First Colombian Experience with Mavacamten in Obstructive Hypertrophic Cardiomyopathy: A Brief Imaging Report

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Introduction

Hypertrophic cardiomyopathy (HCM) is a myocardial disease, usually of genetic origin, characterized by increased left ventricular wall thickness unexplained by abnormal loading conditions.[1] Dynamic left ventricular outflow tract (LVOT) obstruction occurs in approximately 70% of patients, either at rest or with provocation, and is closely associated with symptoms and adverse clinical outcomes.[2,3]

The relief of the obstructive gradient remains a major therapeutic goal in obstructive HCM. Mavacamten, a cardiac myosin inhibitor, has been shown to reduce LVOT gradients and improve symptoms and functional capacity in symptomatic obstructive HCM.[4,5] After its regulatory authorization in Colombia in August 2025, mavacamten became a new therapeutic option in this setting.

We present, to our knowledge, the first published Colombian case of obstructive HCM treated with mavacamten, with serial clinical and echocardiographic follow-up during the first 10 weeks of therapy.

Case Presentation

A 74-year-old man was referred for exertional dyspnea, chest pain, and presyncope for more than three months. On examination, blood pressure was 110/60 mmHg, heart rate was 84 beats/min, respiratory rate was 15 breaths/min, and oxygen saturation on room air was 96%. Cardiac auscultation revealed a mid-systolic ejection murmur that increased with Valsalva and transition from squatting to standing.

Transthoracic echocardiography showed asymmetric hypertrophy, predominantly involving the interventricular septum, with a diastolic septal thickness of 15 mm (Table 1). Left ventricular ejection fraction (LVEF) was 65%, whereas global longitudinal strain (GLS) was at the lower limit of normal, with segmental impairment involving the septal and lateral walls (Video 1). Elongated mitral valve leaflets with systolic anterior motion (SAM) were observed, with an end-systolic mitral coaptation-to-septum distance of 9 mm, suggesting a high likelihood of dynamic obstruction (Figure 1). Color Doppler showed LVOT flow acceleration, with a resting peak gradient of 18.3 mmHg that increased to 78.7 mmHg with Valsalva (Figure 2 and Video 2). Genetic testing identified a MYBPC3 variant.

Initial treatment with metoprolol succinate (100 mg daily) achieved heart rates of 55 to 60 beats/min but was poorly tolerated because of persistent symptoms, cold extremities, diaphoresis, and systolic blood pressure of 90 to 100 mmHg, limiting further optimization of conventional therapy. Because of persistent symptoms and dynamic LVOT obstruction, septal myectomy was considered. However, after a second opinion abroad, the patient started mavacamten 5 mg once daily, obtained in North America before local availability in Colombia. During follow-up, metoprolol was replaced with bisoprolol (5 mg daily) due to better tolerability.

After six weeks, echocardiography showed a modest reduction in septal thickness and indexed left ventricular mass, while LVEF and GLS remained stable (Table 1 and Video 3). SAM persisted, with continued LVOT flow acceleration. The resting peak gradient was 29.6 mmHg and increased to 39.6 mmHg with Valsalva (Figure 3). At that time, the patient was receiving bisoprolol (5 mg daily). Clinically, chest pain had resolved, dyspnea had improved, and no hypotensive episodes were reported.

At ten-week follow-up, while receiving mavacamten (5 mg daily) and bisoprolol (5 mg daily), the patient was asymptomatic, in New York Heart Association (NYHA) functional class I, and exercising regularly four times per week. A new echocardiography showed further reduction in septal thickness and indexed left ventricular mass (Table 1). The mitral coaptation-to-septum distance increased to 13 mm, consistent with a lower likelihood of dynamic obstruction, while LVOT flow acceleration markedly decreased. The resting peak gradient was 18.2 mmHg and did not increase with provocative maneuvers (Figure 4 and Video 4); LVEF and GLS remained stable (Video 5). Given the favorable clinical and hemodynamic response, the same regimen was continued, with periodic follow-up planned.

Discussion

This case is relevant not only because it represents, to our knowledge, the first published Colombian case of mavacamten use in obstructive HCM, but also because it illustrates appropriate real-world patient selection, limitations of conventional therapy, and the value of serial echocardiography for treatment decisions (Figure 5 and Table 1).

Keywords

Hypertrophic Cardiomyopathy; Ventricular Outflow Obstruction; Myosins; Echocardiography

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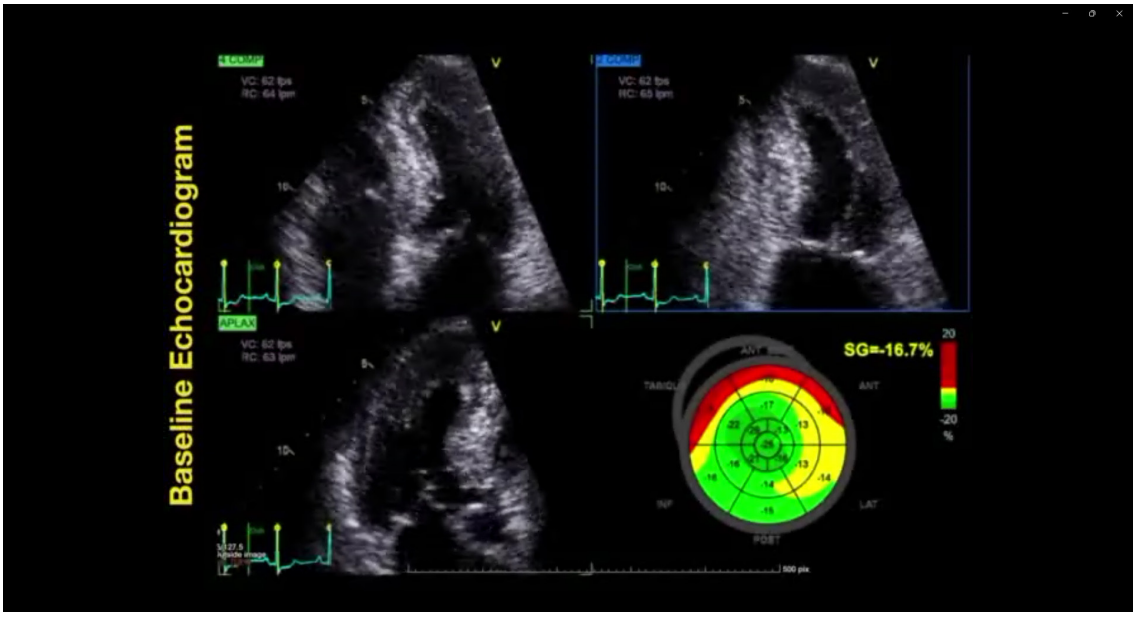


Video 1 – *Baseline left ventricular systolic function. Link: http://abcimaging.org/supplementary-material/2026/3903/2026-0061_video_01.mp4*

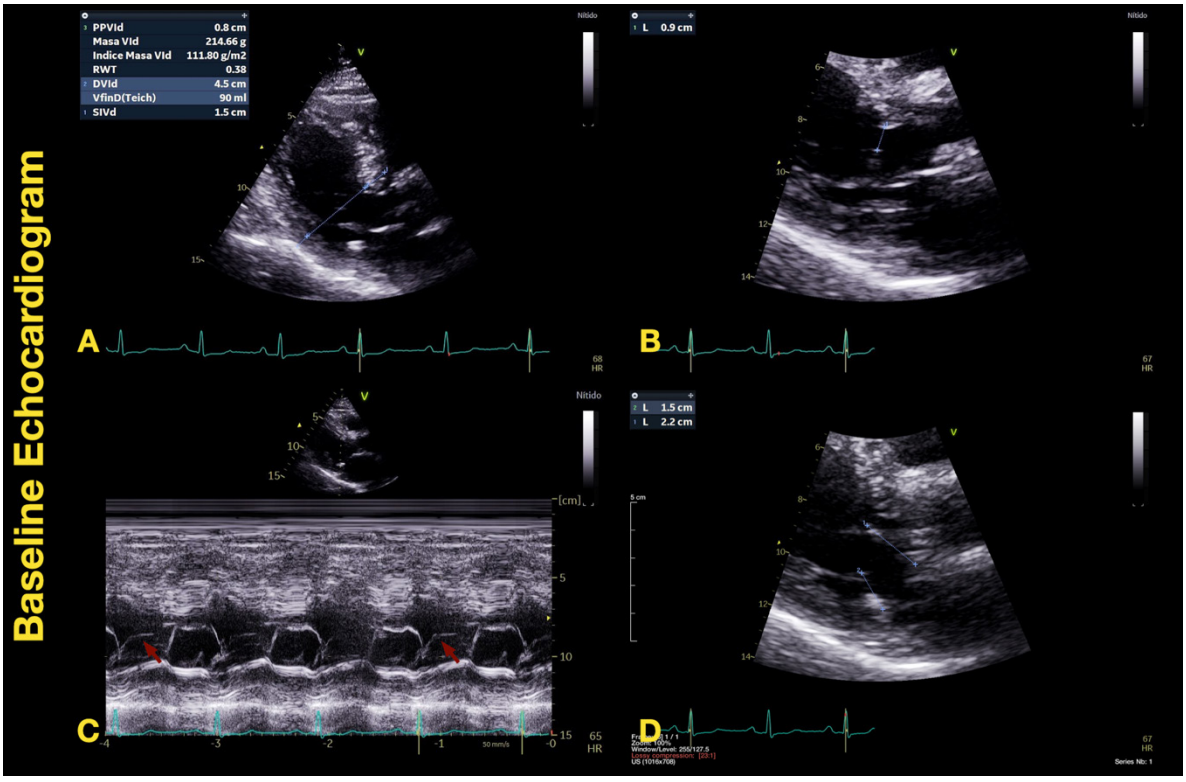


Figure 1 – *Baseline echocardiogram. A. Basal septal hypertrophy (15 mm) and mildly increased left ventricular mass (112 g/m²); B. Coaptation-to-septum distance of 9 mm; C. SAM on M-mode (red arrows); D. Elongated mitral leaflets.*

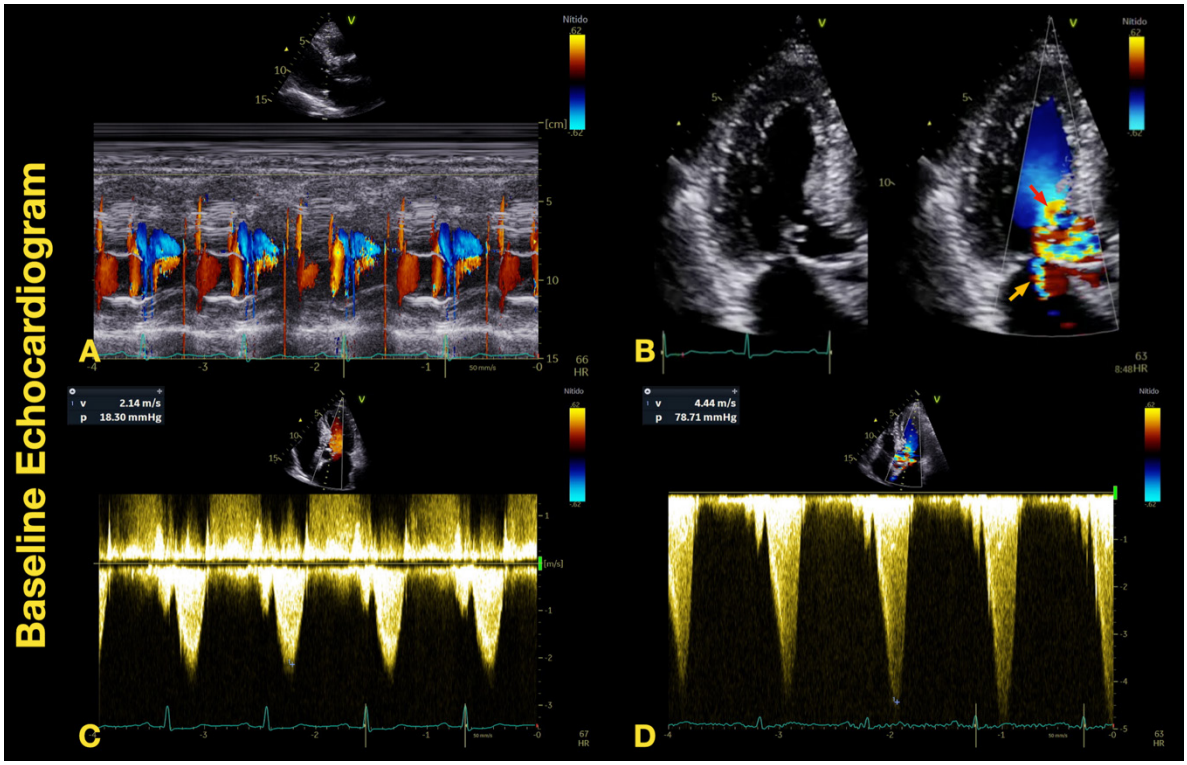
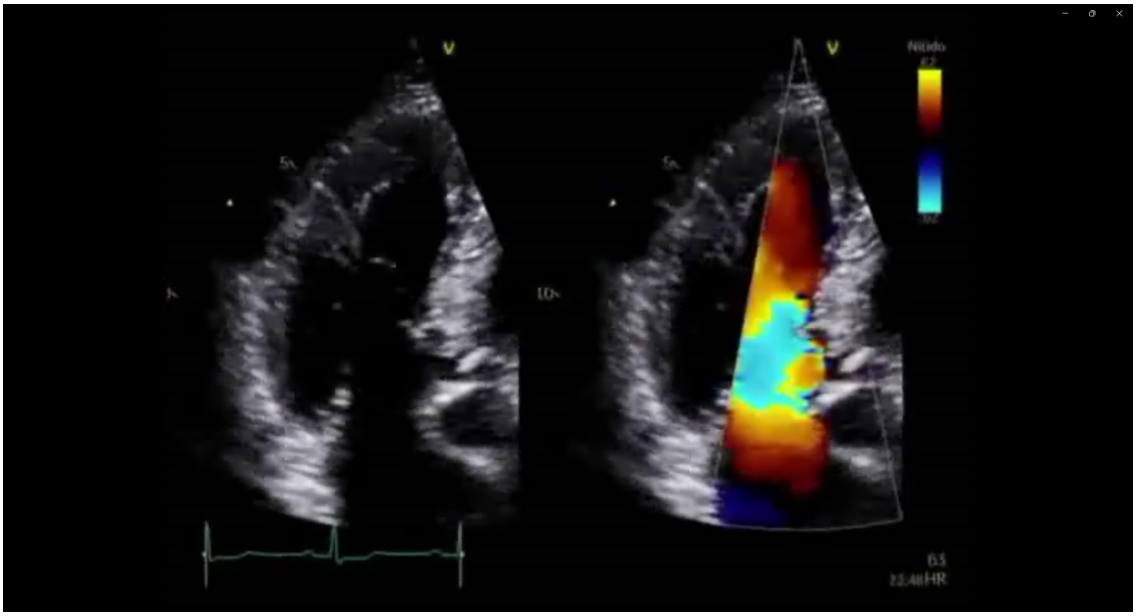


Figure 2 – *Baseline LVOT obstruction. A. Flow acceleration related to SAM; B. Apical 3-chamber LVOT flow acceleration; C. Resting peak gradient (18.3 mmHg); D. Valsalva peak gradient (78.7 mmHg).*

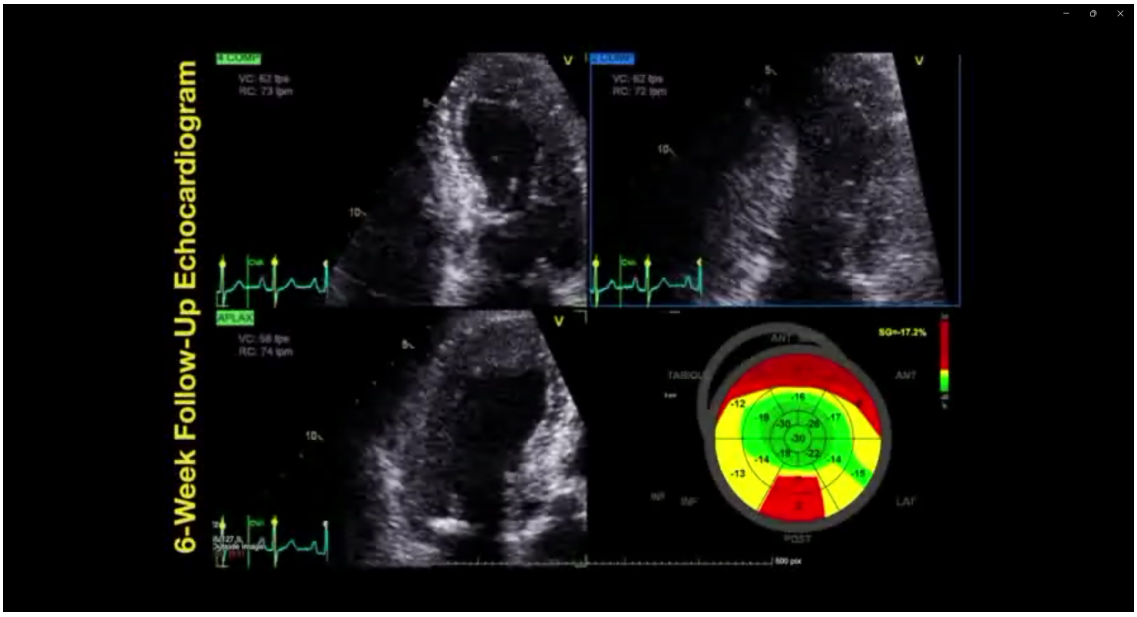


Video 2 – *Baseline LVOT obstruction. Link: http://abcimaging.org/supplementary-material/2026/3903/2026-0061_video_02.mp4*

Table 1 – Echocardiographic Variables Across Follow-Up

Parameter	Baseline echocardiogram	6-Week Follow-Up	10-Week Follow-Up
LV wall thickness (mm)	15	12	11
Indexed LV mass (g/m²)	112	81	79
LVEF (%)	65	66	60
GLS (%)	−16.7	−17.2	−16.9
Resting LVOT peak gradient (mmHg)	18.3	29.6	18.22
Valsalva LVOT peak gradient (mmHg)	78.7	39.6	18.62
Mitral-to-septum end-systolic distance (mm)	9	5	13

GLS: global longitudinal strain; LVEF: left ventricular ejection fraction; LV: left ventricular; LVOT: left ventricular outflow tract.



Video 3 – *Six-week follow-up echocardiogram. Link: http://abcimaging.org/supplementary-material/2026/3903/2026-0061_video_03.mp4*

A major clinical concern in HCM is its association with heart failure symptoms and sudden cardiac death. Among prognostic markers, maximal left ventricular wall thickness and dynamic LVOT obstruction are particularly relevant. In a meta-analysis including 12,146 patients, both variables were associated with cardiovascular death (Hazard Ratio [HR]: 1.42, 95%CI 1.06 to 1.89, and HR: 1.52, 95%CI 1.11 to 2.07, respectively) and sudden cardiac death (HR: 3.17, 95%CI 1.64 to 6.13, and HR: 2.41, 95%CI 1.55 to 3.73, respectively), underscoring the importance of recognizing and treating the obstructive phenotype.[6]

Mavacamten, the first myosin inhibitor approved for HCM, reduces excessive contractility, lessens dynamic LVOT obstruction, and has shown consistent benefits in symptomatic obstructive HCM, including improved symptoms, functional capacity, and quality of life under close echocardiographic monitoring.[5,7] A central teaching point of this case is the role of serial echocardiography during therapy. The follow-up showed early clinical and hemodynamic improvements, with progressive reduction in the provoked LVOT gradient, preserved LVEF, stable GLS, less prominent SAM, increased mitral coaptation-to-septum distance, and reduced LVOT flow acceleration. This behavior is consistent with results of the EXPLORER-HCM trial, in which mavacamten reduced post-exercise LVOT gradient by 36 mmHg *versus* placebo and improved symptoms, exercise capacity, and NYHA functional class.[8]

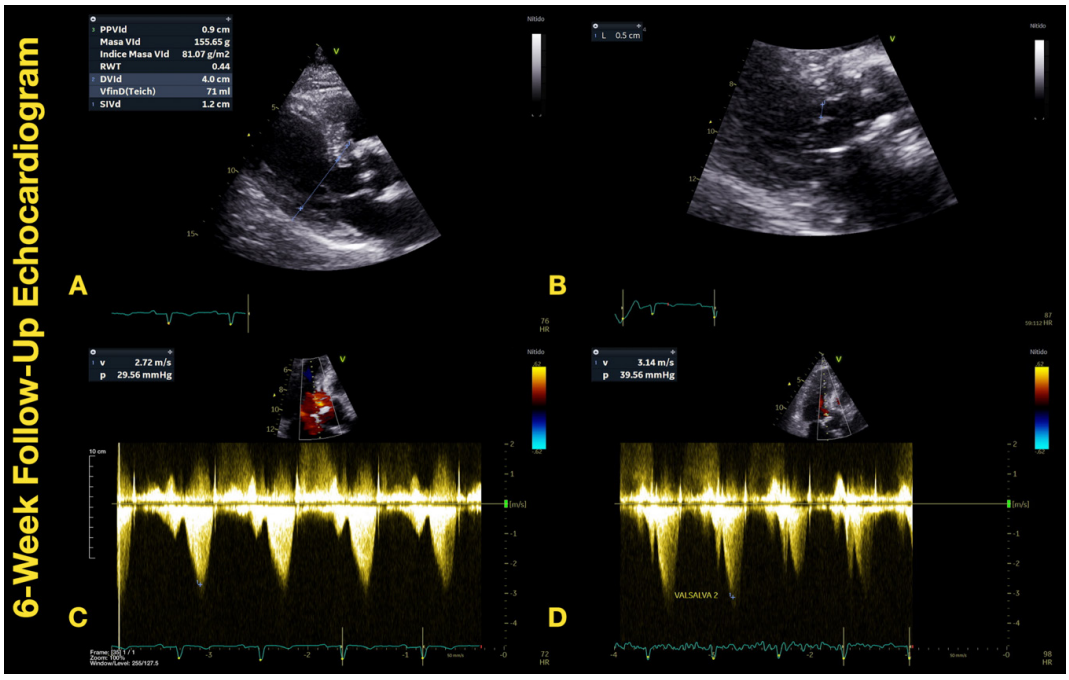


Figure 3 – *Six-week follow-up echocardiogram. A. Septal thickness (12 mm) and indexed left ventricular mass (81 g/m²); B. Coaptation-to-septum distance (5 mm); C. Resting peak gradient (29.6 mmHg); D. Valsalva peak gradient (39.6 mmHg).*

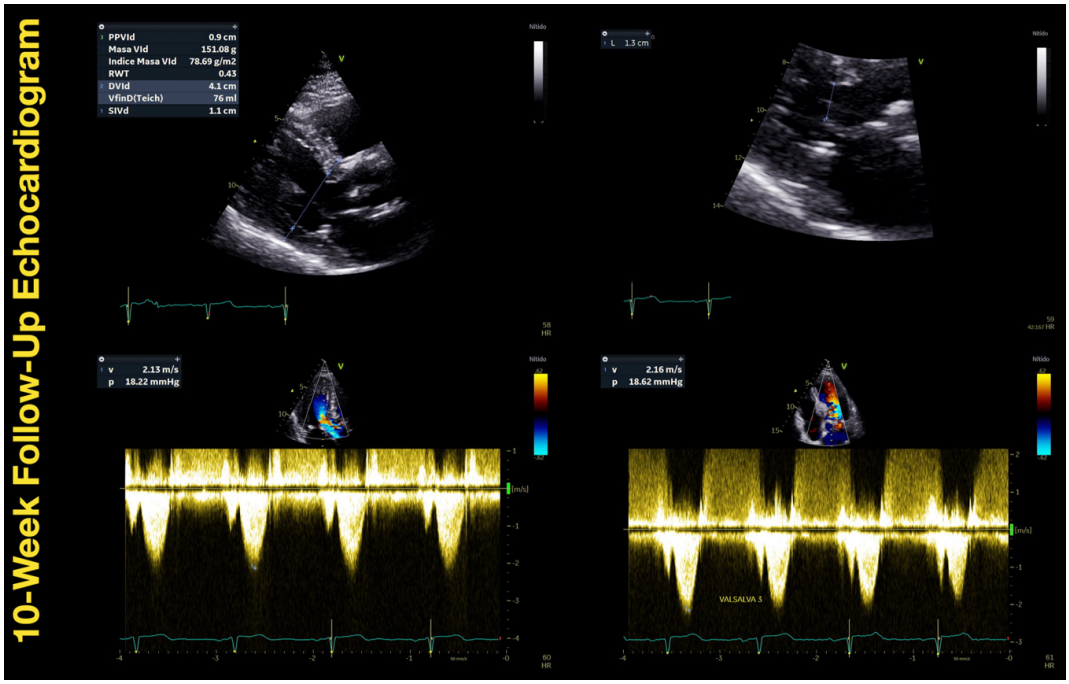
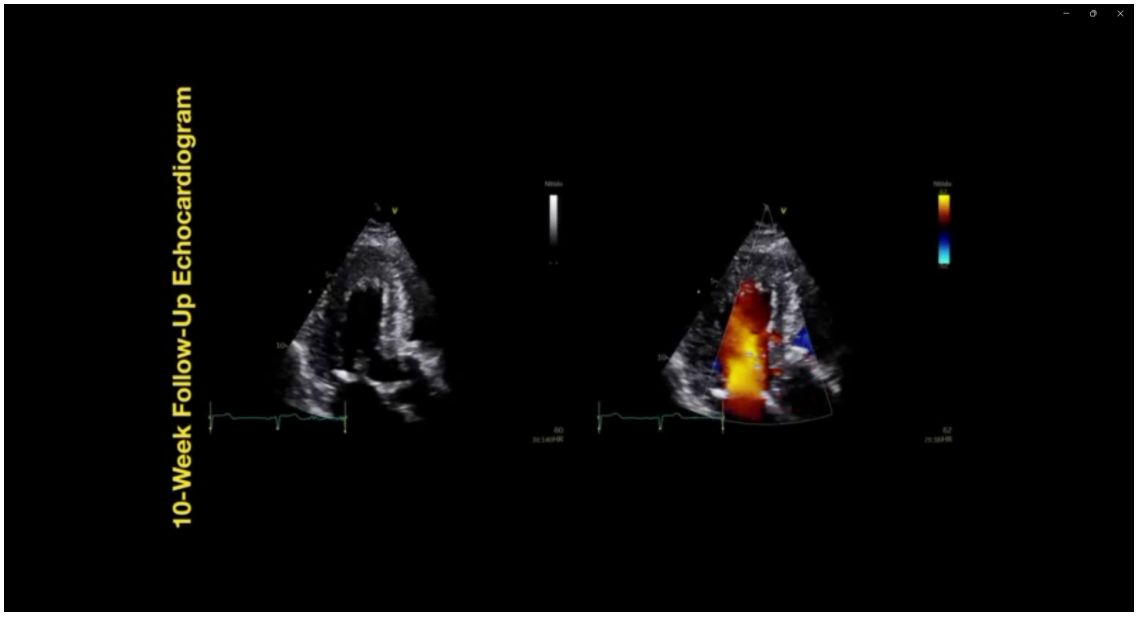
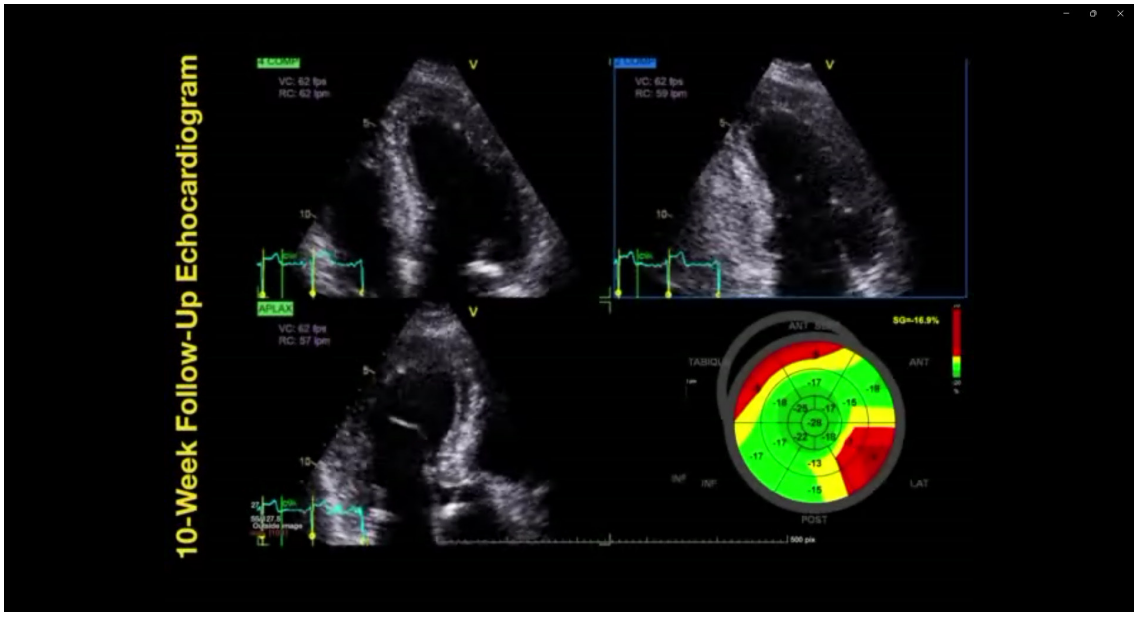


Figure 4 – *Ten-week follow-up echocardiogram. A. Septal thickness (11 mm) and indexed left ventricular mass (78.69 g/m²); B. Coaptation-to-septum distance (13 mm); C. Resting gradient (18.22 mmHg); D. Valsalva gradient (18.62 mmHg).*



Video 4 – *Ten-week follow-up echocardiogram. Link: http://abcimaging.org/supplementary-material/2026/3903/2026-0061_video_04.mp4*



Video 5 – *Ten-week follow-up left ventricular systolic function. http://abcimaging.org/supplementary-material/2026/3903/2026-0061_video_05.mp4*

Another interesting finding was the downward trend in septal thickness and indexed left ventricular mass. Similar structural changes have been reported in an EXPLORER-HCM substudy assessed by cardiac magnetic resonance.[9] In our patient, a comparable pattern was observed during follow-up. Although this is a single-case observation and should be interpreted cautiously, it remains illustrative.

From a practical standpoint, this case supports the concept that echocardiography-guided follow-up facilitates real-world initiation and monitoring of mavacamten in

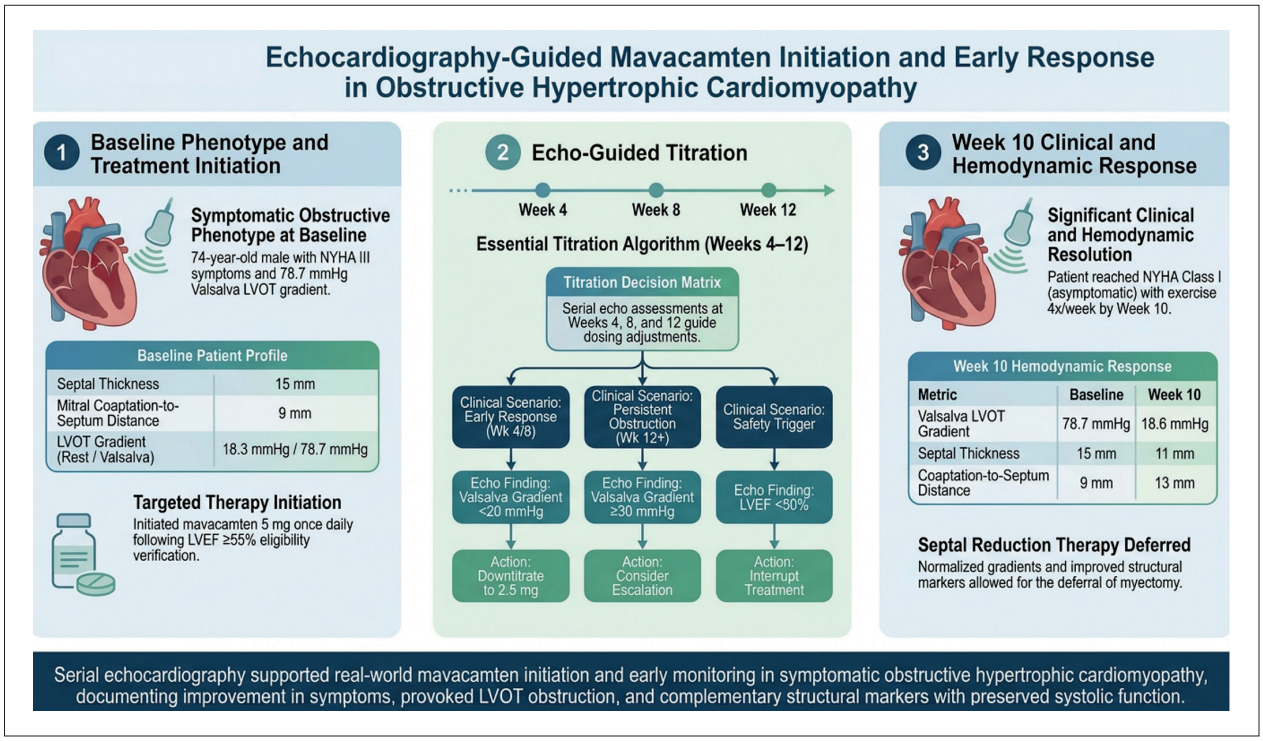


Figure 5 – *Echocardiography-guided initiation and early dose adjustment of mavacamten in a patient with symptomatic obstructive HCM, showing improved symptoms, reduced provoked LVOT gradient, and increased mitral leaflet–septal distance with preserved left ventricular systolic function. LVEF: left ventricular ejection fraction.*

selected patients with symptomatic obstructive HCM. In this setting, symptoms, LVEF, provoked LVOT gradient, SAM, and mitral coaptation-to-septum distance may provide a useful framework for longitudinal assessment.

Conclusion

This case illustrates that mavacamten may provide early clinical and hemodynamic benefits in selected patients with symptomatic obstructive HCM. In our patient, improvement in symptoms and LVOT obstruction occurred with preserved LVEF and stable GLS, supporting the value of an echocardiography-guided approach in real-world care.

Author Contributions

Conception and design of the research: Vasquez-Rodriguez JF, Idrovo-Turbay A, Reyes-Toledo RE, David-Pardo DG, Acuña-Olmos J; acquisition of data: Vasquez-Rodriguez JF, Idrovo-Turbay A, Acuña-Olmos J; analysis and interpretation of the data: Vasquez-Rodriguez JF, Reyes-Toledo RE; statistical analysis: Vasquez-Rodriguez JF; writing of the manuscript: Vasquez-Rodriguez JF, Reyes-Toledo RE, David-Pardo DG; critical revision of the manuscript for intellectual content: Vasquez-Rodriguez JF, Idrovo-Turbay A, David-Pardo DG, Acuña-Olmos J.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

This study received no external funding.

Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Fundación Cardioinfantil – Instituto de Cardiología under the protocol number 32-2026. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

During the preparation of this work, the author(s) used ChatGPT (OpenAI) for English language editing and grammar review, and to assist in creating the original Figure 5 layout from author-provided content and instructions. The author(s) reviewed and edited the content as necessary and assume full responsibility for the article's content.

Availability of Research Data

Data cannot be made publicly available because this manuscript is a single-patient case report. No additional research dataset was generated. Public sharing of patient-level data could compromise confidentiality despite de-identification. All data necessary to interpret the case are included in the manuscript.

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