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LETTER TO THE EDITOR

Left ventricular perforation associated with vent catheter insertion without transesophageal echocardiography guidance

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To the Editor,

Malposition of a left ventricular (LV) vent catheter may lead to serious complications, including ventricular laceration, pseudoaneurysm, perforation, and acute aortic regurgitation (AR), making prevention essential [1, 2]. Such malposition has been demonstrated by transesophageal echocardiography (TEE), with fixation against the papillary muscles observed in 3.5% of cases and apical impaction in 5.5% [1]. Postoperative LV rupture secondary to vent catheter insertion has also been reported, particularly in the context of ischemia-related myocardial vulnerability [3]. We report a case of intraoperative LV perforation associated with vent catheter insertion despite the absence of apparent myocardial fragility, likely due to near-apical impaction of the catheter tip.

A 70-year-old woman (146 cm, 54 kg) with hypertension underwent thoracoscopic-assisted minimally invasive cardiac surgery (MICS) for atrial septal defect (ASD) closure. Coronary angiography revealed no stenosis. Transthoracic echocardiography demonstrated a relatively small LV cavity, with an end-diastolic diameter of 36 mm and an ejection fraction of 62%, without regional wall motion abnormalities. The interventricular septal and posterior wall thicknesses in diastole were both 8 mm, with no evidence of LV wall thinning or AR.

After the induction of general anesthesia, a TEE probe was inserted, and cardiopulmonary bypass (CPB) was initiated via right femoral cannulation. Following pericardial opening, a 14-Fr polyvinyl chloride (PVC) LV vent catheter (Cardinal Health, Dublin, OH, USA) was smoothly advanced into the LV cavity via the right superior pulmonary vein. Although ventricular arrhythmias and pulsatile blood return were observed, the catheter position was not confirmed using TEE. While dissecting the ascending aorta for cross-clamping, sudden bleeding revealed LV perforation with the catheter tip protruding into the pericardial space, without hemodynamic changes (Fig. 1a). Suction through the vent catheter was functioning normally, and the catheter was left in place, with the aorta promptly cross-clamped. Cardiac arrest was achieved uneventfully with antegrade cardioplegia, as there was no AR. In the presence of significant AR, we would have administered antegrade cardioplegia initially, followed by aortic opening and selective cardioplegia after the onset of ventricular fibrillation. After left atriotomy and ASD closure, inspection through the mitral valve revealed the vent catheter passing between the mitral chordae tendineae (Fig. 1b). The catheter passed between the papillary muscles and penetrated the apical-inferior wall, resulting in an approximately 5 mm defect, which was closed with two mattress sutures. Intracardiac air was evacuated using cardiotomy suction, and the atriotomy was closed. Additional de-airing was performed via the aortic root cannula before and after cross-clamp removal. The patient was successfully weaned from CPB without difficulty. The postoperative course was uneventful, with no evidence of bleeding from the perforation site, LV aneurysm formation, or air embolism.

LV perforation occurred despite no apparent myocardial fragility, indicating the need for routine TEE guidance. In the transgastric view, the catheter should be directed toward the LV apex, with the tip floating freely within the LV cavity without impinging on the apex or papillary muscles [1].

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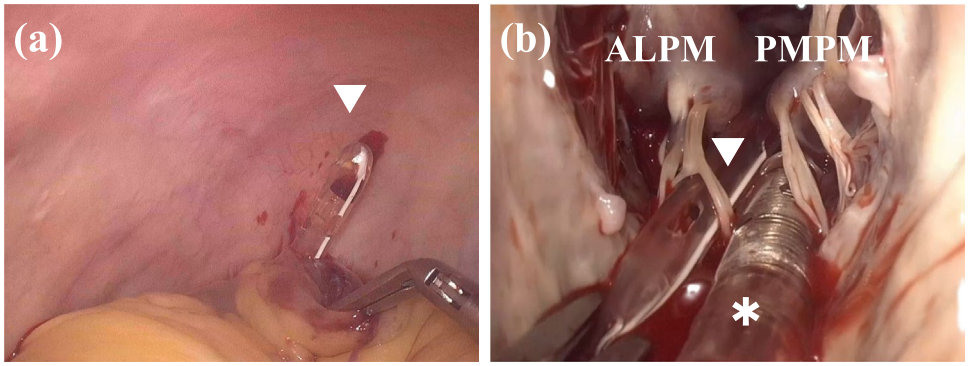

Fig. 1 Intraoperative views. **a** Vent catheter tip protruding through the left ventricular myocardium (arrow). **b** Catheter passing between the mitral chordae (arrow). Intracardiac air was evacuated by cardiotomy suction (asterisk). *ALPM* anterolateral papillary muscle; *PMPM* posteromedial papillary muscle

Three factors may explain the perforation in this case. First, a small LV cavity, as seen in ASD, may predispose to injury. Short stature and a small LV cavity are possible predictors for LV perforation [3, 4], likely because the catheter tip can advance farther and more easily impinge on the endocardium. Second, technical limitations of MICS, including limited exposure and inability to palpate the catheter, may increase the risk of vent-related complications [2]. Third, passage of the vent catheter between the mitral chordae tendineae may have contributed. In a porcine study, advancement of an ablation catheter through an introducer sheath facilitated myocardial perforation compared with advancement without a sheath, likely due to restricted movement, reduced buckling, and increased axial rigidity [5]. In this case, entrapment of the vent catheter within the mitral chordae tendineae may have similarly restricted tip mobility, resulting in localized excessive force on the endocardium. In addition, the use of a PVC catheter, which may be relatively stiff compared with softer catheter materials, such as polyurethane or silicone, might have further contributed to this effect.

Immediate removal of the catheter before aortic cross-clamping could be hazardous. Blood drainage from the venous cannula or active suction through the vent catheter may create a negative pressure gradient across the perforation site, allowing air entrainment into the LV cavity. In this case, catheter removal was performed only after cardiac isolation.

Despite the vent catheter tip having protruded into the pericardial space, its function appeared to be preserved, possibly because drainage from the proximal portion of the catheter is more effective than that from the distal tip [6]. Even in the presence of ventricular perforation, some degree of drainage may have been maintained through the proximal side holes. Accordingly, even if AR had been present, a certain degree of LV decompression might still have been achieved.

Continuous TEE confirmation of vent catheter position is essential. Hypothermia during CPB can increase catheter stiffness, while LV decompression may allow further advancement toward the apex, potentially resulting in endocardial injury. Because recognition of catheter-related injury may be delayed, the development of new or increasing pericardial effusion should raise suspicion of LV perforation.

In conclusion, routine TEE confirmation of vent catheter position may help prevent such mechanical complications, and even in routine and familiar operative settings, anesthesiologists and cardiac surgeons should proceed carefully and deliberately, without relying excessively on familiarity with the procedure.

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Declarations

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