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Acute Mesenteric Ischemia in Severe Coronavirus-19 (COVID-19): Possible Mechanisms and Diagnostic Pathway

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Thromboembolic complications are being increasingly recognized in coronavirus-19 (COVID 19) pneumonia (1). Apart from deep venous thrombosis and pulmonary embolism (PE), acute mesenteric ischemia (AMI) has been reported in severe COVID-19 patients (2). AMI is a devastating complication with high mortality rate, so high suspicion, early recognition, and timely treatment is essential to avoid morbidity and mortality associated with this disorder.

The exact pathological mechanism underpinning the complication of AMI in COVID-19 is not known at present. Putatively, four mechanisms, in isolation or in varying combinations could account for this fulminant complication in severe COVID-19. First, a coagulation disorder (hypercoagulability) induced by systemic inflammatory state, endothelial activation, hypoxia and immobilization may lead to mesenteric vascular thrombosis. The evidence available at present has not conclusively demonstrated large mesenteric vessel (arterial or venous) thrombosis. Preliminary pathological evidence has shown bowel necrosis with small vessel thrombosis involving the submucosal arterioles, thereby pointing to an in-situ thrombosis of small mesenteric vessels rather than an embolic event (2).

Second, elevated levels of von Willebrand Factor have been reported in severe COVID-19. von Willebrand Factor is released from Weibel-Palade bodies in response to endothelial damage. Vascular endothelium expresses angiotensin converting enzyme 2, the target receptor for severe acute respiratory syndrome 2 (SARS-CoV-2), which possibly explains the endothelial cell tropism of SARS-CoV-2 and subsequent endothelial dysfunction or damage with resultant vascular thrombosis (3).

Third, expression of angiotensin converting enzyme 2 on enterocytes of small bowel, the target receptor for SAR-Cov-2, may result in intestinal tropism and direct bowel damage.

Lastly, shock or hemodynamic compromise which is commonly associated with severe COVID-19 pneumonia may lead to a nonocclusive mesenteric ischemia.

A vigilant and systematic approach is needed to suspect, diagnose and manage this otherwise fatal complication of severe COVID-19.

Patients with severe COVID-19 complicated by AMI may present with abdominal pain, nausea/vomiting, diarrhea, abdominal distention or worsening systemic status (sepsis).

Blood tests may reveal elevated lactate levels and fibrin degradation products (D-dimer). However, both these tests are nonspecific and may be elevated in severe COVID-19 without AMI (4).

Imaging has a vital role to play in timely detection of AMI and is the mainstay of diagnosis.

Although readily available, abdominal radiographs lack sensitivity and specificity. Ultrasound avoids the use of ionizing radiation but is also nonspecific. Computed tomography angiography (CTA) is the imaging study of choice to diagnose AMI. CTA is usually performed to detect PE in severe COVID-19 patients. CTA done for detection of PE may need to be extended to cover the abdomen so that both the chest and abdomen are scanned in the same examination. This might come at the cost of higher radiation dose but given the seriousness of AMI the trade-off is worth it.

On CT, thick-walled, edematous and dilated bowel (>3 cm) should raise the suspicion of AMI. Presence of pneumatosis intestinalis or portal venous gas suggests bowel ischemia. But presence of pneumatosis must be interpreted with caution as it may be present secondary to mechanical ventilation in patients of severe COVID-19. Nonenhancing thick bowel suggests bowel infarction. Frank perforation presents as discontinuity of bowel wall with localized air containing collection (5).

In conclusion, precise knowledge of the occurrence of AMI in COVID-19 patients is essential for ordering appropriate diagnostic testing at a low threshold level and quick decision making with regard to intensity of thromboprophylaxis to reduce the morbidity and mortality associated with this disorder.

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