Acute and Transient ST Segment Elevation During Bacterial Shock in Seven Patients Without Apparent Heart Disease*

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Acute elevation of the ST segment in several ECG leads was observed in seven patients with bacterial shock during the course of therapy. Six patients had bacterial pneumonia, one had acute cholecystitis, and none had a previous history of heart disease. At the onset of the ST elevation, all patients were receiving dopamine infusion, which in four of them was inadvertently increased shortly before the ECG changes. The ST elevation was not associated with chest pain, pericardial friction rub, or acute changes in the heart rate, or arterial blood pressure. In four patients the maximum ST elevation was ≥ 5 mm. In each instance the ST segment returned to the isoelectric line within 24 hours, and subsequent development of Q waves or changes in the QRS was not observed. Although the existence of an acute pericarditis or an acute myocarditis as possible causes of the ST elevation cannot be fully ruled out, the sudden onset, prominent magnitude, and brief duration of the ST elevation are perhaps more indicative of an acute ischemic event, possibly related to a transient coronary vasospasm induced by the dopamine infusion.

Electrocardiographic changes in the course of shock have been demonstrated in patients with or without ischemic heart disease and in the experimental laboratory. These changes usually reflect subendocardial ischemia and are found most frequently in hemorrhagic shock. They are presumably caused by a decrease in coronary perfusion pressure, severe anemia, or both.

To our knowledge, the occurrence of an acute and transient elevation of the ST segment in patients with bacterial shock submitted to a variety of therapeutic regimens has not been previously reported. We describe such changes in the ECGs of seven patients without history of heart disease who had received dopamine. We discuss the possible causes of these acute ST elevations in light of the clinical, ECG, laboratory, and hemodynamic findings, and consider the likelihood of dopamine as the triggering factor.

Case Reports

Case 1

This was a 57-year-old man, a heavy smoker, who had no relevant past medical history. Three days prior to admission, he had fever, right pleuritic chest pain, asthenia, and progressive dyspnea. On admission he was hypotensive and had clinical signs of shock and of respiratory insufficiency. Chest x-ray examination demonstrated a right upper lobe pneumonia, and a normal cardiac size. The ECG was within normal limits (Fig 1). Treatment was begun with penicillin G, intravenous (IV) fluids, and dopamine (3.20 µg/kg/min), which resulted in a progressive improvement. Two hours and 15 minutes later, however, a sudden, asymptomatic ST segment elevation occurred, and the dopamine infusion was stopped. Subsequent ECGs revealed a return of the ST segment toward the isoelectric line which was already apparent 3½ hours later. The patient was discharged 17 days after admission.

Case 2

A 46-year-old woman with a 16-year history of bronchial asthma had developed fever, cough, chills, left pleuritic chest pain, and increasing dyspnea 24 hours before admission. On arrival at the emergency room she was hypotensive and was in severe respiratory distress. Chest x-ray film revealed a left lower lobe pneumonia and a normal heart size. The ECG was interpreted as normal. She was mechanically ventilated and treated with IV fluids, methylprednisolone, penicillin, gentamycin, and furosemide. In view of the poor response, a dopamine infusion was initiated at 4.0 µg/kg/min and increased shortly thereafter to 10.0 µg/kg/min. Four hours later an acute elevation of the ST segment was noted, which prompted discontinuation of the dopamine infusion. Return of the ST segment to the isoelectric line was seen on the ECG taken 11 hours later. The patient was extubated four days later and discharged 20 days after admission.

Case 3

This was a 31-year-old woman with a past history of...
cholelithiasis. Two days before admission she experienced precordial pain, fever, chills, asthena, cough, and progressive dyspnea. In the hospital she presented with clinical signs of shock and respiratory insufficiency and with physical and radiologic evidence of a right lower pneumonia. An ECG showed no significant abnormalities. Treatment was started with IV fluids, penicillin G, and gentamycin. Dopamine was added shortly thereafter at 3.0 \( \mu g/kg/min \). Two hours later an acute, asymptomatic elevation of the ST segment was observed. The dopamine drip was stopped after realizing that its infusion rate had been inadvertently increased to approximately 40 \( \mu g/kg/min \). Sixteen hours later, however, these ECG changes had reverted. She was discharged 14 days after admission.

**Case 4**

This was a 48-year-old man, with a past history of chronic lung disease. Twenty-four hours prior to admission, he complained of left pleuritic chest pain, dyspnea, fever, chills, asthenia, nausea, vomiting, and diarrhea. Clinical signs of shock and marked respiratory insufficiency were apparent on admission. A less than 1.0-mm ST segment elevation in leads 2 and V₆ through V₈ was the only abnormality in the ECG (Fig 2). Physical examination and chest x-ray film showed evidence of a left lower lobe pneumonia. Intravenous fluids, penicillin G, and tobramycin were administered, followed by a dopamine infusion at 1.6 \( \mu g/kg/min \). Twelve hours later, an asymptomatic acute ST segment elevation developed, associated with an accidental increase in the dopamine dose to approximately 30 \( \mu g/kg/min \). The dopamine infusion was immediately discontinued. The sequential ECG changes showed a progressive reversal of the ST segment elevation, already remarkable one hour after its onset. He was discharged 18 days after admission.
Case 5

This 34-year-old man had a history of pulmonary tuberculosis at the age of 17 years. Forty-eight hours prior to admission he complained of left pleuritic chest pain, dyspnea, fever and asthenia. On arrival at the hospital he showed clinical signs of shock and acute respiratory distress, and had clinical and radiologic evidence of a left lower lobe pneumonia. A 12-lead ECG was normal except for a moderate prolongation of QT interval (Fig 3). Therapy included penicillin G, digoxin, furosemide, and dopamine at 5.6 μg/kg/min, with which a rather quick clinical improvement was appreciated. Three hours later, however, an acute and painless ST segment elevation was noted, associated with an accidental increase in the dopamine rate to approximately 45 μg/kg/min, which was immediately stopped.

Subsequent ECGs showed a progressive return of the ST segment to the isoelectric line, already significant within the first two hours after stopping the dopamine infusion. The patient was discharged 19 days after admission.

Case 6

This 44-year-old man, a smoker, was affected by a systemic collagen disease. He had begun to experience asthenia, fever, chills, nausea, and vomiting, pleuritic chest pain, and dyspnea two days prior to admission. He was admitted in mild respiratory distress, was hypotensive with clammy skin, and had clinical and radiologic signs of right lower lobe pneumonia. An ECG was within normal limits. Fluid replacement, antibiotics, and dopamine constituted the initial treatment. The dopamine infusion, at 14.0 μg/kg/min, had to be discontinued six hours later upon observation of an acute asymptomatic elevation of the ST segment, which gradually returned to predopamine level within the ensuing 20 hours. The patient left the hospital 15 days after admission.

Case 7

This was a 59-year-old man, a smoker, with a past history of chronic lung disease. Three days before admission he experienced nausea, vomiting, abdominal pain, fever, and chills. On arrival at the emergency room he had signs and symptoms of acute cholecystitis and shock, reversed less than two hours after beginning therapy with fluids and dopamine at 3.0 μg/kg/min. Two and one-half hours later, immediately following a flushing of the dopamine infusion line, however, an oppressive discomfort in his chest, some respiratory difficulty, nausea associated with clammy skin, a moderate increase in blood pressure, and an elevation of the ST segment were noted. The dopamine infusion was immediately stopped, and full recovery from the acute event was noted 50 minutes later, when the ST segment had returned to almost the isoelectric line (Fig 4).

The patient underwent a cholecystectomy for acute cholecystitis within 24 hours and was discharged from the hospital 15 days later.

Discussion

We report the observations of transient ST elevation during treatment of bacterial shock in seven patients who had no history of heart disease. In four patients the maximum ST elevation was equal or greater than 5 mm. In all patients, the ST segment returned to the isoelectric line within 24 hours, and in five it decreased to 50 per-
cent of the maximal elevation within the first six hours.

Although duration of the ST elevation varied, in no instance did it exceed 24 hours. In addition, no changes in the QRS complex or development of Q waves were seen. Flattening or slight inversion of T waves, however, was documented in five patients.

The significance of these ST segment changes is not clear to us. The contention that they may represent transmural ischemia produced by an acute coronary vasoconstriction is disputed by the lack of correspondence between the ECG distribution of the ST elevation observed in our patients and that caused by occlusion of a major coronary branch. As opposed to our findings, furthermore, ECG changes due to coronary spasm have a much shorter duration and very seldom involve primarily the lateral leads. On the other hand, an acute reduction of the coronary blood flow as a cause of these ST elevations is strongly suggested by the fact that during the elevation each patient was receiving dopamine, a drug which, according to some experimental studies, may produce increase in coronary vascular resistance. Nayler et al have demonstrated local coronary vasoconstriction when dopamine, even in small doses, was injected directly into a coronary artery of a dog. Apparently, such vasoconstriction is due to stimulation of α-receptors and can be abolished by α-blocking agents. When dopamine is administered systemically, however, the vasoconstrictor effect on coronary arteries may be masked by the vasodilation that follows increases in myocardial oxygen demands made by increases in heart rate, arterial blood pressure, and contractility. The marked, sudden increase in dopamine dose in four patients just before the ECG changes, the fact that the ST segment began to decline within 30 minutes after stopping the dopamine infusion, and the immediate ST elevation seen in patient 7 following an accidental bolus of dopamine further suggest a possible case-effect relationship between the drug and the ECG changes.

Myocardial ischemia associated with the use of dopamine, if present in our patients, was probably not related to an increase in myocardial oxygen demands, as previously documented in experimental and clinical myocardial infarction, but to the cited dopamine-linked coronary vasoconstrictor effect, inasmuch as most patients experienced little changes in arterial blood pressure and heart rate during the ST elevation.

We recognize that the ECG changes recorded in our patients are at least partially compatible with the diagnosis of pericarditis. Upward concavity of the ST elevation, presence of peaked T waves in several patients, and involvement of the leads most frequently affected by pericarditis are sugges-

Figure 4. Sequential ECGs (case 7). Admission ECG (top to bottom) 0 hr, just before cessation of dopamine administration and immediately after accidental flushing of the dopamine infusion tubing, shows prominent ST segment elevation in lateral leads, not observed in subsequent ECGs at 50 min, 6.0 hr, and 40.0 hr later. Generalized flattening of T waves apparent in ECG on day 5 (5 d). Although no previous ECG was available for comparison with first (0 hr), the ST segment was isoelectric before its elevation on the lead monitored on the oscilloscope.
tive of such a condition. The existence of pneumonia in six patients, not uncommonly associated with pericarditis, offers additional support for this cause. Here, too, however, several conflicting elements may preclude the certainty of this diagnosis. First, the acuteness of onset of the ST segment change for the highest ST elevation was reached in less than 30 minutes in each patient. Second, the duration of the ST elevation was limited to less than 24 hours, compared with the typical slow evolution of these changes in acute pericarditis. Third, in each instance, chest pain and pericardial friction rub were absent. Fourth, the ST elevation in four patients was equal to or greater than 5 mm, clearly more than that usually observed in pericarditis.

Since ST elevation has also been identified during acute myocarditis and the coexistence of pneumonia and myocarditis is well known, it is conceivable that such an association might have occurred in our patients, explaining the ST elevation. It is doubtful, however, because the ST elevation in acute myocarditis is often accompanied by QRS changes and followed by a noticeable T wave inversion, simulating an acute myocardial infarction.

We concluded, therefore, that in the patients described, the dopamine probably produced ischemia in a myocardium, normal or already affected by an infectious process, due either to an excessive dose or to a special sensitivity.

The practical importance of our findings can be centered on the need for careful and continuous monitoring of the ECG in patients receiving IV dopamine, particularly during the course of bacterial shock related to pneumonia, and considering the convenience of stopping its infusion in the event of an elevation of the ST segment. Moreover, due caution should be exercised to avoid accidental increases in the perfusion rate or flushing of IV tubing containing the drug. Further studies are necessary, however, to provide more convincing evidence of the participation of this drug on the genesis of the cited ECG changes.

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448 BRUGUES TERRADELLAS ET AL
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