Priapism in an Otherwise Healthy Man with SARS-CoV-2: Case Report and Literature Review

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Abstract
COVID-19 disease causes acute respiratory infection – pneumonia. It is associated with an increased risk of complications such as hypercoagulopathy, which leads to thromboses. We present a case of a young man presenting with typical SARS-CoV-2 symptoms (fever, cough, fatigue, and dyspnea), who experienced ischemic priapism, most probably due to thrombosis of penile vessels caused by the novel coronavirus infection. After prompt treatment of the priapism with punctures and irrigation, lasting penile detumescence was achieved. However, despite younger age, lack of serious comorbidities and administration of anticoagulants, priapism was followed by a fatal pulmonary embolism some days later.

Keywords
hypercoagulopathy, priapism, SARS-CoV-2

INTRODUCTION
Currently, the world is affected by an ongoing pandemic of a novel coronavirus causing severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).[1] Higher complication and mortality rates are reported in older population and in individuals with underlying comorbidities[2], while children and young people remain at low risk[3]. According to last publications, morbidity and mortality among young and middle age people may increase, probably due to new strains of the virus or genetic predispositions.[4] Thromboembolism rates of COVID-19 patients are high and associated with higher risk of death.[5] While not the only reason, thrombosis of the vessels of penis may cause priapism – prolonged erection lasting more than 4 hours without sexual stimulation, associated with a risk of erectile function impairment. Ischemic priapism is reported in patients with CO-

VID-19 infection, predominantly in patients aged over 60 years, but also in younger people.[6]

CASE REPORT
We present a case of a young male – 44 years old without any remarkable previous medical history. He presented with symptoms that started 5 days prior, which at the beginning were mild – subfebrile temperature, fatigue, headaches, muscle pain, light cough. He was started on paracetamol, vitamin supplements and azithromycin 500 mg once daily. Gradually, the cough increased and the day before admission to hospital, he felt shortness of breath and experienced erection of the penis without sexual stimulation. The erection persisted more than 15 hours and started to be painful and the dyspnea worsened, so he was referred to our hospital for evaluation. At admission, the laboratory results showed normal white
blood cells (WBC), lymphopenia – 0.5 G/l, normal values of neutrophils, hemoglobin, and thrombocytes. Liver enzymes were slightly elevated, electrolytes and renal function – normal. CRP was elevated 25 times and INR – 1.32 (Table 1).

Arterial blood gas analyses: from a. radialis – saturation (SpO₂) 89%, pH 7.36; from corpora cavernosa of penis – SpO₂ 56%, pH 7.03.

Unenhanced CT scan indicated bilateral diffuse ground glass opacities in all lung segments – viral pneumonia affecting between 40% and 50% of the lung parenchyma. Fig. 1

ECG – sinus tachycardia (102 b/m).

Nasopharyngeal PCR test for COVID-19 – positive.

An ischemic priapism was diagnosed and immediate intervention under local anesthesia with lidocaine was performed – bilateral puncture of the cavernous bodies with blood aspiration, followed by irrigation with saline and heparin solution. The needle was gauge 21. Lasting detumescence was achieved right away and the episode of priapism was solved (Fig. 2).

The treatment included saline infusions, antibiotics, gastroprotection, NSAIDs, methylprednisolone, enoxaparin 60 mg/0.6 ml once daily. At the beginning, oxygen (5 l/min) was given through an oxygen mask achieving SpO₂ 97% and improvement of his condition. After 5 days his condition started to decline and low saturation level despite prone position and maximal oxygen on face mask necessitated the application of high-flow oxygen non-invasive positive pressure ventilation (60 l/min; 90% O₂). The subsequent radiological examinations showed worsening of the lung findings (Fig. 3) and laboratory tests (Table 1).

At 13 days after admission, the patient experienced chest pain, cyanosis and decline of saturation and his blood gas analyses, CT chest scan and D-dimers indicated bilateral submassive pulmonary thromboembolism. Heparin infusion treatment was initiated as instructed by consultation with a cardiologist. The patient’s condition rapidly declined the next day, he was intubated and died 8 hours later.

**DISCUSSION**

There are three types of priapism: ischemic “low blood flow” priapism, characterized by a minimal to absent arterial inflow, leading to pain and rigidity; non-ischemic “high blood flow”; and stuttering priapism. In our case, the patient had low flow priapism – he experienced rigidity and

**Table 1.** Laboratory results dynamics – admission, day 4 and day 13

<table>
<thead>
<tr>
<th>Indicator, reference range</th>
<th>Hospital admission</th>
<th>Day 4</th>
<th>Day 13</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC, 4.1–11 G/l</td>
<td>4.9</td>
<td>7.86</td>
<td>17.37 H</td>
</tr>
<tr>
<td>Neutrophils, 2.0–7.8 G/l</td>
<td>4.1</td>
<td>6.89</td>
<td>15.5 H</td>
</tr>
<tr>
<td>Lymphocytes, 0.6–4.1 G/l</td>
<td>0.5 L</td>
<td>0.55 L</td>
<td>0.89</td>
</tr>
<tr>
<td>Hemoglobin, 140–180 g/l</td>
<td>177</td>
<td>157</td>
<td>148</td>
</tr>
<tr>
<td>Thrombocytes, 140–440 G/l</td>
<td>164</td>
<td>485 H</td>
<td>407</td>
</tr>
<tr>
<td>Urea, 2.8–7.2 mmol/l</td>
<td>7.2</td>
<td>4.6</td>
<td>12.5 H</td>
</tr>
<tr>
<td>Creatinine, 74–110 umol/l</td>
<td>104</td>
<td>76</td>
<td>142 H</td>
</tr>
<tr>
<td>Potassium, 3.5–5.1 mmol/l</td>
<td>4.7</td>
<td>3.9</td>
<td>5.5</td>
</tr>
<tr>
<td>Glucose, 4.1–5.9 mmol/l</td>
<td>7.17 H</td>
<td>7.94 H</td>
<td>16.24 H</td>
</tr>
<tr>
<td>CRP, &lt;0.50 mg/dl</td>
<td>13.37 H</td>
<td>18.29 H</td>
<td>9.44 H</td>
</tr>
<tr>
<td>ASAT, &lt;50 U/l</td>
<td>94 H</td>
<td>66 H</td>
<td>37</td>
</tr>
<tr>
<td>ALAT, &lt;50 U/l</td>
<td>141 H</td>
<td>82 H</td>
<td>35</td>
</tr>
<tr>
<td>Procalcitonin, &lt;0.50 ng/ml</td>
<td>0.4</td>
<td>1.11 H</td>
<td>2.64 H</td>
</tr>
<tr>
<td>D-Dimer, &lt;300 ngFEU/ml</td>
<td>832</td>
<td>601 H</td>
<td>5600 H</td>
</tr>
<tr>
<td>INR, 0.8–1.2</td>
<td>1.32 H</td>
<td>1.46 H</td>
<td>1.77 H</td>
</tr>
</tbody>
</table>

H – high, L – low
Figure 2. Puncture of cavernous bodies and saline and heparin irrigation – stages of the procedure.

Figure 3. Dynamics of chest X ray at 6 days (left) and 11 days (right).

pain and the pH and SpO$_2$ of the cavernous blood analyses proved ischemia. The mechanism is sinusoidal thrombosis and veno-occlusion with little or no cavernosal blood flow.[8] The causes of ischemic priapism are often unclear, but the main subcategories include haematological/thrombotic causes, drugs/pharmacological agents, intracorporal injection of pharmacostimulants, neurological causes, and malignancy.[9]

One of the serious COVID-19 disease complications is venous thrombosis. Overall thrombosis rate was reported to be 21% among hospitalized patients, in ICU – 31%; overall pulmonary embolism rate was 13%, in ICU – 19%.[5] The mortality rate among patients with thromboembolic incidents was found to be significantly higher than among patients without thrombosis. In our case, we have a patient with identified coronavirus disease, severe pneumonia and hypercoagulopathy – priapism and subsequent pulmonary thromboembolism leading to death – despite young age, absence of concomitant diseases and thromboprophylaxis with low-molecular-weight heparin. There are several case reports for priapism as an urological complications of COVID-19 infection usually in patients aged over 60 years, but also in younger men.[6,10] In the published cases, priapism was not associated with other risk factor like neurological or malignant disease or administration of potentially priapism provoking drugs, this was also valid for our patient.

In our case the treatment of priapism was prompt right after the patient admission, which is important, as the time from onset to treatment is crucial for a successful outcome.[11] The current practices include puncture, irrigation and injection of diluted sympathomimetic drug (phenylephrine).[6,11] In our case, we did not inject sympathomimetic drug because of patient’s tachycardia. Puncture and irrigation were enough to achieve lasting detumescence.
CONCLUSION

Hypercoagulopathy in the course of SARS-CoV-2 is a relatively common complication leading to higher morbidity. One possible urological manifestation is ischemic priapism, which should be treated according to current practices, additional systemic antithrombotic therapy may be needed.

REFERENCES