

## Paraplegia after combined general and epidural anesthesia : a case report

G. CEKIC (\*, \*\*), M. DE KOCK (\*), Y. KREMER (\*) and J.-L. SCHOLTES (\*)

**Abstract :** Neurological complications after epidural anesthesia performed for abdominal surgery are uncommon, but of important consequence with significant morbidity. Paraplegia is very rare and may be a result of multiple factors. We report a case of elective colectomy under combined general and epidural anesthesia for a carcinoma. An epidural infusion was used for intra-operative and post-operative analgesia. During induction of anesthesia, the patient was asystolic for a few seconds and during the first postoperative day, a hypotensive episode was registered. He then developed a sensory-motor deficit in the legs. A spinal cord infarction at the level of T10 extending to T2 was diagnosed with magnetic resonance imaging. The association of hypotension as a cause of spinal cord infarction is discussed. The factors that may have contributed to paraplegia and preventive neuroprotective strategies are reviewed.

**Key words :** Epidural anesthesia ; complication ; spinal cord infarct ; post-operative paraplegia.

### INTRODUCTION

Paraplegia as a consequence of epidural anesthesia is very rare and may be the result of multiple factors, including epidural-intradural-subdural abscess, spinal hematoma, toxic injection, trauma from the needle and spinal cord ischemia (1). Various etiologies may compromise the spinal cord perfusion but two principal pathophysiological mechanisms are implicated: prolonged hypotension or arterial insufficiency and mechanical factors (2). The present report describes a case of postoperative paraplegia after colectomy under combined general and epidural anesthesia. The differential diagnosis, possible etiological factors and association with epidural anesthesia are discussed.

### CASE REPORT

A 72-year-old male with four days of primarily right-sided abdominal pain, nausea and anorexia presented to the Emergency Department. His

medical history was significant for dyslipidemia and paroxysmic atrial fibrillation for which he didn't take any anticoagulant therapy and his only medication was a cholesterol lowering drug. The patient did not report any problems with defecation or gastrointestinal hemorrhage. Examination of the abdomen revealed no mass or tenderness. A thoracic-abdominal scan demonstrated a mass in the right colon without further suspect lesions. A total colonoscopy performed in the surgical department showed an obstructing tumor in the ascending colon. Biopsies taken at his level showed a well-differentiated colonic adenocarcinoma occurring in a previously existing villous adenoma. An elective right hemicolectomy with an end-to-end ileo-transverse anastomosis was programmed.

The preoperative evaluation showed normal blood biochemistry and ECG. An echocardiographic exam performed one year earlier showed a preserved left ventricular function. The carotid bruits were normal. Mallampati classification depicted a Class 4 status, suggesting a difficult intubation. The patient underwent a bowel preparation with an oral Polyethylene Glycol-Electrolyte solution and was allowed to drink water until 2 hours before the intervention.

In the operating room, a 500 ml cristalloid infusion was initiated and an epidural puncture was performed at the T5-T6 interspace using a 18-gauge

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Tuohy needle. An epidural catheter was easily inserted, and, after an uneventful epidural test dose of 3 ml lidocaine with epinephrine 1/200000, induction of general anesthesia was initiated with sufentanil 0,3 µg/kg and propofol 1.5 mg/kg. As soon as manual mask ventilation was ascertained, muscle relaxation was achieved with atracurium 0,5 mg/kg. Before laryngoscopy, i.v. lidocaine 60 mg and an additional dose of propofol 50 mg was administered.

On laryngoscopy, the Cormack and Lehane score was 3. Following a rather difficult intubation, the ECG showed that the patient was asystolic. Cardiopulmonary resuscitation was started by external cardiac compressions, manual ventilation through the endotracheal tube, and 0.5 mg of intravenous atropine. Within 1 minute, the heart resumed a normal sinus rhythm.

At the start of surgery a bolus dose of Bupivacaine 0,5% 5 ml with Sufentanil 5 µg was injected through the epidural catheter and a continuous infusion of Bupivacaine 0.125% with Sufentanil 0.2 µg/ml was started at a rate of 7 ml/h.

Surgery was uneventful and lasted 2 hours 42 minutes at the end of which, the patient was extubated. Hemodynamics during the procedure were stable with a mean blood pressure superior to 70 mmHg. Total fluid infusion during surgery consisted of 2000 ml of crystalloids and 500 ml of colloids. Estimated blood loss was 250 ml and urine output was 300 ml. In the post-anesthesia care unit a PCEA device was started with a continuous infusion rate of 5 ml/h of 0.0625% Bupivacaine with 0.1 µg/ml Sufentanil with possibility of a 5 ml bolus administration every 50 minutes. Motor function of the legs was preserved and the upper sensory level of the epidural was at T8. During the postoperative period hemodynamic and neurological parameters remained stable, the patient received an additional amount of 1500 ml crystalloid until oral fluid intake was allowed i.e. 24 hours later.

In the morning of the first post-operative day, the patient was mobilised with the physiotherapist and complained of abdominal pain. After verification of the puncturing site of the epidural catheter, the level of sensory block, and a test dose of 3 ml lidocaine with epinephrine, a bolus dose of 5 ml of bupivacaine 0,25% was injected through the epidural catheter. The patient then presented an orthostatic hypotensive episode, and became confused. The lowest systolic blood pressure recorded by the nurse was 50 mmHg. Immediate return to the supine position improved the clinical condition : the patient's mental status returned to normal and the

blood pressure progressively reached values superior to 100/70 mmHg with the help of a 500 ml colloid infusion and ephedrine. An ECG recording performed at that time was normal. Neurological examination showed a decrease of sensation and motility in the left and right lower limbs. The measured upper sensory level at that time was at the T6 dermatome. Hypotension was suspected to be caused by a high level of the epidural anesthesia., which was subsequently discontinued. Six hours later the patient complained of numbness and paralysis of both lower extremities. The epidural catheter was removed after having checked that the last dose of LMWH was given more than 8 hours before. A new neurological evaluation confirmed a sensory deficit reaching up to the T6 level and motor deficit of lower limbs, however with preservation of a minimal sensation in the legs. Emergency magnetic resonance imaging of the thoracic and lumbar spine showed a T2-weighted hypersignal restricted to the border of the anterior sulcus, starting at T2 and extending to T10, almost exclusively restricted to the grey substance. There was no evidence of spinal hematoma or abscess. The lesion was diagnosed as a central cord infarction (Figs. 1 and 2).

A high-dose methylprednisolone protocol was prescribed as a bolus intravenous infusion of 30 mg per kilogram of body weight over fifteen minutes followed 45 minutes later by an infusion of 5.4 mg per kilogram of body weight per hour for 23 hours. Unfortunately this did not result in a change of the neurological state. Other complications in the post-operative period were : renal insufficiency, pneumonia and a few days stay in intensive care unit for hemofiltration.

On the tenth post-operative day the patient was transferred to a spinal cord rehabilitation ward. Functional outcome after 12 months is unimproved.

## DISCUSSION

Neurological complications following epidural anesthesia are uncommon, with an incidence estimated to be between 1/1000 and 1/1000000 but some of them have catastrophic consequences (3). These include principally traumatic lesions of spinal cord or nerve root, infectious lesions like meningitis or epidural-subdural abscess (0.04 to 0.07%), spinal hematoma (0.04 to 0.05%) and ischemic lesions (4, 5).

The spinal cord is supplied by three arteries that course vertically over its surface : a single anterior spinal artery and paired posterior spinal

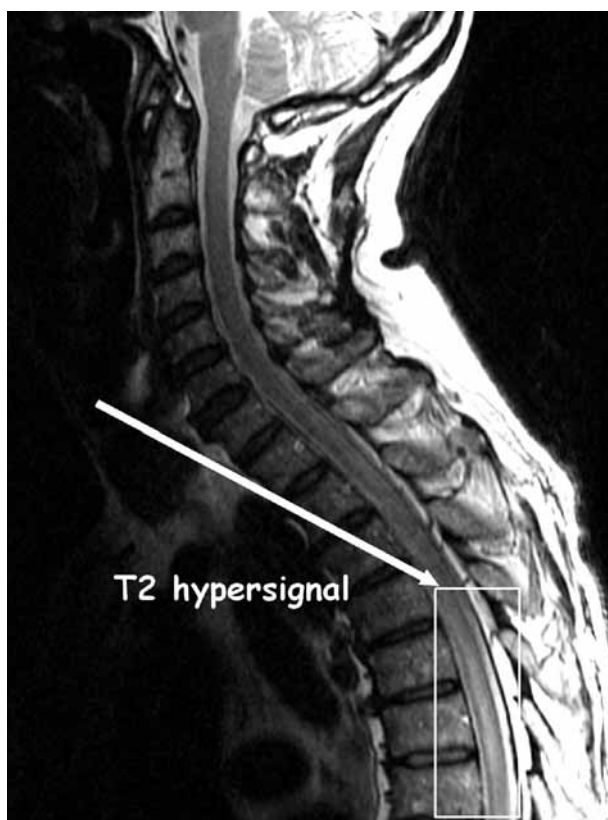


Fig. 1. — Magnetic resonance image (sagittal fast spin echo T2-weighted image) of the thoracic spine showing abnormal signal in the thoracic cord starting approximately at T2 extending to T10, consistent with cord infarct. There is no evidence of spinal/epidural haematoma.

arteries. At each segment, paired penetrating vessels arise from the anterior spinal artery to supply the anterior two-thirds of the spinal cord; the two posterior spinal arteries, which often become less distinct below the midthoracic level, supply the posterior columns. The spinal arteries receive additional blood flow from the intercostal arteries in the thorax and the lumbar arteries in the abdomen. Likewise, at the lower thoracic level, most frequently between T9 and T12, a large radicular anterior medullary artery (Adamkiewicz artery), supplies the anterior spinal artery with some redundancy. Spinal cord ischemia can occur at any level; however, the presence of the artery of Adamkiewicz creates a watershed of marginal blood flow in the upper-thoracic segments (1, 2).

Thus, anterior spinal artery syndrome usually results from interrupted flow in one of its feeders. Causes are multiple. The most frequent include trauma, dissecting aortic aneurysm, aortography, vasculitis related to collagen vascular disease, thoracic surgery, prolonged aortic cross-clamping and a hypotensive crisis. But in all of the cases two

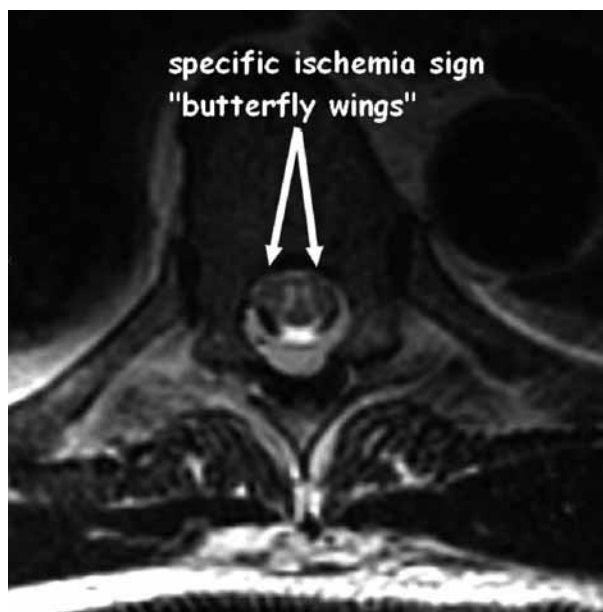


Fig. 2. — Magnetic resonance image (axial cross-sectional view T2-weighted image) at the level of T10 showing the specific spinal ischemia sign "butterfly wings".

principal mechanisms can be distinguished: (1) prolonged hypotension or arterial insufficiency and (2) mechanical factors (2).

Etiological factors that lead to spinal cord ischemia after neuraxial blockade are iatrogenic lesion of the radicular arteries, spinal cord compression by mechanical factors (abscess, hematoma) (6), and arterial insufficiency mechanism. Nevertheless a direct causal relationship of the spinal cord infarction with only epidural anesthesia is unlikely.

The effects of spinal injection on the vertebro-medullary vascularisation, leading to acute spinal cord deficit, have been initially attributed to the local anesthetic itself, e.g. a continuous infusion of bupivacaine with epinephrine, the arterial insufficiency mechanism being secondary to hypotension in addition to the vasoconstriction caused by epinephrine (7).

But many studies indicate a greater vulnerability of spinal neurons to ischemic insult from either cardiac arrest or a severe sustained episode of hypotension rather than to the vasoconstrictors in the local anesthetic solution. In these conditions, development of ischemic necrosis in spinal cord has been well documented in clinical studies (8). Moreover LEE *et al.* assert that a permanent spinal cord damage can be caused as from 2 minutes of spinal artery ischemia (9).

In our case, immediate MRI exam allowed to exclude a compressive phenomenon and to diagnose a central cord infarction. The spinal artery

ischemia has been caused by decreased spinal artery perfusion pressure secondary to severe hypotension likely due to the association of epidural and general anesthesia in a patient having probably arteritic lesions (old age, hypercholesterolemia) and relative hypovolemia due to intestinal preparation and underestimation of perioperative fluid loss. Severe systemic hypotension as a consequence of perioperative cardiac arrest in addition to postoperative hypotension secondary to lower circulatory debit due to the epidural analgesic infusion have all might contribute to the negative outcome.

## CONCLUSION

Although the mechanisms of paraplegia were not totally elucidated in our patient, we should keep in mind that peri-operative prolonged hypotension can produce a reduction of spinal cord blood flow, especially in the presence of cardiovascular comorbidities. Spinal cord infarction did not appear to be related exclusively to central neuraxial anesthesia. Therefore, prophylactic measures should be taken to prevent this devastating complication.

Complete cardiovascular assessment should be realised preoperatively in abdominal surgery, with risk of hypovolemia, in a patient with cardiac comorbidities. Hypovolemia and hypotension in the postoperative period should be aggressively treated. Postoperative neurological evaluation should be systematically realised and ischemia of spinal cord suspected whenever a new neurological sign appears especially after sustained post-operative hypotension.

If a central neuraxial blockade is chosen for postoperative analgesia, blood pressure should be controlled and fluid and vasopressors should be ready for administration before epidural reloading.

Some neuroprotective strategies have already been developed in some surgeries like aortic

aneurism repair, considering the vulnerability of the spinal cord to ischemic insult, e.g. using evoked potential technique, regional lumbar epidural cooling and spinal cord drainage to improve cord perfusion pressure (10). There is a lack of evidence whether these measures would be helpful for other surgical types in patients with comorbidities or at risk for ischemic insults.

Considering the devastating consequence of this complication, it is necessary to further develop standard guidelines for the treatment of spinal cord ischemia.

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