Commentary

Psychosocial Impact of Hypertrophic Cardiomyopathy

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DESCRIPTION

Hypertrophic cardiomyopathy is a kind of cardiac disease that involves thickening of the heart muscle, stiffness in the left ventricle, and abnormalities in the mitral valve. HCM is a chronic, progressive condition with a hereditary aetiology that causes the heart muscle to thicken and contract more than normal, resulting in debilitating and severe symptoms. The most common cause of hypertrophic cardiomyopathy is faulty genes in the heart muscle. The walls of the heart chamber (left ventricle) contract harder and thicker than normal as a result of these genes. The stiffening of the thickened walls is a result of the thickening. With each heartbeat, the amount of blood taken in and pumped out of the body is reduced. Because the cause of hypertrophic cardiomyopathy is uncertain or can be linked to genetics, high blood pressure, or ageing, identifying a high-risk group is difficult. Chest pain, palpitations, shortness of breath, exhaustion, and syncope are some of the symptoms (fainting). Sudden cardiac death is uncommon in people with hypertrophic cardiomyopathy. However, in those under the age of 30, the illness is the most common cause of sudden cardiac death. Most cases of hypertrophic cardiomyopathy are hereditary. The most frequent type of hereditary heart disease is HCM. It can strike at any age, although most people are diagnosed in their forties. Beta-blockers, calcium channel blockers, and diuretics are medications that provide limited and variable relief from symptoms. They may aid function, but they may also have negative side effects. To treat the symptoms of hypertrophic cardiomyopathy and avoid further problems, medications such as beta-blockers, calcium channel blockers, and antiarrhythmic are frequently administered. A septal myectomy, in which a surgeon removes a small portion of the thickened septal wall to broaden the outflow tract (the path blood takes) from the left ventricle to the aorta, may be performed if symptoms continue. Open-heart surgery is required for septal myectomy. It is just a possibility for patients with obstructive HCM who are

experiencing significant symptoms. This procedure is usually intended for younger individuals and those whose medicines are not performing as effectively as they should. Part of the enlarged septum protruding into the left ventricle is removed by the surgeon. This enhances blood flow both inside and outside the heart. Another alternative is alcohol ablation, a cardiac catheterization treatment that involves injecting a small amount of pure alcohol into the septum, forcing it to shrink back to a more normal size and expanding the path for blood flow. This treatment involves injecting ethanol (a form of alcohol) through a tube into the tiny artery that delivers blood to the thickening heart muscle caused by HCM. These cells die as a result of the alcohol. The swollen tissue thins out and returns to its original size. With age, the risks and complications of cardiac surgery rise. As a result, in elderly individuals with concomitant medical issues, ablation may be preferred to myectomy. Mavacamten is a first-in-class cardiac myosin inhibitor that targets a protein that causes muscle thickening by inducing contractions. The therapy restores normal heart function by addressing the aetiology of HCM. Patients who received mavacamten improved their symptoms, physical function, exercise ability, and quality of life in previous trials, including PIONEER-HCM. Clinicians could also check for obstruction of the Left Ventricular Outflow Tract (LVOT), which is a sign of obstructive HCM.

CONCLUSION

Hypertrophic cardiomyopathy (HCM) is a chronic, progressive condition with a hereditary etiology. HCM causes the heart muscle to thicken and contract more than normal, resulting in debilitating and severe symptoms. Symptoms include palpitations, shortness of breath, exhaustion, and syncope. Open-heart surgery is required for septal myectomy. Mavacamten is a first-in-class cardiac myosin inhibitor that targets a protein that causes muscle thickening. The therapy restores normal heart function by addressing the aetiology of HCM.

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