An unusual case of severe colitis after colonoscopy

Dear Sir,

Colitis following endoscopy is extremely rare, and has only been described in patients with an underlying medical condition such as connective tissue disease1,2 or historically in cases of anaphylaxis to agents used to disinfect colonoscopes.3,4 This agent is no longer used in the cleaning cycle.

We describe a case of colitis in an otherwise healthy young female after a straightforward colonoscopy.

A 49 year old female attended our endoscopy department for a colonoscopy. Over the last few months, she had been complaining of symptoms of left sided abdominal discomfort and loose motions, with fatigue. There were no other symptoms of note, and her past medical history, family history and blood tests were unremarkable. She had been screened for connective tissue disease, and these tests were normal.

Bowel preparation over the preceding 24 h with two sachets of Picolax (sodium picosulphate), was uneventful. She was cannulated and given 2 mg of midazolam, and 25 mg of pethidine for sedation and analgesia. The endoscope was sterilised as per our department protocol using a glutaraldehyde-independent regimen.

She tolerated the procedure poorly, despite sedation, and colonoscopy could only be performed to the mid-transverse colon. The colonic mucosa was normal, and random biopsies were taken. The patient was discharged a few hours after the procedure uneventfully.

She subsequently returned to our emergency department that evening with acute onset severe, generalised abdominal pain, tachycardia, and pyrexia. Blood tests revealed a leucocytosis and anaemia. An erect chest radiograph was unremarkable, and stool microscopy and culture were later found to be normal.

An urgent CT scan of the abdomen and pelvis was arranged. This revealed abnormal thickening of the left colon, to splenic flexure level, with fat stranding (Fig. 1). There were no mass lesions, and the right colon was considered to be normal on imaging.

These appearances were considered to be consistent with an acute severe colitis by two independent radiologists.

The patient was admitted and managed conservatively, with intravenous fluids, nil by mouth and analgesia. She responded rapidly to this treatment, and all her symptoms resolved in 1 week. A repeat CT scan of her abdomen was then performed, and showed complete resolution of the previously noted abnormalities (Fig. 2).

She was discharged uneventfully and remains well one year later. Random biopsies taken at the original procedure showed normal colonic mucosa.

This sequence of events after lower gastrointestinal tract endoscopy has been reported previously in the literature, but only in cases with concomitant connective tissue disease and immunosuppression, or in the past when glutaraldehyde was used to disinfect endoscopes.1–5 The incidence of this condition is believed to be about 3–5%, based on retrospective analysis of patient questionnaires.6

In cases of connective tissue disease, the colitis has been suggested to be ischaemic in origin; it is thought that pre-existing disturbance to mesenteric blood flow due to connective tissue disease is made worse by the mechanical effects of colonoscopy on vessels in the colonic mesentery. This may then create a critically ischaemic state in the colonic mucosa, resulting in ischaemic colitis. This was described by Wheeldon et al., who performed mesenteric angiography, with normal results in such a case. Glutaraldehyde-induced colitis was a well-recognised phenomenon.3,4,6

Figure 1 Acute severe colitis, computerised tomogram on admission.
It is thought that inadequate removal of glutaraldehyde during a colonoscopy cleansing cycle allowed the agent to come into direct contact with the colonic mucosa, causing an irritant effect, resulting in colitis histologically indistinguishable from ischaemic colitis. Animal models have demonstrated that application of glutaraldehyde to bowel can cause colitic lesions similar to those observed in humans.7

Hsu et al. reported a series of 7 patients who developed this condition within 48 h of uncomplicated flexible sigmoidoscopies.8 Within a 2 week period, 6 patients developed this condition. A failure was found in the unit's sterilisation protocol, which had led to retention of glutaraldehyde in endoscopes.

Our case differs from previously reported episodes in that colitis occurred after a colonoscopy in a patient with no evidence of immunosuppression or connective tissue disease. Furthermore, glutaraldehyde is not used in our endoscopy unit.

The cause of the acute colitis in our patient is unclear. One possible mechanism of injury in our patient could be a reaction to an unidentified agent on the colonoscope.

Clinicians performing lower gastrointestinal tract endoscopy need to be aware that an acute severe colitis can occur in healthy patients, and in the absence of glutaraldehyde-induced injury. Fortunately, this condition appears to be self-limiting.

References

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