J Anesth (2001) 15:170–172

Journal of
Anesthesia

Spinal cord infarction in a patient undergoing left pyeloplasty in the right lateral kidney position

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Key words Spinal cord infarction · Lateral position · Postoperative complication

Introduction

Postoperative paralysis of the lower extremities is a rare but serious neurologic complication. This event has been reported to occur in a number of specific contexts: in association with the patient's position during operation, from intraoperative prolonged and severe hypotension, or as a complication of epidural anesthesia.

We describe a case of spinal cord ischemia with paralysis of the lower extremity that probably occurred as a complication of patient position for left pyeloplasty and hypotension combined with asymptomatic spinal canal stenosis.

Case presentation

A 60-year-old man with congenital ureteral stenosis underwent left pyeloplasty. Physical examination revealed a well-developed male. His height was 173 cm, and his weight was 67 kg. The medical history included hypertension, which was controlled by a calcium antagonist, and also electrocardiographically detected atrial fibrillation. The latter persisted to the time of surgery. Echocardiography revealed no thrombus in the left atrium. He had not complained of lumbago or any other neurological deficiency before surgery.

His blood pressure was 120/76 mm Hg, and his heart rate was 84 beats/min with irregular rhythm. He received premedication of 0.5 mg of atropine and 25 mg of hydroxyzine i.m. 30 min before the induction of anesthesia.

After the usual preparation and draping of the back, an 18-gauge Touhy needle was introduced into the Th12–L1 interspace by the median approach with the patient in the left lateral decubitus position. Loss of resistance to injection of normal saline was used to identify the epidural space. An epidural catheter was inserted without difficulty. No pain, bleeding, or paresthesia was associated with needle or catheter placement. No blood or other fluid was obtained from the catheter, either spontaneously or with aspiration. After a negative test dose of 2 ml of 2% lidocaine, the catheter was taped in place and the patient was turned to the supine position. An additional 2 ml of 2% lidocaine was injected via the catheter, followed by continuous infusion at 5 ml·h^{-1}.

Geneal anesthesia was induced with intravenous thiopental, and tracheal intubation was facilitated by vecuronium. Anesthesia was maintained with continuous administration of 0.5% sevoflurane and 60% nitrous oxide in oxygen in a semiclosed circuit with intermittent positive-pressure ventilation.

Throughout the operation, the patient was kept in the right lateral position, stretched over a pillow with flexion of the table and elevation of the kidney rest. After the patient was placed in this position, arterial blood pressure decreased to 76/38 mm Hg from a baseline of 140/100 mm Hg, but it rapidly returned to 110/70 mm Hg when 4 mg of intravenous ephedrine was administered. However, blood pressure remained unstable (between 80/58 mm Hg and 112/48 mm Hg) and systolic blood pressure continued at less than 100 mmHg in spite of fluid infusion and intermittent administration of ephedrine during the first 80 min of the operation. Then, a continuous infusion of dopamine was initiated at a rate of 2 to 3 μg·kg^{-1}·min^{-1} to maintain systolic blood pressure above 110 mmHg. The operation lasted for 5 h. The estimated total blood loss was 230 ml. The rectal tem-

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Received: June 29, 2000 / Accepted: February 20, 2001

perature was between 35° and 36°C during the operation. At the end of surgery, the trachea was extubated and continuous epidural infusion of 2% lidocaine was initiated for postoperative analgesia at a rate of 3 ml·h^{-1}. The patient did not complain of postoperative pain, and the upper level of sensory blockade was at approximately Th8 at the discontinuation of general anesthesia.

The following morning, the patient complained of lower extremity weakness and persistent numbness below Th10. Initially the complaint was thought to result from the pharmacologic action of lidocaine, but weakness of the right lower leg and numbness over both sides of the perineal area persisted 1 day after epidural analgesia was discontinued. For 6 days after the operation, these neurological symptoms and constipation persisted, and urinary retention became obvious when the urinary catheter was withdrawn and the patient required it again.

Neurologic evaluation revealed decrease in thermal and pain senses with intact tactile sense in the perineal area and dermatomes Th10–L3 on the right side and in Th10–11 on the left side. Deep tendon reflexes were normal. Babinski's reflex was negative on both sides. Neurological symptoms in this case showed both conus medullaris and epiconus syndromes, but magnetic resonance imaging of the lumbar spine revealed cord swelling in gadolinium-enhanced T2-weighted images, and conus medullaris infarction was diagnosed (Fig. 1). Examination by an orthopedist disclosed unrecognized lumber spinal canal stenosis. Urinary and fecal continence returned 17 days after the operation, and motor and sensory function returned gradually over the next 6 months.

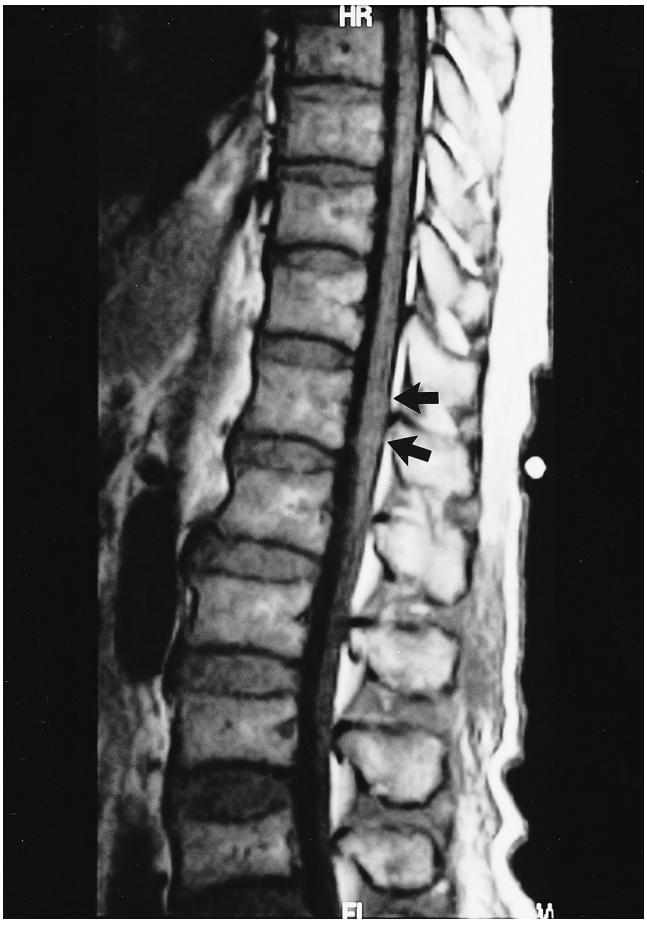

Fig. 1. Plain magnetic resonance imaging (MRI) scan of lumbar spine on 9th postoperative day. *Arrows* show swelling of spinal cord with hyperintense signal in Th1–2 and L1 level in T2-weighted image is consistent with infarction

Discussion

Spinal cord ischemia after operation is rare. Ischemic anterior spinal artery syndrome has been diagnosed in patients with paralysis showing loss of thermal and pain sense with intact tactile sense after various surgical procedures.

On the other hand, direct damage to the radicular spinal arteries, thromboembolism, severe hypotension, or increase in intraspinal pressure can cause spinal ischemia. Hyperpylexia may aggravate spinal ischemia. An increase in intraspinal pressure and severe hypotension can be caused by inferior vena cava (IVC) compression in some patient positions, surgical manipulation, or the use of retractors or packs. Spinal stenosis can facilitate the increase in intraspinal pressure, and it is a risk factor for neurological complication in patients inappropriately positioned under general anesthesia [1].

Amoiridis recently reported a case of spinal cord infarction that became apparent after surgery had been performed with the patients in a hyperlordotic position [2]. Compression of the IVC from such hyperlordosis increases intraspinal pressure [2,3]. For surgery on the left kidney, our patient was in the lateral position, while the table was flexed and the kidney rest was elevated to expose the flank optimally. This maneuver also causes partial obstruction of the IVC, as was reported for the hyperlordotic position, which is associated with a sudden fall in blood pressure and an increase in intraspinal pressure [4–6]. Initially unrecognized spinal canal stenosis in this patient may also have exacerbated the rise in intraspinal pressure caused by positioning while the patient was under general anesthesia [7]. Thus, spinal cord ischemia might have occurred as a result of intraoperative positioning that could cause IVC compression and hypotension, combined with spinal canal stenosis as a predisposing factor.

In epidural anesthesia, addition of epinephrine to local anesthetic solutions has been suggested to compromise blood flow to the spinal cord [7]. Further acci-

dental subdural or subarachnoid administration of high doses of lidocaine can cause neurotoxicity [8,9].

In our case, absence of spinal fluid return upon epidural catheter aspiration cannot in itself exclude subdural catheter placement, but successful epidural placement was confirmed by the absence of sensory blockade just after the test dose of 2 ml of 2% lidocaine was injected. Epinephrine was not used, and direct injury of the spinal cord or epidural hematoma was not diagnosed by clinical course and magnetic resonance imaging (MRI). Thus epidural anesthesia may not be responsible for the present neurological disorder.

The patient had atrial fibrillation, which is thought to be a risk factor for thromboembolism, and preoperative echography showed no thrombus. However, thromboembolism cannot be excluded as a cause of spinal ischemia in this case. Neither hyperpylexia nor direct injury to the spinal artery or aorta is thought to be the cause of this event.

A review by Kane of 36 cases of severe neurologic deficit following epidural block [7] included four patients with lower limb paralysis following prolonged intraoperative hypotension. However, all patients underwent epidural anesthesia with lidocaine solution including epinephrine, and in all cases systolic blood pressure fell below 100 mm Hg for a prolonged period during surgery. In the current case, hypotension persisted for about 80 min during epidural anesthesia with 2% lidocaine without epinephrine. But hypotension itself is not independently responsible for compromised blood flow to the spinal cord, considering that epidural anesthesia frequently is employed to produce controlled hypotension during surgery without inducing any neurologic complications [10].

We conclude that a prolonged lateral position with kidney rest for surgery that could promote IVC compression and hypotension might be avoided in patients with spinal canal stenosis.

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