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Case Report

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Renovascular hypertension in the young, an indirect cause for upper gastrointestinal bleeding - a case report

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Abstract

Renal hypertension is one of the most frequent grounds for secondary hypertension, accounting for 5% of all hypertensive patients. The aetiology lies mainly in renal artery stenosis, with fibromuscular dysplasia and atherosclerosis as the leading causes. In the case of renal hypertension, fibromuscular dysplasia accounts for most of the cases in young women, while atherosclerosis is more frequent among middle-aged men. Discovering the real cause of secondary hypertension can sometimes be a challenge. A 35-year old man, with metabolic syndrome arrives at the emergency department with acute upper intestinal tract bleed and severe anaemia, due to non-steroidal inflammatory drug abuse for persistent headache. The cause of the headache is revealed when the blood pressure is measured and the systolic value amounts to 230 mmHg. Further investigation establishes the diagnosis of metabolic syndrome and target organ damage in the form of microalbuminuria and left ventricular hypertrophy. The second line of diagnostic procedures is directed toward the causes of secondary hypertension. The computerized tomographic angiography reveals a severe single proximal stenosis of the right renal artery, which suggests an atherosclerotic aetiology. The patient remains on medical therapy with controlled blood pressure values. The resolution of the case is ongoing, as there is no immediate indication for interventional therapy. This case not only presents a very rare cause of renal hypertension in the young, due to atherosclerotic renal artery stenosis, but also a very uncommon first symptom in the form of upper gastrointestinal bleed.

Keywords: secondary hypertension, renal artery stenosis, atherosclerosis

Introduction

Most cases of hypertension do not have a precise etiology and are thus defined as essential hypertension. For the rest (~ 10% of the cases) the diagnosis of secondary hypertension arises [1].

Secondary hypertension is considered as a possible diagnosis before the age of 30 and after the age of 50. The definable causes for hypertension are mainly endocrinologic, vascular and renal. Discovering the real cause of secondary hypertension can sometimes be a challenge. Renal hypertension is one of the most frequent grounds for secondary hypertension and accounts for up to 5% of all hypertensive patients. The aetiology lies mainly in renal artery stenosis, with fibromuscular dysplasia and atherosclerosis as the leading causes [2]. In the case of renal hypertension, fibromuscular dysplasia accounts for

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most of the cases in young women, while atherosclerosis is more frequent among middle-aged men.

Case report

A 35 years old patient is hospitalized three weeks prior to the referral in our clinic with upper gastrointestinal bleeding and severe secondary anaemia. This condition was considered to be due to excessive consumption of non-steroidal anti-inflammatory drugs (NSAIDs) for persistent headache. The reason the patient was sent to our clinic was the detection of a very high blood pressure value of 230/100 mmHg. The clinical examination, besides increased blood pressure, doesn't hold unusual findings. The patient is overweight with absent heart or vascular murmurs; also, papilledema was not observed at the ophthalmological examination.

The blood panel shows mild anaemia, normal kidney function with a normal creatinine clearance, normal urinary sediment and summary except for microalbuminuria. The clinical and laboratory findings reveal a patient with metabolic syndrome - hypertensive, overweight, dyslipidaemic (cholesterol level of 263 mg/dl, triglycerides level of 430 mg/dl) and with a carbohydrate metabolism disorder (postprandial blood glucose level reaches 208 mg/dl).

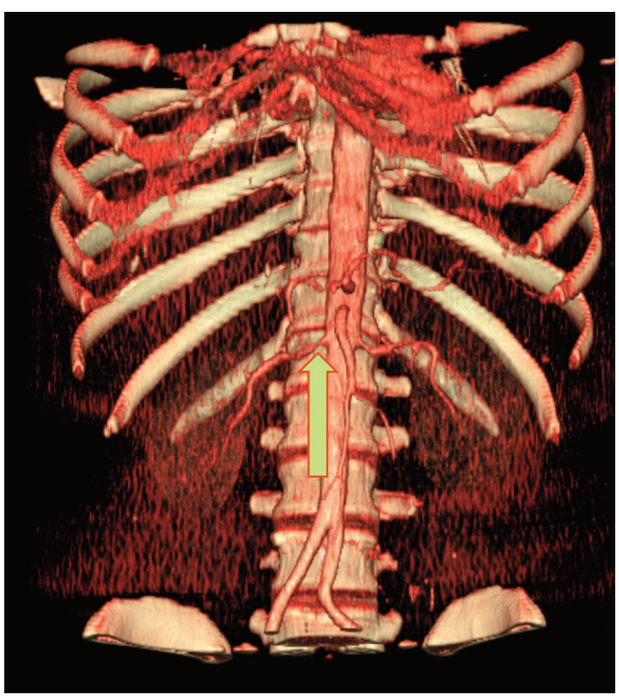

Figure 1. Computerized tomographic angiography. The green arrow indicates an 85% stenosis in the initial segment of the right renal artery.

The ECG tracing indicates the presence of left ventricular hypertrophy, with a Socolow Lion index of 45 mm and a Cornell index of 30 mm. Echocardiography confirms the presence of concentric left ventricular hypertrophy (both ventricular septum and posterior wall of the left ventricle measure 15 mm), left atrial dilation with mild mitral regurgitation and delayed relaxation type of diastolic dysfunction.

Since this is a young 35-year old patient with severe hypertension, we focused on the causes of secondary hypertension. We started our differential diagnosis with the endocrine causes, quickly eliminating pheochromocytoma, as the normetanephrines (40.1pg/ml, N <180pg/ml) and metanephrines (35.9pg/ml, N <90pg/ml) levels were normal. Plasma cortisol level was normal (2.67ug/dl, N= 2.5-12.5 ug/dl) therefore excluding Cushing's syndrome. Conn's syndrome was also out of the question because of the hyperreninaemia (176 ug/ml) and normal blood electrolytes. The exclusion of these three causes of hypertension was facilitated by the normal CT appearance of the adrenal glands. The existence of a thyroid pathology was not confirmed, based on normal hormonal values (TSH= 1.69uUI/ml, N= 0.4-4 uUI/ml).

Moving forward, renal parenchymal hypertension was quickly removed from the diagnosis list, since the abdominal ultrasound did not attract any attention, as the kidneys had the same size, with normal echostructure. Kidney function is normal, summary and sediment are within normal limits, except for the previously mentioned microalbuminuria.

Following our investigations there was an unresolved issue, namely the elevated renin level. At this point we debated the potential causes of hyperreninaemia in this case. Addison's disease does not fit the clinical picture; neither does the patient have any signs of cirrhosis. A discussion regarding the haemorrhage as a source for the hyperreninaemia would have been valid if the renin determination was done close to the bleeding event, which may have triggered the secretion of renin. However, the blood sample was taken four weeks after the upper gastrointestinal bleeding. Malignant hypertension does not appear to be the cause for hyperreninaemia, as the patient has no papilledema or other signs of acute target organ damage. Essential hypertension could come into question due to the metabolic context of the patient and because the patient is past the age of 30, which is slightly beyond the cut-off age for developing secondary hypertension. Renin-secreting tumours are a questionable diagnosis, the CT examination revealed no abdominal masses, but the possibility remains in infracentimetric tumours.

The last differential diagnosis, and one that is much easier to investigate, is renovascular hypertension. Doppler ul-

trasound of the renal arteries showed, to our surprise, increased velocities on both sides (1.9 m/sec for the right renal artery and 2.29 m/sec for the left one) [3]. However, they didn't meet the criteria for significant stenosis (>60% narrowing of the circulating lumen). Velocities corresponding to these stenoses are disputed, but a stenosis of 60% would correspond to a velocity higher than 2.5 m/sec. We turned to a more sensitive investigation, namely computerized tomographic angiography that detected an 80 % stenosis within the first centimetre of the right renal artery through a circumferential noncalcified plaque (Figure 1). There were no other stenoses on distal or contralateral segments. The adrenal glands had a normal aspect and dimension.

Our final diagnosis in this case was: right renal artery stenosis, secondary hypertension complicated with hypertensive heart disease, mixed dyslipidaemia, recent upper gastrointestinal bleeding and mild secondary anaemia.

We opted initially for classical antihypertensive drug therapy. At the last follow-up the patient still had slightly increased blood pressure values and the medication doses were augmented.

Discussion

The diagnostic problem that occurs in this circumstance is whether this is really a case of secondary renovascular hypertension. Is the stenosis truly significant from a hemodynamic point of view? The Doppler exam states the contrary, although showing bilaterally increased velocities. Still, the CT scan, which is considered more sensitive, indicates a unilateral right renal artery stenosis of 85%. Another reason to believe that if the renal stenosis is significant, then it is only unilateral, is the fact that ACEI treatment did not affect the renal function.

The velocities obtained through the Doppler examination can be disputed because the method is rather subjected to an underestimation of speed. Arguments against significant renal artery stenosis are the equal kidney size and borderline velocities.

Or is it just hypertension in the context of metabolic syndrome plus NSAID consumption? The problem in this instance is that the patient is no longer taking NSAIDs, because the headaches subsided after the normalization of blood pressure values.

The possible association between renal artery stenosis (not as severe as to cause renal atrophy) and arterial stiffness in the context of metabolic syndrome solves the problem that the patient doesn't have right kidney atrophy.

The next diagnostic issue is related to the type of renal artery stenosis, whether it is atherosclerotic or fibromuscular dysplasia. The age advocates for the aetiology of fibromuscular dysplasia, but the CT aspect pleads for atherosclerosis with a unique almost ostial stenosis. Fibromuscular dysplasia usually occurs in the distal two thirds of the artery and takes the appearance of multiple stenoses, unless the patient has the very rare type of medial hyperplasia (1-2% of cases). Moreover, the atherosclerotic stenosis is more plausible in the metabolic context with severe hypertriglyceridemia, hypercholesterolemia and obesity.

Another problem is the correct course of treatment for a young patient with atherosclerotic renal artery stenosis. The clinical studies [4] regarding the atherosclerotic stenoses are conducted on older patients, since the medical literature is relatively low in cases of atherosclerotic renal artery stenosis in the young. However, these studies have not necessarily demonstrated a net benefit of angioplasty over drug therapy. It seems that the benefit is greater on fibromuscular dysplasia, with a much lower restenosis rate than in atherosclerosis. Theoretically, angioplasty should be used in case of significant stenosis where the blood pressure is not adequately controlled with drug therapy or where the patient exhibits progressive renal disease, unexplained heart failure, pulmonary oedema, acute malignant hypertension. Our patient does not fit any of these scenarios, so probably conservative treatment is the best option for the moment.

As for the prognosis of the patient, considering that the patient already has right renal artery stenosis on CT, but the Doppler exam describes high velocities for both arteries, it is likely that he will progress towards bilateral stenosis. He already exhibits target organ damage in the form of microalbuminuria and left ventricular hypertrophy. It is highly probable that there are other still undetected systemic atherosclerotic lesions, thus the patient fits into a very high cardiovascular risk group. Only rigorous drug therapy and lifestyle changes may slow the progression of atherosclerosis.

Conclusion

This case is particular through its presenting complaint, with upper gastrointestinal bleeding, due to an excessive consumption of NSAIDs for persistent headache, probably due to the undetected high blood pressure values. The second important feature is the atherosclerotic aetiology of the renal artery stenosis at a young age. Also, the initial Doppler examination indicates bilateral not significant stenosis, yet the CT exam validates only

the right renal stenosis. This conclusion is reinforced by the lack of serum creatinine increase with ACEI administration. The prevalence of LVH in patients with renal artery stenosis is 79% versus 46% in patients with essential hypertension [5]. That may explain why there are no pathological findings on the ophthalmologic examination, coexisting with such a significant left ventricular hypertrophy. The lesson learned from this case is that statistics are just statistics, and even if a certain aetiology might seem improbable, it is not a reason to easily dismiss it.

Conflict of interest statement

The authors of this paper certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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