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Thoracic epidural anesthesia and epidural hematoma

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This report involves a 74-year-old-male who developed a thoracic epidural hematoma with paraparesis on the second postoperative day in conjunction with thoracic epidural anesthesia established before surgery for acute abdominal aortic dissection. The finding indicates that laminectomy can be performed successfully as late as three days after diagnosis of the hematoma, with a complete restitution of neurological function. High-dose steroid treatment may have been a contributing factor for the positive outcome.

It has been shown that postoperative epidural analgesia provides better dynamic pain control and reduces overall mortality compared with traditional postoperative treatment modalities (1–3). However, serious complications such as spinal hematoma with neurological deficits following epidural and spinal anesthesia may lead to a devastating situation for the individual patient.

Spinal hematomas may occur spontaneously or in association with neuraxial anesthesia. Several review articles have addressed the neurological complications after central nerve blocks (4–6). Neurological dysfunction resulting from hemorrhagic complications after a regional blockade is rare. The incidence is believed to be less than 1:150,000 for epidural, and less than 1:220,000 for spinal anesthetics (7). Risk factors include anticoagulation therapy especially with low-molecular weight heparin, and coagulation disorders. In a review by Horlockeret et al. the neurological outcome following spinal hematomas showed that only 38% of the patients had a partial or full recovery (8). The most favorable outcome seemed to occur in patients who underwent laminectomy for epidural hematoma within 8 h of diagnosis (9, 10).

Case report

A 74-year-old male diagnosed with hypertension, non-insulin-dependent diabetes mellitus, and cancer of the prostate was admitted to our hospital for sudden development of severe pain in both legs. The patient was chronically treated with nifedipine 10 mg twice daily, glibenclamide 1.75 mg twice daily, and regular injections of a GnRH-analog for the prostatic neoplasm. For almost a decade the patient suffered from cold and painful legs, but had no history of back pain. Lower extremity perfusion pressures were normal 6 months before admittance. A recent CT-scan of the spine did not confirm any dissemination of the prostate cancer.

On the day of admission, the patient was bicycling when he was suddenly struck by severe pain in both legs. A general practitioner who first examined the patient noted pale and cold legs with a palpable pulse only in the left femoral artery. Arm blood pressure was 210/100 mmHg and heart rate 90 b.p.m. Following hospital admission a thoraco-abdominal CT scan revealed an arteriosclerotic aorta with a 30-mm infra-renal abdominal aortic aneurysm and signs of dissection with a loose intima tear. No signs of an epidural hematoma were present. The patient was subsequently referred for emergency surgery. His cardiovascular condition was stable and he experienced no abdominal pain.

In the operating room his blood pressure was 190/80 mmHg and heart rate 100 b.p.m. Laboratory investigations showed a hemoglobin level of 141 g/l, platelet count of $172 \times 10^9 /l$, activated partial thrombin time of 28 s, and a prothrombin complex of 127%. As a result of stable hemodynamics and little pain the patient received an epidural catheter at the T10–11 level before induction of general anesthesia. The catheter was applied with the patient in the sitting position with an 18-G Tuohy needle and the loss of re-
sistance technique with a 10-ml plastic syringe filled with normal saline. Bone contact was first made, but after redirection of the needle the application was uneventful. The patient reported no paresthesia. A closed-end catheter (0.9 mm) with three lateral orifices was inserted 5 cm into the epidural space. There was no blood or CSF leak through the needle or the catheter.

General anesthesia induction was performed with pentobarbital, fentanyl and rocuronium. Anesthesia was maintained with desflurane and oxygen/nitrous oxide (40/60). Heparin 5000 IU was administered intravenously before aortic cross-clamping, approximately 2 h after application of the epidural catheter. The patient received an infrarenal aorto-bifemoral graft. Thrombendarterectomy was performed bilaterally because of signs of embolization to the femoral arteries. The total intraoperative blood loss was 1500 ml. Two units of packed red cells, 3000 ml of Ringer’s lactate and 1000 ml of hydroxyethyl-starch were given intraoperatively. Surgery lasted approximately 5 h. A 4-mg dose of epidural morphine was administered immediately after anesthesia induction. Thirty minutes later a bolus of mepivacaine 6 ml (20 mg/ml) was followed by an infusion at 6 ml/h. Thereafter no more i.v. fentanyl was administered. The patient was extubated in the operating room and transferred to the intensive care unit (ICU) in a hemodynamically stable and normothermic condition. In the ICU, an epidural infusion was started with a mixture of bupivacaine 2.5 mg/ml and morphine 0.05 mg/ml at 4 ml per hour. Abdominal pain relief was adequate, but the patient complained of intermittent pain in the legs, which was treated with epidural bupivacaine injections. Low molecular weight heparin (enoxaparine 40 mg s.c.) was given 6 h postoperatively. From this point on, no more low molecular weight heparin was administered.

On the first postoperative day the patient was stable and transferred to the ward. The previously experienced leg pain returned, and as leg weakness and anal sphincter dysfunction followed, the epidural infusion was stopped on the second postoperative day. When 6 h had passed without any improvement in motor function, an MRI was performed. The investigation revealed a 30-mm cranio-caudal epidural hematoma at the Th12 level with moderate compression of the spinal cord (Fig. 1). The epidural catheter was then withdrawn and the patient was transferred to the neurosurgical ICU.

As the patient could not lift his legs from the bed, he received betamethasone 16 mg i.v., followed by 8 mg daily on the following two days. The patient’s clinical status improved with less leg weakness and pain. However, on the fifth postoperative day the neurological symptoms again increased. Laminectomy was performed at the Th10–L1 level with evacuation of the hematoma. Postoperatively, the leg pain disappeared and motor function improved.

The patient spent 40 days in the hospital. Eight months later he lives at home, walks several kilometres, and again rides his bicycle.

Discussion

Factors that increase the risk of epidural hematoma formation include both non-pharmacologic and pharmacologic causes. Vascular malformations, a deformed vertebral column, and other anatomical abnormalities may enhance the risk for spinal bleeding (9,11). Other risk factors include the anesthetic technique with a temporary epidural catheter for postoperative pain relief, or catheter removal, which may increase the risk of hematoma formation. The insertion site (thoracic vs. lumbar) and the approach (midline vs. paramedian) of the epidural space are sometimes believed to increase the risk of epidural bleeding. However, a thoracic epidural catheter is no less safe than a lumbar epidural approach (12). There is no evidence that the risk for hematoma formation is lower with a midline compared with a paramedian

![Fig. 1. Thoracic epidural hematoma at the T12 level visualized on a MR image.](image-url)
approach (13). However, the paramedian technique may need more attempts and have a lower success rate compared with the midline technique (14). The relative experience of the anesthesiologist performing the blockade may play a role in the number of punctures attempts and eventually the formation of an epidural hematoma.

The introduction of low-molecular weight heparin in the United States increased the reported incidence of neuraxial hematomas compared with what was reported in Europe (15). This observation initiated the present guidelines for thromboprophylaxis in combination with neuraxial blockade in Sweden and Norway (16, 17), as well as in other countries.

It can be argued that there was no motivation to insert an epidural catheter before general anesthesia in this patient. On the other hand, the patient was in a stable condition, and efficient epidural anesthesia may maintain stable perioperative hemodynamics and provide adequate pain relief with lower postoperative morbidity (18). The preoperative pain that this patient experienced as one of his initial symptoms most likely resulted from vascular ischemia in the lower limbs, and the preoperative CT-scan revealed no signs of spinal stenosis or nerve root compression. No major bleeding had occurred that would have affected his coagulation status and no anticoagulants were given preoperatively.

Following development of an epidural hematoma, an 8-h time limit for emergency laminectomy has been set in order to minimize the loss of motor and sensory function in the lower part of the body (10). It is commonly believed that after this time span an increased risk of permanent damage occurs. The patient in this report underwent surgery 3 days after the diagnosis of the epidural hematoma, and nevertheless had a complete neurological recovery despite the time frame, i.e. surgery can also be performed after more than 8 h. Contributing factors to the positive outcome may have been that complete ischemia did not occur as a result of moderate spinal cord compression. Hentschel and colleagues have reported on two cases with spontaneous resolution of spinal epidural hematomas without surgery (19). Another important fact is that the patient was given betamethasone during the observation period in the neurosurgical ward, which may have contributed to the outcome (20).

Conclusion

A patient who developed an epidural hematoma following thoracic epidural anesthesia with gradual onset of paraplegia regained complete neurological function in the lower part of the body (10). It is commonly believed that after this time span an increased risk of permanent damage occurs. The patient in this report underwent surgery 3 days after the diagnosis of the epidural hematoma, and nevertheless had a complete neurological recovery despite the time frame, i.e. surgery can also be performed after more than 8 h. Contributing factors to the positive outcome may have been that complete ischemia did not occur as a result of moderate spinal cord compression. Hentschel and colleagues have reported on two cases with spontaneous resolution of spinal epidural hematomas without surgery (19). Another important fact is that the patient was given betamethasone during the observation period in the neurosurgical ward, which may have contributed to the outcome (20).

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