Epidural blood patch (EBP) is a commonly performed procedure to alleviate post-dural puncture headache (PDPH). Although very effective, EBP is not free of serious complications. This case is the first report of an EBP that, rather than being absorbed, organized to form a chronic spinal epidural hematoma (SEH), resulting in radicular symptoms. Furthermore, this is also the first report of an SEH undergoing ossification, a phenomenon known to occur with cranial epidural hematomas.

Case Report

A 39-yr-old, healthy woman, G5P4Ab0LC4, was admitted to the delivery room of another hospital in active labor at term. During her previous three deliveries, she received epidural analgesia without difficulty or complications. The patient had no history of back pain or migraine headaches. In the lateral decubitus position, a 20-gauge epidural catheter was inserted through an 18-gauge Tuohy needle. During insertion of the epidural needle, the patient felt a distinct sensation of membrane puncture followed by a postural headache and tinnitus, without nausea or vomiting. No paresthesia or pain was present upon catheter insertion. The patient received a test dose of local anesthetic, yielding almost immediate loss of sensation to spinal dermatome level T4, with motor paralysis and shortness of breath. The catheter was left in the subarachnoid space, and the patient delivered a healthy baby within the hour. The headache was treated with analgesics and hydration, with partial alleviation of symptoms. On the first postdelivery day, the catheter was removed and the patient was discharged to her home. She returned the following day to the emergency room because of exacerbation of symptoms. The patient did not have fever or focal neurologic deficits. A diagnosis of PDPH was made, and an EBP was performed. In the sitting position, at the L3-L4 intervertebral space, the epidural space was located, and autologous blood was injected into the epidural space at the same level as the Tuohy needle until substantial back pressure was felt (total 20 ml). The headache and tinnitus subsided, and the patient was released to her home after a few hours. During the next few hours, the pressure in the patient’s back had changed to radicular back pain. The severity of the radicular symptoms worsened over 2–3 days and reached its maximal magnitude as a severe (visual analog scale score of 8), incapacitating, burning/stabbing pain in her back, radiating to her right leg. Her general physician prescribed analgesics, traditional physiotherapy, chiropractic therapy, Alexander training, and acupuncture, without improvement. Only a year after delivery was she referred for an orthopedic consultation at our hospital.

The orthopedic surgeon referred her for magnetic resonance imaging, which demonstrated a 1.5-cm round lesion compressing the spinal nerve roots at the right neural foramina of L3-L4 and L4-L5 (fig. 1). Surgical exploration during general anesthesia revealed an ossifying organized epidural hematoma at L3-L5. This finding coincided with the anatomical location at which the EBP was performed. Microscopically, the tissue was composed of young bone and osteoid, many osteoblasts and osteoclasts, and vascular stroma. Microbiologic study showed numerous erythrocytes with few polymorphonuclear cells on Gram stain. There was no bacterial growth. Sixty-day patient follow-up showed full recovery.

Discussion

Post-dural puncture headache, first reported in 1898, is a complication of dural puncture during neuroaxial anesthesia or diagnostic lumbar puncture. The incidence after spinal anesthesia is 0.2–24% and up to 30% after lumbar puncture.1 Incidence increases with needle diameter and differs with needle design.2 The typical presentation is of a fronto-occipital headache accompanied by nausea, dizziness, and/or visual disturbances. Typically, the symptoms are exacerbated in the upright position and are relieved when the patient lies down.3 In 90% of cases, symptoms resolve spontaneously within 10 days. Initial treatment is conservative, including non-steroidal anti-inflammatory drugs, paracetamol, and bed rest.2 Two causal hypotheses exist to explain PDPH: (1) downward traction of intracranial structures secondary to cerebrospinal fluid (CSF) leakage through a hole in the dura and (2) the Monroe-Kellie hypothesis: the sum of volumes of the brain, CSF, and intracranial blood is constant.4 Therefore, venodilation compensates for the decrease in CSF volume and causes migraine-like headaches.

In the case of a severe headache unresponsive to conservative therapy, an EBP is recommended. First introduced in 1960 by Gormley, an EBP is the accepted standard treatment of PDPH, carrying 70–98% success rate when conservative treatment fails.1,5 Blood is injected into the epidural space at the same level as the dural puncture until the patient reports pressure or worsening headache or 15–20 ml has been injected.6 Epidural blood increases the CSF pressure by forming a mass effect and stimulates cerebral vasoconstriction. At the same time, a fibrin clot is formed and seals the dural rent, allowing CSF volume to normalize. This generally results in an immediate improvement in symptoms. Magnetic resonance imaging studies confirmed that the mass effect of an EBP resolves over 7 h.6-7 It is therefore generally accepted that the injection of blood to the...
solid spinal epidural mass that caused chronic first reported case of an EBP that organized to form a subsided within 48 h. This may result from the leakage of the adjacent hematoma. Another theory is that damaged vascularized tissues such as bone and dura provoke and initiate tissue responses such as inflammation, repair, and remodeling, resulting in osseous transformation. The development of radicular sympoms might be indirect evidence to a local inflammatory process that also contributed to the ossification. The natural sequence of healing is more rapid in children than in adults and might explain the fact that there are more reports of ossified cranial epidural hematomas in children. Therefore, the patient described here is the first report of an SEH that underwent ossification causing neurologic symptoms. To our knowledge, this is the first reported case of an EBP that organized to form a solid spinal epidural mass that caused chronic radicular symptoms. It is hypothesized that because the dura originates from the endosteum, it actively evokes extradural ossification of the adjacent hematoma. Another theory is that damaged vascularized tissues such as bone and dura provoke and initiate tissue responses such as inflammation, repair, and remodeling, resulting in osseous transformation. The development of radicular symptoms might be indirect evidence to a local inflammatory process that also contributed to the ossification. The natural sequence of healing is more rapid in children than in adults and might explain the fact that there are more reports of ossified cranial epidural hematomas in children.

In the current case, the blood of a mother soon after giving birth was injected into the epidural space. One can thus speculate that this blood, still containing fetal cells, implanted stem cells and/or growth factors in the epidural space. There are clues that umbilical cord blood might contain nonhematopoietic stem cells. At least one study isolated fibroblast-like cells with osteogenic potential in the human umbilical cord blood. Perhaps such cells initiated ossification of the maternal blood in the epidural space rather than being absorbed.

There is no question that EBP, a common procedure with few reported complications, is the treatment of choice for severe PDHP when conservative therapy fails. However, this case should remind anesthesiologists that EBP is an invasive procedure with the potential for morbidity. The development of persistent and progressive back pain or neurologic symptoms in patients after EBP should alert general practitioners and clinicians, because immediate orthopedic investigation of these symptoms may prevent permanent problems.


