound screening in high-risk patients, especially those with positive family history of AAA.

\section{References}


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