Intraoperative Priapism during Neuraxial Anaesthesia Resulting in Trouble to the Urologist: A Case Report

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Abstract

Introduction: Priapism following neuraxial anaesthesia for urological endoscopic procedures is uncommon and may result in delay or even postponement of scheduled operations. An imbalance between sympathetic and parasympathetic nervous system usually contributes to intraoperative penile erection, although local stimulation before complete sensory blockade may also lead to priapism.

Case Report: We discuss a case of successful management of priapism in 47-year-old Chinese male patient listed for transurethral resection of bladder tumor following a subarachnoid block.

Conclusion: With a detailed knowledge about the pathophysiology of intraoperative penile erection, anaesthesiologist can appropriately direct treatment depending on the individual patient and hence minimize the risks of subsequent complications.

Keywords: Priapism, spinal anaesthesia, glycopyrrolate, phenylephrine.

Introduction

Priapism is defined as a persistent penile erection unrelated to sexual excitation, which when left untreated for more than 4 h, will result in edema, risk of abrasion, tissue drying, and eventually, necrosis of the penis [1, 2]. The etiology of priapism can be primary, secondary, or idiopathic [3]. Psychogenic and reflex erections may occur during the early stages of neuraxial anaesthesia when the pathways involved are still incompletely blocked [4]. It is extremely troublesome for the surgeon to proceed with an operation in the presence of penile tumescence, as the likelihood of complications like urethral trauma, and hemorrhage is fairly high.

Case Report

A 47-year-old, 54.1 kg normotensive Chinese male, with the American Society of Anaesthesiologist physical status Class II, with a transitional cell carcinoma of urinary bladder, was scheduled for transurethral resection of bladder tumor (TURBT). The patient was taking fluoxetine for anxiety disorder. The patient had no history of back surgery or injury. Results of routine laboratory analysis, chest X-ray, and electrocardiogram were unremarkable. In the operating room, standard monitoring was instituted. The baseline blood pressure was recorded 120/58 mmHg and heart rate 68 bpm. The patient was given spinal anaesthesia at L3–4 interspace using 25 G Whitacre needle in a single attempt. 2.2 ml of bupivacaine 0.5% with dextrose was injected. On testing with 2.0 ml of bupivacaine 0.5% with dextrose and 10 µg fentanyl were injected intrathecally after the free flow of clear cerebrospinal fluid (CSF) was obtained. Even after 20 min, no motor or sensory block was elicited and a second attempt of spinal block at the same level using 27 g Whitacre needle was made. After second attempt, there was free flow of clear CSF, and 2.0 ml of bupivacaine 0.5% with dextrose was injected. On testing with an ice pack, a sensory level up to T10 dermatome was achieved. The patient was subsequently placed in lithotomy position for the surgery. The surgeon noted penile erection during insertion of the urethroscope and could not proceed further. The patient was sedated with propofol infusion at 20 mg/h and intravenous fentanyl 40 µg. Thereafter, intravenous glycopyrrolate 0.2 mg was given. The surgeon applied cold saline compress to the penis for a further 30 min, but the erection persisted. The surgeon then performed intracorporeal aspiration of blood from the penis. These measures took a total of 50 min and the penile erection finally resolved, allowing the surgery to be successfully accomplished.

Discussion

Priapism is defined as a persistent penile erection unaccompanied by sexual excitation. Sustained priapism, when left untreated for more than 4 h, will result in edema, tissue drying, and necrosis of the penis. The prognosis depends on the time elapsed before starting the therapeutic intervention. The etiology of priapism can be primary, secondary, or idiopathic.
Pathologic conditions causing priapism may be haematoletic such as sicle cell anaemia and polycythemia; traumatic such as pelvic trauma; surgical, neoplastic, and neurologic such as spinal cord injury; infective such as syphilis and urethritis; or pharmacologic such as antidepressants and antihypertensives [3]. Although spinal anesthesia interrupts sympathetic and parasympathetic innervations of the penis, erection may still occur. A possible explanation is a local stimulation from instrumentation of the penis in the presence of incomplete blockade of S2-S4 segments [5]. Psychogenic and reflex erections may also occur under neuraxial anaesthesia when the pathways are still incompletely blocked [4]. These mechanisms might have resulted in priapism in this patient. On a subsequent occasion, the patient was scheduled for TURBT, again experienced incomplete blockade after spinal anaesthesia given by an experienced senior anaesthetist at L4-5 interspace using 25 G Whitacre needle in one attempt. After ensuring free flow of CSF, 2.6 ml of bupivacaine 0.5% with dextrose and 10 µg fentanyl was injected intrathecally. The patient was still able to move both lower limbs with only mild numbness in legs. General anaesthesia was given in view of the partial block. Possible etiologies resulting in incomplete or failed spinal anaesthesia, despite confirmation of the spinal needle in the subarachnoid space with clear, free flowing CSF, include anatomical variations in the ligaments supporting the spinal cord, spinal stenosis, or adhesions within the vertebral canal from previous surgery [6]. These act as longitudinal or transverse barriers to the even spread of local anaesthetic drug. We decided not to investigate further for possible anatomic variations as therapeutic intervention was not appropriate given that he has no spinal symptoms and the only impact this has on him pertains only to the failed spinal anaesthetic for which he can be anaesthetised with general anaesthesia instead. The mechanism of penile erection involves the arterioles, venules, and arteriovenous channels [7]. Detumescence is mediated by the adrenergic stimulation causing constriction of penile venous sinuosoids, opening of emissary veins, and enhancing the blood drainage [8]. Regardless of etiology, intraoperative penile erection should be immediately addressed so as to prevent long-term sequelae such as thrombosis and fibrosis [1]. Various treatment modalities have been recommended for intraoperative penile erection [9]. Traditional methods include increasing the depth of general anaesthesia along with induced hypotension, dorsal nerve block, corporeal aspiration, with or without shunt procedures, and ketamine administration [1]. Induction of hypotension may be hazardous in elderly patients with coronary artery disease and may cause cardiac emergencies [10]. Ketamine has a dissociative effect on the limbic system which leads to penile relaxation. Although it is extensively used for this purpose, complete flaccidity takes a long time, averaging between 90 and 110 min. One major limitation is its propensity to cause hallucinations [8]. Intracorporeal injection of α-adrenergic agonists like phenylephrine 250 µg has been shown to produce rapid detumescence [11]. These activate adrenergic receptors, thereby decreasing blood supply and promoting blood drainage from the corpora cavernosa. Non-specific α-adrenergic agonists such as epinephrine possess additional β1 actions which may result in adverse effects such as hypertensive crisis or pulmonary edema [12]. We avoided intracorporeal injection as it could result in pain, hematoma, infection, or fibrosis. The use of the anticholinergic agent, glycopyrrolate, to treat intraoperative penile erection suggests a parasympathetic cholinergic etiology as well [5]. Glycopyrrolate being a quaternary ammonium compound is preferred in elderly patients as it has less adverse cardiovascular and central nervous system effects and hence tolerated better. Terbutaline, a β2 adrenergic agonist, 0.25–0.5 mg subcutaneous or intravenous, has proven to be effective for detumescence. It relaxes smooth muscles of cavernous tissue, arteries, veins, and tunica albuginea, thereby increasing the blood flow in venules and arteriovenous channels resulting in detumescence [13]. Several other methods like local application of ice pack or ethyl chloride spray have been recommended [14]. In our patient, the surgeon decided to aspirate blood from the corpora cavernosa after the failure of other measures. The success rate of intracorporeal aspiration is around 30%. Surgical shunts have a role mostly instituted when other methods fail. The aim is to provide a shunt between corpus cavernosum and glans penis, corpus spongiosum or a vein to bypass veno-occlusion [9].

Conclusion

With a better understanding of the pathophysiology of intraoperative penile erection, management can be appropriately tailored based on the patient’s profile. We emphasize early treatment as the duration of the erection is the critical factor for the successful detumescence of the penis and minimizing the risks of complications.

References

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