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Cyclosporine in the Treatment of Steroid Resistant Severe Ulcerative Colitis: A Word of Caution

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Abstract: Cyclosporine may be considered as state of the art treatment for steroid resistant severe ulcerative colitis. Up to 30-40% of the patients with severe ulcerative colitis will fail to respond to intravenous steroid treatment and would require surgical treatment. I report a case of steroid resistant severe ulcerative colitis, which failed to respond to cyclosporine due to presence of cytomegalovirus infection causing ischaemic colitis that had been seen on histopathological examination.
Keywords: Ulcerative colitis, Cyclosporine, Steroid resistant, Cytomegalovirus infection

INTRODUCTION

Cyclosporine may be considered as state of the art treatment for steroid resistant severe ulcerative colitis. Cytomegalovirus infection is well known to be associated with the use of cyclosporine in renal transplant patients [2]. I report a case of steroid resistant severe ulcerative colitis, which failed to respond to cyclosporine treatment and histological examination of colectomy specimen showed the presence of cytomegalovirus infection causing ischaemic colitis.

CASE REPORT

An 81-year-old gentleman presented to our unit with a 6-month history of diarrhoea. He was referred as an emergency because over the preceding 72 hours he had developed acute generalised abdominal pain associated with bloody diarrhoea 10 times per day. He had an extensive past medical history including hypertension, previous aortic bifircate graft, left ventricular failure and ischaemic heart disease. In fact he had been admitted the month earlier with chest pain and associated troponin rise. He was on maximal anti angina medication but at the time of admission he was on no treatment for his bowel condition.

On admission he was apyrexial, tachycardic (110 bpm) and his abdomen was generally tender but soft. Rectal examination revealed no abnormality but he was FOB +ve. Admission bloods were unremarkable except for his albumin 27 and CRP 83. Initial investigations were to exclude ischemic colitis, which included a CT abdomen and pelvis and a flexible sigmoidoscopy. The CT showed a pan-colitis and the sigmoidoscopy revealed quite severe inflammation throughout the left colon. Biopsies from this procedure showed non-specific colitis.

Treatment was commenced and, given his chequered medical history, it was felt that operative intervention carried an unacceptable risk. He was started on maximal dose 5-ASA and intravenous hydrocortisone 100mg 6 hourly. After an initial improvement he clinically deteriorated and predfoam enemas were introduced. However, his per rectal bleeding worsened to the extent that he was dropping his haemoglobin and precipitating his angina. At this stage intravenous cyclosporine was commenced, as he was still not improving on maximal steroid dose. During the next week he improved dramatically. His diarrhoea settled to twice daily with no bleeding and his albumin returned to his admission level of 27. Flexible sigmoidoscopy confirmed this clinical improvement, showing areas of regenerating mucosa. However, after the first week he deteriorated again, developing anaemia related angina, and a cardiac angiogram was carried out but found no lesion amenable to angioplasty. At this stage surgical intervention became the only possible option and despite the high risk involved a subtotal colectomy was carried out.

The histology of the colon revealed non-specific colitis with obvious ulceration, crypt abscess formation, and thickening of the sub mucosal layer with associated lymphocytic infiltrate (Fig. 1). This infiltrate was particularly evident in the perivascular regions and abnormal vessel walls were noted with cells displaying an “owl eyed” appearance (Fig. 2). When the specimen was stained to look for cytomegalovirus (CMV) multiple areas were positive (Fig. 3). This finding raised

the question as to whether this patient had CMV colitis from the start or whether it was a consequence of his immunosuppression, with steroids and cyclosporine, and if so whether the CMV infection led to the deterioration in his condition one week after cyclosporine was introduced.

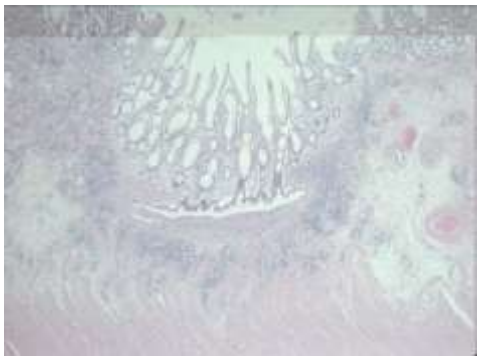

Fig. 1: Low power histology showing Non specific colitis

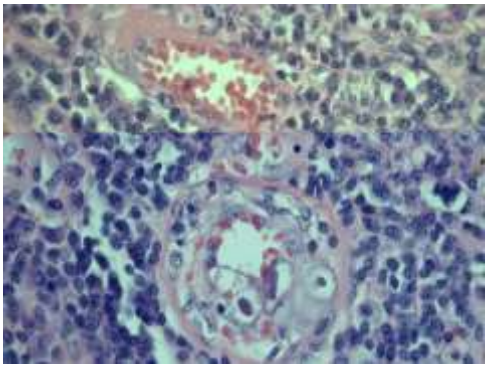

Fig. 2: Perivascular infiltration with cells displaying "owl eyed" appearance

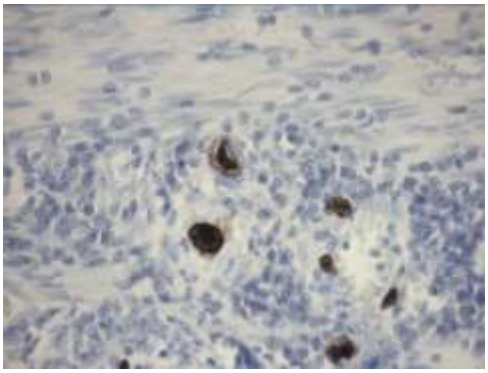

Fig. 3: Specimen stained for Cytomegalovirus showing multiple positive areas

DISCUSSION

Up to 30-40% of the patients with severe ulcerative colitis will fail to respond to intravenous steroid treatment and would require surgical treatment. Cyclosporine offers an alternative to colectomy for these patients. Intravenous cyclosporine induces remission with in 14 days in 50-80% of patients, who fails intravenous corticosteroid. Long-term response rates for responding patients are 40-60% [1, 4, 5]. Serological evidence of secondary cytomegalovirus infection in renal transplant recipients receiving cyclosporine A occurs in 33.3% of patients and in 17% they are clinically symptomatic [2]. Granciclovir can be used for the treatment of cytomegalovirus colitis [3]. In the above case report patient initially responded to intravenous cyclosporine treatment but clinical relapse occur within one week of commencement of cyclosporine treatment and histological examination of colectomy specimen showed cytomegalovirus infection causing vasculitis and ischaemic colitis. Preoperative biopsy did not show any evidence of cytomegalovirus infection. I believe that the cytomegalovirus colitis contributed in the progression of disease resulting in failure to cyclosporine treatment.

CONCLUSION

I conclude that the patients with steroid resistant severe colitis receiving cyclosporine treatment should be monitored closely for cytomegalovirus colitis.

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