Apical Ballooning and Cardiogenic Shock in Obstructive Hypertrophic Cardiomyopathy

Mark V. Sherrid, MD
Division of Cardiology
NYU Langone Health
New York, New York

Daniel G. Swistel, MD
Division of Cardiac Surgery
NYU Langone Health
New York, New York

Sandhya Balaran, MD
Department of Cardiovascular Surgery
Mount Sinai St. Luke's
New York, New York

To the Editor:

We were interested in the case report of Nalluri *et al.*[1] We would like to draw the authors' attention to our report of two patients with hypertrophic cardiomyopathy with known latent obstruction who developed apical ballooning and cardiogenic shock when obstruction became severe and unrelenting.[2] These two patients had shock that was unresolved by intravenous administration of copious fluids, β-blockade, and phenylephrine. Finally, both patients were taken to the operating room in extremis for relief of left ventricular (LV) outflow tract obstruction. Remarkably, in both cases apical ballooning and shock resolved almost immediately after cardiopulmonary bypass. Both patients are alive with normal LV function 10 and 7 years later.

We advanced an explanation for the observed LV dysfunction on the basis of prior research.[2-5] Milder degrees of systolic dysfunction are common in obstructive hypertrophic cardiomyopathy, even when the ejection fraction is normal. In the presence of significant LV outflow gradients, a midsystolic drop in LV Doppler ejection velocities and flow is observed at the entrance of the outflow tract. This has been termed the "lobster claw abnormality" because of its characteristic bifid appearance.[3] It is caused by an abrupt decline of LV longitudinal shortening due to afterload-mismatch.[4] It is compelling evidence of the deleterious mechanical effect of obstruction. Both the abrupt decline of longitudinal systolic shortening and the midsystolic drop in ejection velocities are reversed by relief of obstruction.[4] Ischemia and myocyte energy depletion contribute to LV systolic dysfunction. We posit that our patients' shock was a more severe manifestation of these same phenomena.

Could this be takotsubo cardiomyopathy caused by a neurohumoral mechanism? Prompt recovery within minutes of removal of afterload makes this unlikely.

REFERENCES

1. Nalluri N, Asti D, Anugu VR, Ibrahim U, Lafferty JC, Olkovsky Y. Cardiogenic shock secondary to takotsubo cardiomyopathy in a patient with preexisting hypertrophic obstructive cardiomyopathy. Cardiovasc Imaging Case Reports 2018;2:78-81.
2. Sherrid MV, Balaran SK, Korzeniecki E, Chaudhry FA, Swistel DG. Reversal of acute systolic dysfunction and cardiogenic shock in hypertrophic cardiomyopathy by surgical relief of obstruction. Echocardiography 2011;28:E174-9.
3. Sherrid MV, Gunsburg DZ, Pearle G. Mid-systolic drop in left ventricular ejection velocity in obstructive hypertrophic cardiomyopathy. J Am Soc Echocardiogr 1997;10:707-12.
4. Barac I, Upadya S, Pilchik R, Winson G, Passick M, Chaudhry FA, et al. Effect of obstruction on longitudinal left ventricular shortening in hypertrophic cardiomyopathy. J Am Coll Cardiol 2007;49:1203-11.
5. Sherrid MV, Wever-Pinzon O, Shah A, Chaudhry FA. Reflections of inflections in hypertrophic cardiomyopathy. J Am Coll Cardiol 2009;54:212-9.

https://doi.org/10.1016/j.case.2018.07.009

Conflicts of interest: The authors reported no actual or potential conflicts of interest relative to this document.