Mumps of the Heart

Clinical and Pathologic Features

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This report concerns a patient who died of severe congestive cardiac failure 8 months after the onset of a clinical illness indistinguishable from mumps, characterized by submaxillary gland enlargement, orchitis, pancreatitis, meningitis, and myocarditis. During the past 2 decades, several papers have appeared describing electrocardiographic changes in many patients with mumps,1-5 and clinical signs and symptoms of myocarditis as well as electrocardiographic changes in a few patients with mumps.6-8 A patient dying of mumps myocarditis was recorded in 1932 by Manca,9 who described the autopsy findings in a 21-year-old soldier who died 14 days following the onset of his illness, which occurred during a severe epidemic of mumps in the barracks. Manca's paper appears to be the first to describe pathologic alterations in the heart of a patient dying from a viral myocarditis.10 Recently, Krakower and Roberg11 reported a 4-year-old girl, who died of heart failure 55 days after the onset of an acute illness that may have been mumps. The present communication describes the clinical and pathologic findings in another patient dying from probable mumps myocarditis.

Report of a Patient

A. M. (no. 02-49-32), a 17-year-old school boy, died October 10, 1959, after an 8-month illness. He had been in good health until February 16, 1959, when he noted pain in the left side of his neck. The following day the entire left side of his neck was swollen, and tender "lymph nodes" were palpated at the angle of the left jaw. Two days later he developed fever (104 F.), and the peripheral blood showed 40 polymorphonuclear neutrophils, 47 mature lymphocytes, 10 atypical lymphocytes, 2 monocytes and 1 per cent eosinophils. By February 20, 1959, he had developed severe headache, stiffness of the neck and bilateral testicular tenderness. At this time he was admitted to a local hospital where, in addition, he developed abdominal pain associated with an elevated serum amylase (533 Somogyi units). Frequent ventricular premature contractions also were noted, and he was told that his heart was enlarged. A presumptive diagnosis of mumps was made and he was treated with prednisolone. The white blood-cell count was 10,000/mm3 and the erythrocyte sedimentation rate was 20 mm. in 1 hour. Twenty-five per cent atypical lymphocytes were observed in the peripheral blood smear on February 25. By the end of 2 weeks his symptoms had subsided, but the tachycardia, cardiomegaly, and ventricular premature contractions persisted, and on June 8, 1959, he was admitted to the Clinical Center. His health before this illness had always been good. There had been no previous history of mumps or known exposure to mumps, and there was no history of acute rheumatic fever or scarlet fever. The parents were healthy.

On examination, he was well-developed and appeared well-nourished. The blood pressure was 105/70 mm. Hg. The salivary glands were not palpable. Prominent v waves, which increased on inspiration, were noted in the superficial jugular veins. The heart was very large. The pulmonic-valve closure was palpable, but the apical first sounds were weak. A grade-II/VI "grunting" systolic murmur, which was accentuated on inspiration and practically nonapparent on expiration, was audible along the lower left sternal border. A different grade-II/VI blowing systolic murmur was heard at the apex and in the left axilla. The liver was palpable and tender. The testes were soft.

The hematocrit value, white blood-cell count and differential, sedimentation rate, blood urea nitrogen, serum electrolytes, proteins, calcium, alkaline phosphatase, and transaminase were normal. The serum total bilirubin was 2.0 mg./100 ml. Lumbar puncture disclosed normal pressures: the spinal fluid was clear, but microscopically two red cells and 17 white cells, all lymphocytes, were seen per mm.8 Spinal fluid sugar was 77 mg./100 ml. and protein 25 mg./100 ml. Chest
Figure 1
Posterantuerior roentgenogram of chest.

Roentgenogram (fig. 1) showed generalized cardiomegaly. Electrocardiogram (fig. 2) showed sinus tachycardia, frequent multifocal ventricular premature contractions, and probable atrial hypertrophy. Serum mumps complement-fixation titer was 1:256.

Shortly after admission, the patient developed overt congestive cardiac failure, which responded only temporarily to digitalis and diuretic therapy. The cardiac index measured on July 21 was 1.8 L./min./M.², and the arteriovenous oxygen difference was 8.8 vol. per cent. Prednisolone, 160 mg. daily, was then administered for 1 week, but there was no apparent change in his condition, which progressively deteriorated. He died in severe right- and left-sided heart failure on October 10, 1959.

At autopsy (A59-202), the heart was hypertrophied (weight, 550 Gm.), all chambers were dilated, and the myocardium was soft. Recent and organizing thrombi were present in the apex of each ventricle and in the right atrium. The tricuspid and mitral valvular leaflets and chordae were normal, but their rings were dilated. The semilunar valves were normal. The endocardium of the left atrium and ventricle was mildly thickened. The coronary arteries were normal in origin and distribution and were free from luminal disease.

Microscopically, sections of the heart disclosed diffuse interstitial myocardial fibrosis, small focal areas of myocardial lysis, and, rarely, a few mononuclear cells in the interstitial fibrous tissue and in the focal areas of myocardial necrosis (fig. 3). Most myocardial fibers were hypertrophied, but a few were atrophic. No Aschoff bodies were seen. Stains of the myocardium for fat (oil-red O), glycogen (periodic acid-Schiff, and PAS-diastase), amyloid, and iron were negative. The parotid, submaxillary, thyroid, parathyroid, and adrenal glands were normal. The seminiferous tubules of the testes were severely atrophied. Sections of the liver revealed overwhelming centrallobular congestion and necrosis. The pancreas also showed the lesion of chronic passive congestion. The leptomeninges were focally thickened and contained small foci of mononuclear cells. There was intense generalized subpial and subependymal gliosis, extending into the brain parenchyma, which frequently showed changes of edema.

Electrocardiogram.

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Discussion

The diagnosis of mumps in the present patient appears justified. The clinical picture was compatible, the mumps complement-fixation test was elevated, and the testicular atrophy, myocardial fibrosis and necrosis, and central-nervous-system changes were consistent with previous acute orchitis, myocarditis, and meningoencephalitis, respectively. The necropsy cardiac findings in the patient described differed from those in the patient studied by Manca, but understandably so, in view of the far longer course in the present patient. The heart of Manca’s patient, who lived only 14 days, was neither dilated nor hypertrophied. Histologically, his patient had a fibrinous and leukocytic exudate in the interstitial tissue of the myocardium and the myocardial fibers showed various degenerative changes. The patient reported herein had no significant cellular infiltration in his heart at autopsy, but the cellular response would be expected to have disappeared long before 8 months had elapsed. Had the myocardial inflammatory reaction persisted, a beneficial response to the administration of prednisolone might have been expected. The autopsy findings in the patient described herein, however, are similar to those of the patient reported by Krakower and Roberg.11

According to Saphir and Cohen12 the histologic appearance of the heart in the present patient and in the patients reported by Manca and Krakower and Roberg is consistent with a viral origin. In viral myocarditis degeneration and actual necrosis of isolated or groups of myocardial fibers invariably occur, whereas in isolated or Fiedler’s myocarditis (presumedly nonviral) the interstitial tissue of the myocardium is principally involved and involvement of muscle fibers is rare.12 Saphir and Cohen believed that necrosis of individual myocardial fibers, a prominent histologic feature in the patient herein described, is sufficiently characteristic of viral myocarditis that it may be used as a differential diagnostic feature between isolated and viral myocarditis.

The course, and the radiographic and electrocardiographic features of the present patient, however, were not unlike those described by Levy and Von Glahn13 in patients with “cardiac hypertrophy of unknown cause.” It may be worth while to perform the mumps complement-fixation and skin tests in all patients with obscure myocarditis or idiopathic myocardial enlargement. Certainly, meningoencephalitis due to mumps is seen without the associated

Figure 3

Photomicrographs of heart. Left. Large areas of replacement and interstitial fibrosis are present. Right. Close-up of myocardium showing hypertrophied and atrophied fibers and loose interstitial fibrous stroma. Masson connective-tissue stain (left) and hematoxylin and eosin stain (right); original magnification ×35 (left), ×235 (right).
or antecedent occurrence of parotitis, orchitis, or pancreatitis. It is conceivable that the mumps virus may involve the heart without attacking other organs. The left ventricular endocardium of the present patient and of the patient described by Krakower and Roberg was mildly thickened, a finding frequently observed in the hearts of patients with primary myocardial disease. Recently, Noren et al.\textsuperscript{14} reported positive skin reactivity to mumps virus antigen in patients with endocardial fibroelastosis. This finding would appear to be further evidence that infection with the mumps virus may play a role in the etiology of some forms of heart disease.

**Summary**

The clinical and pathologic findings of a patient who died from diffuse myocardial disease 8 months after an attack of mumps is presented. His illness was complicated by myocarditis, meningoencephalitis, pancreatitis, and orchitis. A study of past reports discloses that electrocardiographic evidence of myocardial involvement in mumps is common, that clinical evidence of myocardial involvement is unusual, and that death from myocardial involvement is extremely rare.

**References**

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