

## CLINICAL VIGNETTE

# High Output Cardiac Failure

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### *Introduction*

High output heart failure is a disease state in which the signs and symptoms associated with heart failure (i.e., dyspnea, lower extremity edema, pulmonary edema, etc.) are manifested in a state of increased cardiac output. The majority of heart failure cases are caused by decreased systolic function combined with low to normal cardiac output. However, in the case of high output cardiac failure, cardiac index is elevated. Some conditions that contribute to high output heart failure include pregnancy, hyperthyroidism, and chronic anemia.<sup>1</sup> Treatment of high output heart failure secondary to anemia focuses on treatment of underlying cause of anemia as well as diuretic therapy for volume overload.

### *Case Report*

The patient is a 48-year-old female with past medical history of severe mitral stenosis s/p mitral valve replacement, polycystic kidney disease s/p renal transplant, obesity s/p gastric bypass, and hypothyroidism who presented to her Internal Medicine Primary Care Physician with increasing weight gain, dyspnea on exertion, and lower extremity edema. She also noted weight gain of 16 lbs. in one week. She also reported intermittent chest tightness with accompanying abdominal swelling making it difficult to fit into her clothes. She admitted to medication non-adherence as she was instructed after mitral valve replacement to take oral furosemide as needed but was afraid to take furosemide due to concern it would injure her transplanted kidney. She denied paroxysmal nocturnal dyspnea or orthopnea.

The patient was advised by her PMD to resume Furosemide 60mg PO daily and to weigh herself daily, increasing administration of furosemide if she had weight gain of > 2 lb. over 24 hours. She began Furosemide 80mg/day two days later when she developed dyspnea at rest. She presented to her PMD again; at that time, she was found to be hypoxic with O<sub>2</sub> sat of 82% on room air. JVP was noted to be elevated, and she had mild bibasilar crackles. She was taken to the Emergency Room (ER). Initial labs in the ER revealed hemoglobin of 7 with her baseline hemoglobin of 10.5. The patient was admitted and given 1 unit of packed red blood cells split with Furosemide 40mg IV following each half unit of packed red blood cells administered. The patient's anemia was secondary to iron deficiency; therefore, in addition to packed red blood cells she was given parenteral iron repletion. Her volume overload was treated with Furosemide 40mg IV BID X 3 days, and she was discharged on Furosemide 40mg PO Qday with oral iron

supplementation. Her hemoglobin was stable upon discharge and work-up for the source of iron loss was deferred for outpatient follow-up.

### *Discussion*

High output heart failure caused by anemia rarely occurs unless there is underlying cardiac dysfunction. The presence of underlying cardiac disease such as current or past history of valvular disease or ventricular dysfunction is usually necessary to cause increased cardiac output in response to low hemoglobin, thereby leading to high output heart failure. Severe anemia such as hemoglobin less than 5 is needed to cause high output heart failure in a patient with previously normal cardiac function and no prior history of heart disease.

The presentation of high output heart failure secondary to chronic anemia is similar to heart failure from other causes with patients complaining of dyspnea, lower extremity edema, and decreased exercise tolerance. However, in addition to the above, physical exam findings are usually significant for widened pulse pressure, flow murmur at LSB, and prominent P<sub>2</sub>.

The mechanism of high output failure secondary to anemia has not been fully elucidated. It is thought to be related to increased myocardial work load.<sup>2</sup> Chronic anemia has been shown to be associated with chamber enlargement and subsequent ventricular dysfunction.<sup>3</sup>

Treatment involves determining the underlying cause of anemia. Patients should be evaluated for iron deficiency as well as vitamin B12 and folate deficiency. In addition, labs that assess renal function, inflammatory markers (CRP, ESR), reticulocyte count, and peripheral smear should also be obtained. Patients with chronic kidney disease and decreased erythropoietin are at-risk for development of high output heart failure.<sup>3</sup> However use of exogenous erythropoietin does not improve outcomes and increases the risk of venous thromboembolism. As such, use of erythropoiesis-stimulating agents is not recommended.<sup>4</sup> Treatment includes transfusion of packed red blood cells, administered in one-half units usually over 3-4 hours followed by diuretic therapy. If the cause of anemia is iron deficiency, parenteral iron infusion is preferred over oral administration and has been shown to improve symptoms, functional capacity, and quality of life.<sup>5</sup>

## Conclusion

The aforementioned patient had end stage renal disease s/p renal transplant as well as recently discovered severe mitral stenosis s/p mitral valve replacement. Both underlying conditions placed her at greater risk for development of high output heart failure secondary to anemia. Patients with prior history of valvular dysfunction and/or underlying renal dysfunction should have special attention paid to chronic anemia. Treatment with diuretic agents alone is insufficient to correct the heart failure. Care should be taken to replete blood loss even in a state of volume overload. Consideration should be made for iron repletion for patients with chronic iron deficiency anemia to avoid development of ventricular dysfunction. High output heart failure secondary to anemia, is important to identify as treatment of anemia is crucial to avoid further cardiac dysfunction.

## REFERENCES

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