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Short Communication

Medical research

Systemic precipitation of underlying arterial thrombosis in COVID-19 infected patients

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ABSTRACT

Pandemic COVID-19 has brought forward a wide array of organ-specific and systemic disorders, one such being raised susceptibility towards development of systemic coagulopathies (venous, arterial or microvascular thrombi), which often contributes to the high incidence of thrombotic complications, such as deep venous thrombosis, pulmonary embolism, associated with common thrombotic arterial complications (eg, limb ischemia, ischemic stroke, myocardial infarction) in critically ill patients. A similar picturealso seems to have been painted in our cases discussed below, whereby underlying SARS CoV 2 infection has predisposed patients to hypercoagulability-related disorders including thrombosis and other fatal vascular events. Making the start of prompt antithrombosis prophylaxis are necessary to ensure better recovery in both the pre-operative and post operative period and regular re-evaluation of basic investigations (PT-INR, aPTT, D-Dimer) to ensure timely action for any change, enabling optimal patient care and recovery.

Keywords: COVID-19; SARS-CoV-2; Thrombosis; Venous Thromboembolism

INTRODUCTION

The pandemic COVID-19 has brought forward a wide array of organ-specific and systemic disorders, both related to its predecessor SARS and some novel to itself. Though many researches are still going on with regards to extrapolation of inherent mechanisms and subsequent treatment, many grey areas still remain. One such feature being raised susceptibility towards development of systemic coagulopathies (venous, arterial or microvascular thrombi) in COVID-19 infected patients- symptomatic or otherwise. ACE2 expressed in host target cells, particularly alveolar

epithelial type II cells is the site of adhesion of this virus. In later stages of infection, when viral replication accelerates, epithelial-endothelial barrier integrity gets compromised. In addition, SARS-CoV-2 also infects pulmonary capillary endothelial cells, accentuating the inflammatory response and triggering an influx of monocytes and neutrophils. Inflamed lung tissues and pulmonary endothelial cells may result in microthrombi formation and contribute to the high incidence of thrombotic complications, such as deep venous thrombosis, pulmonary embolism^[2], with common thrombotic arterial complications (eg, limb ischemia, ischemic stroke, myocardial infarction) in critically ill patients.

A similar picture is also seen to have been painted in cases discussed below, albeit the COVID-19 positive status in the first and last two cases was diagnosed postoperatively. We can make a safe presumption that subclinical COVID-19 infection most likely had precipitated the acute onset of ischaemic stroke and right sided hemiparesis in those cases, with no other underlying risk factors for the same. Whereas, the rest cases are middle aged males, two of them with known comorbidity (Type II DM) were bought to our hospital with complaints of acute onset chest pain or AMI (acute myocardial infarction), with no previous history of similar complaints in the past, which can also be assumed to have been precipitated by underlying SARS CoV 2 infection which is known for predisposing patients to hypercoagulability-related disorders including thrombosis and even fatal vascular events.

Additionally, platelets play an eminent role in primary hemostasis. The adhesion of platelets to subendothelial collagen and von Willebrand factor, and the subsequent aggregation of platelets via fibrinogen cross-bridging, is the basis for initial hemostatic plug formation after vascular disruption. Platelets are also the hemostatic element primarily involved in arterial thrombotic diseases, and to a lesser degree in the development of venous thrombosis^[3]. The majority of anaesthetic agents, including intravenous induction agents, volatile anaesthetics, and local anaesthetics, have been reported to inhibit platelet function as reported by various studies. The magnitude of this inhibitory effect tends to vary amongst different agents. Hence it remains possible that an anaesthetic regimen with potent antiplatelet actions could lead to bleeding complications in a patient who is genetically predisposed, whose environment contains additional haemorrhagic risk factors (e.g., hemodilution, anticoagulants), or who is undergoing a procedure that involves a high risk for bleeding complications (e.g., neurosurgery)

At the same time the start of prompt antithrombosis prophylaxis are necessary to ensure better recovery in both the pre-operative and post operative period and regular re-evaluation of basic investigations (PT-INR, aPTT, D-Dimer) to ensure timely action for any changes enables optimal patient care and recovery.

However, these are only few such cases highlighting underlying thrombotic complications in a COVID-19 infected patient (Table 1), many more instances are required to be studied for designating a proper line of treatment against thrombotic complications in such patients in the future for a more holistic COVID-19 patient care.

The following seven case reports (Fig: Table 1) shares such a scenario and the steps taken with regards to its management.

Declaration of interests

None

Funding sources

None.

	CASE 1	CASE 2	CASE 3	CASE 4	CASE 5	CASE 6	CASE 7
Baseline characteristic s (age, sex, medical history)	60 years Male	57 year Male Type 2 DM	45 year Male	40 year Male Type 2 DM	50 year Male	57 years, Female Rheumato id arthritis	38 year, Male Hypertensi on
Antithrombot ic treatment at the time of arterial events	No	No	No	No	No	No	No
Covid infection: Diagnosis:	RT-PCR for SARS Cov2 positve,	RT-PCR for SARS Cov2 positve,	RT-PCR for SARS Cov2 positve,	RT-PCR for SARS Cov2 positve,	RT-PCR for SARS Cov2 positve,	RT-PCR for SARS Cov2 positve,	RT-PCR for SARS Cov2 positve,
Treatment:	Intubation on vasopressor s	Yes, Non-rebreather mask	Yes, Non- rebreath er mask	Yes, Non- rebreathe r mask	Yes, Non- rebreathe r mask	Intubation &on vasopress ors	Intubation &on vasopressor s

Table: 1: Tabular representation of studied cases:

			5(0)				
VTE associated	No	No	No	No	No	Pulmonar y embolism	No
Treatment	emergency left decompress ive craniotomy under general anaesthesia	Medical	Medical	Medical	Medical	emergenc y explorator y laparotom y	decompress ive craniotomy
Imaging exam	CT Brain (plain) MRI Angiograph y	No	No	No	No	CECT abdomen and pelvis	MRI Angiograph y
Symptomatol ogy at the time of diagnosis	acute onset right sided weakness, deviation of left angle mouth and inability to speak	severe chest pain since one day, retrosternal in origin and associated with sweating. Diagnosed as ACS/STEMI/A WMI- evolved/DM	retroster nal left sided chest pain, radiating to left arm and associate d with sweating Diagnos ed with IWMI	complain s of retrostern al chest pain, radiating to neck and back, associate d with sweating since last 12 hours. diagnosed with ACS/IW MI- evolved/ DM	with complaint s severe chest pain since one day, retrostern al in origin and associate d with sweating Diagnose d with ACS/IW MI- evolved	complaint s of pain over abdomen and constipati on since 1 week	complaints of giddiness and left sided hemiparesis since 3 days and GCS 4/15
Days from disease onset to thrombotic event	2	0	0	0	0	7	3
Arterial thrombosis Thrombus localisation	complete occlusion of left internal carotid artery and poor re- canalisatio n of left middle cerebral artery on MR- Angiograph y	NA	NA	NA	NA	thrombosi s of superior mesenteri c artery and few of its branches on the right side with dilatation of multiple small bowel loops	MRI angiograph y large right MCA and ACA territory infarct in right femoral, temporal and parietal lobes, right ganglio- capsular region and anterior thalamus.

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