CASE REPORTS

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Postpartum Dural Venous Sinus Thrombosis after Postdural Puncture Headache and Epidural Blood Patch

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DURAL venous sinus thrombosis (DVST) is an unusual complication of pregnancy, with an incidence of 1:10,000[1] to 1:25,000.[2] We report a case of postpartum transverse sinus thrombosis after attempted epidural analgesia, complicated by postdural puncture headache (PDPH), and epidural blood patch (EBP).

Case Report

A 22-yr-old gravida 2 para 1 woman was admitted to the hospital in active labor. She had no history of neurologic or hematologic disorders, arthritis, lupus, malignancy, or infection; had no allergies; was a nonsmoker; did not use alcohol; and was taking no medications. Her cervix was dilated 7 cm at the time epidural anesthesia was requested. During attempted epidural needle placement at L_5–S_1, a dural puncture occurred using a 17-gauge Tuohy needle. Further attempts at epidural placement were abandoned because of rapid progression of labor to complete cervical dilatation, with fetal bradycardia during contractions. No epidural catheter was placed, and no epidural medications were administered. With the use of perineal infiltration of local anesthesia, the patient spontaneously delivered a healthy term infant.

Several hours after delivery, the patient experienced the gradual onset of a dull, throbbing headache, which was completely relieved by placing the patient supine, but which returned immediately after sitting or standing. The headache was moderate in intensity the day of delivery. The next day (first postpartum day), the headache was so severe that an EBP was performed, using 12 ml autologous blood placed at L_5–S_1. This procedure failed to relieve her headache. On the second postpartum day, she complained of increasingly severe postural headache, now associated with nausea. A second EBP was performed at the L_5–S_1 level using 20 ml autologous blood. After the second EBP, the patient experienced complete pain relief that lasted 48 hr, but the headache returned to its original intensity on the fifth postpartum day. A definite postural component was noted, but the pain was not completely relieved with the patient recumbent. The headache was now primarily frontal, with some occipital pain and neck stiffness. She reported no nausea and remained afebrile. Because of the continued postural component of the pain, the clinical diagnosis continued to be PDPH. A third EBP was performed using 20 ml autologous blood. In addition, an intravenous infusion of 1 l lactated Ringer's solution that contained 500 mg caffeine was administered for 3 hr. After this procedure, the patient experienced only partial relief of her headache. During the presumed PDPH, there were no vision or hearing changes.

On the sixth postpartum day, the pain increased in intensity and was described as dull and throbbing, exaggerated by sitting or standing, but not entirely relieved by the recumbent position. The patient denied experiencing nausea, vomiting, confusion, incontinence, aphagia, or other neurologic symptoms; there was no papilledema. Because of the continuing headache, which now was no longer postural, a magnetic resonance image was obtained on the tenth postpartum day. It showed thrombosis and occlusion of the left transverse sinus (fig. 1). This was confirmed by a magnetic resonance venogram using the two-dimensional time-of-flight magnetic resonance angiogram technique (fig. 2).

The patient was admitted immediately to the hospital for anticoagulation and neurologic monitoring. Prenatal platelet counts were normal. Anticoagulation was begun using a heparin infusion. Coumadin (5 mg daily) was begun 2 days later, with a goal of maintaining an International Normalization Ratio (for prothrombin time) of 2–2.5. Intravenous meperidine was required for control of her headaches. Protein C, protein S, and antithrombin III concentrations were within normal limits. No laboratory evidence of a lupus anticoagulant or antinuclear antibody was noted. The patient's headaches decreased in severity and were controlled using oral pain medications. She was discharged after 8 days of hospitalization (the 16th postpartum day). Eight days after discharge, her headaches resolved completely. At the 6-month follow-up, no headaches or neurologic sequelae were seen, and the coumadin was discontinued.

Discussion

No previous reports are found of PDPH successfully treated by EBP with subsequent DVST. Because our patient recovered completely (if only temporarily) with EBP, it is likely she initially had a PDPH, followed 48 hr later by DVST.

The diagnostic dilemma posed by our case may be

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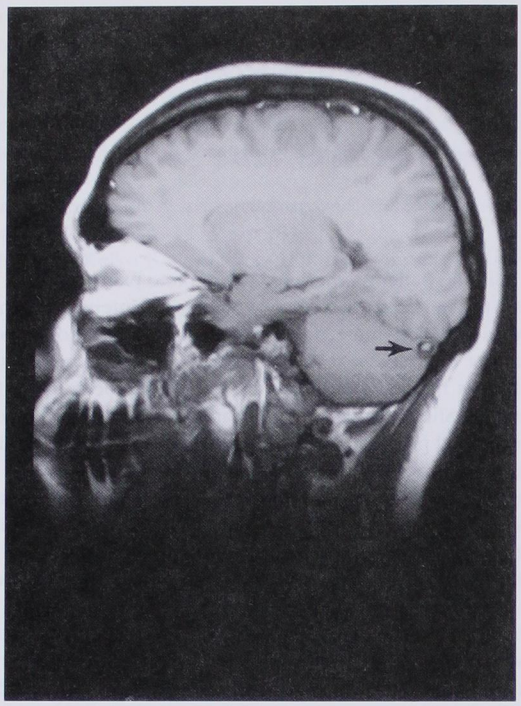

Fig. 1. T1-weighted spin echo sagittal magnetic resonance image shows hyperintensity of the thrombus in the left transverse sinus (arrow).

more common than previously recognized. According to the National Center for Health Statistics, there were more than 4 million live births performed in the United States in 1991,[3] with 16% (approximately 640,000) receiving epidural anesthesia.[4] There is a reported incidence of unintentional dural puncture during epidural placement that ranges from 1% to 7.6%[5,6]; therefore, the occurrence of wet tap would range between 6,400 and 48,640 incidents annually. The incidence of DVST during pregnancy has been reported from 1 in 10,000 to 1 in 25,000,[2] representing approximately 160–400 episodes per year. If epidural anesthesia with subsequent unwanted dural puncture does not affect the incidence of DVST, then, by chance, one might predict that as many as 4.8 patients annually might suffer inadvertent dural puncture followed by DVST. Gibbs§ reported that recent data indicate that 29% of deliveries are performed using epidural anesthesia; this would almost double the numbers discussed earlier. When patients who received intentional dural puncture (*i.e.*, spinal anesthesia or combined spinal-epidural anesthesia) are considered, the number of potential occurrences of dural puncture with subsequent DVST could increase.

The majority of puerperal DVST episodes occur 1–2 weeks after delivery,[1] as was the situation in our case. Because both PDPH and DVST occur in the early postpartum period, there exists the possibility of both misdiagnosis and mistreatment (or delayed treatment) if headache after unintentional dural puncture is actually caused by DVST.

Younker *et al.*[7] reported DVST after vaginal delivery of a primigravida after two failed attempts at epidural catheter placement, both of which resulted in accidental punctures. Unlike our case, the severe headache that developed immediately after delivery did not respond to EBP. Siegle *et al.*[8] reported a similar case of DVST in which no EBP was performed. A patient reported by Ravindran *et al.*[9] experienced sagittal sinus thrombosis after uneventful epidural anesthesia for labor and delivery; an EBP was performed without change in neurologic symptoms. Another report involved postoperative sagittal sinus thrombosis after spinal anesthesia for hemorrhoidectomy in a 35-year-old man.[10] He experienced a severe postural headache on the first postoperative day, but no EBP was performed.

Dural venous sinus thrombosis during the puerperium usually occurs during the first 7 postpartum days.[1] The most frequent presenting symptom is headache.[11,12]

Fig. 2. Two-dimensional (2-D) time-of-flight magnetic resonance angiogram confirms lack of flow in the left transverse sinus (arrows).

§ Gibbs CP: Obstetric Anesthesia: USA. Unpublished data presented at the Society for Obstetric Anesthesia and Perinatology 28th Annual Meeting, Tucson, Arizona, May 1–4, 1996.

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Other neurologic signs include papilledema, focal deficits, seizures, and coma.[13] The mechanism for these neurologic changes is thought to be thrombosis of the cerebral venous sinuses and arachnoid granulations, resulting in inadequate venous and cerebral spinal fluid drainage, with increased intracranial pressure, seizures, and focal neurologic deficits.[2]

Magnetic resonance imaging is a sensitive test for the detection of DVST and may be the investigation of choice when this disorder is suspected.[14] Our patient's magnetic resonance image (fig. 1) showed increased signal intensity instead of the normal flow void in the left transverse sinus. This finding persisted in other planes and other pulse sequences. Dural venous sinus thrombosis results in increased signal intensity instead of the normal flow void.[15] Magnetic resonance angiography (fig. 2) was used to confirm the diagnosis of DVST in our patient. This technique is based on gradient-refocused scans that show increased signal intensity instead of a flow void for flowing blood. These scans are then reconstructed into a three-dimensional format for the magnetic resonance angiogram or venogram; computed tomography may be inadequate, and angiography should be reserved for difficult cases.[16]

Dural venous sinus thrombosis occurs most frequently in association with pregnancy.[12] The etiology of puerperal DVST is unclear, but two of the suggested mechanisms are[1]: (1) damage to venous sinuses due to fluctuations in intracranial pressure during delivery, and (2) an increased thrombotic tendency due to changes in various coagulation factors. The hypercoagulable state of pregnancy, which may contribute to its association with DVST, has been attributed to increased platelet adhesion and an increase in clotting factors (fibrinogen and factors VII, VIII, and X).[13] Other conditions associated with increased risk of DVST include hereditary antithrombin III deficiency[17] and dysfunctional protein C with decreased free protein S concentration.[18] Hereditary protein S deficiency has been associated with DVST in a patient taking oral contraceptives.[19]

Our patient was treated by anticoagulation with intravenous heparin followed by oral coumadin. The goal of therapy is to reduce intracranial pressure and to avoid extension of the thrombus.[13] Although controversial, anticoagulants are indicated in the absence of cerebral hemorrhage or hemorrhagic infarction.[13,20] Bousser *et al.*[11] reported a series of 38 patients with angiographically proven DVST. Four of the 15 untreated patients died; none of the 23 patients treated with anticoagulants died. Extremely severe cases (involving seizure and progressive neurologic deficit) were treated successfully with urokinase.[2,21]

Our case emphasizes the importance of including DVST in the differential diagnosis of headache after dural puncture in the puerperium. The significant role of magnetic resonance imaging techniques in diagnosis is also demonstrated.

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Transient Paraparesis after Postdural Puncture Spinal Hematoma in a Patient Receiving Ketorolac

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SPINAL hematoma is a known, rare complication of spinal and epidural anesthesia. After dural puncture, spinal (epidural, subarachnoid, or subdural) hematoma formation has been described to result in paraparesis in patients with chronic renal failure,[1] liver failure,[2] and thrombocytopenia.[3] In otherwise healthy patients, hematoma formation and paraparesis have occurred with spinal or epidural anesthesia in association with complete anticoagulation,[4] low molecular weight heparin,[5] minidose heparin,[6] antiplatelet drugs,[7] and aspirin.[8] The majority of reported neurologic deficits have been permanent, despite surgical decompression and aggressive rehabilitation.[9]

We present a case of postdural puncture spinal hematoma and paraparesis that occurred in an otherwise healthy woman receiving ketorolac postoperatively and that resolved spontaneously and completely.

Case Report

A 66-yr-old woman (163 cm, 54 kg) was admitted for surgical repair of a third-degree cystocele and second-degree rectocele during spinal anesthesia. Her medical history was significant for hypertension well controlled by propranolol hydrochloric acid and hydrochlorothiazide/triamterene. She also had osteoarthritis, for which she took 75 mg diclofenac sodium (Voltaren, Ciba-Geigy, Ardsley NY) twice daily. She had no history of easy bleeding and previously had five spontaneous vaginal deliveries, an appendectomy, and a vaginal hysterectomy without incident. Preoperative laboratory values consisted of a white blood cell count equal to 3.7 K/ul (with normal differential), a hemoglobin equal to 12.0 g/dl, a hematocrit equal to 37%, and a platelet count of 144,000/ul.

She took her propranolol on the morning of surgery, as instructed during her preoperative evaluation, and discontinued her diclofenac the previous day. She received, as routine premedication, 150 mg ranitidine orally and 2 mg midazolam intramuscularly on call to the operating room. For administration of the spinal anesthetic, the patient was seated and two attempts were made at L4–5 in a right paramedian approach using a 27-g Quincke needle, with only bone and no blood encountered. A third attempt, using a 25-g Quincke needle, resulted in clear, free-flowing spinal fluid. A T-8 sensory level was achieved after injection of 1.5 ml 5% lidocaine. Surgery proceeded uneventfully in the lithotomy position with less than 100 ml blood loss. Intraoperative medications were 1 g cefazolin intravenously, 1.5 mg midazolam intravenously, and 30 mg ketorolac intravenously, which was administered at the end of the procedure, 1 h after lumbar puncture. Ketorolac (15 mg intramuscularly) was administered every 6 h for three doses after this initial dose, and diclofenac was not used perioperatively. No medications were administered for prophylaxis of deep vein thrombosis.

The patient's spinal anesthetic was noted to be resolving before discharge from the postanesthesia care unit and later was noted to be resolved completely with her first request for pain medications 3 h postoperatively. At this time, she received 10 mg morphine intramuscularly. On the first postoperative day after an uneventful evening, the patient's urinary catheter was removed and she ambulated at 7:15 AM without difficulty. At 12:45 PM, she first complained of bilateral lower extremity "aching" without back pain. She received two oxycodone and acetaminophen orally, which relieved the pain. At 5:50 PM, she had an episode of vomiting that was followed by the acute onset of "weakness" in both legs. At 8:20 PM, she related this weakness to the nursing staff, and we examined her.

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