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Ischemic Stroke in COVID-19 Patient: Case Report

Hind Elshareef, Jawaher Enani

King Saud University Medical city, Riyadh, KSA

Corresponding Author: Hind Elshareef

ABSTRACT

A lot of data are emerging regarding the prothrombotic tendency of COVID-19 which led to different guidelines regarding the use of thromboprophylaxis in such cases. We present a case of middle age man with COVID-19 presented with evolving ischemic stroke, he was not known to have risk factor and was successfully treated with thrombolytic therapy.

Keywords: SARS-CoV-2, COVID-19, thromboembolic disease

INTRODUCTION

COVID-19 disease has a wide range of presentation that varies from simple upper respiratory infection to severe ARDS. The novel corona-virus SARS-CoV-2 disease (COVID-19) pandemic, which started last December 2019 in China, is still spreading. [1] It is well established that patients with underlying comorbidities are people with highest risk of severe and life-threatening manifestation such as thromboembolic disease, sepsis and multiorgan failure. [2,3,4] thromboembolic phenomenon here is interesting as immune mediated thrombogenesis might not be unique but it is common in these patients and seen more frequently specially when found accidently as some of the patients screened for research purposes.

CASE REPORT

53 years old male, not known to have any medical illnesses, presented to ED complaining of right-side weakness, power in his limbs was 2/5, slurred speech and disorientation. He came to the hospital within 3 hours of starting of the symptoms. He has history of COVID 19 one month ago, along with his family his swab repeated again and came positive for COVID-19.On examination he was conscious oriented only to his wife not oriented to time nor place, mild dysarthria and had comprehensive and expressive aphasia. Motor system examination revealed only moderate pronation drift in rt upper limb and mild one in rt lower limb, sensory was intact NIHSS was 5 initial Ct brain was normal ASPECT score 10 and Ct Anigo does not show any stenosis or occlusion. Pt received Tissue plasminogen activator (tPA),0.9mg/kg, and transferred to ICU for Neuro monitoring. His vitals and neurological status were observed for 24 hours. The next day CT brain was repeated and revealed interval appearance of left basal ganglia acute infarction involving the lentiform nucleus, caudate body and anterior limb of the internal capsule [figure 1]. But his weakness was improving the power became 4/5. NIHSS score was 3. No new complains so he was transferred to the general word for further monitoring by our neuro physicians.

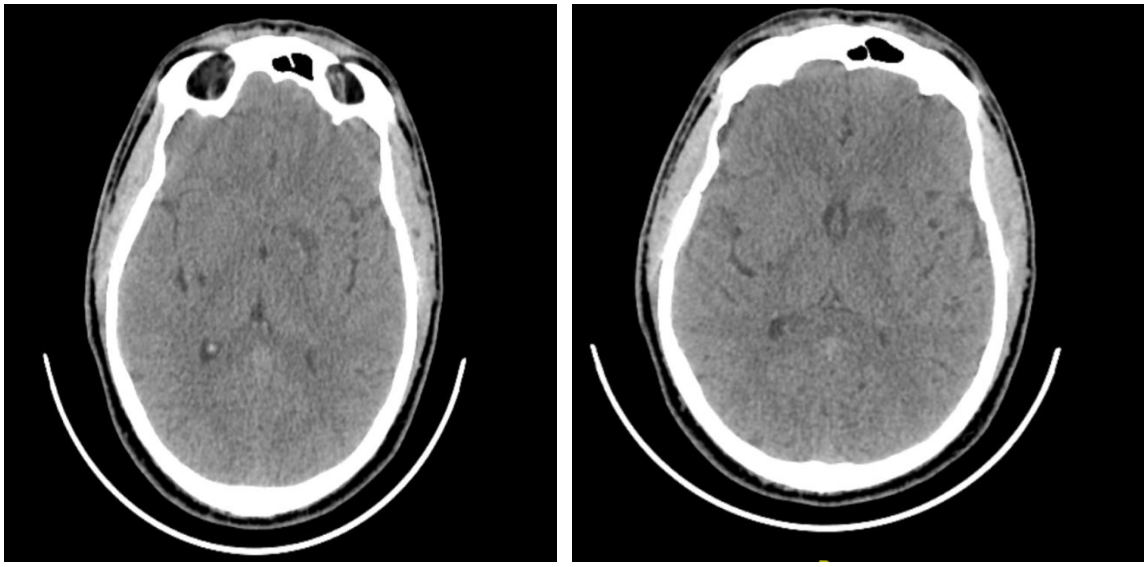

Figure 1: CT Brain

There is interval appearance of left basal ganglia acute infarction involving the lentiform nucleus, caudate body and anterior limb of the internal capsule.

Table 2: Sepsis-Induced Coagulopathy (SIC) score

Category	Parameter	0 point	1 point	2 point
Prothrombin time	PT-INR	≤1.2	>1.2	>1.4
Coagulation	Platelet Count ($x10^9$/L)	≥150	<150	<100
Total SOFA	SOFA four items	0	1	≥2

The total Sequential Organ Failure Assessment (SOFA) is the sum of the four items (respiratory SOFA, cardiovascular SOFA, hepatic SOFA and renal SOFA)

DISCUSSION

SARS-CoV-2 is a single-strand RNA coronavirus, which enters human cells mainly by binding the angiotensin-converting enzyme 2, [6] which is highly expressed in lung alveolar cells, cardiac myocytes and the vascular endothelium. Dutch study by Klok F et.al was able to recruit 184 patients with covid-19 pneumonia admitted to an intensive care unit (ICU) found a 49% cumulative incidence of thrombotic complications, including arterial and venous thrombosis. [5] So, it is safe to say it is common finding that require attention and serious consideration when we deal with a massive pandemic like this.

Although the exact mechanisms of COVID-19 induced thrombosis is not well established, it is agreed that Covid-19 causes massive inflammation boosting cytokines, which increase the liver's production of clotting factors in particular fibrinogen which is remarkably elevated in covid-19 patients leading to amplification of the coagulation process. [7] Another theory is activation of endothelial cells is thought to be the primary driver for the increasingly recognized complication of thrombosis. Viral inclusion bodies have been identified in endothelial cells in a variety of organs, from lung to gastro-intestinal tract. [11] Furthermore, prolonged hospital stay, and pre-existing comorbidities can contribute to venous thromboembolism. Knowledge of the incidence of this complication in COVID-19 is important for justification of high dose thromboprophylaxis, especially in patients admitted to the intensive care unit (ICU). There should be a high suspicion for thrombotic complications and low threshold for definitive imaging, where possible. Regarding the thromboprophylaxis dose it is still an area of debate that need more study

and research. Study by helm j et.al, recruited 184 ICU patients with proven COVID-19 pneumonia all patients were receiving at least standard doses thromboprophylaxis. But still there was 31% incidence of thrombotic complications in ICU patients with COVID-19 [10] which led to their recommendation of increasing the prophylaxis towards high-prophylactic doses, e.g. going from enoxaparin 40 mg OD to 40 mg BID. few studies came up with the idea of risk stratification of patients and then selecting the doses accordingly as suggested by British Thoracic Society based on the study of Tang et al involving 449 patients with severe COVID-19, only 22% received heparin (at standard prophylactic dose in the majority). [8] survival was superior in patients receiving (prophylactic dose) heparin who had a Sepsis-Induced Coagulopathy (SIC) score ≥4.(table 1) [9] other way of risk stratification can be based on factors such as:

Location of patient's care (e.g. critical care)

Disease severity (e.g. need for CPAP, high oxygen requirements (e.g. PaO2/FiO2 ≤40 kPA (300 mmHg))

Clinical features suggestive of possible VTE

CONCLUSION

COVID-19 induced thrombosis is a common finding that has serious implication on patient's outcome. Higher doses of thromboprophylaxis might be the best option to avoid this complication. Clearly clinical trials are needed to establish a better risk stratification and clinical management.

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Declaration of conflicting interest

The authors declare no conflicts of interest in preparing this article.

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