ST Segment Depression on Exercise Testing in Chronic Aortic Regurgitation as a Criterion of Left Ventricular Dysfunction

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SUMMARY

A young male of 23 years presenting with clinical evidence of severe aortic regurgitation (AR) and aortic stenosis (AS) with angina pectoris is described. Electrocardiogram revealed left ventricular hypertrophy and strain pattern consistent with clinical diagnosis. Evaluation of angina on treadmill exercise test revealed ST segment depression of 7 mm in inferolateral leads alongwith typical chest pain. Cardiac catheterization did not show any gradient acrose the aortic valve and only svere AR was seen, but interestingly, injection of the contrast in left ventricle, aortic root, and left coronary artery consistently caused ST segment depression of more than 5 mm alongwith chest pain. The changes reverted to baseline after 5 to 15 minutes with 10 mg sublingual isosorbide dinitrate. Injection in right coronary artery did not cause any ST segment shift and angina. The anatomy of the coronary system was found to be normal. Possible mechanisms of such ischaemic changes without any obstructive pathology in the coronary arteries, and their significance in chronic severe AR are discussed.

INTRODUCTION

Angina pectoris is a classical symptom of aortic stenosis (AS) but may be associated with severe aortic regurgitation (AR). The cause of this with normal coronary arteries has been attributed to subendocardial ischaemia due to reduced supply and demand ratio. [1] This ratio is represented by diastolic and systolic presure time indices (DPTI/SPTI).

There is general agreement that all severely symptomatic patients of chronic AR should be treated surgically [2]. However, there is no consensus regarding the role of surgery in asymtomatic and minimally symptomatic patients. Exercise induced left ventricular (LV) dysfunction has been suggested as the most important marker to identify patients in this group who would do better with surgical treatment [3]. Quantitative biplane angiography, exercise radionuclide ejection fraction, and m-mode echocardiography are the techniques used to select patients for surgery[4] These are costly techniques and, moreover, are not readily available in the poor and developing countries.

We have come acrosss a patient of chronic severe AR who was only mildly symptomatic. The resting LV function was normal. During stress test and angiography, severe magnitude of ST segment depression was observed. The possible mechanisms responsible for these ischaemic changes in the presence of normal coronary system, and the value of these changes with respect to timing of surgery are discussed.

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CASE REPORT

A Young male of 23 years presented with chest pain, both on rest and exertion which was typical of angina pectoris for 6 months, alongwith dizziness and mild syspnoea on exertion for the same duration. There was no history suggestive of acute rheumatic fever, syphilis or any joint affliction in the past. Physical examination revealed pulse rate of 84 beats per minute and blood pressure was 160/40mm of Hg in both the arms in recumbent position. The pulse was collapsing with bisferiens character. There was no evidence of congestive heart failure. Precordial examination revealed a diffuse apical impulse maximum in the 6th intercostal space 1 cm outside the midclavicular line. A systolic thrill was palpable at the upper right sternal border (RSB) and in the suprasternal notch. On auscultation the first beart sound was normal and there was a constant ejection click maximum at the upper left sternal border (LSB) followed by a grade IV/VI ejection systolic murmur maximum at the upper RSB and radiating to both the carotids and suprasternal notch. The second heart sound was normally split with soft aortic component and was followed by an early blowing diastolic murmur extending to 3/4th of the diastole. A third sound (S3) was audible at apex followed by middiastolic murmur. In view of these findings clinical diagnosis of severe AR and AS was entertained.

INVESTIGATIONS :

The haemoglobin concentration was 13.5gm% and erythrocyte sedimentation rate 12 mm in the first hour. Serum biochemistry was within normal limits and serologic tests for syphilis were negative. The electrocardiogram (ECG) showed SV_1 + RV_6 of 82 mm with 1 mm depression of ST segment in leads V_{5-6} (without digoxin). The QRS axis and P waves were normal (fig. 1). Chest roentgenogram in posteroanterior projection showed cardiothoracic ratio of 0.60 and dilatation of ascending aorta. There was no evidence of pulmonary venous hypertension or calcification of the aortic valve.

M-mode echocardiogram showed a normal mitral valve with fine fluttering of the anterior leaflet. The size of left atrium was normal and LV dimensions were 54 mm and 76 mm in endsystole and enddiastole respectively. Ejection fraction was 64%.

Exercise test: To assess the status of angina, a multistage treadmill exercise test was performed. In third stage of the test on Chung's protocol, ST segment depression of 7 mm was observed in leads, II, III, aVF, and V4-6 (fig 1) and the patient experienced chest pain radiating to left arm. Exercise was discontinued at this point. The ECG returned to baseline over a period of more than 15 minutes.

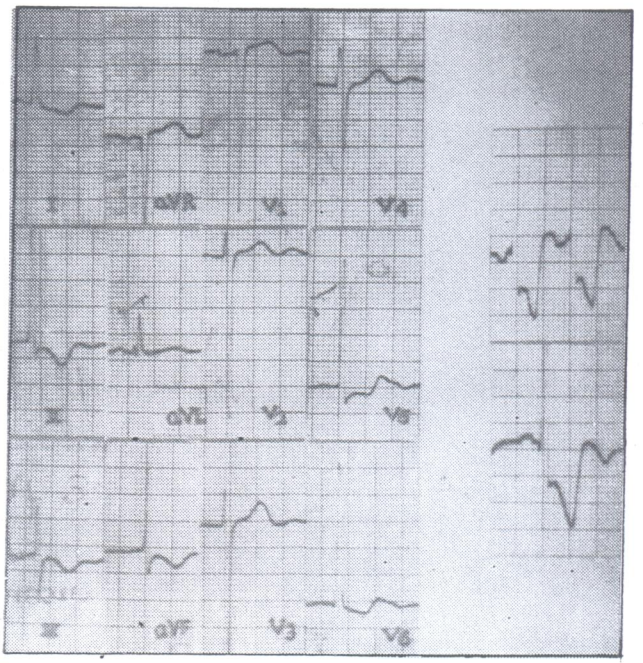

Fig. 1.: On the left is the resting 12-lead ECG; from V_2 to V_6, tracing is on half standardization. There is evidence of severe LVH with strain pattern. On the right is lead V_5 at peak exercise (above) and after 5 minutes of recovery (below) showing 7 mm ST depression.

Cardiac catheterization: The patient was then subjected to cardiac catheterization and coronary angiography. The LV pressure was 160/0, 12 mm of Hg. No gradient was found across the aortic valve. The LV angiogram in oblique view did not show mitral regurgitation and the aortic root angiogram in left lateral view showed Seller's grade IV AR. Interestingly, during LV and aortic root angiography ST segment depression of 5 mm was observed. Post-angiography, LVEDP increased to 20 mm of Hg.

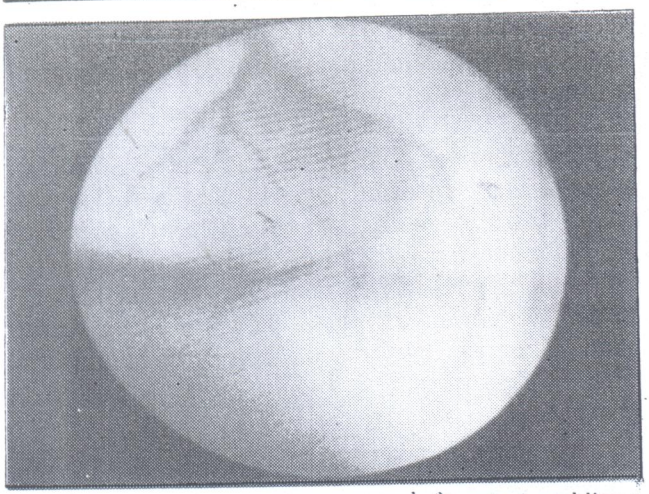

Fig. 2 : Left coronary agiogram in left anterior oblique view showing normal left coronary artery.

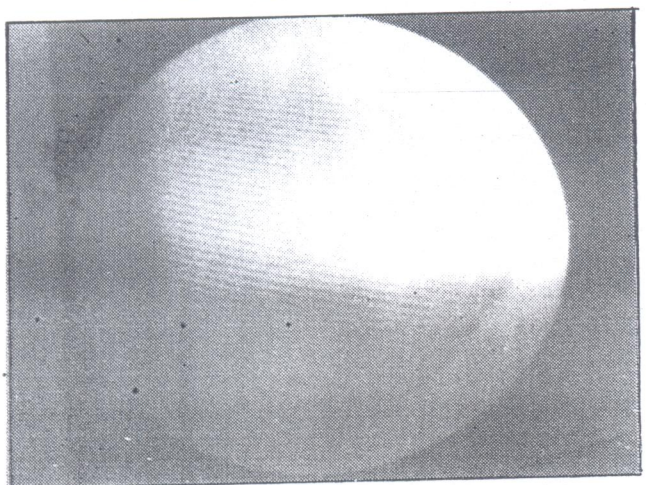

Fig. 3.: Right coronary angiogram in left anterior oblique view showing normal right coronary artery.

Coronary angiography was performed by Judkin's technique. First the left coronary (LC) angiogram was done. The LC tree did not show any significant lesion (fig 2). The contrast injection again caused 5 mm ST segment depression. In addition, severe sinus bradycardia (35 beats per minute) also occurred which disappeared on coughing. The right coronary (RC) system was also normal (fig. 3) and no shift was seen in the ST segment with RC injection. The contrast material used for angiography was Urografin 76.

After catheterization, the diagnosis of severe AR with angina alongwith ischaemic ECG pattern and normal coronary arteries was confirmed. The patient has been advised early aortic valve replacement which he is awaiting.

DISCUSSION

Angina in aortic valve disease occurs more commonly with stenosis than regurgitation. It is usually seen with severe AR and the reported incidence varies from 6 to 29%[4-7]. Some of the atypical features of angina in AR include rest pains, prolonged episodes of pain, poor response to nitrates and occurrence of pain at slow heart rates especially during sleep. Such angina is usually associated with widely patent coronary arteries and has been attributed to reduced supply and demand ratio which is repressented by DPTI/SPTI[1]. Oliveros et al have demontrated that DPTI/SPTI ratio is lower in patients of AR and the magnitude of lowering is related to severity of lesion[8]. It returns to normal following successful surgery of the aortic valve[8]. The reduction in supply is basically due to fall in coronary diastolic pressure (due to aortic runoff) and rise in LVEDP, whereas the demand is increased by LV hypertrophy and dilatation and increase in heart rate. In AS also, the DPTI/SPTI ratio is reduced and the resultant subendocardial ischaemia has been shown to cause fibrosis in this region[9]. In AR, however, fibrosis has been shown to be widespread and not limited to subendocardial zone. The difference has been explained on the basis of differing mechanical factors in these two conditions.[9].

We have observed 7 mm ST segment depression in inferolateral leads during exercise stress test. This was associated with typical pain of angina. The possible explanation for the mechanisn of these ischaemic changes is reduced supply as a result of rise in LVEDP caused by LV dysfunction due to stress. In face of normal coronary anatomy and constant aortic diastolic pressure, rise in LVEDP led to reduction in coronary driving pressure with resultant subendocardial ischaemia. There could have been rise in demand also due to rise in LV systolic pressure and increase in LV contractility with the effect of exercise. The possibility of reduction in supply due to rise in LVEDP is supported by the observation of similar ECG changes during LV, aortic root, and LC angiograms as well. After LV and aortic root angiograms, LVEDP increased from 12 to 20 mm of Hg. Further fortification to this possible mechanism is provided by the fact that injection of contrast in right coronary artery, which mainly supplies the right ventricular

myocardium, did not result into any ST segment shift or angina. Injection of contrast is known to cause depression of LV function and hypotension. It has been shown that cardiac reaction to injection of contrast in normal subjects is different from that in patients with diseased ventricle (coronary artery disease) and may be used as an approximate test of ventricular function[10,11].

Determining the optimal time for operative treatment of chronic AR has been for years a perplexing problem in clinical cardiology. Since the average survivial of patients after development of symptoms of angina, dyspnoea, and presyncope or syncope is only 3 to 5 years, [4,12,13] most clinicians agree that severely symptomatic cases of AR should undergo valve replacement. Irreversible changes in LV function are present in a subset of such patients; even after successufl surgical correction of AR, this subset of patients may develop congestive heart failure or have persistent cardiomegaly as well as depressed LV function [1,14-17]. Therefore, in order to minimise the risk of postoperative dysfunction, every effort should be made to operate upon the patient before serious LV dysfunction occurs [2,14-17]. In contrast, there is no consensus regarding role of operation in patients with minimal or no symptoms. Among this group also, there are some patients who have exercise induced LV dysfunction. It has been shown that in patients without exercise induced LV dysfunction, 5-year survival rates are not different in medically as compared to surgically treated groups (87% vs 86%). [8] However, when exercise induced LV dysfunction is present, surgically treated patients survive longer than their medically treated counterparts [18]. Thus it appears that valve replacement in all asymptomatic patients would subject many to the inherent risks of operation and of prosthetic heart valves [19].

It is concluded, therefore, that with the present state of valve technology and art of surgery, time has not yet arrived to recommend surgery to all patients of asymptomatic and minimally symptomatic severe AR [20]. It is important, therefore, to identify patients at a stage when the resting LV function is normal and only exercise induced LV dysfunction is present. Although the most precise method for assessment of LV function is quantitative biplane angiography, it cannot be readily employed in serial fashion. Two other techniques, radionuclide angiography and M-mode echocardiography, are safe and noninvasive tools for serial evaluation of such cases [2,3,21]. However, radionuclide studies are not readily available in poor and developing countries.

The value of treadmill stress test in predicting postoperative LV function and long-term survival has been pointed out by Bonow et al [22] They have demonstrated that the duration of exercise on treadmill may be of more prognostic value than radionuclide ejection fraction or resting echocardiographic endsystolic LV dimension and fractional shortening. We have found that ST segment response to exercise as well as angiography may help as a marker to identify patients with exercise induced LV dysfunction. It requires a comprehensive study to establish this as a criterion for timing of surgery in asymptomatic and minimally symptomatic patients of severe AR. It will provide us a cheap, simple, and noninvasive tool for serial evaluation of such patients.

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