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Case Report

Spontaneous Hematoma and Covid-19: Diagnosis and Management, A Case Report

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Abstract

The aim of our presentation is to warn against these bleeding complications and to discuss their etiological diagnosis and management.

Keywords: Covid-19; Bleeding; Chronic Renal Failure

Introduction

The bleeding complications of Covid-19 are poorly described in the literature, although they potentiate the severity of the disease. In addition, spontaneous muscle hematomas are a frequent and serious complication of anticoagulant treatments. We report a case of spontaneous hematoma occurring of a patient with Covid-19.

The aim of our presentation is to warn against these bleeding complications and to discuss their etiological diagnosis and management.

Observation

Mr. T., 58 years old, hypertensive and chronic kidney disease on hemodialysis, was seen urgently for polypnea, fever at 38.5° C, deterioration of general condition and severe asthenia. On admission, laboratory tests found slight leukocytosis at 16,000 GB / mm3 with neutrophilia and lymphopenia, CRP at 146 mg / l, fibrinogen at 6.2 g / l, d-dimer at 890 IU, AST at 96 IU / l and ALT at 89IU / l. PCR of the nasopharyngeal swab showed the presence of Sars Cov-2. The CT chest objected pulmonary damage estimated at 40%.

The diagnosis of Covid-19 infection was retained and the patient is managed with specific treatment based on ciprofloxacin 400mg IV infusion BID, cefotaxim 1g IV TID , dexamethasone 6mg OD and Enoxiparin 0.4IU Od subcut as well as oxygentherapy 9L / min. On day 4 of evolution, the patient reported a throbbing pain in the left groin, radiating throughout the ipsilateral lower limb, not giving way under simple analgesics, then the appearance of a red patch occupying the described region. As well as difficulty of walking. A muscle assessment revealing a CK at 450 IU / l. The soft tissue ultrasound showed hemorrhagic suffusion extending from the left inguinal region to the calf, measuring approximately 14 cm in diameter. Doppler ultrasound ruled out a possible aneurysm and confirmed the presence of a large hematoma on the inside of the left lower limb. Computed tomography (CT) found a blood collection in the interstitial tissues diffused from the left groin to the big toe. The medium-term course was favorable marked by the gradual disappearance of myalgia, resorption of the hematoma after one month and resumption of physical activity.

Discussion

This clinical presentation of a muscle hematoma occurring in a dialysis patient suffering from covid-19 posed an etiological diagnostic problem of acute muscle pain resistant to usual analgesics, it may be necessary for thrombophlebitis or an aneurysm, imaging currently corrects the diagnosis [4,5].

If the CT scan is not performed urgently, the soft tissue ultrasound can be used to point to an etiology. Doppler ultrasound is today a diagnosis method of choice, Non-invasive, inexpensive, reliable, reproducible but operator-machine and patient-dependent [19]. This method makes it possible to carry out a very precise anatomical mapping of the arteriovenous axes of the lower limbs.

In our clinical case, Doppler ultrasound found no hemodynamic abnormalities and ruled out acute arteritis or thrombophlebitis.

In the literature, CT scan is the standard diagnostic examination and our observation confirms these data [1-4,6-9]. The hematoma initially appears as a fluid collection taking the contrast. Its density is typical of acute hemorrhage.

Nevertheless, COVID-19 may cause a severe inflammatory state leading to the disorder of hemostasis. It is also linked with various coagulation abnormalities, such as an increase in procoagulant factor levels, including fibrinogen, D-dimers, and factor VIII that have been associated with higher mortality and increased risk of venous thromboembolism (VTE). This combination of inflammation and thrombosis can be referred to as thromboinflammation or COVID-19 associated coagulopathy. Sepsis-induced coagulopathy and disseminated intravascular coagulopathy (DIC) have been reported with severe disease, mainly in non-survivors. However, most patients with severe infection of SARS-CoV-2 develop thrombosis rather than bleeding, while bleeding is commonly seen in DIC. Severe COVID-19 increases fibrinogen levels and factor VIII activity which is not seen in patients with DIC [20,21].

Therefore, patients with COVID-19 have a high tendency to develop acute thrombotic events, including VTE, acute stroke, acute myocardial infarction, and clotting of the ECMO (extracorporeal membrane oxygenation) and CRRT catheters [23-28]. Even though It commonly causes thrombotic complications, bleeding complications of COVID-19 due to coagulopathy and use of anticoagulation are less commonly reported. Erdinc., *et al.* [22] reported a case of a patient with COVID-19 complicated by spontaneous retroperitoneal lbleeding and massive deep vein thrombosis (DVT), which was later complicated by compartment syndrome. To the best of ourknowledge, a spontaneous bleeding occurring in hemodialysis patients is rarely reported in the current literature. This case emphasizes that COVID-19 induced hypercoagulable state can cause massive thrombosis, and patients might need anticoagulation therapy. However, clinicians should also consider the risk of hemorrhagic complications of the disease and be cautious when administering anticoagulant therapy in selected cases, especially for dialysis patients.

In addition, we observed that the patient with chronic renal insufficiency, receiving Calciparin (unfractionated heparin "UFH") during the dialysis sessions having received a preventive anticoagulation based on low molecular weight heparin (LMWH), being able to increase the risk of bleeding. There are many indications for anticoagulation in patients with renal insufficiency due to the high prevalence of cardiovascular diseases, thrombotic complications, especially in nephrotic syndrome, but also in relation to the various vascular accesses necessary for hemodialysis or circuitsextracorporeal.

The management of prophylactic or therapeutic anticoagulation is difficult in patients withchronic renal failure, due to their tendency to hemorrhagic diathesis and renal elimination of some anticoagulants.

NHF exerts its anticoagulant activity by binding to antithrombin, inducing a conformational change in the latter, which increases its anticoagulant activity approximately 1000 times. This is exerted by mainly inhibiting factors IIa and Xa [10].

It is mainly metabolized by the endothelial and reticuloendothelial system and the inactive metabolites are eliminated via the urinary route. Its bio-availability is highly variable and its half-life is in the range of 1 hour to 1 hour 30 minutes after intravenous administration. Its handling requires regular dosing of activated partial thromboplastin time (APTT), or even anti-Xa activity in the event of baseline abnormal APTT.

In addition to the risk of bleeding associated with its use, UFH can induce osteopenia and immuno-allergic thrombocytopenia (heparin-induced thrombocytopenia or HIT) due to the presence

of antibodies that recognize heparin-related platelet factor4., resulting in activation of platelets and coagulation which may lead to venous and/or arterial thrombosis. HIT can occur in 1% to 3% of patients on UFH, requires discontinuation of heparin and the introduction of another immediate-acting antithrombotic [11].

As for LMWHs, they are obtained by fractionating FNH by different chemical or enzymatic processes. They more specifically inhibit factor Xa and have excellent bioavailability. They induce less osteopenia and TIH than FNH [11]. In most cases, blood monitoring is not necessary but, if necessary, their anticoagulant activity can be measured by measuring the anti Xa activity.

LMWHs are metabolized by the kidney, which is why there is a possible cumulative effect with repeated administration of LMWH in patients with chronic renal failure [10]. Before any administration of LMWH, we strongly recommend calculating creatinine clearance (Ccr) according to the Cockroft-Gault formula (formen: 1.23 x [weight x (140-age)/creatinemia in µmol/l]; for women 1.03 x [weight x (140-age)/serum creatinine]) especially in the elderly, or serum creatinine may be abnormally low due to decreased muscle mass.

While a retrospective study of 620 patients with a Ccr <60 ml/min treated either with UFH or with two subcutaneous injections of enoxaparin, did not demonstrate a significant difference between the two groups of patients, enoxaparin increased nevertheless 2.5 times the risk of bleeding in the subgroup of patients with severe renal failure (Ccr <20 ml/min) [12].

The duration of anticoagulation with LMWH also seemed to be an important risk factor since only 3.1 % of patients anticoagulated for a duration of ≤ 3 days with enoxaparin had a hemorrhagic episode whereas, beyond three days, the prevalence was 15.5%[12].The use of LMWHs in patients with patients with severe renal impairment (Ccr <30 ml/min) at beyond three days of treatment therefore requires regular dosing of anti-Xa activity and halving of the doses or a single daily administration of LMWHs.

In the event of prolonged administration, monitoring of the anti-Xa activity is also recommended in cases of moderate renal insufficiency (Ccr between 60 and 30 ml/min). NHF is used for the prevention of thrombosis of the extracorporeal circuits during the hemodialysis session, although LMWHs are increasingly replacing it in this indication. Although LMWHs are not dialyzed, their use in this indication seems as safe as that of UFH [13]. In our presentation, the combination of UFH and LMWH and the duration of administration of 10 days may be responsible for the increased risk of bleeding.

On the other hand, our dialysis patient received a high dose of fluoroquinolones (800mg/day) while it should not exceed 300mg/day, which could be responsible for the occurrence of intense myalgia which may be linked to acute rhabdomyolysis, because La ciprofloxacin is considered to be a weak inhibitor of CYP3A4.

In fact, rhabdomyolysis has been reported in COVID-19. So far, there have been a few cases of COVID-19 with rhabdomyolysis. It is still a rare but potentially fatal presentation. SARS-CoV-2 has been isolated from several organs and raises the possibility that the virus infects striated muscles and potentially leads to muscle breakdown [17]. There have also been reports of the association of rhabdomyolysis with viral infection such as influenza and severe acute respiratory syndrome [15,16]. Hyperkalemia, acute renal failure, metabolic acidosis, disseminated intravascular coagulation, compartment syndrome, arrhythmias and cardiac arrest are potential complications of rhabdomyolysis [14].

In our patient, there was no obvious cause for rhabdomyolysis. He did not report any strenuous activity for a few days before the presentation, no signs of trauma. After ruling out common causes, we came to the conclusion that rhabdomyolysis in our patient was probably associated with COVID-19 and/or fluoroquinolone overdose. Our patient's COVID-19 was diagnosed by the standard real-time reverse transcription-PCR (rRT-PCR) test. Jin and Tong reported the first case of rhabdomyolysis in COVID-19 as a late presentation [18].

They stressed that focal muscle pain, checking CK and myoglobin levels are important in detecting rhabdomyolysis in COVID-19 patients.

Conclusion

Bleeding complications with large deep or parietal hematoma may occur during the management of SARS-CoV-2 infection, occurring in chronic renal failure, this prompts us to be vigilant in the use of anticoagulants. Rhabdomyolysis can be an initial extrapulmonary manifestation of COVID-19, with very few cases de-

scribed in the literature to date, as it can be caused by overdose of quinolones. In any case, it is essential to add the routine CK assay in the initial screening laboratories while evaluating a suspected COVID-19 infection with myalgia and generalized weakness, which

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