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Priapism in a patient with coronavirus disease 2019 (COVID-19): A case report

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ABSTRACT

Thromboembolic complications related to SARS-CoV-2 have been extensively reported. They include deep vein thrombosis, pulmonary embolism, ischemic stroke, and acute coronary syndrome. Penile thrombosis has not been reported as a thrombotic complication of SARS-CoV-2 infection with hypercoagulability.
Here we describe a case of priapism as a thromboembolic complication in a patient with COVID-19 who recovered from acute respiratory distress syndrome (ARDS). We discuss the underlying pathophysiological mechanisms mainly related to an hypercoagulability state. Emergency management consisted on an intracavernosal injection of the sympathomimetic agent ethylephrine and cavernosal blood aspiration. The patient experienced no recurrences under thromboprophylaxis by enoxaparin 40 mg twice daily.

1. Introduction

The new severe acute respiratory syndrome coronavirus 2 (SARS CoV-2) causing coronavirus disease 2019 (COVID-19) has led to an international health crisis. Nearly 5% of patients require intensive care unit (ICU) management, and the clinical presentation is dominated by acute respiratory distress syndrome (ARDS) [1]. In addition to life-threatening respiratory distress, other potentially fatal complications include hypercoagulability with a high rate of thromboembolic complications of up to 31% in ICU patients, requiring a specific anticoagulation strategy [2]. Deep vein thrombosis and pulmonary embolism are the most common forms of thrombosis, whereas ischemic stroke and acute coronary syndrome have been reported less frequently [2,3].

Priapism is a medical emergency potentially related to focal thrombosis due to hypercoagulability and hyperviscosity. To the best of our knowledge, penile thrombosis has not yet been reported in patients with COVID-19.

Here we report a case of priapism as a clinical expression of hypercoagulability and hyperviscosity induced by SARS CoV-2 infection.

2. Case report

A 62-year-old man with a history of left inguinal surgery and appendectomy presented with asthenia, fever, dry cough, and diarrhea. Clarithromycin was given by his general practitioner because of suspected bacterial infection. Two days later he experienced acute dyspnea requiring mobile emergency medical unit management. At emergency medical team arrival, he had isolated respiratory failure and was promptly intubated and mechanically ventilated after receiving etomidate and succinylcholine for rapid sequence induction combined with midazolam and sufentanil for sedation. He then experienced on-scene hemodynamic failure, for which he received intravenous fluid resuscitation and 0.1 μg/kg/min of norepinephrine. Chest computed tomography (CT) and CT angiography performed before ICU admission showed diffuse and bilateral involvement of the pulmonary parenchyma with areas of frosted glass, thickening of the interlobular septa creating a crazy paving pattern, and parenchymal condensations. No sign of proximal pulmonary embolism was identified.

Upon ICU arrival, blood pressure was 116/82 mmHg, heart rate 100 beats per minute, and body temperature 38.5 °C. He had severe ARDS with an initial PaO_2/FiO_2 ratio at 66 with 100% FiO_2. The physical examination revealed previously unidentified priapism, in the absence of urinary catheterization. The patient was sedated and unable to answer questions about pain. Clinically, the two corpora cavernosa were rigid, while the glans was flaccid. Table 1 reports the laboratory tests performed at ICU arrival. SARS CoV-19 infection was confirmed by real-

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Table 1
Clinical laboratory results at ICU arrival.

	Patient	Laboratory Standard
Hematology		
Leukocytes, G/	7.2	3.8–11
Hemoglobin, g/dL	11.4	13–18
Hematocrit, %	33.5	39–53
Neutrophils, G/L	6.25	1.4–7.7
Lymphocytes, G/L	0.63	1.0–4.8
Monocytes, G/L	0.27	0.15–1.0
Platelets, G/L	170	150–400
Creatinine, μmol/L	81	62–106
Urea, mmol/L	7	3.0–9.2
Troponin T, ng/mL	0.081	< 0.014
NT-pro-BNP, pg/mL	2898	< 900
Creatine phosphokinase, IU/L	1253	20–200
Bilirubin, μmol/L	11	< 21
C- reactive protein, mg/L	178	< 5
Procalcitonin, ng/L	5.45	< 0.5
Ferritin, μg/L	2674	13–400
Prothrombin, %	91	70–130
Activated partial thromboplastin time, %	1.20	< 1.20
Fibrinogen, g/L	7.14	1.56–4.00
D-Dimer, ng/mL	2210	< 500
Blood gas analysis, Day 1 (FiO_2 100%)		
pH	7.36	7. 35–7. 45
$PaCO_2$, mmHg	52	35–45
PaO_2, mmHg	66	85–100
CO_2 total, mmol/L	30	22–29
O_2 saturation, %	89	95–98
Cavernosal blood gas analysis, Day 1 (FiO_2 40%)		
pH	6.98	
$PaCO_2$, mmHg	121	
PaO_2, mmHg	68	
CO_2 total, mmol/L	30	
Lactate, mmol/L	10	

NT-pro-BNP, N-terminal pro-brain natriuretic peptide; CO_2, carbon dioxide; O_2, oxygen; $PaCO_2$, partial pressure of CO_2 in arterial blood; PaO_2, partial pressure of O_2 in arterial blood.

time reverse transcriptase-polymerase chain reaction on a bronchial aspirate sample.

Appropriate management for ARDS was provided and an ice pack was applied to the penile area. After 4 h of persistent erection, penile blood aspiration recovered dark blood clots suggesting low-flow priapism. This hypothesis was confirmed when the cavernosal blood gas analysis showed acidosis with a pH of 6.98 and partial pressure of carbon dioxide of 121 mmHg; partial pressure of oxygen was 68 mmHg (Table 1). In addition to cavernosal blood aspiration, an intracavernosal injection of the sympathomimetic agent ethylephrine was performed. Thromboprophylaxis was by enoxaparin 40 mg twice a day.

Finally, the patient was successfully extubated after 14 days of mechanical ventilation. No other localizations or local recurrence of embolic thromboembolism occurred in the ICU of after discharge to a ward.

3. Discussion

Priapism is defined as a penile erection that persists beyond 4 h and is unrelated to sexual interest or stimulation [4]. The pathophysiological mechanisms are classified as ischemic, arterial, or stuttering. Ischemic priapism is the most common form. The mechanism of ischemic priapism is persistent corporal smooth-muscle relaxation inducing subtunic vein compression and impeding sinusoidal outflow. Once the intracorporal pressure increases above the mean arterial pressure of the cavernosal arteries, no inflow of blood can occur [5]. Arterial priapism is rare, and chiefly related to local trauma. Stuttering priapism is the result of obvious factors such as an acute sickle cell crisis. In addition to immediate local complications specifically related to the ischemic mechanism, long-term complications can be expected in all presentations. Thus, the identification and management of ischemic priapism is a medical emergency [6].

In our patient, the clinical presentation, blood gas findings, and presence of dark blood clots at cavernosal blood aspiration strongly support ischemia-related priapism [6]. The only surprising finding of a cavernosal oxygen partial pressure measurement above 30 mmHg can be easily explained by the 40% FiO_2 at the time. No alternative diagnosis to ischemia was identified: the priapism was inaugural, and the patient did not receive treatments known to promote priapism, such as propofol sedation [7].

Furthermore, in patients with SARS-CoV-2 infection, thromboembolic complications have been identified at the time of hospital admission [2]. Indeed, COVID-19 patients demonstrate the simultaneous presence of all the elements of the Virchow's triad promoting local thrombosis. The first component is hyperviscosity, which may be related to increased plasma viscosity, blood cell count elevation, and/or impaired blood cell passage through capillaries. SARS CoV-2 enters cells by binding to the ACE-2 receptor on the surface of cells including endothelial cells, and vasculopathy has been documented during autopsies [8]. The second component is hypercoagulability [9], which occurs during the massive inflammatory response to SARS-CoV-2 infection, with increases in C reactive protein, procalcitonin, LDH, D dimer, fibrinogen, and ferritin as seen in our patient. Antiphospholipid antibodies have been identified in patients with COVID-19 [10]. The third component is endothelial dysfunction related to structural vessel wall damage caused by cytokinin and free radicals [11]. The cytokine storm induces damage-associated molecular patterns, causing focal microvascular inflammation, which triggers endothelial activation and, finally, prothrombotic conditions.

3.1. Limitations

Although the arguments supporting a causal link between COVID-19 and priapism, as well as the ischemic mechanism for priapism, are very strong in our case, reports of further cases would strengthen the evidence.

4. Conclusion

The clinical and laboratory presentation in our patient strongly suggests priapism related to SARS-CoV-2 infection. This medical emergency should be recognized by healthcare professionals and treated promptly to prevent immediate and chronic functional complications.

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Ethics approval

Not applicable.

Consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available.

Availability of data and material

Not applicable.

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Code availability

Not applicable.

Declaration of Competing Interest

None to disclose.

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