Brief Report

Cauda equina syndrome after spinal anaesthesia in a patient with severe vascular disease

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Purpose: Spinal anaesthesia is selected for many lower extremity surgical procedures each year in the United States with a high degree of safety and efficacy. Even when adverse neurological outcomes have occurred, anatomical abnormality or coagulopathy have been implicated in the majority of cases. Epinephrine is used in high concentrations in many of these anaesthetics to increase the duration and intensity of the block. Although epinephrine is known to decrease spinal cord blood flow, its use in normal patients has not caused complications. We report a case where spinal anaesthesia with bupivacaine and epinephrine resulted in anterior spinal artery compromise and the development of a cauda equina syndrome postoperatively.

Clinical Features: A 57-yr-old man with severe coronary artery and peripheral vascular disease was scheduled for incision and drain of an abscess of the left thigh. He received an atraumatic dural puncture and injection of 12.5 mg bupivacaine with 0.2 ml 1:1000 epinephrine. During onset, he experienced a severe, painful sensation of the thighs which resolved with development of the block. Postoperatively, he was noted to have exacerbation of proximal muscle weakness and decreased perineal sensation and rectal tone. Subsequent EMG studies demonstrated proximal neuron loss consistent with cauda equina syndrome, presumed to be related to insufficiency of the anterior spinal artery.

Conclusion: Routine use of epinephrine in spinal anaesthesia for patients with multi-organ vascular disease should be considered carefully because of the possibility of vascular insufficiency of the spinal cord which would be exaggerated by the vasoconstrictive effect of epinephrine.

Objectif : Aux États-Unis, la rachianesthésie est choisie pour de nombreuses interventions chirurgicales des membres inférieurs et comporte un haut niveau d'innocuité et d'efficacité. Même quand des incidents neurologiques se sont produits, l'anomalie anatomique ou la coagulopathie a été incriminée dans la majorité des cas. L'épinéphrine est utilisée en concentrations élevées dans beaucoup d'anesthésiques rachidiens afin d'accroître la durée et l'intensité du bloc. Bien que l'épinéphrine soit connue pour diminuer le débit sanguin de la moelle épinière, son usage chez les patients normaux n'a pas causé de complications. Nous signalons ici un cas où la rachianesthésie avec bupivacaïne et épinéphrine a causé une atteinte de l'artère spinale antérieure et le développement postopératoire du syndrome de la queue de cheval.

Aspects cliniques : Un homme de 57 ans, présentant une sévère coronopathie et une maladie vasculaire périphérique, devait subir l'incision et le drainage d'un abcès sur la cuisse gauche. Il a reçu une ponction durale atraumatique et une injection de 12,5 mg de bupivacaïne avec 0,2 ml 1 : 1 000 d'épinéphrine. Pendant l'induction, il a éprouvé une douleur pénible à la cuisse, douleur qui est disparue à mesure que s'installait l'anesthésie. Après la chirurgie, on a remarqué une accentuation de la faiblesse des muscles proximaux, une diminution de la sensation du périnée et du tonus rectal. Des examens EMG subséquents ont montré une déperdition neuronale proximale caractéristique du syndrome de la queue de cheval, que l'on a présumé reliée à l'insuffisance de l'artère spinale antérieure.

Conclusion : L'emploi courant de l'épinéphrine dans la rachianesthésie de patients présentant une pathologie vasculaire pluriorganique doit être attentivement évalué, vu la possibilité d'une insuffisance vasculaire de la moelle épinière qui pourrait être accentuée par l'effet vasoconstricteur de l'épinéphrine.

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CAUDA equina syndrome is a rare complication of spinal or epidural anaesthesia[1] characterized by proximal lower extremity muscle weakness, sensory loss, sexual dysfunction and sphincter dysfunction of the bowel and bladder. A variety of causes have been reported.[2-4] We report a case in which spinal anaesthesia with epinephrine in a patient with diffuse vascular insufficiency was associated with cauda equina syndrome postoperatively.

Case Report

A 57-yr-old man was scheduled for incision and drainage of a mass of the right thigh. His past medical history was remarkable for non-insulin dependent diabetes, 80 pack-years of smoking, coronary artery bypass surgery, previous cerebrovascular accident and aorto-bifemoral bypass. During recovery from aortic reconstruction, he developed chronic renal insufficiency, a neurogenic bladder, and acute respiratory failure. Echocardiography prior to discharge revealed moderate left ventricular impairment. In the month before presentation of the mass, he noted some dyspnea with exertion, chest pain with heavy activity as well as claudication with walking more than one block. Review of the old medical records revealed a neurological examination which indicated 4/5 muscle weakness of the proximal muscles of the lower extremity. Haemoglobin was 10.9 $g \cdot dl^{-1}$, haematocrit was 33 and white count was 11,000$\cdot mm^{-3}$

After discussion of options, a spinal anaesthetic was selected. A right radial arterial line was placed and he was prehydrated with 500 ml lactated Ringers solution. Blood pressure (BP) was 130/70 mmHg, hear rate (HR) was 70 bpm and respiratory rate (RR) 12$\cdot min^{-1}$. With the patient in a sitting position, a 22 gauge Quincke needle was used to inject 12.5 mg plain bupivacaine with 0.2 mg epinephrine at the L_{3-4} interspace. There were no paraesthesia during injection of the local anaesthetic solution. He was returned to the supine position. After two-three minutes, he described an unpleasant sensation on the anterior surface of both thighs, which he described as "severely painful warmth". The BP was 120/70 mmHg, HR was 78 bpm and RR was 12$\cdot min^{-1}$. This sensation resolved within five minutes. The sensory level of the block was T_{10}. An abscess was identified, a drain was placed and the wound was loosely closed. There was no haemodynamic instability during the procedure.

On arrival in the post-anaesthesia care unit (PACU), he was noted to have an L_1 sensory level. After 110 min, he was discharged with apparently normal sensation and some motor function in the lower extremities. The following morning, he was unable to ambulate because his hips were too weak. Neurological examination revealed decreased sensation over the perineum, anterior and lateral thighs, and decreased motor function of the hips and thighs (2-3/5). Deep tendon reflexes were diminished at the knees but active at the ankle. Previous neurogenic bladder and self-catheterization made assessment of bladder sphincter function difficult to assess. There was decreased rectal sphincter tone. A magnetic resonance scan (MRI) revealed no abnormality of the spine.

Over the next several days, there was some return of perineal sensation and motor function of the lower extremity muscles. Rectal sphincter tone returned to baseline. The EMG at 10 days revealed conduction disturbance in the proximal region of L_5 and S_1, which was felt to confirm pre-existing neurological defects. The suspected aetiology of the increase in neurological deficit was anterior spinal artery vascular insufficiency. He was transferred to a chronic rehabilitation unit. Rehabilitation was complicated by lower extremity ischaemia and aorto-enteric fistula which was found to track to the site of the initial abscess. Subsequent EMG studies were refused by the patient. After several months and three additional operations the patient died from cardiopulmonary failure. Autopsy was refused by the family.

Discussion

The most likely explanation of the outcome in this case is transient ischaemia of the anterior spinal artery, which led to the cauda equina syndrome. Ischaemia of the cauda equina may occur because of the limited collateralization of the anterior spinal artery.[1] Although the anterior spinal artery is a continuation of the vertebral artery, it is discontinuous in the thoracic and lumbar regions, and fed by branches of adjacent vessels. The lower thoracic and lumbar cord is often supplied by a single branch from the abdominal aorta, the artery of Adamkiewicz without collateral supply. This deficiency of collateral blood flow makes the cauda equina susceptible to ischaemia.

The unique aspect of this case is the severe vasculopathy. There was clinical evidence of multi-organ microvascular insufficiency (kidney, myocardium, bladder, lower extremities, central nervous system). Likewise, there may have been vascular insufficiency of the spinal cord. The additional effect of adding epinephrine to the subarachnoid space may have induced ischaemia, especially if there was disturbance of the artery of Adamkiewicz from previous aortic reconstruction. His pre-existing lower extremity weakness was further evidence of possible vascular insufficiency of the spinal cord.

Epinephrine is known to potentiate the analgesic quality of subarachnoid bupivacaine[5] and was selected for this reason but this is balanced by theoretical disadvantages. Epinephrine is known to decrease blood flow in the spinal cord.[6] Nevertheless, despite widespread use, little evidence for toxicity or permanent injury exists. There have been, however, case reports where paraesthesia[3] have been attributed to epinephrine.

Other causes of spinal cord injury cannot be excluded. Although massive injections of local anaesthetics,[8] decreased compliance of the epidural or subarachnoid space,[4] air in the epidural space[9] and herniation of nucleus pulposus material[10] have been associated with the cauda equina syndrome, none of these occurred. In addition, the spinal column was normal by MRI scan.

In conclusion, we report a case of cauda equina syndrome which we believe was related to anterior spinal artery insufficiency secondary to a subarachnoid injection of bupivacaine and epinephrine in a patient with severe vascular disease. Routine use of epinephrine for spinal anaesthesia in patients with multi-organ vascular disease should be considered carefully.

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