ISSN 2515-8260 Volume 09, Issue 02, 2022

Priapism presenting in acute spinal cord injury

[1]Dr. Kumar Lakshman, [2]Dr. Triza Kumar Lakshman, [3]Dr. Raju Paul Manjooran, [4]Dr. Dominic Anto, [5]Dr. Abhishek Vijayan, [6]Dr. Rajeev Aravindakshan

[1]Assistant Professor, Department of Neurosurgery, Adichunchanagiri Institute of Medical Sciences, BG Nagara, Mandya, Karnataka, India
[2]Associate Professor, Department of OBG, Adichunchanagiri Institute of Medical Sciences, BG Nagara, Mandya, Karnataka, India
[3,4]Professor, Department of Neurosurgery, Pushpagiri Institute of Medical Sciences, Thiruvalla, Kerala, India
[5]Assistant Professor, Department of Neurosurgery, Government Medical College, Thiruvananthapuram, Kerala, India
[6]Professor, Department of Community Medicine, All India Institute of Medical Sciences, Mangalagiri, Andhra Pradesh, India

Corresponding Author:
Dr. Kumar Lakshman

Abstract

Background: The frequency and timing of priapism in acute spinal cord injury (SCI) and the level, or levels, most likely to be associated with priapism are debated.
A spinal cord injury (SCI) is defined as damage to the spinal cord caused by an insult resulting in the transient or permanent loss of usual spinal motor, sensory, and autonomic function.
Methods: This is prospective observational study done in PES Institute of Medical Sciences, Kuppam, from February 2019 to February 2020. A total of 15 cases were studied. The patients selected for study are those who presented with history of cervical /thoracic spine injury with complete neurological deficits, following trauma. Based on detailed history and thorough clinical examination diagnosis of priapism in spinal cord injury was made. **Investigations done:** Complete Blood counts, Blood grouping, Coagulation profile Peripheral Smear, Renal function tests, Serology, Corporeal Blood gas analysis, CT Cervical/thoracic spine, MRI cervical spine with whole spine screening.
Results: A total of 15 patients who presented with spinal column injuries with neurological deficits Quadriplegia or paraplegia, following trauma were included in the present study. Eleven of the 15 patients had priapism. Four out of the 15 patients presented with quadriplegia but without priapism. Among the 11 patients, 4 patients presented with Paraplegia with priapism secondary to complete cord transection of the lower cervical/high thoracic cord. Seven patients presented with quadriplegia and priapism secondary to complete cord transection of the cervical cord. All 15 patients underwent spine stabilization surgery along with surgical decompression of the spinal canal and conservative management for priapism. In 6 patients, the priapism subsided spontaneously within 12hours, in two pts it subsided within 24hrs for in two pts it lasted for 36 hours while in one patient it lasted for 4 days post spinal canal decompression.
Conclusion: Most common site of SCI leading to Priapism in our study were at cervical & high Thoracic cord with complete cord transaction. However it may be concluded that

priapism is not seen in all cases of complete cord transection and never seen in patients with paresis.

Keywords: SCI, priapism, acute spinal cord injury

Introduction

Priapism is persistent erection of the penis [1]. The term priapism was derived from the Greek god Priapus, son of Aphrodite. His father was Zeus, and when Hera, the wife of Zeus, heard of the pregnancy she cursed the child, such that, when the boy was born with oversized genitals, he was rejected by Aphrodite. Priapus was then brought up by shepherds who noticed that in his vicinity, flowers would bloom and animals would copulate furiously. He was thus made a god of fertility and his giant phallus was made a symbol of power [2, 3].
Priapism has been reported in the ancient papyrus of the Pharaonic Egypt and pescriptions for its treatment are found in Ebers Papyrus [4]. The earliest record of priapism in modern literature was by Petraens in 1616, in an article entitled "Gonorrhoea, Satyriasis et Priapisme" [5] and the first account of priapism appearing in the English literature was by Trife in 1845 [6]. There were subsequent isolated case reports of this mysterious illness and the various unsuccessful attempts at management. In 1914, Frank Hinman published his seminal article on the pathophysiology of this unique condition, and his work was carried on by his son who postulated that venous stasis, combined with increased blood viscosity and ischaemia, played an important part in its development.

Epidemiology and Aetiology

Approximately 14% of vertebral column fractures result in damage to the cord. Of these, 50% will be incomplete, of whom 50% will walk by the time they leave hospital, if managed appropriately [1, 7].
Injury to the spinal column (fractures and/or dislocations) may be associated with immediate SCI, incomplete or complete. In others there may be injury to the spinal column, which is not associated with any neurological deficit. Patients with potentially unstable injuries to the spinal column must be transferred, transported and examined with in-line spinal immobilisation to prevent excessive movement at the level of an unstable injury. If excess movement at the level of an unstable fracture or dislocation is not prevented, there can be secondary damage to the spinal cord, which could lead to the patient with no initial SCI developing an incomplete or complete SCI that was potentially preventable (or a patient with an incomplete SCI becoming complete). Secondary deterioration of neurological function in patients with SCI is uncommon, occurring in about 4% of patients [8, 9]. Some men with acute SCI never develop priapism [9].

Methodology

1. **Study design:** This is prospective observational study done in PES institute of medical sciences, Kuppam.
2. **Study setting:** PES institute of medical sciences, Kuppam
3. **Study population:** The patient selected for study are those who presented with history of cervical spine injury following road traffic accidents/fall from height.
4. **Sampling method:** Purposive sampling.
5. **Sample size:** A total of 15 cases were studied.
6. **Inclusion criteria:** All male patients presenting to ER with history of trauma to spine and presenting with acute onset of quadriplegia/paraplegia
7. **Exclusion criteria**

A) Female patients

ISSN 2515-8260 Volume 09, Issue 02, 2022

B) Pre-existing penile deformity,
C) Pre-existing impotence due to any cause.
D) Traumatic brain Injury causing neurological deficit
E) Age <18yrs or >60yrs
F) Patients presenting to ER with history of trauma to spine and prior history of Sickle cell Anemia,
G) History of previous penile surgery
H) Patients presenting with previous history of spine surgery

8. **Study tools:** The patient selected for study are those who presented with history of spine injury (cervical/Thorax) following trauma (road traffic accidents/Fall from height). Based on detailed history and thorough clinical examination diagnosis of priapism in spinal cord injury is made.

Investigations done: Complete Blood counts, Blood grouping, Coagulation profile Peripheral Smear, Renal function tests, Serology, Corporeal blood gas analysis.

CT Cervical/thoracic spine

MRI cervical spine with whole spine screening.

9. Method of collection of data

Detailed history, thorough clinical examination and diagnosis of spinal cord injury was made. Presence or absence of priapism in patients with neurological deficits were noted and subsequently followed up for its subsidence.

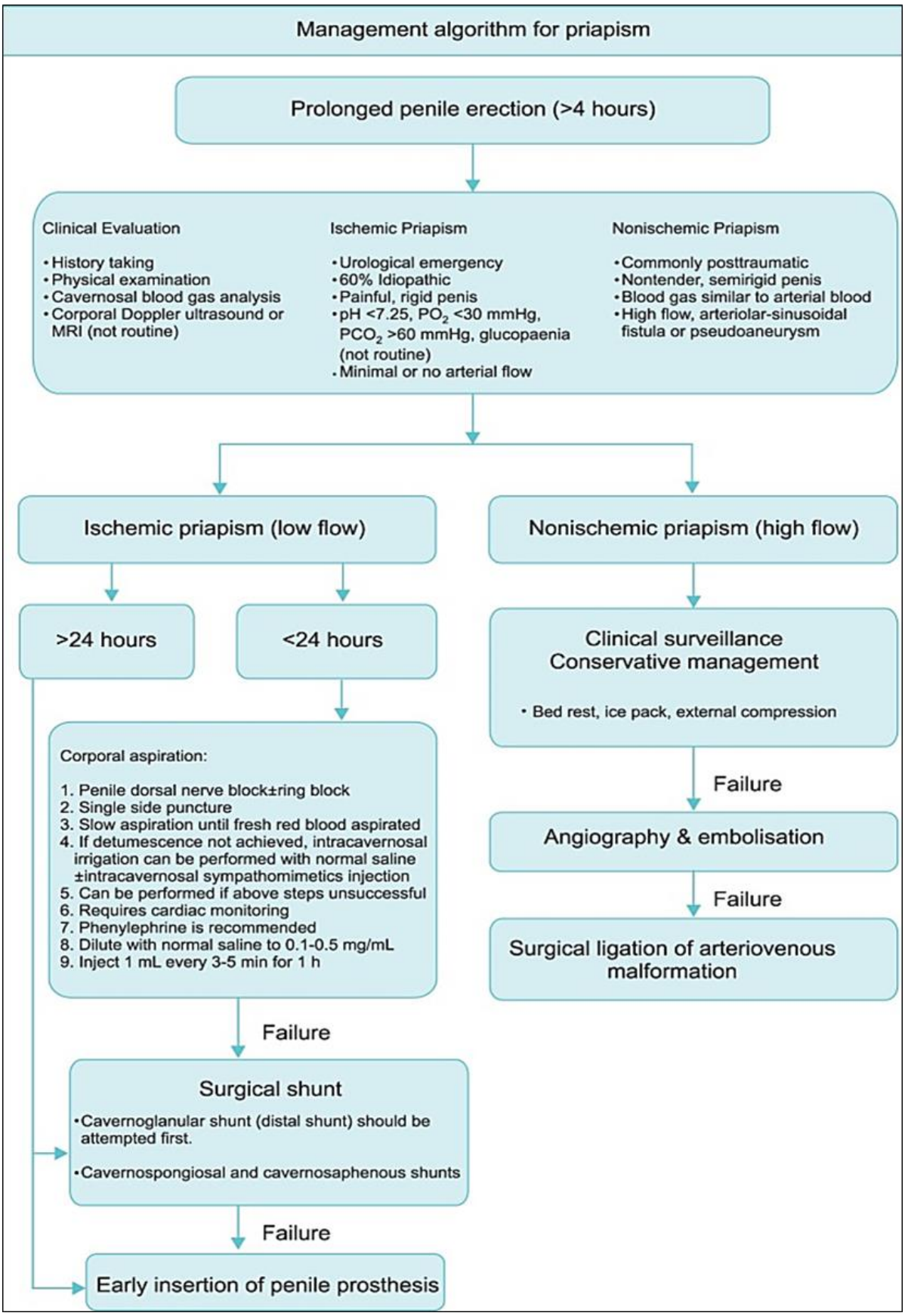
10. Statistical analysis of data

Of the 15 patients with complete motor deficit (quadriplegia/paraplegia), eleven patients presented with priapism. All 15 patients underwent elective spinal stabilization with decompression of the spinal canal. In 6 patients, the priapism subsided spontaneously within 12hours, in two pts it subsided within 24hrs, in two pts it lasted for about 36 hours while in one patient it lasted for 4 days post spinal canal decompression. Four of the 15 patients presented with quadriplegia but without priapism.

Sl. no	Age	Mode of injury	Duration of presentation to ER after RTA	Upper limb power	Lower limb power	Level of sensations	Priapism at the time of presentation	Type of Priapism (Ischeamic/ Non ischeamic	American Spinal Injury Association (ASIA) Impairment Scale (AIS)	Level of Injury	Outcome
1	28yrs	RTA	3hours	Bilateral 3/5	Bilateral 0/5	Absent below the level of nipples	present	Non Ischeamic	A	C6 #	Priapism resolved spontaneously after 4 days
2	31yrs	RTA	10 hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	C4#	Priapism resolved spontaneously within 4hrs
3	24yrs	RTA	3hours	Bilateral 5/5	Bilateral 0/5	Absent below the level of nipple	present	Non Ischeamic	A	C7#	Priapism resolved spontaneously within 36hrs

4	19yrs	Fall from height	6 hours	Bilateral 5/5	Bilateral 0/5	Absent below the level of sternal notch	present	Non Ischeamic	A	T1#	Priapism resolved spontaneously within 24hrs
5	22yrs	Fall from height	12 hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	C4#	Priapism resolved spontaneously within 10hrs
6	46yrs	RTA	4 hours	Bilateral 5/5	Bilateral 0/5	Absent below the level of nipples	present	Non Ischeamic	A	T2#	Priapism resolved spontaneously within 12hrs
7	38yrs	RTA	2 hours	Bilateral 5/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	T1#	Priapism resolved spontaneously within 8hrs
8	22yrs	Fall from height	7 hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	C6#	Priapism resolved spontaneously within 36hrs
9	34yrs	RTA	6 hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	C5#	Priapism resolved spontaneously within 10hrs
10	19yrs	Fall from height	5 hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	C4#	Priapism resolved spontaneously within 12hrs
11	21yrs	Fall from height	2 hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	present	Non Ischeamic	A	C5#	Priapism resolved spontaneously within 24hrs
12	32yrs	RTA	48hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	Absent		A	C4#	
13	29yrs	RTA	36hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	Absent		A	C5#	
14	19yrs	Fall from height	10hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	Absent		A	C3#	
15	35yrs	Fall from height	28hours	Bilateral 0/5	Bilateral 0/5	Absent below the level of neck	Absent		A	C5#	

ISSN 2515-8260 Volume 09, Issue 02, 2022



ISSN 2515-8260 Volume 09, Issue 02, 2022

The ASIA impairment scale (modified from the Frankel Classification)	This is an internationally recognized scale to classify the clinical extent of SCI. Impairment is classified from A–E, the estimated. Incidence of each type of injury in brackets and bold.
A-Complete	No sensory or motor function is preserved in the sacral segments S4-S5 (45%).
B-Incomplete	Preservation of sensory but not motor function below the neurological level and includes the sacral segments S4-S5 (15%).
C-Incomplete	Preservation of motor function below the neurological level. More than half of key muscles below the neurological level have a muscle. Grade of 3 (10%).
D-Incomplete	Preservation of motor function below the neurological level. More than half of key muscles below the neurological level have a muscle. Grade of 3 (30%).
E-Normal	Sensory and motor function is normal.
Neurological level	The level of a SCI is the lowest level of the spinal cord with normal sensation and motor function on both sides of the body.
Tetraplegia	This is attributable to a lesion or injury within the cervical spinal cord. There is incomplete or complete loss of motor (sensory functioning the arms, the torso, pelvic organs, and the legs).
Paraplegia	This is attributable to a lesion or injury within the thoracic, lumbar, or sacral spinal cord. There is incomplete or complete loss of motor. (Sensory function of the torso, pelvic organs, and the legs). The level of injury will affect which of these are affected. Arm function is preserved.
Anterior spinal artery syndrome	The anterior spinal artery runs as a single artery anterior to the cord and supplies the anterior 2/3 of the cord. Transection therefore produces sparing of the dorsal columns (Figs 3 and 4), resulting in paralysis and loss of pain and temperature with preservation of proprioception, fine touch and vibration.
Brown-Sequard syndrome	This is caused by lateral cord damage. It may occur because of osteophyte impaction on half of the cord producing sensorimotor damage at the level of the injury. There is ipsilateral loss of motor function, fine touch, proprioception and vibration and contralateral loss of pain, and temperature below this level.
Cauda equina syndrome	Bladder and bowel dysfunction associated with upper motor neurone symptoms and signs in the legs caused by injury to the lumbosacral nerve roots.
Central cord syndrome	Results from bleeding, infarction, or oedema to the central grey matter of the spinal cord. This is most common in the cervical region where it presents as upper motor neurone signs in the legs and mixed upper and lower motor signs in the arms with loss of pain and Temperature sensation in the arms. Sacral nerve fibres are positioned laterally in the cord and the patient may demonstrate sacral Sparing of sensation. This indicates incomplete cord damage and therefore offers the theoretical chance of some recovery of the cord.
Posterior cord syndrome	Produces loss of vibration and proprioception. This is associated with damage to the posterior spinal artery and is very rare.

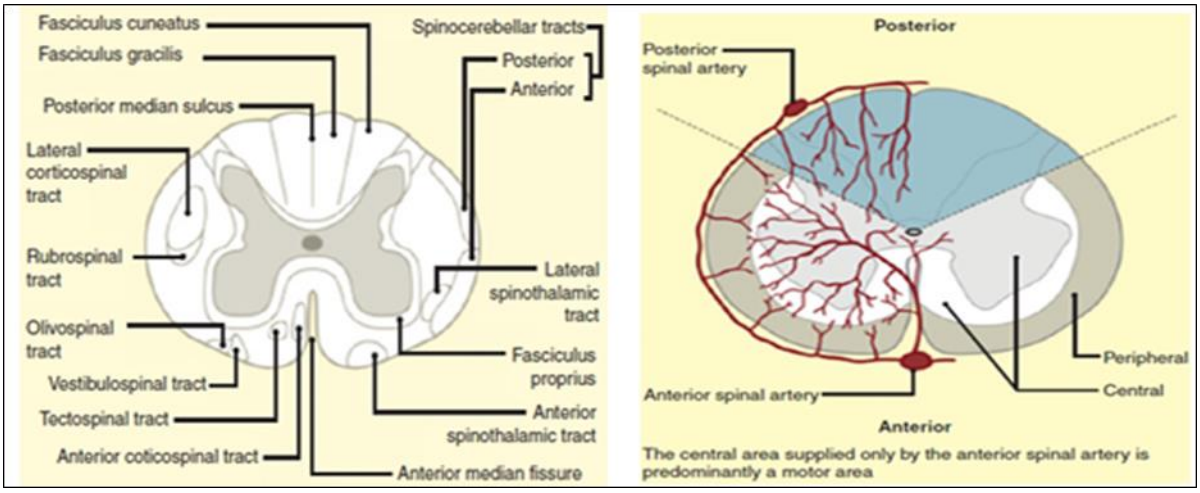
Definitions and clinical syndromes associated with SCI that have been agreed by the International Standards for Neurological and Functional Classification of SCI. ASIA, American Spinal Injury Association.

Discussion

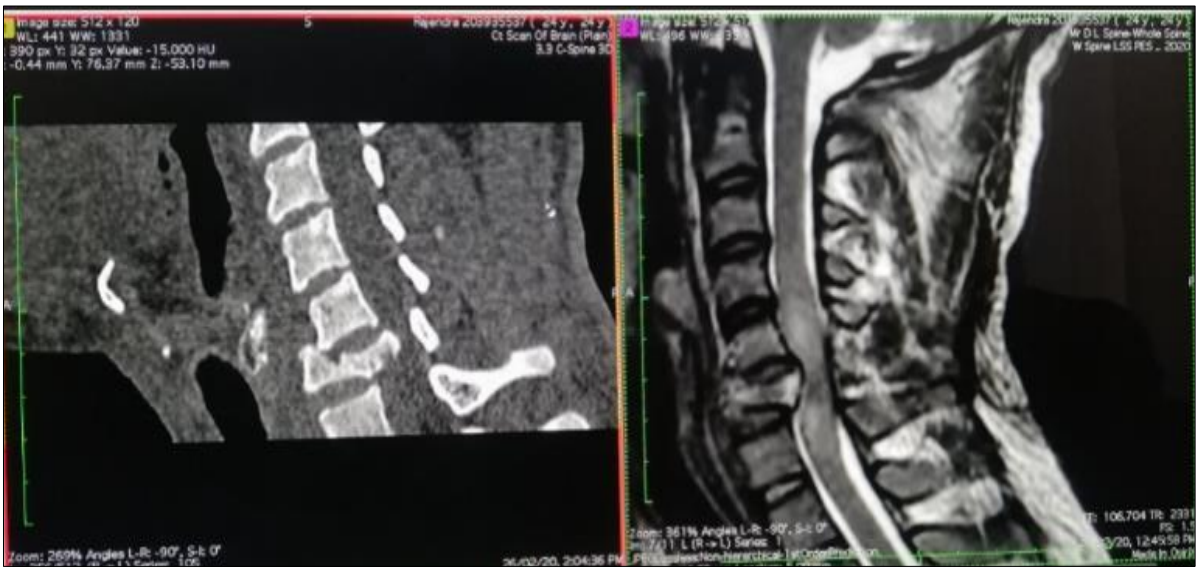
Anatomy

The spinal canal contains the spinal cord in a potential space filled with epidural fat and blood vessels. The size of this potential space varies considerably along the length of the canal. It is narrowest in the mid-thoracic region and unstable injuries here easily impact onto the cord, particularly retropulsion of bone fragments, leading to a high chance of complete cord injury [7].

ISSN 2515-8260 Volume 09, Issue 02, 2022



(A)Schematic diagram showing the arrangement of spinal tracts and (B) blood supply of section of spinal cord



(A) CT of cervical spine showing C5/6 bilateral facet displacement with disc disruption and extensive soft tissue disruption. Posterior displacement fracture fragment of C6 vertebrae is demonstrated. (B) MRI of the same patient showing marked spinal stenosis due to impingement of the posterior fracture fragment on the thecal sac there is cord swelling and contusion and a focal intramedullary haematoma at the level of C6/7, the cord oedema extends form C4/5 to C7.

The anterior spinal artery is particularly vulnerable to retropulsion of bone and disc fragments and may give increase to cord infarction in watershed areas, particularly at C6/7 level. Classification of priapism: Congenital and acquired Priapism. Acquired has been further subdivided into two types: (a) low-flow (ischaemic) or (b) high-flow (non-ischaemic) [10]. Low-flow priapism is more common; typically the penile shaft is firm, the glans penis is soft and the priapism is painful [11]. Congenital neonatal priapism is also a recognised clinical entity. Although most cases are idiopathic, birth trauma resulting from forceps delivery, respiratory distress syndrome, umbilical artery catheterisation, polycythaemia, and congenital syphilis are other known causes. Most of the reported cases of congenital priapism have been successfully treated conservatively, although the erectile dysfunction in the adulthood has not been assessed. Idiopathic priapism is used to classify those without a known cause and it is thought to be precipitated by a normal penile erection, sexual stimulation, or prolonged sexual activity. Interestingly, before the introduction of intracavernous injection of vasoactive drugs for erectile dysfunction in 1984, most patients did not have a known cause of priapism and a third of these were classified as idiopathic.

ISSN 2515-8260 Volume 09, Issue 02, 2022

Mechanisms

A neuronal as well as disregulatory basis to priapism has been proposed [12]. Neurogenic priapism is seen in patients with spinal cord injury [13], cauda equina compression syndrome [14], and for centuries has been noticed in the victims of hanging [15].

It is assumed that the mechanism of priapism in most patients with SCI is that abrupt loss of sympathetic input to the pelvic vasculature leads to increased parasympathetic input and uncontrolled arterial inflow directly into the penile sinusoidal spaces [7].

The parasympathetic nuclei in the sacral erection center are activated and erection is achieved through the cavernous nerves when tactile stimulation of the genital organs are transmitted by pudendal nerves. Psychogenic stimulation results from audio-visual or imaginary stimuli and depends on the modulation of the spinal erection centers (T11-L2 and S2-S4). In patients with spinal cord lesion above the T9 level, psychogenic erection is usually absent. When the lesion occurs at the sacral level the psychogenic erection component is preserved but the reflex mechanism is not [13].

The sympathetic outflow arises from the thoracolumbar spine, that is, the spinal cord from approximately T2 to the conus (L1-2). The sympathetic outflow to the penis and clitoris arises from the lowest levels of the spinal cord, the conus, which is the reason why a lesion at any level in the spinal cord from the brainstem to the conus can be associated with priapism [7].

The finding that sympathetic nervous system plays an important part in normal detumescence and the activation of neural reflex mechanisms during erections suggest a possible pathogenic role for the nervous system in priapism [16, 17].

Priapism of the clitoris, although much rarer than its male counterpart, has been reported sporadically in the literature. It is commonly associated with drugs like trazadone, citalopram, bromocriptine, olanzapine [18], and fluoxetine [19], pelvic malignancies, blood dyscrasias, or retroperitoneal fibrosis [20]. Priapism has also been reported following spinal shock, that is, it has been reported in what initially appeared to be a complete spinal cord lesion; the spinal cord lesion resolved [21].

In all 11 patients who presented with priapism following traumatic spinal cord injury were of Non Ischemic type as analyzed by their corporeal blood gas analysis. In six patients it subsided spontaneously (with bed rest/Ice pack/external compression) within 12 hrs. while in 2 patients it lasted for 24hrs. In three patients an additional corporeal aspiration was performed to facilitate detumescence and the priapism resolved within 36 hrs. in two out of the three patients while in one patient it persisted for 4days during which 3 doses of phenyl ephrine 100mg was instilled after corporeal aspiration and the patient finally had detumescence. We attribute that in 3 patients in whom the non-ischemic priapism lasted for more than 36 (up to 96hrs in one patient) had a component of spinal shock resulting in combination of non ischeamic priapism with ischeamic priapism like picture.

Spinal shock is the loss of reflexes below the level of SCI resulting in the clinical signs of flaccid areflexia and is usually combined with hypotension of neurogenic shock. There is a gradual return of reflex activity when the reflex arcs below redevelop, often resulting in spasticity, and autonomic hyperreflexia. This is a complex process and a recent four-phase classification to spinal shock has been postulated: areflexia (Days 0-1), initial reflex return (Days 1-3), early hyperreflexia (Days 4-28) and late hyperreflexia (1-12 months) [1].

Results

A total of 15 patients who presented with spinal column injuries with neurological deficits Quadriplegia or paraplegia, following trauma were included in the present study.

Eleven of the 15 patients had priapism. Among the 11 patients, 4 patients presented with

Paraplegia with priapism secondary to complete cord transaction of the lower cervical/high thoracic cord. Seven patients presented with quadriplegia and priapism secondary to complete cord transaction of the cervical cord. All 11 patients underwent spine stabilization surgery along with surgical decompression of the spinal canal and conservation management for priapism. In 6 patients, the priapism subsided spontaneously within 12hours, in two pts it subsided within 24hrs for in two pts it lasted for 36 hours while in one patient it lasted for 4 days post spinal canal decompression. Four out of the 15 patients presented with quadriplegia but without priapism.

Conclusion

Priapism occurs in a proportion of men with acute traumatic SCI. The proportion of men with acute SCI that develop priapism is not known. Priapism can be associated with injury to any part of the spinal cord from the foramen magnum to the conus. A turgid semi-erect or erect priapism is always associated with a complete (American Spinal Injury Association A) motor and sensory paraplegia. Priapism probably occurs at the moment of, or very shortly after, complete SCI. This is high-flow (arterial) priapism. The priapism settles spontaneously usually within a few hours, occasionally up to few days after the SCI. It, only, rarely requires medical treatment.

It may be concluded that priapism is not seen in all cases of complete cord traction and never seen in patients with paresis.

We require much larger case series to understand the pathophysiology and management of non-ischemic priapism.

References

1. Bonner S, Smith C. Initial management of acute spinal cord injury. Contin Educ Anaesth Crit Care Pain. 2013 Dec;13(6):224-31.
2. Papadopoulos I, Kelâmi A. Priapus and priapism. From mythology to medicine. Urology. 1988 Oct;32(4):385-6.
3. Hodgson D. of gods and leeches: treatment of priapism in the nineteenth century. J R Soc Med. 2003 Nov;96(11):562-5.
4. Shokeir AA, Hussein MI. The urology of Pharaonic Egypt. BJU Int. 1999 Nov;84(7):755-61.
5. Hinman F. priapism: report of cases and a clinical study of the literature with reference to its pathogenesis and surgical treatment. Ann Surg. 1914 Dec;60(6):689-716.
6. Tripe J. Clinical contributions from private practice: Case of continued priapism. The Lancet. 1845 Jul 5;46(1140):8-9.
7. Song P, Moon K. Priapism: Current Updates in Clinical Management. Korean J Urol. 2013 Dec 1;54:816-23.
8. Todd NV. Priapism in acute spinal cord injury. Spinal Cord. 2011 Oct;49(10):1033-5.
9. Cherian J, Rao AR, Thwaini A, Kapasi F, Shergill IS, Samman R. Medical and surgical management of priapism. Postgrad Med J. 2006 Feb;82(964):89.
10. Sadeghi-Nejad H, Seftel AD. The etiology, diagnosis, and treatment of priapism: review of the American Foundation for Urologic Disease Consensus Panel Report. Curr Urol Rep. 2002 Dec;3(6):492-8.
11. Todd NV. Priapism in acute spinal cord injury. Spinal Cord. 2011 Oct;49(10):1033-5.
12. Burnett AL. Pathophysiology of priapism: dysregulatory erection physiology thesis. J Urol. 2003 Jul;170(1):26-34.
13. Utida C, Truzzi JC, Bruschini H, Simonetti R, Cedenho AP, Srougi M, *et al.* Male infertility in spinal cord trauma. Int Braz J Urol. 2005 Aug;31(4):375-83.

14. Ravindran M. Cauda equina compression presenting as spontaneous priapism. J Neurol Neurosurg Psychiatry. 1979 Mar;42(3):280-2.
15. Gallagher JP. A lesson in neurology from the hangman. JSC Med Assoc 1975. 1995 Jan;91(1):38.
16. Levine JF, Saenz de Tejada I, Payton TR, Goldstein I. Recurrent prolonged erections and priapism as a sequela of priapism: pathophysiology and management. J Urol. 1991 Apr;145(4):764-7.
17. Chin JL, Sharpe JR. Priapism and anesthesia: new considerations. J Urol. 1983 Aug;130(2):371.
18. Bucur M, Mahmood T. Olanzapine-induced clitoral priapism. J Clin. Psychopharmacol. 2004 Oct;24(5):572-3.
19. Cc BM, Wl D, Pj B. Nefazodone-induced clitoral priapism. Int. Clin. Psychopharmacol. 1999 Jul;14(4):257-8.
20. Guven EO, Kilciler M, Bedir S, Avci A, Ozgok Y. Transitional cell carcinoma of the clitoris: direct implantation or metastasis. Int. Urol. Nephrol. 2007 Sep;39(3):829-31.
21. Spinal Cord Injuries: Practice Essentials, Background, Anatomy. 2022 Mar 3 [cited 2022 Mar 20]; Available from: https://emedicine.medscape.com/article/793582-overview