DOI: 10.7860/JCDR/2024/72561.19555

Case Report

Internal Medicine Section

Management of Hypertrophic Obstructive Cardiomyopathy with Concurrent Hypertension and Intracerebral Haemorrhage: A Case Report

SANGRAM MANGUDKAR[1], VARUN LINGINENI[2], VIJAYASHREE S GOKHALE[3], PONVIJAYA M YADAV[4], ANKIT SHOKEEN[5]

ABSTRACT

Hypertrophic Obstructive Cardiomyopathy (HOCM) is primarily a hereditary genetic disorder that presents difficulties in managing hypertension using a wide range of antihypertensive medications. It is crucial to steer clear of any substance that reduces preload or afterload in this situation, making beta-adrenergic receptor antagonists the ideal option. Hereby is a case of a 41-year-old who reported to the Medicine Outpatient Department (OPD) with complaints of weakness in the right upper and lower limbs for two days and altered sensorium. The patient was recently diagnosed with hypertension, and a Computed Tomography (CT) scan showed a left gangliocapsular bleed with a 3 mm midline shift. Echocardiography revealed HOCM. The patient was started on antioedema drugs and metoprolol. A repeat CT scan showed a decrease in the size of the bleed and surrounding oedema. The patient's consciousness improved, and blood pressure came under control. He was discharged with antihypertensives for regular follow-ups. Overall, this case underscores the complex interplay between HOCM and hypertension, the challenges in managing hypertension in HOCM patients, and the critical role of beta-adrenergic receptor antagonists in this setting. It highlights the need for a tailored approach to hypertension management and the importance of early recognition and appropriate intervention in patients with HOCM presenting with complications such as intracranial haemorrhage.

Keywords: Hypertrophic cardiomyopathy, Intracraial bleed, Metoprolol

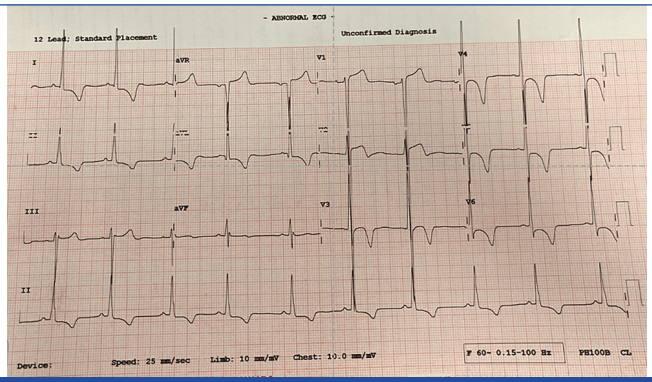
CASE REPORT

A 41-year-old male presented to the Medicine Outpatient Department (OPD) with a rapid onset of weakness in the right upper and lower limbs that started two days ago, accompanied by altered sensorium. There was no complaints of the patient experiencing fever, vomiting, vision impairment, or any other related cardiovascular or neurological symptoms. There were no documented instances of hypertension or diabetes mellitus, and there was no notable family history. Upon examination, the patient did not have a fever, his pulse rate was 74 beats per minute, and his blood pressure was 180/100 mmHg.

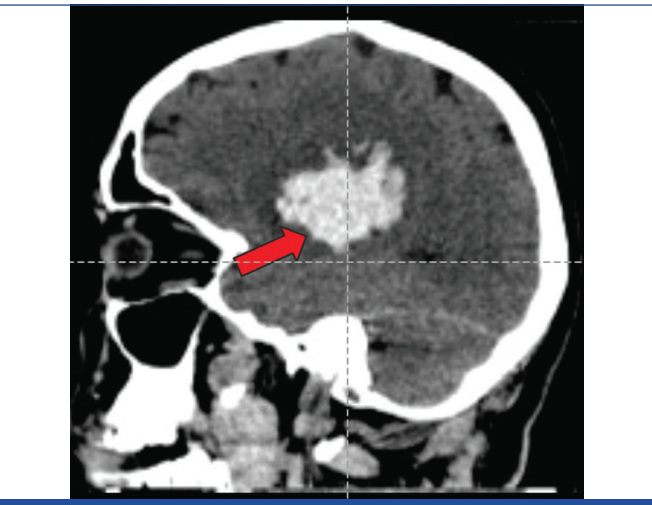
The physical examination was normal. Upon examination, the patient's Glasgow Coma score was E2V2M4, and a detailed neurological assessment suggested 0/5 power in the muscles of the right upper and lower limbs. The reflexes in the right-side upper and lower limbs were exaggerated, while the entire left-side was normal. Other Central Nervous System (CNS) examinations could not be performed due to the patient's uncooperative behaviour. Fundus examination revealed early changes of papilloedema. Routine blood investigations were normal. The Electrocardiogram (ECG) showed sinus tachycardia, a pattern of Left Ventricular Hypertrophy (LVH), and deep Q-wave and T-wave inversions noted in all leads [Table/Fig-1]. There was no systolic anterior motion of the leaflet.

The CT brain showed a well-defined hyperdense area (HU: 60-68) measuring approximately 36*50*27 mm Craniocaudal (CC) Anteroposterior (AP) Transverse (TR) in the left gangliocapsular region, thalamus, and corona radiata, suggestive of intraparenchymal haemorrhage [Table/Fig-2]. This was associated with significant perilesional oedema causing mass effect, resulting in effacement of surrounding sulci, the Sylvian fissure, the left lateral ventricle, and a midline shift of 3 mm to the right [Table/Fig-3].

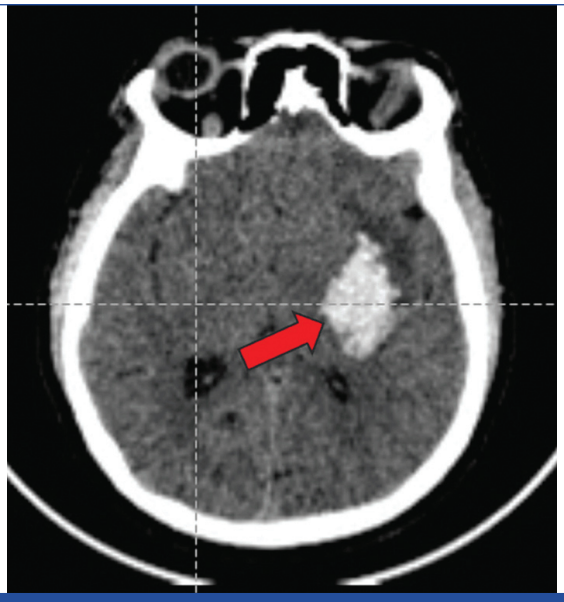
Echocardiography revealed moderate concentric Left Ventricular Hypertrophy (LVH) with Hypertrophic Obstructive Cardiomyopathy (HOCM) [Table/Fig-4]. The patient was treated with injections of



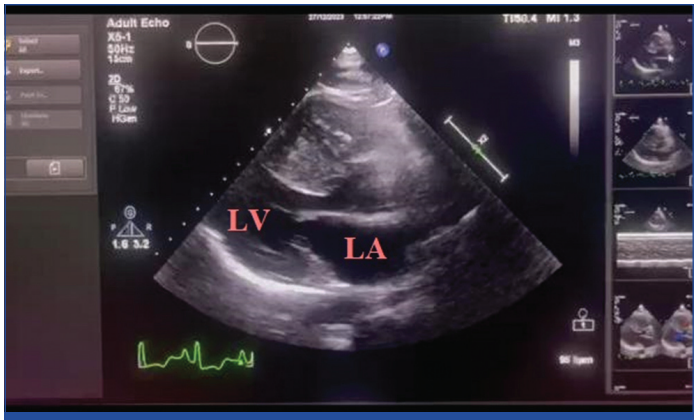
[Table/Fig-1]: ECG showed sinus tachycardia, LVH pattern, deep Q wave, and T wave inversion in all leads.



[Table/Fig-2]: CT brain (sagittal section), a well-defined hyperdense (HU: 60-68) area of approximate size 36*50*27 mm (CC *AP *TR) noted in the left gangliocapsular region, thalamus, and corona radiata suggestive of intraparenchymal haemorrhage.

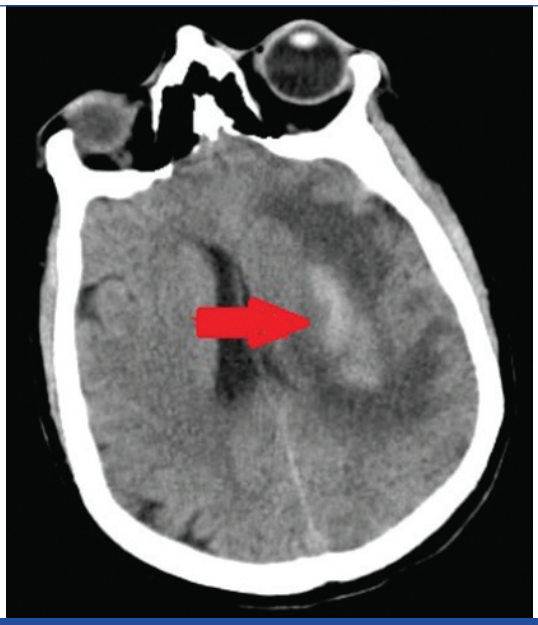


[Table/Fig-3]: CT brain (axial section), there is significant perilesional oedema with a mass effect in the form of effacement of the surrounding sulci, sylvian fissure, the left lateral ventricle, and a midline shift to the right.



[Table/Fig-4]: Echocardiography (Parasternal long axis view): showing concentric hypertrophy of left ventricle.

mannitol, levetiracetam, and metoprolol. A repeat CT scan showed a decrease in the size of the bleed to 21*18*20 mm (CC*AP*TR) and surrounding oedema [Table/Fig-5]. The patient demonstrated gradual improvement in consciousness, and the power of the right upper and lower limbs improved over two weeks. The patient is now on regular follow-up.



[Table/Fig-5]: CT brain (axial section): there is a significant decrease in the size of bleed and surrounding oedema when compared to [Table/Fig-1,2].

DISCUSSION

The HOCM is a genetically predisposed heart condition where the septal wall of the myocardium thickens, leading to Left Ventricular Outflow Tract (LVOT) obstruction due to systolic anterior motion of the mitral valve [1]. The prevalence of HOCM ranges from 0.02 to 0.2% in Western and Asian countries [2]. It was previously considered a genetically targeted disease but often remained undetected until symptoms such as heart failure, stroke, atrial fibrillation, or sudden cardiac death occurred [2]. Unfortunately, there have been no major randomised trials for the treatment of HOCM, and the consensus is to treat symptomatic patients only. Historically, HOCM was associated with a poor prognosis, including cardiac failure, thromboembolic events, malignant arrhythmias, and death [3,4]. However, in the past 20 years, advanced treatment options have significantly improved outcomes, allowing many patients to lead normal or extended lives with a better quality of life and reduced mortality rates associated with HOCM [5].

A recent study suggests that individuals with co-morbidities may experience a more significant impact on their survival than the effect of HOCM alone [6]. Hypertension is commonly found in 30 to 50% of patients with HOCM. Structural myocardial damage in patients with hypertensive cardiomyopathy and those with HOCM is similar but more prominent in HOCM. In the current case, the patient did not had any co-morbidities but presented with a hypertensive emergency [7]. This case involves newly detected hypertension alongside HOCM and a gangliocapsular bleed. Hypertension is widely recognised as one of the most prevalent cardiovascular disorders affecting approximately one billion individuals worldwide [8]. The co-existence of both conditions complicates the diagnosis and treatment process. Hypertension can lead to LVH due to increased strain on the heart, resulting in either concentric or eccentric enlargement, increased left ventricular contraction, and impaired diastolic function.

In addition to beta-blockers, various antihypertensive medications may not be recommended for patients with HOCM. Angiotensin Converting Enzyme (ACE) inhibitors and nitrates should be avoided as these medications can worsen Left Ventricular Outflow Tract (LVOT) obstruction by reducing afterload [1]. There were no significant differences in clinical outcomes, such as five year survival rates or cardiac death, among individuals with HOCM [9]. In a study by Luo Q et al., it was concluded that patients with HOCM and a history of hypertension had fewer syncopal episodes and a lower prevalence of sudden cardiac death in the family compared to those with HOCM without hypertension. Similar to this case report, index patient had no previous syncopal episodes and no significant family history [10]. Previous studies have shown no difference in the presence of LVOT obstruction between HOCM patients with and without hypertension, but the prevalence of LVOT obstruction was significantly higher in patients with hypertension [10,11]. In a study by Wang Z et al., it was found that another significant consequence of hypertension is a cerebral accident, leading to a gangliocapsular bleed on the right-side in patients with HOCM, as seen in index patient [12]. Beta-blockers and verapamil/diltiazem are the initial recommended medications for treating HOCM and hypertension. These medications help alleviate heart obstruction while regulating blood pressure. The five year survival rate in individuals with HOCM and hypertension is generally poor.

CONCLUSION(S)

Individuals diagnosed with HOCM may initially show no symptoms, although the first clinical signs may present as atrial or ventricular arrhythmias. Furthermore, the course of the disease can be further complicated by cerebrovascular accidents, often in the form of intracerebral bleeding. It is crucial to note that favourable outcomes can be achieved in patients with HOCM if they adhere to prescribed antihypertensive medications. Timely identification and

intervention play a crucial role in improving the overall prognosis of these patients. Therefore, early diagnosis and prompt initiation of appropriate therapeutic measures are essential components in the comprehensive management of HOCM, ensuring not only symptom relief but also optimising long-term health outcomes for affected individuals.

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PARTICULARS OF CONTRIBUTORS:

1. Professor, Department of Internal Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune, Maharashtra, India.
2. Resident, Department of Internal Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune, Maharashtra, India.
3. Professor, Department of Internal Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune, Maharashtra, India.
4. Resident, Department of Internal Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune, Maharashtra, India.
5. Student, Department of Internal Medicine, Dr. D. Y. Patil Medical College, Hospital and Research Centre, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Varun Lingineni,
D4 1906, Mahindra Antheia, Nehru Nagar, Pimpri, Pune-411018, Maharashtra, India.
E-mail: varunbhaskar004@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: May 01, 2024
- Manual Googling: May 16, 2024
- iThenticate Software: May 21, 2024 (11%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Apr 30, 2024**
Date of Peer Review: **May 14, 2024**
Date of Acceptance: **May 22, 2024**
Date of Publishing: **Jun 01, 2024**