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# Mitral Valve Prolapse in a Family Practice Setting

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Mitral valve prolapse (MVP) syndrome is a relatively new clinical entity and fairly commonly encountered in clinical practice. The symptoms are often so vague that it is frequently not diagnosed or misdiagnosed for completely unrelated conditions. The recognition of MVP is important since the symptoms as well as the arrhythmias produced may be responsive to propranolol and the incidence of bacterial endocarditis is known to be increased. This paper reports on 25 patients with MVP in a family practice setting and compares this experience to recent literature on this problem.

In this study 25 patients are reviewed, all of whom presented to the Ohio State University Family Medicine Clinic for initial evaluation of their symptoms and were diagnosed as having mitral valve prolapse (MVP) syndrome. In this paper, the prolapse syndrome is discussed and the patient profiles are compared to the data in the current literature. The purpose is to make the primary care physician aware of the diagnosis and management as well as the incidence of this syndrome in a nonselected primary care population.

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## Definition and Etiology

Mitral valve prolapse or click-murmur syndrome has been described by many investigators.[1-3] The syndrome is classically found in a young female in the third or fourth decade of life with the symptoms of dizziness, palpitations, syncope, and chest pain, and is found to have a late systolic murmur with a midsystolic click. The prolapse may also be asymptomatic and the echocardiogram is usually diagnostic.[4]

The definitive cause of mitral valve prolapse and its syndrome is unclear; however, autopsy findings have shown a degenerative process in which abnormal mucoproteins are deposited in the mitral valve leaflets causing them to prolapse into the left atrium during systole.[5] This in some way may be related to the symptoms. In the past, investigators have attributed the symptoms to left ventricular dysfunction, papillary muscle dysfunction, and cardiomyopathy.[2]

Familial incidence has been well documented suggesting genetic predisposition, and the syndrome has been found to occur in higher proportions of patients with other genetic syndromes such as Marfan syndrome.[3]

## Materials and Methods

All 25 patient charts were retrieved from the files of the Ohio State University Family Practice Center, using number 7466 of the International Classification of Health Problems for Primary Care (ICHPPC) code. Each patient voluntarily presented to the clinic for initial evaluation of his/her symptoms and none were referred. Echocardiograms were performed and interpreted by the Division of Cardiology. The tabulated information was abstracted from the patient charts. The patients represented the practice of three full-time faculty and 12 residents in training. The cases were all diagnosed within a 15-month period. The total number of patient visits to the clinic during this period was 13,814. The total number of new patients seen was 2,260.

**Table 1. Patient Data**

| | |
|---|---|
| Number of patients | 25 |
| Sex ratio, Female/Male | 13/12 |
| Age | 16 to 64 years (27 years)* |
| Race, Caucasian/Black | 21/4 |
| Interval between symptom onset and diagnosis | 1 month to 36 years (7 months)** |
| History of childhood murmur | 2 |
| Rejection from armed service or by insurance company due to murmur | 1 |

*median age
**average number of months

## Clinical Profile

The 25 patients were between the ages of 16 and 64 years of age, with a mean age of 27 years. The sex distribution was essentially equal and there was a racial predominance of Caucasians (Tables 1 and 2). The majority of the patients presented with multiple symptoms—the most common being chest pain, palpitations, syncope, and rapid heart rate (Table 3). This symptom complex is similar to that reported elsewhere in the literature.[1] Interestingly, one patient presented with fever of unknown origin and subsequently was diagnosed as having subacute bacterial endocarditis two months after uneventful anal fistulectomy. Mitral valve prolapse was diagnosed after her endocarditis hospitalization. Three of the patients were asymptomatic and diagnosis of mitral valve prolapse was suspected on auscultation alone during physical examination for athletic or preemployment physicals; these were confirmed by echocardiogram.

Twenty-two of the patients had late systolic murmurs and nine of these had the classical late-systolic murmur preceded by a mid-systolic click (Table 4). One investigator pointed out that the auscultatory findings are related to posture, and these findings may be missed if the patient is not examined in the sitting, squatting, and standing positions.[1]

The interval between the onset of symptoms and the diagnosis of mitral valve prolapse averaged seven months, with a range from one month to as much as 36 years (Table 5). Twelve of the patients had no prior diagnosis attributed to their symptoms while the remaining patients carried a variety of diagnoses prior to the discovery of their mitral valve prolapse (Table 6).

No familial relationship could be determined from this study due to the lack of pertinent family history; however, the father of one patient had

| Table 2. Age at Diagnosis | |
|---|---|
| **Years** | **Number** |
| Less than 10 | 0 |
| 11 to 20 | 5 |
| 21 to 30 | 11 |
| 31 to 40 | 5 |
| 41 to 50 | 2 |
| 51 to 60 | 1 |
| 61 to 70 | 1 |
| Over 70 | 0 |

| Table 3. Presenting Symptoms | |
|---|---|
| Chest pain | 12 |
| Palpitations | 10 |
| Syncope | 7 |
| Rapid heart rate | 6 |
| Dizziness | 4 |
| Fatigue | 2 |
| Fever | 1 |
| Asymptomatic | 3 |

| Table 4. Auscultatory Findings | |
|---|---|
| Late systolic murmur with midsystolic click | 9 |
| Late systolic murmur only | 13 |
| Midsystolic click only | 2 |
| No auscultatory findings | 1 |

| Table 5. Interval Between Symptom Onset and Diagnosis | |
|---|---|
| 1 month | 8 |
| 5 months | 1 |
| 7 months | 2 |
| 10 months | 4 |
| 13 months | 4 |
| 2 years | 2 |
| 36 years | 1 |
| Asymptomatic | 3 |

died unexpectedly at 30 years of age, and he reportedly had symptoms which were consistent with the MVP syndrome.

Chest pain was the most commonly encountered symptom in this series and was usually sharp, left precordial pain associated with shortness of breath. In a 57-year-old patient, who previously had been diagnosed as having angina pectoris, nitroglycerin and other vasodilators had no effect on her chest pain; this should have aroused some suspicion concerning the diagnosis of angina.

Palpitations were also frequently reported by patients and these were attributed to ectopic ventricular and atrial beats although only three of the patients had these abnormalities on the electrocardiogram. Syncope has been thought to be possibly related to arrhythmias; however, the only arrhythmias identified in the patients with syncope in this study were sinus arrhythmia and sinus tachycardia. Fatigue occurred in a few of the patients and was a difficult symptom to manage. No satisfactory explanation for the mechanism of fatigue in the MVP syndrome could be found in the literature.

## Diagnostic Methods

The echocardiogram is a safe, noninvasive, accurate method of identifying mitral valve prolapse.[2] In the study population, the echocardiogram identified 13 patients with mitral valve prolapse and an additional five patients with probable mitral valve prolapse. One patient presented with a history and classical physical findings of mitral valve prolapse but had a negative echocardiogram. This has been reported in the literature.[6] The remainder of the patients had clinically diagnosed mitral valve prolapse and did not have echocardiograms as part of their evaluation (Table 7).

The electrocardiograms of patients with mitral valve prolapse have been shown to be abnormal in many instances and may be related to myocardial disease.[7] In most series, nonspecific ST-T changes and premature beats were the most common findings.[7] Of the 17 patients in this series who had

| Table 6. Previous Diagnosis to which Symptoms were Attributed | |
|---|---|
| Anxiety neurosis | 3 |
| Angina pectoris | 1 |
| Hypertension | 1 |
| Hiatal hernia | 1 |
| Costochondritis | 1 |
| Endocarditis | 1 |
| Mental retardation | 1 |
| Depression | 1 |
| Rheumatic fever | 2 |
| Hyperthyroidism | 1 |
| No previous diagnosis | 12 |

| Table 7. Echocardiographic Findings | |
|---|---|
| Echo definitely consistent with mitral valve prolapse | 13 |
| Echo probably consistent with mitral valve prolapse | 5 |
| Clinical findings consistent with mitral valve prolapse but echo negative | 1 |
| Clinical findings consistent with mitral valve prolapse but no echo obtained | 6 |

| Table 8. Electrocardiographic Findings | |
|---|---|
| Nonspecific ST-T changes | 3 |
| Left atrial hypertrophy | 1 |
| Sinus tachycardia | 1 |
| Sinus arrhythmia | 2 |
| Premature auricular contractions | 1 |
| Premature ventricular contractions | 1 |
| Normal electrocardiogram | 7 |
| Electrocardiogram not obtained | 8 |

| Table 9. Treatment | |
|---|---|
| Subacute bacterial endocarditis prophylaxis | 24 |
| Inderal therapy | 12 |
| Inderal response | 7 |
| Inderal resistance | 5 |
| Psychiatric referral | 1 |

electrocardiograms as part of their evaluation, ten were abnormal. ST-T changes were seen in three patients and premature beats in another three. Seven were felt to be entirely normal (Table 8). Holter monitoring can identify patients with arrhythmias but only a few of the patients had 24-hour tracings, and these were normal. Stress electrocardiograms have also been shown to elicit arrhythmias.[7,8]

Angiocardiography may confirm prolapse but it is invasive and associated with increased morbidity and is now rarely necessary since the advent of echocardiography. None of the patients were referred for angiographic evaluation.

## Treatment and Prognosis

The treatment of MVP syndrome should be directed at several problems: prevention of arrhythmias, relief of symptoms secondary to arrhythmias, prophylaxis against bacterial endocarditis, and supportive psychotherapy.

Propranolol (Inderal) in moderate doses has been shown to be effective in treating some arrhythmias and chest pain in mitral valve prolapse.[9] Twelve of the patients were treated with 40 to 160 mg/day of propranolol, and about half had subjective improvement in their symptoms, while the remaining did not have any response (Table 9). Therefore, propranolol should be reserved for those with documented arrhythmias and not used randomly.

The MVP patient has been shown to be at a greater risk for the development of bacterial endocarditis.[10] All of the patients in this study had been placed on subacute bacterial endocarditis (SBE) prophylaxis except the patient who developed endocarditis prior to the diagnosis of mitral valve prolapse.

Supportive psychotherapy is essential to the management of the patient with mitral valve prolapse since the treatment is less than optimal for the vast majority of symptoms. One investigator studied the natural history of the disease and felt that most patients should be assured that the disease is probably benign.[11]

The incidence of sudden death has been reported to be low and the prognosis is good unless mitral insufficiency, life-threatening arrhythmias, or bacterial endocarditis intervene.[12] A recent study on the management of mitral valve prolapse concluded that in addition to SBE prophylaxis, frequent follow-up should be done to evaluate the degree of mitral insufficiency.[13] Surgery should be considered for significant mitral regurgitation.

## Comment

Mitral valve prolapse is not uncommon. In this series 22 of the 25 patients were symptomatic; however, a recent investigation showed that mitral valve prolapse is present in a significant percentage of preselected, asymptomatic, healthy young women and, thus, may be one of the more common cardiac abnormalities.[14] Because of its recent discovery, it is assumed that a large percentage of practicing family physicians may not be aware of the syndrome or its method of diagnosis. For this reason, a retrospective study of the primary care population at the Ohio State University was undertaken, which identified 25 patients with MVP, one of whom had already developed significant morbidity as a result of her prolapse.

These patients were all diagnosed within a 15-month period and, interestingly, there were more cases of mitral valve prolapse identified during this period than there were abnormal Pap smears or rectosigmoid lesions, both of which are considered commonly encountered clinical problems in a primary care setting. The patient profiles also compared favorably to others reported in the literature.

Since the manner in which the prolapsed leaflet produces symptoms is unknown, and since familial incidence is documented, it is interesting to speculate that the noncardiac symptoms as well as some of the cardiac symptoms may be related to an associated inherited neuropsychiatric disturbance; hence the reason why many MVP patients are often misdiagnosed for either psychiatric syndromes or for other medical diseases. It is understandable, then, why all patients do not respond to propranolol since propranolol has been shown to be of value only in treating some arrhythmias and perhaps chest pain resulting from these arrhythmias.

Since it is the family physician who most often encounters the patient with nonspecific complaints, awareness of the MVP syndrome, as well as the ability to diagnose it, is therefore an essential skill of the modern family physician.

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