Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Renovascular Hypertension Secondary to Hypoplasia of Renal Artery: A Case Report

Khaoula Bourzeg[1*], Moulay Achraf Choukri[1], Rim Zerhoudi[1], Abdelkhalek Chetioui[1], Abdelmajid Bouzerda[1], Ali El Khatouri[2]

[1]Head of Cardiology Department, Cardiology Department, Avicenne Military Hospital, Marrakesh, Morocco
[2]Head of Medical Department, Cardiology Department, Avicenne Military Hospital, Marrakesh, Morocco

DOI: 10.36347/sjmcr.2022.v10i04.036 | **Received:** 27.02.2022 | **Accepted:** 31.03.2022 | **Published:** 30.04.2022

***Corresponding author:** Khaoula Bourzeg
Head of Cardiology Department, Cardiology Department, Avicenne Military Hospital, Marrakesh, Morocco

Abstract **Case Report**

The hypertension of the adult subject remains idiopathic in most cases, but the discovery of abnormalities in the standard workup should lead to the search for secondary cause. Among them hypoplasia of the renal arteries which remains a rare disease. It is characterized by hypotrophy of tubular appearance of these arteries; its clinical consequences are derived from the intrarenal hemodynamic repercussions. Its diagnostic approach is complex and requires the availability of angiography to determine the features of the renal vessels. The following case illustrates the diagnostic process of a 50-year-old man suffering from hypertension since the age of 45 years with renal failure revealing hypoplasia of the right renal artery with hypotrophy of the right kidney.

Keywords: Hypoplasia of renal arteries, kidney hypotrophy, renovascular hypertension, renal failure.

INTRODUCTION

Renal artery hypoplasia continues to be a confusing clinical problem, due to the low prevalence of this condition. The main manifestation of hypoplastic and stenotic arteries is the appearance of renovascular hypertension [1]. The descriptions in the literature of this disease are limited to some case reports with compromise primarily of the thoracic and abdominal aorta.

CASE PRESENTATION

A 50-year-old male was admitted to the cardiology consultation for a control of hypertension. The patient's cardiovascular risk factors are hypertension treated by Valsartan 160mg, chronic smoking at 30 pack year weaned 5 years ago. There was no history of diabetes, dyslipidemia, or family history of CVD. There is no notion of taking medication or Licorice. The history of his hypertension goes back to 5 years ago when he was diagnosed and managed as essential hypertension without complementary studies then the patient is lost from sight until the age of 50. In the physical examination, it was found that the patient was in fair general conditions and had the following vital signs: blood pressure of 161/92 mmHg, heart rate of 60 beats per minute, 19 breaths per minute. The body weight was 71 kg for a height of 170 cm and the body mass index was 24,6 kg/m2. Cardiorespiratory examination was normal as well as the rest of the clinical examination.

Electrocardiography showed sinus rhythm of 61 beats/minute, with a right heart axis, left ventricular hypertrophy and repolarization disorder with negatives T waves in lateral leads (figure1).

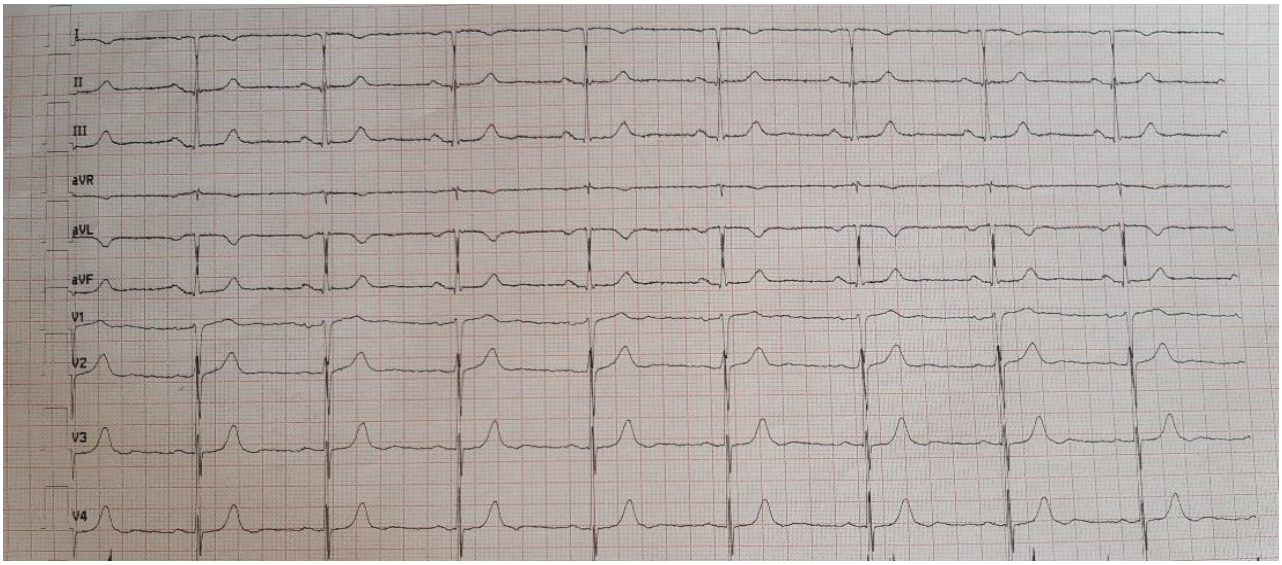

Fig-1: Electrocardiography showed sinus rhythm of 61 beats/minute, with a right heart axis, with left ventricular hypertrophy and repolarization disorder with negative T waves in lateral leads

Bloods tests showed a normocytic normochromic anemic at 11 g/dl, platelets at 220000/µL. An optimal international normalized ratio (INR) of 1, 02. The level of urea was 1.24g/l and creatinine at 34 mg/l with a glomerular filtration rate of 20ml/min. the potassium level was at 4,2 mmol/l and the natremia at 137 mmol/l. A microalbuminuria/creatinuria ratio at 0, 38. The level of triglycerides was 0,85 g/L, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) levels were within normal limits. The rest of the biological workup was within normal limits including aspartate aminotransferase (AST), alanine aminotransferase (ALT) cytobacteriological examination of urine and the PSA level.

A complement by ultrasound with Doppler of renal arteries was necessary which documented as an important finding: right kidney with reduced dimensions, hypotrophy with increased echogenicity, without dilation of the collecting system, masses, or calculi while the left kidney was without abnormality. The study reported that the left intrarenal vessels, both arterial and venous, had a normal appearance, but it been not possible to visualize the right vessels.

For this reason, the nephrology group of the hospital decided to carry out an Angio-scanner of renal arteries with a protocol of nephroprotection protocol and subsequent hemodialysis. This study determined the presence of hypoplasia of the right renal artery.

For the nephrologist the patient is not yet at the stage of hemodialysis, and they have recommended a close follow-up of the patient. And after discussions with them we were able to keep the patient on Valsartan 160mg with the addition of amlodipine at a dose of 10 mg with an adapted diet and adequate physical activity.

Ambulatory blood pressure monitoring was adopted with good blood pressure control at 1 month of treatment intensification.

DISCUSSION

The concept of an intimate causal relationship between hypertension and renal disease has developed slowly over the past 125 years. Bright [2], in 1836, noted the association of cardiac enlargement and chronic renal disease, while Mahomed [3] postulated in 1881 that high arterial pressure was a cause of Bright's disease. Congenital malformation of the renal artery, producing hypertension by the Goldblatt mechanism [4], is undoubtedly a rare entity. In 1954, Howard *et al*. [5], reporting on 6 hypertensive patients with unilateral renal disease, described a case of a thirty-nine-year-old man from whom a non-functioning left kidney was removed. Pathologic examination of the specimen disclosed that the main renal artery was absent, and, in its place, several small arteries entered the hilus of the kidney.

Also, renal artery hypoplasia continues to be a confusing clinical problem, due to the low prevalence of this condition. This has restricted the development of a specific definition and an adequate clinical approach [1]. The term congenital renal hypoplasia indicates the presence of a child-sized kidney with a preserved excretory function, in contrast with the functional alterations related to vascular malformations characteristic of renal artery hypoplasia [6]. Although its pathophysiology is not clearly known, both congenital renal hypoplasia and hypoplasia of renal arteries have a common pathophysiology, related to defects in embryogenesis, which include alterations in the development of the nephrogenic blastema of metanephrogenic ducts or primary vascular deficiencies [1]. This situation can be explained through the phenomenon described by Goldblatt [4], in which, in

animal models with only one kidney, the occlusion of one of the three main branches of the renal arteries resulted in a significant increase in the levels of renin and appearance of secondary arterial hypertension [7].

Therefore, it is not uncommon to find an association between renal artery hypoplasia concomitant with aortic hypoplasia and a variable frequency between 33% and 81% [8]. However, both conditions are extremely rare and unusual [9]. This pathology may debut clinically with acute renal deterioration or appear as a hypertension picture of difficult management in young patients.

Arteriography is the study of choice, since it allows to define whether the hypoplasia is congenital or is a case of renal atrophy, taking into account that, in hypoplasia, the renal artery is present, although with a smaller size and length, as defined by Love *et al*.[10].Frequently, the most prominent angiographic features of this pathology consist in the presence of a renal artery of a smaller size from its origin to the bifurcation, which implies a narrowing with tubular contours without focal stenoses.

Finally, it is important to stress that, among the management strategies for this condition, there are no clinical guidelines. its therapeutic approach should be based on the patient's clinical condition and on the degree of organic involvement. For the surgical treatment of hypertension, it should be noted that in many cases of many cases of unilateral renal disease, nephrectomy may not successful unless it is performed relatively early in the course of the disease. This may be due to the fact that sustained hypertension itself, by continuing to increase stress on the walls of the blood vessels, eventually causes irreversible damage to the arterioles [11].

CONCLUSION

A congenital anomaly of the renal artery can diminish the blood supply to the kidney, producing hypertension by the Goldblatt mechanism. The incidence of this entity is probably higher than might be inferred from the literature. Laboratory data (urinary infection, proteinury, leucocytury, hematury) are just a hint in diagnosing renovascular hypertension that must lead to further investigation.

REFERENCES

1. Paz, J. D. Ñ., Burbano, J. D. O., & Duque, N. H. (2018). Renovascular hypertension secondary to congenital hypoplasia of renal arteries in middle adult patient. About a case. *Revista Colombiana de Nefrología*, *5*(1), 68-73.
2. Bright, R. (1836). Cases and observations illustrative of renal disease accompanied with the secretion of albuminous urine. *Guy's Hospital Report*, *10*, 338-340.
3. Mahomed, F. (1981). Chronic Bright's disease without albuminuria. Guy's Hosp. *Rep., third series*, 25; 295, 1881.
4. Goldblatt, H., & Lynch, J. (1934). Hanzal, RF and summerville, WW: Studies on experimental hypertension. I. The production of persistent elevation of systolic blood pressure by means of renal ischemia. *Jr. Exper. Med*, 347-379.
5. Howard, J. E., Berthrong, M., Gould, D. M., & Yendt, E. R. (1954). Hypertension resulting from unilateral renal vascular disease and its relief by nephrectomy. *Bulletin of the Johns Hopkins Hospital*, *94*(2), 51-85.
6. Cha, E. M., Kandzari, S., & Khoury, G. H. (1972). Congenital renal hypoplasia: angiographic study. *American Journal of Roentgenology*, *114*(4), 710-714.
7. Brueggemeyer, C., Farber, M. S., & Ramirez, G. (1984). Goldblatt phenomenon in a single kidney: the importance of subselective renal vein renins. *Southern Medical Journal*, *77*(10), 1312-1314.
8. Odero, A., Bozzani, A., Arici, V., & Agozzino, M. (2008). Hypoplasia and fibromuscular dysplasia of infrarenal abdominal aorta with downstream aneurysm: case report and review of the literature. *Journal of Vascular Surgery*, *48*(6), 1589-1592.
9. Love, L., & Rosiers, R. J. D. (1966). Angiography of renal agenesis and dysgenesis. *American Journal of Roentgenology*, *98*(1), 137-142.
10. Des, Rosiers, R.J. (1966). Angiography of renal agenesis and dysgenesis. The American Journal of Roentgenology, Radium Therapy, and Nuclear Medicine, *98*(1); 137-42.
11. Isaac, F., Brem, T. H., Temkin, E., & Movius, H. J. (1957). Congenital malformation of the renal artery, a cause of hypertension. *Radiology*, *68*(5), 679-688.