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Pain of Delayed Traumatic Splenic Rupture Masked by Intrapleural Lidocaine

WILLIAM W. POND, M.D.,* GREGORY M. SOMERVILLE, M.D.,* SIONG H. THONG, M.D.,*
JAMES A. RANOCHAK, M.D.,† GREGORY A. WEAVER‡

Intrapleural instillation of bupivicaine has been used to control pain after surgery on the gallbladder,[1–6] breast,[1,2] kidney,[1,2] and lung,[7,8] as well as to control pain from multiple rib fractures. We report a previously unreported potential complication along with a possible explanation of visceral pain relief. We further describe the use of intrapleural lidocaine as opposed to the use of bupivicaine as used in other studies.

CASE REPORT

A 56-yr-old, 188-cm, 96-kg man was involved in a motor vehicle accident, sustaining fractures of his left radius, ulna, and ribs two and four through nine. A chest radiograph performed on admission showed left pleural fluid, a questionably widened mediastinum, and multiple left rib fractures. Computed tomography of the chest and abdomen failed to substantiate injury to the mediastinal structures or abdominal organs, including spleen.

A chest tube thoracostomy drained 100 ml sanguinous fluid and was connected to negative closed drainage. The dyspnea present since the accident failed to improve after pleural fluid drainage.

Because of chest trauma and a history of chronic bronchitis, the patient was closely observed for possible deteriorating pulmonary function (table 1). Because of a 74-pack-year smoking history, he had a chronic productive cough with chronic bronchitis. Three years previously, ventricular ectopy and questionable myocardial ischemia had warranted cardiac catheterization, which was normal.

Because of chest pain, the patient's ability to cough, breathe deeply, and clear secretions was severely limited. Clinical pulmonary deterioration was treated with an intravenous aminophylline infusion, aerosolized metaproterenol treatments, attempted deep breathing and cough, and narcotic analgesics.

In spite of vigorous management over the following 5 h, the patient showed marked inability to clear secretions or breathe deeply. Consideration was given to tracheal intubation, mechanical ventilation, and systemic analgesics. A decision was made to provide regional analgesia *via* an intrapleural anesthetic.

A left intrapleural epidural catheter was inserted over the T-5 rib in the anterior axillary line. An initial test dose of 3 ml of 0.5% bupivicaine was given, followed by a bolus dose of 17 ml. Over the following 30 min, the patient showed marked decrease in chest pain, although pain from the left radial and ulnar fractures now became apparent. An infusion of lidocaine (16 mg/ml) through the catheter was begun at a rate of 2.1 mg/min. Over the following 2 h, the patient was able to breath more easily and to clear thick mucoid secretions. One dose of narcotic analgesics per 8 h was used for arm pain until the fractures were reduced. Thereafter, no narcotic analgesics were necessary, and the patient's mental status was continuously clear. One unit of packed red blood cells was given in view of a hematocrit of less than 30%.

The intrapleural lidocaine infusion was continued uneventfully for the next 8 days with the infusion rate adjusted to maintain a serum lidocaine level less than 5 μg/ml. The degree of analgesia was judged to be good, although supplemental doses of 10 ml bupivicaine 0.5% were necessary upon two occasions for coughing and respiratory treatments. Hematocrit remained stable at 34–35%.

Gradual improvement of the respiratory status continued. On the sixth day, the patient was transferred from the intensive care unit, and cautious ambulation was begun. The intrapleural lidocaine infusion was tapered and then discontinued in the afternoon of the eighth day after initiation of therapy. The catheter was removed and an occlusive dressing was applied.

During the evening of the same day, the patient became restless and diaphoretic, and complained of chest and upper abdominal pain. Electrocardiogram and chest radiographs were unchanged. Arterial blood pressure was 138/90 mmHg, and the hematocrit 31%.

The possibility of angina was entertained and 0.4 mg sublingual nitroglycerine was given; the patient experienced no pain relief, but the arterial blood pressure declined precipitously to 80/60 mmHg. Apparent hypovolemia was treated successfully with intravenous crystalloids and two units of packed red blood cells. Physical examination revealed diffuse upper abdominal tenderness. Abdominal radiographs were negative for ileus or free intraperitoneal air. Computed tomography of the abdomen now showed splenic rupture with a subcapsular hematoma. A splenectomy was performed without incident. The patient was discharged from the hospital 10 days later.

DISCUSSION

Multiple rib fractures cause severe patient discomfort with respiration; consequently, there is impairment of ability to breathe deeply, cough, and clear secretions. In this instance, chronic bronchitis, chronic obstructive pulmonary disease, pulmonary contusion, and multiple rib fractures caused deteriorating pulmonary function in spite of aggressive bronchodilator therapy, chest physiotherapy, and narcotic analgesics. Intrapleural administration of local anesthetic can relieve pain from multiple-rib fractures[9] and improve ventilatory capacity that is decreased due to pain.[3] Intrapleural anesthesia decreased the patient's pain and improved pulmonary function, thus preventing the need for tracheal intubation and mechanical ventilation.

* Staff Anesthesiologist.
† Staff Surgeon.
‡ Research Technician.
Received from the Departments of Anesthesia and Surgery, The Lutheran Hospital of Fort Wayne, Inc., Fort Wayne, Indiana.
Address reprint requests to Dr. Pond: Department of Anesthesia, The Lutheran Hospital of Fort Wayne, Inc., 3024 Fairfield Avenue, Fort Wayne, Indiana 46807.

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Blunt trauma may cause splenic rupture that is acute (85–90% of cases), delayed (10–15%), or occult (less than 1%).[10] Delayed splenic rupture occurs when there is a delay between trauma and intraperitoneal bleeding. In three-quarters of these patients, the quiescent period is less than 2 weeks, ending by rupture of a slowly enlarging capsular hematoma or by release of a temporary tamponade of a minor laceration. Hemodynamic signs relate to rapidity and severity of intra-abdominal hemorrhage. Generalized upper abdominal pain, which is usually present, may localize to the left upper quadrant or be referred to the left shoulder. This patient manifested signs of mild hypovolemia and slow splenic rupture. Pain due to the expanding subcapsular hematoma did not become evident until after the discontinuation of the intrapleural anesthesia.

Chest and abdominal wall pain relief is postulated to occur by diffusion of the local anesthetic from the pleural space through pleura to the intercostal nerves.[1,10] Diffusion occurs easily medial to the angle of the ribs posteriorly, where there is no interposed intercostalis intimus muscle. This provides a satisfactory explanation for relief of chest wall and abdominal wall incisional pain.

This does not, however, explain visceral pain relief; therefore, we postulate the following explanation. General visceral autonomic afferent fibers are sensitive to tension and contraction. Their peripheral processes (dendrites) are distributed with pre- and postganglionic sympathetic efferent fibers, but the afferent fibers are not interrupted in autonomic ganglia. The path of sympathetic supply to the spleen is *via* spinal nerves T 6-10, white rami communicantes, thoracic sympathetic ganglia, greater splanchnic nerve, and celiac and splenic plexuses. The sympathetic chain lies posterolateral to the vertebral bodies and anterior to the necks of the ribs, immediately medial and adjacent to the pleura.[12] Intrapleural anesthetics diffuse through the pleura to interrupt visceral afferent autonomic fibers at this level. This provides a plausible explanation for intrapleural anesthesia providing not only somatic but visceral analgesia for gallbladder surgery and renal surgery.

Intrapleural bupivicaine was administered to obtain initial analgesia; analgesia was maintained by continuous infusion of lidocaine, 16 mg/ml (1.6%) at a rate of 8 ml/h (2.2 mg/min.) Lidocaine was felt to be very safe, because systemic absorption from the intrapleural space could never be greater than the rate of infusion, which is safe even when infused intravascularly; *e.g.*, for cardiac dysrhythmias. Lidocaine was used instead of bupivicaine because a laboratory assay is readily available, toxic levels are well known, and nursing personnel are comfortable with lidocaine infusions.

A continuous infusion of intrapleural lidocaine produced excellent pain relief for a patient with multiple left rib fractures. The pain of delayed splenic rupture was masked until discontinuation of the intrapleural lidocaine. Visceral splenic pain is probably blocked by transpleural diffusion of anesthetics to thoracic sympathetic fibers.

TABLE 1. Blood Gas Table

Day	pH_a	Pa_{CO_2} (mmHg)	Pa_{O_2} (mmHg)	Supplemental O_2
Admit (0)	7.30	50	50	Nasal 4 l/min
+1	7.32	51	66	35% mask
+1	7.30	52	57	35% mask
+1	7.35	45	47	4 l/min*
+1	7.38	46	53	Nasal 4 l/min†
+2	7.42	42	51	Nasal 4 l/min
+3	7.44	44	54	Nasal 4 l/min
+5	7.45	43	62	Nasal 4 l/min
+9	7.50	33	62	Nasal 3 l/min‡

* One hour after intrapleural catheter insertion.
† Four hours after intrapleural catheter insertion.
‡ Obtained during episode of abdominal pain.

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