CASE REPORT

Priapism following spinal anesthesia in urological procedures

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ABSTRACT

Priapism following spinal anaesthesia for urological procedures is a rare occurrence .It is a troublesome complication which may pose a challenge to the urologist in proceeding with the surgery or even may result in cancellation of the elective procedure. We present a case of occurrence of priapism in a hypertensive patient posted for transurethral resection of prostrate following subarachnoid block and the way we managed it. Studies in the past two decades have mentioned various techniques of treating this intraoperative complication, e.g. the intracorporeal injection of vasopressors, subcutaneous or intravenous terbutaline and intravenous glycopyrrolate. In our case, we successfully dealt this complication with intravenous glycopyrrolate.

Key Words: Priapism; Spinal anesthesia; Phenylephrine; Glycopyrrolate

Citation: Natarajan P, Remadevi R., Rao PB, Ramachandran TR. Priapism following spinal anesthesia in urological procedures. Anaesth Pain & Intensive Care 2012;16(1):47-50.

INTRODUCTION

Priapism can be defined as a persistent penile erection unrelated to sexual excitation which when left unmanaged for more than four hours will result in edema, risk of abrasion , tissue drying and necrosis of penis.[1,2] The cause of priapism can be primary, secondary or idiopathic.[3] Priapism under spinal and epidural anaesthesia is reflexogenic, especially if the sympathetic blockade extends above the midthoracic level or it could be both reflexogenic and physhogenic.[1,4] The reflex stimuli may arise from the stimulation of the pudendal nerve (S2,3,4) by instrumentation before complete sensory blockade. We present a case of priapism after spinal anesthesia, which was successfully managed by us.

CASE REPORT

A 59 year old male was admitted to our hospital with benign prostatic hypertrophy for TURP surgery. Routine preoperative evaluation revealed that he was a known hypertensive for four years, controlled with Tab. amlodipine 5 mg once a day. Lab analysis showed normal blood and urine results. Chest X ray and ECG were also normal. He was given spinal anaesthesia with 25 G Quincke spinal needle at L3-4 space with 2.5 ml of bupivacaine 0.5% to achieve a sensory loss upto a level of T 10 dermatome.

The patient was positioned in lithotomy, prepped and draped. The surgeon passed the 26 F urethroscope through the urethra and within 5 minutes a severe rigid erection developed. The urologist could not proceed further hence the urethroscope was removed. We waited for nearly 30 minutes for spontaneous detumescence which did not occur. Inj glycopyrrolate 0.2 mg IV was given. Intracavernosal injection of phenylephrine was avoided because of his known hypertensive status. An option of perineal urethrostomy was given to patients

relatives for which they were not willing. The surgery was called off and patient was shifted to recovery room and monitored. He remained pain free throughout. Gradual spontaneous detumescence occurred over the following four hours. The patient was taken up for surgery the next day under general anaesthesia which was uneventful.

DISCUSSION

Priapism can be defined as a persistent penile erection unrelated to sexual excitation which when left unmanaged for more than four hours will result in edema, risk of abrasion , tissue drying and necrosis of penis.[1,2] The cause of priapism can be primary, secondary or idiopathic.[3] Priapism with primary etiology is not accompanied by a disorder responsible for a prolonged erection, may be of physical or psychological origin.[4] Secondary priapism is caused by factors directly or indirectly affecting the penile erection.[4] These may be hematologic e.g. sickle cell anemia, polycythemia, leukemia and coagulopathies; traumatic and surgical, e.g. spinal cord injury, penile trauma or pelvic/perineal trauma; neoplastic e.g. metastasis, myeloma, prostatic cancer or penile cancer;, neurologic e.g. herniated lumbar disc, multiple sclerosis or spinal cord tumors; infective e.g. prostatis, urethritis, syphilis, malaria or dibetes mellitus; or pharmacologic e.g. verapamil, nitroglycerine, heparin, haloperidol, prazosine and many more.[3] However, penile erection under spinal and epidural anaesthesia is reflexogenic, especially if the sympathetic blockade extends above the midthoracic level or it could be both reflexogenic and psychogenic.[1,4] The reflexogenic stimuli arise due to stimulation of the pudendal nerve (S2,3,4) with instrumentation before onset of complete sensory blockade. Another possible explanation is incomplete blockade of sacral segments of the spinal cord during spinal anaesthesia.[6,8] While it may be psychogenic being a result of exaggerated auditory sensation during second stage of anaesthesia.[4]

The mechanism of penile erection is a very complex phenomenon. In the flaccid state, the arterioles are partially closed, while the venules and the arteriovenous channels remain open, providing an unimpeded drainage of the arterial inflow.[1] Any reflexogenic or psychogenic stimuli will result in stimulation of sacral parasympathetic outflow, causing relaxation of the corporal arterioles and partial closure of the venules and arteriovenous shunts with subsequent engorgement of the corpora leading to erection.[1,7] The effects of the sympathetic and parasympathetic nervous system on the male sexual organ is complementary. Activation of the alpha 1 adrenergic receptors produces ejaculation while activation of the M3 cholinergic receptor type produces erection.

Normally the erection subsides after sympathetically mediated arteriolar constriction with the reduction of inflow and enhanced venous drainage.[1] Detumescence is mediated by the adrenergic stimulation that causes a constriction of penile venous sinusoids, opening emissary veins and thereby increasing blood drainage.[9]

Understanding the mechanism of erection has revolutionized the treatment of priapism. Various studies have described treatment options for intraoperative priapism.[11] Traditional methods include deepening the plane of general anaesthesia with a simultaneous induction of hypotension, dorsal nerve block, paralysis, corporeal aspiration with or without shunting procedures and ketamine administration.[1]

Induction of hypotension with sodium nitroprusside or deep general anaesthesia may result in lowering of arterial blood pressure in elderly patients with coronary artery disease and can precipitate a cardiac emergency.[12] The injection of 8 ml of 0.25% of bupivacaine into the subpubic space to block the dorsal nerve of penis has been found to be effective.[13,14] Ketamine has been frequently used to treat penile erection,[4,15-17] has a penile relaxing property probably secondary to its dissociative effect on the limbic system, however complete flaccidity occurred only after 25-110 min, representing a limiting factor.[18,19]

Aspiration with a non heparinised syringe into the base of one of the corpora cavernosa has a success rate of around 30%.[20] Aspiration can be combined with flushing the cavernosa with normal saline to clear the sludged blood.[11] Surgical shunts are done only when all the consecutive measures fail. The aim of the surgical treatment is to provide a shunt between corpus cavernosum and glans penis, corpus spongiosum or a vein so that the obstructed veno-occlusive mechanism is bypassed.[11] Shunt between corpora cavernosum and glans, such as the Winter's procedure, is reasonable initial procedure, although its success in maintaining detumescence has been questioned.[21] In failed and resistant cases, a more definitive shunt like the cavernospongiosum shunt can be performed which has a success rate of around 75%.[20,22,23]

Intracorporeal injection of phenylephrine 250

micrograms has been recommended by certain authors. Detumesence occurred rapidly in all patients with a single injection.[24,25] These agents produce detumescence by decreasing the blood supply to or increase blood drainage from the corpora cavernosa through activation of the adrenergic receptors. The pure alpha 1 agonistic activity lacks adverse cardiac effects such as hypertensive crisis or pulmonary edema.[26] This makes it a safer drug when compared to epinephrine, norepinephine, metaraminol which has additional beta 1 action responsible for the adverse systemic and cardiac effects.[1]

The use of terbutaline subcutaneously or intravenously (0.25-0.5 mg) have been recommended by certain authors. It is thought to relax the entire smooth muscle of the corpora cavernosum resulting in flaccidity of the entire penis and relaxation of the tunica albugenia, thereby increasing the blood flow in the venules and arteroivenous channels and produces detumescence.[4] Injection of intracavernosal phenylephrine may cause pain, hematoma, infection, fibrosis of the penis and accidental intravenous injection may cause a severe change in the hemodynamic status of the patient, the reason for which the drug was avoided in our patient and glycopyrroate was used.

The use of intravenous glycopyrrolate to treat intraoperative penile erection in patients receiving continuous spinal anaesthesia suggests a parasympathetic cholinergic etiology.[28] The authors who studied the use of glycopyrrolate have found it to be a safe drug that can be used in patients with coronary artery disease or in situations where cardiovascular stability is deserved.[28] This was the reason that we preferred to use this drug in our patient, with complete but slow positive effect.

CONCLUSION

Although intropeative priapism is an unusual condition, it warrants serious and urgent attention. Therapy should be quickly initiated to enhance the venous drainage of the engorged corpora cavernosa, otherwise prolonged venous stasis leads to viscosity and sludging, that may result in irreversible impairment of the various routes of venous return.

Glycopyrrolate produced detumesence due to its anticholinergic property, which may be the etiology in the above case, and it proved to be safe and reliable even in a hypertensive patient who encountered priapism following spinal anesthesia.

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BOOK REVIEW

Handbook for Anesthesia Technicians

Department of anesthesia, Agha Khan University Hospital (AKUH) Karachi has pioneered in producing a very handy, informative and easy to understand handbook for anesthesia technicians. The first edition of this book has received a highly favourable acclaim from the anesthesia community of the country, and it has fulfilled a long standing need of technicians for an introductory, concise book covering the core elements of anesthesia practice. The book is edited jointly by Dr. Robina Irshad Khan and Samuel Nawab and comprises of chapters on applied basic sciences, basics of anesthesia as well as specialty anesthesia including pediatric anesthesia, thoracic anesthesia, cardiovascular anesthesia, neuro anesthesia and regional anesthesia. Dedicated chapters have been included on pain management, resuscitation and medical ethics. The book consists of 23 chapters, each written by a member of the faculty of AKUH.

The book is printed on good quality white paper and a number of full colour illustrations and photographs supplement the text throughout. The book has been published and distributed on a non-profit basis, that's why it is nominally priced at Rs. 120.00 only. It is highly recommended source of contemporary anesthesia knowledge for every anesthesia technician.