Bulletin of Environment, Pharmacology and Life Sciences
Bull. Env. Pharmacol. Life Sci., Spl Issue [2] 2023: 455-460.

Online ISSN 2277-1808
Journal's URL:http://www.bepls.com
CODEN: BEPLAD

REVIEW ARTICLE **OPEN ACCESS**

Hypertrophic Cardiomyopathy in India: An Update

Abhijeet Shelke, Suhas Mule and Rjaehs Bobade
Department of Cardiology, Krishna Institue of Medical Sciences, Krishna Vishwa Vidyapeeth, Deemed To Be University, Karad

ABSTRACT

Studies have concluded that "the outcome of LV that is not enlarged has either a preserved or increased ejection fraction". Additionally, studies have revealed that hypertrophy is frequently associated with LVDD. The majority of individuals with this type of disease is typically have a mild progression for this condition. Thus, in our review, we were trying to discuss and review HCM in terms of genetics, pathology, clinical features, and management.
Key words: *LV, HCM, Genetic, Pathology, Clinical Feature, Management.*

Received 30.09.2023 Revised 25.10.2023 Accepted 31.11. 2023

INTRODUCTION

Studies have also shown that "hypertrophic cardiomyopathy (HCM) is a genetic disorder of the heart muscle cells. Here, cells of heart is bigger in size than it should be". Further research has shown that it is usually not symmetrical in shape, and it is the most significant involvement often occurring in the basal interventricular septum (IVS), which is next to the aortic valve (AV) due to the proximity of these two structures".[1] Studies have further concluded that, on occasion, it is localized to distinct MC locations, such as the apex, midportion, or posterior wall of LV. Other times, it also affects the whole LV. Studies have also concluded that, at the level of the cell, cardiac myocytes are hypertrophied and disorganized. They are also separated from one another by patches of interstitial fibrosis, which separates them from one another.[1]

Thus, studies concluded that HCM is a disease that does not have a definite pattern of distribution that can be determined based on the geographic location, ethnicity, or gender of the patient. Furthermore, studies showed that it has been estimated that the prevalence of HCM among adults in the general population ranges from 0.16% to 0.29%, which translates to around 1:625–1:344 people.[2,3,4,5] In addition, studies found that, when measured using ECG or another imaging technique, an adult patient's LVED wall thickness of greater than 13 mm is indicative of HCM.[2] Studies have further concluded that the diagnostic criteria should include a LVWT of less than 15 millimeters, in accordance with the European Society of Cardiology's recommendations.[6] Studies have concluded that even if it is therapeutically beneficial, its prevalence is based on the detection of HCM. Among them, studies showed that the age-dependent expression of HCM is one that stands out as especially notable. [7,8] Since the expression of HCM mutations, according to several studies, is dependent on age, it is expected that it would be higher in those who were older [1]. Authors have concluded through their study that "when more sensitive imaging methods & genetic testing were done in more members of the family then they found a significantly higher estimatation upto of 0.6% (1:167) ".[9,10,11] Although studies have also concluded that HCM may have a delayed onset, the diagnostic threshold is 13 mm. [8,12] Furthermore, studies have shown that, because of this, the individuals in issue may not get a diagnosis for HCM. On the other hand, HCM may be the result of phenocopy conditions, which may account for five percent to ten percent of clinically proven instances of HCM in children.[13,14,15] Studies have also found that it might be hard to tell the difference between primary (HCM) and secondary hypertrophy when there are other conditions going on at the same time that can cause HCM, like arterial hypertension or aortic stenosis.[1] These conditions include arterial hypertension, aortic stenosis, and narrowing of the aorta.[1] Thus, in our review we were trying to discuss & reviewed HCM.

GENETIC ASPECT

Studies have further concluded that, in this disorder single mutation is the main cause of illness, despite the variable penetrance and expression of the condition. However, studies have also concluded that this

does not rule out the possibility of many mutations simultaneously producing the disease.[16] Studies have also concluded that the phenotype may vary quite a bit from one individual to the next as a result of the interplay between the mutation that caused the condition and a wide variety of other genetic and nongenetic factors. Studies have also concluded that about sixty percent of patients who have been diagnosed with HCM have a clear-cut condition that runs in their families.[16] Additionally, studies have also concluded that there have been reports of autosomal recessive inheritance as well as X-linked inheritance, although neither type of inheritance is very prevalent which increases the likelihood of phenocopy condition.[17,18,19] In addition, studies also concluded that syndromic conditions like Noonan syndrome(NS) and Anderson-Fabry disease(AFD) may also manifest themselves.[19,20,21] Furthermore, studies also concluded that there is no investigation into the phenocopy conditions, which are sometimes commonly referred to as the gene copy conditions.[19] Additionally, studies have revealed that HCM has a complicated genetic and molecular foundation, which Christine and Jonathan Seidman's ground-breaking study has largely shed light on. In a study, the finding of the p.Arg403Glu mutation in the MYH7 gene opened the way for important discoveries that followed.[22] Studies have also concluded that these findings were made after the p.Arg403Glu mutation was found.[23] Studies have also been shown through studies that HCM is a genetically varied disease. This is because different mutations have been found in the key causal genes, which all code for sarcomere proteins. This was accomplished as a result of the finding that HCM is a genetically diverse disease.[23] Among the causal genes that have so far been identified, MYH7 and MYBPC3 (myosin-binding protein C) are the two that are responsible for the vast majority of cases. Studies have also concluded that they are responsible for approximately half of the patients who have been diagnosed with familial HCM."[24,25,26,27] Researchers have also found that "mutations in TNNT2, TNNI3 (cardiac troponin I), and TPM1 (tropomyosin) are very rare alomst 10%." [26,27,28,29] "Studies have shown that , other changes like in ACTC1 (cardiac-actin), MYL2 (myosin light chain 2), MYL3 (myosin light chain 3), and CSRP3 (cysteine and glycine-rich protein 3) have been found to be the reasons for it."[30,31,32]

PATHOLOGY [30,31,32]

Additionally, studies have revealed that to categorize the mechanical processes that take place in HCM, one of four different sets of interlocking mechanisms may be employed. The mutation is by far the most important concern. Additionally, studies have found that "initial or proximal phenotypes are those that are the direct result of phenotypes produced by the mutations on the structure and function of the sarcomere proteins."[30] Studies have also found that these phenotypes are those that are the result of those phenotypes that are directly a result of the mutations. Studies have also found that the intermediate (or secondary) phenotypes include the changes that happen at the molecular level because of changes made to the structure and function of the sarcomere protein. Examples of the latter kind of impact include changes in gene expression as well as the activation of signaling pathways like the MAPK and TGFB1 pathways.[31] Both of these are examples of epigenetic effects. These phenotypes are the result of the disruption of a large variety of secondary molecular processes in the heart, such as the activation of the HSP. These phenotypes are the result of the disturbance of a wide variety of secondary molecular processes.[32] The clinical phenotypes of HCM are the result of the molecular and histological changes outlined in the preceding paragraphs (quaternary). Studies have also found that it is important to remember that there is a difference in how HCM cases caused by a sarcomere protein mutation work compared to conditions that look like these cases. Furthermore, studies have also concluded that "VH results from storage of materials such as glycogen , functional problems in myocytes, such as decreased contraction in cases of HCM; however, mutations of this kind are rather uncommon." [30,31,32]

Figure 1 is showing that "mutation in the sarcomere, which is comprised of Z disks, thick filaments, and thin filaments, is the primary source of the problem. A primary defect may take the form of a mutation in the amino acid sequence of the sarcomere protein or the absence of a sarcomere protein. This can then lead to a series of initial (or proximal) defects, such as changed levels of the sarcomere protein, calcium sensitivity, or ATPase activity. Studies have also found that these first problems cause a chain of intermediate or secondary changes at the molecular level, such as altered transcriptomics or signaling pathways, which lead to the expression of those changes. These changes may be attributed to the initial defects. Furthermore, studies have found that myocyte hypertrophy and fibrosis are two examples of the histological and morphological changes that the second set of molecular changes that take place in the myocardium may be responsible for. In addition to this, studies have concluded that tertiary phenotypes are possible terms for these changes. The clinical manifestations of HCM are often referred to as quaternary phenotypes. These clinical manifestations are the end result of the molecular and histological changes that occur during HCM. Heart failure and cardiac arrhythmias are two examples of these conditions".[1]

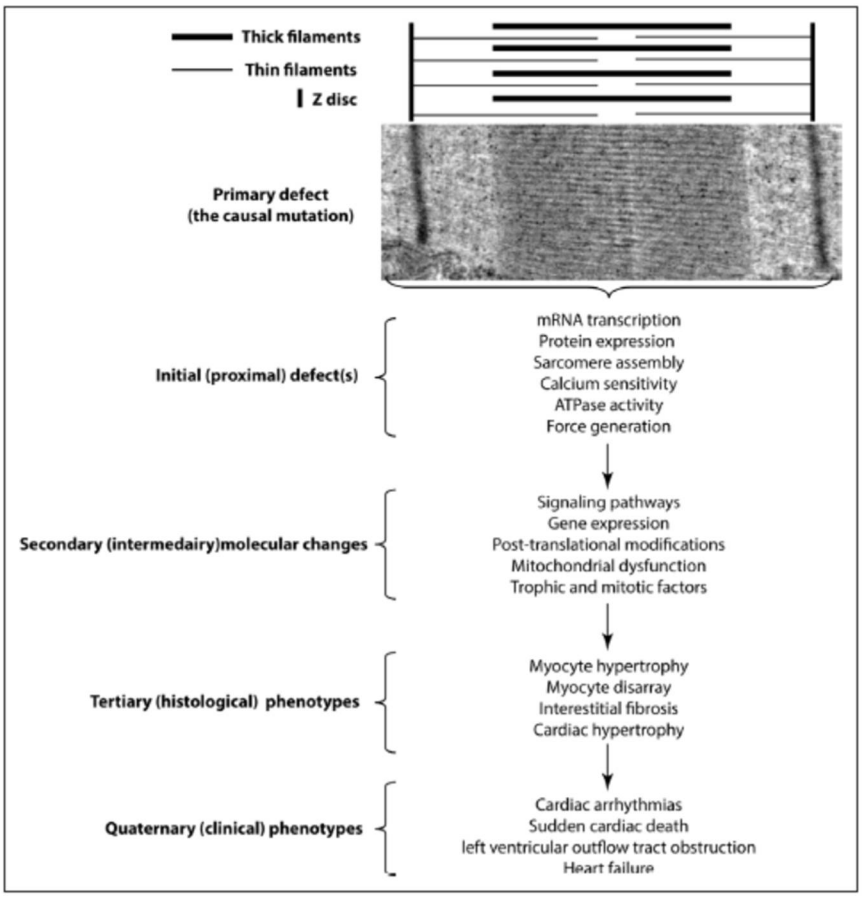

FIGURE 1: PATHOGENESIS

CLINICAL FEATURE [1]

Studies have also concluded that 4 major pathophysiologic condition are as follows:-

1. Diastolic Ventricular Dysfunction (DVF)
2. Obstruction to LVOT
3. Imbalance between Myocardial Oxygen Supply (I-MOS)
4. Cardiac Arrhythmia(CA)

1. DVF

Researchers have also found that this common form of HCM [33] raises the LVEDP [34], which in turn raises the pressures in the left atrium, pulmonary veins, and pulmonary capillaries.[35] Furthermore, studies revealed that an increase in the LVDP may cause a variety of symptoms, such as orthopnea, peripheral edema, exercise intolerance, and exertional dyspnea. It is also possible for this to bring on HF despite the heart's ejection fraction being normal.[1]

2. LVOT Obstruction

Researchers have also found that when these patients engage in physical activity, the obstruction of the LV outflow tract, often known as the LVOT, becomes much worse. Studies also concluded that patients who have been diagnosed with HCM have an obstruction of the LVOT while they are at rest, which accounts for around one-third of all cases. When the patient is at rest, studies have shown that the LVH that occurs in the other third of patients is not provokable and does not produce obstruction. This is in contrast to the first one-third of patients who have a provokeable obstruction.[36,37,38] Studies have further concluded that patients with severe obstruction almost always suffer from EVDP as well as exertional dyspnea. Some of these patients go on to develop the most severe form of HF as a direct result of their condition. Studies have also found that severe obstruction, with or without ventricular arrhythmia, may cause exertional or immediately postexertional syncope.[38]

3. MOS

Researchers have also discovered that patients with HCM frequently report experiencing ischemic chest pain (ICP).[39] This discomfort may or may not be defined by the symptoms that are typically associated

with angina pectoris. Studies also proved that this symptom occurs when there is an imbalance between the MOS and the demand for oxygen. This happens because less blood flows through the thick-walled, intramural coronary arteries, which have narrowed lumens and walls that have thickened in the myocardium. [39]

4. CA

Researchers have also found that recurrent nonsustained ventricular tachycardia frequently causes palpitations, presyncope, and syncope, some of the cardinal clinical signs. Studies have also concluded that "it is not uncommon for patients to have SV and ventricular ectopic beats(VEB), and nonsustained ventricular tachycardia (NVT) may be seen in 20% to 30% of patients". [40,41] Studies have also found that "nonsustained VT is a major risk factor for SCD because it can lead to VF, which is the most common cause of the condition.[40,42,43] Furthermore, studies have shown that a severe LVOT obstruction may also be the cause of syncope.[42,43,44,45] Additionally, studies concluded that HCM is characterized by ventricular arrhythmias(VA), the underlying mechanisms of which are mainly unclear. The "VR that is linked with HCM, interstitial fibrosis, myocardial ischemia, and myocyte disarray are all potential mechanisms".[1]

MANAGEMENT [1]

1. Clinical Assessment [1]

Researchers have also found that the assessment approach for patients with HCM includes a thorough physical examination, a 12-lead ECG, and extensive echocardiographic testing as part of the process. Cardiopulmonary exercise testing, MRI, and cardiac rhythm monitoring are all elective treatments; nevertheless, the latter is useful in patients who may have a lesser tolerance for exercise. It is recommended that patients, including those who are asymptomatic, have frequent reevaluations of their conditions. Since they are symptomatic or only have minor symptoms, the great majority of patients with HCM do not need pharmacological treatment.[1]

2. Pharmacotherapy [1]

Also, researchers have found that "beta-adrenergic receptor blockers, which don't have any intrinsic sympathetic activity, were first used to treat HCM in the 1960s.[1] Since that time, studies have concluded that "they have remained the foundation of the pharmaceutical treatment of symptomatic patients. They are effective in the alleviation of ICP and may reduce exercise-induced LVOT obstruction and the dyspnea that results from it as well".[1] Researchers have also found that taking the negative inotropic drug disopyramide with a beta-blocker may make the symptoms of people who have LVOT obstruction even worse. Furthermore, studies concluded that in "patients who are unable to take beta-blockers or who do not react to them, L-type calcium channel blockers, such as verapamil or diltiazem, may be helpful".[1] Furthermore, studies revealed that "in order to prevent hypovolemia, hypotension, and the development or provocation of LVOT obstruction, diuretics may be administered in patients with HCM, pulmonary congestion, and outright heart failure. However, minimum effective dosages and careful monitoring are necessary". In addition, studies have also concluded that the most effective treatment for newly diagnosed cases of atrial fibrillation is cardioversion. Anticoagulation treatment for the long term is required since both chronic and paroxysmal atrial fibrillation are considered to be risk factors for thromboembolism".[1]

CONCLUSION

We've come to the conclusion that since HCM was first classified more than 50 years ago, genetic testing, cardiac imaging, the prevention of severe arrhythmias, cardiac surgery, and interventional cardiology have all made progress in helping doctors find and treat people with HCM. Because of this advancement, we now have access to superior treatment alternatives. It is feasible that greater annotation of the human genetic variants and their variable relationship to clinical expression will make it simpler to identify people who carry dangerous variants. This would be a positive development in the field of human genetics. It will supply much-needed information on the prognosis of the expanding number of such people, many of whom are relatives of patients with HCM and who contain the same mutation but who do not seem to have any evident signs of HCM. In the not-too-distant future, people in this category could serve as test subjects for therapeutic interventions designed to either halt or significantly decrease the progression of phenotypic positive conversion.

REFERENCES

1. Marian, A. J., & Braunwald, E. (2017). Hypertrophic cardiomyopathy: genetics, pathogenesis, clinical manifestations, diagnosis, and therapy. Circulation research, 121(7), 749-770.

2. Maron, B. J., Gardin, J. M., Flack, J. M., Gidding, S. S., Kurosaki, T. T., & Bild, D. E. (1995). Prevalence of hypertrophic cardiomyopathy in a general population of young adults: echocardiographic analysis of 4111 subjects in the CARDIA study. Circulation, 92(4), 785-789.
3. Maron, B. J., Mathenge, R., Casey, S. A., Poliac, L. C., & Longe, T. F. (1999). Clinical profile of hypertrophic cardiomyopathy identified de novo in rural communities. Journal of the American College of Cardiology, 33(6), 1590-1595.
4. Hada, Y. (1987). Prevalence of hypertrophic cardiomyopathy in a population of adult Japanese worker as detected by echocardiographic screening. American Journal Cardiology, 59, 183-184.
5. Zou, Y., Song, L., Wang, Z., Ma, A., Liu, T., Gu, H., ... & Hui, R. (2004). Prevalence of idiopathic hypertrophic cardiomyopathy in China: a population-based echocardiographic analysis of 8080 adults. The American journal of medicine, 116(1), 14-18.
6. Muneretto, C., Elliott, P., Anastasakis, A., Borger, M., Borggrefe, M., Cecchi, F., ... & Watkins, H. (2014). 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy. European Heart Journal, 35(39), 2733-2779.
7. Charron, P., Dubourg, O., Desnos, M., Isnard, R., Hagege, A., Millaire, A., ... & Komajda, M. (1997). Diagnostic value of electrocardiography and echocardiography for familial hypertrophic cardiomyopathy in a genotyped adult population. Circulation, 96(1), 214-219.
8. Niimura, H., Bachinski, L. L., Sangwatanaroj, S., Watkins, H., Chudley, A. E., McKenna, W., ... & Bjornsdottir, H. (1998). Mutations in the gene for cardiac myosin-binding protein C and late-onset familial hypertrophic cardiomyopathy. New England Journal of Medicine, 338(18), 1248-1257.
9. Bick, A. G., Flannick, J., Ito, K., Cheng, S., Vasan, R. S., Parfenov, M. G., ... & Seidman, C. (2012). Burden of rare sarcomere gene variants in the Framingham and Jackson Heart Study cohorts. The American Journal of Human Genetics, 91(3), 513-519.
10. Maron, B. J., Peterson, E. E., Maron, M. S., & Peterson, J. E. (1994). Prevalence of hypertrophic cardiomyopathy in an outpatient population referred for echocardiographic study. The American journal of cardiology, 73(8), 577-580.
11. Semsarian, C., Ingles, J., Maron, M. S., & Maron, B. J. (2015). New perspectives on the prevalence of hypertrophic cardiomyopathy. Journal of the American College of Cardiology, 65(12), 1249-1254.
12. Niimura, H., Patton, K. K., McKenna, W. J., Soults, J., Maron, B. J., Seidman, J. G., & Seidman, C. E. (2002). Sarcomere protein gene mutations in hypertrophic cardiomyopathy of the elderly. Circulation, 105(4), 446-451.
13. Sachdev, B., Takenaka, T., Teraguchi, H., Tei, C., Lee, P., McKenna, W. J., & Elliott, P. M. (2002). Prevalence of Anderson-Fabry disease in male patients with late onset hypertrophic cardiomyopathy. Circulation, 105(12), 1407-1411.
14. Seo, J., Kim, M., Hong, G. R., Kim, D. S., Son, J. W., Cho, I. J., ... & Chung, N. (2016). Fabry disease in patients with hypertrophic cardiomyopathy: a practical approach to diagnosis. Journal of human genetics, 61(9), 775-780.
15. Chimenti, C., Pieroni, M., Morgante, E., Antuzzi, D., Russo, A., Russo, M. A., ... & Frustaci, A. (2004). Prevalence of Fabry disease in female patients with late-onset hypertrophic cardiomyopathy. Circulation, 110(9), 1047-1053.
16. Greaves, S. C., Roche, A. H., Neutze, J. M., Whitlock, R. M., & Veale, A. M. (1987). Inheritance of hypertrophic cardiomyopathy: a cross sectional and M mode echocardiographic study of 50 families. Heart, 58(3), 259-266.
17. Branzi, A., Romeo, G., Specchia, S., Lolli, C., Binetti, G., Devoto, M., ... & Magnani, B. (1985). Genetic heterogeneity of hypertrophic cardiomyopathy. International journal of cardiology, 7(2), 129-133.
18. Hartmannova, H., Kubanek, M., Sramko, M., Piherova, L., Noskova, L., Hodanova, K., ... & Kmoch, S. (2013). Isolated X-linked hypertrophic cardiomyopathy caused by a novel mutation of the four-and-a-half LIM domain 1 gene. Circulation: Cardiovascular Genetics, 6(6), 543-551.
19. Marian, A. J. (2016). Challenges in the diagnosis of Anderson-Fabry disease: a deceptively simple and yet complicated genetic disease. Journal of the American College of Cardiology, 68(10), 1051-1053.
20. Gelb, B. D., Roberts, A. E., & Tartaglia, M. (2015). Cardiomyopathies in Noonan syndrome and the other RASopathies. Progress in pediatric cardiology, 39(1), 13-19.
21. Elliott, P., Baker, R., Pasquale, F., Quarta, G., Ebrahim, H., Mehta, A. B., & Hughes, D. A. (2011). Prevalence of Anderson–Fabry disease in patients with hypertrophic cardiomyopathy: the European Anderson–Fabry Disease Survey. Heart, 97(23), 1957-1960.
22. Pare, J. A. P., Fraser, R. G., Pirozynski, W. J., Shanks, J. A., & Stubington, D. (1961). Hereditary cardiovascular dysplasia: a form of familial cardiomyopathy. The American journal of medicine, 31(1), 37-62.
23. Geisterfer-Lowrance, A. A., Kass, S., Tanigawa, G., Vosberg, H. P., McKenna, W., Seidman, C. E., & Seidman, J. G. (1990). A molecular basis for familial hypertrophic cardiomyopathy: a β cardiac myosin heavy chain gene missense mutation. Cell, 62(5), 999-1006.
24. Millat, G., Bouvagnet, P., Chevalier, P., Dauphin, C., Jouk, P. S., Da Costa, A., ... & Rousson, R. (2010). Prevalence and spectrum of mutations in a cohort of 192 unrelated patients with hypertrophic cardiomyopathy. European journal of medical genetics, 53(5), 261-267.
25. Kaski, J. P., Syrris, P., Esteban, M. T. T., Jenkins, S., Pantazis, A., Deanfield, J. E., ... & Elliott, P. M. (2009). Prevalence of sarcomere protein gene mutations in preadolescent children with hypertrophic cardiomyopathy. Circulation: Cardiovascular Genetics, 2(5), 436-441.
26. Erdmann, J., Daehmlow, S., Wischke, S., Senyuva, M., Werner, U., Raible, J., ... & Regitz-Zagrosek, V. (2003). Mutation spectrum in a large cohort of unrelated consecutive patients with hypertrophic cardiomyopathy. Clinical genetics, 64(4), 339-349.

27. Richard, P., Charron, P., Carrier, L., Ledeuil, C., Cheav, T., Pichereau, C., ... & Komajda, M. (2003). Hypertrophic cardiomyopathy: distribution of disease genes, spectrum of mutations, and implications for a molecular diagnosis strategy. Circulation, 107(17), 2227-2232.
28. Thierfelder, L., Watkins, H., MacRae, C., Lamas, R., McKenna, W., Vosberg, H. P., ... & Seidman, C. E. (1994). α-Tropomyosin and cardiac troponin T mutations cause familial hypertrophic cardiomyopathy: a disease of the sarcomere. Cell, 77(5), 701-712.
29. Kimura, A., Harada, H., Park, J. E., Nishi, H., Satoh, M., Takahashi, M., ... & Sasazuki, T. (1997). Mutations in the cardiac troponin I gene associated with hypertrophic cardiomyopathy. Nature genetics, 16(4), 379-382.
30. Mogensen, J., Klausen, I. C., Pedersen, A. K., Egeblad, H., Bross, P., Kruse, T. A., ... & Børglum, A. D. (1999). α-cardiac actin is a novel disease gene in familial hypertrophic cardiomyopathy. The Journal of clinical investigation, 103(10), R39-R43.
31. Poetter, K., Jiang, H. E., Hassanzadeh, S., Master, S. R., Chang, A., Dalakas, M. C., ... & Epstein, N. D. (1996). Mutations in either the essential or regulatory light chains of myosin are associated with a rare myopathy in human heart and skeletal muscle. Nature genetics, 13(1), 63-69.
32. Geier, C., Gehmlich, K., Ehler, E., Hassfeld, S., Perrot, A., Hayess, K., ... & Özcelik, C. (2008). Beyond the sarcomere: CSRP3 mutations cause hypertrophic cardiomyopathy. Human molecular genetics, 17(18), 2753-2765.
33. Stewart, S., Mason, D. T., & Braunwald, E. (1968). Impaired rate of left ventricular filling in idiopathic hypertrophic subaortic stenosis and valvular aortic stenosis. Circulation, 37(1), 8-14.
34. Sen-Chowdhry, S., Jacoby, D., Moon, J. C., & McKenna, W. J. (2016). Update on hypertrophic cardiomyopathy and a guide to the guidelines. Nature Reviews Cardiology, 13(11), 651-675.
35. Biagini, E., Spirito, P., Rocchi, G., Ferlito, M., Rosmini, S., Lai, F., ... & Rapezzi, C. (2009). Prognostic implications of the Doppler restrictive filling pattern in hypertrophic cardiomyopathy. The American journal of cardiology, 104(12), 1727-1731.
36. Braunwald, E., & Aygen, M. M. (1963). Idiopathic myocardial hypertrophy without congestive heart failure or obstruction to blood flow: Clinical, hemodynamic and angiocardiographic studies in fourteen patients. The American Journal of Medicine, 35(1), 7-19.
37. Maron, B. J., Rowin, E. J., Maron, M. S., & Braunwald, E. (2017). Nonobstructive hypertrophic cardiomyopathy out of the shadows: known from the beginning but largely ignored… until now. The American Journal of Medicine, 130(2), 119-123.
38. Maron, M. S., Rowin, E. J., Olivotto, I., Casey, S. A., Arretini, A., Tomberli, B., ... & Maron, B. J. (2016). Contemporary natural history and management of nonobstructive hypertrophic cardiomyopathy. Journal of the American College of Cardiology, 67(12), 1399-1409.
39. Maron, B. J., Wolfson, J. K., Epstein, S. E., & Roberts, W. C. (1986). Intramural ("small vessel") coronary artery disease in hypertrophic cardiomyopathy. Journal of the American College of Cardiology, 8(3), 545-557.
40. Monserrat, L., Elliott, P. M., Gimeno, J. R., Sharma, S., Penas-Lado, M., & McKenna, W. J. (2003). Non-sustained ventricular tachycardia in hypertrophic cardiomyopathy: an independent marker of sudden death risk in young patients. Journal of the American College of Cardiology, 42(5), 873-879.
41. Adabag, A. S., Casey, S. A., Kuskowski, M. A., Zenovich, A. G., & Maron, B. J. (2005). Spectrum and prognostic significance of arrhythmias on ambulatory Holter electrocardiogram in hypertrophic cardiomyopathy. Journal of the American College of Cardiology, 45(5), 697-704.
42. Elliott, P. M., Poloniecki, J., Dickie, S., Sharma, S., Monserrat, L., Varnava, A., ... & McKenna, W. J. (2000). Sudden death in hypertrophic cardiomyopathy: identification of high risk patients. Journal of the American College of Cardiology, 36(7), 2212-2218.
43. Christiaans, I., Van Engelen, K., Van Langen, I. M., Birnie, E., Bonsel, G. J., Elliott, P. M., & Wilde, A. A. (2010). Risk stratification for sudden cardiac death in hypertrophic cardiomyopathy: systematic review of clinical risk markers. Europace, 12(3), 313-321.
44. O'Mahony, C., Jichi, F., Pavlou, M., Monserrat, L., Anastasakis, A., Rapezzi, C., ... & Hypertrophic Cardiomyopathy Outcomes Investigators. (2014). A novel clinical risk prediction model for sudden cardiac death in hypertrophic cardiomyopathy (HCM risk-SCD). European heart journal, 35(30), 2010-2020.
45. Nienaber, C. A., Hiller, S., Spielmann, R. P., Geiger, M., & Kuck, K. H. (1990). Syncope in hypertrophic cardiomyopathy: multivariate analysis of prognostic determinants. Journal of the American College of Cardiology, 15(5), 948-955.

CITATION OF THIS ARTICLE

Abhijeet Shelke, Suhas Mule, and Rjaehs Bobade. Hypertrophic Cardiomyopathy in India: An Update. Bull. Env. Pharmacol. Life Sci., Spl Issue [2]: 2023: 455-460.