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Case Summary
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Case Report: Herpes Simplex Virus Causing a Flare of Ulcerative Colitis

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Abstract

Ulcerative colitis is a relapsing-remitting inflammatory bowel disease. We present a case of a flare of proctitis due to primary herpes simplex 2 infection. We then outline the most common causes of proctitis, particularly infectious proctitis, highlighting the importance of obtaining a detailed sexual history in all patients with a flare of inflammatory bowel disease. Finally, we discuss the considerations and challenges of balancing immunomodulation and antiviral therapy in a viral flare of inflammatory bowel disease.

Case Presentation

A 24-year-old man presented with four days of intermittent fevers >38°C and frequent bloody diarrhoea. This was on a background of a five-year diagnosis of Ulcerative Colitis (UC) with known moderate rectosigmoid involvement, initially treated with oral and Per Rectal (PR) mesalazine, which he had subsequently self-ceased.

A month prior to presentation, he developed PR bleeding and mucous stools. Outpatient colonoscopy revealed mildly active chronic proctitis. He was treated with a course of prednisolone and recommenced mesalazine. After initial partial improvement his symptoms worsened.

Blood tests demonstrated elevated C-Reactive Peptide (CRP) (peak of 82.2 mg/L) and abnormal liver enzymes (bilirubin 9, ALT 123, AST 63, GGT 125, ALP 63) but normal leukocyte count. Abdominal X-ray was unremarkable. Blood, urine and faecal cultures were negative. Flexible sigmoidoscopy detected severe Mayo 3 colitis in the distal 5 cm of the rectum only, with a dusky appearance and deep ulceration with mucopurulent exudate not typical of UC (Figure 1). Subsequent computed tomography scan excluded perforation and ischaemia.

He was empirically treated with intravenous ceftriaxone, metronidazole and hydrocortisone. Azathioprine was commenced after several days. Whilst his temperatures defervesced and CRP improved, he had ongoing symptoms, prompting consideration of alternative diagnoses. Subsequent questioning revealed an episode of unprotected receptive anal sex with a male a fortnight prior to the flare. Testing for sexual transmitted infections returned positive results for Herpes Simplex Virus 2 (HSV2) both on polymerase chain reaction testing of the biopsy and on serology (IgM and IgG positive). HIV testing was negative. Valaciclovir was commenced on day 7 and the patient's condition improved. Salvage therapy with infliximab was started four days later due to persistent rectal pain and PR bleeding. HSV immunostaining requested retrospectively on biopsies from time of initial diagnosis was negative. Review of biopsies confirmed chronic architectural changes consistent with UC. Thus a diagnosis of flare of UC due to primary HSV2 infection was made. After treatment with induction infliximab and a six-week course of antivirals, repeat colonoscopy revealed significant improvement, with Mayo 1 disease in the distal 2 cm of rectum, and essentially normal histology (Figure 2). This, in conjunction with marked clinical improvement, allowed de-escalation of therapy to azathioprine and mesalazine.

Discussion

Common aetiologies of proctitis include inflammatory bowel disease, infections, and ischaemia; additionally, diversion colitis and radiation-associated proctitis have also been described [1]. Although clinicians often focus on enteric pathogens, infections can be sexually or non-sexually transmitted, and there has been a resurgence of the former in the setting of unprotected anorectal intercourse. This is especially prevalent in men who have sex with men, but is increasingly also seen

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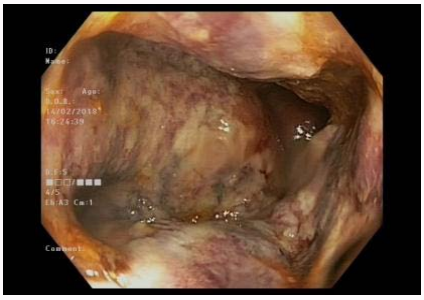

Figure 1: Flexible sigmoidoscopy. The mucosa in the rectum for 5 cm appeared severely erythematous, oedematous and ulcerated in a diffuse pattern.

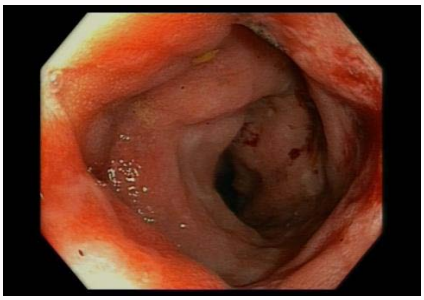

Figure 2: Follow-up colonoscopy at 6 weeks.

in the heterosexual population. The commonest cause of infectious proctitis is gonorrhoea, followed by chlamydia, herpes, and syphilis [2]. Enteric pathogens such as *Campylobacter*, *Shigella*, Salmonella, Giardia, and *Entamoeba histolytica* are also implicated in proctocolitis and can be transmitted by oro-anal sex [3].

HSV infection may be contracted through oro-anal or anoreceptiveintercourse [2]. Infections are predominantly due to HSV2 (13% of rectal HSV infections are caused by HSV1) [3]. It is characterized by small, typically multiple, vesicular lesions involving the perianal skin and anal canal, which may extend into the rectum. Lesions develop 1 to 3 weeks post exposure, ulcerate, crust and heal after two weeks [1]. Symptoms include anal pain, tenesmus and constipation, and systemic features such as fever and inguinal lymphadenopathy [2]. Other manifestations such as urinary symptoms, sacral paraesthesias and impotence may present acutely [2]. The virus becomes latent once antibody production occurs. Most episodes of HSV proctitis are primary infections although latent virus can be reactivated, resulting in recurrent, usually milder, episodes [1]. Diagnosis is most sensitive with PCR assays. Endoscopically HSV procitits is typified by the presence of ulcers, vesicles, and mucopurulent exudates [1]. Treatment entails 7 days to 10 days of antiviral therapy with aciclovir or valaciclovir, however longer courses of suppressive therapy may be necessary in immunocompromised patients.

Cytomegalovirus (CMV) and *Clostridium* difficile are well-recognized pathogens causing flares of colitis in inflammatory bowel disease. Immunomodulation is sometimes increased in addition to antiviral/antibiotic therapy to control inflammation, although this remains consensus-based due to a paucity of literature [4,5]. It has been suggested that anti-tumour necrosis (TNF) therapy such as infliximab may reduce replication/reactivation in CMV-associated flares. Although not described in other case reports of HSV proctitis, these principles were applied to our case where symptoms were refractory to antivirals alone, with excellent clinical response to infliximab.

The key lesson from this case is that even when encountering what appear to be straightforward diagnoses, clinicians must consider a broad range of differentials and take comprehensive histories to target appropriate investigations and ensure optimal patient care. Additionally, flares of inflammatory bowel disease due to viral infection present a unique challenge in terms of balancing immunosuppression and inflammation with immunomodulators.

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