Ulcerative Colitis After Anesthesia with Desflurane and Sevoflurane

To the Editor:

The pathogenesis of inflammatory bowel disease (IBD) remains unknown, but is likely multifactorial. Suspected factors include genotype, immune system dysregulation, intestinal barrier dysfunction, and microbial flora.[1] To our knowledge, no associations between IBD and inhalational anesthetics have been reported. Here we describe a patient who underwent anesthesia on separate occasions with desflurane and sevoflurane and on each occasion developed bloody diarrhea of several weeks duration. The patient was subsequently diagnosed as having ulcerative colitis (UC).

A 48-year-old man was admitted to our hospital with complaints of bloody diarrhea and weight loss. The patient said that he was having 12–13 episodes of diarrhea per day. His medical history was significant for two surgical procedures.

Two years prior to the present visit, at a hospital affiliated with ours, the patient had undergone nasal septoplasty for obstructive sleep apnea. According to the discharge summary, desflurane was used as the surgical anesthetic. The patient said that after the surgery he began to experience bloody diarrhea and went to a general practitioner who, without performing laboratory tests, diagnosed the diarrhea as infectious and prescribed a week-long course of antibiotics. The bloody diarrhea, however, persisted for ≈1 month

The patient's second operation was a lumbar discectomy that had been performed at our hospital 46 days prior to the present visit. During that operation sevoflurane was used. The patient said that on the day after surgery he began to have diarrhea. During the following 2 weeks the diarrhea increased in frequency and became bloody, and continued up to the time when he applied to our clinic.

On physical examination the patient's temperature was 38.5°C and his heart rate was 92 beats per minute. Other physical findings were normal except for a slight sensitivity to palpation of the abdomen. Laboratory findings were as follows: hemoglobin 9.9 g/dL, hematocrit 37%, leukocyte count 11.1×10^3/mL, platelets 392,000/μL, glucose 104 mg/dL (normal 70–105 mg/dL), aspartate aminotransferase 8 IU/L (normal 5–40 IU/L), alanine aminotransferase 7 IU/L (normal 5–40 IU/L), alkaline phosphatase 101 IU/L (normal 80–280 IU/L), gamma glutamyl transferase 31 IU/L (normal 10–50 IU/L), total protein 5.67 g/dL (normal 6.3–8.4 g/dL), albumin 3.34 g/dL (normal 3.8–5.1 g/dL), erythrocyte sedimentation rate 78 mm/h, and C-reactive protein 107 mg/dL (normal 0–8 mg/dL). Other laboratory findings were normal. Stool specimens were examined for ova, parasites, *Clostridium difficile* toxin, and *cytomegalovirus*; none of these were found. Stool cultures showed no pathogenic bacteria.

On colonoscopy, the mucosa in the descending colon, sigmoid colon, and rectum was edematous, granular, and fragile without interruption. In the lumen were seen mucus, exudates, and hemorrhagic secretions. The biopsy specimen showed inflammation at the lamina propria with infiltration by lymphocytes, plasmocytes, and eosinophils. The goblet cells were few in number. Crypt abscesses and morphologic distortion of the crypts were visible.

The patient was diagnosed as having severe UC and was hospitalized for treatment with mesalazine (oral and enema, 4 g/day each) and prednisolone (oral, 60 mg/day). By the fifth day of treatment the patient's frequency of defecation decreased to three per day, and blood and mucus were no longer apparent in the stools. One month later, follow-up colonoscopy showed mucosal healing.

A literature search revealed that a connection between IBD and inhalational anesthesia might be provided by interleukin-17. Fujino et al[2] found that in patients with IBD interleukin-17 expression was increased in the mucosa and serum compared to normal controls. More recently, Tylman et al[3] reported that in patients undergoing anesthesia with either sevoflurane and fentanyl or propofol and remifentanil, plasma interleukin-17 levels decreased in both groups during surgery, but after surgery the levels increased more rapidly in the sevoflurane group. The findings of these studies are difficult to interpret, given that among the events that lead to UC, it is not yet clear whether interleukin-17 should be considered a cause or an effect.[4]

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