Journal of Cardiology Cases (2010) 2, e74—e77

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Case Report

Dynamic narrowing of left ventricular outflow tract—Possible mechanism of latent left ventricular outflow tract obstruction

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Received 10 March 2010; accepted 23 March 2010

KEYWORDS
Latent left ventricular outflow tract obstruction;
Alcohol septal ablation;
Stress echocardiography

Summary It has been reported that left ventricular outflow tract (LVOT) obstruction can be provoked in patients even without significant left ventricular hypertrophy. We experienced a 74-year-old man with mild degree of left ventricular hypertrophy and latent LVOT obstruction which was successfully treated by alcohol septal ablation. LVOT was not narrow at end-diastole, but proximal septum was protruding further into LVOT during the ejection period, producing a dynamic narrowing of the LVOT. Alcohol septal ablation did not reduce the interventricular septal thickness nor enlarge LVOT. However, it limited the excursion of proximal septum. The effect of the treatment suggested the importance of the dynamic nature of LVOT in the mechanism of latent LVOT obstruction in this case.

Introduction

Latent left ventricular outflow tract (LVOT) obstruction is a condition that causes significant narrowing of LVOT when it is exposed to sympathetic stimulation such as exercise load. It has been reported that even mild degree of left ventricular hypertrophy can cause LVOT obstruction [1]. In the present case, mild left ventricular hypertrophy presumably due to hypertension with latent LVOT obstruction was successfully treated by alcohol septal ablation. This case could provide an insight into pathology of this disorder.

Case report

A 74-year-old man had been well until approximately one year previously when he presented with dyspnea on effort.

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doi:10.1016/j.jccase.2010.03.006

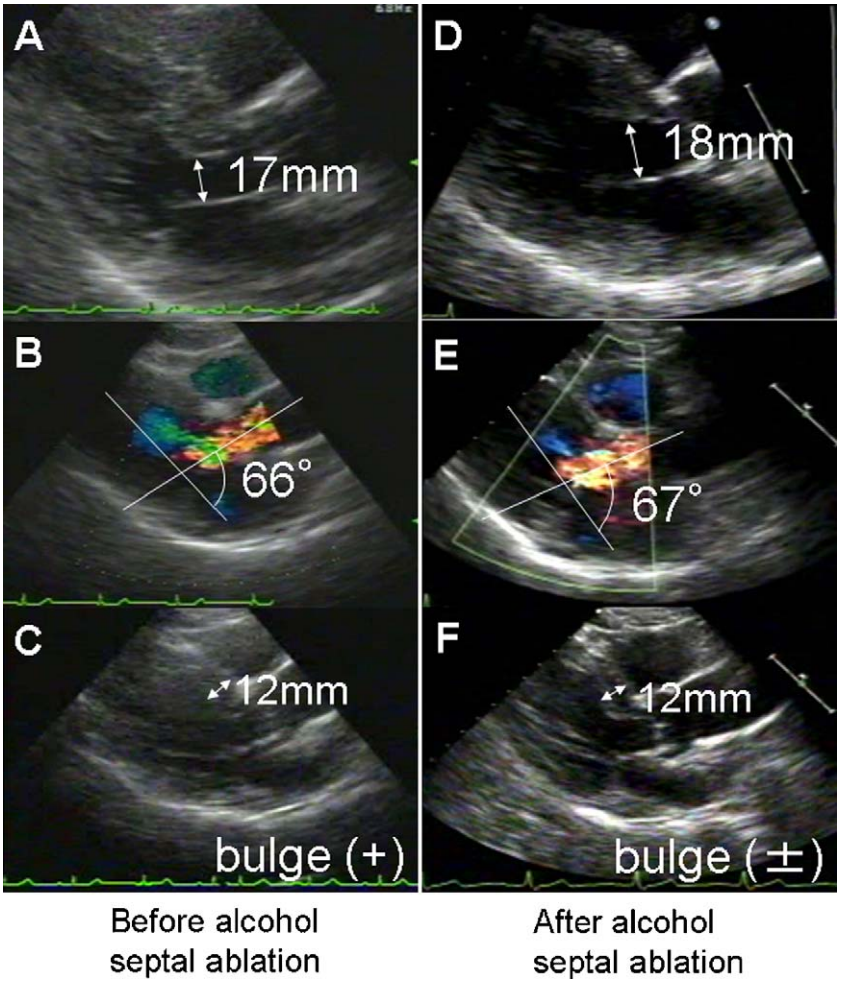

Figure 1 Comparison of left ventricular outflow tract (LVOT) features between before and after the alcohol septal ablation. (Before the procedure) (A) LVOT diameter; (B) angle of attack; (C) proximal septal thickness and bulge. (After the procedure) (D) LVOT diameter; (E) angle of attack; (F) proximal septal thickness and bulge.

He had been hypertensive for approximately 10 years but not on treatment. He did not have an obvious family history of cardiovascular disease. Standard echocardiogram demonstrated left ventricular ejection fraction of 76% and interventricular septal thickness of 12 mm with mild proximal septal bulge (Fig. 1). Transmitral *E*/*A* ratio was 0.53 and deceleration time of E wave was 320 ms, indicating abnormal relaxation pattern of left ventricular filling. The proximal septal bulge was protruding further into the LVOT during the ejection period, producing dynamic narrowing of the LVOT (LVOT diameter reduced from 17 mm at end-diastole to 12 mm at end-systole) (Fig. 2). The peak flow velocity at LVOT was 2.4 m/s. Systolic anterior motion (SAM) of mitral leaflet and mitral regurgitation were not found. The angle between the direction of LVOT flow and left ventricular long axis on parasternal long axis view (angle of attack) was 66 degrees (Fig. 1). He was able to exercise on a treadmill (Bruce protocol) for only 5.3 min before he experienced shortness of breath and almost fainted. His systolic blood pressure dropped from 132 to 70 mmHg, while his heart rate increased from 68 to 102 beats per minute. No systolic wall motion abnormality was induced. The peak flow velocity at LVOT climbed up to 8.2 m/s at peak stress, indicating exercise induced LVOT obstruction. However, SAM of mitral leaflet and mitral regurgitation were not induced even after exercise loading.

The latent LVOT obstruction was treated by alcohol septal ablation, although the basal septum was not markedly hypertrophied. Before the alcohol septal ablation, LVOT pressure gradient was 24 mmHg and augmented to 158 mmHg by ventricular extra systole. Immediately after ethanol injection into septal branches, LVOT pressure gradient decreased to 1 mmHg and was augmented only to 4 mmHg by ventricular extra systole (Fig. 3). His symptoms dramatically improved soon after the procedure.

Six months later, he remained free of symptoms. The echocardiogram demonstrated that the thickness remained unchanged, although the shape of the proximal septum bulge appeared to be rather rounded off. LVOT diameter at end-diastole was 18 mm, which was almost the same as before the alcohol septal ablation (Fig. 1). However, the proximal septum was not protruding into the LVOT during ejection period as far as it was before the ablation (LVOT diameter at end-systole was 15 mm), resulting in the relief of dynamic narrowing of LVOT (Fig. 2). The peak flow velocity at LVOT was 1.4 m/s. The angle of attack was 67 degrees

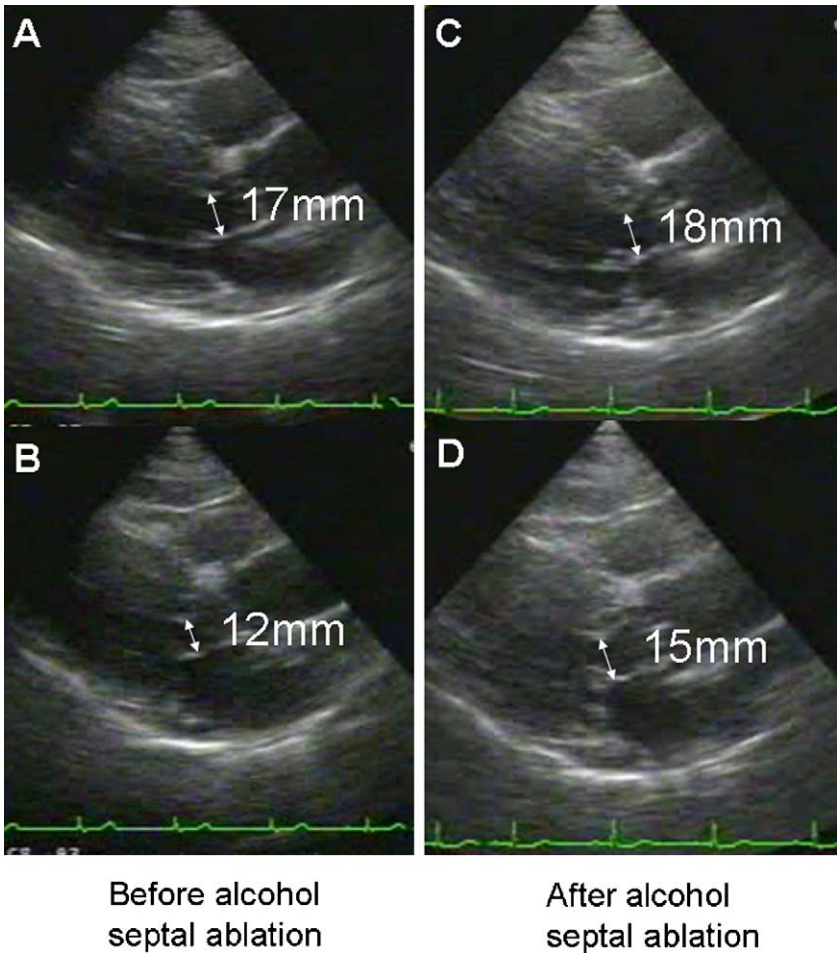

Figure 2 Loss of dynamic narrowing of left ventricular outflow tract (LVOT) after alcohol ablation. The proximal septum was protruding further into LVOT and LVOT was narrowing from the onset of ejection (A) to the end of ejection (B). After the procedure, the proximal septum lost its excursion and LVOT was not significantly narrowing from the onset of ejection (C) to the end of ejection (D).

(Fig. 1). He remained on a treadmill for 8 min. His heart rate increased from 62 to 112 beats per minute, but the peak flow velocity increased only to 3.6 m/s at peak stress. Then, his systolic blood pressure increased from 137 to 239 mmHg. The pharmacological therapy did not change before and after the treatment.

Discussion

Hypertrophic cardiomyopathy (HCM) is a common cause of latent LVOT obstruction. Maron et al. have shown that LVOT obstruction can be induced by stress test in more than half of HCM patients with no evidence of obstruction at rest in the large-scale HCM cohort study [2]. Vaglio et al. have demonstrated an adverse impact of latent LVOT obstruction on survival [3]. The overall average mortality rate was approximately 2% per year. Moreover, they have shown that 18% of the patients with mild symptoms developed advanced symptoms, indicating the progressive nature of this disorder. LVOT obstruction can be provoked in various other conditions, including acute myocardial infarction [1], takotsubo cardiomyopathy [4], and even sigmoid septum [5]. This fact implies that this disorder may be under-diagnosed in the clinical setting. Identification of latent LVOT obstruction is of clinical significance, because it not only defines the possible mechanism for symptoms of heart failure, but also creates the therapeutic option for relief of the symptoms.

Several factors have been proposed to be linked with the mechanism for the induction of LVOT obstruction. Nakatani et al. have shown that proximal septal bulge, narrow LVOT, and large angle of attack were predictive of latent LVOT obstruction, suggesting the mechanistic importance of these factors [6]. The proximal septal bulge morphologically narrows LVOT. The narrow LVOT increases flow velocity at LVOT which produces local pressure drop by the Venturi effect, drawing anterior mitral leaflet toward LVOT. In addition, a large angle of attack increases drag forces on to the anterior mitral leaflet. SAM of mitral leaflet has been known to contribute to the narrowing of LVOT. The significance of these factors has been confirmed in another study of surgical septal myectomy, demonstrating that LVOT enlarged and ejection flow became more parallel to mitral leaflet after successful septal reduction [7]. In the present case, however, LVOT obstruction appeared to be induced not through SAM of mitral leaflet, but rather by the protrusion of prox-

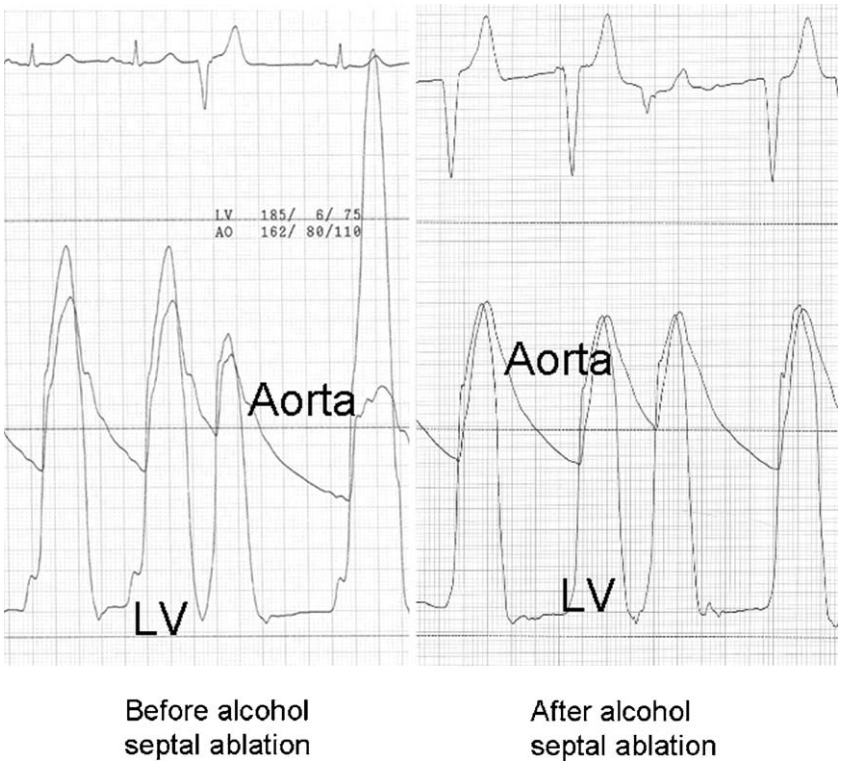

Figure 3 Simultaneous pressure curves of left ventricle and aorta.

imal septum during systole, because SAM of mitral leaflet was not manifest even after exercise loading. The present case had all the three factors determined by Nakatani, but the alcohol septal ablation did not modulate the factors. Instead, the treatment limited the excursion of the proximal septum, without any reduction in its thickness. This diminished the dynamic narrowing of LVOT, presumably leading to the relief of latent LVOT obstruction.

Conclusions

In the present case, a mild degree of left ventricular hypertrophy with latent LVOT obstruction was successfully treated by alcohol septal ablation which limited the excursion of proximal septum. It is suggested that dynamic narrowing of LVOT is mechanistically important for the induction of this disorder in this case.

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