

Colonic Mrsa Complicating Ulcerative Colitis

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Citation

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Abstract

Ulcerative colitis is well known to be exacerbated by infections most common being bacterial and viral especially CMV, clostridium difficile and fungal infections. First recognized almost 70 years ago,¹ enterocolitis due to Staphylococcus aureus has been described as both a complication of antibiotic therapy and as occurring in individuals with predisposing conditions but no previous antibiotic treatment. We describe here a unique case of flare up of ulcerative colitis in a young amphetamine user. This was subsequently found to be due to MRSA (methicillin resistant staphylococcus aureus). He was treated successfully.

CASE REPORT

A 24 year old man presented with 3 weeks history of profuse watery diarrhoea, fever and lower abdominal pain. One week before admission the stool frequency increased to 20 times/day accompanied by bleeding per rectum and vomiting. He abused amphetamine for the past 6 years. He had not travelled abroad recently and denied unprotected sexual contact. HIV and hepatitis C status was unknown and he was on no medications.

Physical exam elicited temperature of 39°C, tenderness in left iliac fossa and signs of dehydration. Haemoglobin was 10.1 g/dl (13.5-17.5 g/dl), white blood cells of $28 \times 10^9/L$ ($4-11 \times 10^9/L$) with neutrophilia $25.7 \times 10^9/L$ ($2.0-7.5 \times 10^9/L$), CRP of 289 mg/L (0-10 mg/L), albumin was 21 g/L (35 – 50 g/L). Stool cultures were negative (no clostridium difficile). Abdominal film showed 6 cm dilated transverse and descending colon.

Flexible sigmoidoscopy revealed gross mucosal edema, diffuse erythema and multiple punched out ulcers in rectum and sigmoid colon (Fig A).

Figure 1

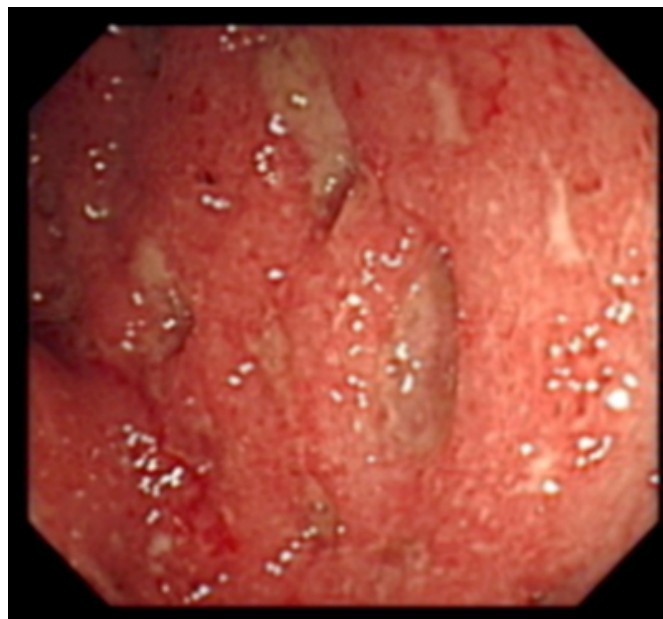


Fig A shows edematous sigmoid with multiple punched out ulcers.

Biopsy excluded ischemic, pseudo-membranous and CMV colitis. CT scan showed inflamed colonic wall (Fig B)

Figure 2

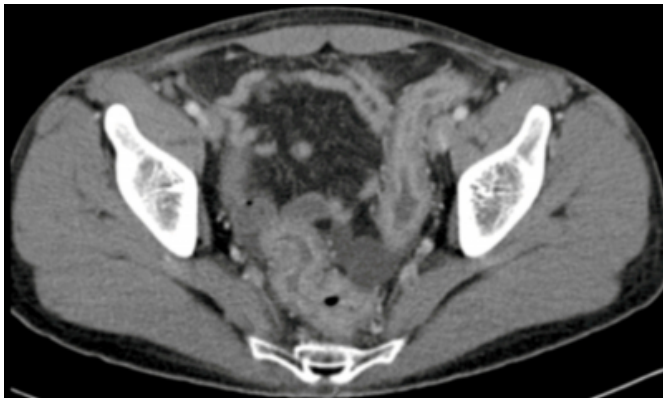


Fig B shows evidence of pancolitis.

He was treated with intravenous steroids, metronidazole and ciprofloxacin with a poor response. On 4th day his diarrhoea worsened (> 40 times/day) with a fall in albumin to 18 g/L and no improvement in CRP. He underwent a careful repeat sigmoidoscopy and biopsy for culture, and surgical consultation.

Figure 3

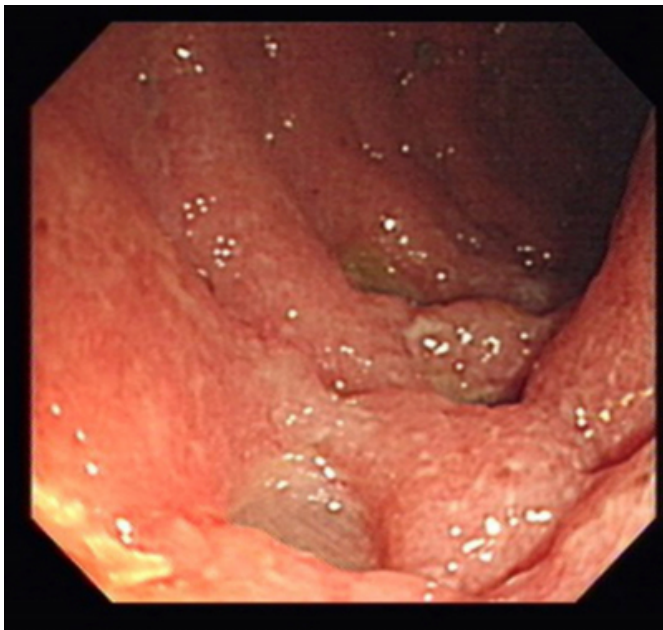
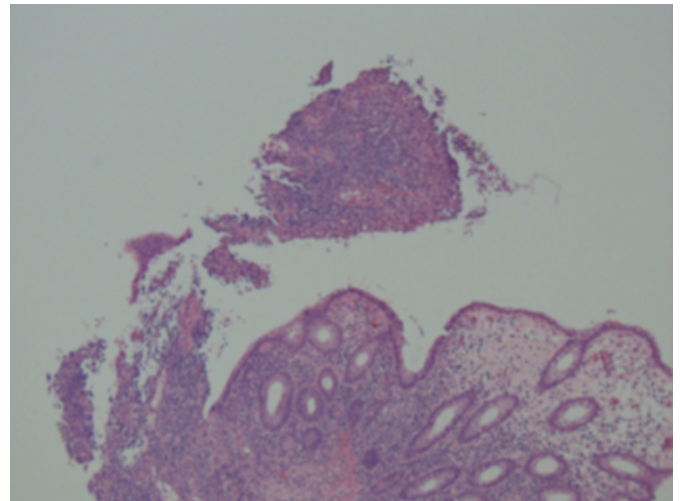


Fig C reveals worsening of ulceration and inflammation without any pseudomembranes.

Figure 4



The histology showed foci of acute cryptitis, crypt abscess formation and surface ulceration with granulation (Fig C). The lamina propria showed severe acute and chronic inflammatory cell infiltrate including neutrophils, lymphocytes, plasma cells and scattered eosinophils in keeping with infectious etiology. Due to the presence of plasma cells and mild crypt distortion ulcerative colitis was also considered as a differential diagnosis.

The tissue specimen from repeat sigmoidoscopy cultured methicillin resistant staphylococcus aureus (MRSA). MRSA was also isolated from nose and groin swabs. He was started on oral vancomycin and made a good recovery in few days. A diagnosis of acute ulcerative colitis with superadded MRSA infection was made. The patient initially defaulted from ambulatory care but at 6 months he was doing well.

UC exposes patients to the risk of opportunistic bacterial and viral infections. The most commonly seen are cytomegalovirus and clostridium difficile. There has been a case report of MRSA exacerbating ulcerative colitis². It can occur either at first or subsequent presentation. The unique point in this case was the growth of MRSA in sigmoid tissue culture in the presence of negative stool cultures.

How this patient contracted MRSA is unclear, although it is likely that he was infected in the community. There is some unproven link of increased risk of MRSA infection amongst amphetamine users³. An increased prevalence of MRSA infection (OR 1.39, 95% CI 1.21 to 1.60) has recently been seen amongst in-patients with IBD compared to patients with other GI conditions⁴. The risk of dying from MRSA is about twice as high in patients with inflammatory bowel disease as in other GI conditions (OR 2.22, 95% CI 1.83 to

2.69) ⁴. Possibility of superadded MRSA infection should be considered in unresponsive patients. Obtaining tissue culture is vital in this group of patients if stool cultures are unhelpful.

References

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