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Diagnosis of "Bursting Heart Syndrome" Due to an Ilio-Caval Fistula in the Emergency Department: A Case Report

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Abstract: **Case Report**

"Bursting Heart Syndrome" is a clinical condition resulting from a sudden increase in venous return associated with peripheral arterial insufficiency, caused by an arteriovenous fistula typically located between the caval system and the aorta, most commonly in aorto-caval fistulas. These represent one of the least recognized complications of abdominal aortic aneurysms encountered in the emergency setting. This syndrome may go unrecognized and can rapidly lead to cardiac arrest. Among these fistulas, those involving communication between the inferior vena cava (IVC) and the common iliac artery (ilio-caval fistulas) are extremely rare. Apart from aortic aneurysms, etiologies are predominantly traumatic and iatrogenic, although other causes have been reported. Clinical presentation is variable and may result in delayed diagnosis. However, specific clinical and imaging findings can establish the diagnosis. Doppler ultrasound, owing to its immediate availability in the emergency department, and contrast-enhanced computed tomography angiography (CTA) of the abdomen are essential diagnostic imaging modalities. Doppler ultrasound often allows visualization of the site of communication between the artery and the vein, while CTA confirms the diagnosis. Treatment is surgical, either via open or endovascular approaches. We report the case of a 69-year-old female patient, followed in cardiology for multivalvular heart disease for six years, with a history of uterine fibroids with recurrence, and treated for a leg ulcer, who presented to the emergency department with worsening dyspnea associated with orthopnea and abdominal pain. On examination, she exhibited signs of global heart failure. Given the abdominal pain, an urgent ultrasound examination was performed, demonstrating a dilated IVC with a "yin-yang" sign on color Doppler, associated with dilatation of the iliac veins, more pronounced on the right side. A rapid Doppler survey of the veins of both lower limbs revealed arterialization of flow within the deep and superficial venous systems, with multiple bilateral pulsatile varicose clusters exhibiting arterialized flow. Careful assessment of the walls of the major abdominal vessels identified a breach in continuity establishing communication between the right common iliac artery and the IVC, located immediately above the confluence of the two common iliac veins and a few millimeters distal to the aortic bifurcation. In the context of pulsatile varicosities, hypotension, development of distal cyanosis of the lower limbs, and worsening peripheral venous insufficiency, together with Doppler ultrasound findings, the diagnosis was retained as a contributing cause of cardiac decompensation in this patient. Emergency abdominal CTA was indicated; however, hemodynamic deterioration with associated renal failure necessitated immediate admission to the intensive care unit, preceding the patient's death due to cardiogenic shock. Early diagnosis and surgical management of such fistulas prior to the onset of shock may double survival rates, underscoring the importance of prompt recognition [1-5]. Subsequently, a brief review of the literature is presented.

Keywords: Iliocaval Arteriovenous Fistula (AVF), High-Output Heart Failure (HOHF), Bursting Heart Syndrome, Aorto-Caval Fistula, Ilio-Caval Fistula.

INTRODUCTION

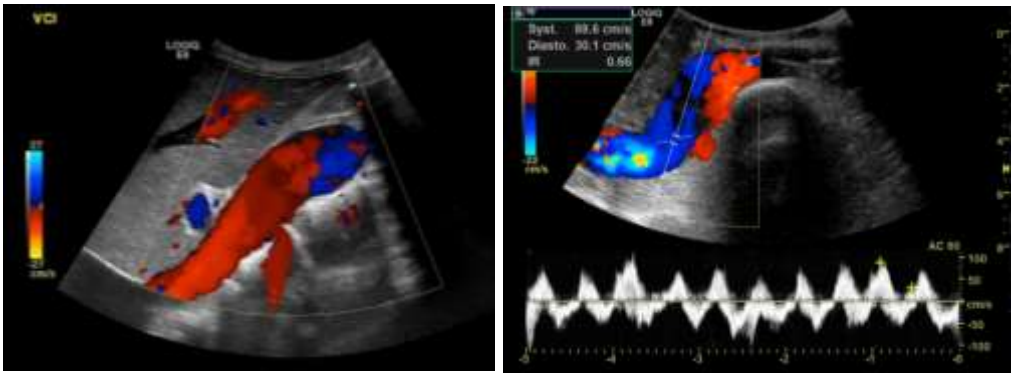
Abdominal arteriovenous fistulas (AVFs) are life-threatening vascular complications that remain challenging to manage. The first description of a major abdominal arteriovenous fistula was reported by James Syme in 1831 [1]. These conditions are rare and most commonly complicate abdominal aortic aneurysms. Among these fistulas, those establishing communication

between the inferior vena cava (IVC) and the common iliac artery (ilio-caval fistulas) are extremely rare. Etiologies are predominantly traumatic or iatrogenic, although other causes may be encountered. Clinical presentation is variable, and diagnosis may be overlooked; however, certain specific clinical and imaging features can guide diagnosis. Doppler ultrasound often enables localization and visualization of the site of communication between the artery and the vein, while contrast-enhanced computed tomography angiography (CTA) confirms the diagnosis. Treatment is surgical, either via open or endovascular approaches [1-5].

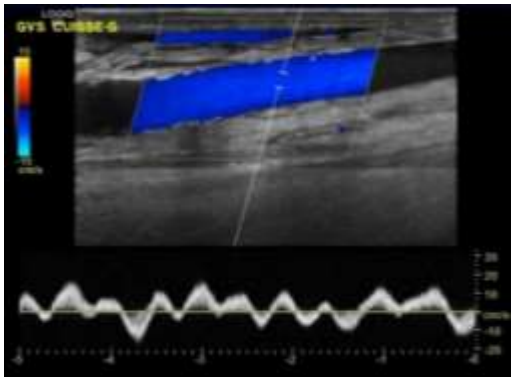
Case Report

A 69-year-old female patient, with no known history of diabetes or hypertension, had a medical history notable for rheumatic multivalvular heart disease followed for six years, with two recent episodes of decompensated global heart failure associated with atrial fibrillation during the current year. She also had treated peripheral hypothyroidism and a history of uterine fibroids operated on 16 years earlier, with current recurrence. Additionally, she had been treated for a leg ulcer secondary to chronic venous insufficiency of the lower limbs, with favorable evolution. She presented to the emergency department with worsening dyspnea, abdominal pain, and exacerbation of lower limb edema and painful varicosities, along with recurrence of the leg ulcer. Clinical examination revealed a soft abdomen, orthopnea, fever (39°C), lower limb edema, and signs of both right- and left-sided heart failure. Chest radiography demonstrated findings consistent with acute pulmonary edema, pulmonary infection, and a small pleural effusion. An urgent abdominal ultrasound was performed. The examination was technically limited due to orthopnea and was conducted in a near-seated position. Nevertheless, in addition to findings of congestive hepatopathy, chronic left nephropathy, and pelvic masses most likely related to uterine fibroids, it demonstrated a markedly dilated inferior vena cava measuring 28 mm in maximal anteroposterior diameter and 56 mm in maximal transverse diameter. Color Doppler evaluation revealed a “yin-yang” sign, indicating bidirectional flow, with turbulent arterialized flow more pronounced in the distal segment near the confluence of the iliac veins. This was associated with marked dilatation of both common and external iliac veins, more prominent on the right side. A rapid Doppler survey of the veins of both lower limbs demonstrated arterialization of flow within both the deep and superficial venous systems, particularly in the common femoral veins and great saphenous veins bilaterally, with multiple pulsatile and painful varicose clusters involving both the great and small saphenous territories, showing arterial flow on Doppler imaging. Careful examination of the major abdominal vessels identified a small breach in vascular continuity forming a fistulous communication between the right common iliac artery and the IVC, located immediately above the confluence of the two common iliac veins and just distal to the aortic bifurcation. On color Doppler imaging, this communication demonstrated a high-velocity turbulent jet entering the IVC. A previous abdominal CTA performed six months earlier had shown significant parietal thickening of the abdominal aorta and its terminal branches without evidence of aortic or iliac aneurysm, along with partially calcified atherosclerotic changes of the right common iliac artery at the same level as the current fistula. In the presence of pulsatile varicosities, hypotension, development of distal cyanosis in the lower limbs, and worsening peripheral venous insufficiency, together with Doppler ultrasound findings, an ilio-caval arteriovenous fistula was considered a contributing cause of cardiac decompensation, in addition to pulmonary infection and the natural progression of her valvular disease.

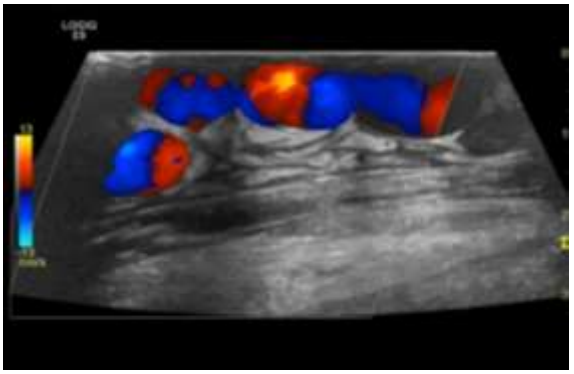
Abdominal CTA was indicated; however, the patient presented with dehydration and severe renal failure, which progressed to shock requiring admission to the intensive care unit. Unfortunately, the patient died within hours of admission.



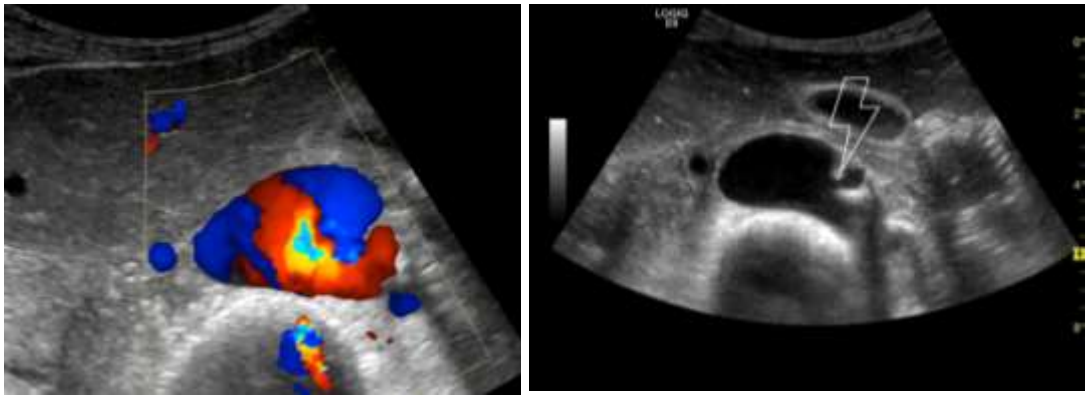
A: Yin–yang sign with turbulent flow and dilatation of the inferior vena cava (IVC), demonstrating arterialized flow



B: Arterialized flow in the great saphenous vein



C: Varicose pulsative veins of the lower Limb



D: Ilio-caval fistula on B-mode ultrasound (arow), with marked turbulence at the level of the fistula on color Doppler

DISCUSSION

An arteriovenous fistula (AVF) is defined as an abnormal direct communication between an artery and a vein, generating a systolic–diastolic shunt. The most common location of acquired AVFs is the extremities. Aorto-caval fistulas involve communication between the aorta and, most frequently, the infrarenal inferior vena cava (IVC). They may also occur between the aorta and iliac veins, between iliac arteries and their corresponding veins, and more rarely between the aorta and renal veins, or between an iliac artery and the IVC (ilio-caval fistulas). These represent severe complications with challenging management and a life-threatening prognosis [1-5].

From an epidemiological perspective, ilio-caval fistulas are exceptional. Their

incidence ranges from 0.4% to 1.4% of all AVFs and they most commonly occur following penetrating trauma or lumbar disc surgery [6-8]. Historically, the mortality associated with this condition has ranged from 16% to 66% [9, 10].

Several studies have investigated the physiology of AVFs, demonstrating a relationship between blood flow and fistula size. For small fistulas, with a cross-sectional area less than 1.5 times the arterial diameter, proximal arterial flow increases approximately fivefold, while distal arterial flow direction remains preserved. In contrast, in large fistulas (greater than three times the arterial diameter), there is a marked increase in proximal arterial flow, whereas distal arterial flow may be reduced or even reversed [1].

Regardless of their size, central AVFs involving major abdominal vessels can produce distal hemodynamic consequences. This is primarily related to the "vascular steal" phenomenon, whereby blood preferentially flows into the low-pressure venous system through the fistula rather than into the distal arterial bed, resulting in distal arterial insufficiency. This phenomenon is particularly frequent in cases of severe atherosclerosis affecting arteries distal to the fistula site, especially in proximal AVFs [11–14].

In spontaneous acquired fistulas, particularly those complicating aortic aneurysms, chronic inflammation of the adventitia with wall necrosis leads to adhesion with the adjacent vein. Arterial wall calcifications do not prevent fistula formation [1].

The pathophysiological consequences of AVFs depend on the magnitude of shunt flow. The larger and more proximally located the fistula, the greater the arteriovenous shunt. AVFs divert blood flow, leading to downstream ischemia. Proximal arterial flow increases, particularly during diastole, while venous return becomes centripetal and pulsatile. Distal arterial pressure decreases, and capillary resistance exceeds that of the fistula, resulting in reversed and retrograde flow with relative ischemia of the distal territory.

Distal venous pressure remains higher than that at the fistula site, maintaining centripetal venous flow. However, in large, high-flow fistulas, distal venous pressure may fall below that at the fistula, resulting in centrifugal (retrograde) flow extending to the first competent venous valve. Over time, progressive venous dilatation leads to valvular incompetence, further perpetuating retrograde flow toward the periphery [15].

Understanding the pathophysiological changes associated with central AVFs is essential for prompt diagnosis, both clinically and radiologically. The initial effect of a central AVF is a decrease in peripheral vascular resistance, accompanied by a marked increase in cardiac output. This increase results from both augmented venous return and compensatory mechanisms aimed at maintaining peripheral perfusion, including renal sodium and water retention mediated by secondary hyperaldosteronism.

The net effect is a significant increase in total blood volume, as the venous system becomes pressurized and venous dilatation markedly increases vascular capacitance. As right heart function progresses toward high-output cardiac failure, pulmonary hypertension may develop. In cases of end-stage right heart failure due to an AVF, abrupt reduction of fistula flow (e.g., by occlusion) may precipitate hemodynamic collapse. Renal dysfunction has also been described, likely resulting from both reduced renal plasma flow and renal venous hypertension [5, 6].

Iliocaval fistulas are broadly classified into three categories based on their origin: Iatrogenic Injury: This is an increasingly reported cause, most frequently occurring as a rare complication of lumbar disc surgery [1, 6]. Vascular injury is estimated to occur in up to 0.05% of these spinal procedures, where

surgical instruments can inadvertently injure the great vessels lying directly anterior to the vertebral column [16]. The clinical signs of the fistula may not be immediately apparent, often presenting weeks, months, or even years after the initial surgery [16].

Spontaneous Aneurysmal Rupture: The erosion and rupture of an atherosclerotic iliac artery aneurysm into the adjacent, adherent iliac vein or IVC is a primary cause of spontaneous fistulas. This specific complication is reported in less than 1% of all abdominal aortic aneurysms but can lead to a very large shunt and acute, severe symptoms [20]. In one case, a 70-year-old man complained of sudden groin pain, collapsed, and was found to be in cardiac arrest; the cause was a ruptured 7 cm iliac aneurysm that had fistulized into the iliac vein [16, 18-20].

Trauma: Penetrating injuries to the abdomen or pelvis can also result in the formation of a traumatic AVF Other etiologies include infectious and neoplastic causes [16]. The clinical picture can range from subtle and chronic to dramatic and acute. Chronic Presentation: Patients with iatrogenic or slowly expanding fistulas may present with a gradual onset of symptoms over months. These include progressive shortness of breath on exertion, orthopnea, and signs of severe fluid retention such as bilateral lower limb edema, abdominal distension from ascites, and congestive hepatomegaly [16].

Acute Presentation: Spontaneous rupture of an aneurysm into the venous system can cause a sudden and catastrophic presentation. Patients may experience acute abdominal, back, or groin pain, followed by rapid hemodynamic collapse, shock, and even cardiac arrest [18]. In some cases, the presentation can mimic septic shock with multi-organ failure, confounding the initial diagnosis [19, 20].

Key Physical Findings: Regardless of the acuity, a thorough physical examination can reveal critical clues.

Abdominal Bruit: A highly suggestive sign is the presence of a loud, continuous, "machinery-like" murmur or a palpable thrill over the abdomen, typically in the lower quadrants or periumbilical region [1, 5].

Cardiovascular Signs: Cardiovascular examination may reveal tachycardia and a wide pulse pressure; one patient had a heart rate of 100/min and a blood pressure of 100/48 mmHg [1]. Signs of severe heart failure, including elevated jugular venous pressure and pulmonary rales, are common.

Peripheral Signs: Venous hypertension in the lower extremities may lead to edema, stasis dermatitis, and varicose pulsatile veins. A multi-modality approach is essential to confirm the diagnosis, assess the hemodynamic impact, and plan treatment. A transthoracic echocardiogram is a crucial initial investigation. It typically reveals signs of severe volume overload, such as dilation of the right atrium and right ventricle, a plethoric and dilated IVC, and often severe tricuspid regurgitation. Left ventricular function is usually preserved or hyperdynamic, at least initially [16]. Computed Tomography Angiography (CTA): CTA of the abdomen and pelvis is the diagnostic modality of choice [15, 18]. It is fast, non-invasive, and provides detailed anatomical information, with reported sensitivity and specificity approaching 100% [16]. The pathognomonic finding is the early and dense opacification of the iliac vein and IVC during the arterial phase of contrast injection, confirming the fistulous connection. CTA can precisely define the fistula's location, size, and its relationship to an underlying aneurysm, which is critical for planning repair [1].

Invasive Hemodynamic Assessment: While not always necessary for diagnosis, right heart catheterization (RHC) can provide definitive physiological proof of HOHF. It will demonstrate a markedly elevated cardiac output and a low Systemic Vascular Resistance (SVR). A high mixed venous oxygen saturation (SvO2) is also characteristic

due to the shunt; one case reported an SvO2 of 86% [5]. The authors of one report hypothesized that a standard "shunt run" with oxygen saturation sampling may fail to show a step-up in oxygen saturation in the IVC if the shunt volume is extremely high, causing rapid mixing of blood in the right atrium [17]. Management requires initial hemodynamic stabilization followed by definitive closure of the fistula. Initial Stabilization: Patients are often critically ill and require intensive care. Management involves standard heart failure therapies like diuretics to manage volume overload. The use of vasopressors and inotropes can be challenging in the setting of extremely low SVR [20]. In one case of cardiac arrest, resuscitative endovascular balloon occlusion of the aorta was attempted proximal to the fistula, but the patient did not regain spontaneous circulation [18].

Definitive Repair: The primary goal is to close the fistula, thereby restoring normal hemodynamics. This can be achieved via endovascular or open surgical techniques. Endovascular Repair: This has become the first-line treatment for many patients, offering a less invasive approach with lower morbidity and mortality [16, 22]. In one reported case, the defect was repaired by coil embolization of the iliac arteries as well as placement of an endovascular graft [20].

Open Surgical Repair: Surgery is reserved for cases where endovascular repair is anatomically unsuitable (e.g., very large fistula, complex anatomy) or in emergent situations [16]. In one case, a midline laparotomy was performed, and due to difficulty in dissecting the densely adherent vessels, the iliac artery was ligated proximal and distal to the fistula, with arterial continuity restored using an interposition Dacron graft [16].

Successful closure of the fistula leads to a dramatic and immediate reversal of the adverse hemodynamics. In one case, after repair, the patient's cardiac output dropped from 14.88 L/min to 8.7 L/min, and mixed venous oxygen saturation decreased from 86% to 69% over three days [20]. Patients typically experience a rapid improvement in their heart failure symptoms and can be weaned from hemodynamic support [5]. In one surgical case, the patient's recovery was uneventful with resolution of symptoms, and he was discharged on the 6th postoperative day [16]. Follow-up imaging with CTA is used to confirm a durable repair with no residual fistula or endoleak [22].

CONCLUSION

Iliocaval arteriovenous fistula is a rare but potentially fatal cause of high-output heart failure. Its varied presentation, from chronic congestive symptoms to acute cardiac arrest, requires a high degree of clinical suspicion, particularly in patients with a history of lumbar surgery or aortoiliac aneurysms. The presence of an abdominal bruit is a vital clinical clue. Ultrasound and CTA are the cornerstone of diagnosis, providing rapid and definitive anatomical information. With timely diagnosis and definitive treatment - increasingly performed via less invasive endovascular techniques- the underlying shunt can be corrected, leading to a complete reversal of the heart failure syndrome and an excellent prognosis.

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