Perspective

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A Note on Hypertrophic Cardiomyopathy

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Perspective

Hypertrophic Cardiomyopathy (HCM) is a rather common condition seen by anaesthesiologists in the perioperative phase. HCM was thought to be a rare condition fifty years ago. However, our understanding and capacity to diagnose HCM patients has vastly increased in recent years. Patients with HCM have a wide range of genotypic and phenotypic characteristics. A subset of these patients does, in fact, have the HCM genotype but not the manifestation (left ventricular hypertrophy). Pharmacotherapy to control symptoms, implantable cardiac defibrillators to manage malignant arrhythmias, and surgical myectomy and septal ablation to reduce the left ventricular outflow obstruction are all options for these individuals. For the perioperative care of these patients, an accurate diagnosis is critical. Left ventricular hypertrophy, left ventricular outflow tract gradients, systolic and diastolic function, and mitral valve morphology and function are typically used to make the diagnosis. Cardiac magnetic resonance imaging can also be used for diagnosis.

Hypertrophic Cardiomyopathy (HCM) was first described in 1868, its functional consequences in 1957, Left Ventricular (LV) asymmetric and especially septal hypertrophy in 1958, and its familial nature in 1960.

Hypertrophic Cardiomyopathy (HCM) is characterised by asymmetrical LV thickening without chamber dilation that occurs in the absence of a known aetiology (e.g. aortic valvular stenosis [AS], hypertension), and is accompanied with myocardial fibre disarray:

1) Myocardial cell disarray, in which cells are arranged in a disordered pattern rather than in a typical parallel pattern.
2) Coronary microvasculature failure due to increased wall/lumen ratio; and
3) remodelling alterations are the key underlying structural abnormalities in HCM.

These changes can also affect myocytes, fibroblasts, and the interstitium, and are not restricted to areas of LVH and myocardial remodelling that occur as a compensatory mechanism. These changes take years to manifest before symptoms appear.

Impaired coronary reserve, diastolic dysfunction, supraventricular and ventricular dysrhythmias, and sudden death are all symptoms of HCM patients' disorganised myocyte pattern, increased wall/lumen ratio of coronaries, and remodelling alterations.

Treatment and outcomes—medical and surgical

The existence and amplitude of LVOTO in HCM patients have been shown to be independent predictors of sudden death and all-cause cardiac mortality.

Given the mechanisms underlying myocardial contraction (calcium ions binding to troponin C and excitation-contraction coupling), as well as the fact that obstruction in HCM is dynamic and lethal dysrhythmias are common, a variety of medical regimens have been used in these patients with the goal of altering the natural history.

Through negative inotropy, the purpose of medical care with these medications is to reduce or abolish the LVOT gradient. The majority of medications used to treat symptomatic HCM patients relieve symptoms by lowering or eliminating the LVOT pressure gradient. Because it reduces the dP/dT (change in pressure over time), -adrenergic blockade can help with dyspnea and angina symptoms. This, in turn, causes a decrease in LVOTO.

These drugs help reduce LVOTO during exercise by dampening the sympathetic response, making them useful for managing symptoms and lowering the risk of sudden cardiac death. The medications should be titrated depending on symptom alleviation and an endeavour to avoid any substantial drug-related side effects. Perhexiline, a myocyte energy supplement, has recently been proven to reduce diastolic dysfunction and symptomatology.

