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Postoperative Myocardial Ischemia Possibly Masked by Epidural Fentanyl Analgesia

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Epidural and intrathecal opioid administration has proven to be an effective analgesic therapy for a variety of painful states. These include cancer, the postoperative period, the first stage of labor, posttrauma, and myocardial infarction.[1] This report describes a patient with a history of coronary artery disease who had asymptomatic myocardial ischemia while being treated with a continuous epidural infusion of fentanyl for postcholecystectomy pain. It is postulated that the epidural fentanyl infusion may have masked the pain of myocardial ischemia.

CASE REPORT

An 85-yr-old, 66-kg man was admitted to the hospital with a 1-week history of right upper quadrant abdominal pain. A diagnosis of acute cholecystitis was made. Despite treatment with antibiotics he did not improve and was scheduled for cholecystectomy.

The patient had an 8-yr history of coronary artery disease with exertional angina and on four prior occasions had suffered subendocardial infarctions, the last of which occurred 23 months prior to admission. On each of these occasions he had substernal chest pain with radiation to the left arm. During each subendocardial infarction, his electrocardiogram (ECG) showed temporary ST-segment depression and T-wave inversion in leads V2–V4 without progression to Q waves (fig. 1). His creatine phosphokinase (CPK) MB isoenzyme fraction was mildly increased with each episode. Prior to the incident reported here, he had had angina once or twice per month and had been walking 2–3 miles each day, using sublingual nitrates prior to exertion. His past medical history was otherwise unremarkable. He had no history of diabetes mellitus. His admission ECG revealed a first degree atrioventricular (AV) block and slightly reduced voltage.

The patient had an uneventful cholecystectomy. The anesthesia for his operation was continuous lumbar epidural anesthesia (2% lidocaine) combined with general endotracheal anesthesia, fentanyl (20 μg/kg), and oxygen. The V5 lead was monitored intraoperatively and demonstrated no evidence of ischemia. Postoperative pain was treated with a continuous epidural infusion of fentanyl (5 μg/ml saline) infused at 10 ml/h. With this regimen he was pain-free except for minimal discomfort during coughing. The ECG during the immediate postoperative period and on the 1st postoperative day were unchanged from admission.

On the 2nd postoperative day, while still in the intensive care unit, his ECG monitor showed ST-segment depression. A 12-lead ECG revealed 2–3-mm ST-segment depression and T-wave inversion in leads V2–V5. (fig. 2). He was completely awake and alert and had no pain. Intravenous nitroglycerin (250 μg/min) resulted in normalization of the ST segments, but the T waves remained inverted. His CPK measurement after this episode of myocardial ischemia peaked at 505 units, with a 4% MB isoenzyme fraction (normal is less than 5%). He remained in the ICU for 3 days after this episode without recurrence of ST-segment depression. After the ischemic event, he was treated with nifedipine, propranolol, and isosorbide dinitrate. The isosorbide dinitrate was started after the 2-day infusion of nitroglycerin was discontinued. Epidural fentanyl was discontinued on the 4th postoperative day.

The ECG T-waves remained inverted for the remainder of his hospitalization. He had no episodes of symptomatic myocardial ischemia during this hospitalization, and the remainder of his recovery was uneventful. He was discharged home on the 15th hospital day. He continued to have New York Heart Association class II angina after discharge.

DISCUSSION

A variety of treatments for the pain of myocardial ischemia have been used. In 1899 Francois-Franch advocated sympathectomy for relief of angina pectoris.[2] Cervical sympathectomy, however, relieved symptoms in only 60% of the cases treated.[3] The discovery of the thoracic cardiac nerves led to more extensive and/or better localized sympathectomy by a variety of means, including paravertebral neurolysis, posterior rhizotomy, and thoracic ganglionectomy. As many as 80% of patients treated by these methods have been reported[2] to be angina free after treatment.

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Modern therapy for myocardial ischemia pain has centered on the pharmacologic manipulation of myocardial oxygen supply and demand using nitrates, β-adrenergic blockers, and calcium channel blockers. However, even with modern therapy, patients having acute myocardial ischemia may experience excruciating discomfort, which may be refractory to intravenous opioids. As newer modes of analgesic therapy, such as spinal narcotics (intrathecal or epidural), have been explored, there has been renewed interest in the myocardial pain pathways.

The afferent nerve supply of the heart begins as undifferentiated or beaded nerve endings in the myocardial walls. These are unmyelinated A delta and C fibers that course along the major vessels, their branches coalescing to form the cardiac plexus lying between the aortic arch and the trachea. From the cardiac plexus, the afferent impulses are transmitted in the cardiac sympathetic nerves to the lower two cervical and upper four thoracic sympathetic ganglia, where the nerve cell bodies are located.[4] The central axon from the sympathetic ganglia transmits the afferent impulses to the substantia gelatinosa of the dorsal horn of the spinal cord. In the substantia gelatinosa are synapses with interneurons and centrally projecting neurons. The impulses are relayed *via* the spinothalamic tracts to the thalamus and the cerebral cortex, where awareness of pain occurs. It is in the substantia gelatinosa

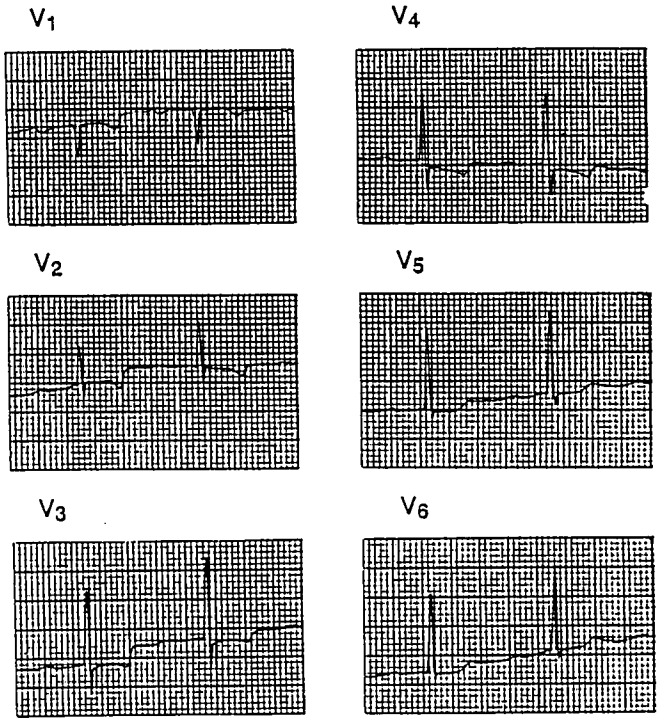

FIG. 1. ECG demonstrating ST depression and T-wave inversion in leads V2–V4 during the patient's previous subendocardial myocardial infarction.

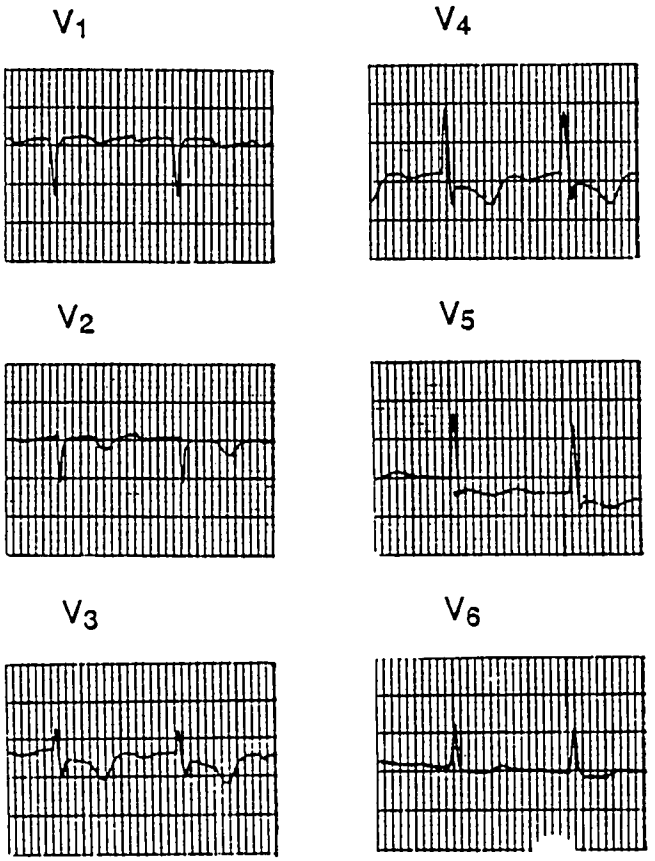

FIG. 2. ECG demonstrating second postoperative day ST depression and T-wave inversion in leads V2–V5 prior to the institution of nitroglycerin infusion.

that intraspinal narcotics have their effect in modulating myocardial ischemia pain.[5]

The first report of clinical application of intraspinal opioid administration for cardiac pain was in 1979 when Pasqualucci *et al.*[6] reported the use of intrathecal morphine sulfate for the treatment of the pain of acute myocardial infarction. Morphine sulfate 0.5 mg was injected, and the analgesia obtained was compared to control patients receiving intravenous or intramuscular injections of morphine or pentazocine. Superior analgesic effects were observed with intrathecal administration and forty times less drug was needed. In 1985 Skoeld *et al.* reported the first use of lumbar epidural morphine sulfate in patients with acute myocardial infarction.[7] Analgesia was maintained by the epidural injection of 1–2 mg morphine at 4–12-h intervals.

Further evidence for this pain pathway and its spinal modulation comes from two other reports. Clemensen *et al.* reported seven patients who continued after coronary artery bypass graft to have angina and were successfully treated with chronic epidural morphine administered *via* an implanted system for 3–11 months.[8] Murphy and Gibes

reported ten patients with intractable angina pectoris despite medical therapy who were successfully treated with implanted epidural dorsal column stimulation electrodes.[9] Six of the ten patients continued to have successful pain amelioration with observation of up to 5 yr.

The patient in the current case report could well have had "silent" or asymptomatic myocardial ischemia unrelated to the epidural fentanyl. Silent ischemia is being recognized with increasing frequency in patients with coronary artery disease, and asymptomatic myocardial infarctions occur in the majority of patients suffering postoperative infarctions.[10,11] However, the current patient's history of painful myocardial ischemia in the past and similar ECG findings with this episode suggest that sensation of pain from myocardial ischemia was blocked by the continuous epidural infusion of fentanyl.

Pain is a valuable warning signal of myocardial ischemia, and there are dangers to unrecognized and untreated ischemia, such as arrhythmias and progression to infarction. This report suggests that patients at high risk for postoperative myocardial ischemia who are treated with spinal opioids analgesics may be at risk for the masking of myocardial ischemia pain by the analgesic effects of the spinally administered opiates. In these patients, a high index of suspicion must be maintained, and they should have increased surveillance to enhance their safety. Surveillance may include appropriate ECG monitoring (*e.g.*, V5 lead), preferably with on-line ST-segment analysis and observation for secondary manifestations of myocardial ischemia such as decreased blood pressure or cardiac output, dyspnea, or increased pulmonary artery wedge pressure. These patients may then receive the benefits of spinal opioid analgesia with less risk of undetected myocardial ischemia.

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Epidural Abscess Associated with Epidural Catheterization: A Rare Event? Report of Two Cases with Markedly Delayed Presentation

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Epidural abscess is a rare condition, with an incidence of approximately 0.2 to 1.2 per 10,000 hospital admissions per year.[1] The incidence of epidural abscess associated with placement of epidural catheters is unknown, but considering the few published reports, apparently is very rare.[2–5] In addition, in these reports the abscess formation generally was acute in onset, occurring soon after epidural catheter placement. We report two cases of epidural abscess formation after placement of epidural catheters for pain management. In both, the manifestation of the abscess did not occur until nearly 1 month after catheter insertion.

Case Reports

Case One. A 71-yr-old man was referred to our Pain Clinic for the treatment of severe burning pain associated with a resolving acute herpes zoster infection involving the T5–T6 dermatome. His acute lesions were mostly healed, but he was taking oxycodone and acetaminophen with codeine without pain relief.

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Key words: Anesthesia techniques: epidural. Complications: infections. Pain: reflex sympathetic dystrophy; herpes zoster.