LUMBOVERTEBRAL SYNDROME AFTER EXTRADURAL BLOOD PATCH

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SUMMARY
We describe a patient who developed an immobilizing lumbovertebral syndrome after an extradural blood patch and who was hospitalized with a suspected extradural abscess. An infectious aetiology of the persistent backache could be excluded and the patient recovered with analgesics and physiotherapy. The probable aetiology is discussed.

KEY WORDS

CASE REPORT
A 40-yr-old patient was scheduled for repair of torn anterior fibulotalus and fibulocalcaneal ligaments. In her past medical history it was notable that twice before she had been admitted to hospital for treatment of lumbovertebral syndromes elicited by weight-lifting. As a result of this previous condition, 5 years previously she was obliged to change her profession from a nurse to a medical clerk. Since then, she had never had a severe backache, although she regularly suffered from migraine. Otherwise, she was in good health. According to the patient's wishes extradural anaesthesia was planned.

After oral preanaesthetic medication with midazolam 15 mg, the patient was alert but calm upon arrival in the operating room. With the patient in the lateral position, the first attempt at placing an 18-gauge Tuohy extradural needle resulted in accidental penetration of the dura at the L3-4 level. Therefore, single-shot spinal anaesthesia was performed with plain 0.5% bupivacaine 17.5 mg via the Tuohy needle. The further intraoperative course was uneventful.

On the morning of the first day after operation, the patient complained of nausea. On the same afternoon, severe frontal and occipital headache occurred which were made worse by sitting and improved on lying down. A low cerebrospinal fluid (CSF) pressure headache was diagnosed. Using conservative treatment with initial bed rest, oral analgesics and adequate hydration, which included increased coffee drinking, the postdural puncture headache (PDPH) improved markedly. The patient, who had a unilateral plaster of Paris dressing, began walking with crutches and was allowed home 8 days after operation, with minimal headache.

With the greater activity and cessation of bed rest, the PDPH worsened. After initially refusing, the patient finally decided to accept an extradural blood patch (EBP) on day 12 after operation. After monitoring the patient with a sphygmomanometer, ECG and pulse oximeter, she was placed in the left lateral decubitus position. Using strict aseptic techniques, the interspace of the previous dural puncture was identified and an 18-gauge Tuohy needle was inserted into the extradural space. Thirty millilitre of blood was aspirated aseptically through a 16-gauge i.v. catheter placed in a large vein at the antecubital fossa. This blood was injected slowly into the extradural space, with pauses after 15 and 20 ml. After 25 ml had been injected, the patient reported the onset of discomfort in the back, whereupon the extradural injection was stopped. The patient was kept recumbent for 90 min and a total of 1200 ml of Ringer lactate solution was infused. Then the patient was able to sit up and walk around with her crutches with only a minor headache. When she went home she complained of a light backache.

Thirteen days later, she presented at the emergency unit with an immobilizing backache and was admitted with suspected extradural abscess. A small degree of backache had persisted since the performance of the EBP and, 3 days before the consultation in the emergency unit, her condition worsened without any recognizable cause. She localized the backache to the lower lumbar region with radiation into the ventral side of both limbs, and into the inferior part of the abdomen. The pain was constant, but became worse with movement of the lumbar spine and eventually led to complete immobilization of the patient. She did not experience pain on coughing and did not have paraesthesiae or restricted sensory and motor function. The patient was afebrile but did have nocturnal sweating. Clinical examination showed pain on pressure and percussion in the lumbar and sacral region, which was maximal at the L5 level. In addition to distinct paravertebral myalgia, especially on the right side,
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there was restricted movement of the lumbar spine, but the remainder of the detailed neurological examination was normal. Haematological examination, including erythrocyte sedimentation rate and differentiated white blood count, was normal. Computed tomography of the lumbar spine did not indicate the presence of an abscess, or pathological alterations of the osseous structures or the discs. Lumbar myelography was normal, as were cytology and biochemical and microbiological examinations of the CSF.

The patient was mobilized with crutches and analgesic therapy and after distinct recovery occurred she was allowed home 6 days later. After 2 weeks, she no longer had back pain and had no related pain in the 12 months since then.

DISCUSSION

Accidental dural puncture occurs in 1.5–3% of attempted extradural anaesthesia [1]. Norris, Leighton and DeSimone demonstrated that identifying the extradural space when the needle bevel is orientated parallel to the longitudinal fibres significantly reduced the risk of PDPH if dural puncture occurred but, because of the large size of the needles used, PDPH is a common complication of dural puncture even with this more suitable technique [2]. When conservative treatment fails, EB is recommended [3]. A high success rate and a low incidence of complications after EB have made this method the treatment of choice for severe PDPH [4–6]. Although residual backache is a relatively frequent side effect of EB [4, 6], to our knowledge an immobilizing lumbovertebral syndrome has not been described after EB. Several factors may have contributed to this complication in our patient.

First, the reactive process in the extradural space after the injection of blood may have irritated her sensitive back. Unfortunately, little is known about the mechanisms leading to organization or resorption of an EB or a haematoma and the metabolic products set free during this process. Samuni and colleagues have proposed a “site specific” Fenton mechanism of free radical cytotoxicity [7]. The clinical importance of this finding has been shown by Angel and co-workers in a study on the aetiological role of free radicals in haematoma-induced flap necrosis where iron or iron-bound polymers (perhaps haemoglobin) were implicated in the generation of free radicals, which may have caused tissue destruction [8]. The role of free radicals after EB is unclear, because the aetiology of the backache seen after EB has not been defined. In Angora goats, DiGiovanni, Galbert and Wahle found a similar tissue reaction after placement of uncotted autologous blood in the extradural space, as was seen after diagnostic lumbar puncture [9]. However, only 2 ml of blood was injected in animals weighing up to 60 kg. Inadvertent puncture of an extradural vessel, as observed in one animal of the small control group, may lead to extravasation of a similar amount of blood. Even an extradural puncture without incident does not exclude the occurrence of a small extradural haemorrhage, as was shown in postmortem specimens of patients after continuous extradural anaesthesia [10].

Second, in so far as the large volume of blood injected into the extradural space is concerned, in an EB study with Tc-99m labelled red blood cells, Szeinfeld and co-workers [11] found that a volume of 15 ml produced a sufficient spread of the blood in the extradural space and concluded that greater volumes [12] are not necessary and probably unwarranted. Abouleish and colleagues reported a success rate of 89% in their patients with EB, with only 7–10 ml of blood injected [4]. Based on these studies, an injection volume of 25 ml for EB seems, in retrospect, to be unnecessary and unwise, and this large volume may have influenced the complication in our patient.

Third, important additional factors were the unilateral plaster of Paris dressing and the difficulty of walking with crutches. It should be noted that the dramatic worsening occurred 3 weeks after mobilization of the patient, 13 days after the end of PDPH, and the patient showed a distinct recovery while still using crutches; nevertheless, we believe that the abnormal walking cannot be excluded as a significant aetiological factor for the severe backache.

In conclusion, this complication after EB does not place into question the value of therapeutic EB for severe PDPH. Nonetheless, it provides a note of caution and argues in favour of using smaller volumes of blood for EB. We recommend a slow extradural injection of 15 ml or less if the patient feels radicular pain or significant back pain. If severe backache occurs after extradural or spinal anaesthesia or after EB, urgent investigation and therapy are mandatory. Although no pathological processes were found in our patient, severe backache after extradural block should be attributed to an extradural abscess or other serious pathological process until proved otherwise.

REFERENCES


