of this agent in vitro. These cells may produce interferon, the quantity which is proportional to their number. Although the mechanisms involved in the phenomenon described in this paper could not be specifically defined, the possibility that elaboration of interferon was the critical factor merits serious consideration because of the finding that the larger the number of cells in the cerebrospinal fluid and, by inference, the greater the amount of interferon present, the lower was the rate of virus recovery.

The quantity of protein in the spinal fluid, one index of inflammation in meningitis, did not appear to exert a significant effect on the frequency with which ECHO 9 virus could be isolated. Failure to correlate protein content with the incidence of viral recovery suggests that the possibility of isolating the agent was not related merely to the presence of an inflammatory reaction alone but that it was dependent, in the main, on the number of cells and possibly the amount of interferon which they produced in the cerebrospinal fluid.

Conclusions

The frequency of recovery of ECHO 9 virus from the spinal fluid of patients with aseptic meningitis appears to be inversely related to the number of cells present in cases in which neutralizing antibody cannot be demonstrated.

Even in the presence of specific antibody, the number of cells in spinal fluid still appears to play a significant role in determining the incidence of isolations of ECHO 9 virus.

Although the specific mechanisms involved in this phenomenon cannot be defined, the possibility that interferon production by the cells in the spinal fluid is the critical factor merits consideration and study.

The age and sex of patients, duration of illness, and concentration of protein in cerebrospinal fluid do not appear to be of significance in determining the rate of ECHO 9 virus recovery from spinal fluid.

References


Ruptured Interventricular Septum

Successful Repair After Myocardial Infarction

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RUPTURE of the interventricular septum is an uncommon but usually lethal complication of acute myocardial infarction. It is estimated that 1% to 2% of deaths attributable to acute myocardial infarction are associated with septal perforation. The average interval between septal rupture and death is 2 weeks, with 50% of the deaths occurring within the first week. Only 3 patients with this lesion are known to have survived more than 5 years.

Until recently, the only treatment available for septal rupture consisted of those measures commonly employed in the treatment of congestive heart failure. Cooley was the first to suggest the surgical closure of interventricular septal rupture with utilization of cardipulmonary bypass. He closed a 3- X 2-cm. septal defect in a 49-year-old man 11 weeks after infarction. The patient showed remarkable improvement for a few weeks but died of sepsis on the 45th postoperative day. Shickman et al. reported the surgical repair of a ventricular septal defect by a closed technique 8 weeks after infarction. However, the patient died on the seventh postoperative day. Recently, Dobell et al. reported surgical closure of a septal perforation 5 months after myocardial infarction. Their patient demonstrated marked improvement and was discharged from the hospital. However, he succumbed to another myocardial infarction on the 30th postoperative day.

The patient whose case is reported here has survived 8 months after closure of an acquired ventricular septal defect due to myocardial infarction and she is now asymptomatic. She is the first patient reported to have survived an extended period after surgical intervention.

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Report of a Case

A 61-year-old Indian female noted right substernal pain of 3 days' duration in April, 1961, but did not seek medical attention for one week. During this time, she noted edema of the feet and legs, 3-pillow orthopnea, and non-clinical dyspnea. She was admitted to the Public Health Service Indian Hospital at Pawnee, Okla. Examination at that time revealed a Grade 4 systolic murmur and thrill over the left sternal border in the fourth left intercostal space. Signs of congestive heart failure were evident. The initial series of electrocardiograms revealed R-S-T junction elevation in leads V_{3}, through V_{6}, with subsequent late inversion of T wave and diminution in the amplitude of R in V_{6}. The patient had been examined 6 years previously because of a transient "stroke." A cardiologist's examination at this time had revealed no murmur. She had been treated intermittently in the preceding 2 years for hypertension. Two months after admission she had improved sufficiently to be discharged from the hospital, but increased dyspnea and pedal edema necessitated her hospitalization at the University of Oklahoma Hospitals in August. At this time the left border of cardiac dulness was in the anterior axillary line and a thrill was palpated in the third through fifth intercostal spaces in the left parasternal region. There was an associated Grade 4/6 holosystolic murmur in this area transmitted to the axilla and heard over the entire precordium and in the back. A ventricular gallop was audible. A left parasternal impulse was palpable. Additionally, signs of right and left ventricular failure were present. The patient was mildly hypertensive, Retinopathy was absent. The electrocardiogram showed evident healing of the previously diagnosed anteroseptal infarction. In addition, incomplete right bundle branch block (BBB) was present. Chest x-ray examination showed cardiac enlargement, pulmonary congestion, and right pleural effusion.

Initial symptomatic improvement was noted after bedrest, digoxin, and diuretic therapy. Accumulation of pleural effusion necessitated thoracostomy on 2 occasions. Later, evidences of increasing congestive heart failure became apparent. Cardiac catheterization and selective angiographic studies were performed on Sept. 8. A 4:1 left-to-right shunt at the ventricular level was demonstrated (table and Fig. 1). The patient was operated on Sept. 20, 1961, approximately 8 months after her initial attack. The heart was exposed through a midline sternotomy incision and the patient was placed on total cardiopulmonary bypass with the utilization of a rotating disk oxygenator at flows varying from 2,500 to 3,000 cc. per minute. The heart was diffusely enlarged. No evidence of myocardial scarring was noted. A thrill was present, localized over the apex. A ventriculotomy was done from the midportion of the right ventricle to the apex. A 2- x 1.5-cm. oblong defect was visualized in the apical portion of the anteroseptal septum. The margins of the defect were made up of smooth scar tissue. It was closed with 4-0 silk mattress sutures (Fig. 2). No residual shunt was apparent and the ventriculotomy incision was closed with 3-0 silk suture. Cardiac action remained good throughout the procedure and the ventricle was surged for venous inflow as the patient reexamed. Symptoms and signs of congestive heart failure were absent. The murmur was unchanged from the previous examination. Chest x-ray examination showed a decrease in heart size. Pulmonary congestion was absent. The electrocardiogram showed complete RBBB. A dye-dilution curve was obtained from the brachial artery after right atrial injection. There was no demonstrable left-to-right shunt. When last seen in June, 1962, 8 months after surgery, 14 months after infarction, the patient was asymptomatic.

Comment

Antemortem diagnosis of acquired ventricular septal defect myocardial infarction can be made with reasonable certainty, but this is the first patient in whom operative intervention has proved successful with survival more than a few weeks postoperatively. This patient's ability to survive a 4:1 left-to-right shunt was undoubtedly due to the nature of the infarction. The initial electrocardiogram suggested anteroseptal infarction, and subsequent tracings showed evidence of muscle healing, with the area of infarction becoming well localized. In this patient, as in the patient presented by Cooley et al., the rim of the perforation was surrounded by firm scar tissue with adjacent healthy cardiac muscle. The apical location of the septal rupture was characteristic of an acquired defect in
will have well-localized infarcts and can be supported medically until cardiac-catheterization studies and surgical closure can be performed.

Summary

A 61-year-old patient who had a ventricular septal defect after acute myocardial infarction was found to have a 4:1 shunt by cardiac catheterization and selective angiocardiography. Surgical closure was accomplished with the utilization of cardiopulmonary bypass and resulted in marked clinical improvement. Dye dilution studies 4 months postoperatively revealed no evidence of residual shunt. The patient is asymptomatic 8 months after surgery.

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References