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J.T.: A Case of Mitral Valve Prolapse

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Mitral Valve Prolapse

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History continued from Summary of previous hospital records.

Mr. J.T. is a fifty-two year old white male who was diagnosed as having mitral valve prolapse four years ago, and who, on September 9, 1981, presented to the Hines Veteran's Administraion Hospital for evaluation of a cardiac arrhythmia.

During a hospitalization for a displaced shoulder in 1974, the patient, who was otherwise asymptomatic, was told that a routine electrocardiogram had shown that his "heart wasn't beating right", and he was placed on quinidine by his private physician which was discontinued three months later. Three years later in 1977 the patient was admitted to Hines for a lumbar laminectomy and disc removal at which time his admission electrocardiogram showed a right bundle branch block and multifocal premature ventricular contractions with coupling and occasional ventricular tachycardia. He was placed in the Coronary Care Unit at that time and treated with pronestyl and quinidine which were ineffective in suppressing his arrhythmia, after which he was placed on propranolol which was found to partially control his arrhythmia. Two echocardiograms done at that time showed no abnormalities, however, a third echocardiogram done after exercising the patient on a treadmill showed mitral valve prolapse. After his spinal surgery the patient was discharged on fourty milligrams of propranolol Q.I.D.

Since January of 1978 he began to notice progressive dyspnea on the exertion of climbing two-and-one-half flights of stairs,as well as dizziness or lightheadedness when he stands up suddenly, however he denies ever having experienced any orthopnea, or dependent

edema. Also, from January of 1978 until March of 1981 he has experienced occasional episodes of chest pain which he describes as "a metal rod pressing across the front of the chest", which can come on at any time and which last from ten to fifteen minutes and are associated with dyspnea but not diaphoresis. The patient reports no episode of chest pain since March of 1981.

On July 7, 1981 while working on a ladder he reports that he had a "dizzy spell" and fell off the ladder, breaking his right arm at the radius. At that time he was taken to Good Samaritan Hospital where an electrocardiogram revealed that the patient was having multifocal premature ventricular contractions with bigeminy and coupling, and a right bundle branch block. He was then treated with lidocaine and placed in a monitored bed. An echocardiogram done at that time showed no valvular abnormalities. Attempts to control occasional short runs of ventricular tachycardia with quinidine proved fruitless and after four days the patient was transferred to Hines for further treatment where his propranolol was continued at fourty milligrams Q.I.D. and he was discharged on July 13,1981.

After discharge computerized axial tomography (CAT scan) of the patient's brain done as an outpatient showed a questionable low density right cerebral hemisphere thought to be secondary to artifact, slight enlargement of the left lateral ventricle, and minimal atrophy of the left cerebral hemisphere. Also done as an outpatient at that time was a treadmill stress electrocardiogram which was stopped at one-and-one-half minutes because the patient had a run of ventricular tachycardia (five premature ventricular contractions in a row).

Since his discharge on Jult 13, 1981 the patient experienced increased shortness of breath and dyspnea on exertion which became severe enough to cause him to stop his work as a plumber because he reports that he "just couldn't fake it anymore." He was readmitted on September 9, 1981 for observation and further evaluation of his arrhythmia. His only medication at the time of admission was fourty milligrams of propranolol Q.I.D.

In viewing his past medical history Mr. J.T. reports that as a child he contracted measles, mumps, and chickenpox, and he recalls that he also contracted frequent sore throats leading to a tonsilectomy at age 16. He denies ever having contracted rheumatic fever, scarlet fever, or diptheria. In 1975 he had a bilateral herniorrhaphy and in 1977 he had a laminectomy and L_5-S_1 disc removal. He denies any personal history of hypertension, diabetes mellitus, or hyperlipidemia, but reports that he has smoked one to one-and-one-half packs of cigarettes per day for the last thirty-two years. His alcohol consumption consists of an occasional beer, and he has drunk about ten cups of coffee per day for the last twenty years. He has until recently worked as a plumber for the last thirty years, a trade which he began after serving three years in the Navy where he was stationed in Norfolk, Virginia.

In reviewing his family history Mr. J.T. reports that his mother suffered a heart attack at age fifty-two, but is still alive today at age ninety. He reports that his father died at age fifty-two of a liver abscess. He reports that he has a brother who is alive at age fifty-nine who has been told that his heart has frequent premature ventricular contractions. He has three sisters aged fifty, sixty-five,

and seventy, who are all alive and doing well. He reports that he is happily married and has four sons and one daughter, ages sixteen through twenty-seven, who are all alive and doing well. He denies any family history of hypertension, diabetes mellitus, or cancer.

Mr. J.T. reports that he has no known allergies or drug sensitivities.

In a systems review the patient denied any recent fever, sweating, or weight loss but did report that he has felt a general body weakness for the last several months. He denies any integumental changes or lesions. The patient has worn eye glasses for fifteen years for correction of far-sightedness, he has partial dentures, and he gets occasional headaches. He denies any hearing problems, nasal problems, recent sore throats or dysphagia. He reports no neck stiffness or masses. He denies any chronic cough or hemoptysis and reports that he has no shortness of breath at rest. He denies any nausea, vomitting, diarrhea, or constipation, and has not noted any hematemsis, melena, or blood in his stools. The patient denies any nocturia, dysuria, or hematuria. He reports that since his ruptured lumbar disc in 1977 he has been left with a slight weakness of his left leg. He denies any arthritis or muscular pains. He reports no lymphadenopathy, abnormal bleeding, or abnormal bruising. He reports that he had two episodes of syncopy approximately four or five years ago before his spinal surgery, but denies any other such related events other than the aformentioned falling off a ladder in July of 1981. He also reports that while walking he has recently noticed that he finds himself falling to the right side, often causing him to slightly stumble in order to catch his balance. The patient denies any paresthesias, seizures, tremors, or

memory loss.

On physical examination Mr. J.T. was a slightly obese, well developed, white male who was in no apparent distress. He was cooperative, alert, and well oriented. His temperature was 97.8°F orally, his pulse rate was fifty-two beats per minute, his respiratory rate was eighteen per minute, and his blood pressure was 130/84. His height was five feet eleven inches and he weighed two hundred and ten pounds.

He had normal integument and was normocephalic. His extraocular muscle movements were intact, his pupils were equal and reacted to light both directly and consentually, his sclera were non-icteric, and his fundi showed no pathology. His external ears, as well as ear canals and tympanic membranes, were normal. His nose had a midline septum and normal mucosa. Examination of his mouth and pharynx showed that the patient wore partial dentures, had good oral hygene, non-injected mucosa, and no observable lesions. The patient's neck had a normal range of motion, and no lymphadenopathy, thyromegaly or masses were palpable. There was no tracheal deviation or jugular venous distension. His carotid pulses were 2+ bilaterally with a good upstroke and no bruits were heard. Examination of his chest and lungs showed an increase in the anterior to posterior diameter of his chest wall. There was a surgical scar in the lumbar area of his back, he had no back tenderness and no scoliosis was appreciated. There was symmetrical expansion of the thorax on inspiration, and his lungs were clear to ascultation and percussion, although his breath sounds were somewhat distant bilaterally. He had no gynecomastia, breast masses, or axillary adenopathy. Cardiac examination revealed no lifts or heaves, there was

no palpable point of maximal impulse, and his heart sounds were distant. His heart rhythm was irregularly irregular with a normal S_1 and S_2, no S_3, and a positive S_4. There was also a systolic click heard best at the left sternal border at the sixth intracostal space. The click was noted to move later in systole when the patient moved from a standing position to a squatting position. No murmer was heard. The patients abdomen was slightly obese, soft and non-tender to palpation, without masses or organomegaly and with normal bowel sounds. Examination of the patient's extremities showed a splint on his right arm and hand, no edema, and no clubbing or peripheral cyanosis. His right arm and hand were tender to palpation. His radial femoral, and pedal pulses were all bilaterally 2+. He had a normal circumcised penis, and small testicles. On rectal examination he had good sphincter tone, no masses, a normal sized prostate, and occult blood negative stool.

Neurological examination showed cranial nerves II through XII to br grossly intact. Deep tendon reflexes of biceps, brachioradialis, patellar, and achilles were all bilaterally 2+ and symmetrical. The patient's station and gait were normal, and he was able to turn without difficulty. He was able to walk on his heels and on his toes without difficulty also. A positive Rhomberg sign was noted in that when standing with feet together and eyes closed, the patient fell to the right. Motor strength in his extremities was found to be normal and symmetrical, and no sensory abnormalities were noted to the modalities of pinprick, light touch, and vibration sense.

Electrocardiogram on admission showed a rate of sixty per minute, a right bundle branch block, multifocal premature ventricular contractions

with coupling and trigeminy. An admitting chest x-ray showed cardiomegaly, but no other abnormalities were noted.

On admission the patient's CBC showed a hemoglobin of 14.9 gm%, hematocrit of 43.1%, erythrocyte count of 4.98 million, mean cell volume of 87 FL., mean corpuscular hemoglobin of 29.9 picograms, mean corpuscular hemoglobin concentration of 34.6%, and a leukocyte count of 8,600/cmm. A differential white cell count showed neutrophils 69%, band cells 5%, monocytes 3%, lymphocytes 21%, and eosinophils 2% with adequate platelets. The patient's prothrombin time was 12.9 seconds with a control of 12.4 seconds, and his partial thromboplastin time was 23.0 seconds. Admitting blood chemistries showed a sodium of 138 mEq/L, potassium of 4.9 mEq/L, Chloride of 103 mEq/L, CO_2 of 26 mEq/L, glucose of 96 mg/dL, blood urea nitrogen of 17mg/dL, SGOT of 16 I.U., alkaline phosphatase of 65 I.U., inorganic phosphorus of 65 mg/dL, uric acid of 5.4 mg/dL, creatinine of 1.0 mg/dL, calcium of 9.6 mg/dL, total cholesterol of 203 mg/dL, total bilirubin of 0.5 mg/dL, direct bilirubin of 0.1mg/dL albumin of 4.6 gr/dL, LDH of 269 I.U., and total protein 7.5 gm/dL. The patient's urinalysis showed a clear yellow unine with a specific gravity of 1.020, and a pH of 5.0. The urine was negative for glucose, blood, ketones, protein, and bile, and a slight amount of mucus was seen on microscopic examination. The patient also had a non-reactive RPR.

A problem list for Mr. J.T. at the time of admission should include the following:

1) Disease of the heart.

   Etiology- unknown.

   Anatomy- mitral valve prolapse, right bundle branch block.

   Manifestations- systolic click, premature ventricular contractions, ventricular tachycardia, chest pain, dyspnea on exertion, dizziness, light headedness, history of syncopy.

   Functional class- II.

2) Rule out nervous system dysfunction.

3) Healing fracture of right arm.

4) History of herniated lumbar intervertebral disc with lumbar laminectomy and disc removal in 1977.

5) History of bilateral ingruinal herniorrhaphy in 1975.

6) History of tonsilectomy at age sixteen.

Although the overall cause of Mr. J.T.'s heart disease is unknown in the definite sense, it is quite probable that all of his present symptoms and manifestations could be attributed to his mitral valve prolapse. His case of mitral valve prolapse seems to be idiopathic in origin in that no firm etiology is evident. Mr. J.T. does mention the fact that as a child he contracted frequent sore throats, and although he denies ever having contracted rheumatic fever, it is possible that one or more of his sore throats were streptococcal in origin, thus leading to a subclinical case of rheumatic fever. Such

an occurance could have caused valvular damage not readily detectable at the time, and since rheumatic fever has been shown to be definitely associated with mitral valve prolapse, it should be considered as a possible cause of his condition.[1] Another etiology of mitral valve prolapse to be considered in this patient is that of ischemic heart disease which has been shown by some to be possibly related to mitral valve prolapse.[2]

The manifestations of the patient's heart disease are all consistent with those of mitral valve prolapse. A systolic click which moves later in systole with maneuvers which increase the return of blood to the heart is a hallmark of mitral valve prolapse. Such a click is often, but as in this case not always, associated with a systolic murmer.[3] Premature ventricular contractions, as well as ventricular tachycardia are common manifestations of mitral valve prolapse, although the site of origin of such arrhythmias is a source of great controversy.[4]

Chest pain is a very common manifestation of mitral valve prolapse. It has been seen in up to sixty-one percent of such patients, and is usually not, but may be, related to exertion.[5] Because chest pain, arrhythmias, and conduction abnormalities such as this patient's right bundle branch block, are also seen in ischemic heart disease (as is mitral valve prolapse), coronary artery disease must be ruled out in this patient. For this reason the next diagnostic step in this patient would be a cardiac catheterization, coronary angiogram, and left ventriculogram to rule out coronary artery disease, ventricular wall motion abnormalities secondary to ischemic heart disease, and to

further assess the extent of mitral valve prolapse.

Complaints of dizziness, lightheadedness, and dyspnea are not uncommon in mitral valve prolapse. Such complaints may be related to the associated arrhythmias, but in some cases are related to anxiety neurosis, which according to some researchers has a significant association with mitral valve prolapse.[6]

Mr. J.T.'s report of dizziness and lightheadedness as well as his reports of frequent "falling to the right side" while walking, and his two episodes of syncopy several years ago leave some question as to whether these events represent hemodynamic manifestations of his arrhythmia, or are more directly neurologically oriented such as in the case of vertigo or cerebellar dysfunction. The patient upon further questioning was not able to satisfactorily resolve this question, and thus one is left with the impression that there are both hemodynamic and neurological aspects to his complaints. This is quite possible in that, aside from the hemodynamic derangements which can occur during associated cardiac arrhythmias, mitral valve prolapse has been associated with syncopy, stroke, and cerebral emboli.[7] The questionable results of computerized axial tomography (CAT scan) of the patient's brain may support such a possibility.

Another possible explanation of the patient's frequent right-sided falling episodes, which is brought to mind by the patient's positive Rhomberg sign, is that of posterior nerve root damage done by his herniated intervertebral disc, or possibly by damage done during the surgical removal of the disc.[8] The patient does report some perceived left sided leg weakness, however, his normal and

symmetrical muscle strength, and normal vibratory sense upon examination, would tend to speak against this possibility.

Diagnostically speaking a repeat CAT scan of the patient's brain should be obtained in order to further clarify the possible abnormalities of the previous CAT scan.

The problem of the patient's healing fracture of his right arm seems to be resolving appropriately over time, however, X-rays of the arm are in order to accurately assess the amount of callous formation, and to help the physician in instructing the patient on the appropriate extent of rehabilitation and usage of the limb.

The problems of the patient's previous surgeries of lumbar laminectomy and intervertebral disc removal, bilateral herniorrhaphy, and tonsilectomy seem to be resolved and can be considered as inactive.

The patient underwent a cardiac catherization on September 11, 1981. A ventriculogram showed the left ventricle to be normal size and showed normal contractibility with an ejection fraction of sixty-eight percent. The patient's mitral valve prolapse was noted, but no mitral regurgitation was evidenced. His left main coronary artery, as well as left anterior descending and circumflex arteries were normal and without lesions. His right coronary artery was found to have mild plaguing in the mid portion of the artery which constituted a thirty percent stenosis. This stenosis was felt to be a non-significant lesion.

During the course of the cardiac catherization the patient underwent an episode of ventricular tachycardia immediately following the second injection of his right coronary artery. This episode was

controlled with a direct current cardioversion and the patient was medicated with intravenous lidocaine and heparin. The patient was placed in the Coronary Care Unit afterwards where he was found to be in his usual dysrhythmia of frequent multifocal premature ventricular contractions, none of which were sustained. The next day the patient reported that he felt fine and was transferred back out to the floor.

As was previously mentioned, there is great disagreement concerning the site of origin of the ectopic impulses which cause the premature ventricular contractions that are common in mitral valve prolapse. Some of the different proposals are: that such ectopic exitations originate in the mitral valve leaflet itself when it is stretched, that stretching of the papillary muscle causes it to initiate the ectopic beats, and that such ectopic exitations originate in the venrticular wall and are brought about by the stimulation of the sudden dumping of the blood which collects in the prolapsed valve leaflet against the side of the ventricle. [9,10]

There is also much discussion of the proper treatment of such arrhythmias. Propranolol has been recommended as the first line drug of choice for several reasons. It can shorten the QT interval which is prolonged in many mitral valve prolapse patients, and it decreases myocardial contratility and cardiac output which tends to decrease the stretch on the prolapsing mitral valve.[11] Propranolol is also useful in controlling the chest pain associated with mitral valve prolapse.[12] Other antiarrhythmic medications have been tried by various researchers to control such arrhythmias, such as quinidine, procainamide, dysopyramide, mexiletine, and diphenylhydantion,

each with a few reports of success.[13] Some such arrhythmias which were unresponsive to medical treatment have been treated successfully by pacemaker overdrive suppression.[14] In the event of severe mitral regurgitaion associated with mitral valve prolapse, a prosthetic valve replacement should be considered, however, some investigators have attempted such replacement of competent mitral valves in patients with severe life-threatening arrhythmias associated with mitral valve prolapse.[15]

In Mr. J.T.'s case ~~it seems as~~ though propranolol at a daily dose of one-hundred and sixty milligrams has been ineffective in controlling his arrhythmia, and because of his present bradycardia his situation would warrant trials on other medications. Since he has already been placed on quinidine and procainamide in the past with no benefit, a trial on one or more of the other aforementioned antiarrhythmic drugs is warranted, possibly in combination with propranolol. Because his arrhythmia is not of such severity to be considered life-threatening, pacemaker overdrive suppression or prosthetic mitral valve replacement should not be considered at this time.

Some physicians have suggested the use of anticoagulants or antiplatelet medications in patients with mitral valve prolapse in an attempt to avoid the cerebral thromboembolic problems associated with this valvular abnormality, however, there is no data to support these beleifs. Because of the potential complications of such medications, their use in this situation is probably of little or no use.[16]

Finally, the patient should be instructed to request antibiotic

prophylaxis for endocarditis prior to any dental or sugical procedures. This, however, is somewhat controversial in patients with mitral valve prolapse who have a click on asculation but no murmer.[17]

Good review of literature and its application to the particular case in question.

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