Functional medicine

An uncommon association with sub-arachnoid hemorrhage in a young man

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Introduction

Priapism is a relatively uncommon condition, with an incidence rate of 1.5 per 100,000 person-years amongst the general population.1 Hence, most healthcare professionals are unfamiliar with this condition.

Key to the evaluation of priapism is to define whether it is ischemic or non-ischemic in nature. Non-ischemic priapism is commonly due to trauma, such as genitourinary injury, leading to uncontrolled cavernous arterial inflow. It is not an emergency and chance of spontaneous resolution is high. Ischemic priapism, on the other hand, occurs commonly due to veno-occlusive causes leading to outflow obstruction, culminating in compartment syndrome of the penis. Ischemic priapism should be treated promptly as a delay in treatment could result in irreversible fibrosis of the corporal tissue.

We describe a case of ischemic priapism presenting in a gentleman with a subarachnoid hemorrhage. Early recognition of this state as a urological emergency is essential to avoid cavernosal fibrosis and the eventual catastrophe of erectile dysfunction.

Case presentation

A 38 year old teacher with no significant past medical history presented with a dissecting right vertebral artery aneurysm, for which he underwent endovascular coiling of the aneurysm and stenting of the right posterior inferior cerebellar. He was subsequently noted immediately post-procedure to have a persistent erection and was referred to the urologist. A collaborative history obtained from the patient's wife revealed that the former did not have any known or family history of haematological dyscrasias or metabolic disorders, nor past history of priapism. The patient also did not use any long-term medications or any illicit drugs including cocaine.

Physical examination showed a fully rigid corpora cavernosa, glans and spongiosum. The endovascular procedure was performed 2 hours prior to urologist review and there were no signs of peri- neal trauma or abdomino-pelvic malignancies. There were also no immediate complications arising from the right femoral artery cannulation for the endovascular procedure.

A full blood count revealed an elevated total white count but a normal haemoglobin level of 14.7 g/dl and reticulocyte count. Cavernous blood aspiration demonstrated ischemic priapism, with a PO2 of 62 mmHg, pCO2 of 66 mmHg and pH of 7.19. The corresponding arterial blood gas showed a pO2 of 222.4 mmHg, pCO2 of 31.6 mmHg and pH of 7.46.

A colour duplex ultrasound of the penis was performed, which demonstrated a patent dorsal vein of the penis with high-resistance inflow within the cavernosal arteries with evidence of diastolic flow reversal. The resistive indices were elevated, measuring up to 1.0. This picture of high resistance within the penis was concordant with the blood gas analysis showing ischemic priapism, seen on the cavernosal aspiration.

As the patient was sedated prior to the procedure with propofol and fentanyl, the initial treatment did not include administration of a dorsal penile block. A total of 300ml of blood was aspirated and 1mg of phenylephrine administrated but the priapism persisted.

Subsequently, the decision was made to give a dorsal nerve
block in preparation for a possible distal corpora-glanular shunt. The penile block was administered with 10ml of 1% Lignocaine. We repeated the intracavernosal blood aspiration, removing a further 120ml of blood, with successful cessation of the priapism.

**Discussion**

Priapism is a pathological penile erection that persists for more than 4 hours and is beyond or not related to sexual stimulation. We report the rare presentation of priapism in a young man who underwent endovascular coiling for his ruptured vertebral artery aneurysm.

Takaku et al. reported a case of stuttering priapism in a man after 3 episodes of subarachnoid haemorrhage (SAH), hypothesizing that the priapism was the result of hypothalamic injury. The paraventricular nucleus of the hypothalamus has been shown to be instrumental in the regulation of erectile function. This area which contains oxytocinergic neurons is susceptible to activation by nitric oxide, which is vital in the regulation of cerebral blood. In our patient, the endovascular procedure may have resulted in a parasympathetic activation leading to neurogenic priapism.

Neurogenic priapism associated with spinal cord injury is classically high flow, due to increased parasympathetic stimulation and increased afferent blood flow. Although our patient presented with ischemic priapism, the high peak systolic velocity alluded to a possibility that there could be high-flow priapism which subsequently built up to a compartment syndrome picture whereby the penile vasculature inflow became greater than what the efferent vasculature could support. This is also consistent with the patient's intracavernosal pO2 levels, which is higher than what we would expect of a pure low-flow priapism. In contrast, the classic appearance of low-flow priapism on penile Doppler sonography involves low cavernosal blood flow with a high-resistance, low-velocity trace. However, our patient's peak systolic velocity of 32.0cm/s and end-diastolic velocity of 2.06cm/s were characteristic of a high flow etiology. Wu et al. also made a similar observation that high-flow priapism secondary to a neurogenic cause could become ischemic when left untreated.

Intra-operative erections have been treated successfully with the administration of a dorsal penile block. The combination of intracavernosal aspiration and dorsal penile block also proved to be effective in the management of our patient's ischemic priapism. Lidocaine is an amide anaesthetic, which blocks fast voltage-gated sodium channels in the cell membrane of postsynaptic neurons. Dorsal nerve blocks have been shown to abolish reflex erections, hence it is likely that the lignocaine interrupted the afferent excitatory drive, hence discontinuing the high arterial influx of blood.

Whilst distinction of the cause of the priapism is important, it is perhaps secondary to the recognition of ischemic versus non-ischemic priapism. We hereby propose close observation with a view for early treatment of neurogenic priapism, whilst traditionally associated with high flow ischemia, resulted in ischemic priapism in this case.

**Conclusion**

Cerebrovascular accidents are rarely associated with priapism. In an unconscious or sedated patient who is unable to express himself, it must be identified early by the vigilant healthcare worker. High flow priapism may eventually result in ischemia, hence close observation with a view of early intervention is advised. Dorsal penile nerve blocks should be administered in patients with priapism which have a neurogenic origin regardless of conscious status.

**Consent**

Informed consent was obtained from patient.

**Conflict of interest**

The authors declare that they have no conflict of interest.

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