

Subvalvular membrane on the left ventricular outflow tract: multidetector computerised tomography imaging

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In this report, we describe a patient with a subvalvular membrane on the left ventricular outflow tract. Discrete subvalvular membrane is a cause of left ventricular outflow tract narrowing. Multidetector computerised tomography can demonstrate the anatomical three-dimensional view of this region and guide for surgery. (Folia Morphol 2011; 70, 4: 315–317)

Key words: subvalvular membrane, left ventricular outflow tract, multidetector computerised tomography

INTRODUCTION

Subvalvular membrane is a rare cause of left ventricular outflow tract (LVOT) narrowing [6]. This pathology can have different degrees of LVOT obstruction and symptoms. The clinical diagnosis is reliably confirmed with multidetector computerised tomography (MDCT). We present a case of subvalvular discrete membrane, an unusual cause of LVOT obstruction, with 16-row MDCT three-dimensionally.

CASE REPORT

A 22-year-old woman presented with a complaint of chest pain and intermittent palpitation continuing for 6 months. Biochemical and haematological parameters were in the normal range. Electrocardiogram showed sinus tachycardia. Chest X-ray was normal. Physical examination revealed a 2/6 systolic ejection murmur. In the echocardiographic study a discrete membrane was seen at the left ventricular outflow tract subvalvular localisation (Fig. 1). 16-row multidetector computed tomography clearly demonstrated a discrete subvalvular membrane three-dimensionally (Fig. 2). The patient was submitted to surgical treatment (Fig. 3). The membrane was resected in a cardiopulmonary bypass operation.

DISCUSSION

Left ventricular outflow tract narrowing may occur at the supra-ventricular, valvular, or subvalvular levels [11, 14]. Subvalvular LVOT obstruction can be separated into 4 distinct categories based on the aetiology of the obstruction: septal hypertrophy; fibrous membrane, web, or tunnel; abnormal papillary muscle attachment to the mitral valve; and redundant or accessory mitral valve tissue [12, 16]. This subvalvular narrowing is an additional resistive factor for the aortic and arterial impedance that may dramatically influence the left ventricular performance.

Rare causes of LVOT obstruction have been reported in the literature and include subaortic membrane, tunnel subaortic stenosis, various congenital anomalies (e.g. pulmonary atresia with intact interventricular septum associated with suprasystemic right-ventricular pressure with leftward displacement of the interventricular septum leading to LVOT obstruction), Tako-Tsubo cardiomyopathy (stress cardiomyopathy or transient left ventricular apical/midventricular ballooning syndrome), cardiac amyloidosis, and ectopic thyroid tissue (struma cordis) [1, 2, 4, 6, 7, 9, 10]. LVOT obstruction can be associated with a variety of other cardiac anomalies.

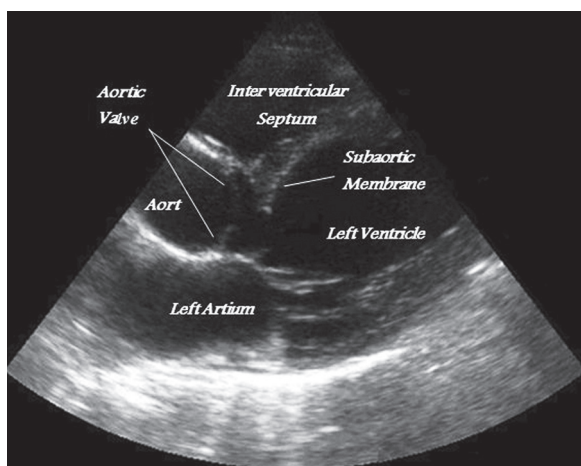


Figure 1. Echocardiographic study showing a discrete membrane at the left ventricular outflow tract subvalvular localisation.

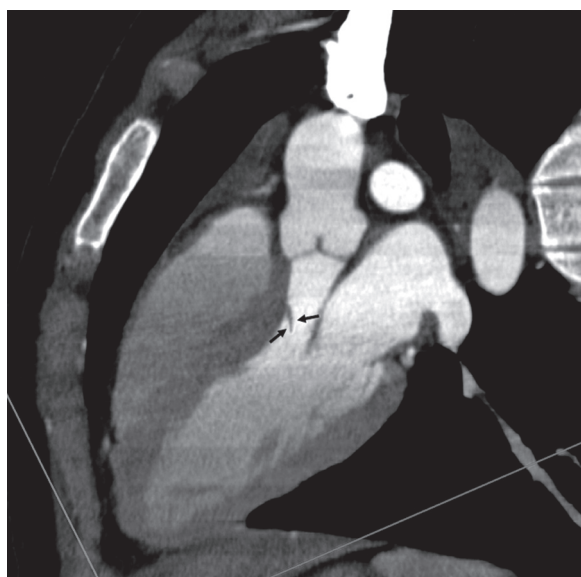


Figure 2. Sagittal cardiac multidetector computerised tomography view showing membrane (arrows) on the left ventricular outflow tract.

Clinical manifestations are exercise intolerance, chest pain, syncope, and tachycardia, and they are usually due to left ventricular outflow tract obstruction [13].

Discrete subaortic stenosis, which accounts for 8–30% of all forms of ventricular outflow tract obstruction, may present as a crescent shape of fibroelastic tissue protruding to the LVOT. There is clinical and experimental evidence that discrete subvalvular aortic stenosis is an acquired lesion [8]. Morphological abnormalities of the outflow tract,

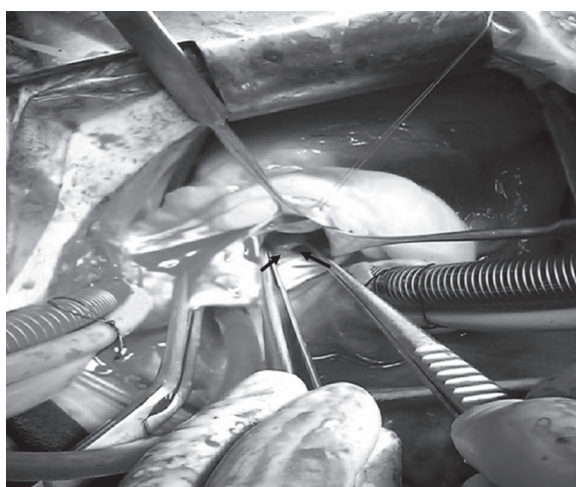


Figure 3. Operative findings; the subvalvular discrete membrane.

persistence of embryonic endocardial cushion tissue, and abnormal flow pattern have been proposed as the primus movens of subvalvular fibrous tissue proliferation [3, 5, 15, 17].

The present patient had LVOT narrowing secondary to an unusual anomaly of the aortic subvalvular membrane. Echocardiogram showed a membranous structure at the subvalvular region. The three-dimensional 16 row MDCT provided direct visualisation of the subaortic membrane, and the images correlated well with surgical findings. In our patient we did not find a significant LVOT obstruction, and no other abnormalities were present. The aberrant structure noted in the present patient was easily resected in the cardiopulmonary bypass operation.

CONCLUSIONS

Subvalvular membrane is a rare cause of LVOT narrowing. MDCT could be a useful tool for assessing discrete subaortic stenosis with anatomical details and guiding surgical interventions.

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