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Wide acquired arteriovenous fistula between main renal artery and interlobar vein treated with nephrectomy

Case Report

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Abstract: A case of renal arteriovenous fistula between the main renal artery and interlobar vein diagnosed 25 years after percutaneous renal biopsy was presented. A 62-year-old female was referred to a urologist with dilatation of the left renal pyelocalyceal system diagnosed after abdominal ultrasonography, while intravenous urography did not confirm that finding. Historically, she underwent renal biopsy 25 years ago without any complication. Her hypertension was well controlled during the last 10 years, although three antihypertensive drugs with occasional additional diuretics were necessary during the last 6 months. Color Duplex Ultrasonography, arteriography and Multi-Slice Computed Tomography revealed the presence of renal arteriovenous fistula between the main renal artery and interlobar vein, as well as severe dilatation of all interlobar veins, renal, ovarian and adrenal vein on the left side. Urological and vascular surgeons operated to ligate the fistula and preserve the kidney. However, it was not possible to reach the fistula inside the kidney and nephrectomy was performed.

Keywords: *Renal arteriovenous fistula • Hypertension • Hematuria • Nephrectomy*

1. Introduction

Renal arteriovenous fistula (AVF) is a pathological communication between the intrarenal artery and vein. If communications are multiple, they are considered to be congenital malformations. Solitary communications are generally an acquired AVF. It is usually a consequence of direct trauma of the intrarenal vessels during renal biopsy, percutaneous nephrostomy, renal surgery, or blunt trauma. A third entity is idiopathic AVF, similar to acquired AVF, but without an evident cause [1].

AVF is a well-known complication of native and transplant renal biopsy. The incidence of AVF is more frequent in renal allografts, reaching 17% in some instances, but is significantly smaller after native kidney biopsies [2,3].

Although most AVFs are of little clinical importance, gross hematuria and hemodynamic changes due to high shunt flow and loss of kidney function may occur and require treatment. Over time, arterial blood fills the vein and forms a pseudoaneurysm, which can cause compressive lesions in the parenchyma, or penetrate the renal collecting system, leading to hematuria. Also, the shunting of blood through the fistula can provoke steal syndrome, which may induce deterioration of glomerular filtration rate (GFR) [4,5].

This process can last more than 30 years and may be followed by severe renal vein dilatation, hypertension, high-output cardiac failure and peripheral

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thromboembolism. However, about 80% of acquired AVFs undergo thrombosis and heal spontaneously [6]. The majority of acquired AVFs are either asymptomatic, or show transient symptoms. The most common physical findings are abdominal bruits, hypertension and cardiomegaly, while hematuria is relatively rare. The principal methods of diagnosis are color duplex sonography (CDU), computerized tomography (CT), magnetic nuclear imaging (MRI) and arteriography [7,8].

Treatment of renal AVFs must be individualized for each patient with regard to symptoms, manifestations of the fistula and overall co-morbidity. In the majority of cases, endovascular intervention is the procedure of choice and should be attempted at the time of arteriography. The use of small catheters permits successful embolization and reduces ischemia of the renal cortex. The success rate of embolization is approximately 88%. Complications of the procedure include renal infarction, hemorrhage and coil migration. Surgical treatment should be limited for giant high-flow and symptomatic AVFs. Alternative therapeutic options are arterial ligation, partial or total nephrectomy [9-11].

2. Case report

A 62 year-old female with renal arteriovenous fistula (AVF) is presented. In 1981, the patient underwent nephropexy on the right side due to nephroptosis. During hospitalization, biopsy of the supraclavicular lymph nodes was done on the left side. Pathological examination proved specific, tuberculous inflammation and the patient received anti-tuberculous therapy during the next 6 months. In 1986, she underwent percutaneous renal biopsy on the left side, due to mild proteinuria and microscopic hematuria. Pathohistological examination revealed unspecified chronic glomerulonephritis. She was not given immunosuppressive therapy and remained asymptomatic for the next 25 years, except for intermittent microscopic hematuria. Several years after renal biopsy she developed hypertension, which was well controlled with an ACE inhibitor and a beta-blocker. However, during the last 6 months three antihypertensive drugs with occasional additional diuretics were necessary for hypertension control. Due to flank pain, an abdominal ultrasound examination was performed in general practice and marked dilatation of the left renal pyelocalyceal system was noticed (Figure 1).

She was advised to visit a urologist, who indicated intravenous urography (IVU). However, IVU showed a slightly smaller left kidney without signs of urinary tract obstruction. As repeated abdominal ultrasonography confirmed the presence of hypoechoic lesions in the renal medulla, color duplex ultrasonography (CDU) was performed. The CDU examination revealed a massive high- flow vascular structure in the lower pole (Figure 2) and she was hospitalized at our clinic.

At admission, physical examination revealed a moderately obese woman with a Karnofsky score of 80%. A systolic-diastolic murmur was present on the left flank. The blood count was normal, except for fewer thrombocytes (110 x 10^9 L). The complete blood biochemical analysis was normal. Chest radiography revealed signs of left ventricular hypertrophy (LVH). M-mode echocardiography proved concentric LVH with increased wall thickness, a non-dilated chamber and an ejection fraction of 55%. The increased end-diastolic wall thickness of more than 11 mm was considered significant.

Arteriography and Multi-Slice Computed Tomography (MSCT) confirmed the diagnosis of giant renal AVF, with severe dilatation of all interlobar veins, as well as the renal, ovarian and adrenal vein. The fistula was located between the main renal artery and the upper interlobar vein (Figures 3 and 4). Surgery was performed by a urologist and vascular surgeons to ligate the fistula and to preserve the kidney. The retroperitoneal space was approached through the total medial laparotomy. The left renal vein was enormously wide, similar to vena cava. The lateral left renal vein tributaries, adrenal and ovarian vein were also dilated. These veins were immediately ligated. After that, the left renal artery and accessory renal artery for the lower pole, were identified and secured. The renal hilum was approached, but the fistula was inside the kidney so nephrectomy was performed. After removal of the kidney, greatly dilated veins were seen inside the renal tissue. The AVF was 10mm wide, located between the main renal artery and vein (Figure 5). After surgery, blood pressure normalized without any antihypertensive medication, several

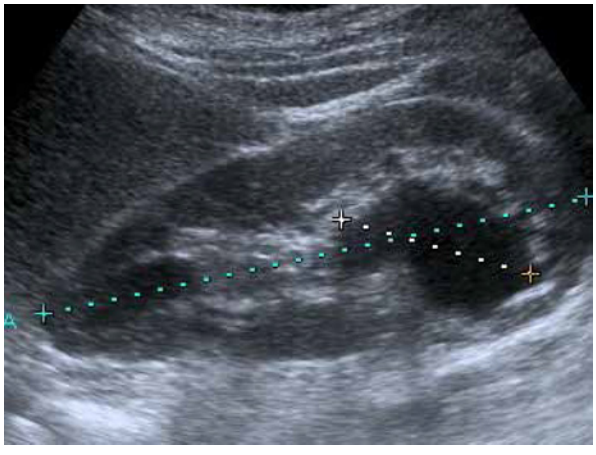

Figure 1. Ultrasonography of the left kidney showing anechoic lesion in the lower pole obtained with a 3.5-MHz transducer.

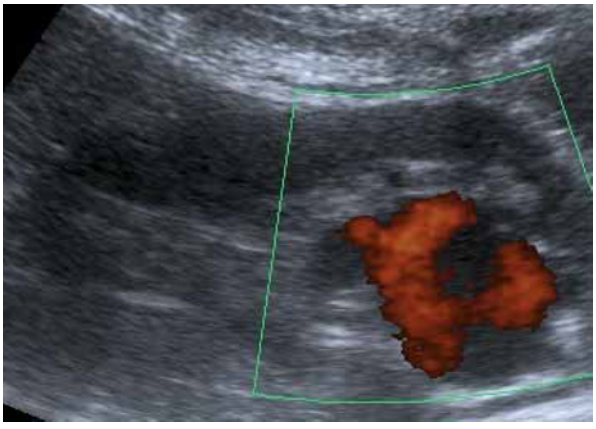

Figure 2. Color duplex ultrasonography image demonstrates an area of intense and high-flow vascular structure in the lower pole, with high velocity (165.5 cm/s) and low resistant index (0.38).

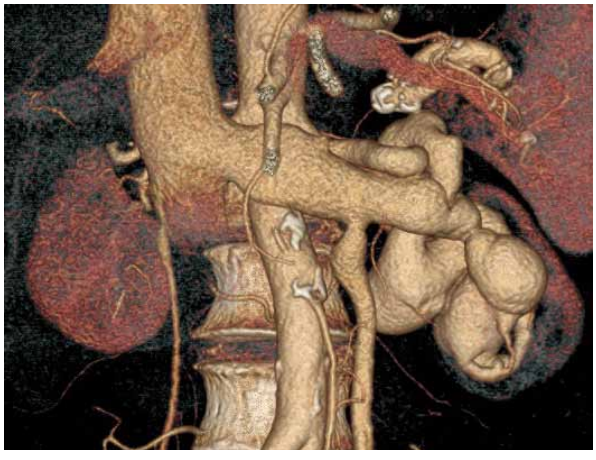

Figure 3. Multi Slice Computed Tomography revealed giant AVF of the left kidney with severe dilatation of all interlobar veins, renal, ovarian and adrenal vein.

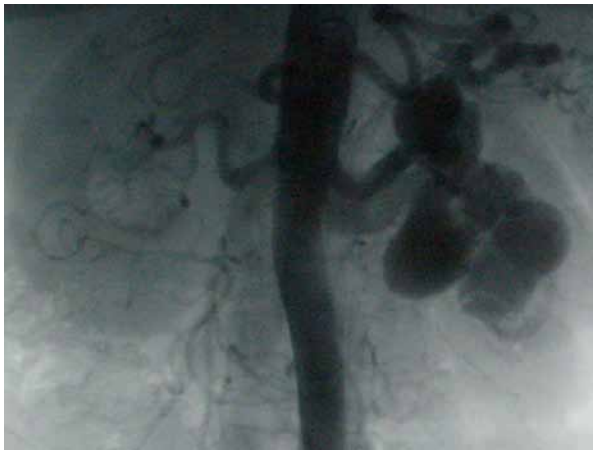

Figure 4. Arteriography showing wide AVF between renal artery and upper interlobar vein.

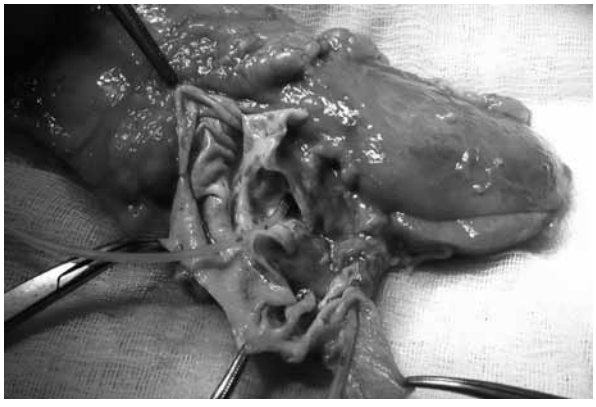

Figure 5. Macroscopic prepartation of AVF located between the main renal artery and vein. The catheter passes through the wide AVF.

repeated urine analyses were without microscopic hematuria, while function of the remaining right kidney was normal.

3. Discussion

Renal biopsy has lowered the complication rate over the past few years and is considered a safe procedure [3]. However, mild complications of renal biopsy, such as micro or gross haematuria, are common but usually resolve within weeks. According to several publications, the incidence of AVF after renal biopsy varied between 4% and 17% for both native and transplanted kidneys [2-5]. Depending on size, growth and blood flow rate the clinical presentation can be very distinct. The time period between AVF formation and clinical presentation is quite variable, but in most cases clinical symptoms of AVF appear during the first few months after biopsy. Rarely, this process can last more than 30 years and be followed with severe complications, such as renal vein dilatation, hypertension, high-output cardiac failure and peripheral thromboembolism [6,7].

Our case is one of the only described to date, where serious clinical complications due to the AVF developed 25 years after renal biopsy. During that time the AVF enlarged to such an extent that neither transcatheter embolization nor conservative surgical treatment was possible. We had to do a nephrectomy, which had beneficial effects regarding elimination of hypertension, haematuria and high output cardiac failure.

In conclusion, in certain cases with hypertension, haematuria and high output cardiac failure, we have to consider kidney AVF as a potential cause of these complications, even though the onset is several decades after kidney biopsy.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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