

Vascular Cases of High Output Heart Failure Masquerading as Low Output Heart Failure

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Citation: Imbriano LJ, Maesaka JK (2021) Vascular Cases of High Output Heart Failure Masquerading as Low Output Heart Failure. J Urol Ren Dis 06: 1238. DOI: 10.29011/2575-7903.001238.

Received Date: 05 November, 2021; **Accepted Date:** 11 November, 2021; **Published Date:** 15 November 2021

High Output Heart Failure (HOHF) is a rare curable disorder that shares signs and symptoms with the more dangerous and lethal low output heart failure (LOHF or congestive heart failure - CHF). The rarity of HOHF, and similar symptoms shared between HOHF and LOHF may lead to inappropriately characterize dyspnea on exertion and peripheral edema as LOHF. HOHF occurs in patients with normal hearts. These patients are challenged with an increased blood return to the right heart, stretching the right atrium and right ventricle which compensate with an increase in heart rate and stroke volume to fill the arterial tree which is “underfilled” because of the rapid return of blood to the right heart. Contrary to the cardiomyopathy of LOHF most causes of HOHF are curable [1,2]. Symptoms common to both HOHF and LOHF include dyspnea on exertion, decreased exercise tolerance, cough, edema, orthopnea, tachycardia, palpitations, and hypotension. Subtle differences such as “warm extremities” and a “bounding pulse” with wide pulse pressure imply the presence of HOHF, whereas the extremities in LOHF are cold and dry, and the hypotension has a narrow pulse pressure. HOHF can occur in obesity, liver disease, hyperthyroidism, thiamine deficiency (Beriberi) [3], severe anemia [4], arterio-venous fistulas (A-V fistulas) [5], septic shock, Paget’s disease, lung disease and pregnancy.

In the hospital setting, the symptom complex simulates many possibilities and the diagnosis of HOHF often becomes one of exclusion after failure of common remedies to resolve the hypotension associated with it. The measurement of right ventricular end diastolic pressure, cardiac output, pulmonary artery pressure, systemic vascular resistance, cardiac enzymes, and echocardiography will document “heart failure” but not necessarily distinguish which type, LOHF or HOHF. The finding of normal or elevated cardiac output should raise suspicion of HOHF. [6] Mistakenly treating HOHF with diuretics can exacerbate the problem. The patient’s history and diet habits may point to thiamine deficiency, while the physical exam may reveal evidence of vascular shunting. Blood cultures may exclude septic

shock. Bone x-rays, bone scans, and thyroid hormone testing can exclude Paget’s disease and hyperthyroidism. In the outpatient setting, patients with HOHF often present serendipitously with non-specific mild symptoms of dyspnea on exertion, hypotension, and palpitations that have been worsening for an extended period of time. Three patients with a history of renal disease were referred to our outpatient nephrology office for evaluation of “heart failure”. Each one was considered to have early onset LOHF which proved to be erroneous after the physical exam. The auscultation of bruits was the key to making the diagnosis of HOHF related to arterio-venous shunting of blood.

Case 1

A 17-year-old female high school student was referred to the renal office. She had been admitted 35 years earlier to the county medical center because of foamy urine, diffuse lower extremity soft pitting edema and an eight-kilogram weight gain over the preceding 2 months. Her urine albumin excretion was just over 7000 mg/24 hours. Serum albumin 1.4 gm/dl. All other lab parameters were normal, including glucose, blood urea nitrogen (BUN), creatinine, liver function tests, electrolytes, ANA, anti-DNA, C3, C4, CH50, hepatitis profile, CMV, and cryoglobulin level. Chest x-ray and renal sonogram were normal. She was not taking any medication, and her past medical history was normal. Consent for a kidney biopsy was obtained from her mother. Using ultrasound guidance and sterile technique, a left kidney biopsy was obtained using a 16-gauge Vim-Silverman biopsy needle. A one-half inch vertical skin incision was closed with one 4-0 silk suture transversally.

The biopsy revealed minimal change nephropathy. Prednisone was prescribed at 40 mg/day for 30 days, and slowly weaned off over the next 6 weeks. She responded completely in follow up visits and was lost to follow up after weaning. At the present time she was referred to the renal office for increasing edema, dyspnea on exertion, severe palpitations, and easy fatigue.

Her history revealed the prior treatment for minimal change disease as well as two normal pregnancies in her 20's. The onset of these symptoms developed slowly over a year and were impacting her job as a schoolteacher. Current lab data reveals blood urea nitrogen (BUN), creatinine, hemoglobin and urinalysis normal. The physical exam revealed blood pressure (BP) 94/50, heart rate (HR) 114, respiratory rate (RR) 22, and normal temperature. Sitting behind her on the examining table, auscultation of the lungs revealed no rales or wheezing, but a loud continuous bruit over the lower left lung area and costo-vertebral angle. There was a small atrophic scar, in the shape of a cross. When asked if that was a tattoo, she said that it was the site of a kidney biopsy many years prior. A CT of the abdomen and pelvis (CT-AP) with contrast revealed a large arterio-venous fistula (A-V fistula) in the left kidney, obviously created by the trauma of the kidney biopsy. [7] She was referred to interventional radiology for embolization of the A-V fistula. Within 10 days of embolization her symptoms abated, and she was relieved of high output heart failure.

Case 2

A 64-year-old female was referred for the evaluation of diabetic nephropathy, hypertension, edema, proteinuria and occasional microhematuria on urinalysis, which was ascribed to atrophic vaginitis. She had T2DM for over 20 years, mild retinopathy, and peripheral neuropathy. Her blood glucose and glycohemoglobin (HgbA1C) was tightly controlled by her endocrinologist, and blood pressure was treated with a calcium channel blocker and diuretic. She denied chest pain, hemoptysis, or g.i complaints. But over the preceding 2 months she developed dyspnea on exertion, new onset edema, a rapid thready pulse and decreased physical activity. She had been to a "walk-in clinic" 10 days earlier for "cellulitis" of the right forearm and was taking amoxicillin, 500 mg, three times per day. At this visit, blood pressure on the left arm was 102/48, HR 104, RR 18, lungs clear, heart in rapid, regular rhythm. She had 1+ edema of the lower legs. Abdomen exam was negative. No rash present. Examination of the site of the "cellulitis" of the right arm revealed an enlarged, very warm, thick arm, but no redness, tenderness, or fluctuation. Touching a pulsatile "mass" in the forearm revealed a strong bruit and thrill. She was referred to a vascular surgeon after a CT of the arm revealed a hyper vascular mass. Biopsy of the mass revealed renal cell carcinoma. A CT of chest, abdomen and pelvis revealed a right renal mass, consistent with renal cell carcinoma. [8] The vascular mass in the forearm was a metastasis of renal cell carcinoma and was removed. 10 days later the right kidney was removed. Her symptoms of HOHF resolved within 2 weeks. Unfortunately, she developed pulmonary and cerebral metastases within 2 years and expired.

Case 3

A 50-year-old male had developed biopsy proven membranoproliferative glomerulonephritis in his late 20's. He progressed to End Stage Renal Disease (ESRD) and received hemodialysis via a left upper extremity A-V fistula. Four years later he received a deceased donor kidney transplant, which provided stable renal replacement function for the next 20 years. He currently has a stable serum creatinine of 1.8 mg/dl, consistent with stage 3A CKD. He was referred at this time for symptoms of dyspnea on exertion, tachycardia, weakness, and unable to walk 2 blocks. BP was 96/48, HR 124, lungs clear, no edema. He denied chest pain, fever, chills, nausea, vomiting, or diarrhea. Examining his left arm revealed a serpiginous, large, pseudo-aneurysmal fistula with intense thrill and bruit. It appeared to be another case of high output heart failure. Using the Nicoladoni-Branham sign, applying pressure to the distal forearm where arterial inflow to the fistula existed caused the thrill and bruit to immediately diminish, and the heart rate decreased to 96. This was consistent with an A-V fistula delivering excessive amounts of blood to the right ventricle. He subsequently had the fistula ligated with resolution of symptoms [5,9,10].

Summary

The three cases represent different scenarios for vascular-related high output heart failure. High Output Heart Failure occurs when a normal heart receives too much blood and must work overtime to pump the blood out to the periphery. There is increased right ventricular end diastolic pressure, wide pulse pressure, tachycardia-palpitations and normal or increased cardiac output.

In the three cases above (renal biopsy created, a hypervascular renal cell cancer metastasis, and a highly functioning A-V fistula), blood in an artery bypassed the normal "resistance vessels" or arterioles. The normal sequence of blood flow is artery → arterioles ("resistance vessels") → capillaries → venules → veins. The resistance vessels offer a slowing down of blood return to the venous system. But vascular anomalies bypass this circuitry, with blood going from the artery directly into the vein, bypassing the "resistance vessels / arterioles", delivering large amounts of blood rapidly to the right ventricle which must increase heart rate to maintain systemic BP. Similar cases are described after stab wounds or bullet wounds. HOHF from A-V fistulas or arterio-venous malformations are likely to be diagnosed during physical examinations. HOHF is not common but should be suspected in patients with "symptoms of heart failure" accompanied by prior trauma, persistent hypotension, wide pulse pressure, and warm extremities.

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