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## CLINICAL VIGNETTE

# Unilateral Paraparesis after Spinal Anesthesia

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### Case

A 55-year-old man with lower urinary tract symptoms underwent a transurethral resection of prostate (TURP) under spinal anesthesia. He had a history of spinal stenosis status post L3-L5 laminectomy, cervical radiculopathy, non-insulin dependent diabetes mellitus, obstructive sleep apnea and moderate asthma. Preoperative vital signs, complete blood count, basic metabolic panel, electrocardiogram and echocardiogram were within normal limits.

Two years prior to presentation, the patient had undergone urgent decompressive L3-L5 laminectomy for a 4 week history of right lower extremity numbness and weakness. He had regular follow-up with neurosurgery postoperatively. The neurosurgery services most recent note 1 month prior to surgery noted an absence of sensation to light touch in the RLE, but otherwise strength was noted to be intact. The preoperative examination 1 week prior to surgery noted no focal deficits, and that the patient was able to lift heavy objects without issue. A recent MRI showed that despite the L3-L5 laminectomies, there remained moderate spinal canal narrowing and mild to moderate R neural foramina narrowing from L2-L5.

On the day of surgery, a fully informed consent with a discussion of the risks, benefits and alternatives of anesthesia was conducted with the patient. The patient's preference was for neuraxial anesthesia and he understood the risks associated with it and elected to proceed. Using a premade spinal kit from Medline, 1.4cc of 0.75% Bupivacaine in 8.75% dextrose was injected intrathecally along with 15mcg of fentanyl and an epinephrine wash through a 25g pencil point spinal needle. The placement was not technically challenging, needing only 1 attempt and no abnormalities other than a midline lumbar scar was noted. The anesthesiologist reported that they went one level above the upper limit of the midline scar.

After sensory blockade to level T8 was obtained, sedation with a propofol infusion @ 50 mcg/kg/min was started and the case proceeded uneventfully. The patient received gentamicin 3mg/kg (240mg) for antibiotic prophylaxis. The patient was positioned low lithotomy for a total of 78 minutes and the total operative time was 64 minutes. A fluid deficit of less than one liter was noted at the conclusion of the case. 1200ml of Lactated Ringer's solution was given and the estimated blood loss was

25 ml. The patient was taken to the recovery area in stable condition at the conclusion of the case.

Two hours after arrival to the recovery room, and 4 hours after the spinal was placed, the patient was still unable to move his R lower extremity at all, and still had some residual weakness on the left. 8 hours after the initial spinal placement, the patient continued to have 0/5 strength in his RLE and only 1/5 strength in his LLE prompted stat MRI which was essentially unchanged from prior. It specifically showed no acute process such as a hematoma, abnormal fluid accumulations or damage to any structures. Neurology was consulted, who localized the deficit along the right side going as high as T8, and thus recommended a Thoracic MRI which was also negative for any acute process.

Over the next 4 days, the patient's neurologic examination would wax and wane, but never returned to baseline. At the time of discharge from the hospital on POD # 4, he had 4/5 strength in the R hip and knee and 2/5 strength in the R ankle. The final diagnosis from Neurology, was Right Leg Weakness of unclear etiology and he was discharged home with home physical therapy.

Two weeks after the initial insult, the patient had a follow-up visit with his PCP. He reported no subjective improvement in his symptoms, and that he needed to use a walker to ambulate. He had 2-3/5 strength in the muscle groups of the R lower extremity and absent sensation below the knee on the right side, while being neurologically intact on the left side.

### Discussion

Spinal anesthesia has been successfully used for major surgical procedures below the diaphragm for over 120 years, with the first spinal anesthetic administered by August Bier in 1898 using the local anesthetic cocaine.<sup>1</sup> Until the last decade, spinal anesthesia was the preferred anesthetic technique for TURPs and still is when monopolar TURP is to be performed.<sup>2</sup> This is due to the unique risk of TURP syndrome that could manifest during the procedure when using a monopolar electrocautery that requires an electrically inert solution, but due to the characteristics of the solutions used can lead to hypervolemia, mental status changes, pulmonary edema, transient vision loss, hyponatremia/hypo-osmolality and other complications.<sup>3</sup>

Bipolar TURP was introduced in the mid-2000s and randomized trials showing its effectiveness began to be published in 2009-2010.<sup>4</sup> Since the bipolar electrocautery unit contains both electrodes, the risk of electrical propagation is not a major concern. This allows the use of isotonic irrigating fluid, such as normal saline or lactate ringer, eliminating the risk of electrolytic disturbance from systemic uptake and also metabolism to toxic metabolites of the previously used irrigation fluids.<sup>4</sup> This reduces the need to perform the procedure under spinal anesthesia to monitor for the neurologic complications of TURP syndrome.

This case highlights one of the most often discussed and the most dreaded complications of neuraxial anesthesia, major neurological injury. Prolonged nerve injury after neuraxial anesthesia is in fact a very rare complication. Moon et al reviewed 1.7 million neuraxial anesthetics and found only 4 cases (1 per 425,000) of paraparesis.<sup>5</sup> Other sources have quoted the incidence of paraplegia to be 1 per 100,000, and note that the mechanism of a severe injury is likely multifactorial and difficult to identify.<sup>6,7</sup>

Possible etiologies for the neuraxial related paraplegia include direct needle trauma, the injection of foreign substances into the CSF (see the cases of Woolley and Roe from 1947 and the chloroprocaine induced neurotoxicity that occurred in the 1980s), profound hypotension of the spinal cord, anterior spinal cord ischemia or a combination of factors such as hypotension, mechanical obstruction, vasculopathy and/or hemorrhage leading to ischemia and irreversible cord damage.<sup>8-10</sup> Other causes - which were ruled out in our patient by imaging, physical exam or other methods - include cauda equina syndrome and epidural hematoma.

For the patient described, his history of spinal stenosis, spinal surgery and pre-existing neuropathy all put him at increased risk of neurologic injury. Hebl et al showed that patients with spinal stenosis are at increased risk of neurological complications after neuraxial anesthesia, however, did not report the relative contribution of anesthetic and surgical factors.<sup>11</sup> A history of spine surgery by itself does not increase the risk of neurologic injury. However, the presence of scar tissue, adhesions, hardware and/or bone grafts may lead to changes in CSF flow patterns resulting in unequal spread, uptake and termination of action of local anesthetics.<sup>11,12</sup> The 2015 ASRA guidelines on Neurologic complications associated with regional anesthesia take these risk factors into account and give guidance that in patients with known severe spinal stenosis, a thorough risk-to-benefit be considered before neuraxial is performed. They do note that it is unclear what contribution can be attributed to anesthetic factors, surgical factors and factors related to the natural progression of the disease.<sup>13</sup>

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