Severe mitral regurgitation after intracardiac repair of tetralogy of Fallot: a rare complication and management

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Abstract

Tetralogy of Fallot (TOF) with additional ventricular septal defect (VSD) forms a difficult surgical subset. Commonly, additional VSD is in the muscular septum and direct visualization may be difficult during surgical repair especially in arrested heart. Consequently, direct closure of these defects is performed based upon preoperative imaging and/or intraoperative transoesophageal echocardiogram. We hereby report an unforeseen occurrence of traumatic acute severe mitral regurgitation after TOF repair possibly during closure of additional muscular VSD. We discuss the possible mechanism of this unprecedented complication, which was promptly diagnosed and managed with good surgical outcomes.

Keywords: Tetralogy of Fallot • Ventricular septal defect • Mitral regurgitation

INTRODUCTION

Surgical correction of tetralogy of Fallot (TOF) has established its safety in the current era. A majority of the postoperative problems are related to the anatomic characteristics of the patient such as hypoplastic pulmonary arteries, anomalous coronary artery crossing right ventricular outflow tract (RVOT), additional muscular ventricular septal defect (VSD) and associated anomalies. Unprecedented improvement in the surgical techniques and growing experience of cardiac surgeons worldwide has led to gradual decline in the occurrence of postoperative complications. Nonetheless, the surgical technique of closing small muscular VSD remains far from satisfactory, and as a result, having residual VSD after otherwise successful surgical repair is not uncommon. We recently encountered an unanticipated complication of closure of such a muscular VSD during successful repair of TOF in a child. We discuss the possible mechanism and details of the surgical repair.

MATERIALS AND METHODS

A 2-year old girl with the diagnosis of TOF with additional muscular VSD underwent surgical correction. She underwent complete repair of TOF by transtrial approach and the additional muscular VSD present was closed with pledgetted 5–0 Prolene suture. The intraoperative transoesophageal echocardiogram (TOE) immediately after coming off bypass did not show any residual VSD or RVOT gradient but trivial mitral regurgitation (MR) of uncertain aetiology was evident. The initial postoperative course was uneventful and she was discharged on postoperative day 5. However, on the evening of postoperative day 9, the child developed sudden-onset breathlessness while playing with her friends and the symptom worsened rapidly. At presentation in the hospital, she had severe respiratory distress and was immediately intubated. The clinical findings and chest radiograph were consistent with acute pulmonary oedema. After initial stabilization, transthoracic echocardiogram was done, which revealed severe MR secondary to chordal rupture leading to flail anterior mitral leaflet (Fig. 1). The child was immediately taken for mitral valve repair. Cardiopulmonary bypass (CPB) was instituted with the ascending aorta and bicaval cannulation. After cross clamping the aorta, the heart was arrested with cold blood cardioplegia and the right atrium was opened. The interatrial septum was incised anteriorly and was reflected on a posteriorly based flap. On inspection, the chordae belonging to the anterolateral papillary muscle complex were found ruptured leading to prolapse of A1 and P1 segments of the mitral valve. On close evaluation, it was seen that the Prolene suture used for closure of additional muscular VSD from the right ventricular side had trapped the tip of the anterolateral papillary muscle on the left ventricular (LV) side. The additional muscular VSD stitch along with its pledget was removed from the right ventricular side.

Two neochordae were constructed with 6–0 polytetrafluoroethylene sutures and fixed over the tip of papillary muscle and used to suspend the A1 and P1 segment of the mitral valve with the help of pledgets. Anterolateral commissuroplasty was done with pledgetted 5–0 Prolene suture. Saline insufflation test was done, which revealed trivial-to-mild MR with good coaptation of...
A1 and P1 segments of the mitral valve leaflets. The muscular VSD was closed with pledgeted 5-0 Prolene sutures. The interatrial septum and right atrial incision were closed and aortic cross clamp released after deairing. The child was weaned from CPB and the procedure was completed in a routine manner. Post-operative TOE showed trivial-to-mild MR, no mitral stenosis or residual VSD and normal LV function.

Postoperative course was uneventful and the child was extubated on Day 2, shifted to the ward on Day 4 and discharged on Day 7. Transthoracic echocardiogram done on Day 6 just before discharge revealed trivial MR, no mitral stenosis and normal LV function.

**DISCUSSION**

Additional ventricular septal defects (VSDs), usually muscular, are reported in 3–15% of patients with TOF [1]. It is often difficult to locate these VSDs intraoperatively from the right ventricular side due to heavy trabeculations. There can be multiple defects in the right ventricular side merging to form a single defect in the LV side. These are usually identified by passing a right angled clamp through the defect and locating the tip of the clamp on the LV side by viewing through the subaortic VSD. Different techniques are used for muscular VSD closure as with direct pledgeted sutures, horizontal mattress sutures or a three-pledget technique [2]. Kapoor technique (double patch) [3] and the approach can be from right atrium, right ventricle or left ventricle [4]. Hybrid VSD closure techniques have also been successful for closure of muscular VSDs. But these additional muscular VSDs can often complicate the postoperative course with a residual or a missed shunt.

We have experience in managing a large number of patients with multiple muscular VSDs with and without RVOT obstruction. But traumatizing the mitral valve apparatus with the VSD suture is unknown to us. Traumatic entrapment of the mitral valve apparatus must have happened during closure of the muscular VSD. We strongly feel that the collapsed ventricle displacing the papillary muscle may be the reason for it being caught in the suture. In retrospect, the presence of trivial MR visualized during TOE after the first surgery might have been due to trapped anterolateral papillary muscle, functionally impairing complete mitral valve closure. Thus, we postulate that the additional muscular VSD stitch had accidentally trapped the tip of the anterolateral papillary muscle (Fig. 2), which later on must have given way due to the increased contractile force of the left ventricle as the child became more active resulting in ruptured chordae, leading to prolapse and severe MR. This case highlights the importance of careful visualization and suture placement while closing muscular VSD. The mitral valve apparatus can be visualized either through the larger subaortic VSD or through the left atrium by means of an atrial septal defect either pre-existing or a surgically created one. In addition, the importance of prompt identification and management of an extremely rare cause of deterioration in the early postoperative period cannot be over emphasized.

**Conflict of interest:** none declared.

**REFERENCES**


