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Mitral Valve Prolapse : An Aero Medical Problem

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Twelve cases of mitral valve prola (MVP) seen at the Institute of Aviation M cine are presented. 66% were from the fly branch, mean age 24 years. 33% were fr ground duty branches, mean age 39 years. cept one, all cases had mid to late syste murmur in the apical region and/or along left sternal region. Click was heard in 75% cases. 58% presented as ECG abnormality wh was either in the form of Twave changes in inferolateral leads or cardiac arrhythmi Ventricular Premature Beats (VPB) or Sup Ventricular Premature Beats (SVPB). In 2 cases papillary muscle dysfunction due coronary heart disease (CHD), was consider to be the cause of MVP. Echo cardiograp proved the diagnosis of MVP in 83% case Left Ventricular Angiogram was done in 50% the cases and it confirmed the diagnosis MVP. In this paper, aeromedical problems e countered in cases of MVP are discussed a relevant literature is reviewed.

MITRAL valve prolapse (MVP) is describe as a clinical syndrome resulting from divers pathogenic mechanisms of the mitral valv apparatus. It is also known by various other term such as systolic—click murmur syndrome, Barlow syndrome, floppy valve syndrome and billowin mitral leaflet syndrome[4]. During the last decade a so lot of interest has been generated in this entit Arrhythmia, endocarditis or sudden death are we known associations of this syndrome[4]. Brown et a in 1973 reported ten pilots with MVP who develope ventricular tachycardia and multifocal ventricula premature beats (VPB) while sustaining 4—6 G o positive acceleration (+Gz). It is because of these associated complications that MVP amongst aircrew is considered more hazardous. While evaluating such cases, the flight surgeon faces two problems (a) Does the patient have MVP, and (b) if so wha should be the disposal? Aeromedical problems

associated with MVP, problems with regard to the diagnosis and disposal of the aircrew with this disability have been highlighted.

Material and Methods

Twelve serving Air Force personnel were investigated at the Institute of Aviation Medicine, Bangalore. Out of these 8 (66%) belonged to flying branch and 4 (34%) were from ground duty branch. Their mean age was 24 and 39 years respectively. initial presentation for evaluation was as follows (a) ECG abnormality with systolic murmur heart (non-ischaemic group) : 8 (66%) (b) ischaemic heart disease (IHD) (Old stabilised) : 4 (34%). All were subjected to a detailed clinical examination. Haemoglobin, blood count, blood sugar, urea, uric acid and cholesterol were estimated in all the cases. X-ray chest to exclude evidence of cardiomegaly or cardiac aneurysm was done in all the subjects. A 14 lead ECG, resting and after double Master's exercise was recorded. A maximal stress testing on a computerised viagraph treadmill as per Bruce protocol[3] was done in the non-ischaemic group. In the group with ischaemic heart diseases submaximal treadmill stress testing was done. In cases of ECG abnormality (66%), twenty four hour ambulatory ECG recording on a Cardiodyne type tape recorder was done. This was later replayed and tracings analysed for any ST-T changes or pattern of arrhythmias.

In 10 cases (83%) echocardiographic studies were done. In 50% cases in addition to echocardiogram, left ventricular angiogram was also done. In two cases electrophysiological studies were undertaken in order to determine the nature of ECG abnormality. These three investigations were done at the Cardio Thoracic Centre, Pune.

Observations

Details of the 12 patients with mitral valve prolapse are summarised in Table I.

All subjects were asymptomatic at the time of evaluation. All were on the taller side of the scale for height and weight average height being 174 cms. One case had Marfanoid features, but metacarpal index was normal. Non-ischaemic cases (n=8) were referred primarily for evaluation of ECG abnormality in 7 cases (88%) and evaluation of systolic murmur at the apex in one case (12%). Cases with IHD (n=4) were referred for their periodic review. In non-ischaemic group, except one all cases had mid or late systolic murmur either at the apex or along the left lower parasternal region or over both the areas. Non-ejection click at the apex was heard in 75% cases in this group and in the IHD group out of the 4 cases 75% had non-ejection click and all had mid to late systolic murmur at the apex. In both the groups click and murmur became prominent in upright posture and after exercise. There was no clinical or radiological evidence of cardiomegaly.

As seen from the table I, ECG abnormality was in the form of T wave changes in the inferior or infero-lateral leads (50%) seen on resting or after double Master's two step exercise with extension seen after stress testing. In one case there was QS pattern in V 1 to V4. Two cases had presented with ventricular premature beats. Three cases with T wave changes, in addition had VPB or supra ventricular ectopic beats (SVE). IHD group had stabilised pattern of inferior wall infarction, infero posterior infarction and in one case sub endocardial infarction.

In 25% cases ambulatory monitoring revealed additional information which was in the form of bigeminal or trigeminal rhythm in one case and intermittent WPW syndrome was detected in another case. Echocardiographic studies were suggestive of MVP in these cases where it was recorded (83%). However, in 50% of the total cases, LV angiogram was recorded which confirmed the diagnosis of MVP. In addition to these, in two cases electrophysiological studies were done in order to prove the nature of ECG abnormality. In one case multiple bypass tracts were demonstrated, which were responsible for WPW syndrome. In the other case ectopic beats were found to be ventricular in origin.

Table - I

Clinical details

Patient Case Number	Age	Clinical presentation	General examination	Cardiac findings
1. IK	19	Asymptomatic	Tall Built (186 cms)	Non-ejection click + systolic murmu Parasternal region I/6.
2. AB	21	-do-	Tall Built (175.5 cms)	Systolic murmur mid and late, apex and 4th Lt. sternal space II/6.
3. SC	21	-do-	Average (167.5 cms)	Non-ejection click systolic murmur a the apex I/6.
4. DK	29	-do-	Tall (175 cms)	Non-ejection click + systolic murmu MA and LSR I/6.
5. RC	26	-do-	Tall (175 cms)	NAD
6. KA	22	-do-	Tall (179 cms)	Non-ejection click + systolic murmu (Mid and Late).
7. P	23	-do-	Average (168.5 cms)	Non-ejection click late systolic murmu at apex.
8. RS	26	-do-	Average (170 cms)	Non-ejection click Mid and Late systolic murmur.
9. RS	37	Old IHD	Tall (177 cms)	Non-ejection click Mid and Late systolic murmur.
10. VJ	42	-do-	Tall (180 cms)	Non-ejection click Mid systolic murmur at the apex.
11. NL	50	-do-	Average (170cms)	Systolic murmur at the apex. Grade II/VI.
12. RR	44	-do-	Tall (174 cms)	Non-ejection click +

ECG Findings	Mitral Echogram	IV Angio	Electrophysiological studies	24 hour Ambulatory monitoring
T ↓ II, III, avF, T ↓ V4 V6 VPB + some in bigeminy	Mitral leaflet prolapse (posterior)	Mild MVP (post)	Short AH and MV intervals (Multiple track)	Intermittent VPB and WPW Syndrome.
Multiple ectopic beats, some in Bigeminy and Trigeminy	MVP (post)	MVP Confirmed	Confirm VPB	VPB in Bigeminy and Trigeminy
QS in V1 - V4	-do-	-do-	—	NAD
T ↓ III, III (R), avF, avF(R) ST ↓ (1.5 mm) V4-V6 (DMT) Nodal rhythm	-do-	-do-	—	Nodal beats seen
Multiple VPB	-do-	—	—	VPB
T ↓ II, III, III (R), avFR, V5&V6	MVP	—	—	NAD
T ↓ II, III, avF, V4-V6	MVP	-	—	NAD
NAD	MVP	MVP	—	NAD
Suggestive of Inf. & Post infarction.	MVP	--		—
T ↓ II, III, avF, avF (R) T ↓ V4-V6	MVP	MVP	—	
QS in II, III, avF VPB+	—	—	—	—
Inf. Wall on supravent. Ectopic after DMT.	—	—	—	—

Discussion

The actual prevalence of MVP is not really known. Barlow et al' have reported the incidence of MVP between 10-17%. It has been diagnosed in all age groups. All cases in the present series (non IHD group) were asymptomatic and referred for evaluation due to ECG abnormality, systolic murmur or both together. Devereux et al[4] have also found most of the cases in their series to be asymptomatic. Symptoms like chest pain, dyspnoea, light headedness and dizziness, syncope, palpitation and psychiatric problems have been reported in cases with MVP [6-9]. Auscultation usually leads to the earliest suspicion of MVP. Presence of non-ejection click and or a mid or late systolic murmur are present in almost all the cases[6-8]. In the present series non-ejection click was heard in 75% cases and a mid or late systolic murmur was heard in 91% cases (n=11). As described earlier ECG abnormality was the first abnormal finding seen on ECG in 88% cases in the non-ischaemic group (n=7).

A variety of ECG abnormalities have been described in MVP. Apart from arrhythmias, the commonest ECG abnormalities are non-specific ST-T wave abnormalities in the form of ST depression and T wave inversion in resting ECG or after DMT or stress testing. ST-T changes are commonly seen in the infero-lateral leads. Other less common abnormalities include infarct patterns, prominent U-waves, prolonged QT interval, ventricular tachycardia, supraventricular tachycardia and atrial fibrillation [7-9]. In the present series the commonest ECG abnormality in the non-ischaemic group was Twave inversion in the infero-lateral leads (50%), followed by VPB in two cases. In one case QS pattern suggestive of anterior wall infarction was seen in V1 to V4. Cases with IHD had unequivocal findings of inferior, infero-posterior or sub endocardial infarction. With increasing use of treadmill stress testing in asymptomatic individuals one would expect that more patients with asymptomatic form of MVP will be discovered. This fact is supported in the present series when stress testing revealed abnormalities in the form of T inversion, ST depression or appearance of VPBs for the first time, in 25% cases of non-ischaemic group, in addition to their original ECG abnormality.

Echocardiography has been extremely helpful in detection of MVP in 83%, i.e., in all these cases where it was done (n=10). The most characteristic echocardiographic finding is an abrupt mid systolic posterior buckling motion of the mitral valve. A less specific echo abnormality is holosystolic posterior "hammocking" motion of the mitral leaflets, which can be reproduced in some cases with milder form of MVP who have isolated non-ejection systolic click. Using standard M-mode technique, the ultrasonic beam fails to traverse the postero-medial commisural scallop of the posterior leaflet of the mitral valve, which is the most frequently involved position of the mitral valve. Two dimensional real time echocardiographic studies have helped to define the mitral motion abnormality in MVP[8].

In 50% cases in the present series, LV angiogramwas done to arrive at the final diagnosis of MVP. LV angiogram continues to be considered by some as "gold standard" for the diagnosis of MVP. The characteristic finding is in the area of mitral valve in right anterior oblique view where the posterior mitral leaflet is seen to bulge into the left atrium. In equivocal cases left anterior oblique ventriculography can be quite helpful[8, 10].

Ambulatory ECG monitoring is occasionally helpful in the detection of arrhythmias in patients, with MVP[8]. A number of complications like bacterial endocarditis, progressive mitral regurgitation, left ventricular failure, ventricular tachycardia, ventricular fibrillation, embolism and sudden death have been described[5,6,8,10]. Stresses encountered in aviation and space travel, e.g., hypoxia, vagal and sympathetic stimulation are arrhythmogenic and are especially hazardous in individuals with MVP[10]. A combination of increased left ventricular after load and decreased left ventricular volume which occurs with −G forces could also have potentially disastrous consequences[10]. The application of +G forces in either the X or Z plane has been demonstrated to decrease left ventricular volume and to increase

systolic and diastolic pressure in the ascending aorta. Thus, acute severe mitral regurgitation might result from high 'G' loading of an individual with MVP who had little or no mitral incompetence under normal circumstances. The sudden occurrence of severe mitral regurgitation is frequent y haemodynamically disastrous especially in the presence of normal size of LV and LA. Rupture of elongated chordae tendinae might occur with high Gz loading in which case the resulting acute severe mitral regurgitation would not be alleviated by removal of 'G' forces. In addition under +G stresses ventricular arrhythmia may develop where no other form of stress testing has unmasked ominous arrhythmias[7,10]. Thus it can be concluded that diagnosis of MVP is more important in flying personnel than in those who are not involved with flying. All efforts should be made to confirm this diagnosis, before a proper disposal is given.

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