Acute Constrictive Epicarditis Following Infectious Mononucleosis

Case Report

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IT IS KNOWN that infectious mononucleosis may be complicated by myocarditis and pericarditis. This report concerns a young man in whom pericarditis and cardiac tamponade developed into acute constrictive epicarditis in the course of this disease. The clinical diagnosis of constriction was proved by hemodynamic studies and by operation, which was followed by complete recovery.

Cardiac involvement in infectious mononucleosis was reported as early as 1914 by Kirkland and Pruen.\(^1\)\(^-\)\(^2\) They described "cervical adenitis with cardiac complications" in 14 out of 60 patients. However, the evidence for heart involvement was confined to electrocardiographic changes. The Armed Forces Institute of Pathology in Washington has reported that six out of 19 fatal cases of infectious mononucleosis had myocarditis. Pericarditis was first reported in 1946 by Evans and Graybiel.\(^4\) One of their patients was a 20-year-old man in shock due to massive pericardial effusion.

Case Report

Our patient was a 24-year-old man (W.P.), who was admitted to the Toronto Western Hospital on July 24, 1959. Six weeks previously he had a sore throat, followed by slight fever, cough, and pain in the chest. He developed shortness of breath, fullness in the abdomen, vomiting, weakness, and decreasing urinary volume.

On admission he was acutely ill with dyspnea, cyanosis, and jugular venous distention. His blood pressure was 112/105 mm. Hg. No paradoxical pulse was noted. The apical rate was 95 per minute and regular. The heart sounds were of good quality. There was dullness to percussion with diminished breath sounds over the right chest posteriorly. The liver edge was at the level of the umbilicus. His temperature was 101 F.

His chest x-ray on admission (fig. 1) showed blunting of the right costophrenic angle and a globular appearance to the heart. The electrocardiogram on July 27, 1959, showed negative T waves in the limb leads as well as negative T in $V_a$.

Laboratory data on admission were as follows: Hemoglobin 90 per cent, white cell count 7,000, with 35 per cent lymphocytes and 15 per cent atypical monocytes. Paul Bunnell agglutination was positive in a dilution of 1:1792. The non-protein nitrogen was 64 mg. per cent. The total proteins were 5.6 Gm., with a normal electrophoretic pattern. The urine was normal on admission and remained so throughout his hospital stay.

Two weeks after admission he developed peripheral edema, bilateral pleural effusions, and a paradoxical pulse of about 15 mm. Hg. The voltage was decreased in the limb leads of the electrocardiogram. On August 10, 1959, he became unconscious twice after severe coughing. The same day 600 ml. of straw colored fluid were removed from his pericardium.

His chest x-ray immediately following pericardiocentesis showed a small amount of air in the pericardial sac (fig. 2).

The pericardial aspiration was followed by transient clinical improvement. Three days after the first pericardiocentesis he developed thrombocytopenic purpura with a platelet count of 70,000. The bone marrow film was compatible with a hemolytic process, although the hemoglobin remained around 90 per cent.

For the first 4 weeks, the patient's daily urinary output was less than 1,000 ml., despite repeated injections of Thiomerin, oral diuretics, and varying amounts of Prednisone. At the end of that time he had a diuresis, during which he lost most of his peripheral edema but retained his pleural and pericardial effusions. The diuresis was followed by a marked drop in his serum sodium and chlorides to 123 and 72 mEq. per liter respectively. He also developed an acute psychosis with hallucinations. After a week of intensive treatment, the low-sodium syndrome was corrected, his psychosis cleared, and the thrombocytopenic purpura disappeared. In August, the patient had eight pleural and four pericardial aspirations. In September and most of October his condition was stationary. Figure 3 is representative of his many chest x-rays during this period. His weight was...
Circulation. Volume XXIII, February 1961

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steady around 185 lb. in spite of oral diuretics and repeated injections of Thiomerin. The liver remained at the level of the umbilicus. The jugular venous distention was unchanged, and his pulse pressure rarely exceeded 30 mm. Hg.

Two and a half months after admission, on October 6, a cardiac-output estimation was done by the dye-dilution technic (fig. 4). The appearance time was 20 seconds. The cardiac output was 11.12 liters per minute. The cardiac index was 5.25 liters per minute per M.² Blood volume estimation was within normal limits.

Toward the end of October his condition deteriorated. He started to gain weight, despite continued diuretic treatment, and again developed gross peripheral edema and ascites. His weight reached a maximum of 206 lb. On November 11, 3½ months after admission, he was transferred to the Metabolic Unit where urinary sodium estimations were done. In figure 5 the urinary output and sodium excretion, together with the dietary sodium intake and weight, are shown. With each injection of Thiomerin diuresis with maximal sodium excretion occurred. Between injections the urinary output was low and the sodium excretion practically zero, this despite a sodium intake of less than 400 mg. a day. His weight fell on this rigid sodium restriction from 206 to 183 lb. and then remained stationary. He still had jugular venous distention, pleural effusion, pericardial effusion, and gross ascites requiring abdominal paracentesis several times. The liver remained at the level of the umbilicus.
Eight weeks after the first study, a repeat cardiac output estimation was done (fig. 6). The appearance time, which had been 20 seconds, was now prolonged to 27 seconds. The cardiac output fell from 11.12 to 4.19 liters per minute. Fluoroscopic examination showed almost total absence of cardiac pulsations. A diagnosis of acute constrictive pericarditis was made and the patient was referred for surgery (D.R.W.).

Before operation, on December 2, 1959, 4,500 ml. of ascitic fluid and 1,500 ml. of right pleural fluid were removed. When the left chest was opened by an anterior fourth interspace incision, another 1,200 ml. of fluid were obtained. There was almost no cardiac movement on inspection and palpation.

The thickened pericardium was incised and approximately 300 ml. of blood-stained fluid were obtained. There was a moderate amount of granulation tissue between the visceral and parietal layers of the pericardium. As the parietal pericardium was removed, there was little improvement in cardiac movement and it was apparent that the primary constrictor was the epicardium. The adherent, thickened visceral layer of the pericardium was then removed from the left side of the heart, the right ventricle, and part of the right atrium. With the release of the constricting epicardium there was immediate improvement in cardiac filling and function, and moderate dilatation occurred.

The postoperative course was uneventful. His pulse pressure, which preoperatively rarely exceeded 30 mm. Hg widened to 65 mm. Hg. The paradoxical pulsation and jugular venous distention disappeared. The pleural effusions and ascites cleared, and the liver was no longer palpable. He required no further diuretics, and the urinary sodium excretion became normal.

The chest x-ray (fig. 7) taken 2 weeks postoperatively was normal. The electrocardiogram showed deep inversion of precordial T waves.

The patient was discharged after 5½ months in the hospital, and on a follow-up visit in March 1960 he had no symptoms and examination was within normal limits.
Discussion

We have attempted to rule out any underlying disease process other than infectious mononucleosis that could have accounted for this patient’s acute constrictive pericarditis. Thirteen smears and cultures and guinea pig inoculations from his pleural, pericardial, and ascitic fluid and from his sputum failed to show tubercle bacilli. Tuberculin tests have remained negative. Psittacosis has been ruled out by agglutination. Virus studies were made by inoculating sputum, throat washings, urine, stool, blood, pleural fluid, pericardial fluid, and pericardial tissue. No virus was isolated in over 13 passages in monkey kidneys and human amnion, chic embryo, and virus cultures. Lupus erythematosus cells were absent on repeated examinations from the peripheral blood and bone marrow. The pathologic report on the epicardium and pericardium, removed at operation, showed only chronic inflammatory tissue having no specific characteristics.

By March 1957, 12 cases of infectious mononucleosis with pericarditis had been reported. The review by Shugoli\(^5\) stated that six of them had a friction rub, one a massive pericardial effusion, and the others only electrocardiographic evidence of acute pericarditis. Gardner\(^6\) reported one case of acute pericarditis associated with infectious mononucleosis and Hutchinson\(^7\) three cases.

Sixteen cases have been reported in the literature in which infectious mononucleosis alone seems to be the etiologic agent responsible for acute pericarditis. These cases are all well documented with clinical, electrocardiographic, and radiologic evidence of acute pericarditis; however, only two of these patients\(^4,5\) had massive pericardial effusions requiring pericardiocentesis. Our case is reported because it shows an interesting sequence of events: acute pericarditis followed by tamponade and constrictive epicarditis within 5 months of the onset of infectious mononucleosis.

Summary

The case history of a 24-year-old man is reviewed, who had pericarditis, cardiac tamponade, and many other manifestations of infectious mononucleosis. Cardiac-output estimations during the course of the illness showed a marked drop in his cardiac index. This, together with other findings, was interpreted as being due to the development of constrictive epicarditis. At operation the epicardium was densely adherent to the underlying myocardium. After extensive resection of both layers of the pericardium the patient made an uneventful recovery.

Acknowledgment

We wish to thank Dr. A. Rapoport, Director of the Metabolic Unit, Toronto Western Hospital, Toronto, for carrying out the metabolic studies in this case.

References

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Circulation. 1961;23:257-260
doi: 10.1161/01.CIR.23.2.257

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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