Severe Intraoperative Shock Related to Mesenteric Traction Syndrome

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Mesenteric traction syndrome is defined as arterial hypotension, facial flushing, and tachycardia related to mesenteric traction. We describe a case of mesenteric traction syndrome refractory to catecholamine and vasopressin infusions. The patient, who had Crohn disease, developed severe distributive shock after mesenteric traction while undergoing resection of an intestinal inflammatory mass, accompanied by facial flushing and unaltered readings for pulse oximetry, capnography, and bispectral index monitoring. The absence of tachycardia in this case was attributed to long-term use of timolol. Arterial pressure returned to baseline level after approximately 30 minutes, and the postoperative period was uneventful. (A&A Case Reports. 2017;8:51–54.)

Mesenteric traction syndrome (MTS) is defined as transient arterial hypotension, facial flushing, and tachycardia because of mesenteric traction.[1] Its pathophysiology includes an increase in prostacyclin serum levels, which causes the hemodynamic changes and facial flushing.[2] We describe a severe case of MTS during colorectal surgery that was refractory to intravenous (IV) infusions of catecholamine and vasopressin.

CONSENT FOR PUBLICATION

The patient reviewed the case report and gave written permission for the authors to publish the report.

CASE DESCRIPTION

A 66-year-old woman who had been undergoing pharmacological treatment for Crohn disease (Montreal phenotypic classification A3L3B2) for 4 years was scheduled for intestinal resection. Abdominal computed tomographic scans showed stenosis of the rectosigmoid junction, parietal stenosis, and thickening of the terminal ileum, including the ileocecal valve, with ileoileal and ileosigmoid fistulas. During the preoperative briefing, the surgeon stated that the extent of the intestinal resection would be defined during the procedure and that total colectomy was a possibility.

During a physical examination, the patient was found to have glaucoma and right eye amaurosis because of traumatic retinal detachment. She had no history of allergies or smoking. Her surgical history included an uneventful cesarean delivery, retinopexy, and humeral osteosynthesis. Her medications for chronic conditions included azathioprine (150 mg/d), infliximab (100 mg every 8 weeks), omeprazole (20 mg/d), folic acid, cyanocobalamin, calcitriol, and timolol eye drops (stopped the night before surgery). A laboratory workup showed the following findings: hemoglobin level 11.3 g/dL, hematocrit value 32%, leukocyte count 36 000 cells/mm^3, and serum albumin level 3.1 g/dL. Electrocardiography, transthoracic echocardiography, and chest radiography showed no abnormalities.

Once in the operating room, the patient underwent cardioscopy and pulse oximetry. In addition, a noninvasive blood pressure measurement was taken, and cortical electrical activity was measured with a bispectral index (BIS) monitor (Aspect Medical Systems, Norwood, MA). A peripheral 18-G IV catheter was placed in the right arm. Ciprofloxacin (400 mg) and sufentanil (5 µg) were administered. An epidural catheter was placed in L1-L2, and 0.5% ropivacaine 10 mL and fentanyl 100 µg were administered. General anesthesia was induced with fentanyl (250 µg IV), propofol (120 mg), and cisatracurium (8 mg). The trachea was intubated with a 7.5-mm cuffed tube via a King Vision Video Laryngoscope (King Systems, Noblesville, IN). Hydrocortisone (300 mg IV) was then administered. Ultrasound-guided internal jugular vein puncture was performed by inserting a double-lumen 7F catheter. Metronidazole (1.5 g) was administered intravenously. Anesthesia was maintained with sevoflurane (end-tidal concentration 2.0%), and muscle relaxation was maintained with IV boluses of cisatracurium (2 mg).

Ten minutes after mesenteric traction was begun, the patient's arterial blood pressure (ABP) started to decrease progressively from 95/60 to 70/45 mm Hg. In an attempt to stabilize the ABP, we administered 1 L IV colloids and 10-mg IV boluses of ephedrine (total dose 50 mg); despite these actions, the ABP decreased to 45/30 mm Hg within the next 15 minutes.

The central venous pressure also decreased to 2 mm Hg (initially 11 mm Hg), but the end-tidal CO_2 level remained unaltered (30 mm Hg). Intense facial and peripheral flushing also was noted. No electrocardiographic or heart rate abnormalities were observed. Two 10-µg IV boluses of epinephrine were administered, but mean arterial pressure (MAP) increased by <10%. The Figure shows the timeline of events.

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The possibility of distributive shock was considered, and an IV infusion of norepinephrine (0.1 µg/kg/min) was started and titrated. A catheter was inserted into the left radial artery for continuous monitoring of ABP and for drawing blood. At that point, MAP was 38 mm Hg, despite norepinephrine infusion (1 µg/kg/min). An IV infusion of vasopressin (0.08 U/min) was started via an Infusomat pump (B. Braun Melsungen AG, Melsungen, Germany), which resulted in a slight increase in MAP (approximately 10%). An arterial blood gas analysis showed a significant reduction in the Pao_2/fraction of inspired oxygen ratio (87.3) and respiratory acidosis (pH—7.25/$Paco_2$—56.6/HCO_3—20.8). Electrolyte values were normal.

Sufficient sevoflurane was administered to maintain a BIS of 60. Mean urinary flow was 2.4 mL/kg/h. Considering the patient's severe hemodynamic instability, the surgeon decided to perform a Hartmann colostomy instead of primary reconstruction to shorten the surgical time.

Forty minutes after the onset of shock, the MAP started to increase progressively. Infusion rates were titrated until the MAP reached 65 mm Hg, which occurred approximately 1 hour after the onset of the initial symptoms. When hemodynamic stability had been maintained for 30 minutes, morphine (2 mg) and 0.2% ropivacaine (16 mg) were administered through the epidural catheter. Dipyrone (2 g) and ondansetron (8 mg) also were administered intravenously.

The degree of neuromuscular blockade was monitored with acceleromyography (TOF-Watch; Organon Ltd, Dublin, Ireland). Atropine (0.75 mg) and neostigmine (1.5 mg) were administered intravenously, and when the T4/T1 ratio reached 1.0, tracheal extubation was performed. At that time, the patient's BIS was 97, her MAP was 77 mm Hg, and her heart rate was 79 bpm. She had an uneventful recovery in the postanesthesia care unit.

The surgical specimen obtained included the terminal ileum (65 cm), cecum (18 cm), right colon, and part of the sigmoid colon (17 cm). Intense adhesions were present, along with an abundance of mesentery covering the intestinal tract (mostly the terminal ileum). Histopathologic analysis of the ileum showed narrowing, wall thickening, and alterations in color and texture at different points throughout the interior, which is consistent with active Crohn disease. There was no sign of abscess.

DISCUSSION

MTS is defined as transient arterial hypotension, facial flushing, and tachycardia because of mesenteric traction.[1] In the present case, the MTS diagnosis was established on the basis of clinical features and risk factors. Anaphylaxis to latex was considered in the clinical differential diagnosis of distributive shock. There were no risk factors in the patient's medical history to support this, however, and the patient improved despite exposure to latex; therefore, this possibility was ruled out. The possibility of toxin release from an abscess after mesenteric manipulation was excluded because neither computed tomography nor histopathologic examination demonstrated abscess, and postoperative evolution was uneventful.

The remarkable lack of associated significant tachycardia in this case of MTS may be explained by the patient's long-term use of timolol eye drops, which, despite being administered topically, may have had systemic cardiovascular effects.[3] We considered the possibility that a sympathetic block related to the epidural block might have contributed to the drop in ABP; however, the authors of

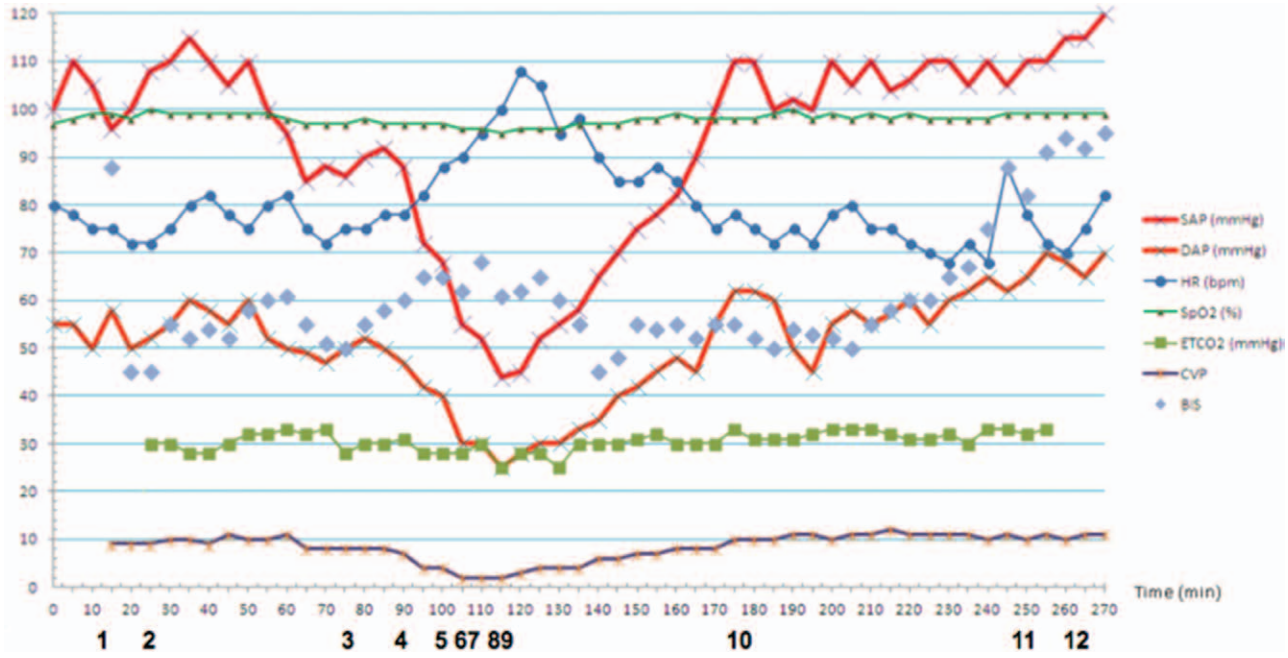

Figure. Timeline of events. BIS indicates bispectral index; CVP, central venous pressure; DAP, diastolic arterial pressure; $ETCO_2$, end-tidal CO_2; HR, heart rate; SAP, systolic arterial pressure; Spo_2, arterial oxygen saturation. 1, Epidural block (0.5% ropivacaine 10 mL plus fentanyl 100 µg). 2, General anesthesia induction. 3, Mesenteric traction. 4, Fluid administration. 5, Ephedrine bolus 10 mg IV (×5). 6, Epinephrine bolus 10 µg IV (×2). 7, Norepinephrine infusion initiated. 8, Left radial artery catheter placement. 9, Vasopressin infusion initiated. 10, Vasopressin infusion interrupted. 11, Norepinephrine infusion interrupted. 12, Tracheal extubation.

one study showed that receiving a supplemental thoracic epidural block was not associated with a greater reduction in ABP in MTS cases.[4] Considering that only 10 mL ropivacaine 0.5% was administered initially through the epidural catheter and that hemodynamic instability began >1 hour afterward, it seems unlikely that sympathetic block was a major contributory factor.

Before 1985, a hypotensive response to mesenteric traction was attributed to an autonomic reflex, such as afferent sympathetic stimulation, response to the retention of blood volume by the capacitance vessels, and direct cardiac vagal inhibition.[5–7] When these hemodynamic changes were assessed with pulmonary artery catheters during 20 aortic surgeries, investigators saw an increase in cardiac output and a decrease in systemic vascular resistance, sometimes lasting >30 minutes.[1] The average decrease in MAP was 20 mm Hg, but the values ranged from 3 to 57 mm Hg. Considering the 5- to 10-minute lapse between mesenteric traction and the onset of hypotension, a hormone-mediated mechanism was proposed.

In 1988, this hormone hypothesis was confirmed, and evidence appeared to indicate that prostacyclin may play a role in mediating the hemodynamic changes and facial flushing associated with MTS.[2] Not only was a significant increase in prostacyclin levels (measured as the metabolite 6-keto-$PGF_1\alpha$) seen after mesenteric traction, it also was shown that preoperative administration of ibuprofen could block this response. Despite this evidence, the role of neurologic reflexes was not excluded because there was also some degree of hypotension in the ibuprofen-treated group. A correlation between the 6-keto-$PGF_1\alpha$ serum levels and the degree of flushing was also confirmed.

In 1989, a research group showed that the levels of thromboxane B2, prostaglandin E2, and histamine are not increased significantly in patients undergoing abdominal aortic reconstructive surgery, further reinforcing the theory that prostacyclin plays a central role in MTS.[8] In addition, another study confirmed that ibuprofen prevents increases in prostacyclin levels.[9] Nevertheless, a delayed increase in thromboxane B2 levels was detected in the placebo group, which was considered a regulatory (vasoconstrictor) response to the increased synthesis and release of prostacyclin and vasodilation.

A more recent study showed that prostacyclin release causes not only arterial hypotension, facial flushing, and tachycardia but also a decrease in the Pao_2/fraction of inspired oxygen ratio, which is probably because of an increased intrapulmonary shunt related to vasodilation.[10] The compensatory hormonal vasopressor response also has been studied. Prostacyclin release was found to be associated with an increase in the serum levels of renin, arginine-vasopressin, epinephrine, and tromboxane A2 which contributes to the hemodynamic stabilization seen within approximately 30 minutes of mesenteric traction.[10]

The benefits of using a vasopressin infusion to treat vasodilatory shock have been well demonstrated. Vasopressin reduces norepinephrine requirements by maintaining cardiac output and oxygen delivery and consumption.[11] In cases of MTS, the absence of hemodynamic improvement during vasopressin infusion may be explained by the preexisting high levels of endogenous arginine-vasopressin,[12] which make additional doses ineffective.

In one reported case, a ketorolac infusion was used to successfully treat MTS,[13] and in a prospective study, a flurbiprofen infusion was used to treat this syndrome.[14] It is not clear, however, whether nonsteroidal anti-inflammatory drugs can treat MTS after it has already been established. In contrast, infusion of the opioid remifentanil has been shown to be a risk factor for MTS.[15]

Preoperative infusion of ibuprofen or flurbiprofen seems to be the best way to prevent or mitigate MTS. Preoperative use of H1 and H2 blockers may decrease the incidence of arrhythmias and reduce actions needed to reach hemodynamic stability.[16]

This case is not the first reported of severe MTS in a patient with an inflammatory bowel disease.[17] These diseases are characterized by an imbalance in proinflammatory mediators, such as tumor necrosis factor-α, prostaglandin I2 (PGI2), and prostaglandin E2, and local increases in leukocyte recruitment.[18,19] Some patients develop treatment tolerance and get clinically worse, with associated increases in interleukin-17 expression and serum PGI2 levels.[18–20]

Patients scheduled for surgical treatment of an inflammatory bowel disease are usually in an advanced stage of the disease; they present with a predominantly T helper-17 inflammatory response and increased concentrations of interleukin-17 and PGI2.[20] Although more studies are necessary, we suggest that this particular group of patients could be at a greater risk for massive releases of prostacyclin, which could potentially increase the incidence and severity of MTS.

MTS is a diagnosis of exclusion in the differential diagnosis of distributive shock occurring during intra-abdominal surgery because it currently is not feasible to determine prostacyclin levels intraoperatively in a timely fashion; however, the initial treatment of distributive shock does not require the diagnosis of MTS be made. The importance of recognizing MTS is that it is self-limited compared with other causes. Successful treatment intraoperatively does not mandate continued monitoring and treatment in the intensive care unit postoperatively. Also, the administration of anti-inflammatory agents intravenously may play a role in future management.

DISCLOSURES

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