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Unusual Cause of Weakness of the Lower Extremity Following Vaginal Delivery under Epidural Analgesia: Iliopsoas Muscle Strain

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Neurologic sequelae are among the most feared complications of epidural anesthesia in obstetric patients. Any patient who develops postpartum weakness or paralysis in the lower extremities after having received an epidural anesthetic presents a problem with the differential diagnosis. Is this weakness a manifestation of neurologic injury that has resulted from the epidural anesthetic or a manifestation of maternal diseases? Has traumatic labor and delivery caused nerve damage, resulting in weakness of the lower extremity?¹⁻⁶ We describe a case in which transient weakness of the lower extremities occurred following vaginal delivery under epidural analgesia. This unusual complication resulted from bilateral iliopsoas muscle strain and was not related to neurologic injury of the epidural anesthetic itself or nerve damage from traumatic labor and delivery. Epidural anesthesia, however, was implicated as a contributing factor to the muscle injury.

REPORT OF A CASE

A 43-year-old, gravida 2, para 1, woman was admitted to the hospital in active labor at term gestation. Her antepartum course was benign, and there were no complicating factors in her medical history. A previous vaginal delivery had been uneventful. After a 4-5 cm cervical dilatation occurred, the vertex fetal presentation, an epidural block was performed with the patient in the sitting position. The epidural space was entered with a 17-gauge Tuohy needle at the L3-4 intervertebral space using a midline approach and was identified by loss of resistance technique with a single attempt. No back pain or paresthesias

occurred during needle insertion. A test dose of 0.25% bupivacaine, 3 ml, was tolerated well, and a total of 8 ml of the same local anesthetic solution was injected. An epidural catheter was threaded into the epidural space without difficulty or elicitation of paresthesias and was fixed with approximately 2 cm of the catheter remaining in the epidural space. Analgesia throughout labor was satisfactory. The vital signs remained stable throughout a slow course of labor eventually requiring augmentation by oxytocin for hypotonic uterine contractions. A supplemental dose of 5 ml 0.37% bupivacaine was injected through the epidural catheter 2 h after the initial dose, and an additional 10 ml of 0.25% bupivacaine was given approximately 3 h after the second dose when the patient was transferred to the delivery table and placed in the lithotomy position for vacuum extraction. Second stage labor continued for 2 h, despite strenuous bearing-down efforts by the patient, with marked flexion of the hips on the pelvis similar to fetal posture *in utero*. The patient was able to exert maximal uninhibited expulsive efforts to each contraction every 2-3 min, lasting 30-45 s, with good motor strength under a degree of epidural analgesia. After having labored for a total of more than 10 h, the patient delivered a 7-pound, 11-ounce infant, Apgar score of 9 and 9 in 1 and 5 min, respectively, with the assistance of vacuum extraction. Following repair of a midline episiotomy, the patient was able to move herself from the delivery table to the bed approximately 1 h after having been placed in the lithotomy position.

Approximately 8 h after delivery, the patient complained of bilateral lower-extremity weakness and groin pain during assisted walking. An examination by the anesthesiologist at this time revealed a moderate inability to flex both thighs at the hip and tenderness in the groins. Passive flexion of the thighs elicited bilateral groin pain. Knee and ankle movements were intact. There were no sensory deficits to pinprick and touch. Her deep tendon reflexes and sphincter control were normal. A pelvic examination performed by the obstetrician revealed no abnormal masses or evidence of hematoma. Coagulation studies were normal.

On the second postpartum day the patient was completely unable to move her thighs. A neurologist and an orthopedic surgeon were consulted, and their respective patient evaluations reiterated the previous findings mentioned above: loss of flexor strength of the thighs bilaterally and no evidence of motor loss of any other muscle groups. There were no sensory deficits. All reflexes were normal. Based on these findings, a diagnosis of bilateral iliopsoas muscle strain was made without proceeding to further diagnostic studies. Physiotherapy following 2-3 days of bed rest was recommended.

The patient improved slowly with these conservative measures, and

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by the sixth postpartum day she was walking with a walker. On the eighth day she was able to climb a flight of stairs with the aid of a cane, and on the ninth day she was finally discharged home, able to walk without assistance. About 2 months after returning home she reported that her strength of the thighs and ambulation were completely normal, and there was no residual pain.

DISCUSSION

Prolonged lower-extremity weakness occasionally may be a result of unduly prolonged neural blockade from an epidural anesthetic. Sensory and motor blockade exceeding 60 h have been reported following epidural anesthesia with 0.5% bupivacaine.⁷ In our case, however, a prolonged neural blockade was not likely, since she had a full return of sensation, and the weakness was limited to a single muscle group.

Motor weakness resulting from various neurologic causes is always accompanied by other neurologic deficits such as sensory, reflex, or bladder and bowel dysfunction, signaling actual damage to the neural structures. Motor deficits after epidural anesthesia may result from varying degrees of trauma, compression, or ischemia of the spinal cord or nerve roots. Direct trauma to the spinal cord or nerve roots usually is accompanied by pain on needle insertion or catheter insertion or upon injection of local anesthetic and is followed by localized sensory or motor deficits.¹ Paraplegia resulting from compression of the spinal cord or nerve roots by the epidural hematoma may occur in the presence of clotting abnormalities or in patients receiving anticoagulants. Symptoms of cord or nerve root compression include backache or radicular pain, sensory deficits, and sphincter dysfunction, in addition to motor weakness. Epidural abscess likewise may cause cord compression and resultant paraplegia. Back pain, tenderness, fever, and leukocytosis may arouse suspicion of the abscess. Anterior spinal artery syndrome, a complication of cord ischemia, is characterized by lower-extremity weakness. Hypotension and epinephrine-containing local anesthetic have been implicated as a cause of this syndrome. Chemical agents, including certain local anesthetic preparation, have been cited as the cause of adhesive arachnoiditis with subsequent neurologic deficits such as weakness of the lower extremity, impaired sphincter control, and sensory deficits.⁴

Since the weakness in our patient was symmetric bilaterally and involved proximal muscles of the pelvis, intrinsic spinal cord lesions should be considered in the differential diagnosis. Such lesions are spinal cord tumors, which can cause significant sensory deficits, and impairment of sphincter control in the bilateral involvement of the spinal cord.⁶ Epidural hemangiomas often enlarge during pregnancy and subsequently cause symptoms of cord compression.⁸ Multiple sclerosis in pregnancy may present with weakness of the lower extremity accompanied by visual disorders and loss of coordination. Sporadic cases

of relapsing idiopathic polyneuritis in pregnancy has been presented with weakness of the extremities, progressing to facial palsy and respiratory depression.⁹ In our case, acute onset of the weakness and fast recovery without any neurologic deficits discounted the possibility of the intrinsic spinal cord lesions.

Trauma to the lumbosacral plexus or peripheral nerve during childbirth can result in weakness of various muscle groups of the lower extremity.⁵ Such trauma is caused by compression of the descending fetal head or by instrumentation with obstetric forceps. The weakness involves the distal muscle group, and sensory deficits are noted in the distribution of the individual nerve. Obturator nerve palsy by a descending fetal head can result in weakness of adduction of the thigh and sensory loss of the medial aspect of the thigh. Peroneal nerve palsy resulting in foot-drop has been associated with improper use of knee stirrups. Cases of paraplegia following labor also have been attributed to large intervertebral disc protrusion.¹⁰ A case of moderate iliopsoas weakness associated with femoral neuropathy was reported following prolonged labor and subsequent cesarean section.¹¹ The femoral nerve palsy resulted in paralysis of extension of the leg in addition to weakness of flexion of the thigh, sensory loss in the anterior aspect of the thigh and medial aspect of the leg, and loss of patellar reflex.

Other etiologic considerations include rare myopathic states, such as muscular dystrophy, myotonia congenita, and familial periodic paralysis. These muscular disorders may present paralysis of limb muscles without sensory deficits but are characterized by family history, insidious onset, and progressive course of the disorders.

In our case, the consistent finding of the weakness without any neurologic deficits in the absence of any myopathy history, and elicitation of pain and tenderness in the groin suggest the possibility of muscle damage. Since the weakness involved inability to flex the thighs at the hips, attention was directed to the iliopsoas muscles, which are the major flexors of the thighs. Iliopsoas muscle injury is uncommon and usually is caused by activity requiring violent contraction of the muscle itself.^{12,13} Bilateral iliopsoas strain without associated neuropathy, presented as transient lower-extremity weakness following labor, has not been described previously. Although the exact mechanism of the muscle strain in our patient is not entirely clear, we suspect that repeated strenuous bearing-down efforts with severe flexion of the hips during a protracted second stage labor may have been responsible for the injury. The patient may have been unable to perceive pain arising from the muscle at the time of injury because of epidural analgesia, and this may have contributed to further muscle injury by permitting repeated muscle contractions in the absence of appropriate pain perception. This also explains how the patient was able to move herself off the delivery table without complaining of pain.

In summary, although bilateral weakness of the lower extremities following an epidural anesthetic raises the suspicion of neurologic complication of the epidural anesthesia, lower-extremity weakness following vaginal delivery may result from iliopsoas muscle strain without associated neurologic injury as in our case. Accurate diagnosis of this complication, which can be made primarily on the basis of physical examination alone, may obviate the need for unnecessary and often costly diagnostic studies and can assure the patient of a benign prognosis and eventual recovery.

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Isoflurane for Neuroanesthesia: Risk Factors for Increases in Intracranial Pressure

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Isoflurane often is used for neuroanesthesia because it has the least effect on cerebral blood flow (CBF)[¶] and cerebrospinal fluid pressure^{1,2} of the currently available volatile anesthetics. While animal studies have tended to reinforce this clinical impression,³ some clinicians have questioned whether isoflurane should be labeled as the agent of choice for neuroanesthesia.⁴ We have been impressed that an occasional patient with an intracranial mass lesion will have increased intracranial pressure (ICP) when isoflurane is administered, despite prior institution of modest hyperventilation (fig. 1). We undertook this study

to identify the risk factors that make intracranial hypertension a likely event during isoflurane anesthesia.

MATERIALS AND METHODS

Fourteen unpremedicated patients (ages 42-73 years) were scheduled for elective craniotomy for excision of malignant supratentorial neoplasms. The protocol was approved by our institution's human studies committee. Computed tomographic (CT) scans of patients' heads were obtained within 3 days before operation and were interpreted by a neuroradiologist with regard to size, type, and location of lesion; the presence of midline shift; the degree of effacement of the lateral ventricles; and the extent of cerebral edema surrounding the tumor. Peritumor edema was quantitated on a visual scale ranging from 0 (no edema) to 3+ (edema present throughout the cerebral hemisphere).⁵

General anesthesia was induced with thiopental (3 mg/kg iv) and maintained with nitrous oxide, 70% in oxygen. Endotracheal intubation was facilitated with pancuronium, 0.1 mg/kg iv and was performed after a second dose of thiopental, 2 mg/kg, iv, plus lidocaine, 1.5 mg/kg, iv. Ventilation was controlled to maintain end-tidal carbon dioxide tension (PETCO₂) at approximately 26 mmHg (Beckman LB2®). A radial arterial catheter was inserted either before or immediately after induction of general anesthesia.

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