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Hypercoagulability in COVID-19: A rare case of DIC in SARS-CoV2 in the Emergency Department

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Case Description

67-year-old nursing home resident with PMH of MI, diabetes, hypertension, seizures, and left AKA presents to the ER due to altered mental status and hypoxia. Nursing home staff reported patient had fallen from bed, and during evaluation, he was found to be altered and hypoxic to 80% on room air. He is normally wheelchair-bound but participates in nursing home activities and well-oriented at baseline. Upon arrival, patient was encephalopathic with GCS 10 and no focal deficits. His vital signs were T 36°C, HR 110, BP 119/102, RR 22, and SpO2 96% on 15L nonrebreather. He had dry mucous membranes, clear breath sounds with good respiratory effort, soft abdomen, and no other skin or joint findings. He did not tolerate any oxygen weaning, but since he was protecting his airway, he was transitioned to Salter nasal canula 15L. Given the ongoing pandemic and hypoxia of unknown etiology, COVID-19 nasopharyngeal PCR swab was performed. ABG revealed pH 7.43, pCO2 31.5, and paO2 at 87.8. Although a normal paO2, this was drawn while patient was on a nonrebreather, and thus it was more telling of his hypoxemia.

A 1-view AP chest x-ray in the resus room demonstrated hazy opacities in right upper lobe, perihilar, and infrahilar regions, reported as nonspecific findings for atypical or multifocal pneumonia (Figure 1). ECG revealed sinus tachycardia with new T-wave inversions in V2-3 and slightly prolonged QTc 512. Sepsis workup was initiated with blood cultures, urinalysis, and empiric treatment with vancomycin, ceftriaxone, flagyl, and 1L LR bolus for pneumonia. A dose of methylprednisolone 40mg was additionally given for suspected COVID-19 infection. CT head showed 2 small areas of acute to subacute infarcts in right inferior parietal and inferior cerebellum (Figures 2-3). Neurology team favored these as incidental findings that did not explain his encephalopathy.

As listed in Table 1, abnormalities in the metabolic panel primarily were the hemolyzed K 8.4 and Cr 1.72 (baseline 0.3-0.4). Despite the hemolyzed specimen, due to his prolonged QTc and the magnitude of the potassium elevation. His whole blood K later returned at 3.1. His CRP was 14.0, ESR 20, LDH 2005, mild total bilirubinemia 3.0 without significant transaminitis, and haptoglobin <30. Keppra level was supratherapeutic at 57.6 mg/L. He also had a leukocytosis at 13.9 with lymphocytopenia at 0.14, hemoglobin 13.2, and platelets 52. More importantly, he had an elevated coagulation profile with INR at 2.68, PT 27.8s, and PTT 44s. High-sensitivity troponins and BNP later returned significantly elevated at 1649 and 808, respectively. As our patient was not on any anticoagulants and presenting with a perceivably pro-thrombotic state with suspected hemolysis, there was concern for TTP vs. DIC. A bedside ECHO showed normal EF left ventricular function, but right ventricle appeared slightly enlarged with mild septal bowing. No pericardial effusion was visualized. His inferior vena cava was collapsed, so he was given an additional bolus. CTPE could not be pursued as patient had documented severe iodine allergy. DIC panel subsequently revealed d-dimer >20 and low fibrinogen <60. During this workup, patient's PCR swab resulted as positive for COVID-19. He was given 2U cryoprecipitate while hematology was consulted for possible TTP. Despite his encephalopathy, AKI, thrombocytopenia, and evidence of MAHA from lab findings, hematology favored DIC due to his overall pro-thrombotic state, prolonged coagulation parameters, and a clear precipitant of sepsis, likely from COVID-19. Patient was admitted to the MICU for further management.

Discussion

- The cytokine storm precipitated by SARS-CoV2 virus leads to activation of platelets and the coagulation cascade.
- Elevated D-dimer and PT have been significantly associated with poor prognosis in a retrospective study by Tang et al.
- Treatment of the underlying disease as always been the hallmark of DIC management, but this is difficult to do with the lack of evidence-based therapeutics available for the treatment of COVID-19

Conclusion

- Testing a coagulation profile upon presentation for suspected COVID-19 patients can help identify a dysfunctional hemostatic system as a sign of a hyperinflammatory response.
- Early detection of DIC is paramount in order to promptly initiate appropriate therapies as well as to determine a valuable prognostic indicator.
- While our frontline physicians are becoming more familiarized with clinical characteristics related to SARS-CoV2, the abnormalities in coagulation, especially leading to DIC, in critically ill patients still warrant attention and further in-depth research

Differential Diagnosis

Altered mental status	Hypoxic respiratory failure
Post-ictal state	Multifocal bacterial pneumonia
Intracranial hemorrhage	Aspiration pneumonia
Ischemic stroke	Pulmonary embolism
Sepsis-induced encephalopathy	Pneumothorax/hemothorax
Hypoxia-induced encephalopathy	ARDS
Severe electrolyte derangements (hyponatremia, hypercalcemia	COVID-19 pneumonia

Table

Labs	Results	Reference Ranges
Sodium (Na)	129	135-145 mmol/L
Potassium (K)	8.4	3.5-5.0 mmol/L (hemolyzed)
Whole blood potassium	3.1	
Chloride (Cl)	96	98-111 mmol/L
BUN	58	10-25 mg/dL
Creatinine (Cr)	1.72 (baseline 0.3-0.4)	<1.2 mg/dL
Calcium	8.0	8.6-10.4 mg/dL
ALT	17	<52 IU/L
AST	94 (hemolyzed)	<35 IU/L
Albumin	3.4	3.2-4.6 g/dL
Total bilirubin (Tbili)	3.0	<1.2 mg/dL
Direct bilirubin	0.5	0-0.3 mg/dL
High-sensitivity (hs) troponin	1649	<18 ng/L
Brain natriuretic peptide (BNP)	808	<50 pg/mL
Creatine kinase (CPK)	316	<250 IU/L
C-reactive protein (CRP)	14.0	<0.5 mg/dL
Erythrocyte sedimentation rate (ESR)	20	<20 mm/Hr
Lactate dehydrogenase (LDH)	2005	<250 IU/L
Ferritin	204	24-336 ng/mL
Haptoglobin	<30.0	30-200 mg/dL
WBC	13.9	3.8-10.6 K/uL
Absolute lymphocyte count (ALC)	0.14	1.10-4.0 K/uL
Absolute neutrophil count (ANC)	13.62	1.80-7.70 K/uL
Hemoglobin (Hgb)	13.2	13.5-17.0 g/dL
Hematocrit	39.9	41-53 %
Platelets (plts)	52	150-450 K/uL
INR	2.68	<2.0
Prothrombin time (PT)	27.8	12.1-14.5 sec
Partial thromboplastin time (PTT)	44	22-36 sec
D-dimer	>20.0	<0.67 (age-adjusted) ug/mL
Fibrinogen	<60	200-450 mg/dL
Levetiracetam (Keppra)	57.6	12.0-46.0 mg/L

Imaging

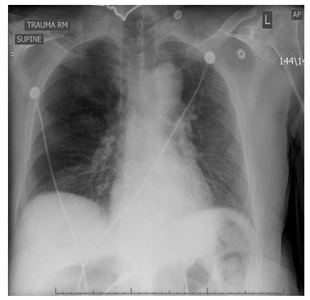

Figure 1. Chest x-ray image

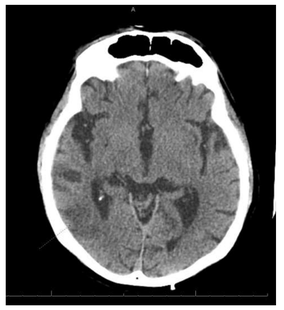

Figure 2. Computed Tomographic image of right inferior parietal lobe infarct

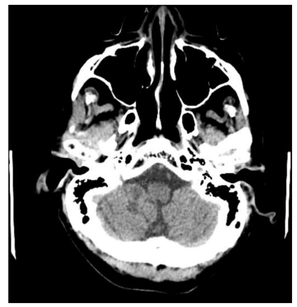

Figure 3. Computed Tomographic image of right inferior cerebellar infarct

Citations

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Case Follow-up

- Infectious disease determined patient was not a candidate for remdisivir or tocilizumab
- Patient clinically deteriorated with uptrending lactic acid, troponins, worsening renal failure, persistent DIC, and eventually into septic shock
- Nursing home Director of Nursing confirmed patient's previous desires to be DNAR
- Patient developed wide-complex tachycardia and passed away 2 days later