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Chronic adhesive arachnoiditis after repeat epidural blood patch

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ABSTRACT
Epidural blood patching is an effective treatment for postdural puncture headache but has potential risks. Arachnoiditis is a very rare disabling condition and few cases have been described following an epidural blood patch. We present a case of chronic adhesive arachnoiditis in a parturient treated with a repeat epidural blood patch. A healthy 29-year-old woman had an accidental dural puncture following epidural insertion during labour. Initial treatment of postdural puncture headache with an epidural blood patch was ineffective and was therefore repeated. She gradually developed severe neurological symptoms consistent with arachnoiditis confirmed with magnetic resonance imaging. Despite intensive multimodal treatment with analgesics and physiotherapy, her neurological condition remains unresolved two years later. This serious but rare complication should encourage caution when treating parturients with postdural puncture headache with a repeat epidural blood patch.

Keywords: Postdural puncture headache; Epidural blood patch; Arachnoiditis

Introduction

An epidural blood patch (EBP) has a reported efficacy of 32–73% in managing postdural puncture headache (PDPH).[1] The procedure is often performed 48 h after the onset of symptoms.[2] The optimal amount of autologous blood for an EBP would appear to be 20 mL.[1] The procedure is considered to be safe, although complications do occur and can be serious.[3–6] Repeat EBP may improve success rate but is controversial.[3,7] We describe a case of chronic adhesive arachnoiditis (CAA) that we believe may have been caused by the use of a repeat EBP.

Case report

A healthy, 29-year-old woman had an uncomplicated pregnancy. Labour proceeded normally and epidural analgesia was requested. Alcohol 70% without chlorhexidine was used for skin preparation. An 18-gauge Touhy needle was inserted at the L3–4 interspace and using a loss of resistance to saline technique an accidental dural puncture (ADP) occurred. In accordance with departmental guidelines, the epidural catheter was advanced intrathecally and used during labour providing satisfactory pain relief with intermittent boluses of bupivacaine 0.25%. The total dose administered was 15 mg. The catheter remained in place for 7 h and was removed immediately after delivery. No paraesthesiae were recorded.

Twelve hours after delivery, the patient developed a severe PDPH. An EBP was performed approximately 36 h after ADP using 25 mL of autologous blood injected at the L2–3 interspace. Her headache was substantially relieved but returned the following day. Two days after the first EBP, a second EBP was performed using 30 mL of autologous blood. At the end of the procedure, the patient experienced symptoms of pressure in the lower back. Unfortunately, the spinal level of the second EBP was not documented. The same experienced anaesthesiologist performed both procedures without

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technical difficulty using loss of resistance to saline. Skin preparation for the EBP and autologous blood collection was performed using alcohol 70% with chlorhexidine 0.5% on both occasions.

Approximately 7 h following the second EBP, the patient experienced lumbar pain and radiculopathy in both legs as well as pain radiating to the upper thoracic region. Subsequently, her neurologic symptoms improved and she was discharged from hospital. Five days later, she returned with low back pain, lumbosacral radiculopathy and inability to walk. Magnetic resonance imaging (MRI) showed changes consistent with arachnoiditis and an intradural haematoma extending from T12 to S2 which was more pronounced on the left side and combined with clumping of the nerve roots (Fig. 1). Initially, conservative analgesic therapy improved her condition slightly but during the following weeks her symptoms worsened. She developed loss of pain and cold sensation on her right foot, difficulty in standing and walking; the low back and leg pain worsened. The department of pain medicine was involved and she received additional analgesic therapy with only minor improvement. A second MRI scan performed one month later showed radiological evidence of arachnoiditis although the haematoma had reabsorbed. The patient received further treatment with anti-inflammatory drugs, corticosteroids and physiotherapy without significant symptomatic relief.

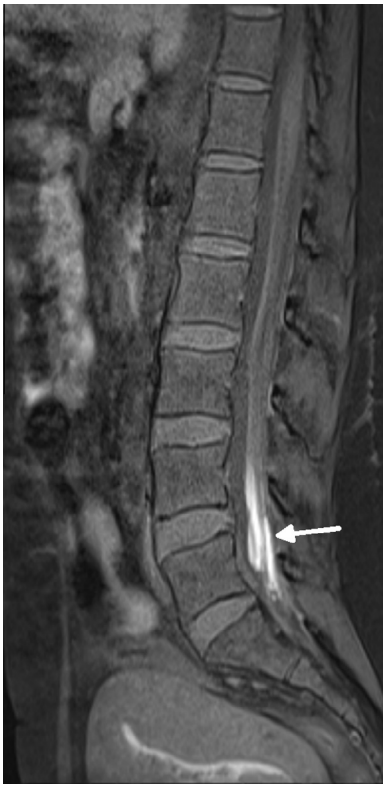

Fig. 1 Sagittal T1-weighted MRI scan without contrast. Intradural blood extends from T12 to S2. The maximal haematoma width is 10 mm at L5 (arrow).

Five months later, despite physiotherapy and social support, her ability to walk was restricted to 40 m due to severe pain. She was partly dependent on wheelchair for daily activities and a third MRI scan was unchanged. One year later, her back pain had improved but she still had severe pain in her legs and remained partly dependent on the wheelchair in spite of multimodal pain therapy. Follow-up MRI two years after the incident did not show further improvement and her physical condition remains unchanged.

Discussion

Arachnoiditis following EBP is extremely uncommon. In this case, the repeat EBP was the most likely cause of arachnoiditis with continued neurological deficit. However, skin preparation with chlorhexidine 0.5% in 70% alcohol or the intrathecal catheter may have played a causative role.[8–10]

Epidural analgesia for labour using local anaesthetics without preservatives has not been reported to be associated with CAA.[8] In a retrospective study of severe neurological complications after central neuraxial blockade (CNB), arachnoiditis was reported following spinal block in only two cases.[11] Long-lasting neurological deficit following CNB in the obstetric population has been reported in only one in 240 000.[12] Reversible neurological injury was reported in one in 6700 cases without any certain correlation to the CNB and no cases of arachnoiditis were found.[12]

Accidental dural puncture is known to complicate epidural anaesthesia with an incidence of about 1%.[2,13] The incidence of PDPH following ADP varies between 50% and 85%.[2,13] The mechanism of PDPH is not fully understood but is thought to be caused by leakage of cerebrospinal fluid (CSF) from the puncture site leading to decreased CSF volume, downward traction on pain-sensitive structures and compensatory vasodilatation of intracranial vessels.[14] The symptoms usually start within 48 h of ADP.[13] Headache is severe and postural and often combined with neck stiffness, altered auditory perception and sensitivity to light[14,15]; it often resolves spontaneously within one week.[15] Various treatments have been tried, but the most effective remains an EBP.[16] According to a recent Scandinavian survey, 86% of parturients with PDPH received an EBP.[2] The most common side effect of the procedure is low back pain, while other transient rare complications include fever, seizures, radicular pain and aseptic meningitis.[6]

Chronic adhesive arachnoiditis is an extremely rare condition and in only a few cases has EBP been implicated as the cause.[3–6,8] The diagnosis of CAA is difficult because of varied symptomatology and the lack of a