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Aphthous colitis induced by non-steroidal antirheumatic drugs

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NSAIDs are commonly used drugs. Their gastrointestinal toxicity is well known [1–7] but incompletely understood, and has led to the development of COX-2 inhibitors [8]. We describe the case of a young man with NSAID colopathy.

A 42-year-old banker was admitted to hospital with diffuse abdominal cramps and bloody diarrhoea after three days' intake of diclofenac (3 × 50 mg) for back pain. There was no history of previous disease or foreign travel. Stool cultures were repeatedly negative. The medication had already been discontinued.

Laboratory findings and abdominal sonography were normal. Upper endoscopy showed erosive inflammation of the duodenum. Coloscopy revealed a severely inflamed Bauhin's valve and aphthous lesions in the entire colon and especially the proximal segment. Biopsies demonstrated colitis with some neutrophils. No granulomas suggestive of chronic inflammatory bowel disease were present.

The diarrhoea subsided completely. At the patient's request we performed follow-up coloscopy after six weeks which showed complete normalization.

Gastrointestinal lesions following therapy with NSAIDs are common [1–4]. The connection between intake of NSAIDs and the development of gastric ulcers with and without *Helicobacter pylori* infection has been extensively discussed [4–17].

In contrast, little is known of NSAID colopathy [18–21] despite its apparent relative frequency. A British case control analysis showed that 74% of patients with newly diagnosed colitis had used NSAIDs previously [22].

In a 1987 study leukocyte scintigrams showed inflammation of the small intestine in ⅔ of rheumatic patients participating in the study [23]. Other studies described aphthous lesions [19], ulcers [18] and strictures [21].

In view of these facts, endoscopic diagnosis of aphthous lesions may pose a diagnostic problem when patients are on medication with NSAIDs. It is impossible to distinguish between pharmacologically induced colitis and Crohn's disease. It is, however, easier to differentiate it from ulcerative colitis because damage from NSAIDs is segmental, is not restricted to the colon and is localised mainly in the proximal part of the colon, in contrast to the distally located ulcerative colitis. An additional complexity arises from the fact that exacerbations of Crohn's disease induced by NSAIDs have been described in the literature [23–25]. For this reason, patients with inflammatory bowel disease are usually advised to avoid NSAIDs.

Hopes of using histology as the gold standard to distinguish Crohn's disease from NSAID colitis have not been fulfilled. The typical eosinophils and granulomas are found only in some biopsies from patients with Crohn's disease. Japanese and Belgian studies have concluded that histological differential diagnosis between Crohn's disease and other forms of colitis has only appoximately 86% sensitivity [26–28].

Pharmacologically induced colitis, especially NSAID colopathy, may exhibit a wide spectrum of histological injuries – ulcers, eosinophil colitis, collagenous colitis, lymphocytic, ischaemic colitis and mild unspecific colitis [29]. One sign pointing to NSAID colopathy is location in the proximal colon [30].

Considering all these facts, only the following criteria are useful for the diagnosis of colopathy induced by non-steroidal anti-inflammatory drugs:

- clinical symptoms
- exposure to NSAIDs
- endoscopic lesions with a predominance to the proximal colon
- regression after withdrawal of medication

In our case the rapid subsidence of clinical symptoms and endoscopic injuries after withdrawal of medication points to NSAID-induced colopathy. A first manifestation of Crohn's disease activated by NSAIDs cannot be ruled out and can only be shown during follow-up.

We advised the patient to avoid not only NSAIDs but also COX-2 inhibitors. Studies on gastrointestinal side effects of COX-2 inhibitors have shown that ulcers develop under COX-2 inhibitors as well [16, 17]. Moreover, there are signs that COX-2 inhibitors have selective effects only in the stomach and duodenum.

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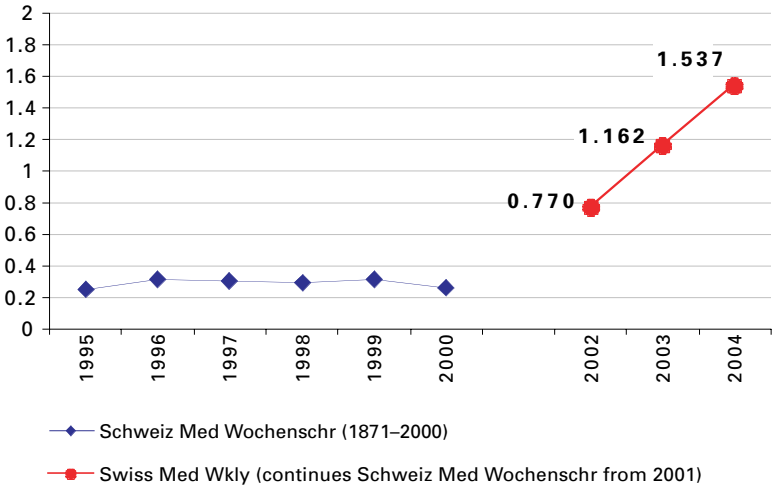

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