Hibernating kidney – a case report

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A 75-year-old woman presented with a two-month history of progressive dyspnea on exertion. On physical examination, her blood pressure was 148/90 mmHg, jugular venous pressure was 10 cmH₂O with a prominent V wave, right ventricular heave was present and she had a

systolic murmur at the right upper sternal border that varied with inspiration. The patient had a pulsatile liver, mild ascites and systolic bruits over both kidneys, with the diastolic component on the left side.

Key Words: Aneurysm; Arteriovenous malformation; Coil embolization; Pulsatile liver; Tricuspid regurgitation

A 75-year-old woman presented to the Vascular Medicine Clinic at Mayo Clinic in Rochester (Minnesota, USA) after experiencing an approximately two-month history of progressive shortness of breath on exertion. She denied any history of orthopnea, paroxysmal nocturnal dyspnea, chest pain or lower extremity edema. Her medical history included hyperparathyroidism, for which she underwent parathyroidectomy, longstanding systemic hypertension, hyperlipidemia and obesity. A physical examination revealed that the patient was comfortable and in no distress at rest. Her blood pressure level was 148/90 mmHg, and her heart rate was 90 beats/min and regular. Neck examination revealed a jugular venous pressure of 10 cmH₂O with a prominent V wave. Palpation of the precordium revealed a right ventricular heave. A systolic murmur was noted at the right upper sternal border that varied with inspiration. She had a pulsatile liver, mild ascites and systolic bruits over both kidneys, with the diastolic component on the left side. Findings on the rest of her physical examination were unremarkable. Laboratory studies revealed a creatinine level of 2.3 mg/dL. A chest x-ray revealed cardiomegaly, right ventricular enlargement, a prominent right hilum and pulmonary vascular congestion. An electrocardiogram revealed atrial tachycardia with variable atrioventricular block at a heart rate of 105 beats/min, premature ventricular complexes, right ventricular bundle branch block and left anterior fascicular block. An echocardiogram revealed severe biatrial enlargement, mild left ventricular enlargement with restrictive hemodynamics and an ejection fraction of 56%, mild right ventricular enlargement with severe tricuspid valve regurgitation, and an estimated right ventricular systolic pressure of 61 mmHg.

An abdominal computed tomography angiogram revealed a single left renal artery that branched to the upper pole, with a 3.2 cm × 2.5 cm calcified aneurysm and mural thrombus coursing inferiorly (Figure 1). The aneurysm communicated with an enlarged left renal vein. The right renal artery also had a 4.5 cm aneurysm at the lower pole, in direct communication with the markedly dilated right renal vein. The anterior and posterior

Figure 1) Aneurysmal changes with calcification of the wall of the arteriovenous malformations (arrows)

right renal veins were enlarged and involved with the arteriovenous malformation (AVM). The appearance and rate of contrast mixing in the renal artery and vein in these AVMs suggested high-volume left-to-right shunts that were likely congenital in origin (Figure 2). A technetium-99m mercaptoacetyl triglycine renal scan showed 51% function of the right and 49% function of the left kidney, suggesting an equal contribution to renal function by each kidney. Other causes of cardiomyopathy were excluded. Further inquiry revealed a history of an abdominal bruit in 1970, at which time an arteriogram revealed bilateral congenital renal AVMs.

Right heart catheterization showed a systolic/diastolic/mean pulmonary artery (PA) pressure of 54/24/36 mmHg, mean right atrial pressure of 14 mmHg and a PA wedge pressure of 16 mmHg. Thermodilution cardiac output was increased at 9.9 L/min, with a cardiac index of 4.58 L/min/m². Mean PA resistance was 7.63 Wood units/m², arterial oxygen saturation was 90% and PA oxygen saturation was 76%

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Figure 2) Reconstructed images showing bilateral arteriovenous malformations

(Table 1). After 9 ng/kg/min prostacyclin infusion, the patient's mean PA pressure decreased to 29 mmHg, cardiac output increased to 13.3 L/min, and her PA resistance decreased to 4.69 Wood units/m².

Consideration was given to nephrectomy in an attempt to relieve the left-to-right shunt; however, given the bilateral AVMs occupying a large area of renal parenchyma, it was not attempted because of the risk of dialysis. The patient underwent successful coil embolization of the left renal AVM, eliminating the left-to-right shunt and improving renal perfusion. The patient's creatinine improved to 1.3 mg/dL, tricuspid regurgitation decreased and functional capacity improved following coil embolization (Figure 3). These benefits were sustained one year after treatment.

DISCUSSION

A long-term increase in PA blood flow can affect PA distensibility (1) and induce vascular remodelling from high shear forces and mechanical vascular strain. There are increased levels of endothelin in patients with left-to-right shunt. Systemic AVMs are associated with pulmonary hypertension and high output cardiac failure. Repair of arteriovenous fistulas can improve pulmonary hypertension (2-6). The present case is the first report of bilateral congenital renal AVMs that led to heart failure late in life, and the first case to document improvement in symptoms and renal function with endovascular treatment.

CONCLUSION

Congenital AVMs may manifest as congestive heart failure (CHF) even late in life, and should be considered in the

TABLE 1

Right heart catheterization

Pulmonary artery pressure*, mmHg	54/24/36
Mean right atrial pressure, mmHg	14
Pulmonary artery wedge pressure, mmHg	16
Cardiac output, L/min	9.9
Cardiac index, L/min/m ²	4.58
Mean pulmonary artery resistance, Wood units/m ²	7.63
Arterial oxygen saturation, %	90
Pulmonary artery oxygen saturation, %	76

*Pulmonary artery pressure presented as systolic/diastolic/mean

Figure 3) Arteriogram before and after coil embolization of the arteriovenous malformation

differential diagnosis of CHF in persons of all ages. In the absence of common causes of CHF, cardiomyopathy should not be considered idiopathic without a thorough peripheral vascular examination. Treatment of congenital AVMs, even in the elderly, may result in CHF improvement.

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