A case of priapism following intrathecal morphine injection in a dog

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Abstract

Background: Priapism refers to prolonged erection unrelated to sexual stimulation, with severe sequelae unless treated. In humans, it is a rare complication associated with epidural or spinal opioid administration. Its pathophysiology is unclear. This is the first report of priapism following neuraxial anesthesia in dog.

Case Description: An intrathecal morphine injection (30 mcg/kg) at L5–L6 for postoperative analgesia was given at the end of surgery for removal of cutaneous mastocytomas of the abdomen and left axillary lymphadenectomy. Painless penile erection occurred 2 hours later and lasted 6 hours, before spontaneously resolving 7–8 hours after the injection. No pain or other adverse events (e.g., nausea, urinary retention, and itching) were recorded. Recovery was complete without treatment.

Conclusion: Painless, self-resolving priapism is a rare complication associated with intrathecal morphine injection in dogs.

Keywords: Dog, Intrathecal morphine, Priapism.
hub of the needle. Preservative-free morphine (30 μg/kg) was diluted with 0.6 ml of sterile saline solution and administered in a single bolus over 20–40 seconds. Recovery was uneventful without initial complications, and the dog was brought into the ward. 2 hours after tracheal extubation, a painless penile erection began and lasted 6 hours, before spontaneously resolving 7–8 hours after the spinal injection. No pain or other adverse events (e.g., nausea, urinary retention, or itching) were recorded. Recovery was complete without sequelae and the dog was discharged from hospital the morning after the surgery. The follow-up physical examination 1 week later revealed no abnormal findings and the owners reported no sequelae.

Discussion
This is the first case report of priapism after intrathecal morphine injection in a dog. The parasympathetic nervous system (PNS) controls erection, while the sympathetic nervous system (SNS) influences ejaculation and termination of an erection (Pybus et al., 1984). Erection is produced by blocking sympathetic vasoconstriction, whereas detumescence results when parasympathetic vasodilatory action is impaired. Priapism refers to full or partial penile erection that lasts for several hours and is unrelated to sexual arousal. Its pathophysiology is unclear but is thought to stem from hemodynamic dysfunction of the penis, resulting in persistent erection. One explanation for its cause is that there is a sort of autonomic imbalance between the SNS and the PNS that leads to excessive, persistent blood engorgement (Pelavski et al., 2006). The underlying cause of postoperative priapism remains elusive. Theoretically, any drug or drug combination that affects the neurovascular system or the central nervous system may cause priapism (Appell et al., 1977; Gottlieb and Lustberg, 1977; Chin and Sharp, 1983).

In the human medical literature, priapism has been reported as a complication of central regional anesthesia with local anesthetics (Van Arsadelen et al., 1983; Tsai and Hong, 1990; Burnett, 2003; Pelavski et al., 2006; Nair et al., 2019). Epidural anesthesia is thought to produce a potential sympathetic blockade at the lumbar level, without simultaneous complete parasympathetic blockade of the sacral spinal cord. This may result in high flow (nonischemic) priapism (Van Arsadelen et al., 1983; Tsai and Hong, 1990; Burnett, 2003; Pelavski et al., 2006). The same mechanism may explain the occurrence of priapism after spinal injection: the local anesthetic is diluted by the cerebral spinal fluid, resulting in less concentration in areas distal to the injection site. Another possible mechanism is selective inhibition of sympathetic innervation of the penis by spinal anesthesia, which results in unopposed parasympathetic activity and ultimately penile erection (Nair et al., 2019).

As largely described in humans, epidural (Torda et al., 1980; Rawal et al., 1983) and intrathecal opioids (Pybus et al., 1984; Wiesenfeld-Hallin and Soderstern, 1984; Cunicelli et al., 2021) seem to have a key role in priapism. Sustained erections and incapacity to ejaculate have been reported after single-shot epidural administration (Torda et al., 1980; Rawal et al., 1983) and via catheter in healthy men (Ruan et al., 2007). Wiesenfeld-Hallin and Soderstern (1984) noted that as intrathecal morphine increased, naloxone decreased the number of intromissions prior to orgasm in male rats. To our best knowledge, there are no reports of systemic morphine-causing priapism. Furthermore, neither prolonged penile erection nor inability to ejaculate was reported in males following IV or IM opioid injection (Torda et al., 1980; Rawal et al., 1983; Wiesenfeld-Hallin and Soderstern, 1984). These findings corroborate the opioid-induced spinally mediated mechanism that decreases sympathetic response to sexual stimulation (Pybus et al., 1984). However, sexual functioning is variably affected by opioids. Reduction in testosterone levels, decreased libido, erectile dysfunction, and delayed ejaculation are noted to occur frequently in chronic opioid users (Mirin et al., 1980; Gulliford, 1998), and impotence and decreased libido are often seen in patients under long-term intrathecal morphine therapy (Ruan, 2007).

Our hypothesis is that the anesthetic protocol we used did not lead to the occurrence of priapism in this patient. Dexmedetomidine was shown to have protective effects against ischemia-reperfusion injury in an experimental rat model of priapism (Köfükçu et al., 2021), and it has been successfully used in the treatment of intraoperative penile erection in men (Guler et al., 2021). Although priapism is sometimes associated with propofol-based anesthesia in men (Corten et al., 2017), it is improbable that the propofol timing and dosage we used for anesthesia induction could have been responsible for this complication. In our opinion, the cause of priapism is ascribable to the intrathecal morphine the dog received as postoperative analgesia. A probable cause was the blockage of sympathetic outflow from the sacral spinal cord through a spinally mediated mechanism. Another possible mechanism was an increased nitric oxide (NO)-mediated vasodilation. NO is released from vascular endothelium and mediates the increase of blood flow and the relaxation of smooth muscle in the corpora cavernosa, causing erection. Morphine is believed to cause an increase in endothelial production of NO (Pourshanazari et al., 2011) and might contribute to the development of priapism. These conclusions are concordant with studies in humans (Ruan et al., 2007; Cunicelli et al., 2021).

The morphine dosage we used has been reported in previous studies on orthopedic surgery with bupivacaine (Sarotti et al., 2013, 2016, 2019) without the occurrence of priapism. The same dose of morphine (without local anesthetics) was effective in managing perioperative pain in dogs undergoing major thoracic or cranial abdominal surgery (Lardone et al., 2022).
and it is known to reduce the need for rescue analgesia in dogs undergoing cervical and thoracolumbar spinal surgery (Novello et al., 2008). Considering all the above, the cranial spread was expected to be sufficient to produce adequate analgesia in terms of quality and duration. Although its frequency is extremely rare in dogs and uncommon in men, many anesthetists, including ourselves, often add a low dose of morphine to lumbar epidural or spinal injection without risking the complication of priapism. It is unclear what could have increased the susceptibility of the patient to developing such a complication. It seems that the site of injection in relation to the autonomic outflow from the spinal cord plays an important role in priapism development as all previously reported cases, including ours, involved lumbar central regional anesthesia where the injection or the catheter tip could potentially be adjacent to the sympathetic outflow of the L1–L2 nerve roots.

We were unable to find published data on spinal morphine at recovery. In our experience, intrathecal morphine is more often associated with adverse effects when administered at the end of surgery as postoperative analgesia. While general anesthesia may hide some complications (e.g., itching or nausea) timing could be another crucial factor. We speculate that a high spinal concentration of opioids (without local anesthetic), together with sudden sympathetic activation during recovery from the general anesthesia, may have contributed to the priapism in this patient. Although pain is commonly reported during low-flow priapism in men (Corten et al., 2017), no pain was observed in our patient and no analgesics were administered. The complication self-resolved without any treatment. Although priapism is rare, veterinarians should be aware of this complication and initiate prompt therapy as needed.

Conflict of interest
The authors declare that the study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Authors’ contributions
EL: data management and manuscript preparation; VG: data collection; and PF: interpretation and correction of the manuscript.

References


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