Hyperlordosis as a possible factor in the development of spinal cord infarction

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A patient developed persistent symptoms and signs suggestive of partial spinal cord infarction after an operation involving the use of the hyperlordotic position. This position involves extension at the waist, such that both the head and feet are below the level of the waist. It is employed to increase surgical access to the abdomen. Where this position is adopted for a prolonged surgical procedure, existing risk factors for spinal cord ischaemia should urge caution in the use of epidural analgesia.

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CASE REPORTS

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A patient developed persistent symptoms and signs suggestive of partial spinal cord infarction after an operation involving the use of the hyperlordotic position. This position involves extension at the waist, such that both the head and feet are below the level of the waist. It is employed to increase surgical access to the abdomen. Where this position is adopted for a prolonged surgical procedure, existing risk factors for spinal cord ischaemia should urge caution in the use of epidural analgesia.

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We report a case of spinal cord infarction occurring after a prolonged sub-total pancreatectomy where the patient was in the hyperlordotic position. The vulnerability of the spinal cord’s blood supply, whilst not generally an issue for this procedure, may become so where the patient remains in this position for prolonged periods. The causative mechanism in this case is uncertain but potential risk factors associated with the development of this uncommon and potentially devastating complication are discussed with reference to the literature. Such factors should caution against the use of epidural analgesia for procedures where the hyperlordotic position is to be employed.

Case report

A 43-year-old man with a history of alcohol-induced chronic pancreatitis associated with insulin-dependent diabetes presented with chronic abdominal pain. A difference in the skin temperature over the anterior surface of his trunk corresponding to the 6th to 11th thoracic dermatomes was noted on examination. This was thought to be a sympathetic component of his chronic abdominal pain. The pain was being treated with paracetamol, tramadol, dihydrocodeine and dothiepin. He had a BMI of 24. Investigations showed a normal full blood count (haemoglobin 15.1 g dl⁻¹), blood clotting, urea and electrolytes, liver function tests, ECG and chest radiograph. The glycosylated haemoglobin (HbA1c) was 10.1%.

The patient was admitted to hospital and with him awake, a thoracic epidural was placed at the level of the 5th thoracic intervertebral space for control of his chronic pain using an infusion of bupivacaine 0.1% 4 ml h⁻¹, with patient-controlled boluses of 4 ml no more often than every 20 min. Apart from a sensory block extending above the 4th thoracic dermatome on one occasion and better analgesia on the left side, this period was uneventful.

The patient was scheduled for a sub-total pancreatectomy. Anaesthesia was induced 5 days later using thiopental, vecuronium and phenoperidine, with enflurane in oxygen-enriched air for maintaining anaesthesia. The previously sited epidural remained, having provided satisfactory pain relief since insertion.

The operation lasted 7 h, for 6 h of which the patient was in the hyperlordotic position, with the head and legs extended below the level of the waist by 25–30° from the horizontal. The intraoperative period was uneventful, with good cardiovascular control. The preoperative arterial pressure was 110/80 mm Hg, which was maintained throughout apart from two 5-min intervals where it fell to a systolic pressure of 90 mm Hg. An epidural infusion of bupivacaine 0.1% with diamorphine 40 μg ml⁻¹ was run throughout at 4 ml h⁻¹. Vasoconstrictors were not used.
Measured blood loss was 1900 ml, and there was a good urine output. I.V. fluid replacement was with a combination of crystalloid and colloid. Blood sugar control was maintained with a potassium/insulin/glucose infusion at 11–13 mmol litre\(^{-1}\) apart from a brief period early on when it reached 21 mmol litre\(^{-1}\).

After surgery, the patient’s trachea was extubated and he was transferred to the intensive care unit (ICU) where after 2 h his arterial pressure fell to 75/30 mm Hg for 5 min, which was treated with 400 ml human albumin solution 4.5%. His haemoglobin concentration was subsequently 6.8 g dl\(^{-1}\) and clotting was deranged, with a prothrombin time of 4.5%. His haemoglobin concentration was subsequently 6.8 g dl\(^{-1}\) and clotting was deranged, with a prothrombin time of 27 s and kaolin cephalin clotting time of 55 s. There was no evidence of bleeding and the haemoglobin recovered to 11.8 g dl\(^{-1}\) after transfusion of three units of blood. He was able to move his toes and upper limbs well but a persistently high neural block at T6 bilaterally and unsatisfactory analgesia required re-siting of his epidural at T8/9, awake, on the ICU, which was uneventful. This provided good pain relief for 18 h. However, before the patient was discharged from the ITU the next day, the epidural catheter had to be removed as the patient complained of a burning pain in his back and left leg after each epidural bolus, despite the catheter having been withdrawn slightly. Boluses consisted of one 10 ml bolus of bupivicaine 0.25% administered by a doctor, as well as patient-controlled boluses of 4 ml.

Over the ensuing days the patient complained of persistent numbness of his left buttock and discomfort in his left groin. On examination there was no groin pathology, power in both legs was normal and he was fully mobile although after discharge from hospital he subsequently complained of weakness at this time. He had impaired temperature sensation posteriorly from T8 to L2, and inconsistent alterations anteriorly.

Because of the persistent nature of the sensory loss and dysaesthesia, the patient had magnetic resonance imaging (MRI) scans on the fifth and seventh days after surgery. The images on the fifth day were degraded by patient motion, but did show swelling and an abnormal signal within the anterior spinal cord from T3/4 to T9/10. There was enhancement after i.v. gadolinium, suggesting an acute rather than chronic pathological process. This was confirmed on the second scan 2 days later. No fluid collection was seen in the epidural space and there were no features to suggest haematoma or abscess formation within the cord or epidural space. The findings suggested infarction/ischaeemia of the cord.

Two weeks later the patient was examined by a consultant neurologist after he had complained of numbness in his left leg and buttock and over the posterior part of his chest. He also described pain in his left groin that radiated to his left testicle. He was mobile and had no bladder or bowel symptoms or motor deficit. He had brisk leg reflexes, left more than right, and three beats of clonus from each ankle. He had a reduced sensation in his left leg, which extended up to the low thoracic region. The neurologist felt the symptoms were those of an anterior spinal cord infarction. No treatment was believed to be of benefit and none was given.

Five months later the patient was seen by a second neurologist, who found altered sensation to the level of T6, predominantly on the left-hand side but with some new spread across the midline, especially over the lower abdomen. Motor function was normal, with no wasting, and normal power. Reflexes were brisk with extensor plantar responses, and there were no longer any unpleasant sensations. The examination was otherwise unchanged since the patient’s discharge from hospital, and the conclusion was the same.

**Discussion**

We report a case of partial infarction of the spinal cord occurring in a patient who spent 6 h in the hyperlordotic position during an operation for sub-total pancreatectomy. This resulted in the development of numbness extending up to the 6th thoracic dermatome, including the left buttock and leg, and initial dysaesthesia of the left groin and testicle. After 5 months the only new neurological signs remaining were abnormal sensation up to T6, including the left buttock and leg, with some spread across the midline. Before surgery, differences in skin temperature had been detected between T6 and T11 on both sides of the abdomen. The subsequent MRI findings indicated an acute ischaemic event in the anterior spinal cord, which resulted in a permanent neurological deficit consistent with infarction of the spinal cord.

Although the diagnosis of spinal cord infarction is primarily clinical, early MRI scanning is necessary where an epidural is in use to rule out the presence of a haematoma or abscess. However, the association between epidural anaesthesia and spinal cord infarction in the absence of an epidural haematoma is unproven. In 1999, Mayall and Calder\(^1\) described paraplegia resulting from spinal cord puncture with extensive haematoma formation, and Bromage and Benumof\(^2\) described paraplegia after possible intracord injection of air. Both cases had readily attributable mechanisms to account for the subsequent neurological deficits described and were associated with repeated attempts at insertion. Neither of these factors was associated with the neurological injury in our case where both epidural insertions were performed on the first attempt, with the patient awake.

The dysaesthesia occurring during epidural boluses in our patient may be explained by the preservation of sensory modalities carried in the dorsal columns, with abolition of pain and temperature sensation carried in the lateral spinothalamic tracts.\(^3\) Misinterpretation of residual posterior column sensory input in the setting of spinothalamic tract dysfunction may serve as a mechanism for the genesis of dysaesthesias within the CNS.\(^3\) The anterior spinal artery also supplies the lateral horn cells of the sympathetic...
nervous system and the anterior horn cells. The absence of motor signs in our patient would indicate preservation of supply to the anterior horn, indicating ischaemia in the region of the radicular branches of the anterior supply. The lower thoracic and lumbar regions rely on a radicular arterial supply, including the artery of Adamkiewics, vulnerable because of its acute angle of entry into the cord. In a review of 44 cases of spinal cord infarction, the mean level of sensory deficit was at T8. The testicular dysesthesia in our patient indicates the T8 dermatome, where the ‘break’ in the operating table was positioned. The mid-thoracic region is another area of vulnerability where the vascular supply is reduced, albeit offset by a smaller cross-sectional area, lower grey to white matter ratio and hence reduced metabolic demands. It is significant that there are no functioning communications between the anterior and posterior circulations of the spinal cord.

Skouen and colleagues described a persistent motor deficit that became evident on the day after surgery. The contribution of an increase in pressure in the inferior vena cava (IVC) as a consequence of prolonged hyperlordosis was considered to be a contributory factor. Intraoperative hypotension, in this case compounded by epidural anaesthesia, was thought significant although of brief duration. Intraoperative hypotension was not a significant factor in our case, although a similar episode to that in Skouen and colleagues’ patient occurred in our patient in the ITU after surgery.

Hypotension sufficient to reduce flow in hyperlordosis may not have to be extreme. In animal experiments, spinal cord blood flow diminished at a mean arterial pressure of 50 mm Hg, being unmeasurable at 25 mm Hg. Sclerosis of the spinal artery is more likely in a diabetic, as in our case, and would contribute to the vulnerability of flow in such a vessel. The HbA1c of 10.1% before surgery indicates poor diabetic control and the editorial by Renck remarks on the significance of diabetes in reports of the development of this complication. Any increase in cerebrospinal fluid pressure by reducing the pressure gradient between the aorta and the spinal vessels would compromise flow to the spinal cord. An increase in venous pressure transmitted to cerebrospinal fluid by compression of the IVC and mechanical kinking resulting from hyperlordosis would augment this factor, especially where epidural compliance is reduced. Amoiirdis and colleagues reported early-onset paraplegia where spinal stenosis served to reduce epidural compliance, and again hyperlordosis and IVC compression compounded the risk for spinal ischaemia. In another report, delayed paraplegia occurred because of venous infarction from thrombosis of spinal veins, resulting in a relentless progression of symptoms. The aetiology in this instance differs but in all these cases prolonged hyperlordosis was a common factor.

In their review of over 17 000 central blocks, Dahlgren and Tornebrandt found 10 permanent neurological sequelae resulting from epidural blockade. Of these, five had a polyneuropathy or non-specific neurological symptoms. Two of these patients had diabetes, and two suffered periods of intraoperative hypotension.

The contribution of a period of anaemia is also hard to quantify. Our patient lost approximately 2 litres of blood, and had a starting haemoglobin of 15.1 g dl⁻¹. In a man weighing 80 kg, his final haemoglobin concentration was anticipated to be around 9 g dl⁻¹. The first haemoglobin concentration measured on the ICU was 6.8 g dl⁻¹ for which the policy is to transfuse to a haemoglobin concentration of 8–10 g dl⁻¹, so this was promptly done on arrival. However, the next haemoglobin measurement was 11.8 g dl⁻¹ so we feel that the first measurement was taken on a diluted specimen and the significantly deranged clotting at this time supports this. Albeit of unlikely significance in this case, we know of no reports indicating anaemia as a contributory factor other than in vaso-occlusive conditions.

There is no current treatment that has been shown to improve the outcome after infarction of the spinal cord. The results of the second National Acute Spinal Cord Injury Study in the USA suggested an improvement in neurological function with high-dose steroids in lesions resulting from blunt trauma only. In the review by Cheshire and colleagues, approximately 25% of cases showed good recovery, 50% some recovery and 25% no recovery at all.

Epidural anaesthesia is of undoubted benefit at improving postoperative pain control, with a low incidence of neurological complications. However, if these do develop, they are readily ascribed to the use of epidural anaesthesia. Any suggestion of pre-existing neurological dysfunction should therefore be fully documented before surgery and the benefits of using an epidural should be discussed with the patient in the light of any other factors that may influence the blood supply to the cord during the perioperative period. We therefore caution against the use of epidural analgesia where the hyperlordotic position is envisaged for prolonged periods of time where there are such identifiable risk factors.

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