Papillary Muscle Rupture of the Mitral Valve Complicating Removal of a Permanent Transvenous Electrode

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We report a patient who developed acute mitral valve papillary muscle rupture following removal of an infected permanent transvenous electrode. Of additional interest is the fact that the patient was paced from the left ventricle for 20 months after the catheter electrode had passed through a patent foramen ovale at the time of insertion.

Since the introduction of transvenous cardiac pacing by Furman and Schwedel in 1959, numerous reports of unusual complications have appeared. To our knowledge, rupture of a papillary muscle or any valvular component has not been reported previously. This report describes a case of mitral valve papillary muscle rupture upon removal of a transvenous pacing electrode. Furthermore, this patient is of interest in that she underwent transvenous permanent cardiac pacing with the electrode positioned in the left ventricle, having passed through a patent foramen ovale, for a period of 20 months.

CASE REPORT

This 88-year-old woman had been observed in the Cardiac Clinic at the New England Medical Center Hospitals since 1965 for third degree atrioventricular block and episodes of lightheadedness. She had repeatedly refused permanent transvenous pacemaker implantation and was managed with isoproterenol (Protensol). She was admitted to the New England Medical Center Hospitals on January 28, 1969 after experiencing a Stokes-Adams episode. Cardiac examination revealed blood pressure 150/80 mm Hg and irregular pulse of 48 beats per minute. S1 varied in intensity. S2 was "physiologically split." There was a grade II/VI blowing murmur that occurred in early systole. It was loudest at the apex and radiated to the axilla. Crepitant rales were heard over both lung fields. There was no peripheral edema. The electrocardiogram revealed third degree ativoventricular block. Chest x-ray films revealed moderate left ventricular enlargement. On the following day, the patient underwent implantation of a permanent transvenous Mansfield* electrode. A Mansfield fixed rate generator pack was implanted in the right pectoral region.

Following intracardiac positioning of the pacing electrode, the electrocardiogram revealed the pacemaker to be functioning well and producing a "right bundle branch block" pattern. The pacing threshold was 1.5 milliamperes. An anterior chest x-ray film revealed the electrode tip to be in the area of the right ventricular apex. No lateral film was obtained at that time. Twenty-eight days after electrode insertion, the patient had a Stokes-Adams attack, and intermittent capture was noted on the electrocardiogram. She was readmitted for evaluation and repositioning of the electrode. After the procedure was completed, the anterior and lateral chest x-ray films (Fig 1) suggested that the electrode had crossed a patent foramen ovale and was in the left ventricular apex; the electrocardiogram again demonstrated a "right bundle branch block" pattern. Since the pacemaker at that time appeared to be functioning well, it was elected to leave the electrode in that position. In August of 1970 she underwent generator pack replacement and two months following discharge she had to be readmitted because of tissue breakdown over one corner of the generator pack with a sinus draining purulent material. She was noted to have low grade fever, and group A beta hemolytic streptococci was cultured from the draining sinus. Six blood cultures showed no growth at ten days. The patient was started on intravenous cephalothin (Keflin) therapy, and on her fourth hospital day, exteriorization of the generator pack was performed. Ten days later, a replacement electrode was introduced through the left cephalic vein. An electrocardiogram now revealed the pacemaker to be functioning well and producing a "left bundle branch block" pattern. Fluoroscopy suggested that the electrode tip was located in the right ventricular apex. A new generator pack was implanted in the left pectoral region subcutaneously. On the following day removal of the original electrode from the infected area on the right was attempted. Constant traction was applied. A sudden "give" occurred, and the catheter was removed. Myocardial tissue was noted adhering to the tip of the electrode. Auscultation at this time revealed a harsh grade III/VI pansystolic murmur at the apex. The patient's blood pressure, heart rate, and respirations were unchanged. She was sent to the intensive care unit for observation and monitoring. Thirty-six hours later, the patient suffered respiratory arrest and could not be resuscitated. At no time prior to death did her cardiac status change, and there was no sign of congestive heart failure. Postmortem examination was limited to the heart. There was diffuse coronary artery disease with no gross evidence of recent myocardial infarction or endocarditis. The posterior papillary muscle of the mitral valve was disrupted (Fig 2). Microscopic examination revealed fresh necrosis with an inflammatory cell infiltrate at the margins of the disrupted muscle. There was no histologic evidence of bacterial infection. There was a patent foramen ovale.

DISCUSSION

Bacterial infection is generally an uncommon complication with transvenous pacing.* Erosion through the skin of either the generator or the electrode can occur with secondary infection. If the infection cannot be adequately controlled with appropriate therapy, removal

*Manfield Generator and Endocardial Catheter System, Adcole Corporation, Waltham, Massachusetts.
The case presented here involved disruption of the papillary muscle of the mitral valve consequent to the removal of the transvenous electrode. No similar occurrence involving the mitral or tricuspid valve has been previously reported. Electrode adherence to the tricuspid valve and chordae tendineae has been reported.\(^2\)\(^3\)\(^4\) As a result, disruption of the tricuspid valvular apparatus upon forceful removal of the electrode would appear to be a definite risk. When removal of the pacing electrode becomes necessary and resistance is encountered, it has been recommended that the incarcerated electrode be removed by constant traction.\(^6\)\(^7\)\(^8\)\(^9\)\(^10\)

In this patient, longterm pacing was achieved successfully for 20 months with the electrode in the left ventricle, having passed through a patent foramen ovale. The potential hazards involved in pacing within the left ventricular chamber include thrombus formation, systemic embolization, loss of capture, and ventricular arrhythmias secondary to instability of the electrode. Anterior and lateral chest x-ray films at the time of placement are helpful in establishing the correct electrode position.\(^11\)\(^12\)

The patient presented in this report had a suppurative infection involving the site of generator implantation and that portion of the transvenous electrode in the subcutaneous tissues. It was felt that not only generator pack replacement, but also removal of the contaminated catheter were necessary. Removal of the catheter electrode resulted in acute papillary muscle rupture.

Removal of a permanent transvenous electrode, particularly beyond the first week of insertion, carries the potential risk of disruption of the tricuspid or mitral valvular apparatus. If electrode removal is mandatory and difficulty is encountered during attempts at nonsurgical removal, thoracotomy and direct surgical removal of the electrode should be considered.

REFERENCES

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Spectrum of ECHO Virus 1 Disease

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The occurrence of aseptic meningitis probably due to ECHO virus 1 disease has been reported together with significant myopericarditis, abnormal liver function tests, conjunctivitis, lymphadenopathy, pyuria and hematuria. Virus isolation and identification was from the spinal fluid. No previous report of such isolation in an adult exists in the literature.

ECHO virus 1 was first isolated from the stools of Egyptian children by Melnick and Aagren in 1952.1 ECHO virus 1 infection has been implicated in central nervous disease, especially aseptic meningitis, pericarditis, pleurodynia, diarrhea, undifferentiated upper respiratory infection, paralytic disease, exanthema, myalgia and ocular disease.2 However, a review of the literature would indicate that there have been no reports of isolations of ECHO virus 1 from the cerebrospinal fluid (CSF) of an adult with aseptic meningitis. Further, there have been no reports of ECHO virus 1 aseptic meningitis with simultaneous involvement of the pericardium, myocardium and liver.

CASE REPORT

History: The patient, an 18-year-old white diabetic youth, was originally seen by one of us (L.A.S.) on October 15, 1969. His illness had begun seven days earlier with generalized aching and an oral temperature of 101°F. He became afibrile for several days and on the day of his examination his temperature climbed back to 104°F and he was nauseated, vomited several times and complained of a splitting headache. He had photophobia and severe retrobulbar pain. He did not have a sore throat, chest pain or cough and no diarrhea or urinary frequency.

The patient’s diabetes was ushered in by an acute, severe episode of acidotic diabetic coma nine years ago. He had been taking 62 units of NPH insulin daily with a 2800 calorie diabetic diet. He had no interim history of acidosis, acetonuria or hypoglycemia.

The patient’s system review was negative for congenital or rheumatic heart disease. He had no previous history of jaundice or infectious mononucleosis. There was no recent history of parenteral skin penetration, shell fish ingestion, or contact with children or infants with any febrile disease or diarrhea. He was admitted to the hospital on the same day at 2PM.

Physical Examination: The patient was an acutely ill white youth. His temperature was 102°F (rectally), his pulse 96 per minute and regular with respiratory rate of 24 per minute and a blood pressure of 100/80 mm Hg. His upper and lower eyelids were swollen and the conjunctivae were suffused, but the sclerae were not icteric. There was adenopathy, particularly one gland in the left anterior cervical triangle was swollen, painful but easily movable. There was similar lymphadenopathy of both axillae. There was no neck vein distention. The lungs were clear to percussion and auscultation. The patient had a grade 1 systolic murmur over the apex and aorta. This same murmur was heard before this acute illness and remained unchanged in character and intensity. It was not transmitted to the axilla but transmitted to the neck. The patient did not have paradoxic pulse. There was evidence of nuchal rigidity. The remainder of the physical and neurologic examination was within normal limits.

Laboratory Data: Urinalysis revealed evidence of occult blood (Hemastix), 2+ sugar (Clinitest tablets), negative test for acetone and protein, specific gravity of 1.013 with a pH of 7.4. Microscopic examination of the urinary sediment revealed two to three white blood cells and one to two red blood cells per high-powered field. He had a hemoglobin of 15.1 gm per 100 ml, hematocrit 43 percent, white blood cells 8600 with 70 percent neutrophils, 10 percent band forms, 13 percent lymphocytes, 2 percent atypical lymphocytes and 5 percent monocytes. A lumbar puncture performed on admission revealed an opening pressure of 180 mm Hg, a closing pressure of 120 mm Hg and normal fluid dynamics. The cerebrospinal fluid (CSF) glucose was 120 mg/100 ml (normal 40 to 80 mg/100 ml), a concomitant blood glucose