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Case Report

Diclofenac suppositories and acute ischaemic proctitis

N Arya, M J G Hawe, C Ozo

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Nonsteroidal anti - inflammatory drugs are widely prescribed because of their high efficacy as both anti-inflammatory and analgesic agents. The adverse effects of NSAIDs such as damage to the gastroduodenal mucosa, renal toxicity and bronchospasm are well known. In recent years, many cases have been reported of NSAID induced damage to the small and large intestine.[1,2] We report a case of acute ischaemic proctitis following diclofenac suppository.

CASE REPORT A fifty three year old healthy male patient was admitted for elective repair of his left inguinal hernia. He had a history of peptic ulcer symptoms following ibuprofen tablets, but no lower gastro-intestinal symptoms.

Under spinal anesthesia, Lichtenstein mesh repair of the inguinal hernia was performed. He had a 100 mg diclofenac suppository in the immediate post-operative period for pain relief, with no other medications given per rectum. There was no intra -operative or post-operative hypotension.

Five hours after the operation he complained of crampy abdominal pain, associated with six to seven episodes of haematochezia over the next twelve-hour period. He remained hemo-dynamically stable. Flexible sigmoidoscopy showed congested erythematous rectal mucosa with confluent erosions extending to 15 cm from the anal verge. The proximal rectal mucosa above 15 cm was macroscopically normal.

Coagulation screen, platelet count, CRP and ESR were within the normal range. The serum levels of Protein C and Functional Protein S were normal and a test for activated Protein C resistance was negative. The stool culture revealed no evidence of *Salmonella, Shigella, Campylobacter, Escherichia coli* 0157 or *Cryptosporidium oocyst* and it was negative for *Clostridium difficle* toxin A.

He was discharged home on prednisolone suppositories. Histology of the inflamed rectal mucosa showed widespread areas of acute ischaemic necrosis and ulceration which were limited predominantly to the superficial half of the mucosa. There were fibrin thrombi in the mucosal and submucosal capillaries (Figure). The biopsy of macroscopically normal proximal rectal mucosa showed no evidence of inflammation.

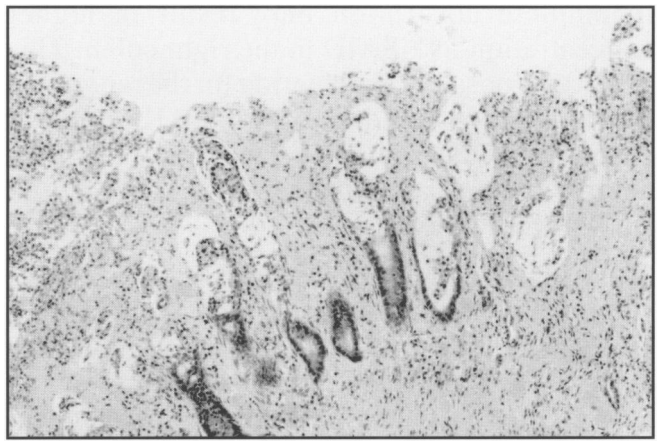

Fig Rectal biopsy showing necrosis of superficial half of the mucosa.

Prednisolone suppositories were subsequently discontinued and he remained asymptomatic. Eight weeks later, flexible sigmoidoscopy and biopsy showed complete clinical and histological resolution of the lesions.

Department of Surgery, Mid Ulster Hospital, Magherafelt, Co. Londonderry, BT45 5EX.

N Arya

M J G Hawe

Department of Pathology, Antrim Area Hospital, 45 Bush Road, Antrim, Co. Antrim, BT41 2RL.

C Ozo

Correspondence to Mr Arya, Department of Vascular Surgery, Level 5, Belfast City Hospital, Lisburn Road, Belfast BT9 7AB.

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DISCUSSION

The exact mechanism of NSAID-induced damage to the colonic/rectal mucosa is not fully understood. The inhibition of prostaglandin synthetase in the colonic mucosa causes decrease in the level of endogenous protective prostaglandins and increased production of Leukotriene B4.[1,3] Another significant factor may be due to local toxicity owing to the high local concentration of NSAID. Increased use of slow-release NSAID preparations associated with incomplete absorption may result in higher concentration of NSAID in the right colon. This provides a possible explanation for the report that the maximum incidence of NSAID-induced stricture and ulceration is on the right side of the colon .[1] It also explains the rectal lesions caused by NSAID suppositories[4,5]

There have been previous reports of NSAID suppositories induced rectal lesions presenting as tenesmus and bleeding.[4,5] The endoscopic appearance of erosions and ulcers progressing to stenosis may be related to the duration of treatment with NSAID suppositories. At endoscopy, the appearance of NSAID proctitis may be difficult to differentiate from infectious colitis or idiopathic inflammatory bowel disease.

A review of histology of 11 cases of NSAID-induced colitis suggests a remarkable similarity to ischemic colitis.[1] In other cases, it showed features of non specific colitis, with mixed inflammatory cell infiltrate and focal erosions.[2,4] Absence of crypt architectural distortion differentiates NSAID colitis from ulcerative colitis.[1]

In our case the pathological picture was classical of acute ischaemic proctitis. Infectious causes were excluded and there was no thrombogenic tendency or pen-operative hypotension. The absence of any other etiology, the rapid anatomic and histological resolution along with classical pathology suggest that the lesions were caused by diclofenac suppositories.

In spite of widespread use of NSAID suppositories, the number of cases of rectal lesions reported is small. It is quite possible that a large number of rectal lesions remains undetected or misinterpreted. NSAID should be considered as a cause of acute ischaemic proctitis and haematochezia in the appropriate clinical setting.

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