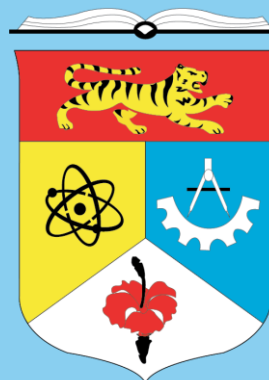


COVID Render Me Paralyzed!

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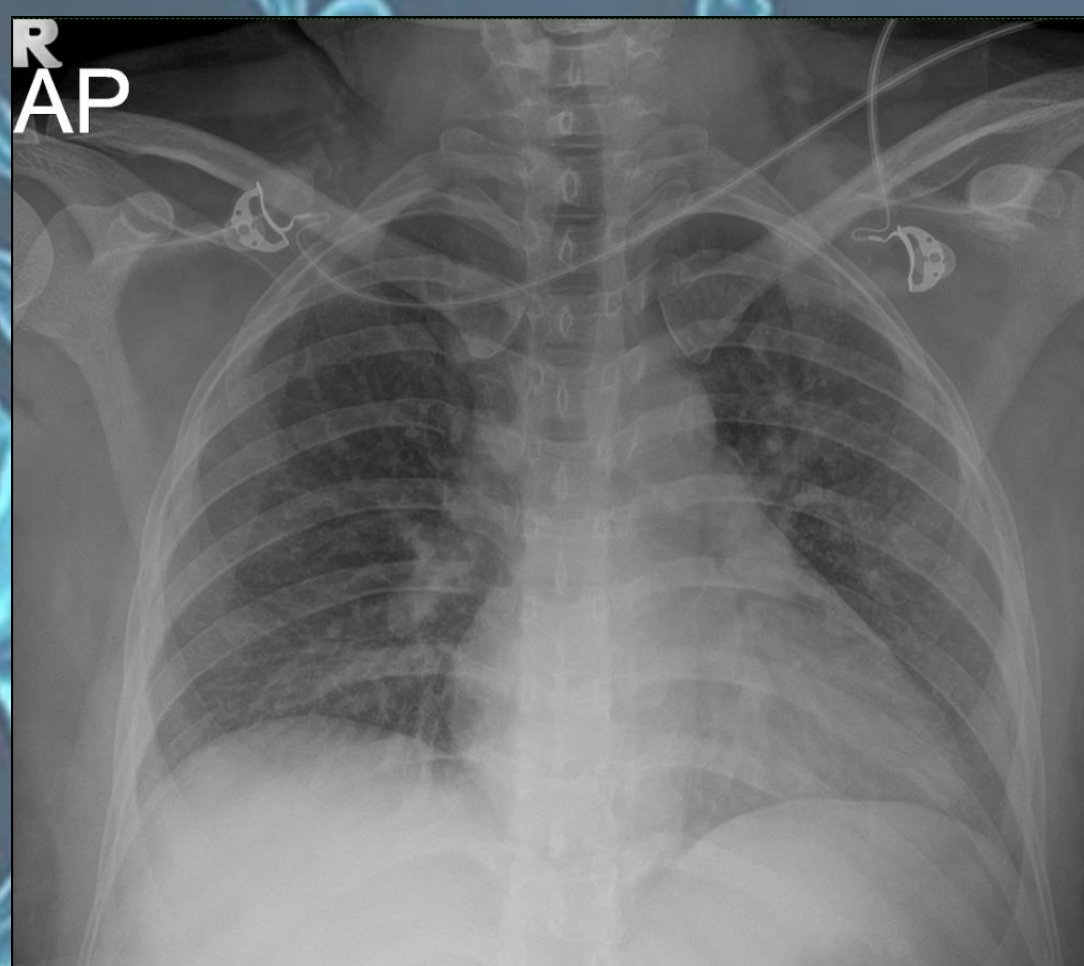
INTRODUCTION

Severe Coronavirus-19 (COVID-19) infection is known to increase risk for venous thromboembolism. Meanwhile, cerebral venous thrombosis (CVT) is an uncommon thromboembolic event with multiple etiologies. Extrapulmonary manifestations of COVID-19 is usually concomitantly appear with pulmonary symptoms. However, there is paucity of literature describing neurological manifestations in COVID-19 with the absence of pulmonary findings. We present a patient with focal neurological deficit and subsequently tested positive for COVID-19.

CASE REPORT

A 34-year-old lady with no past medical history presented to the ED with sudden onset of left sided body weakness that was associated with slurring of speech. It occurred while she was resting, and she denied any history of fall or trauma. She denied having any respiratory symptoms or headache. She revealed no known sick contact or COVID-19 contact, recent travel and took no oral contraception. She has no family history of autoimmune conditions. On arrival, she was tachycardic with a low-grade temperature of 37.9 and a blood pressure of 130/85 mmHg. She saturated well without oxygen supplementation. Physical examination showed facial asymmetry with left sided hemiparesis and upgoing left Babinski reflex. Stroke alert was immediately activated. CT brain plain and perfusion reported superior sagittal sinus thrombosis. Subsequently, she was started on subcutaneous enoxaparin. Chest X-ray done showed clear lung field.

During observation in ED, she developed 2 episodes of generalized tonic-clonic seizure that lasted for 1 minute and was aborted with intravenous (IV) diazepam. She was then started on IV Levetiracetam and the seizure was controlled. Her initial laboratory studies are shown in Table 1, revealed leukocytosis without lymphopenia, otherwise normal renal and liver functions. Other inflammatory markers were not raised. A nasopharyngeal swab testing for COVID-19 antigen yielded a positive result, and eventually confirmed with reverse transcriptase polymerase chain reaction (RT-PCR) for COVID-19. Throughout admission in the ward, she was fit free. Screening tests for a thrombophilic state were within normal range. She was fully recovered after 2 weeks of admission and was discharged well with oral anticoagulant.



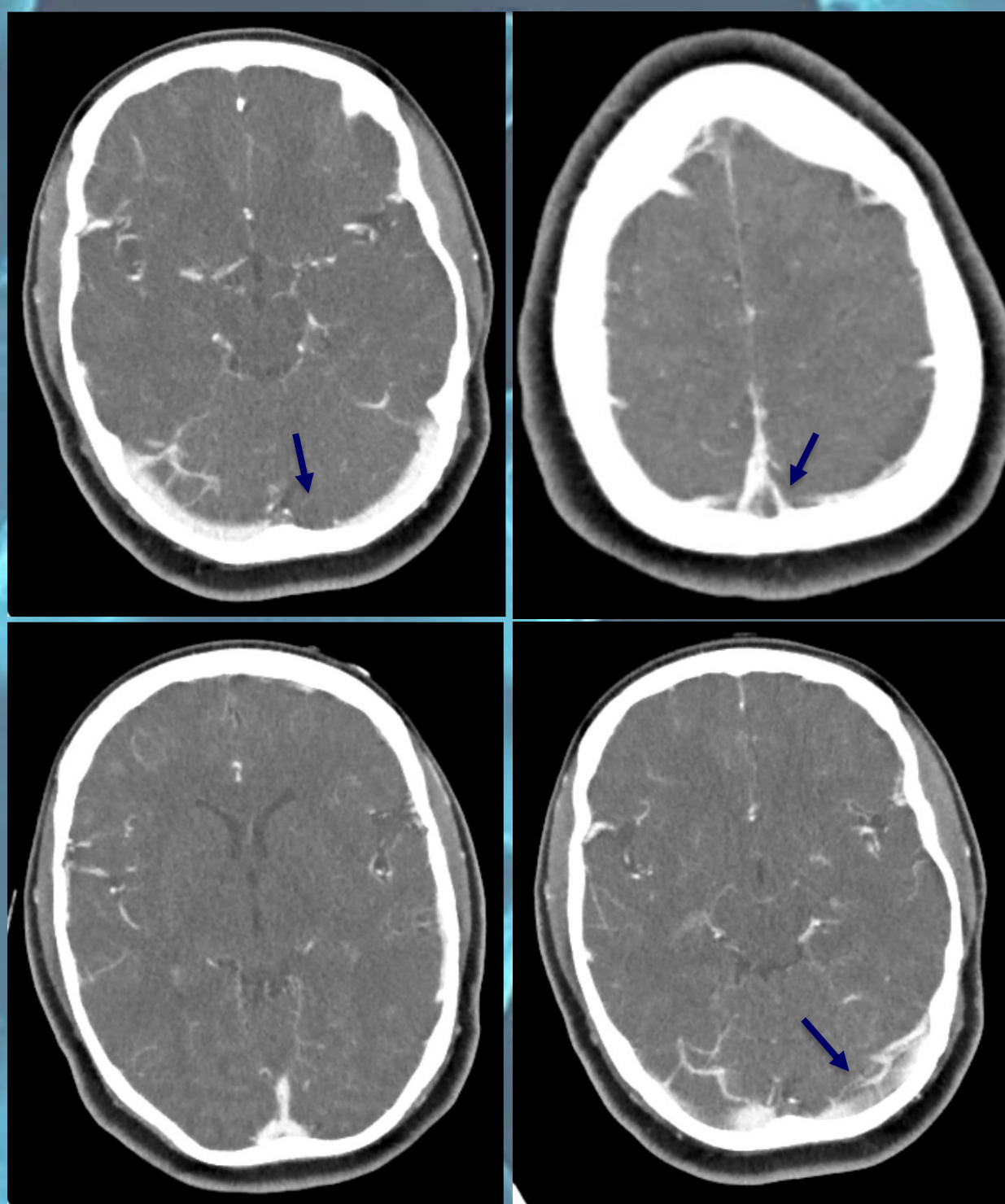
Chest X-Ray shows bilateral minimal peripheral haziness with reticular changes

DECLARATION OF CONFLICT

There is no conflict of interest.

DISCUSSION

CVT has been reported in literature as one of the emerging complications of COVID-19 infection, not as an initial presentation of the disease to emergency department (ED). The intricate link between hemostatic and immune system during the COVID-19 infection are likely to contribute to CVT. Pro-inflammatory state during the COVID-19 may result in immunothrombotic dysregulation leading to excess formation of vascular thrombi. It has known that the virus has neurotropic potential. It is suspected that the direct invasion of the CNS, as well as the cytokine storm contribute to the pathogenesis of CVT.



CT Brain showed hyperdensity of superior sagittal sinus and the cortical veins at the vertex with filling defects

CONCLUSION

Thromboembolic incidents are frequently occurred in COVID-19 infection. An understanding of the complex interplay between these conditions is necessary for the need to consider CVT in young patients presenting with focal neurological deficit during the COVID-19 pandemic in the absence of respiratory symptoms.

ACKNOWLEDGEMENT

Department of Emergency Medicine HCTM

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