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Spontaneous iliac arteriovenous fistula, high-output heart failure, and cardiac arrest

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Introduction:

High-output heart failure is a potentially life-threatening condition that can lead to cardiac arrest. The most common causes of this condition are obesity, liver disease, arteriovenous shunts, lung disease, and myeloproliferative disorders, however the exact prevalence remains uncertain [1]. Here we describe an unusual case of cardiac arrest as a consequence of high-output heart failure, secondary to rupture of an iliac artery aneurysm into the common iliac vein, with arteriovenous (AV) fistula formation.

Case Presentation:

A 70 year-old male presented to the Emergency Department via EMS after report of cardiac arrest. EMS found patient at home in PEA, CPR was performed with 1 round of epinephrine administered, and he was intubated in the field. Patient regained spontaneous circulation after 1 round of CPR, and arrived to the ED unresponsive but with a pulse. Patient's wife reported that patient had been complaining of groin pain. He walked outside reportedly to smoke cannabis when his wife heard him collapse on the ground, found him unresponsive and called 911. Patient's wife provided no further history of recent illnesses, and stated patient did not regularly see any physicians or take any medications.

On presentation, his vital signs were blood pressure 97/63, heart rate of 160 beats per minute, temperature of 94.3 Fahrenheit rectally, with respiratory rate of 22 breaths per minute and pulse oximetry 100% on ventilator. His body mass index (BMI) was 29.0 kg/m².

Physical examination revealed an ill-appearing, unresponsive male with skin cool, dry and pale with mottling noted. ETT was in place, pupils were 3 mm and equal, round and reactive to light. On auscultation heart was tachycardic with irregular rhythm, breath sounds were coarse bilaterally with symmetric chest expansion. Abdomen was soft with normal bowel sounds, no masses or distention noted. Musculoskeletal exam did not reveal any significant deformity or peripheral edema. Neurologic exam was limited due to unresponsiveness and GCS of 3.

An electrocardiogram revealed atrial fibrillation at 138 beats per minute with rightward axis, diffuse T wave inversions and ST depressions in inferior and lateral leads. Lab work was completed revealing a high-sensitivity troponin level of 469, arterial blood gas with pH 7.04, PCO₂ of 42, pO₂ of 400 with bicarbonate of 11. Lactate was elevated at 5.6. Basic metabolic panel was significant for potassium of 2.5, and complete blood count revealed a leukocytosis of 16.3, with elevated hemoglobin and hematocrit of 16.4 and 49.9 respectively. There were no other significant electrolyte derangements, and the rest of lab work including hepatic enzymes, lipase, ethanol, salicylate, creatine kinase were all unremarkable. Patient was taken immediately to obtain CT scan studies of the head, cervical spine, chest, abdomen and pelvis with contrast. Results were significant for 7 cm left common iliac artery aneurysm with rupture into the common iliac vein with arteriovenous (AV) fistula communication between the two vessels. A 4.8 cm infrarenal abdominal aortic aneurysm was also noted with no signs of leakage. There was no evidence of acute pulmonary emboli or pleural effusions. Repeat ECG again revealed atrial fibrillation at 171 beats per minute with rightward axis, again with ST depressions in leads V4 through V6, with new ST depressions in leads I and aVL, and new ST elevations in leads v1 and v2. Medications administered included normal saline 1 L IV fluid bolus, potassium 20 mEq IV and 40 mEq via OG tube, magnesium 2 g IV, ceftriaxone 1 g IV, and fentanyl 50 mcg IV. Cardizem infusion was initiated at 5 mg/hr given atrial fibrillation with rapid ventricular response.

Given CT scan findings, call was immediately placed to vascular surgery team at the nearest hospital within system. Patient was stabilized for transfer, tachycardia persistent in low 130s with blood pressure 105/69. Transport arrived within the hour for transfer of patient by helicopter for emergent intervention with endovascular aneurysmal repair. At receiving facility patient was brought immediately to the OR where he again sustained PEA cardiac arrest approximately 2 hours after departure. Despite insertion of resuscitative endovascular balloon for aortic occlusion proximal to AV fistula, patient could not regain spontaneous circulation with minimal cardiac electrical activity, and patient was pronounced deceased.



Figure 1: Computed tomography with IV contrast demonstrating iliac artery aneurysm with arteriovenous fistula

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Discussion:

High-output heart failure is a less common form of heart failure and is the result of the heart's inability to keep up with metabolic demand. This is distinct from more common forms of heart failure which are a result of systolic or diastolic dysfunction. Patients with high-output heart failure have normal cardiac function with decreased vascular resistance, often a consequence of another underlying disease process. This condition has two main physiologic causes: an increase in oxygen consumption from increased metabolism leading to an increase in the body's demand for blood, and a decrease in systemic vascular resistance often due to bypass of the arteriolar and capillary bed leading to increased flow into venous circulation, and increased venous return [1]. The increase in cardiac output creates substantial myocardial stress leading to heart failure.

Arteriovenous shunt formation is among the most common reasons for high-output heart failure. Arteriovenous (AV) fistula creation is most often encountered in patients with end stage renal disease who require access for dialysis. Even among these patients, high-output heart failure is a rare complication [2]. Iliac AV fistula refers to the formation of an abnormal communication between the iliac artery and vein, often a result of operative or penetrating trauma, or less often, spontaneous aneurysmal rupture. Aneurysm of the iliac artery is the most common form of aneurysm after abdominal aortic aneurysms, which is itself often a consequence of prolonged and uncontrolled blood pressure elevation [3]. Complaints of lower abdominal or flank pain are common, with clinical features including bruit, pulsatile mass, and edema. Iliac AV fistula is a rare diagnosis that can carry a mortality rate of up to 60% [4].

Diagnosis of an iliac arteriovenous fistula can be made using angiography, duplex and color doppler sonography, MRI, and CT. Although angiography is considered the most definitive diagnostic technique, CTA is often considered a superior diagnostic tool given that it is rapid, minimally invasive, accurate, and less operator-dependent. CTA is often the preferred as it is readily available in most emergency departments, and has sensitivities from 90 to 100% and specificities from 90 to 100% [5]. Treatment options vary from conservative management through compression therapy, or more definitively through open or endovascular repair with vascular surgery [6]. The principal objectives of treatment are arterial repair and removal of venous communication to prevent further arterial dilation, rupture, and progression of sequelae such as high-output heart failure [7].

Conclusions:

We reported a case of cardiac arrest as a result of high-output heart failure due to iliac AV fistula. The patient in this case developed an iliac AV fistula as a result of iliac aneurysmal rupture, leading to high-output heart failure, cardiac remodeling, arrhythmogenesis and subsequent cardiac arrest. Early detection of this condition is crucial, as is expeditious operative intervention by vascular surgery team.