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Priapism in COVID-19: A thromboembolic complication

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ABSTRACT

SARS-CoV-2 (COVID-19) infection is frequently associated with thromboembolic complications. In this case report, we describe the diagnosis and management of priapism as a thromboembolic complication of severe COVID-19.

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1. Introduction

As of mid-August 2020 the CDC reported approximately 5.5 million cases of Coronavirus Disease 19 (COVID-19) in the US [1]. In addition to complications such as pneumonia and acute respiratory distress syndrome (ARDS), more attention has been focused on the pro-thrombotic properties of the disease. Even with prophylactic anticoagulation, the incidence of venous thromboembolism (VTE) is between 16%–27% [2–5]. Lamamri et al. reported a case of priapism as a thromboembolic complication of COVID-19 [6].

Priapism is an erection that persists beyond or is unrelated to sexual stimulation and typically only involves the corpora cavernosa. The potential for penile fibrosis and permanent impotence makes the condition a medical emergency. Though there are many potential causes, the pathophysiology is believed to be related to excess contractile neurotransmitter release, impaired relaxation of intracavernosal smooth muscle, failure of intrinsic detumescence, or, as postulated for our patient, obstruction of draining venules [7].

In this report, we describe a case of primary ischemic priapism in a patient with severe COVID-19 complicated by ARDS.

2. Case report

A 69-year-old man with a history of obesity presented with one week of cough, congestion, dyspnea, anorexia, and generalized weakness. He was initially prescribed amoxicillin-clavulanate and a methylprednisolone taper by his primary care physician for suspected acute

sinusitis. He developed worsening respiratory distress, prompting his coming to the Emergency Department. On presentation, his temperature was 98.9° F, HR 78 beats/min, RR 25/min, BP 135/71, and SpO₂ 94% on 40 L heated high flow nasal cannula (HHFNC) at 80% FiO₂. He subsequently tested positive for SARS-CoV-2. CXR on admission showed bilateral multifocal interstitial and airspace opacities. He was treated with dexamethasone (10-day course) and supplemental oxygen via HHFNC. The patient's respiratory status worsened, and he required intubation using etomidate, fentanyl and rocuronium. Mechanical ventilation was begun and sedation was maintained with propofol and fentanyl. Hypotension ensued and norepinephrine was added for pressure support.

Over the next 12 h, he declined to PaO₂/FiO₂ 149.0 on 100% FiO₂, and prone ventilation was begun. Sedation with propofol and cisatracurium was initiated for ventilator compliance. Upon supination the following afternoon, nursing noted an erection. Ice packs were placed but the erection persisted over the following 3 h with rigidity of the corpora cavernosa and flaccid glans. Urology was consulted and genital ultrasound showed normal arterial and venous Doppler flow and compressible dorsal penile veins with no identified thrombosis. Due to continued suspicion for ischemic priapism, a cavernosal blood gas was obtained, revealing pH 6.93, pO₂ <30.1, and pCO₂ >98.3, consistent with ischemic priapism. Subsequently, 21-gauge needles were placed in bilateral corpora cavernosa to drain blood until urology arrived and assisted in administration of intracavernosal 250 µg/mL phenylephrine. Complete detumescence was achieved with 10 doses in 3-min intervals or 2500 µg phenylephrine over 30 min. The patient was subsequently started on an intravenous heparin drip. The patient had a prolonged course of severe ARDS in the ICU and ultimately died; however, he experienced no further thromboembolic complications, and priapism did not recur.

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3. Discussion

Priapism is divided into three subtypes: ischemic (low-flow/veno-occlusive), nonischemic (high-flow/arterial), and stuttering [8]. Resolution of ischemic priapism within 24 h is associated with better outcomes [9]. In conscious patients, pain typically differentiates ischemic from nonischemic priapism. However, in our sedated patient, we had to rely on cavernosal ABG findings. ABG criteria suggestive of ischemia include $pO_2 < 30$, $pCO_2 > 60$, and $pH < 7.25$, all of which occurred in our patient.

Case reports suggest that propofol can cause ischemic priapism in individuals undergoing procedures [10–12], including confirmation by rechallenge with propofol [12]. However, our patient received propofol for more than 12 h prior to pronation without issue or reoccurrence in subsequent weeks of propofol infusion. Additionally, the prevalence of coagulopathy in COVID-19 and the associated risk of mortality requires greater attention to its likelihood as the precipitating factor.

Thromboembolic complications are frequent in COVID-19. In a study of 3334 patients in New York City, Bilaloglu et al. observed a 16% rate of thrombosis, 29.4% in ICU patients, using routine screening practices. Mortality in patients with thrombotic events was double that of those without thrombosis (43.2% vs. 21.0%, $p < 0.001$), including a multivariable hazard ratio of 1.82 for VTE and 1.99 for arterial thromboembolism (ATE) [3]. In a smaller study of 150 COVID-19 patients, Helms et al. reported an odds ratio of 2.6 ($p < 0.035$) for thrombotic complications in COVID-19 ARDS patients compared to those with non-COVID-19 ARDS [13].

Though the evidence has suggested an association between COVID-19 and hypercoagulability, there is a lack of high-quality evidence supporting routine therapeutic anticoagulation. Consequently, the American Society of Hematology encourages participation in one of “multiple randomized controlled trials” rather than empirically using anticoagulation with hospitalized patients with COVID-19 [14].

Declaration of Competing Interest

None

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