Apical Hypertrophic Cardiomyopathy with Microfistulas: Correlation Between Coronary Flow Steal, Positive Inotropic Effect, and Normalization of T-Wave Polarity

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Introduction

Apical hypertrophic cardiomyopathy (HCM) with microfistulas is a rare condition, and the incidence of multiple microfistulas draining into the left ventricle (LV) ranges from 0.07% to 0.09%.[1, 2] Diagnosis can be established via contrast ventriculography, coronary angiography, and echocardiography.[3-6]

Two-dimensional echocardiography with color and pulsed-wave Doppler allows visualization of microfistula flow and measurement of diastolic flow velocity (DFV) using the same approach as for the left anterior descending artery (LAD).[7]

In apical HCM with microfistulas, electrocardiography (ECG) may show negative T waves of varying depths; this finding has been attributed to LV hypertrophy itself, myocardial ischemia, and coronary flow steal caused by the microfistulas.[8-10]

The interpretation of changes in T-wave polarity, whether at rest or under increased metabolic demand, remains controversial, limiting the utility of this finding as an indicator of ischemia.[11-13] In this context, negative T waves may become positive during physical or pharmacological stress echocardiography; however, the mechanism underlying this phenomenon remains uncertain.[14]

Case report

We report the case of a 61-year-old woman with hypertension and dyslipidemia who smoked and was undergoing irregular medical treatment. She sought cardiology assessment due to fatigue during strenuous exertion, without chest pain. She was in good general condition, with normal lung sounds, regular heart rhythm, a heart rate (HR) of 60 bpm, an audible fourth heart sound, no murmurs, and no edema; her blood pressure measured 190/90 mmHg. ECG showed sinus rhythm with T-wave inversion (Figure 1A). Echocardiography revealed marked HCM, predominantly apical, without dynamic obstruction. The patient was prescribed losartan, amlodipine, and a statin and advised to quit smoking.

Four months later, she was referred for dobutamine stress echocardiography to assess myocardial ischemia. At baseline, myocardial contractility was normal. The LAD was clearly visualized, and DFV measured 54 cm/s (Figure 2A). Multiple other flow signals were observed in the LV apical region, but DFV was not recorded.

During stress, DFV in the LAD was recorded at a HR of 127 bpm and measured 99 cm/s (Figure 2B), whereas color flow in the microfistulas appeared attenuated or suppressed. The test concluded at a HR of 151 bpm; no ischemic manifestations or arrhythmias occurred (Video 1), and the negative T waves seen on the baseline ECG became positive (Figure 1B). Despite these results, the attending physician requested coronary angiography, which showed normal, large-caliber coronary arteries; ventriculography was not performed at that time (Figure 3).

She remained stable over the following five years, despite reporting fatigue during strenuous exertion. She maintained controlled blood pressure, normal LV ejection fraction, and normal LV global longitudinal strain, with grade II diastolic dysfunction (Table 1). The ECG showed marked T-wave inversion (Figure 1C), with variations in T-wave depth compared to the ECG performed five years earlier. She was referred for repeat supine bicycle stress echocardiography; the test began with a HR of 62 bpm, reached a workload of 75 watts, and achieved a peak HR of 115 bpm. The test was terminated due to physical exhaustion; no arrhythmias or signs of myocardial ischemia occurred, and the T waves progressively became positive on ECG (Figure 1D).

The highest flow velocities were obtained by optimizing alignment with the Doppler cursor. At baseline, DFV measured 56 cm/s in the LAD and 140 cm/s in the microfistula (Figures 2C and 2D). The prominent diastolic flow through the microfistula (Figure 2E) was completely suppressed when the HR reached 108 bpm, whereas flow through the LAD remained clearly visible in the LV apical region (Figure 2F and Video 2).

Keywords

Hypertrophic Cardiomyopathy; Stress Echocardiography; Electrocardiography.

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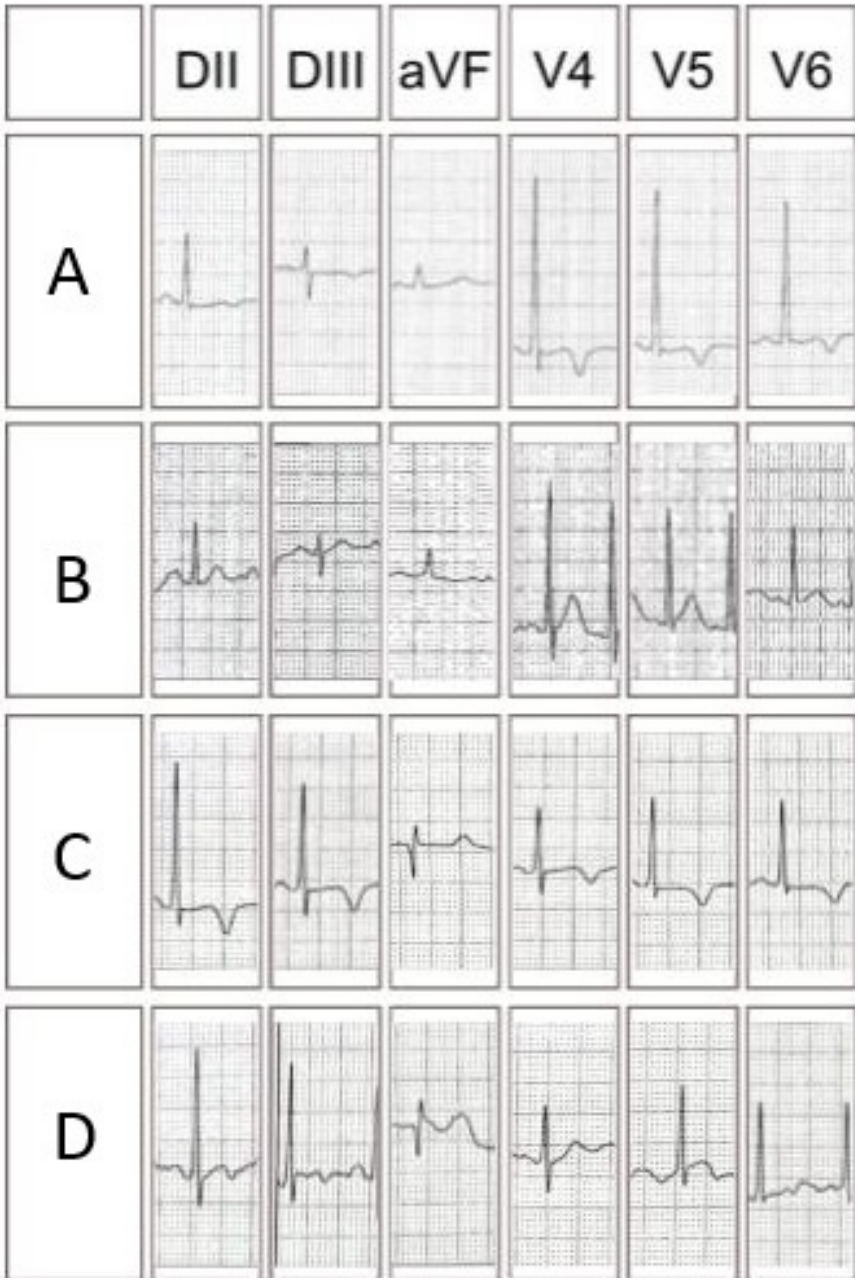


Figure 1 – *Electrocardiographic tracings. Ventricular repolarization abnormality at baseline (A) and normalization of T-wave polarity during dobutamine stress echocardiography (B). After five years, the ventricular repolarization abnormality persists at baseline (C), and T waves become positive during physical exertion (D).*

Discussion

Microfistulas constitute a vascular labyrinth, representing the persistence of embryonic sinusoids that are typically obliterated over the course of normal development. In apical HCM with microfistulas, a plexiform communication occurs between the coronary artery and the LV cavity, a rare association that often raises the possibility of myocardial ischemia. Symptoms suggestive of angina and the presence of a murmur may or may not be observed.[2-4, 6, 14]

The ratio of stress DFV to resting DFV yields coronary flow velocity reserve, which was low (< 2) in the LAD, a finding that could have resulted from impairment of either

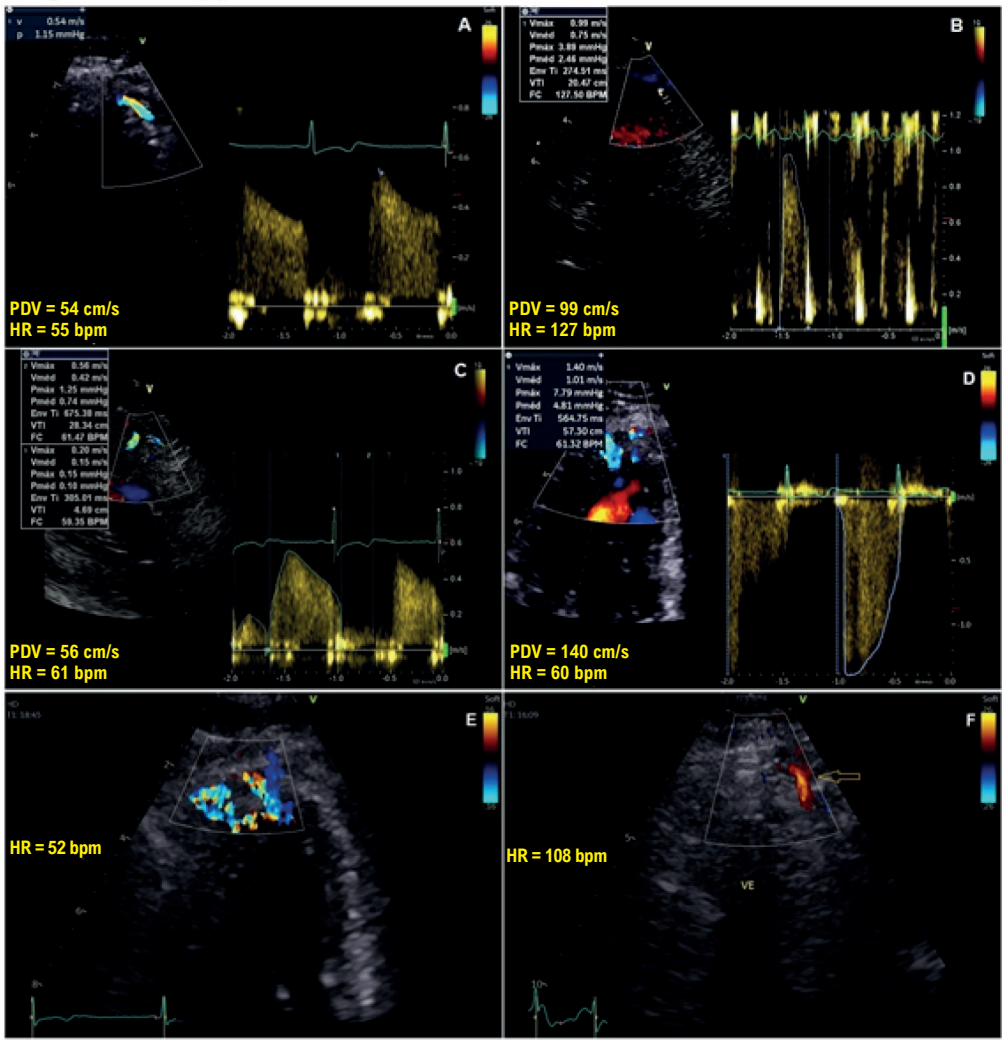


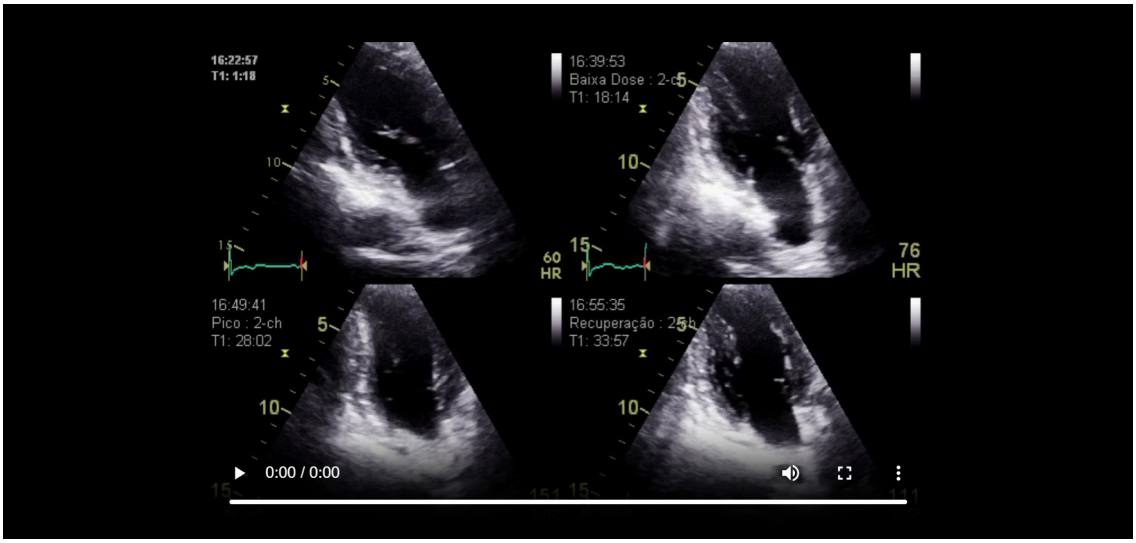
Figure 2 – *Pulsed-wave Doppler echocardiography of the LAD at baseline (A) and during dobutamine stress (B). Baseline pulsed-wave Doppler measurements in the LAD (C) and the microfistula (D). Prominent color Doppler flow through the microfistulas into the LV at baseline (E) is completely suppressed by the positive inotropic effect during physical exertion, whereas flow in the LAD (arrow) is preserved (F). HR: heart rate; PDV: peak diastolic velocity.*

the epicardial coronary arteries or the microcirculation.[7] In our case, although dobutamine stress echocardiography at maximal HR was negative for ischemia, a false-negative result was suspected.

Given the possibility of myocardial ischemia in the presence of ECG T-wave pseudonormalization, coronary angiography was a plausible indication; however, it revealed large, normal epicardial coronary arteries.

The value of T-wave polarity changes as an indicator of myocardial ischemia during stress is controversial. However, even at rest, conditions that might cause some form of ischemic memory are absent in our case.[15] False-positive diagnoses of ischemia based on prominent ECG abnormalities should be avoided in patients with apical HCM and microfistulas. It is worth noting that the depth of these negative T waves can vary over time during routine follow-up.[8,14] This was observed in our case, although no specific explanation for these changes was identified.

During dobutamine stress echocardiography, flow through the microfistulas appeared to be suppressed, but this was not given due attention at that time. Exercise stress echocardiography was performed using a supine bicycle, allowing for real-time echocardiographic monitoring. Once the HR reached 108 bpm, microfistula flow was completely suppressed; simultaneously, the T waves became positive



Video 1 – *Dobutamine stress echocardiography was negative for myocardial ischemia at maximal heart rate. Link: http://abcimaging.org/supplementary-material/2026/3903/2026-0090_video_01.mp4*

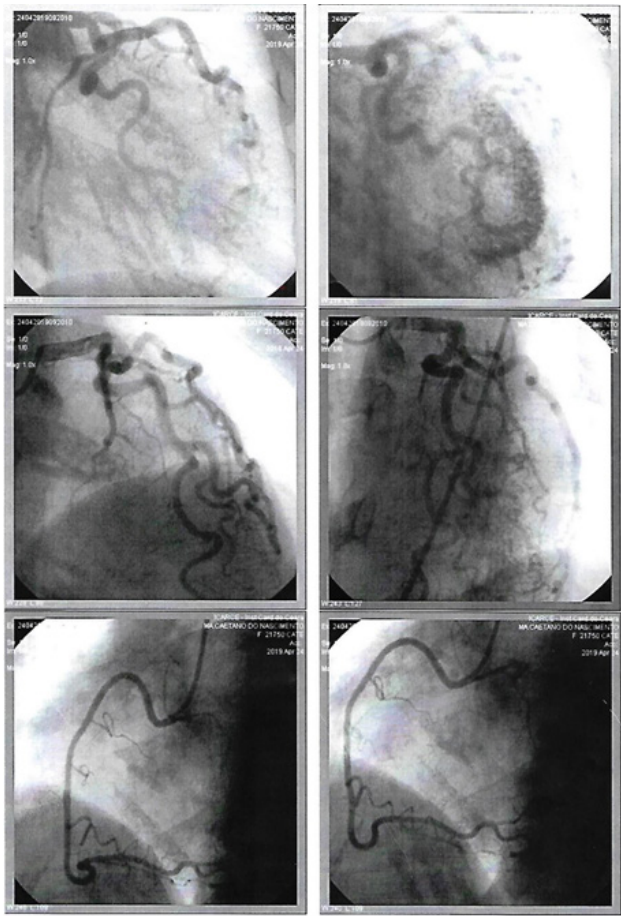
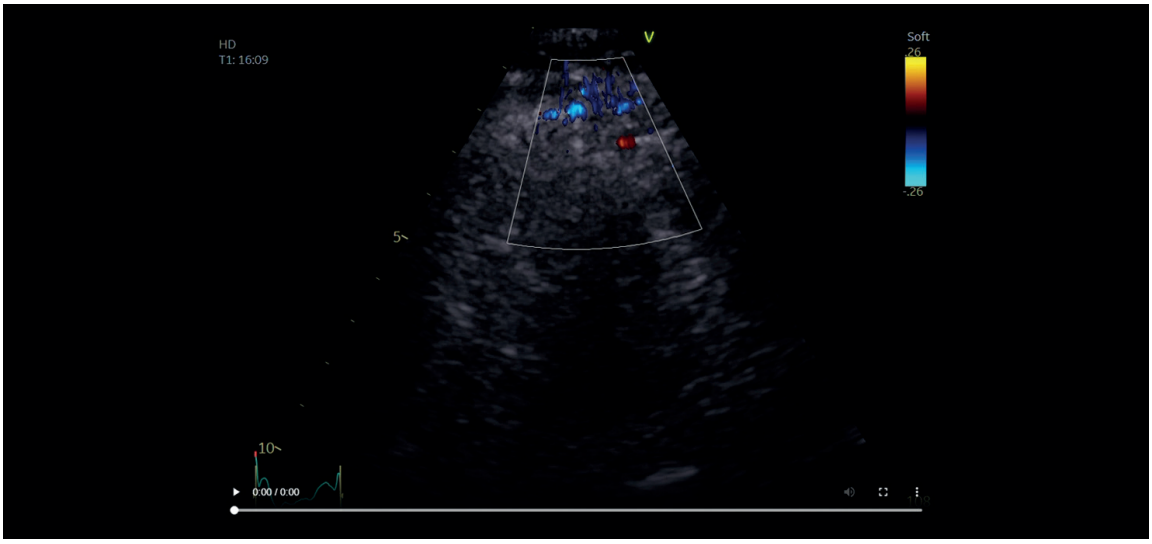


Figure 3 – *Normal epicardial coronary arteries.*

Table 1 – Baseline echocardiographic variables measured before exercise stress echocardiography

Left ventricular ejection fraction, Simpson's method (%)	65
Left ventricular global longitudinal strain (%)	−18.7
Left atrial volume index (mL/m²)	31
Left atrial reservoir strain (%)	27
Mitral Doppler E wave (cm/s)	9
Mitral E/A ratio	1.21
Mitral E/E' ratio	16



Video 2 – *During physical exertion, flow through the microfistulas is completely suppressed, whereas flow through the LAD is preserved. Link: http://abcimaging.org/supplementary-material/2026/3903/2026-0090_video_02.mp4*

on ECG, suggesting a correlation between these events. Conversely, flow through the LAD remained clearly evident.

Even in the presence of grade II diastolic dysfunction, the positive inotropic effect on the LV during exercise stress echocardiography did not result in the suppression of diastolic flow through the epicardial coronary artery. However, the consequent rise in intracavitary pressure and the shortening of diastole likely contributed to the complete suppression of flow within the microfistulas. This microfistula flow has been considered coronary flow steal because there is no communication with the coronary network; rather, blood flows directly into the LV cavity without perfusing the myocytes.[4,10]

In our case report, we observed, for the first time, that this flow steal ceased following a moderate increase in LV contractile performance, which could contribute to the magnitude of clinical manifestations and the prognosis. However, we cannot infer that the suppression of flow steal is the sole determinant of normalization of T-wave polarity on ECG.[16]

Conclusion

In this case of apical HCM with microfistulas, the change in ECG T-wave polarity was not consistent with pseudonormalization or myocardial ischemia, as it occurred in the presence of normal epicardial coronary arteries and coincided with suppression of coronary flow steal through the microfistulas, despite the positive inotropic effect.

Author Contributions

Conception and design of the research and writing of the manuscript: Abreu JS; acquisition of data: Abreu JS, Diógenes TCP; analysis and interpretation of the data: Abreu JS, Abreu MEB; critical revision of the manuscript for intellectual content: Chagas ISM, Feitosa MPM.

Potential Conflict of Interest

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

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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 Research Ethics Committee of Universidade Estadual do Ceará under protocol number 6.312.526 (approved on 09/20/2023). 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

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

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