CASE PRESENTATION
A 67-year-old man presented to the emergency room complaining of profuse diarrhea, tenesmus and urgency over a 24 h period. The diarrhea was initially nonbloody but had subsequently turned bright red in color. Past medical history included only type II diabetes mellitus with no known complications. He had no significant history of gastrointestinal disease. Medications included metformin and glyburide. There was no history of nonsteroidal anti-inflammatory drug use, and the use of metformin long preceded the diarrhea. The patient had no significant history of gastrointestinal disease. Medications included metformin and glyburide. There was no history of nonsteroidal anti-inflammatory drug use, and the use of metformin long preceded the diarrhea. The patient had no significant history of gastrointestinal disease.

DISCUSSION
Chemical colitis is not uncommon. Several reports of various substances causing colonic inflammation exist. These include soap, lye, ammonia, glutaraldehyde, potassium permanganate,
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Figure 1) Hydrogen peroxide proctitis. A Endoscopic appearance of the distal rectum demonstrating the ’snow-white sign’ and mucosal erythema and edema; B Linear erosions and white plaques in the proximal rectum

vinegar, chloroxylenol and chlorhexidine (11-14). While all these chemicals likely have a direct irritant and/or caustic effect on the mucosa, the mechanism of damage by hydrogen peroxide to colonic mucosa is unique.

The pathogenesis of hydrogen peroxide colitis has been reported to be secondary to penetration of the chemical into the mucosa with subsequent production of oxygen \((2\text{H}_2\text{O}_2 \rightarrow 2\text{H}_2\text{O} + \text{O}_2)\). Experimental models have shown that after seconds of exposure, the colon becomes distended, white and bloodless. Microscopically, minute gas cysts form as a result of oxygen production in both mucosa and submucosa within 1 min of exposure, followed by vascular congestion, focal hemorrhage and ulceration (15). Gas bubbles infiltrate the large veins and lymphatics and can be noted in the portal vein, right side of the heart, liver, lungs and inferior vena cava. Fatal gas embolisms have also been reported (15-17).

Reports of hydrogen peroxide colitis are sparse in the literature. In 1951, Pumphery (18) reported two patients with acute colitis following hydrogen peroxide enema. Sheehan (19) delineated the pathogenesis of the disease by animal experimentation, after describing another case of hydrogen peroxide colitis (19). By 1981, when Meyer et al (20) reported three more patients with hydrogen peroxide colitis, the use of hydrogen peroxide enema for various therapeutic and diagnostic procedures was already uncommon.

Renewed interest in this phenomenon occurred in the past two decades, after four reports of a total of 22 cases of inadvertent iatrogenic hydrogen peroxide exposure to the colon. All of these 22 cases were attributed to contamination of the air-water channels of the colonoscope (8-10,21). Most episodes occurred in relation with a ‘final rinse’ of the channels using 2.0% glutaraldehyde solution (in contrast to a previously used 0.2% solution) and were generally associated with inadequate rinsing of the channels. A second source of glutaraldehyde proved to be from within the tubing used to connect the water bottles to the endoscopes. Similar to the hydrogen peroxide cases, patients presented with tenesmus, bright red blood per rectum and abdominal cramping. Pathology was indistinguishable from ischemic colitis. Another relatively recent report (23) of glutaraldehyde colitis involved six cases that were attributed to an automatic scope-disinfecting machine. The authors stated that the problems could be avoided by repeatedly changing rinse water, force-air drying the channels and washing endoscopes before use (23). This was confirmed in a study in 2001 that found that after machine disinfection, there was no significant glutaraldehyde residue in the scope if the scope was soaked for 5 h in saline and if the channels were rinsed (24).

A more unfortunate aspect to this case is the anxiety state that led to the self-induced proctitis. Waiting lists for almost all procedures and consultations continue to grow in Canada, but a particular need has been identified in oncology (25-27). These waits are difficult to avoid under the current economic and resource limitations and, therefore, special attention should to be paid to the emotional burden that a new diagnosis of cancer may impose. Certainly in our patient, his belief was that his condition was imminently fatal and that two months was far too long to go with hormonal treatment alone. With appropriate follow-up and proper counseling as to the nature, course, prognosis and treatment of the specific disease, it may be possible to avert such potentially disastrous situations.

REFERENCES


Waiting-list induced proctitis: The hydrogen peroxide enema


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