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Spinal Cord Infarction after Surgery in a Patient in the Hyperlordotic Position

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PARAPLEGIA after epidural analgesia and surgery has been reported secondary to a neurotoxic effect of highly concentrated local anesthetic accidentally injected into the subarachnoid space, ischemic anterior spinal artery syndrome attributed to inadvertent arterial damage during surgery, or to a vasoconstrictive effect of additives to local anesthetics.[1,2] However, in three reports[1,3,4] and in the current case, a patient's hyperlordotic position during an operation in the pelvis seemed to play a major role in developing ischemic infarction of the spinal cord. This is the first report that includes detailed electrophysiologic and magnetic resonance imaging (MRI) findings that illustrate the pathophysiologic nature of this complication of surgery.

Case Report

A 43-yr-old man with infiltrating carcinoma of the bladder underwent radical resection of the bladder, prostate, and seminal vesicles with *en bloc* lymphadenectomy followed by formation of an ileal bladder. Preoperative history included cigarette smoking and increased concentration of uric acid (7.7 mg/dl), cholesterol (291 mg/dl), and triglycerides (445 mg/dl). Blood pressure was 120/70 mmHg. To maintain postoperative analgesia, an epidural catheter (20-G) was introduced between the 3rd and 4th lumbar laminae, advanced for 4–5 cm, and tested in its epidural site with an injection of 5 ml 0.5% bupivacaine preoperatively. The operation lasted 10 h. Throughout the operation, the patient was placed in a hyperlordotic supine position with a 17° head- and trunk-down tilt (vertex at L1) and the legs extended 17° at the hips to raise the pubis. Continuous intraarterial monitoring of systemic blood pressure documented 90/50 mmHg as the lowest recording. Throughout the procedure, the systolic pressure was less than 100 mmHg for only 15 min in the premedication phase. Blood loss of 7 l during the operation was replaced by autologous blood, fresh frozen plasma, 0.9% NaCl, and colloids. Toward the end of the operation, 10 mg morphine in 10 ml 0.9% NaCl was instilled through the epidural catheter, and 2 h later, 0.25% bupivacaine was started at a rate of 4 ml/h, after an initial test dose of 4 ml 0.5% bupivacaine. The patient was sedated, and his lungs were ventilated until the following morning, at which time, on first awakening after extubation, he complained of a complete loss of sensation in the left lower limb and right leg and foot. A severe weakness of both lower limbs was noticed, making him unable to stand. Initially, this was thought to be due to the pharmacologic action of bupivacaine, but the weakness persisted when the epidural analgesia was discontinued on the 3rd postoperative day. There was no evidence of progression of the neurologic deficit after awakening. A plain MRI scan of the lumbar spine on the 3rd day and a plain and gadolinium MRI scan of the lumbar and thoracic spine on the 4th day failed to demonstrate any spinal cord abnormality. However, in the dorsal third of the vertebral bodies of L2 and L3, an area of hyperintense signal was visible in T2- and T1-weighted images (figs. 1A and 1B). The same day, examination of cerebrospinal fluid showed lymphocytosis (22/mm^3), a slightly elevated total protein (0.97 g/l, Lowry) and no intrathecal IgG production, indicating a partial breakdown of blood-cerebrospinal fluid barrier.

On the 7th day, physical examination revealed hypalgesia and hypesthesia in dermatomes L1–L3 on the left and L4–S1 on the right and analgesia and anesthesia in L4–S5 on the left and S2–S5 on the right. A complete flaccid paralysis of the left lower extremity and paresis grade IV for hip flexion and grade III for hip, knee, and ankle extension as well as for ankle and knee flexion on the right were found (grades according to Medical Research Council). Ankle reflexes were absent bilaterally; patellar reflex was normal on the right and absent on the left. The surface skin temperature of the left foot was 34.6°C and of the right foot 28.5°C. He had an indwelling urinary catheter and was incontinent of feces. Nerve conduction studies revealed small compound muscle action potentials and slow motor nerve conduction velocities in the right and left tibial and peroneal nerves. No F-responses were recorded in the abductor hallucis and extensor digitorum brevis muscles bilaterally. No spontaneous or

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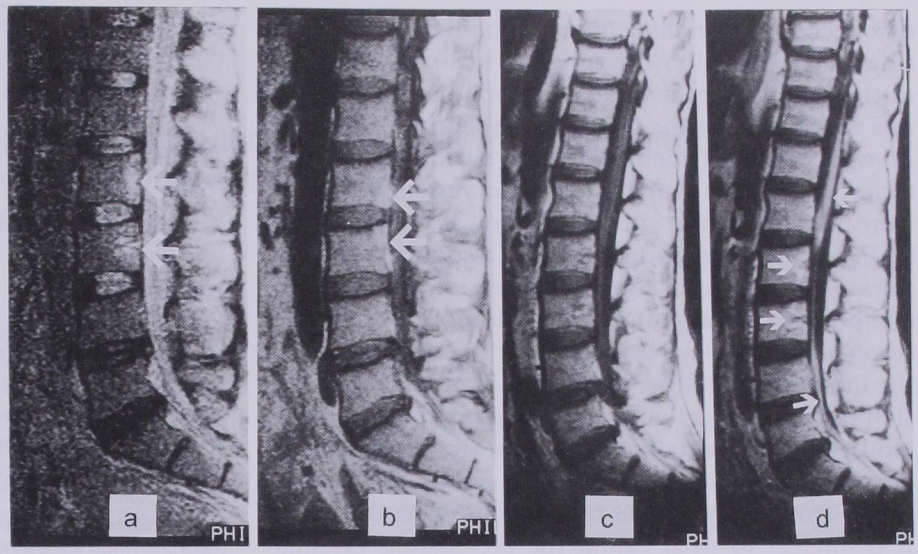

Fig. 1. Plain magnetic resonance imaging (MRI) scan of lumbar spine on 3rd day: (*A*) TR 1800 and TE 90 and (*B*) TR 560 and TE 30. Hyperintense signal in the dorsal third of the vertebral bodies of L2 and L3 in T2- and T1-weighted images consistent with infarction. On the 27th day, MRI without (*C*) TR 560 and TE 30 and with (*D*) TR 560 and TE 30 gadolinium-DTPA demonstrates gadolinium-enhancement of lumbosacral spinal cord and dura mater and less enhancement of vertebral bodies L2 and L3.

voluntary activity was recorded from paralyzed muscles. On the 14th day, compound muscle action potentials were more reduced and finally unobtainable on the 28 day, whereas orthodromic sensory nerve action potentials of the left medial plantar nerve remained well preserved. Needle electromyography revealed massive spontaneous activity in all paralyzed lower limb muscles as well as the left paraspinal musculature of L5 (multifidus).

On the 27th day, the T1-weighted image with gadolinium-DTPA showed an area of contrast enhancement in the lumbosacral spinal cord, adjacent meninges, and dorsal third of the vertebral bodies of L2 and L3 (fig. 1D), whereas in the noncontrast images, the hyperintense signal of the vertebral bodies of L2 and L3 had disappeared (fig. 1C). These findings are consistent with an ischemic infarction of spinal cord, dura mater, and the vertebral bodies of L2 and L3.[5]

Discussion

The occurrence of paraplegia after various surgical procedures performed during perioperative epidural analgesia or anesthesia was estimated to be 1:11,000.[2] In many cases, immediate postoperative paraparesis was attributed to a direct toxic effect of the local anesthetic or acute ischemia. A neurotoxic effect can be postulated if the highly concentrated anesthetic used for epidural analgesia has been injected inadvertently into the subarachnoid space. Ischemic lesions of intraspinal nervous tissue can be caused by a decrease in systemic BP, direct damage to radicular spinal arteries, or an increase in intraspinal pressure. The latter situation can occur after injection of large amounts of local anesthetic *via* an epidural catheter or with inferior vena cava (IVC) compression, *e.g.*, due to surgical packs and retractors, a pregnant womb, or a patient's hyperlordotic position.[1–4] Under these circumstances, the addition of spinal stenosis can facilitate the increase of intraspinal pressure.[1] Subacute paraparesis develops in cases of epidural hematoma or abscess.[2]

In the current case, a lesion of the cauda equina or the spinal cord, or both, was likely because paraplegia occurred with impairment of perianal sensation and incontinence. The increased skin temperature of the left leg could be explained by impaired sympathetic outflow after damage to the intermediolateral nucleus. Electrophysiologic testing proved an anterior horn or root lesion and, concerning the sensory pathways, a supraganglionic site of the lesion. It was only 4 weeks after onset and 2 weeks after clearly diagnostic electrophysiologic results that contrast-enhanced MRI showed the infarction of the spinal cord. However, in the initial MRI, the abnormal signal of the dorsal aspects of the vertebral bodies of L2 and L3 (fig. 1) could have been interpreted as ischemic infarction.[5]

Ischemic anterior spinal artery syndrome often has been diagnosed in paraparesis after various surgical procedures performed during epidural anesthesia, because of the dissociated sensory loss observed clinically.[6–8] Although this typical dissociation was not present in our case, MRI demonstrated an ischemic infarct of the spinal cord and of dura mater and the posterior parts of two vertebral bodies (fig. 1). The large extent of the infarcted area explained the mild cerebrospinal fluid lymphocytosis and increased protein concentration. Because the dura mater and the posterior vertebral bodies are supplied by branches of more than one radicular artery along their intraspinal courses,[5] an increase of intraspinal pressure resulting in a sealing-

off effect of the foramina intervertebralia must be considered etiologically. The vertebral infarctions located in the dorsal aspects of the vertebral bodies support this suggestion. In the three cases reported by Yuh *et al.* with lesions of abdominal aorta or of segmental arteries at their origin, the ischemic vertebral lesions were located in the anterior aspect of the vertebral bodies or near the endplate.[5] The assumed increased intraspinal pressure could have resulted from IVC compression because of the hyperlordotic supine position, as previously suggested for three other cases in the literature.[1,3,4] However, IVC or iliac vein compression due to surgical packs or retractors cannot be excluded. Experimentally, it has been shown that an increase of intraabdominal pressure can be transmitted to the epidural space *via* the IVC and its collaterals.[9] It is unclear to what extent the preexisting vascular risk factors contributed to the ischemic lesion. Local anesthetic was not given during the operation and only 10 mg morphine (10-ml volume) was instilled through the epidural catheter toward the end of the operation. Two hours later, 0.25% bupivacaine was started at a rate of 4 ml/h. Accidental injection of local anesthetic into the subarachnoid space was unlikely because the blood pressure had remained stable. More likely, epidural analgesia did not play any role for the development of spinal cord infarction in our case.

We conclude that a prolonged hyperlordotic supine position for surgery that could promote an IVC compression and aggravate a lumbar canal stenosis should be avoided when possible.

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References

1. Skouen JS, Wainapel SF, Willock MM: Paraplegia following epidural anesthesia: A case report and a literature review. Acta Neurol Scand 1985; 72:437–43

2. Usubiaga JE: Neurological complications following epidural anesthesia. Int Anesthesiol Clin 1975; 13:1–153

3. Bromage PR: "Paraplegia following epidural analgesia": A misnomer. Anaesthesia 1976; 31:947–9

4. Teague CA, Urquhart-Hay D: Spinal thrombophlebitis after prostatectomy with hypotensive anaesthesia: A case report. NZ Med J 1974; 80:654

5. Yuh WTC, Marsh EE, Wang AK, Russell JW, Chiang F, Koci TM, Ryals TJ: MR imaging of spinal cord and vertebral body infarction. AJNR Am J Neuroradiol 1992; 13:145–54

6. Gaudin P, Lefant D: Paraplégie après anesthésie péridurale pour chirurgie vasculaire. Ann Fr Anesth Reanim 1991; 10:468–71

7. Adriani J, Naragi M: Paraplegia associated with epidural anesthesia. South Med J 1986; 11:1350–5

8. Urquhart-Hay D: Paraplegia following epidural analgesia. Anaesthesia 1969; 24:461–70

9. Usubiaga JE, Wikinski JA, Usubiaga LE: Epidural pressure and its relation to spread of the anaesthetic solutions in epidural space. Anesth Analg 1967; 46:440–5