

# COVID-19 associated acute limb ischemia in a patient on therapeutic anticoagulation, a case and literature review

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## Introduction

The novel coronavirus of 2019 (SARS-CoV-2) has been found to cause multiple complications across several organ systems in patterns not typically observed in previous iterations of the virus. Hemostatic mechanisms have been noted to be significantly deranged in particular, resulting in a DIC-like picture with elements of coagulopathy as well as hypercoagulability. [1]

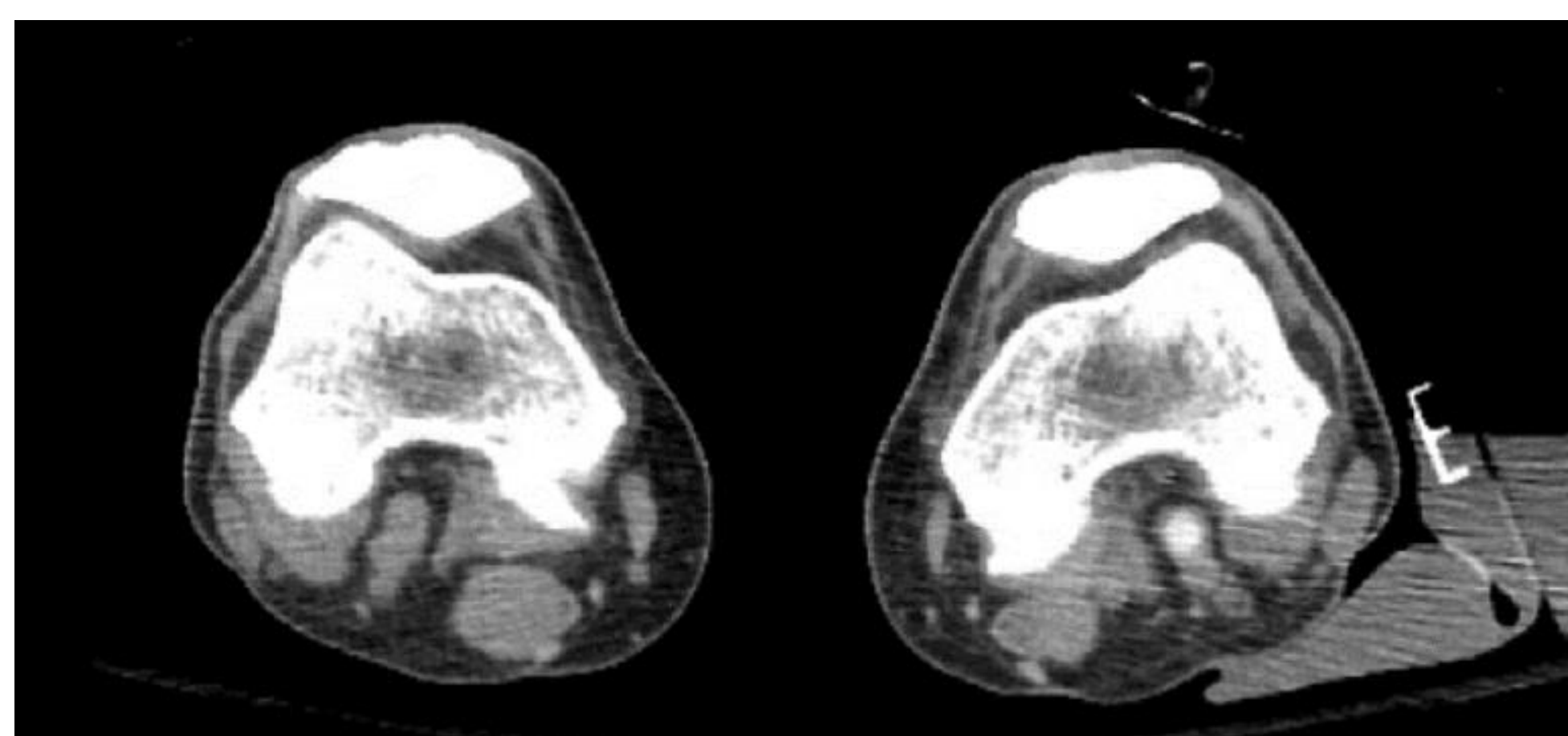
## Case Presentation

A 65-year-old man with a history of hypertension, hyperlipidemia, tobacco use, chronic kidney disease, and diabetes presented from a correctional facility with hypoxia. COVID-19 was confirmed. He was started on therapeutic anticoagulation with enoxaparin in lieu of an elevated D-dimer ( $>7,955$  ng/mL). Oxygen requirements increased, mental status deteriorated, and platelets began falling raising concern for Heparin-Induced Thrombocytopenia (HIT) versus Diffuse Intravascular Coagulation (DIC). Heparin products were stopped. Direct oral anticoagulant (DOAC) was initiated. He later became unable to tolerate oral medications. Fondaparinux was initiated. He was later found to have acute ischemia of the right lower extremity. He underwent four-compartment fasciotomy and surgical thrombectomy but required an above-the-knee amputation the following day. He later passed away due to cardiac arrest.

Figure 1. Admission Chest X-Ray



Figure 2. CT-Angiogram of the Lower Extremities



## Discussion

The hemostatic derangements seen in COVID-19 are heavily noted but ill-explained as of yet. Studies show up to 24% of hospitalized patients experienced some form of thromboembolic event. [1] Arterial thrombosis in COVID-19 as well as DIC is rare, even more so while on anticoagulation. [2] COVID-19 patients are subject to immobility, but pathological studies have also shown endothelitis with viral inclusion bodies seen under light microscopy as well as complement-induced microvascular damage. [3] Also noted is a relative deficiency in antithrombin III (ATIII), a primary component of heparin and heparin-like medication mechanisms. [4] Thromboelastography (TEG) demonstrated again decreased ATIII levels as well as increased fibrinogen, D-Dimer, Factor VIII, and von Willebrand Factor – all of which are consistent with a hypercoagulable state. [5]

## Conclusion

There is a clear association between COVID-19 and thrombotic events. Additional investigation is needed to fully elucidate the mechanism and prevention of this “COVID-19 Associated Coagulopathy (CAC).” Thorough, daily physical exams should be performed on all patients with COVID-19 to assess for venous or arterial insufficiency. Heparin and heparin products should be used with caution given the observed relative deficiency in ATIII. Alternative anticoagulants such as DOACs or direct thrombin inhibitors may convey benefit in further studies.

## References

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