Ischemic colitis after COVID-19 mRNA vaccine

An 82-year-old woman with a history of hypertension and osteoarthritis was admitted with abdominal pain and hematochezia that developed 7 days after she had received her second COVID-19 mRNA vaccination (BioNTech, Germany). Severe lower abdominal tenderness was present with decreased bowel sounds. Abnormal laboratory findings were leukocytosis (18 000/µL; normal 4500–10 500/µL) and increased C-reactive protein (50 mg/dL; normal < 5 mg/dL). Abdominal ultrasound revealed massive edema of the colon but no atherosclerosis of the aorta or narrowing of the pelvic arteries. Sigmoidoscopy revealed deep ulcers, luminal narrowing, and frank mucosal necrosis starting at the rectosigmoid and extending into the entire sigmoid colon (Video 1, Fig. 1). Additional extensive evaluation including stool cultures and studies for viral and autoimmune diseases did not reveal any other cause for the ischemic colitis. Histology showed frank necrosis with lymphocytic infiltration of the lamina propria, in addition to occluded vessels with inflammatory infiltrates, without eosinophilia or pseudomembranes. The patient was treated with intravenous fluids and metronidazole for 5 days. Her symptoms slowly subsided and she was discharged home on day 7. The temporal relationship, in the absence of any other inciting factors, medications, or abnormal anatomic vessel abnormalities, suggests that the causative event for the ischemic colitis was the mRNA vaccination. The SARS-CoV-2 spike is a key pathogenic factor of this novel coronavirus, allowing the virion to attach itself to the cells and then deliver its genetic contents inside the cytoplasm [1]. Some COVID-19 vaccines are a form of genetic therapy that use nanoparticles carrying mRNA to transfect the cell introducing the genetic material encoding the S-spike inside. SARS-CoV-2 spike protein dysregulates prothrombin and fibrinogen chains [2]. The S-spike also phosphorylates ACE2, leading to
MAPK signaling activation with phosphorylation of Erk, p-38, and JNK and subsequent platelet activation, release of coagulation factors, and secretion of inflammatory cytokines [2]. This inflammatory state may further enhance the thrombophilia per se or in the setting of vaccine-induced thrombocytopenia, autoimmune vasculitis or result in arterial or venous thrombosis in different parts of the body.

**Competing interests**

The authors declare that they have no conflict of interest.

**References**


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