Vocal Fold Paralysis Following Spinal Anesthesia

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pinal anesthesia is a commonly used alternative to general anesthesia for surgical procedures of the lower extremities, perineum, or lower abdomen. Cranial nerve palsy is a rare complication of spinal anesthesia. The incidence is not well defined; however, older literature reports an incidence of 1 in 200 to 1 in 1200 cases.1 The most commonly affected is the abducens nerve, which usually presents between the second and fifth postoperative day with diplopia.2 Palsies of the oculomotor, facial, trigeminal, and trochlear nerves, as well as transient fifth postoperative day with diplopia.2 Palsies of the oculomotor, facial, trigeminal, and trochlear nerves, as well as transient cranial neuropathies have also been reported following lumbar puncture, myelography, spontaneous intracranial hypotension,6 and inadvertent dural tear during lumbar spine surgery.9 Lower cranial neuropathies following instrumentation of the dural space have not been described to our knowledge. Herein we present 4 cases of vocal fold paralysis (VFP) that seem to have been caused by spinal anesthesia.

Report of Cases

Patient 1
A woman in her 50s underwent a total arthroplasty of the right knee under spinal anesthesia and conscious sedation. A right central venous catheter was placed in the internal jugular vein, and no instrumentation was introduced by mouth. On postoperative day 4, she noted onset of hoarseness and choking with liquids; she believed that her voice had been normal immediately after surgery. She was discharged to home but was readmitted 2 weeks after surgery with a wound infection. Bedside laryngoscopy performed at that time revealed a left VFP. Her hoarseness persisted, and stroboscopy performed 3 months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold. This finding was confirmed on repeat stroboscopy in the office. She subsequently underwent computed tomography (CT) of the neck, which did not reveal any lesions along the recurrent laryngeal nerve. A laryngeal electromyography was performed at 3 months following surgery. This showed evidence of denervation of the right thyroarytenoid (innervated by the recurrent laryngeal nerve) and cricothyroid muscle (innervated by the superior laryngeal nerve), consistent with a right vagal (as opposed to recurrent laryngeal) neuropathy. The patient subsequently underwent injection medialization with methylcellulose with satisfactory improvement in voice. Three months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold with a small pseudocyst; voice continued to be adequate. At 1-year follow-up, the right vocal fold was mobile but with some signs of residual paresis, including increased size of the pseudocyst.

Patient 2
A woman in her 60s underwent a total arthroplasty of the right knee under spinal anesthesia and conscious sedation. A right central venous catheter was placed in the internal jugular vein, and no instrumentation was introduced by mouth. On postoperative day 4, she noted onset of hoarseness and choking with liquids; she believed that her voice had been normal immediately after surgery. She was discharged to home but was readmitted 2 weeks after surgery with a wound infection. Bedside laryngoscopy performed at that time revealed a left VFP. Her hoarseness persisted, and stroboscopy performed 3 months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold. This finding was confirmed on repeat stroboscopy in the office. She subsequently underwent computed tomography (CT) of the neck, which did not reveal any lesions along the recurrent laryngeal nerve. A laryngeal electromyography was performed at 3 months following surgery. This showed evidence of denervation of the right thyroarytenoid (innervated by the recurrent laryngeal nerve) and cricothyroid muscle (innervated by the superior laryngeal nerve), consistent with a right vagal (as opposed to recurrent laryngeal) neuropathy. The patient subsequently underwent injection medialization with methylcellulose with satisfactory improvement in voice. Three months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold with a small pseudocyst; voice continued to be adequate. At 1-year follow-up, the right vocal fold was mobile but with some signs of residual paresis, including increased size of the pseudocyst.

Patient 3
A woman in her 30s underwent a total arthroplasty of the right knee under spinal anesthesia and conscious sedation. A right central venous catheter was placed in the internal jugular vein, and no instrumentation was introduced by mouth. On postoperative day 4, she noted onset of hoarseness and choking with liquids; she believed that her voice had been normal immediately after surgery. She was discharged to home but was readmitted 2 weeks after surgery with a wound infection. Bedside laryngoscopy performed at that time revealed a left VFP. Her hoarseness persisted, and stroboscopy performed 3 months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold. This finding was confirmed on repeat stroboscopy in the office. She subsequently underwent computed tomography (CT) of the neck, which did not reveal any lesions along the recurrent laryngeal nerve. A laryngeal electromyography was performed at 3 months following surgery. This showed evidence of denervation of the right thyroarytenoid (innervated by the recurrent laryngeal nerve) and cricothyroid muscle (innervated by the superior laryngeal nerve), consistent with a right vagal (as opposed to recurrent laryngeal) neuropathy. The patient subsequently underwent injection medialization with methylcellulose with satisfactory improvement in voice. Three months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold with a small pseudocyst; voice continued to be adequate. At 1-year follow-up, the right vocal fold was mobile but with some signs of residual paresis, including increased size of the pseudocyst.

Patient 4
A woman in her 40s underwent a total arthroplasty of the right knee under spinal anesthesia and conscious sedation. A right central venous catheter was placed in the internal jugular vein, and no instrumentation was introduced by mouth. On postoperative day 4, she noted onset of hoarseness and choking with liquids; she believed that her voice had been normal immediately after surgery. She was discharged to home but was readmitted 2 weeks after surgery with a wound infection. Bedside laryngoscopy performed at that time revealed a left VFP. Her hoarseness persisted, and stroboscopy performed 3 months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold. This finding was confirmed on repeat stroboscopy in the office. She subsequently underwent computed tomography (CT) of the neck, which did not reveal any lesions along the recurrent laryngeal nerve. A laryngeal electromyography was performed at 3 months following surgery. This showed evidence of denervation of the right thyroarytenoid (innervated by the recurrent laryngeal nerve) and cricothyroid muscle (innervated by the superior laryngeal nerve), consistent with a right vagal (as opposed to recurrent laryngeal) neuropathy. The patient subsequently underwent injection medialization with methylcellulose with satisfactory improvement in voice. Three months later (6 months after injury), stroboscopy was repeated, revealing some return of motion of the right vocal fold with a small pseudocyst; voice continued to be adequate. At 1-year follow-up, the right vocal fold was mobile but with some signs of residual paresis, including increased size of the pseudocyst.

CONCLUSIONS AND RELEVANCE Intracranial hypotension resulting in vagal neuropathy is a possible complication of spinal anesthesia and appears to be the cause of transient vocal fold palsy in these 4 cases.

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IMPORTANT: Transient upper cranial neuropathy is a well-described complication of spinal anesthesia. Lower cranial neuropathies, in particular vagal neuropathies, have not been described. We present 4 cases of vocal fold paralysis (VFP) in 3 patients following spinal anesthesia and discuss a proposed mechanism.
Vocal Fold Paralysis Following Spinal Anesthesia
Case Report/Case Series

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Vocal Fold Paralysis Following Spinal Anesthesia

Case Report/Case Series

Stroboscopy performed 1 week later confirmed the finding of an immobile right vocal fold. The patient was prescribed steroids according to a tapering protocol, and a neck CT scan was performed with results within normal limits. The patient did not return for follow-up because her voice slowly returned to normal over several months. She returned 2 years later with new hoarseness that began 3 days after a second normal vaginal delivery with combined spinal epidural anesthesia. Stroboscopy performed 5 days postpartum revealed a fully mobile right vocal fold and an immobile left vocal fold. Results of CT of the neck and chest were within normal limits. A neurologic evaluation and autoimmune workup failed to reveal any cause for neuropathy. The patient was treated with steroids according to a tapering protocol and watchful waiting. At 6-month follow-up, she reported that her voice quality was back to normal; stroboscopy revealed only mild residual paresis of the left vocal fold.

Discussion

At first glance, these cases of VFP may be classified as idiopathic despite the coincidence of onset because spinal anesthesia is generally not considered in the differential diagnosis of vocal fold immobility. However, a causal relationship may be considered in light of the documented occurrence of upper cranial neuropathy as a complication of spinal anesthesia, especially in the absence of important confounding factors. The onset of symptoms, ranging from immediate to 7 days after surgery in our patients, is consistent with previously reported cases of cranial neuropathy following spinal anesthesia. So too is the transient nature of these vocal fold palsies.

Cranial neuropathy following dural puncture is thought to be due to reduction of cerebral spinal fluid volume and consequent decreased intracranial pressure. This is hypothesized to cause traction on the nerve at the level of the brainstem or possibly compression of the nerve by surrounding structures (eg, brain parenchyma, blood vessels).1,10 This idea is supported by the fact that transient cranial neuropathies have also been reported in spontaneous intracranial hypotension.8 In our patient who underwent laryngeal electromyography, this was suggestive of a vagal lesion, given the evidence of denervation of the cricothyroid muscle (innervated by the superior laryngeal nerve in addition the thyroarytenoid), rather than an isolated neuropathy of the recurrent laryngeal nerve; this lends support to the hypothesis of traction on the vagus nerve rather than an idiopathic palsy, the majority of which are recurrent nerve phenomena. Holes in the dura can continue to leak following puncture, and continued egress of spinal fluid may result in a delayed presentation of the neuropathy, as was the case in 3 of our 4 cases. Because of this, combined with the lack of recognition of spinal anesthesia as a potential cause, these neuropathies may be overlooked or attributed to other factors.

Conclusions

Vocal fold paralysis seems to be a potential complication of spinal anesthesia and should be considered in patients presenting with dysphonia shortly after such a procedure. In the absence of other findings on workup, these patients may expect complete or near-complete functional recovery weeks to months after onset.

REFERENCES

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