Central arterial thrombus in the setting of therapeutic anti-coagulation with rivaroxaban in a COVID-19 positive patient

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INTRODUCTION

• In patients diagnosed with COVID-19 coagulation abnormalities demonstrate a hypercoagulable state; however the pathogenesis of the COVID-19 induced hypercoagulability is incompletely understood.
• This case is a demonstration of a patient with diagnosed COVID-19 who developed a central arterial thrombus in the setting of therapeutic anti-coagulation with rivaroxaban.

CASE DISCUSSION

77 year-old male with past history significant for heart failure, atrial fibrillation on anti-coagulation with rivaroxaban, chronic obstructive pulmonary disease on oxygen, insulin-dependent type 2 diabetes mellitus, chronic kidney disease, hypertension, hyperlipidemia and hyperaldosteronism presented 3 days of shortness of breath at rest, dry cough and diarrhea.

• Vital signs were within normal limits with SO2 95% at 4L/min O2 via nasal cannula as his baseline
• Initial blood work showed elevated CRP to 25.5, elevated lactic acid to 2.9, elevated D-dimer at 1,271, and positive PCR for SARS-CoV-2 via nasopharyngeal swab.
• Chest X ray showed lobar infiltrate compatible with bacterial pneumonia, treated with a 7-day course of ceftriaxone and azithromycin.

His coronavirus disease 2019 (COVID-19) specific treatment was a 5 day course of atazanavir 400 mg daily, convalescent plasma transfusion once, 3 days of IV methylprednisolone 40 mg twice daily, vitamin C and zinc supplementation.

His inflammatory markers including CRP, d-dimer, ferritin, and lactic acid were steadily decreasing and he was improving clinically, so he did not receive treatment with Tocilizumab.

The patient had been maintained on rivaroxaban throughout his hospitalization.


tocilizumab

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methylprednisolone 40 mg twice daily, vitamin C and convalescent plasma transfusion once, 3 days of IV treatment was a 5 day course of

ceftriaxone and azithromycin.
The patient's neurological status did not improve throughout his hospitalization.

The patient's neurological symptoms progressed and a repeat CT the next day revealed a clot expanding into the previously patent right MCA.

An MRI of the brain showed a large acute infarct in the right frontal and parietal lobes, as well as the caudate head.

Imaging at this time also showed interval increase in cytotoxic edema with local mass effect, without significant midline shift, herniation or hemorrhage.

His rivaroxaban was held and he was started on 81 mg aspirin.

A TTE showed no evidence of thrombi within the cardiac chambers.

Coagulation study panel indicated increased activity of Factor VIII, von Willebrand Factor, and Protein S, and downtrending D-dimer at 661.

The patient’s neurological status did not improve and family changed goals of care to comfort-measures-only.

DISCUSSION

This case demonstrates the complexity of the hypercoagulable state that has been observed to develop in patients with diagnosed COVID-19.

Early recognition of hypercoagulability via periodic coagulopathy laboratory analysis of may better guide anti-coagulation strategy in patients with COVID-19.

REFERENCES

3. Hypercoagulability/hypercoagulopathy