Renal Artery Stenosis (RAS) Case study

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ABSTRACT

Renal Artery Stenosis (RAS), is one of the causes of secondary hypertension; there are many causes of renal artery stenosis, as atherosclerosis of the renal artery which account for 90% of cases of RAS; fibromascular dysplasia accounts for 10% of RAS. Various causes of thrombophilia either due congenital causes or acquired causes and can lead to RAS. Our patient was presented by acute attack of epistaxis and hypertension. Angiography of the Renal Arteries, are showed no sign of renal artery stenosis. However, the right kidney showed upper pole infarction, and the left kidney showed evidence of functional lower pole renal artery stenosis, although there is no anatomical stenosis detected in angiography. Work up for the cause of thrombophilia did not help in the diagnosis, which may be due to an undiscovered cause of thrombophilia.

Keywords, secondary hypertension, fibromascular dysplasia, atherosclerosis.

INTRODUCTION

Renal artery stenosis (RAS) is the major cause of renovascular hypertension, it is increasingly recognized as an important cause of chronic renal insufficiency and end-stage renal disease. In older individuals, atherosclerosis (ATH) is by far the most common etiology of renal artery stenosis. Renal-artery stenosis may occur alone (isolated anatomical renal-artery stenosis) or in association with hypertension, renal insufficiency (ischemic nephropathy), or both. As the renal artery lumen progressively narrows, renal blood flow decreases and eventually compromises renal function and structure. In the elderly, the possibility of an increase in the prevalence of renal artery stenosis and ischemic nephropathy, clinicians dealing with renovascular disease...
(RVD) need noninvasive diagnostic tools and effective therapeutic measures to resolve the problem successfully. The risk of cardiovascular events in adults depends more on the degree of hypertension than on its cause. A decrease in renal perfusion pressure activates the renin–angiotensin system, which leads to the release of renin and the production of angiotensin II; this has direct effects on sodium excretion, sympathetic nerve activity, intrarenal prostaglandin concentrations, and nitric oxide production; and causes renovascular hypertension, (1).

Atherosclerotic RAS accounts for 90 percent of cases of renal-artery stenosis and usually involves the ostium and proximal third of the main renal artery and the perirenal aorta. In the lesion site, endothelium permeability to plasma macromolecules (e.g., low-density lipoprotein [LDL]) increases, turnover of endothelial cells and smooth muscle cells increases, and intimal macrophages increase. When atherogenic lipoproteins exceed certain critical levels, the mechanical forces may enhance lipoprotein insudation in these regions, leading to early atheromatous lesions. Atherosclerotic RAS is independent of sex, In patients with renal artery stenosis, the chronic ischemia produced by the obstruction of renal blood flow produces adaptive changes in the kidney that are more pronounced in the tubular tissue. These changes include atrophy with decreased tubular cell size, patchy inflammation and fibrosis, tubulosclerosis, atrophy of the glomerular capillary tuft, thickening and duplication of the Bowman capsule, and intrarenal arterial medial thickening (2). The GFR is dependent on angiotensin II and other modulators that maintain the autoregulation system between the afferent and efferent arteries and can fail to maintain the GFR when renal perfusion pressure drops below 70-85 mmHg. Functional impairment of autoregulation, leading to a decrease in the GFR, is not likely to be observed until arterial luminal narrowing exceeds 50%. Blood pressure, measured distal to renal artery stenosis, was found to be less than 90% relative to aortic pressure, that was associated with significant renin release from the affected kidney.

Fibromuscular dysplasia accounts for less than 10 percent of cases of renal-artery stenosis, and 90 percent of cases of fibromuscular dysplasia involve the media, affect girls and women between 15 and 50 years of age, frequently involves the distal two thirds of the renal artery and its branches, and is characterized by a beaded, aneurysmal appearance on angiography. Intimal and periarterial fibromuscular dysplasia is commonly associated with progressive dissection and thrombosis, whereas medial fibromuscular dysplasia progresses in 30 percent of patients and is rarely associated with dissection or thrombosis. Fibromuscular dysplasia rarely leads to renal-artery occlusion. The cause of fibromuscular dysplasia is unknown, although many theories have been advanced, including those involving a genetic predisposition, smoking, hormonal factors, and disorders of the vasa vasorum as risk factors. (3).
Case study

Mr. M.M.A , 36 year old patient presented to our clinic 72 hours after an acute attack of epistaxis and headache, occurring during sleep. He was conscious and was transferred to another hospital, to control epistaxis, he was diagnosed as hypertension and secondary epistaxis. Blood pressure on admission was 220/120, this was the first time for the patient to know that he was hypertensive, he treated by lasix IV 40 mg, beta blocker & calcium channel blocker (amelodepine), blood pressure came down to 160/100 then to 150/95, epistaxis was controlled by internal nasal compression.

The patient sought our advice, There was no history of fever, chills, rash, vomiting, diarrhea, chest pain, dyspnea, peripheral edema, cough, abdominal pain, gastrointestinal bleeding, hemoptyis, slurred speech, change in mental status, unilateral weakness, or loss of consciousness. He did not smoke or use alcohol or other recreational drugs.

On physical examination, the patient was overweight. No icterus, rash, purpura, petechiae, or lymphadenopathy were detected. The carotid pulses were normal and without bruits. The lungs were clear; a systolic murmur was present. No hepatosplenomegaly was found. There was peripheral edema +ve. No lateralization signs were detected, blood pressure 150/95, pulse 78/bpm. regular & equal in both arms.

RESULTS OF INVESTIGATIONS
The following investigations were carried out:--

ECG & ECHO heart were normal.

CBC revealed, normocytic, normchromic anemia, with increase reticulocytic count.

Hematocrit, 24. Platelet count 221,000 /cmm.

Urine, no protenuria. no sugar. &no RBCs no casts.

24 hour urinary VMA 2mg. (n 1.5-5).

Serum creatinine, 0.9 mg. /dl. S.Na 139 & S.K 4.8 mEq/l.

Serum uric acid 7.1mg/dl.

Blood suger curve was normal.

Serum protein electrophoresis was normal. The prothrombin time and partial-thromboplatin time-measures of coagulation function were not prolonged. Factor V Leiden, Wild type was normal.
Lupus-anticoagulant activity was normal.

Anti-phospholipids antibodies with solid-phase ELISA tests to detect anti-cardiolipin (aCL) antibodies, were normal.

Serum cholesterol, 212 mg/dl; serum triglycerid, 180 mg dl&LDL, 170mg/dl.

Renal ultrasonography revealed that the right kidney was 11.2 cm in length and the left kidney 11.1 cm, with normal parenchyma and no hydronephrosis or masses.

Angiography of the Renal Arteries, showed no signs of renal artery stenosis. However, the right kidney showed upper pole infarction, and the left kidney showed evidence of functional lower pole renal artery stenosis, although there is no anatomical stenosis detected in angiography.

**Fig (1):** Renal angiography: trans femoral angiography catheter is seen intra-arterial.

**Fig (2):** Right kidney: there is absent vasculature in the arterial phase in the upper pole of right kidney, most probably due to renal infarction of upper pole. Normal vasculature of lower pole and rest of kidney is evident.
Fig (3): Right kidney: there is absent vasculature in the arterio-venus phase in the upper pole of the right kidney, most probably due to renal infarction of the upper pole. Normal vasculature of lower pole and rest of kidney is evident.

Fig (4): Left kidney: arterial phase: delayed filling of lower pole.

Fig (5): Left kidney: left kidney: arterio-venus phase; non filing of vasculature of lower pole.
DISCUSSION:

Classic clinical clues that suggest the diagnosis of renal-artery stenosis include the onset of stage 2 hypertension (blood pressure >160/100 mm Hg) after 50 years of age or in the absence of a family history of hypertension, hypertension associated with renal insufficiency (especially if renal function worsens after the administration of an agent that blocks hospital admissions for heart failure, and drug-resistant hypertension (defined as blood pressure above the goal despite treatment with at least three drugs of different classes at optimal doses); (4).

Our patient had old unsuspected right upper lope renal infarction, and functional left lower renal branch stasis of blood flow, this may be due to a hypercoagulable state, leading to a thrombophilia, defined as a tendency to develop thrombosis as a consequence of predisposing factors that may be genetically determined, acquired, or both, (5).

Congenital conditions of Thrombophilia: they include some of the inherited abnormalities of the anticoagulant mechanisms, such as:-

* Antithrombin (AT). [Guidelines on investigation and management of thrombophilia (6)].

* Protein C (PC), and protein S (PS) deficiencies, (7).

* Activated PC (APC) resistance phenomenon attributable (or not) to the presence of the factor V (FV) Leiden mutation, which may be defined as a poor response of plasma to the anticoagulant action of APC (8).

* Congenital dysfibrinogenemia is a risk factor for venous and arterial
thrombosis,\(^9\).

* Mutation G20210A in the prothrombin gene, which may produce hyperprothrombinemia \(^10\).

It is generally accepted that they range from AT deficiency (the most severe), to PC/PS deficiencies (intermediate severity), to APC resistance (the least severe).

Acquired conditions of Thrombophilia:

1- anti-phospholipid antibody syndrome, the anti-phospholipid antibody syndrome is characterized by repeated positive tests for lupus anticoagulant (LA) and/or solid-phase anti-phospholipid antibodies and by thrombocytopenia and fetal loss \(^11\).

2- Moderate hyperhomocysteinemia, which may be caused by a congenital deficiency of the enzymes involved in its metabolism, but it may also be attributable to a poor dietary intake of vitamins that act as cofactors (folic acid and B\(_{12}\)), and therefore, it may be easily and effectively treated by dietary supplementation, \(^12\).

Laboratory diagnosis of thrombophilia was based on investigation of the plasmatic anticoagulant pathways to detect antithrombin, protein C, and protein S deficiencies and on the search for dysfibrinogenemia and anti-phospholipid antibodies/lupus anticoagulants. Recently, laboratory investigations have been expanded to include activated protein C (APC) resistance, attributable or not to the presence of the factor V Leiden mutation; hyperprothrombinemia attributable to the presence of the prothrombin gene mutation G20210A; and hyperhomocysteinemia attributable to impairment of the relevant metabolic pathway because of enzymatic and/or vitamin deficiencies \(^13\). Because most of the tests are not reliable during anticoagulation, it is preferable to postpone laboratory testing until after discontinuation of treatment.

The condition of increase in plasma viscosity leads to, impaired blood flow resulting from alterations in the rheologic properties of the blood \(^14\). Blood viscosity varies according to the hematocrit, red-cell aggregability, and plasma viscosity. As blood viscosity increases, a nonlinear increase in shear stress in the walls of small blood vessels, particularly at low rates of shear, results in damage to the fragile vascular endothelium of the kidney blood vessels. Conditions associated with a high hematocrit, such as polycythemia, and hyperleukocytic states, including acute and chronic forms of leukemia, may
result in clinically apparent hyperviscosity of the blood, (14-16), but in this case, both white-cell and platelet counts were normal. The patient had a normocytic anemia and a normal serum iron level; thus, the quantity of bleeding had not resulted in iron deficiency. There was on splenomegaly nor cyanosis was noted.

It is appears from what has been mentioned that we did not reached a concrete diagnosis for this case, this is due to our inability to have a complete scope of all disease etiologies’ as what has been mentioned in the ‘Holy Quran’ “You have only been given a small part of knowledge”.

CONCLUSION

Our patient how present by acute attack of epistaxis and hypertension. Angiography of the Renal Arteries, showed no sign of an renal artery stenosis. However, the right kidney showed upper pole infarction, and the left kidney showed evidence of functional lower pole renal artery stenosis, although there is no anatomical stenosis detected in angiography. Work up for the cause of thrombophilia did not help the in diagnosis, which may be due to an undiscovered cause of thrombophilia.

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ضيق الشريان الكلوي

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بعد مرض ضيق الشريان الكلوي من أهم أساليب ارتفاع الضغط الدم الثانوي ووجودو أساليب
عدد ضيق الشريان الكلوي مثل صلب الشريان ويشمل 90% من الحالات المسجلة وأيضا مرض
اختلال نحو عضلات وانسجة الشريان الكلوي ويشمل 10% من حالات ضيق الشريان الكلوي.

هناك أيضا أسباب تؤدي إلى مثل تلك الحالات مثل اختلال التخثر بالدم. عند عرض المريض
على العيبية الخارجية تبين أنه يعاني من نزيف حاد من الأذن وارتفاع ضغط الدم ثم عمل أشعه بالصبغة
على الشريان الكلوي ولم يوجد في الأشعة أي ضيق بالشريان الكلوي ولكن وجود إصابة في الفص
العلوي للكليتين، الكلية اليسرى أظهرت اختلال وظيفي لسريان الدم بالشريان الكلوي مع عدم وجود
ضيق حقيقي. لم تظهر التحاليل سبب الاختلال الوظيفي لسريان الدم بالشريان الكلوي اليسر.