

CASE REPORTS

17. Muramatsu S, Mizuno Y, Murayama H, Ikemoto S: Hereditary antithrombin III deficiency with a superior sagittal sinus thrombosis: Evidence for a possible mutation starting in the mother of the propositus. *Thromb Res* 1990; 57:593-600

18. Roos KL, Pascuzzi RM, Kuharik MA, Shapiro AD, Manco-Johnson MJ: Postpartum intracranial venous thrombosis associated with dysfunctional protein C and deficiency of protein S. *Obstet Gynecol* 1990; 76:492-4

19. Heistering M, Rimpl E, Illiasch H, Turck H, Kyrle PA, Lechner K, Pabinger I: Cerebral sinus thrombosis in a patient with hereditary protein S deficiency: Case report and review of the literature. *Ann Hematol* 1992; 64:105-9

20. Gates PC: Cerebral venous thrombosis. A retrospective review. *Aust N Z J Med* 1986; 16:766-70

21. Eskridge JM, Wessbecher FW: Thrombolysis for superior sagittal sinus thrombosis. *J Vasc Interv Radiol* 1991; 2:89-93

Anesthesiology
1997; 86:490-4
© 1997 American Society of Anesthesiologists, Inc.
Lippincott-Raven Publishers

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

J. C. Gerancher, M.D.,* Ronald Waterer, M.D.,† Joseph Middleton, M.D.†

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

* Assistant Professor, Wake Forest University Medical Center, Department of Anesthesia, Winston-Salem, North Carolina.

† Anesthesiologist, Forsyth Anesthesia Associates, Medical Park Hospital, Winston-Salem, North Carolina.

Received from Wake Forest University Medical Center and Forsyth Anesthesia Associates, Medical Park Hospital, Winston-Salem, North Carolina. Submitted for publication July 25, 1996. Accepted for publication October 6, 1996.

Address reprint requests to Dr. Gerancher: Assistant Professor, Wake Forest University Medical Center, Medical Center Blvd., Winston-Salem, North Carolina 27157-1009.

Key words: Anesthetic techniques. Spinal complications, paraplegia. Neurologic measuring techniques: MRI.

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.

CASE REPORTS

The patient was alert, oriented, and in no distress except for a bilateral "dull, bone-aching" calf pain. Vital signs were normal. No operative site bleeding was present. A cranial nerve and upper extremity examination was normal. She had 5/5 strength to all lower extremity muscle groups except 4/5 strength with dorsiflexion of the left foot and 3/5 strength in both extensor hallucis longus muscles. The patient was unable to stand on either leg alone and failed a trial of ambulation secondary to lower extremity weakness. Deep tendon reflexes were 2+ throughout. Sensation to touch, pinprick, and temperature was intact. Bowel and bladder function was normal.

Strongly suspecting a diagnosis of spinal hematoma, we obtained a neurosurgery consult and arranged for an emergency magnetic resonance image (MRI) to evaluate the patient for surgical decompression. All nonsteroidal antiinflammatory drugs (NSAIDs) were discontinued. An MRI without contrast was interpreted as "acute/subacute hemorrhage which is epidural with a mass effect from L2-3 downward and with some component of subarachnoid and subdural blood" (fig. 1). This study was completed 7 h after the patient's first complaint of calf pain and 43 h after the dural puncture. Our neurosurgery consultant found no indication for surgical intervention, citing the patient's stable neurologic examination. He recommended, instead, frequent neurologic examinations and 10 mg dexamethasone intravenously every 6 h. Of note, a second platelet count at this time was 80,000/ul, and hemoglobin and hematocrit had decreased to 10.1 g/dl and 30.9%, respectively. Prothrombin time and partial thromboplastin time were normal, and a measurement of bleeding time was not obtained.

During the next 2 days, the patient made a progressive and full recovery from her paraparesis. By the time she returned from the MRI suite, her calf pain had resolved. She was first able to ambulate without assistance 24 h after her initial complaint of weakness. She left the hospital 4 days postoperatively on a tapered dose of oral dexamethasone. A repeat MRI examination was not performed. She had no sequelae at a follow-up of 3 months.

Discussion

Two features of this case are especially unusual: the occurrence of spinal hematoma in the absence of anticoagulation and the full recovery of neurologic function without surgical decompression.

Six previous cases have described spinal hematoma occurring after dural puncture in the absence of anticoagulation or coagulopathy (table 1).^{8,10-14} All of these reports have involved at least some difficulty during dural puncture or the return of bloody cerebrospinal fluid. This case report is similar to the others in that spinal anesthesia, although successful, was not accomplished on the first attempt. Of note, this case is the first to report spinal hematoma after dural puncture with a small-gauge spinal needle, and use of a small-gauge spinal needle did not prevent hematoma formation.

Ketorolac therapy in our patient is an interesting aspect of this case report because NSAIDs, in general, are

known to cause a qualitative platelet defect and because ketorolac, specifically, was shown recently to worsen platelet function in patients during spinal anesthesia.¹⁵ Neither ketorolac nor diclofenac is known to cause thrombocytopenia like that first apparent in our patient at the time spinal hematoma was diagnosed. We speculate that this decrease in platelet count may be the result of platelet consumption at the surgical site and in the spinal hematoma itself. In one of the other six case reports listed in table 1, the patient took "aspirin and other anti-inflammatory drugs," whereas aspirin and NSAID use was not reported by the authors of the remaining cases.

These seven cases suggest that difficult or traumatic lumbar puncture is a risk factor for spinal hematoma formation. However, a conclusion cannot be drawn as to whether ketorolac therapy (or NSAID therapy, in general) places patients who received spinal anesthetics at greater risk as well.

The transient nature of our patient's symptoms is the second interesting feature of this case report. Only three previous case reports describe paraparesis secondary to spinal hematoma after dural puncture or epidural anesthesia that resolved without surgical decompression (table 2).^{3,16,17} With surgical decompression, resolution has been achieved, and this has been well described. In their review of surgery for spontaneous spinal hematoma, Foo *et al.*¹⁸ showed recovery of spinal hematoma to be dependent on the degree and nature of the preoperative neurologic deficit. These authors found that complete recovery after surgery occurred in 42% of incomplete sensorimotor lesions, in 26% of incomplete sensory/complete motor lesions, and in 11.3% of complete sensorimotor lesions. The incomplete and limited nature of our patient's neurologic deficit, then, might suggest a reasonable prognosis for recovery, had surgery been performed.

Her prognosis for recovery after conservative therapy in the absence of surgical decompression does not appear to be predicted by the limited literature available. In their recent review of the literature, Owens *et al.*⁹ found that the majority of patients with known postdural puncture spinal hematoma and paraparesis were treated surgically (16 of 30 patients) and, despite surgical decompression, most deficits remained permanent (only 6 of these 16 patients had "good" outcomes). The remaining 14 patients were not treated surgically. Only two of these patients treated with conservative therapy had a "good" recovery.

Spinal hematoma is certainly not the only cause of

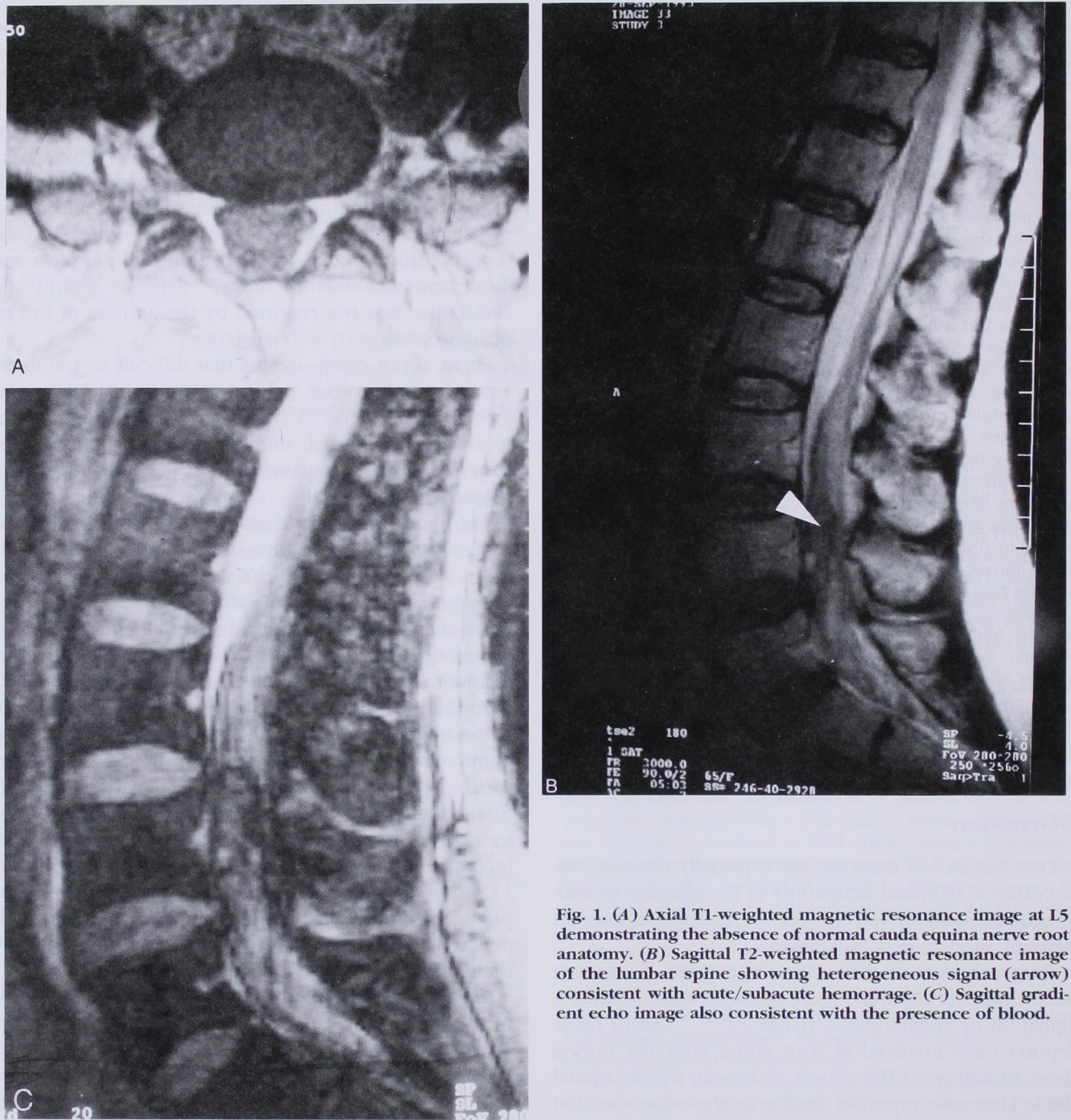


Fig. 1. (A) Axial T1-weighted magnetic resonance image at L5 demonstrating the absence of normal cauda equina nerve root anatomy. (B) Sagittal T2-weighted magnetic resonance image of the lumbar spine showing heterogeneous signal (arrow) consistent with acute/subacute hemorrhage. (C) Sagittal gradient echo image also consistent with the presence of blood.

neurologic deficit after spinal or epidural anesthesia. For example, patient positioning in hyperlordosis has been associated with spinal cord infarction and paraparesis during regional and general anesthesia.¹⁹ Recently, investigators in two clinical studies described a high

incidence of reversible neurologic deficits, uncovered when neurologic symptoms were prospectively sought out after single injection spinal anesthesia with 5% hyperbaric lidocaine.^{20,21} Evidence for a direct effect by the anesthetic agent exists. Irreversible neurologic

Table 1. Spinal Hematoma Following Dural Puncture in the Absence of Anticoagulation or Coagulopathy

Reference	Year	Description	Needle	Difficulty	Bloody?	Time to Symptoms (h)	Outcome
Cooke ¹⁰	1911	Tubercular meningitis	Not reported	Not reported, multiple SAB; 40 ml CSF withdrawn	Yes	Not reported	Died
King ¹¹	1959	Morbid obesity, renal calculus	Not reported	Not reported	Yes	24	Died after laminectomy
Lerner ¹²	1973	Osteomyelitis of the knee	22-g Quincke	Multiple attempts, SAB abandoned	Yes	20	No improvement after laminectomy
Rengachary ¹³	1974	Hip fracture	Not reported	Multiple attempts, SAB abandoned	Not reported	12	Partial recovery after laminectomy
Kirkpatrick ¹⁴	1975	Spinal to evaluate radiculopathy	Not reported	"Minimal difficulty"	No	24	Partial recovery after laminectomy
Greensite ⁸	1980	Knee replacement ASA, NSAIDS	16-g Tuohy	Two attempts, patient's epidural was converted to SAB	Yes	36	No improvement after laminectomy
Gerancher (current study)	1997	a&p repair, ketorolac, diclofenac	25/27-g Quincke	Multiple attempts	No	36	Full recovery

ASA = aspirin; NSAIDS = nonsteroidal anti-inflammatory drugs; a&p = anterior and posterior; CSF = cerebrospinal fluid; SAB = subarachnoid block.

deficits have occurred rarely in cauda equina syndrome after continuous spinal anesthesia with large doses of 5% hyperbaric lidocaine,²²⁻²⁴ and experimentally irreversible conduction block has been described in isolated nerves exposed to 5% hyperbaric lidocaine.²⁵ Only recently, the reversible neurologic symptoms after single injection spinal anesthesia with 5% hyperbaric lidocaine or transient radicular irritation have been clinically recognized²⁶ but appear to be common. The timing of presentation and our patient's initial symptoms of bilateral calf pain are very similar to the transient radicular irritation seen by Pollack *et al.*²⁰ and Hampl *et al.*²¹ in their clinical studies of single injection spinal anesthesia discussed earlier.

Two important differences exist in our case. First, our patient had a motor deficit that prevented ambulation, whereas those patients who experienced transient radicular irritation after single injection spinal anesthesia had only sensory changes and pain. Second, our patient had blood in her epidural, subdural, and subarachnoid spaces documented by MRI. We believe it is likely that this blood caused radicular irritation that presented as calf pain. Although it is possible our patient had an incomplete, transient cauda equina syndrome secondary to a single spinal injection of 5% hyperbaric lidocaine and spinal blood incidentally discovered on MRI, we believe the MRI findings in this case, along with the presentation and timing of symptoms, are most consistent with paraparesis secondary to spinal hematoma.

Our patient's neurologic deficit was transient and limited — so much so that we speculate that, in some cases, postdural puncture spinal bleeding may occur but remain subclinical or unreported. Some patients with less bleeding than our patient might have no symptoms, whereas other patients might present with only transient radicular irritation-type symptoms. We know from the literature that still other patients with spinal hematoma are not so fortunate as ours. These patients have been reported in the literature when their symptoms are progressive and often irreversible. This case report may represent one end of the spectrum of postdural puncture spinal hematoma where symptoms were limited and transient. The late onset of symptoms in this case report, the failure of our patient to bring her new motor deficit to the attention of her caregivers, and the similarity between the clinical presentation of this spinal hematoma and transient radicular irritation syndrome, all serve to underscore the importance of continued vigilance in monitoring our patients for neurologic deficit after spinal and epidural anesthesia.