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BILATERAL SENSORY LOSS, BACK PAIN, AND URINARY RETENTION WITHOUT NEURAL COMPRESSION AFTER MENISCUS REPAIR: A CASE REPORT

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ABSTRACT This presented case report is about a rare neurological complication following right meniscus repair under general anaesthesia. A female in her late 40s with ASA II status underwent an uneventful procedure but developed severe lower back pain, bilateral lower limb sensory deficits and urinary retention postoperatively. She was investigated for cauda equina syndrome. The MRI showed no structural abnormalities or neural compression, indicating the same. The differentials were 1) transient spinal cord ischemia, 2) anaesthetic neurotoxicity, 3) structural abnormalities, 4) inflammatory response, and 5) hypothermia-related injury. No cause for the above differentials was established. This case presents the need for close neurological monitoring in patients following general anaesthesia. Surgeons and anaesthesiologists should know about such rare complications to ensure early intervention and improve patient recovery.

KEYWORDS : *General Anaesthesia, Neurological Complication, Sensory Loss, Urinary Retention, Meniscus Repair.*

BACKGROUND

General anaesthesia is a cornerstone of modern surgical practice. While generally considered safe, it is not devoid of risks. Perioperative neurological complications, though infrequent, can have significant implications for patient outcomes. The incidence of such complications varies, with studies suggesting rates of less than 1% in the general surgical population, though higher incidences are noted in specific surgical contexts (1).

Neurological deficits post-anaesthesia can manifest due to various mechanisms, including spinal cord ischaemia resulting from intraoperative hypotension, mechanical injury, or anaesthetic neurotoxicity. Spinal cord injury during anaesthetic practice, albeit rare, carries a substantial burden in terms of morbidity and mortality. Additionally, concerns have been raised regarding the potential neurotoxic effects of general anaesthetics, particularly in vulnerable populations, with evidence suggesting possible long-term cognitive and behavioural impairments (2,3).

This case report details an unusual presentation of postoperative neurological dysfunction in a patient following routine right knee meniscus repair under general anaesthesia. Despite an uneventful intraoperative course and the absence of structural abnormalities on magnetic resonance imaging (MRI), the patient developed severe lower back pain, bilateral lower limb sensory loss, and urinary retention.

CASE PRESENTATION

A female patient in her late 40s underwent elective right knee meniscus repair under general anaesthesia. The procedure performed was a diagnostic arthroscopy of the right knee, during which she was supine with side support. Her surgery lasted approximately 90 minutes and was uneventful.

The general anaesthesia consisted of a combination of intravenous sedatives, analgesics, and anaesthetic agents carefully titrated to maintain hemodynamic stability. Midazolam (2 mg IV) was administered for anxiolysis and sedation, followed by alfentanil (1 mg IV) to provide short-acting analgesia. Induction was achieved with propofol (160 mg IV), ensuring rapid unconsciousness, while fentanyl (50 micrograms IV) was used for additional analgesia. To minimise postoperative nausea and inflammation, dexamethasone (6.6 mg IV) and ondansetron (4 mg IV) were given. Paracetamol (1000 mg IV) was used prophylactically for pain management, and teicoplanin (400 mg IV) was administered for infection prophylaxis. Additionally, esketamine (10 mg IV) was included to enhance analgesia and reduce opioid requirements.

Throughout the procedure, anaesthesia was maintained with sevoflurane titrated based on patient response and intraoperative monitoring. Respiratory mechanics were meticulously adjusted, with tidal volumes ranging from 387 ml to 534 ml, and minute ventilation maintained between 3.9 L/min and 6.4 L/min to ensure adequate gas exchange. Airway pressures were carefully controlled, with peak pressures fluctuating between 10 cmh2o and 14 cmH2O and plateau pressures maintained between 10 cmH2O and 12 cmH2O. Airway resistance was within normal limits at 7 cmh2o to 10 cmh2o. The ventilator rate was adjusted between 8 bpm and 14 bpm, while the inspiratory time was held constant at 2.5 seconds to optimise lung mechanics.

Oxygenation was well-maintained, with SpO2 levels consistently at 99-100%, indicating effective oxygen delivery. End-tidal CO2 (EtCO2) values fluctuated between 4.8 kPa and 5.33 kPa, confirming adequate ventilation. FiO2 was initially set at 100% before being gradually reduced to 40% to minimise oxygen toxicity risk. The depth of anaesthesia was carefully monitored, with end-tidal sevoflurane (EtSEVO) concentrations ranging from 1.4% to 2.1% and a minimum alveolar concentration (MAC) maintained between 0.7 and 0.9, ensuring sufficient anaesthesia depth without excessive sedation. Her core temperature remained stable, fluctuating between 36.2°C and 36.5°C, eliminating significant hypothermia as a potential contributing factor.

Postoperative Course And Clinical Findings: Immediately following surgery, the patient reported a sudden onset of severe lower back pain. Within a short period, she developed bilateral lower limb sensory loss, loss of motor power in the right leg, and urinary retention. The neurological examination demonstrated notable abnormalities across multiple domains. Motor function assessment revealed reduced power in the right lower limb, indicative of motor impairment. Sensory evaluation showed bilaterally diminished sensation in the left lower limb, specifically affecting dermatomes L1, L2, L4, L5, and S1. Bladder function assessment indicated a loss of sensation of bladder fullness, accompanied by an inability to initiate micturition. This was further evidenced by two unsuccessful attempts at a trial without catheterisation. Additionally, the examination of bowel function identified a loss of anal tone and diminished perianal sensation, as confirmed by digital rectal examination. These findings collectively suggest a significant neurological deficit impacting motor, sensory, bladder, and bowel functions (**Figure 1**).

The patient exhibited several notable physiological parameters during the perioperative period. Blood pressure monitoring using non-invasive blood pressure (NIBP) measurement revealed episodes of hypotension, which may suggest spinal cord hypoperfusion, potentially contributing to neurological dysfunction. The concentration of sevoflurane (EtSEVO) varied between 1.4% and 2.1%, indicating fluctuations in anaesthetic depth that could have influenced post-anaesthetic neurological dysfunction. Oxygen saturation (SpO2) remained consistently between 99% and 100%,

effectively excluding hypoxia as a contributing factor. Additionally, body temperature (TEMP-T1) was maintained within a normal range of 36.2°C to 36.5°C, indicating that significant hypothermia was not a concern. These findings collectively provide insights into potential intraoperative factors affecting neurological outcomes.

Diagnosis: The most plausible explanations included spinal cord ischemia or transient hypoperfusion due to intraoperative hypotension, anaesthetic neurotoxicity from sevoflurane or an inflammatory response triggering transient neurological impairment. There was no evidence of cauda equina syndrome on the MRI spine, ruling out any structural abnormality. There was no evidence of hypothermia intraoperatively.

Treatment

The patient was admitted for neurological monitoring and symptomatic management, with the primary goal of stabilising neurological function and preventing further complications. Immediate interventions included intravenous fluid resuscitation to optimise spinal cord perfusion and mitigate any potential ischemic injury. Blood pressure support was maintained within an optimal range to prevent further hypoperfusion-related damage.

Urinary retention was managed with bladder catheterisation, ensuring proper drainage and preventing complications related to prolonged urinary stasis. Early mobilisation with physiotherapy to prevent deconditioning and optimise motor and sensory function recovery.

OUTCOME AND FOLLOW-UP

Over the following days, the patient exhibited gradual improvement in sensory and motor function, with progressive recovery of bladder control. Follow-up evaluations were scheduled to monitor long-term neurological outcomes and assess residual deficits requiring rehabilitation.

DISCUSSION

General anaesthesia is essential for modern surgery, ensuring analgesia, amnesia, unconsciousness, and muscle relaxation (4). It is known to cause common issues like nausea, vomiting and temporary confusion, which usually resolve quickly. In 0.03% – 0.1%, it is known to cause perioperative neurological deficits, caused by ischemia, mechanical injury or inflammation (5). Such complications may result from spinal cord injury, low blood pressure or nerve compression. Most cases have clear causes, such as prolonged hypotension or pre-existing conditions (6). But this case is different as the patient developed sudden lower limb sensory loss, intense lower back pain and urinary retention after general anaesthesia, without any cause.

Postoperative neurological deficits following non-spinal surgeries are uncommon and often raise concerns about intraoperative factors such as hypotension, ischemia, anaesthetic neurotoxicity, or pre-existing but undiagnosed neurological conditions (7). In this case, the intraoperative anaesthesia monitoring chart provides crucial insights into potential mechanisms contributing to the observed complications. The recorded sevoflurane concentrations ranged from 1.4% to 2.1%, within acceptable anaesthetic depth levels, reducing the likelihood of excessive anaesthetic exposure as a primary cause (8). One of the key intraoperative concerns is the occurrence of hypotension, which could have led to spinal cord hypoperfusion and ischemic injury (9). Although blood pressure data are limited, prolonged episodes of low mean arterial pressure (MAP) could compromise spinal cord perfusion, particularly in patients with borderline vascular status (10). Hypoxia is another potential contributing factor; however, the intraoperative SpO2 remained stable at 99–100%, effectively ruling out significant hypoxic injury (11). Temperature monitoring indicated mild intraoperative hypothermia (minimum 36.2°C), but this level is unlikely to be solely responsible for the patient's neurological symptoms (12).

Bladder and bowel dysfunction, in conjunction with bilateral lower limb sensory loss, raises concerns about cauda equina syndrome; however, the absence of MRI evidence of neural compression contradicts this diagnosis (13).

In conclusion, this case report presents a rare and diagnostically challenging neurological complication following routine GA for right knee meniscus repair. The absence of identifiable causative factors highlights the critical need for heightened awareness of such rare complications. Further research is warranted to elucidate the underlying mechanisms and develop preventive strategies for such rare yet impactful events.

LEARNING POINTS/TAKE HOME MESSAGES

- Unexpected neurological complications can occur after general anaesthesia, even in the absence of neural compression or structural abnormalities, emphasising the need for close postoperative monitoring.
- Transient spinal cord ischemia and anaesthetic neurotoxicity should be considered as differential diagnoses in patients presenting with postoperative sensory deficits, urinary retention, or motor weakness.
- Maintaining intraoperative hemodynamic stability, including optimal blood pressure control, is critical to prevent ischemic complications affecting the spinal cord.
- Early recognition, prompt neurological evaluation, and supportive management can significantly improve patient outcomes in cases of postoperative neurological impairment.

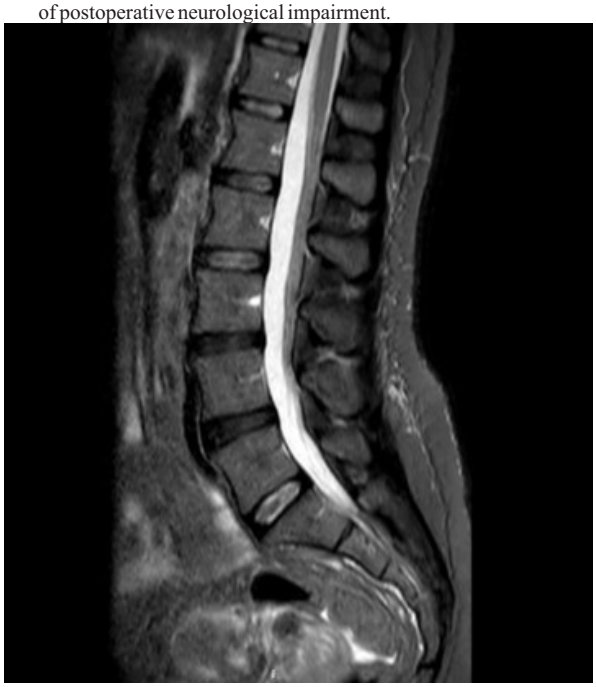

Figure 1: The sagittal lumbar spine MRI does not reveal evidence of spinal cord compression, disc herniation or significant neural impingement. Vertebral alignment remains intact, and no acute abnormalities suggestive of ischemic or mechanical injury. Disc desiccation at L3/L4 and L4/L5. The rest of the intervertebral discs are of normal height and return normal signals, with no evidence of central canal, lateral recess, or exit foramina narrowing.

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