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Suspected Venous Air Embolism during Epidural Anesthesia

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Venous air embolism (VAE) has been reported in many clinical situations,[1–3] including during epidural catheter insertion for obstetric anesthesia.[4] We report a case of suspected significant VAE during administration of steroids into the cervical epidural space.

CASE REPORT

A 40-yr-old man status post–cervical laminectomy in December 1988 and anterior cervical discectomy in April 1989 presented to the Pain Clinic in October 1989 with complaints of neck and arm pain. The patient was scheduled for administration of steroids into the cervical epidural space. Prior to the procedure, the patient's blood pressure was 110/80 mmHg and heart rate was 100 beats per min. With the patient in the sitting position, a 16-G Tuohy needle was advanced at the C7–T1 intervertebral space using the "hanging drop" technique. With loss of resistance, the drop of saline was drawn into the needle. The operator then drew up the local anesthetic and mixed it with Depo-Medrol® (Upjohn).

While the medication was being prepared, the patient became very agitated. He was dyspneic, ashen-colored, and verbalized a feeling of impending doom. No medication had been injected epidurally. The patient complained of shortness of breath and was coughing; with each forcible cough, it was noted that blood projected from the hub of the epidural needle with considerable velocity. The needle was removed; the patient was placed in the supine position; and supplemental oxygen was given *via* occlusive mask. The patient's blood pressure decreased to a low of 82/50 mmHg, and a tachycardia of 120 beats per min was noted. The blood pressure was responsive to crystalloid infusion and 15 mg iv ephedrine. After breathing oxygen for approximately 2 min, the patient was still dyspneic and ashen-colored; a pulse oximeter revealed an initial reading of hemoglobin oxygen saturation (Sp_{O_2}) of 85%. Auscultation of the chest revealed bilateral wheezes without appreciable cardiac murmur. The patient's mental status, Sp_{O_2}, and wheezing slowly improved over the next 40 min. He recovered and was discharged home 1 h and 20 min later.

Approximately 1 month later the patient returned for a repeat procedure. Again, the sitting position was chosen, since we feel this position facilitates midline placement of the needle and enhances the hanging drop sign. At the C7–T1 space, a 16-G Tuohy needle was advanced to the same depth as in the previous procedure; the drop of saline again was noted to be drawn in. The stylet of the needle was immediately inserted and 6 ml 0.375% bupivacaine and 80 mg Depo-Medrol® were drawn into the syringe. We chose not to prepare a mixture of local anesthetic and steroids prior to the procedure, since unintentional dural puncture would necessitate mixing the steroid with 0.9% normal saline instead of local anesthetic. Prior to insertion of the local anesthetic, the needle was aspirated, and the free flow of blood was noted. The epidural needle was removed, and the T1–T2 space was used to reinsert the epidural needle with similar confirmation of epidural space at the previous depth. The medication was subsequently injected after negative aspiration, and a definitive block was noted. The patient tolerated the procedure well and reported good pain relief.

DISCUSSION

The possibility of VAE exists whenever external pressure to an open vein is greater than central venous pressure (CVP).[5] Frequently noted symptoms of VAE in awake patients include dyspnea, faintness, chest pain, and fear of death. Clinical findings associated with VAE are deep inspirations, tachypnea, tachycardia, coughing, gasping, wheezing, cyanosis, and circulatory collapse.[6] Treatment of suspected VAE includes occlusion of the source of air entry, minimization of the amount of air in the circulation, and cardiopulmonary support. To stop entrainment of air, the suspected source in the surgical field should be flooded with saline or nonsurgical sources sought and eliminated. Administration of 100% oxygen both treats hypoxemia and allows reduction in the partial pressure of nitrogen. Removal of intracardiac air can be achieved by aspiration from a previously inserted CVP catheter. Circulatory collapse is treated by placement of the patient in the left lateral decubitus position (Durant's position)[7–8] and if required, administration of vasopressors.

Diagnosis of VAE is made most frequently by precordial Doppler, transesophageal echocardiography, pulmonary artery catheter, or measurement of end-tidal carbon dioxide or nitrogen,[9–12] none of which is commonly used during insertion of epidural catheters. Significant VAE during epidural catheter insertion has not been previously reported, although clinically insignificant VAE has been detected by precordial Doppler during identification of the epidural space and insertion of epidural catheters in obstetric patients.[13] Our patient exhibited several of the signs and symptoms of VAE. Prior surgery to the neck with subsequent surgical scarring could have resulted in a venous malformation with noncollapsible side walls. A 16-G needle passed into this venous space would have resulted in a positive hanging drop sign and made an easy conduit for air entrainment. The increased intrathoracic pressure generated prior to cough would be easily trans-

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mitted to the epidural venous plexus, and thus explain the forceful expulsion of blood from the needle hub seen with coughing. From this experience, we recommend that the stylet of the epidural needle remain in place until just prior to aspiration and injection. This case report should also serve to reinforce the awareness of possible VAE during any invasive procedure in which venous integrity may be disrupted.

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Central Anticholinergic Syndrome Following Glycopyrrolate

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The central anticholinergic syndrome (CAS) is caused by pharmacologic substances that have anticholinergic actions. The central and peripheral manifestations are those of acetylcholine competitive inhibition and appear to involve muscarinic receptors.[1] Of the three muscarinic antagonists commonly used during anesthesia, atropine sulfate and scopolamine hydrobromide are known to cause this syndrome, whereas glycopyrrolate has not been previously reported to do so. We report a case in which glycopyrrolate appears to have been responsible for a particularly severe episode of CAS.

Case Report

A 22-yr-old healthy woman presented in the ambulatory surgery unit for elective diagnostic laparoscopy for pelvic pain. Her past medical and surgical history were unremarkable except for a prior appendectomy under general anesthesia without complications. She denied taking any medications or have any allergies. She denied the acute or chronic use of recreational drugs, alcohol, or tobacco. Family history was negative for chronic illness or drug idiosyncrasy. Her pharmacologic history was corroborated by an accompanying family member. Upon admission she was afebrile, had a blood pressure of 132/80 mmHg, heart rate of 76 beats per min, and a ventilatory rate of 10 breaths per min. Her physical exam was entirely normal. Preadmission laboratory urinalysis, serum electrolytes, blood urea nitrogen, serum creatinine, and complete blood count were normal.

She received 50 mg ranitidine hydrochloride intramuscularly and 30 ml 0.35 M sodium citrate by mouth. An intravenous infusion was begun and 0.2 mg glycopyrrolate administered intravenously. Blood pressure and heart rate remained unchanged. Ten minutes later, she was taken to the operating room where she appeared agitated and began to complain of a severe bilateral temporal headache. Her blood pressure was 170/116 mmHg, heart rate 140 beats per min, and ventilatory rate 20 breaths per min. One minute later these parameters were 156/126 mmHg, 145 beats per min, and 22 breaths per min, respectively. Five milligrams labetalol hydrochloride was administered intravenously, but to no effect. The patient began to writhe on the operating table and stated that her head felt like it was about to explode. Upon her complaint that the operating room was too bright, it was noted that her pupils were widely dilated. Her skin was hot and dry and her oral mucosa was dry. She denied ever having had a similar experience.

A presumptive diagnosis of CAS was made, and 1 mg physostigmine was administered intravenously. Three minutes later the patient was much less agitated and related a marked decrease in the severity of her headache. Blood pressure was now 151/109 mmHg, heart rate 138 beats per min, and ventilatory rate 16 breaths per min. A second

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