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## CLINICAL INFORMATION

# Sudden motor and sensorial loss due to retroperitoneal hematoma during postoperative periods: a case report<sup>☆</sup>



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### KEYWORDS

Epidural anesthesia;  
Flaccid paralysis;  
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hematoma

**Abstract** A 68 year-old male patient was hospitalized for radical prostatectomy. He had no abnormal medical history including neurological deficit before the operation. Prior to general anesthesia, an epidural catheter was inserted in the L3–4 interspace for intraoperative and postoperative analgesia. After surgery for nine hours, he developed confusion and flaccid paralysis of bilateral lower extremities occurred. No pathology was detected from cranial computed tomography and diffusion magnetic resonance imaging no pathology was detected. His thoracic/lumbar magnetic resonance imaging. Intraabdominal pressure was shown to be 25 mmHg, and abdominal ultrasonography revealed progression in the inflammation/edema/hematoma in the perirenal region. The Bromage score was back to 1 in the right foot on the 24th hour and in the left foot on the 26th hour. Paraplegia developed in patients after epidural infusion might be caused by potentiated local anesthetic effect due to retroperitoneal hematoma and/or elevated intra-abdominal pressure.

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### PALAVRAS-CHAVE

Anestesia peridural;  
Paralisia flácida;  
Hematoma  
retroperitoneal

### Súbita perda sensitiva e motora devido a hematoma retroperitoneal durante os períodos de pós-operatório: relato de caso

**Resumo** Paciente do sexo masculino, 68 anos de idade, hospitalizado para prostatectomia radical. O paciente não tinha história médica anormal, inclusive nem déficit neurológico antes da operação. Antes da anestesia geral, um cateter peridural foi inserido no espaço intermédio L3-4 para analgesia no intra e pós-operatório. Após a cirurgia que durou nove

<sup>☆</sup> The study was conducted in the Department of Anesthesiology, Baskent University Istanbul Training and Medical Research Center, Istanbul, Turkey. The study was presented in the Turkish Society of Anesthesiology and Reanimation 48th National Congress held on October 25–29, 2014. Patient Identification: Patient consent was obtained from the patient.

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horas, o paciente desenvolveu confusão e paralisia flácida bilateral dos membros inferiores. Tomografia computadorizada de crânio e imagem de difusão por ressonância magnética não detectaram lesão. Os achados nas imagens de ressonância magnética torácica/lombar eram normais. A pressão intra-abdominal era de 25 mmHg e o ultrassom abdominal revelou progressão de inflamação/edema/hematoma na região perirrenal. O escore de Bromage voltou a 1 no pé direito na 24<sup>a</sup> hora e no pé esquerdo na 26<sup>a</sup> hora. A paraplegia desenvolvida nos pacientes após a infusão epidural pode ter sido causada por um efeito potencializado do anestésico local devido a hematoma retroperitoneal e/ou pressão intra-abdominal elevada.

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## Introduction

The rate of neurological complications following regional anesthesia is 4/10,000.<sup>1</sup> Neurological complications result from factors such as direct nerve injury related to needle or catheter placement, drug-related neurotoxicity, local anesthetic injection into the nerve, neural ischemia.<sup>2-5</sup> Different from the common causes of neurological complications, we herein presented development of paraplegia due to retroperitoneal hematoma in a patient who underwent surgery for prostate cancer.

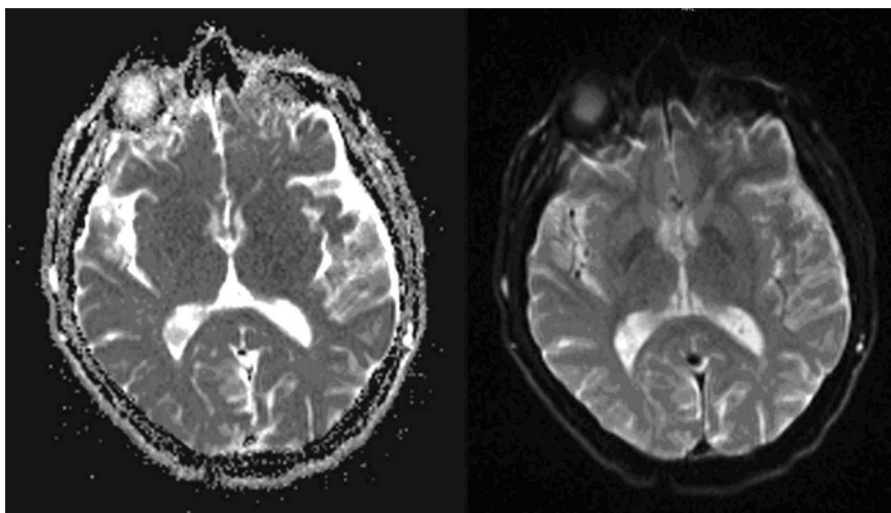
## Case report

A 68 year-old male patient was hospitalized for radical prostatectomy with the pre-diagnosis of prostate cancer, which was considered based on the transurethral resection biopsy performed due to asymptomatic prostate-specific antigen elevation. He underwent surgery with the American Society of Anesthesiologists (ASA) risk score of 1. Acetylsalicylic acid (150 mg.day<sup>-1</sup>), which the patient has been receiving, was discontinued 7 days before surgery. He was monitored at the operating room in accordance with ASA standards; an intravenous route was opened and fluid replacement was started. After cutaneous and subcutaneous infiltration of local anesthesia performed into the L3-L4 intervertebral space using 2 mL of 2% lidocaine solution with the patient in the sitting position, an 18-G epidural catheter was inserted by loss-of-resistance method in one sitting. After negative aspiration and test dose, 7 mL of 0.5% bupivacaine was administered through the epidural catheter. Fifteen minutes later, general anesthesia was performed in the patient, in whom the level of sensory blockade was at T8. The patient, of whom pre-induction blood pressure was 130×70 mmHg, pulse rate was 84 beats/min, and peripheral oxygen saturation (SpO<sub>2</sub>) was 100%, was preoxygenated and general anesthesia induction was performed with 200 mg propofol and 40 mg rocuronium. The anesthesia was maintained with 0.8–0.6% isoflurane in oxygen-air mixture (50/50%). Ninety minutes after epidural bolus dose, 0.25% bupivacaine was administered through the epidural catheter at an infusion rate of 5 mL.h<sup>-1</sup>. During the intraoperative period, 2 units of erythrocyte suspension, 500 mL of colloid, and 4500 mL crystalloid were

administered. The surgical procedure lasted for a total of 3 h. A total of 37.5 mg bupivacaine infusion was performed. The patient woke up without any problem at the end of surgery. Total amounts of urine and blood loss over the course of surgery were 800 mL and 700 mL, respectively. He was kept in the postoperative recovery room for an hour. During transfer to the clinic, his hemoglobin level was 10 g.dL<sup>-1</sup> and hematocrit level was 28.8%. In the postoperative monitoring room, the patient had a blood pressure of 123×60 mmHg, SpO<sub>2</sub> of 99%, pulse rate of 56 beats/min, Visual Analogue Scale (VAS) score of 0, and Aldrete score of 10. His motor examination was normal (Bromage score 1).

After the surgery, the patient was monitored in the clinic using the patient-controlled analgesia form. Postoperative analgesia was performed with 0.1% bupivacaine administered through the epidural catheter at a rate of 5 mL.h<sup>-1</sup>. After surgery for 2nd hour, when his VAS score was 6, the infusion rate was increased to 8 mL.h<sup>-1</sup>. The blood pressure was about 100×60 mmHg and the pulse rate was about 80 beats/min. On the postoperative 6th hour, the infusion rate was decreased to 3 mL.h<sup>-1</sup> since his motor examination was normal (Bromage score 1) and VAS score was 0.

After surgery for 9th hour, an emergency code was called since he developed confusion and motor loss. When the emergency team arrived, the physical examination of the patient revealed confusion, agitation, and meaningless speech. His blood pressure was 78×40 mmHg. However, the Bromage score of the patient was observed to be 4. The epidural infusion was immediately stopped, and after obtaining blood sample for complete blood count, the patient underwent cranial computed tomography and diffusion magnetic resonance imaging (MRI) with the pre-diagnosis of intracranial pathology and/or epidural hematoma-ischemia; however, no pathology was detected (Fig. 1). On the physical examination of the patient, who was transferred to the intensive care unit after surgery for 15th hour, the abdomen was rigid and the Bromage score was 4 for his feet. The findings of thoracic/lumbar MRI of the patient, of whom neurological examination revealed no improvement on the postoperative 15th hour, were reported to be normal (Fig. 2). Intraabdominal pressure (IAP) was measured via a urinary catheter and was shown to be 25 mmHg in the intensive care unit. Since the result of complete blood count revealed a hematocrit concentration of 25%, the



**Figure 1** Cranial computed tomography and diffusion magnetic resonance imaging.



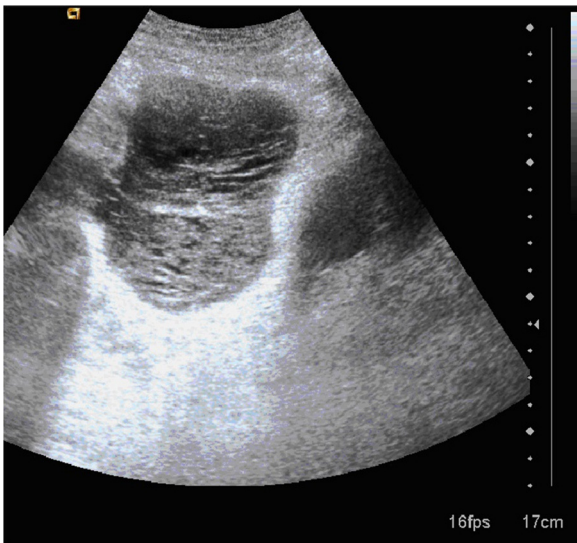
**Figure 2** Thoracic/lumbar magnetic resonance imaging.

patient underwent emergency ultrasonography (US). The abdominal US revealed increased density that might reflect massive inflammation, edema or bleeding in bilateral perirenal regions in the retroperitoneum and hematoma beneath the bladder (Fig. 3). The Bromage score was return to 1 in the right foot on the 24th hour and in the left foot on the 26th hour. Abdominal distension of the patient increased and no gas-stool discharge was observed on the postoperative 3rd day. Creatinine level increased to  $2.3 \text{ mg.dL}^{-1}$  from  $0.9 \text{ mg.dL}^{-1}$  and blood urea nitrogen (BUN) level increased to  $70 \text{ mg.dL}^{-1}$  from  $23 \text{ mg.dL}^{-1}$ . Plane abdominal X-ray performed in the standing position on the postoperative 4th day was normal. Abdominal USG revealed progression in the inflammation/edema/hematoma in the perirenal region. Treatment was started for pyelonephritis; antibiotherapy was arranged. His body temperature did not decrease on the postoperative 6th day monitoring and elevation of BUN-creatinine concentrations persisted incrementally.

Renal scintigraphy performed on the postoperative 7th day revealed bilateral leakage from both renal calyces. Bilateral nephrostomy tubes were placed by the Department of Interventional Radiology and hematoma beneath the bladder was aspirated. On the postoperative 8th day, BUN/creatinine concentrations decreased to  $15/1.3 \text{ mg.dL}^{-1}$ . As the contrast substance passage disappeared during the follow-ups, nephrostomy tubes were removed. The patient was discharged from the hospital on the postoperative 14th day.

## Discussion

In the present case, pelvic compartment and/or increased intrapelvic pressure due to hematoma in the retroperitoneum and hypotension might led to decrease in perfusion pressure in epidural vessels and prolongation in the regression of epidural blockade. In the present case, we primarily



**Figure 3** The abdominal USG.

thought likely development of epidural hematoma at the time of event or, secondly, of drug toxicity; because, clinically, both sudden motor loss and alteration in consciousness developed concurrently. Postoperative paraplegia may result from spinal hematoma, intracord injection, drug toxicity, or compression or impaired perfusion due to epidural air.<sup>6–9</sup> In addition, surgery-related decrease in spinal cord perfusion,<sup>10,11</sup> preexisting spinal metastasis,<sup>7,12</sup> thoracic disc herniation,<sup>13</sup> and temporary anterior spinal artery syndrome<sup>14</sup> may also cause paraplegia. In the present case, since the diagnosis could not be established via cranial computed tomography performed for early diagnosis, we eliminated the pre-diagnosis of epidural vascular compression/ischemia depending on the thoracic and lumbar MRI, which were performed to rule out ischemia and hematoma.

We then considered systemic toxicity due to drug toxicity. Systemic toxicity of local anesthetics was unlikely to cause paraplegia because epidural infusion was decreased at least 10 h before paraplegia was found. Furthermore, the central nerve symptoms such as confusion were caused by hypotension due to postoperative hemorrhage.

The diagnosis was established after intraabdominal pressure was measured 25 mmHg via a urinary catheter and ultrasound revealed retroperitoneum hematoma. We think that some of pregnancy evidence supporting the mechanism. Increased intraabdominal pressure during pregnancy increases blood volume in the epidural venous plexus by compressing the inferior vena cava, decreases cerebrospinal fluid volume, decreases epidural volume, increases epidural pressure, and thereby decreases the need for epidural dose by 30%.<sup>15</sup> We thought that retroperitoneal hematoma occurred in the present case after radical prostatectomy caused sensorial/motor blockade by increasing the intraabdominal pressure and by increasing blood volume in

the epidural venous plexus, decreasing epidural volume, increasing epidural pressure, and thereby decreasing the need for epidural dose.

In the present case report, it was demonstrated that paraplegia developed in patients after epidural infusion might be caused by potentiated local anesthetic effect due to retroperitoneal hematoma and/or elevated intra-abdominal pressure. And early intervention prevents development of irreversible neurological damage.

## Conflicts interest

The authors declare no conflicts of interest.

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