

# High-Output Heart Failure Due to an iliac Arterio-Venous Fistula in Mycotic Aneurysm

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## Abstract

Herein, we report a patient with arterio-venous fistula secondary to mycotic aneurysm, causing high output heart failure. The patient had one-year history of refractory heart failure and recurrent pulmonary edema. This presentation is a good example of curative surgically treatable cause of high-output congestive heart failure.

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**Keywords** • Arterio-venous fistula • Mycotic aneurysm • Heart failure

## Introduction

**M**ycotic aneurysms (MA) account for about 2.6% of aortic aneurysms. Isolated MA of the common iliac artery occur less frequently than those of abdominal aorta.<sup>1</sup> The rupture of isolated iliac aneurysm into systemic vein is very rare.<sup>2</sup> Acute or chronic complications may follow after the development of arterio-venous fistula (AVF), and high-output heart failure is an important long-term complication.<sup>3</sup> These patients on examination may mimic other causes of heart failure (e.g. valvular heart disease, dilated cardiomyopathy, etc.), therefore, careful attention should be paid to noncardiac physical findings, such as thrill and/or continuous murmur over the pulsatile mass.<sup>4,5</sup>

## Case presentation

A 66-yr-old male patient was admitted to the Cardiology Division of Afshar Hospital affiliated with Shahid Sadooghi University of Medical Sciences, Yazd, Iran, with generalized pitting edema, dyspnea, jaundice and abdominal pain. His known medical history included recurrent history of hospitalization because of the episodes of pulmonary edema and refractory congestive heart failure for one year. He had also an aneurysm of the left common iliac artery that was diagnosed six months before. Initial vital sign was normal. General physical examination showed mild to moderate respiratory distress, bilateral jugular vein engorgement, pulmonary congestion, S3 gallop, a grade 3+ systolic murmur on tricuspid area, hepatomegaly and right upper quadrant tenderness, ascites, jaundice, bilateral lower extremity edema predominantly in left side, and venous engorgement of left lower extremity. Seven days after admission, because of the presence of fever, chills, and hypotension he was transferred to the intensive care unit (ICU) with impression of sepsis. Laboratory data then revealed mild microcytic anemia with hematocrit of 30%, mild leukocytosis (WBC: 14000/ $\mu$ l) and with a normal platelet count. During antibiotic therapy, erythrocyte sedimentation rate, after one hour, was

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15mm. The ascetic Fluid and blood cultures were negative. Renal function test revealed azotemia (BUN=80 mg/dl and Cr=4.1 mg/dl). Liver function tests showed mild to moderate liver dysfunction with a total bilirubin of 7mg/dl and direct bilirubin of 4.2 mg/dl that rose to 19.2 mg/dl and 12.2 mg/dl, respectively. Alkaline phosphatase (ALP), aspartate-(AST), and alanine-amino-transferase (ALT) were all within normal range. The international normalized ratio (INR) was 2.2 and his ECG showed normal sinus rhythm.

Chest X-ray showed enlarged heart silhouette with pulmonary congestion. Ultrasonography of the hepatobiliary system confirmed liver congestion with dilatation of hepatic vein and inferior vena cava, and normal gall bladder and biliary ducts. Transthoracic echocardiography showed dilated right atrium, grade III tricuspid regurgitation, and moderate left ventricular dysfunction with an ejection fraction of 35-40%. Coronary angiography demonstrated normal coronary arteries. On cardiac catheterization, the obtained O<sub>2</sub> saturation of low, middle, and high portions of right atrium were 90.3%, 85.4% and 67.3%, respectively. High and low segment O<sub>2</sub> saturations of superior and inferior vena cava were 88.9%, 90.3%, 51.6% and 53.9%, respectively.

Computed tomography scan of the abdomen and pelvis with intravenous and oral contrast showed a large saccular aneurysm measured 8.46×8.28 cm in diameter in left common iliac artery (Fig 1). A repeated physical examination showed a large pulsatile left pelvic mass extended to the inguinal region that associated with bruit and thrills, bounding of the peripheral pulses and precordial hyperactivity. Doppler ultrasonography was performed with impression of fistulated aneurysm as the cause of high output congestive heart failure. Doppler examination revealed a massive aneurysm on the proximal portion of the left common iliac artery with a longitudinal diameter of 10.2 cm and antero-posterior diameter of 7.8 cm associated with 8-mm fistula between the postero-medial portion of the aneurysm and left common iliac vein.

After confirming the diagnosis, the patient was referred to the cardiovascular surgery center. A large pulsatile left iliac artery aneurysm, approximately 10 cm in longitudinal diameters, was observed via a low-midline laparotomy. The necrotic wall was incised after proximal and distal control of the aneurismal segment and compression of the iliac vein-proximal and distal to the fistula. The presence of large amount of vegetations inside the lumen in conjunction with abnormal arterio-venous communication in the postero-medial surface of the aneurismal segment suggested a complicated mycotic aneurysm. With the assumption of an infected



**Fig 1:** CT scan of abdomen and pelvis with IV and oral contrast showing marked dilatation of IVC and a large size saccular type aneurysm (8.48×8.28 cm in diameter) in the left common iliac artery.

source, the aneurismal segment was completely resected and sent for culture and pathological study. After simple repairing of the fistula, a femoro-femoral bypass graft was performed with a 12mm Gel-sealed Dacron. The operation accomplished without complication and the patient recovered well. Pathological reports on the specimen showed necrosis of the aneurismal wall and growth of the *S. aureus*.

Pre-operative antibiotic therapy, vancomycin, cloxacillin continued for seven days, and after surgery and the patient was discharged. 10 days after operation, BUN (19mg/dl) and creatinine (0.9 mg/dl) decreased to normal values. Three months after discharge, the patient was doing well without having any signs or symptoms of congestive heart failure. Chest X-Ray showed normal heart silhouette with no pulmonary congestion and hyperbilirubinemia was subsided (total bilirubin of 0.8 and direct bilirubin of 0.2 mg/d).

## Discussion

High out put cardiac failure is a known entity, most of which is due to anemia, hyperthyroidism or pregnancy.<sup>6</sup> High output heart failure resulting from an AVF is rare, particularly when it results from an iliac mycotic aneurysm. Acute or chronic complications may follow the development of AVF. The immediate concern is massive hemorrhage or arterial insufficiency of the distal circulation. Long-term complications can be local or systemic. Local effects include peripheral arterial insufficiency, edema formation, varicosity, degenerative changes and premature atherosclerosis.<sup>7,8</sup> These changes are considered to be irreversible if the fistula is allowed to persist for more than 1 or 2 years.<sup>9,10</sup> The sys-

temic effects of AVF depend on its size, location and the duration of the shunt and include increased pulse pressure, blood volume and cardiac output. These changes, if left untreated, can result in high output heart failure. Large AVF usually results in progressive heart failure.<sup>8</sup> Alternatively, heart failure may never occur or be delayed for years in the presence of small fistula.<sup>11,12</sup> The presence of a preexisting coronary or myocardial disease may contribute to the development of heart failure.<sup>13</sup> It is also known that the location of the AVF can predict its outcome, as fistulae located centrally, near the heart, are more likely to produce heart failure.<sup>14</sup> Accurate diagnosis and exact location are usually made by angiography or duplex scanning. Occasionally rapid runoff of contrast material through the venous system makes accurate positioning of the AVF impossible. In that case, catheterization and measurement of oxygen saturation may be helpful in locating the site. Although spontaneous regression of AVF can occur, because of the very low incidence of spontaneous healing (< 3%) and the probability of future complications such as bacterial endocarditis and high-output heart failure, it is suggested that diagnosed AVF be repaired as early as possible.<sup>15</sup> Aneurysms either traumatic (pseudoaneurysms), atherosclerotic or mycotic, are very important uncommon causes of AVF.

The term of mycotic aneurysm, coined by Osler, is used to describe any nonsyphilitic infectious aneurysm that develops from endarteritis. In a review of more than 20,000 autopsies performed at Boston City Hospital from 1902 to 1951, aortic aneurysms were found in only 1.5%, with mycotic aneurysms, in turn, accounting for only 2.6% of these cases.<sup>16</sup> However, an increasing number of these aneurysms have occurred in recent years, suggesting that the incidence may be increasing.<sup>17</sup> The most common site of mycotic aneurysms is the femoral artery (38%), followed by the abdominal aorta (31%).<sup>18</sup> Most infected aortic aneurysms in early reports were associated with valvular cardiac infectious diseases before the antibiotic era. With the introduction of antibiotics, however, the cause and location of endarteritis and associated aneurysm formation changed. Currently, about 80% of mycotic aneurysms are the result of microbial arteritis; 3% are estimated to involve infection of a preexisting aneurysm. *Staphylococcus aureus* (30%) and *Salmonella* species (50%) are the predominant organisms in mycotic aneurysms in the post-antibiotic era.

The diagnosis of mycotic aneurysms can be very difficult. Although 48% of all patients (e.g, those in the present series and those reported in the literature) had symptoms before rupture, the symptoms of intact iliac aneurysms often

mimicked urological or neurological diseases. In contrast to persons with abdominal aortic aneurysms, only 36% of all patients had a palpable abdominal or rectal mass before rupture. In 65% of all cases, noninvasive roentgenography failed to show the lesions. An aneurysmal lesion was often (23%) present in the opposite iliac system as well.

Fever and leukocytosis are usually the first findings in 70% of cases, with a palpable aneurysm or back pain constituting the third part of a classic triad of symptoms.<sup>19</sup> However, several patients have presented with nonspecific abdominal or chest pain and no distinctive clinical features. The symptoms of sepsis may be discrete and may easily go unrecognized, especially in the early stages. Pathologically, endarteritis and mycotic aneurysms may exhibit signs of acute or chronic inflammation, necrosis, hemorrhage, or abscess formation, often with an intact intimal lining. Therefore, blood cultures may be negative in up to 50% of cases.<sup>20-22</sup>

Clinical suspicion is fundamental in the diagnosis of mycotic aneurysms. Prompt treatment increases the likelihood of a favorable outcome. The source of infection should be thoroughly investigated, and if warranted, Computed tomography (CT) of the chest and abdomen should be done. CT provides earlier diagnosis and is considered the gold- standard for diagnosis of this disease. The CT scan may show irregular peripheral enhancement of the artery wall, consistent with periarteric inflammation and effusion. Angiography provides the anatomic details of the lesion and adjacent vessels but will not show the peri-arteric extension that CT can demonstrate. Color Doppler ultrasonography can demonstrate the flow from the artery into the abscess cavity.

The management of AVF associated with mycotic aneurysms requires eradication of the source of infection, closure of the fistular duct and maintenance of distal arterial flow by extra-anatomical bypass. Surgery is almost always indicated, since mortality for untreated patients is greater than 90%.<sup>6</sup>

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