

THAT FEELING WHEN YOUR HEART'S HARD WORK IS NOT ENOUGH: HIGH OUTPUT HEART FAILURE

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BACKGROUND

High output heart failure (HOHF) can be caused by chronic condition like severe anemia, hypoxia, obesity, and acutely by septic shock and hemorrhage. Basically, the heart is normal and there's no pump failure. The main problem is the underlying decrease in systemic vascular resistance due to different etiologies that drive-in low blood-pressure and activation of neuro-hormonal mechanisms that favors salt and water retention which cause HOHF [1,2].

It has been reported patients with HOHF displayed hyperdynamic hearts with higher ejection fraction, and eccentric ventricular remodeling and higher echocardiographic estimates of ventricular filling pressures (E/e' ratio), and higher estimated cardiac indexes on echocardiography. Patients with HOHF displayed 2-fold higher cardiac filling pressures coupled with markedly higher pulmonary artery pressures. Elevated cardiac output was related to a greater stroke volume compared with controls. A higher ventricular preload (increased left ventricular diastolic dimension), more complete emptying (higher EF), and lower arterial afterload (decreased systemic vascular resistance) drive the increased stroke volume [3].

CASE PRESENTATION

A 58-year-old obese male presents after developing shortness-of-breath at rest for two days. He reported diet and medications compliance. He has lower extremity edema. He was hypotensive at 83/57. Chest x-ray [Figure 1] revealed vascular congestion with cephalization. BNP was found to be elevated. EKG [Figure 2] and troponin were unremarkable. 2D echocardiography revealed hyper-dynamic left ventricular systolic function with ejection fraction of 84 %, normal left ventricular cavity size, moderately increased left ventricular wall thickness, moderate mid-cavitary obstruction at rest, severe obstruction with Valsalva (peak gradient 103 mmHg) [Figure 3] and moderate pulmonary hypertension, RVSP 63 mmHg.

FIGURE 1: CXR SHOWING VASCULAR CONGESTION WITH CEPHALIZATION AND DIFFUSE INTERSTITIAL INFILTRATE.



FIGURE 2: EKG REVEALING NORMAL SINUS RHYTHM WITH NONSPECIFIC INTRAVENTRICULAR CONDUCTION DELAY.

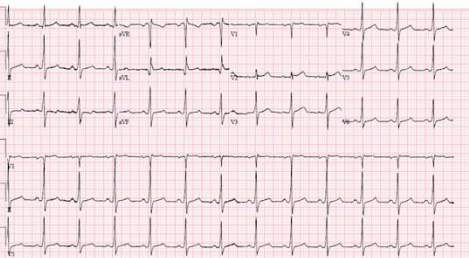
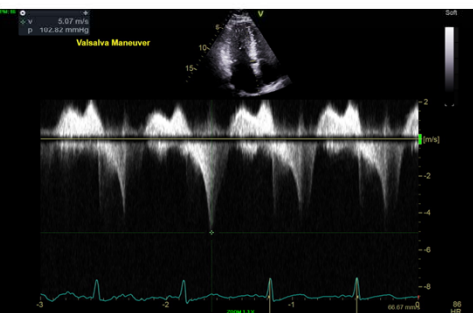


FIGURE 3: 2D ECHOCARDIOGRAPHY SHOWING MODERATE MID-CAVITARY OBSTRUCTION AT REST, SEVERE OBSTRUCTION WITH VALSALVA (PEAK GRADIENT 103 MMHG).



DISCUSSION

Patient has been admitted five times over the past four months with similar presentations. In each admission, he underwent aggressive diuresis and sent home with diuretics after diagnosis of diastolic heart failure. It was noted that patient developed chronic anemia four months before admission and had five subsequent admissions. During this admission, we noticed that even though he has been admitted multiple times at different health facilities, no one entertained the diagnosis of HOHF or worked him up for the anemia. He had been aggressively diuresed and sent home.

Patient's with HOHF can be categorized into two groups with respect to the mixed venous oxygen saturation. Those patients with high mixed venous oxygen saturation include arteriovenous fistula, cirrhosis, carcinoid syndrome, myeloproliferative disorders, hyperthyroidism, sepsis, beriberi. And, those patients with low mixed venous oxygen include morbid obesity, anemia, and chronic pulmonary disease. Our patient's estimated cardiac output was 7.6 L/min and estimated mixed venous oxygen saturation was 10.7%, all speaks for HOHF. The extremely low mixed venous oxygen saturation which is an estimate can be explained by his multiple risk factors including anemia, chronic hypoxemia secondary to tobacco abuse, and obesity. It has been demonstrated that patients with low mixed venous oxygen saturation extract significant amount of oxygen from the capillaries due to the relatively profound hypoxemia. The heart has to pump to accommodate the high oxygen demand state which creates functional out flow obstruction. Patients with outflow obstruction needs the preload to maintain the already compromised outflow open. It's with this basic understanding that patients with HOHF need very cautious and judicious diuresis. The balance between diuresis and avoiding exacerbating outflow obstruction is always a challenge for clinicians specially when the awareness is low for the pathogenesis of HOHF. We believe with the advance of medicine and the advent of the like of Ambulatory Heart Failure Monitoring devises patients would have better fluid status management and less admissions [4].

We were able to understand, diagnose and manage the patient accordingly. Patient cautiously diuresed and euvolemia achieved. Hematology was involved to address the anemia and extensively advised on lifestyle modifications including weight loss and cigarette smoking cessation.

CONCLUSION

This case illustrates patients with HOHF are overlooked and sometime aggressively diuresed which could be deleterious. It's recommended HOHF patients should be diuresed cautiously because preload is needed to avoid functional outflow obstruction. Management should focus on treating the underlying causes [2].

REFERENCES

1. Anand IS, Florea VG. High Output Cardiac Failure. *Curr Treat Options Cardiovasc Med* 2001, 3:2, 151-159.
2. Mehta P.A., Dubrey S.W. High output heart failure. *QJM: An International Journal of Medicine* 2009, 102:4, 235-241.
3. Yogesh N.V. Reddy, Vojtech Melenovsky, Margaret M. Redfield, Rick A. Nishimura, Barry A. Borlaug. High-Output Heart Failure A 15-Year Experience. *Journal of the American college of cardiology* vol. 68, no. 5, 2016
4. Alexander Muacevic and John R Adler, Muhammad A Mangi, Hiba Rehman, Muhammad Rafique, and Michael Illovsky. Ambulatory Heart Failure Monitoring: A Systemic Review. *Cureus*. 2017 Apr; 9(4): e1174.