Congestive Heart Failure Caused by Aortocaval Fistula after Nephrectomy

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Abstract

We treated a 66-year-old man and a 69-year-old woman with high output cardiac failure due to aortocaval fistula. They underwent nephrectomy 40 and 36 years ago, respectively. They suffered from heart failure for a very long time. However, the etiology was not elucidated until recently. The aortogram revealed massive shunt from the aorta to the inferior vena cava via the right renal artery. Three-dimensional computerized tomography clearly delineated the aortocaval fistula. Surgical closure of the fistula promptly improved heart failure. We stress the importance of history taking concerning nephrectomy and the importance of abdominal auscultation in high output cardiac failure. (*Internal Medicine* 40: 1113–1116, 2001)

Key words: renal tuberculosis, computerized tomography

Introduction

Arteriovenous fistulas, anemia, hyperthyroidism, beriberi and Paget's disease might cause high output cardiac failure (1). Arteriovenous fistulas among them can be congenital or acquired. Acquired arteriovenous fistulas occur following gunshot wounds, carcinomas, spontaneous rupture of an aortic aneurysm into the inferior vena cava or surgical procedures: nephrectomy, laminectomy and cholecystectomy (1–3). Arteriovenous fistulas after nephrectomy are rare and there are only a few case reports (4–10). Recently, we treated two cases of congestive heart failure, for which the etiology was unknown for a very long time. Their examination revealed mild aortic valvular regurgitation and huge aortocaval fistula. They had a history of nephrectomy for renal tuberculosis more than 30 years previously. We present these cases as a warning, which illustrates that the etiology of heart failure due to noncardiac origin tends to be overlooked by cardiologists.

Case Report

Case 1

A 66-year-old man had a history of right nephrectomy for renal tuberculosis 40 years earlier. He suffered from exertional dyspnea for ten years. Blood pressure was 116/70 mmHg. Heart rate was 73/min. Systolic and diastolic heart murmur due to aortic and mitral regurgitation was audible on the precordial region and continuous murmur could also be heard on the abdomen. Anemia, hyperthyroidism and beriberi were not found in his clinical examinations. His chest X-ray showed cardiomegaly and lung congestion (Fig. 1). The cardiothoracic ratio was 68%. His ECG showed left ventricular hypertrophy and drug refractory paroxysmal atrial flutter. The left ventricular diastolic dimension was 67 mm and the left atrial dimension was 45 mm, and left ventricular ejection fraction was 68% by echocardiography. Mitral and aortic regurgitation of grade II/IV were also evident on the echocardiogram. Abdominal aortography demonstrated huge aortocaval shunt and a pigtail catheter, the tip of which was easily passed through the channel (Fig. 2). The electrophysiological study showed common type atrial flutter and radiofrequency catheter ablation to the isthmus was successfully performed. The aortocaval connection was surgically closed by a patch at the orifice of the renal artery in the aortic side. Three months after the closure, left ventricular diastolic dimension decreased to 56 mm on the echocardiogram and his symptoms were markedly improved, and no medication was needed 6 months after the procedure.

Case 2

A 69-year-old woman also had a history of right nephrectomy for renal tuberculosis 36 years previously. She had exertional dyspnea for 15 years and the etiology has been unknown. She exhibited pulmonary edema at age 65 and was treated as heart failure by a cardiologist for 28 days in another hospital. After she was discharged from the hospital, she consulted our outpatient clinic. Her blood pressure was 132/60 mmHg and heart rate was 63/min. Diastolic regurgitant murmur was audible on the precordial region and continuous mur-

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Figure 1. Chest X-ray films before and after closure of shunt in case 1.

Figure 2. The aortogram in case 1 showing the huge aortocaval shunt and the presence of the tip of a pig-tail catheter in the dilated right renal artery (the left panel). Three catheter sheaths in the inferior vena cava were used for the electrophysiological study. The aortogram in case 2 showing huge aortocaval shunt through aneurysm (the right panel). IVC: inferior vena cava, AN: aneurysm, Ao: abdominal aorta, Cathe.: catheter.

Figure 3. Three-dimensional computerized tomography in case 2 showing the right renal artery aneurysm and its communication with the inferior vena cava. SMA: superior mesenteric artery.

mur was also heard on the abdomen. Anemia, hyperthyroidism and beriberi were not found in her clinical examinations. The cardiothoracic ratio of the chest X-ray was 69% and the ECG showed left ventricular hypertrophy. The left ventricular diastolic dimension was 56 mm, the left atrial dimension was 54 mm, and left ventricular ejection fraction was 73% by echocardiography. Aortic regurgitation of grade II/IV and tricuspid regurgitation of grade II/IV was also evident. Three-dimensional reconstruction of computed tomography demonstrated right renal artery aneurysm and its communication with the inferior vena cava (Fig. 3). Cardiac catheterization was performed and cardiac index was 8.0 l/min/m². Pulmonary artery pressure was 37/10 mmHg and mean pulmonary wedge pressure was 9 mmHg. The right renal arterial aneurysm and massive aortocaval shunt were demonstrated by aortography (Fig. 2). Transcatheter coil embolization was considered for closure of the shunt, but it was too large for the usual embolization technique. Therefore, surgical closure was chosen. After the fistula was closed surgically, the left ventricular diastolic dimension decreased to 44 mm on the echocardiogram and congestive heart failure was improved.

Discussion

The present two cases had very similar conditions, that is, high output cardiac failure of unknown origin for a very long time, history of right nephrectomy for renal tuberculosis more than 30 years earlier and mild aortic regurgitation. The etiol-

ogy of high output heart failure was aortocaval fistula following nephrectomy. Responsible factors in the development of arteriovenous fistula after nephrectomy may be mass ligation of the renal pedicle (8), hemorrhage (9), infection (10) and neoplasm (3). In the present patients mass ligation of the renal pedicle was probably performed before the kidney was removed and chronic inflammation of tuberculosis was also another important factor for fistula formation. As in the present cases, 70% of fistulas after nephrectomy occur in the right side (7). The mechanism of the high frequency of location in the right side has not yet been clarified. The right renal artery may be longer than the left renal artery, because the right renal artery arises from the abdominal aorta in the left side of the vertebrae, but on the other hand, the left renal artery and the aorta are located in the left side of the vertebrae. These anatomical differences between left and right renal vessels may be related to the right side dominance of occurrence of aortocaval fistula after nephrectomy.

Diagnosis of renal arteriovenous fistula is occasionally made very late. At the time of diagnosis, 90% of patients have a bruit over the flank, 80% have cardiomegaly and 50% have signs of heart failure (11). It was very important for cardiologists to auscultate the abdomen and to pay attention to a history of nephrectomy when they encounter patients with high output cardiac failure of unknown origin. Mild to moderate degree aortic regurgitation as shown in the present cases might not be the main cause of heart failure, but it could be secondary to left heart volume loading in arteriovenous fistula. Low diastolic

blood pressure showing arteriovenous fistula associated with aortic regurgitation was another important physical finding in the present cases. Three-dimensional reconstruction of computerized tomography clearly demonstrated the anatomical communication with the aorta and the inferior vena cava through renal artery aneurysm.

Transcatheter treatment has developed for closure of arteriovenous fistulas using detachable balloon and Gianturco coils (12–16). However, when the connective channel is very large, these materials might migrate to the pulmonary artery. In such cases with high flow an Amplatz spider, an intravascular trapping device, is useful for safer deposition of multiple Gianturco coils (14). However, the Amplatz spider can not be easily obtained in Japan. Therefore, we chose surgical closure of fistula.

Recently, renal tuberculosis necessitating nephrectomy is a very rare condition. However, high output cardiac failure due to arteriovenous fistula following nephrectomy does exist even at the present time, because it can take very long time to develop an arteriovenous fistula with large shunt after surgery. Moreover, other causes of arteriovenous fistula such as, abdominal trauma or inflammation are not so rare even at present. Therefore, we should take this into consideration as one of the important causes of high output heart failure of unknown origin. We must emphasize again that low diastolic blood pressure and continuous murmur on the abdomen are very important signs in making the diagnosis, and three-dimensional computerized tomography is a very useful noninvasive method for the anatomical delineation of the fistula.

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