

CASE REPORT

Hypertrophied Papillary Muscle causing Mid Cavity Left Ventricular Obstruction after Cardiac Surgery

¹Vikas Dutta, ²Ravi Raj, ³Ajay Bahl, ⁴Shyam Singh Thingnum, ⁵Goverdhan Dutt Puri

ABSTRACT

A rare cause of mid cavity left ventricular obstruction can be a hypertrophied and/or a malformed papillary muscle. Hypertrophy of papillary muscle can be atypical presentation of hypertrophic obstructive cardiomyopathy. Most of these patients do not have a resting gradient, but a dynamic gradient can occur in hypovolemia or increased contractile state. We present a case of elderly patient who had a hypertrophied anterolateral papillary muscle and developed mid left ventricular obstruction after weaning the patient from cardiopulmonary bypass.

Keywords: Papillary muscle hypertrophy, Cardiac surgery, Mid left ventricular gradient.

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INTRODUCTION

Left ventricular outflow tract (LVOT) obstruction is commonly seen in patients having hypertrophic obstructive cardiomyopathy (HOCM). Atypical presentation of HOCM can cause mid cavity gradient.¹ A single papillary muscle hypertrophy leading to hemodynamic instability is a rare presentation of HOCM.² We present a case of elderly patient who had a significant mid cavity gradient after coronary artery bypass grafting (CABG), secondary to hypertrophied papillary muscle.

^{1,2}Senior Resident, ³Additional Professor, ^{4,5}Professor

^{1,2}Department of Anesthesia and Intensive Care, Postgraduate Institute of Medical Education and Research, Chandigarh, India

³Department of Cardiology, Postgraduate Institute of Medical Education and Research, Chandigarh, India

^{4,5}Department of Cardiovascular and Thoracic Surgery Postgraduate Institute of Medical Education and Research Chandigarh, India

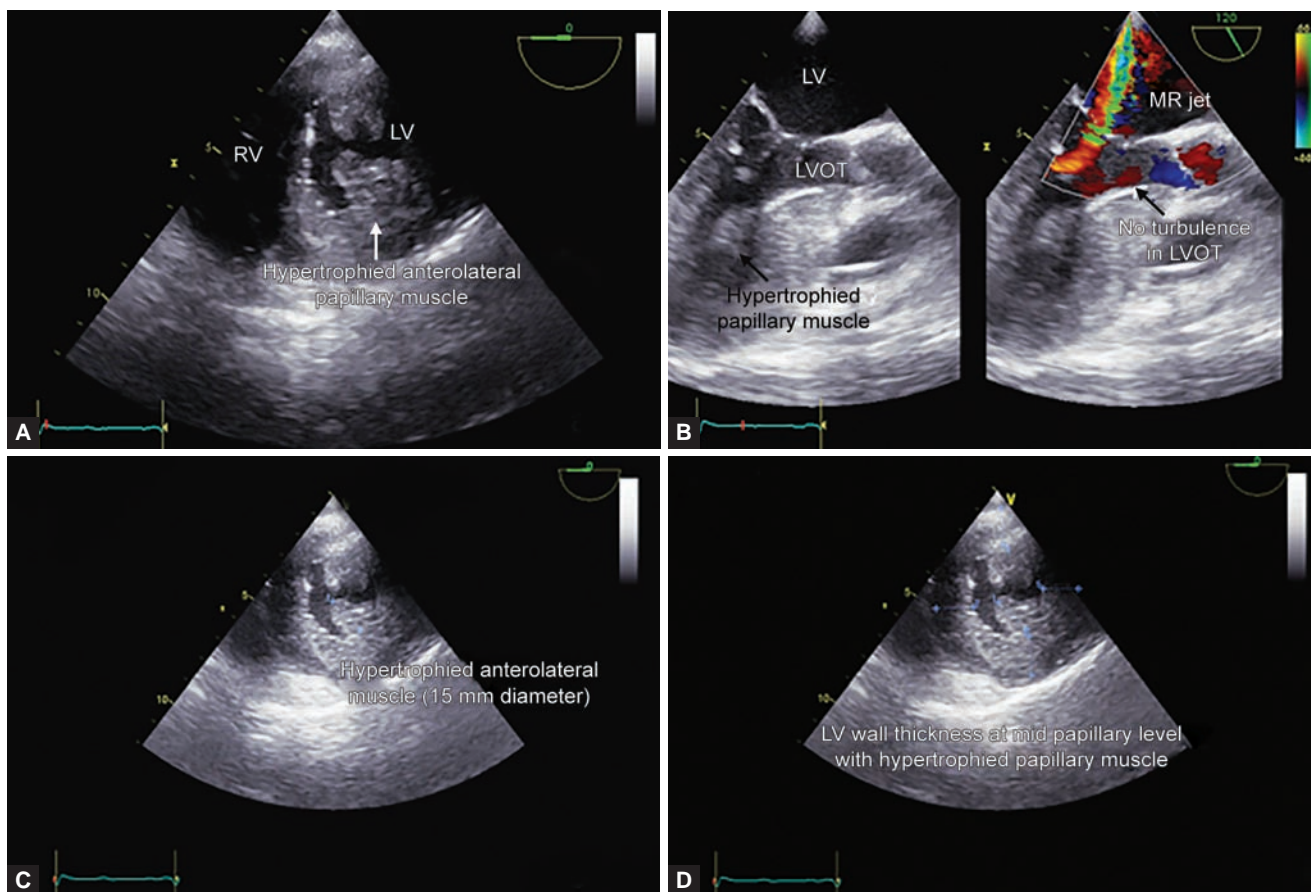
Corresponding Author: Goverdhan Dutt Puri, Professor Department of Anesthesia and Intensive Care, Postgraduate Institute of Medical Education and Research, Chandigarh India, Phone: 7087009509, e-mail: gdpuri007@hotmail.com

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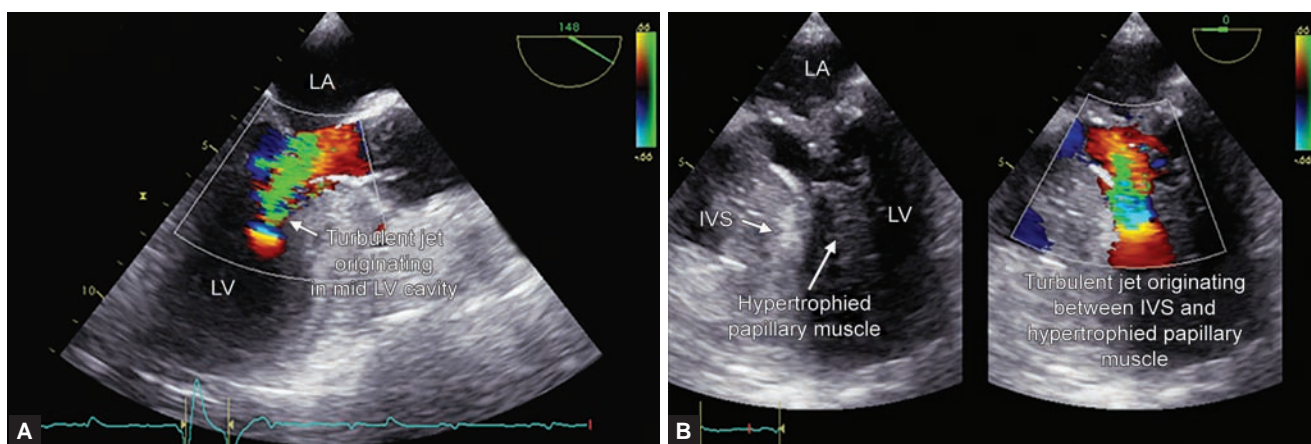
A 73-year-old female with history of angina on exertion and exertional dyspnea was admitted in our cardiac center. Her hypertension and dyslipidemia were well controlled on medication. Patient was not taking any beta blocker. Coronary angiography showed tripple vessel disease with mild mitral regurgitation (MR). Patient underwent on pump CABG. Precardiopulmonary bypass (CPB) transesophageal echo showed mild MR, hypertrophied left ventricle (LV), normal LV systolic function and LV diastolic dysfunction grade II. A prominent anterolateral papillary muscle was noted (Fig. 1A). But no turbulent flow was seen in LVOT (Fig. 1B). The diameter of this hypertrophied anterolateral papillary muscle was 15 mm (Fig. 1C). In two-dimensional echocardiography, the maximum wall thickness of LV was less than 15 mm in all the myocardial segments at mid papillary level (Fig. 1D). Patient was weaned from cardiopulmonary bypass (CPB) with adrenaline 0.05 µg/kg/minute. After coming off CPB, patient developed hypotension which did not respond to increasing dose of adrenaline to 0.1 µg/kg/min. Transesophageal echocardiography (TEE) showed a turbulent flow in mid LV cavity (Fig. 2A, Video 1). On Doppler examination, a peak gradient of 55 mm Hg was recorded. The turbulence was seen between the prominent anterolateral papillary muscle and the ventricular septum (Fig. 2B, Video 2). Adrenaline infusion was decreased to 0.02 µg/kg/minute and phenylephrine was started at 0.2 µg/kg/minute. Bolus of normal saline was given to increase the central venous pressure from 4 to 10 mm Hg. After all these measures, the peak gradient dropped to 44 mm Hg (Fig. 3A). Intravenous metoprolol 5 mg was given and repeated after 5 minutes. After 10 mg of metoprolol dose, the peak gradient dropped to 25 mm Hg (Fig. 3B). No further intervention was done. Trachea was extubated on first postoperative day. Patient was discharged from the hospital uneventfully.

DISCUSSION

Left ventricular outflow tract obstruction and mid cavity gradient are seen in HOCM patients primarily due to septal hypertrophy. Significant resting LVOT obstruction



Figs 1A to D: (A) Hypertrophied anterolateral papillary muscle in short axis view, (B) no turbulence in left ventricular outflow tract in long axis view, (C) diameter of hypertrophied papillary muscle and (D) Left ventricular wall thickness in short axis view

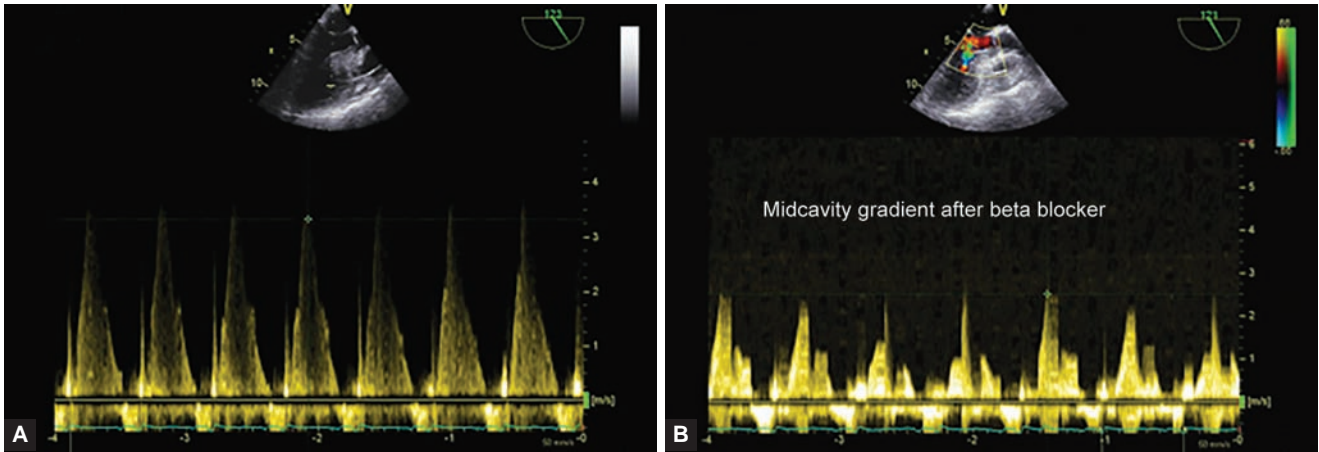


Figs 2A and B: (A) Turbulent jet seen in mid left ventricular cavity, and (B) turbulent flow between hypertrophied anterolateral papillary muscle and interventricular septum

is not seen in majority of these patients, but a dynamic gradient occurs in 25 to 30% of patients, with the resulting pressure gradient being influenced by central blood volume and contractile state.³ Hypertrophic obstructive cardiomyopathy patients sometimes have altered papillary muscle morphology—that is, anteroapical displacement of anterolateral papillary muscle or double bifid papillary muscles. There is an association between altered papillary muscles and resting LVOT gradient which appears to be independent of septal thickness.⁴ Various papillary muscle abnormalities have been reported

in literature like bifid⁵ or octopus papillary muscle⁶, accessory,⁷ or solitary papillary muscle hypertrophy.² Echocardiography can be used to identify the hypertrophied papillary muscle in patients who do not have the traditional LV hypertrophy pattern.⁸

In our case, the hypertrophied papillary muscle did not cause any gradient in the pre-CPB period probably because of low inotropic state. After coming off CPB, when the patient was started on inotropes, a gradient appeared in mid LV cavity during systole. On continuous wave Doppler, MR is characterized by earlier



Figs 3A and B: (A) Decrease in mid left ventricular cavity gradient to 44 mm Hg then to 25 mm Hg and (B) after beta blocker administration

onset, more abrupt initial increase in velocity and a higher peak velocity (> 5.5 m/s) than that of outflow tract signal.⁹ Cardiopulmonary bypass itself can cause marked increase in the levels of circulating catecholamines in arterial blood.¹⁰ The gradient was most probably due to the hypertrophied anterolateral papillary muscle coming in contact with inferoseptal wall during systole. Cha et al¹¹ have defined a prominent papillary muscle as a visually large papillary muscle which occludes the LV cavity during systole or > 1/2 LV end-systolic dimension and when maximal thickness of papillary muscle in short axis view is higher than 11.2 mm. The dimension of papillary muscle in our case was 15 mm. Patients with prominent papillary muscle can have same LVOT and mid cavity gradients as patients having hypertrophied septum.¹¹ Abnormally, thickened papillary muscle can lead to persistent symptoms and heart failure. In these cases, surgery such as papillary muscle realignment is done to relieve the obstruction.¹² In our case, the gradient came down with pharmacological measures and no surgical intervention was done.

CONCLUSION

The hypertrophy of single papillary muscle can cause left ventricular mid cavity gradient which can be diagnosed by intraoperative TEE and managed conservatively.

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