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Protracted heparin-induced priapism after myocardial infarction: a case report

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Abstract

Introduction. Priapism is a condition that often requires emergency treatment to spare erectile function.

Case report. This is a case report on a patient from the Lithuanian University of Health Sciences Kaunas Clinics with a very rare cause of priapism. The 41-year-old patient was hospitalized with a diagnosis of myocardial infarction, which was being treated with stent insertion, followed by conservative therapy with antiaggregants and heparin. A few hours later after the initiation of the treatment, a genitourinary examination revealed a rare case of heparin-induced priapism. The condition was treated conservatively with multiple cold and mechanical pressure applications, followed by blood aspirations, which led to surgical shunt insertion due to ineffective outcomes.

Conclusion. Although the only known pathogenetic mechanism of heparin-induced priapism is associated with heparin-induced thrombocytopenia, the patient's blood test didn't reveal any abnormalities so the real pathogenetic mechanism of this priapism case remains unclear. Clinicians should acknowledge the possibility of priapism development after treatment initiation with heparin and inform their patients about this rare adverse effect.

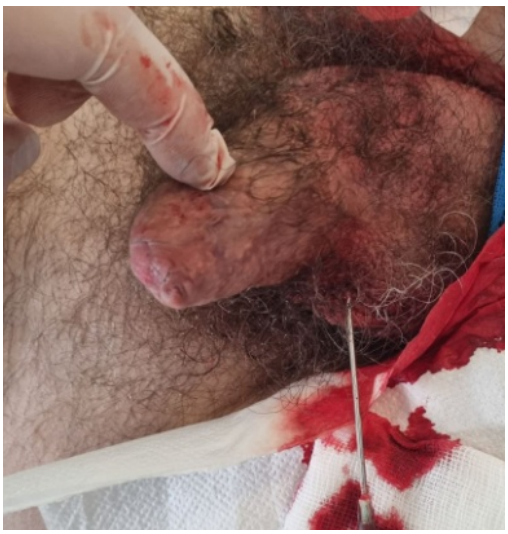
Keywords: priapism, heparin, protracted priapism, drug-induced priapism.

Introduction

Priapism is a rare and dangerous condition that describes the maintenance of more than four hours of non-sexually arisen erection [1]. It is classified into the low flow (ischemic) and high flow (nonischemic) priapism [2]. The causes of this condition include hemoglobinopathies and various hypercoagulable states. Moreover, it may appear as a side effect of some drugs, including vasoactive medications, antidepressants, or cocaine. Very rare causes of priapism include hydroxyzine, drotaverine, low molecular weight heparin [3,4]. Here we present a rare case of heparin-induced priapism which occurred after initiative treatment for myocardial infarction, whose pathogenesis remains unknown.

Case presentation

A 41-year-old Caucasian male presented in the emergency room of our institution with a complaint of a sudden strong pain attack and burning sensation in the chest area. He was hospitalized in the Intensive Care Unit of a Cardiology Department with a diagnosis of the inferior wall myocardial infarction (MIC) with ST-elevation. The current condition indicated coronary angioplasty procedure with stent insertion which was performed, followed by the conservative treatment with antiaggregants (aspirin, ticagrelor) and heparin. A few hours after the conservative treatment initiation, the patient presented for urological examination, complaining of a sudden erection without any sexual stimulation. The patient's medical history included dyslipidemia and arterial hypertension, but no medications were being taken before the hospitalization. The patient denied any alcohol or drug consumption, although he had been smoking for many years. Of note, he had a negative history of sickle cell disease, previous accidents of priapism, PDE-5 inhibitor use, or other erectile dysfunction pharmacotherapy.



Physical exam revealed a fully erect penis with rigid corporeal bodies tender to palpation. As an initial treatment, 20 minutes ice applications and penis mechanical pressure were recommended.

The same day urological examination was repeated due to ineffective conservative treatment when 60 millilitres of dark venous blood was aspirated, followed by 30 more millilitres of lighter blood aspiration from corporeal bodies, which revealed acidosis [Figure 1].

Figure 1. Patient after venous blood aspiration from corporeal bodies

One milligram of adrenaline diluted with nine millilitres 0.9% NaCl solution was injected into the penis root, ice and pressure applications were applied again. The following day, the tenderness, pain, and partial erection reoccurred, the subcutaneous hematoma was present in the penis and scrotum area [Figure 2]. The ring blocks anaesthesia with lidocaine was performed and another 50 millilitres of blood was aspirated.

Figure 2. The following day after the penile blood aspiration, a subcutaneous hematoma in the penis and scrotal area occurred

Since after a while the same symptoms reoccurred for the third time [Figure 3], the distal corporogranular shunt procedure was performed. Under general anaesthesia, a Foley catheter was placed for urethral identification, an incision into the penis head, and hematoma evacuation was performed. After sufficient detumescence, the distal corporogranular shunt incision sites were closed with two vicryl absorbable sutures, Foley catheter was recommended to be removed 4 days after the surgery, hematoma resorption was predicted and sufficient analgesia was assured.

Figure 3. Penis erection reoccurred after blood aspiration

After the surgery, blood inflammatory markers were being followed. Because of leukocytosis, increased CRP, and febrile temperature, antibiotic therapy of cefuroxime 500mg twice a day orally was administered. The abstinence from sexual activity for a month was recommended to prevent any further episodes before his follow-up. The ambulatorial urological examination was indicated after the recovery of myocardial infarction.

Discussion

Priapism is known as more than four hours of lasting erection of the penis which has no associations with sexual stimulation or desire [1]. Ischemic priapism accounts for more than 95% of all priapism episodes and it is marked by rigid corpora cavernous bodies and little or no arterial inflow which is confirmed by blood gas analysis showing acidosis (pH<7.25), hypoxia (pO2<30 mmHg), and hypercarbia (pCO2>60 mmHg) [5-7].

The pathophysiology of ischaemic priapism is idiopathic in most cases. Medication as an etiological factor is responsible for 25% to 40% of cases of priapism which includes a variety of different classes of drugs: antidepressants, antihypertensives (mainly includes alpha-blockers such as prazosin), recreational drugs (cocaine, cannabis, alcohol) [4,5]. However, anticoagulants such as heparin, warfarin, and low molecular weight heparin are proven to cause priapism episodes, but it is considered a very rare side effect [8].

In our case, the priapism occurred to a patient as an adverse effect of the treatment for myocardial infarction with anticoagulant heparin. There were also a few other medications administered during the period of hospitalization, such as 100mg aspirin daily and 90mg ticagrelor twice a day, 25mg metoprolol, 30mg zofenopril, and 60mg atorvastatin, but there is no literature published about any other drugs mentioned above as possible risk factors inducing priapism. Heparin belongs to a list of medications, where priapism stands as a side effect, although this is very rare and only a few publications about heparin-induced priapism have been reported [8,9]. One of the most suitable pathophysiological mechanisms suggests the theory of heparin-induced thrombocytopenia, caused by antiplatelet antibodies which leads to platelet aggregation [8]. Unfortunately, our patient's platelet count was within a normal range the whole duration of hospitalization, so the

pathological mechanism of development of priapism remains unclear.

Conclusion

We presented a rare case of heparin-induced priapism which occurred after the initiation of heparin treatment for myocardial infarction. Since there are only a few case reports published about heparin-induced priapism and the only one widely accepted pathological mechanism includes heparin-induced thrombocytopenia, triggered by antiplatelet bodies, the clear mechanism of priapism occurrence of our patient remains unclear. It is important to remember, that even rare adverse effects of any medication, such as priapism after heparin initiation could occur, and patients must be acknowledged with all the possible outcomes.

Conflicts of interest

There are no conflicts of interest.

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