

Chest Pain of Esophageal Origin during Spinal Anesthesia

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Severe (left) chest pain is a serious problem because of its frequent association with myocardial ischemia or infarction. However, angina-like pain can originate from areas other than the heart, such as the cervical spine,¹ chest wall,² gallbladder, lungs, or esophagus.³⁻⁵ Among these, esophageal origin seems to be not uncommon. Esophageal dysfunction, whether it is reflux esophagitis⁶ or esophageal spasm,⁷ can produce an angina-like pain indistinguishable from that of cardiac origin in quality, distribution, and severity. The purpose of this case report is to call attention to the occurrence of this entity (angina-like pain of esophageal origin) during spinal anesthesia.

REPORT OF A CASE

A 70-year-old man was scheduled for a left below-knee amputation, with spinal anesthesia, because of early gangrene in the left fifth toe. A translumbar aortogram showed a patent femoral artery, but severe infrapopliteal occlusive disease on the left side.

Six months prior to admission, because of unstable angina, the patient had a coronary-artery bypass graft operation (CABG \times 4 vessel grafts). Since his open-heart surgery, he had been doing well, and had taken sublingual nitroglycerine only once. He had adult-onset diabetes and was taking hypoglycemic acetohexamide (Dymelor®) orally. His only other medication was an aspirin-Maalox® mixture (Ascriptin®). The patient had had a cholecystectomy several years previously, and had undergone transurethral resection of the prostate (TURP) with spinal anesthesia three months after CABG.

Physical examination disclosed no abnormality except the scars from his earlier operations. All laboratory data were within normal range except glucose, which was 211 mg/100 ml. An electrocardiogram showed an old inferior myocardial infarction.

After premedication with meperidine, 75 mg, and atropine, 0.4 mg, im, spinal anesthesia was administered with the patient in the sitting position, using tetracaine, 10 mg, 10 per cent dextrose in water, 1 ml, and epinephrine (1:1000), 0.3 ml, at the L3-4 interspace. Analgesia was achieved without difficulty. Oxygen was given via nasal cannula at a rate of 4 l/min. The level of analgesia was T10. Arterial blood pressure and pulse rate decreased slowly from 150/70 torr and 100 beats/min to 110/70 torr and 90 beats/min, respectively. Diazepam, 2.5 mg, was given intravenously.

Approximately 50 min after the induction of anesthesia, the patient began complaining of pain beneath his left lower rib cage. There was no change on the electrocardiogram. Another 2.5 mg of diazepam were given. However, the pain, which was of a pressure type, was becoming severe. The patient was not perspiring, but

his face appeared pale. Systolic blood pressure rose from 110 to 130 torr, and pulse rate from 90 to 100 beats/min. Meperidine was given iv, with no effect (total 30 mg). Sublingual administration of nitroglycerin failed to elicit relief of pain. Total dose of meperidine, 30 mg, diazepam, 7.5 mg, and sodium pentothal, 50 mg, were given during the 30 minutes, with no effect.

At the end of this time, the patient wanted to raise his head. The operating room table was flexed, and the head and back raised about 25 degrees. Several minutes after the position change, the patient felt somewhat better, although the severe pain was still present deep in the left lower chest. At this time, reflux esophagitis was suddenly suspected. Chewable Maalox tables (no. 2, Rorer) were given, with a sip of water. In approximately 4 minutes, the pain was "practically gone," by the patient's description.

While in the recovery room, the patient was interviewed in detail. Although the fact was not stated in his thick medical record, he had had heartburn quite often since open-heart surgery. He had taken sublingual nitroglycerin only once since open-heart surgery, and it had not relieved the chest pain. Tums® or Rolaid® tablets eased the pain, and belching relieved the pain almost immediately. He had had a similar episode of chest pain during the prostatic operation three months previously; it had been relieved by vomiting in the recovery room. Twelve-lead electrocardiogram and serial serum enzyme studies, including determination of creatinine phosphokinase-MB, disclosed no abnormality.

DISCUSSION

It is unlikely that myocardial ischemia or infarction caused this chest pain, in view of the nonresponsiveness to sublingual nitroglycerin administration and negative electrocardiograms and serum enzyme studies. On the other hand, the relief of pain on position change and in response to antacids, and the history, suggest that this pain was probably due to reflux peptic esophagitis, although esophageal acid perfusion test⁸ to produce the same pain was not done in this case.

With reflux esophagitis, the supine position provokes substernal heartburn and other symptoms. The supine position of our patient on the horizontal operating room table during spinal anesthesia may have increased the reflux of gastric juice via the incompetent gastroesophageal junction. This patient had had similar chest pain during TURP. The lithotomy position of the patient during TURP may have further increased reflux by increasing the intraabdominal pressure and, thus, intragastric pressure.

This case reemphasizes the importance of obtaining a detailed history regarding chest pain. When a patient is suspected or known to have peptic esophagitis, simple antacid premedication may prevent the development of angina-like pain during spinal anesthesia, and thus, may save the time and effort needed to differentiate it from pain of cardiac origin.

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Bacteriologic Comparison Between Epidural and Caudal Techniques

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Although there have been several studies of the bacteriologic aspects of epidural technique,¹⁻⁴ there is no study of caudal block. Moreover, many anesthesiologists refrain from using caudal analgesia because of fear of infection.^{5,6} For these reasons we conducted the following study.

METHODS

The Human Research Committee of our hospital approved the protocol of the study, and the patient's consent was obtained. The study consisted of two phases, including a total of 30 patients.

Phase I. Fifteen women of physical status I, ranging in age from 20 to 26 years, were in active labor, necessitating analgesia. In order to minimize the variables, epidural and caudal techniques were simultaneously used for all parturients (the double-catheter technique).⁷ No rectal enema was given during labor. The back of the patient was cleansed by use of povidone-iodine (Betadine®) spray. After one minute of contact of the degerming solution with the patient's skin, the fluid was removed with a sterile swab. For continuous epidural technique, autoclaved sterile epidural trays with disposable 91.5-cm 20-gauge Teflon® epidural catheters§ were used. For continuous caudal technique, the same type of catheter was introduced after the insertion of a 16-gauge Teflon intravascular cannula

into the caudal canal.⁸ The skin at the entrance of the catheters was covered by sterile gauze dressings and carefully sealed by adhesive tape to prevent contamination of the area with blood, amniotic fluid, urine, or fecal matter. No bacterial filters were attached to the catheters. However, filter-needles¶ were used to aspirate the local anesthetics from the corresponding ampules to prevent introducing glass particles into the epidural space. A single sterile disposable syringe was used at each injection into either the epidural or caudal catheter. During the first stage of labor, 0.5 per cent bupivacaine was injected in therapeutic doses through the epidural catheter. To keep the number of injections equal, each time an epidural top-dose was administered, 1 ml of the drug was also injected caudally. During the second stage of labor, 2 per cent chloroprocaine was injected through the epidural and caudal catheters to achieve both abdominal and perineal analgesia.

In the recovery room, initial bacterial cultures were taken from the skin surface around the catheter's entrance. The skin was then decontaminated using 70 per cent alcohol and allowed to dry. The catheters were pulled out under sterile conditions and cultures were taken from the fluid inside the terminal part of the catheter, from the terminal 2 cm of the catheter representing the segment that had been in the epidural space, and from the 2 cm of the catheter extending 0.5 cm from the skin entry, representing the segment that had been in the tissues. From each patient 16 cultures, both aerobic and anaerobic, were taken and incubated at 35-37 C. For aerobes, the specimens were incubated on blood agar plates and identified 48 hours later by Gram-stain morphol-

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§ Desert Pharmaceutical Co., Sandy, Utah 24070.

¶ Monoject 305, Filter Needle (size 20, filter rating 5 µm) by Monoject Co., Division of Sherwood Medical, St. Louis, Missouri 63103.