**CASE REPORT**

In January 1988, a 67-year-old woman was admitted to our hospital with a history of progressive breathlessness during the last two years. She had never smoked and had no history of pulmonary diseases. A chest roentgenogram was made 30 years ago because of a tuberculous contact. She remembered that she had been told that she had an innocent lesion on the chest roentgenogram.

Physical examination at the time of hospital admission revealed mild dyspnea but no other abnormalities. The chest roentgenogram showed an enormous mass with extensive central calcifications (Fig 1). The chest roentgenogram from July 1959 showed the same mass; however, it was smaller and without calcifications. Spirometry, laboratory, and electrocardiographic findings were normal. Computed tomography of the thorax confirmed a large calcified mass in the anterior mediastinum without connections with the heart.

At thoracotomy, a mediastinal tumor, 13 × 11 × 6 cm, weighing 700 g, was removed. The arterial and venous supply arose from subclavian vessels.

Histologic examination of the resected specimen showed a mainly diffuse and focally follicular proliferation of lymphoplasmacytoid cells divided by fibrous tissue with foci of calcified material. The lymphoid proliferation did not respect the borders of the central tumor and invaded the surrounding fatty tissue (Fig 2), leading to the diagnosis of lymphoplasmacytoid NHL of low malignancy, probably as transition from a giant lymph node hyperplasia (Castleman's disease).

**DISCUSSION**

Giant lymph node hyperplasia was initially reported as a solitary mediastinal mass, but multicentric and extranodal disease is now well known. Although the localized form is occasionally asymptomatic, often general symptoms of fatigue, pain, fever, anemia, and sometimes hyperimmunoglobulinemia are present. Histologically, two distinct variants are recognized: the hyaline-vascular type, showing hyalized follicle-like structures with extensive capillary proliferation, and the plasma-cell variant with the presence of sheets of mature plasma cells and normal-to-large-sized follicle centers.

Clinically, nodal changes like Castleman's disease have been found in association with a number of different diseases such as acquired immunodeficiency syndrome (AIDS), Kaposi's sarcoma, rheumatoid arthritis, autoimmune diseases, and in lymph nodes draining Hodgkin's disease or NHL. In terms of differential diagnosis, lymphoplasmacytoid NHL should be considered. Since it is very unlikely to survive for 30 years with an untreated NHL, we regard the initial mass in the anterior mediastinum as a giant lymph node hyperplasia with a malignant transformation to NHL. A relation between the two conditions has already been described, but a transition into NHL has not yet been reported (to our knowledge).

The treatment of choice of giant lymph node hyperplasia is surgical removal, but due to the presence of NHL in this particular patient, radiotherapy was instituted postoperatively. Two years after removal of the tumor, no signs of recurrence were present.

**REFERENCES**


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**Massive ST-Segment Elevation without Myocardial Injury in a Patient with Fulminant Hepatic Failure and Cerebral Edema**

Alan J. Rosenbloom, M.D.†

A 49-year-old woman presented in fulminant hepatic failure. The ECG showed dramatic ST-segment elevation, suggesting diffuse myocardial injury. However, echocardiography, creatine phosphokinase enzyme determinations, and examination of the heart at autopsy (six days later) failed to demonstrate any physiologic, anatomic, or histologic evidence of abnormality. The appearance of ST-segment elevation in this setting should not prompt treatment for cardiac disease or limit the candidacy for liver transplantation of such critically ill patients.

(Chest 1991; 100:570-72)

**CFK = creatine phosphokinase, FHF = fulminant hepatic failure**

The appearance of ECG changes suggestive of myocardial injury in the absence of myocardial cell necrosis, known as pseudoinfarction, has been well described in many textbooks and articles. To the best of our knowledge, however, this is the first report of its association with fulminant hepatic failure (FHF) and cerebral edema.

**CASE REPORT**

A previously well 49-year-old woman presented with nausea, vomiting, and malaise of four days' duration. The ECG obtained on admission and a chest x-ray film were normal. The patient was alert and oriented. Severe acidosis (pH, 7.02; serum bicarbonate, 4.1 mEq/L) and hypoglycemia (glucose, 14 mg/dl) were present. During the next 24 hours she became increasingly jaundiced and obtunded. Extremely high levels of liver transaminases (11,000 to 14,000 IU/L) and severe coagulopathy (prothrombin time, 41 s) were noted. The diagnosis of FHF was made. Large volumes of intravenous fluid, fresh-frozen plasma, and sodium bicarbonate were infused. Thirty-three hours after admission, the patient was transferred to our institution to be considered for liver transplantation.

Her laboratory results on admission to our hospital were remarkable for a serum sodium concentration of 160 mEq/L (normal, 136 to 146 mEq/L) and a serum osmolality of 367 mOsm/kg (normal, 275 to 299 mOsm/kg). Serum magnesium, ionized calcium, potassium, and creatinine and blood urea nitrogen levels were normal. The serum phosphate level was 1.3 mg/dl (normal, 2.5 to 4.5 mg/dl). The total serum bilirubin level was 7.1 mg/dl (normal, 0.3 to 1.5 mg/dl). The chest radiograph showed pulmonary edema.

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The patient was stuporous, with response only to pain. The neurologic examination findings were nonfocal: no clinical signs of brain stem compression were present. A computed tomographic scan of the head showed small ventricles and loss of sulci, suggesting diffuse cerebral edema. A bedside cerebral blood flow study was performed with intravenous xenon 133. This showed a marked, diffuse elevation of flow with an average of 79.8 ml X 100 \text{ min}^{-1} (normal value, adjusted for age and Pco_2, 32 to 48 ml X 100 \text{ min}^{-1}). Endotracheal intubation was performed, and vigorous hyperventilation was begun with an Fio_2 of 50 percent.

The initial ECG showed junctional tachycardia, with ST elevation of up to 2.5 mm, primarily in the lateral V leads (Fig 1). During the

![Figure 1](image1)

**Figure 1.** ECG taken on admission. Note ST-segment elevation in leads V₁, V₆.

![Figure 2](image2)

**Figure 2.** ECG obtained 12 h after admission. Diffuse, marked ST elevation persisted for more than three days on numerous tracings.
next 12 h, ST elevation became diffuse and dramatic (Fig 2). Diffuse myocardial injury, pericarditis, myocarditis, and electrolyte disturbance were the differential diagnoses. An echocardiogram revealed no pericardial effusion and no wall motion abnormality; in fact, the heart was hyperdynamic. Pulmonary artery catheterization data supported this finding, with a mixed venous oxygen saturation of 76 percent and a cardiac index of 5.58 L X kg⁻¹ X min⁻¹. Creatine phosphokinase (CPK) analysis was performed. Three samples were drawn 12 h apart; a fourth, 24 h later. The total values were 1,306, 1,513, 1,345, and 2,368 IU/L, respectively, with MB fractions of 3.6 percent, 1.8 percent, 2.3 percent, and 2.1 percent. In our laboratory, a total CPK level of 0 to 300 IU/L is considered normal. When the total CPK concentration is elevated, we feel that an MB fraction less than 4 percent excludes significant myocardial necrosis.

Multisystem organ failure rapidly developed, and the patient's condition was deemed too unstable for liver transplantation. Despite continued treatment, she died six days later. An autopsy was performed. The examination of the heart revealed no pericarditis or pericardial fluid. Grossly, the endocardium, myocardium, and valves were normal. Small petechiae were present on the epicardial surface. Microscopically, the myocardium appeared normal, and sections of the left anterior descending artery were unremarkable. Massive necrosis of the liver (weight, 450 g) was present with very little fibrosis, reflecting an acute, possibly viral insult.

**DISCUSSION**

There are several possible causes of ST segment elevation in this patient. A particularly serious and often fatal complication of FHF is severe cerebral edema. Abnormal cerebral blood flow has been well documented in FHF. This patient manifested both heightened blood flow and edema. This combination is apparently due to loss of autoregulation of cerebral blood flow in FHF. Many reports have linked catastrophic intracerebral events, such as subarachnoid hemorrhage, head trauma, tumors, meningitis, and stroke, with ST-segment elevation. It is possible that cerebral edema and increased intracranial pressure caused ST-segment elevation.

Abnormality of electrolyte levels, particularly hyperkalemia, can cause ST elevation identical to that present with infarction. Although a number of electrolyte levels were abnormal, most occurred before the ECG changes developed. In addition, the correction of abnormal levels would be expected to reverse related ECG changes; this did not occur.

Abnormal systemic and organ blood flow have been increasingly recognized in hepatic failure states. As already noted, altered cerebral blood flow is often detectable with xenon-tracer studies. Significantly abnormal pulmonary vascular tone appears to be a common cause of ventilation-perfusion mismatching and hypoxia in cirrhotic patients. Resultant hypoxia can be severe, although it is often well compensated for by hyperventilation. Renal failure in the hepatorenal syndrome is apparently related to severe renal cortical vasoconstriction. This phenomenon has also been shown to be important in renal failure accompanying FHF. Arteriovenous shunting has been suggested as the cause of the low systemic vascular resistance seen in hepatic failure. In cases of FHF, extremely low systemic vascular resistance accompanied by frank tissue hypoxia is a poor prognostic sign, signaling the onset of MSOF. Maldistribution of cardiac blood flow and global myocardial ischemia may have occurred by a similar mechanism in this patient. Although cardiac output appeared well preserved, this may have been an artifact of extremely low systemic vascular resistance. The afterload placed on the heart is known to be a major determinant of ejection fraction. With a diminished afterload, as seen in this case, even an ischemic left ventricle will maintain a high cardiac output.

The nonspecificity of even very dramatic ECG changes has been documented repeatedly. The superb review by Marriott, which he describes as "cursory and incomplete," catalogs more than 100 causes of apparent ischemia or infarction on the ECG. Many are related to extracardiac disease.

In conclusion, the cause and significance of the massive ST-segment elevation in this patient remain uncertain. Further ECG, functional, and anatomic studies of the heart in FHF are needed. Thallium scanning may be able to define regional or global ischemia, if present. If cardiac circulatory abnormalities are demonstrated in this setting, it would be particularly interesting to see whether these resolve after liver transplantation.

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Massive ST-Segment Elevation without Myocardial Injury (Alan J. Rosenbloom)