FIGURE 3. Metrizamide cystogram shows smooth internal walls of cyst and no connection with subarachnoid space of the spinal canal.

gut cysts will continue to be appropriately diagnosed and treated with surgical excision: a nonsurgical approach is certainly not as easily recommended when the mediastinal cyst is of soft tissue density on CT scan and is filled with thick, viscid, turbid fluid.3

The techniques and results of percutaneous thoracic biopsy have been well described in the radiologic literature.4,5 To our knowledge, only two mediastinal cysts (one thymic, one pericardial) have been reported in which percutaneous aspiration resulted in disappearance without recurrence.6 Ours is the third such case, but the cyst was of foregut origin. Another option is the transbronchial or transtracheal fine-needle aspiration through the bronchoscope.7 We agree with the recommendation of Pugatch et al8 that thoracotomy can be safely avoided in asymptomatic patients with benign mediastinal cysts. Follow-up chest radiographs should be sufficient if the patient remains asymptomatic.

Performance of cystography for a congenital mediastinal cyst has not been described previously. This technique, similar to renal cystography, is useful in excluding tumor that has undergone cystic degeneration or tumor in the cyst wall. We recommend that metrizamide be used for posterior mediastinal cysts to prevent neurologic sequelae should there be communication with the subarachnoid space, as in a meningocele.

In summary, this case demonstrates the use of percutaneous aspiration for both the diagnosis and the treatment of asymptomatic benign mediastinal cysts. Cystography may be useful for examining the cyst wall and for assessing communication with other mediastinal structures.

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Nalbuphine-induced Pulmonary Edema

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We report the occurrence of acute pulmonary edema in a patient receiving therapy with nalbuphine, a synthetic narcotic analgesic. Other potential causes of acute pulmonary edema were excluded. It is likely that all opiate drugs share the propensity to produce acute pulmonary edema, and nalbuphine resembles other opiates in this regard.

Nalbuphine is a synthetic narcotic analgesic that is structurally similar to oxymorphone and naloxone. Although acute pulmonary edema is a recognized complication of opiate overdose, it has not been reported with the use of nalbuphine.

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Case Report

A 37-year-old asymptomatic healthy woman underwent elective breast biopsy. After an overnight fast, the patient was given a general anesthetic consisting of intravenous thiopental (Pentothal) 300 mg, intravenous succinylcholine 100 mg, and inhalation of nitrous oxide with oxygen. Anesthesia was maintained with enflurane (Ethrane), which was discontinued once intravenous nalbuphine (40 mg) was given at 15 min following induction. Total intravenous fluids given over the previous four hours was 400 ml. During recovery from anesthesia, tachypnea and respiratory distress developed coincident with production of blood-tinted sputum. Fiberoptic bronchoscopic examination revealed only bloody fluid. A chest roentgenogram revealed bilateral parenchymal infiltrates, while arterial blood gas analysis confirmed an increase in the alveolar arterial oxygen gradient. Physical examination 19 days later was unremarkable. Arterial blood gas analysis was normal, as was complete pulmonary function test results. Findings on chest roentgenogram were completely normal.

Discussion

The occurrence of pulmonary edema following elective general anesthesia is unusual in a healthy young patient. Although pulmonary edema can occur following aspiration of gastric contents, this is unlikely since the patient was known to have fasted before this procedure and bronchoscopic examination immediately after onset of symptoms failed to reveal endobronchial gastric contents. The patient received no blood products and the volume of fluid infused was small. We are left to consider the drugs administered, and nalbuphine seems to be the most likely causative agent. This patient received a moderately large dose of intravenous nalbuphine and showed respiratory depression and carbon dioxide retention, which is common in cases of opiate-induced pulmonary edema.1,2

Pulmonary edema complicating opiate overdosage was first described by Osler6 in 1880. Although pulmonary edema was once thought to result from adulterating substances, this entity has been reported before adulteration of narcotics was widespread and cases have occurred following ingestion of legally obtained methadone tablets, albeit in quantities large enough to cause coma.45 It is possible that all opiate drugs share the propensity to produce acute pulmonary edema in cases of overdosage, and this syndrome has been well-documented with morphine, heroin and methadone. We report a case of acute pulmonary edema in which the drug nalbuphine is implicated and suggest that this drug resembles other opiates in causing pulmonary edema during intoxication.

References


Exercise-induced ST-segment Depression and elevation in the same patient

A Case for Mixed Angina

John M. Yackee, M.D.; Reed M. Shneider, M.D.; and Alan G. Wasserman, M.D.

A patient with significant left anterior descending coronary disease is presented who developed significant ST segment elevation and depression at different times under similar testing conditions. Currently proposed explanations for exercise-induced ST segment elevation are discussed. This patient likely represents a case of spasm superimposed on significant obstructive disease, so-called mixed angina.

ST-segment depression during exercise electrocardiography is a well-established criterion for myocardial ischemia. ST-segment elevation during exercise testing is less commonly observed. The latter is generally ascribed to abnormal wall motion in the setting of previous myocardial infarction,1 to transmural ischemia in the setting of coronary spasm,2-4 or severe proximal coronary artery stenosis.5-6 In this report we present a patient who developed marked ST-segment elevation and depression under similar testing conditions at different times.

Case Report

A 65-year-old man was referred for evaluation of substernal chest tightness occurring predictably with swimming and alleviated by rest. There was no history of previous myocardial infarction or episode of chest pain occurring at rest or awakening him from sleep. His coronary artery disease risk factors included hypertension and a positive family history. Physical examination was unremarkable. The resting electrocardiogram revealed increased voltage and was otherwise normal limits. Treadmill exercise electrocardiography was performed with the patient on antianginal therapy. He exercised using a modified Bruce protocol to 6.5 mets, with a peak heart rate 143, and developed chest pain and diffuse (0.3-0.4 mV) ST-segment depression (Fig 1). At peak exercise, the systolic blood pressure fell from 200 mm Hg to 160 mm Hg. The patient was started on therapy with propranolol (20 mg qid) and isosorbide dinitrate (20 mg qid). Subsequent cardiac catheterization revealed a long, irregular 95 percent stenosis of the proximal left anterior descending artery (LAD) involving the origins of the first two diagonal branches and the third septal branch. Result of left ventriculography was normal. Exercise testing was repeated 17 days after the previous test to assess the adequacy of medical therapy. At that time, he exercised to 6 mets with chest pain and 0.4 mV ST-segment elevation at peak exercise in precordial leads V5, V6 (Fig 2). The peak exercise heart rate was 88 and the systolic blood pressure fell from 130 to 110 mm Hg at peak exercise. Due to the markedly positive exercise tests with associated fall in blood pressure, coronary artery bypass surgery was performed with an internal mammary artery graft to the left anterior descending artery and a saphenous vein graft to the first diagonal. The postoperative course was uncomplicated. Exercise electrocardiography was repeated two months after surgery while on therapy with atenolol. He exercised to 7.5 mets with a peak heart rate of 98, without ST changes or a fall in blood pressure, although the patient did complain of mild chest discomfort.

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ST-segment Depression and elevation in same patient (Yackee, Shneider, Wasserman)