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Postoperative paraplegia associated with epidural narcotic administration

Epidural injections of buprenorphine were given for postoperative pain relief to a patient with pulmonary carcinoma who underwent a right upper lobectomy. Paraplegia occurred postoperatively and the patient's neurological status deteriorated after each injection of epidural narcotic. Laminectomies on the third postoperative day revealed an expanded oxidized cellulose (Oxycel) pledget in the epidural space. Neurologic dysfunction after epidural narcotic administration was caused by the oxidized cellulose which had migrated into the epidural space following use for surgical haemostasis and subsequently expanded with the narcotic solution and blood.

Epidural administration of narcotics for the relief of postoperative pain is increasing in popularity. The safety and efficacy of the technique have been described.¹ When a patient receiving epidural narcotics develops neurologic complications in the postoperative period, the anaesthetist is confronted with a complex problem of differential diagnosis. The correct diagnosis must be made in the early stages of the neurologic dysfunction for treatment to be effective.

The following is a report of paraplegia which occurred postoperatively and worsened after each injection of epidural narcotic, but which was unrelated to the narcotic used.

Case report

A 69-year-old, 56 kg man with pulmonary carcinoma was scheduled for a right upper lobectomy. His history

Key words

ANALGESIA: epidural; COMPLICATIONS: paraplegia, oxidized cellulose; ANALGESICS: buprenorphine.

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included hypertension, for which he was being treated with oral nifedipine. The patient denied a history of low back pain or other neurologic dysfunctions. Physical examination was unremarkable. Preoperative laboratory tests were normal.

Utilizing a loss of resistance technique, a 17-gauge Tuohy epidural needle was placed in the L₁₋₂ interspace on the operative day. A 19-gauge epidural catheter was then introduced through the needle without difficulty. Neither blood nor cerebrospinal fluid could be aspirated. The catheter tip was surmised to be positioned 7 cm above the puncture point. No paresthesia was elicited during the placement of the needle or catheter. A test dose of 3 ml of one per cent lidocaine with epinephrine 1/200,000 was injected. There was no evidence of subarachnoid or intravascular injection of the test dose. Fifteen minutes after the test dose, general anaesthesia was induced with thiamylal and succinylcholine, and endotracheal intubation was performed. Controlled ventilation was maintained with one per cent enflurane, 60 per cent nitrous oxide and oxygen. An epidural injection of 10 ml of 0.25 per cent bupivacaine without epinephrine was given to control intraoperative hypertension. The surgical procedure was uneventful except that a fracture of the fifth rib near the costovertebral joint occurred. Haemostasis in the fractured portion was achieved with the packing of oxidized cellulose (Oxycel) pledgets. Operative time was 2 hr 40 min and blood loss 350 ml.

The first epidural injection of 0.1 mg of buprenorphine diluted with saline to 10 ml was administered during the skin closure. The patient was extubated in the operating room and his cardiovascular, respiratory and neuromuscular status were satisfactory.

The second epidural injection of the same dose was given two hours after the operation and the third epidural injection eight hours later. Although repeated injections of buprenorphine were required, pain relief was satisfactory and the patient walked a short distance the next morning. In the afternoon of the first postoperative day he felt slight numbness in both lower extremities but he still could walk. At midnight on the same day he complained of postoperative pain and the fourth epidural

buprenorphine was administered. Pain relief was complete and he was able to sleep.

During the morning of the second postoperative day the patient felt increased numbness in his lower extremities. Unfortunately the patient's physician told him that his symptom could be due to the residual effect of the epidural buprenorphine. That evening urinary incontinence occurred and the anaesthetists were consulted to determine if the patient's condition was related to the epidural narcotics. Physical examination revealed loss of sensation to touch, pain and temperature below T₁₀, complete paralysis of the right lower extremity and incomplete paralysis of the left extremity. Although the role of buprenorphine was thought doubtful, other causes were not obvious. Epidural injection of 3 ml of 12 mg dexamethasone was done, followed by further deterioration of the symptoms. Four hours after epidural dexamethasone administration paraplegia below T₅ was observed. One gram of hydrocortisone was administered intravenously and the epidural catheter was removed. Emergency CT and enhanced CT examinations revealed no abnormality in the spine.

The following morning, on the third postoperative day, the level of sensory loss had reversed to T₇, but a myelogram and a CT myelogram were done which revealed compression at the T₅₋₆ level by a mass in the right epidural space. The mass was initially thought to be a metastatic tumour. Emergency laminectomies of T₅ and T₆ revealed that the mass consisted of expanded oxidized cellulose in the epidural space. The weight of the mass removed was approximately 2 grams.

The day following the laminectomy a typical Brown-Séquard Syndrome was observed, with ipsilateral (right) paralysis and loss of vibration and position sensation, and contralateral loss of pain and temperature. Thereafter, the patient's neurologic dysfunction gradually improved, with only slight numbness of left lower extremity remaining on the 30th postoperative day.

Discussion

Most survey reports indicate that the incidence of severe neurologic deficit following epidural local anaesthetics or narcotics is very low; however, several case reports describing temporary or permanent paralysis following these procedures have appeared. Paraplegia may be due to cord ischaemia or infarct caused by arterial hypotension during surgery, or compromise of blood supply to the cord by the procedure or the condition of the patient, or by production of an epidural or subdural haematoma associated with or without anticoagulant therapy.² Injury to the nerve roots has occurred following inadvertent epidural administration of toxic chemicals or chemical contaminants of anaesthetic or narcotic solutions.

Epidural buprenorphine is effective for postoperative pain relief.^{3,4} It is known to be highly lipophilic and to have an affinity for the opiate receptor approximately twice that of morphine.^{5,6} It is supplied commercially in a preservative-free solution and would seem as safe a choice for epidural analgesia as morphine. Although neurologic changes after epidural morphine have been reported to be transient and insignificant in patients with neurologic disease,⁷ there has been no report of permanent neurologic deficits in normal subjects. In most cases of severe neurologic deficit following epidural anaesthesia, however, aetiological factors were unknown.⁸ Therefore, initially we could not rule out epidural buprenorphine as a cause of neurologic dysfunction.

In this case postoperative paraplegia was caused by a mass of oxidized cellulose which strayed into the epidural space through an intervertebral foramen, from the area of fracture of the fifth costovertebral joint and which expanded with blood and the epidurally injected narcotic solution. Oxidized cellulose has been used in various forms for haemostasis in surgery, with few complications reported.^{9,10} In a case report of blindness following the use of oxidized cellulose in the area of the optic chiasm, a swollen mass of oxidized cellulose exerting pressure on the optic nerve was proposed as a cause for the loss of vision. Removal of the cellulose did not result in the return of vision.⁹ Although there has been no report of spinal cord compression by oxidized cellulose or similar foreign material, the product information for Oxycel warns that tight wadding or packing should be avoided, especially within the bony enclosure of the central nervous system where the extra pressure induced by swelling of the mass of Oxycel may interfere with normal function. Anaesthetists should be familiar with the aetiology and diagnosis of neurologic dysfunction after anaesthetic procedures. Since magnetic resonance imaging is non-invasive, it is the best diagnostic procedure for investigation of paraplegia. Other procedures, such as myelography and epidural injection of contrast medium will worsen symptoms and may be contraindicated in the case of drug or chemical-induced paraplegia. Therefore, precise neurological diagnosis, with the knowledge of the anaesthetic procedure used is required in the early stages for treatment to be effective.

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Résumé

Les injections épidurales de buprénorphine étaient faites en période post-opératoire pour le soulagement de la douleur chez un patient atteint de carcinome pulmonaire ayant subi une lobectomie. Une paraplégie est survenue en période post-opératoire et l'état neurologique du patient s'est détérioré après chaque injection épidurale de narcotique. Au troisième jour post-opératoire des laminectomies ont révélé la présence de tampons de cellulose oxydée et expansée (Oxycel) dans l'espace épidural. La dysfonction neurologique après chaque administration de narcotique dans l'espace épidural était provoquée par l'expansion de la cellulose oxydée qui a migré dans l'espace épidural lors de l'hémostase chirurgicale.