

Chronic intestinal pseudo-obstruction ที่เกิดจาก cytomegalovirus ในผู้ป่วยที่มีภูมิคุ้มกันปกติ

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บทคัดย่อ

Chronic intestinal pseudo-obstruction (CIPO) เป็นอาการแสดงที่พบไม่บ่อยในผู้ป่วยที่มีลำไส้อักเสบจาก Cytomegalovirus (CMV) เรานำเสนอกรณีศึกษาที่พบ CIPO ซึ่งเกิดจาก CMV ในผู้ป่วยที่มีภูมิคุ้มกันปกติ หญิงอายุ 45 ปีมาพบแพทย์ด้วยอาการท้องผูกเรื้อรังและท้องอืด ภาพถ่ายเอกซเรย์ช่องท้องพบการขยายตัวของลำไส้ใหญ่ ในขณะที่ภาพเอกซเรย์คอมพิวเตอร์พบผนังลำไส้ใหญ่ที่มีการหนาตัวแบบรอบด้านบริเวณ hepatic flexure และ transverse colon มีการขยายตัวอย่างมาก การส่องกล้องลำไส้ใหญ่พบแผลลึกที่บริเวณ hepatic flexure และ transverse colon ผลทางพยาธิวิทยายืนยันการย้อมสีภูมิคุ้มกันเชิงบวกสำหรับ CMV ผู้ป่วยมีอาการดีขึ้นหลังการรักษาด้วยยา Ganciclovir และ Prucalopride ควรตรวจสอบสาเหตุทุติยภูมิ เช่น การติดเชื้อ CMV ในกรณีพบ CIPO การเคลื่อนไหวผิดปกติของลำไส้จากการติดเชื้อ CMV อาจเป็นพยาธิสรีรวิทยาที่สำคัญของการเกิดภาวะนี้

คำสำคัญ: ไซโตเมกาโรไวรัส; แผลในลำไส้ใหญ่; ภาวะลำไส้อุดตันเทียมเรื้อรัง

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Cytomegalovirus-induced chronic intestinal pseudo-obstruction in an immunocompetent host

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Abstract

Chronic intestinal pseudo-obstruction (CIPO) is an uncommon presentation of cytomegalovirus (CMV) colitis. We present a case with CIPO caused by CMV in an immunocompetent host. A 45-year-old woman presented with chronic constipation and abdominal distension. A plain abdominal X-ray showed colonic dilatation, while a CT scan demonstrated circumferential wall thickening at the hepatic flexure of the colon and marked dilatation of the transverse colon. Colonoscopy revealed circumferential ulcers at the hepatic flexure and transverse colon, with pathology confirming positive immunostaining for CMV. The patient showed clinical improvement after treatment with ganciclovir and prucalopride. Secondary causes, including CMV infection, should be investigated in cases of CIPO. Dysmotility caused by CMV may represent the essential pathophysiology.

Keywords: Cytomegalovirus; Colonic ulcers; Chronic intestinal pseudo-obstruction

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Introduction

Cytomegalovirus (CMV), a member of the Herpesviridae family, is more commonly found in immunocompromised hosts than in immunocompetent hosts. In immunocompetent individuals, risk factors for CMV include advanced age, steroid usage, and severe concomitant diseases^{1,2}. CMV has been consistently reported as an etiologic agent of Chronic intestinal pseudo-obstruction (CIPO) in both pediatric and adult patients. Histological analysis in these cases frequently reveals abnormalities, damage, or ganglionitis within the myenteric plexus, establishing the neuropathic basis of the motility disorder^{3,4,8}. We present a case of CMV colitis in an immunocompetent host without typical risk factors, who presented with CIPO.

Case report

A 45-year-old woman presented with a two-month history of constipation and right upper quadrant pain. Written informed consent for publication was obtained from the patient on 8 October 2024, and all identifying information was anonymized to protect patient confidentiality. She had experienced abdominal distension for one month and had a weight loss of 6 kg over the past two months. Her medical history was

unremarkable, with no history of drug use, alcohol consumption, substance abuse, or smoking, and no family history of gastrointestinal disease. Physical examination revealed a mildly pale conjunctiva and a distended abdomen with normal bowel sounds. A plain abdominal X-ray demonstrated colonic dilatation and a large amount of fecal content. Computed tomography (CT) revealed circumferential wall thickening at the hepatic flexure of the colon, focal eccentric mucosal thickening, and marked dilatation of the transverse colon containing fecal impaction, associated with diffuse peritoneal fat stranding. Additionally, a large amount of feces was noted along the descending and sigmoid colon. Colonoscopy showed circumferential ulcers at the hepatic flexure and multiple deep ulcers surrounded by nodular lesions in the transverse colon. Pathology revealed chronic ulceration with moderate activity and positive immunostaining for cytomegalovirus (CMV).

The patient was treated with intravenous ganciclovir for 21 days and prucalopride. Repeat colonoscopies performed at 1, 2, and 6 months demonstrated complete healing of the ulcers at 6 months. A follow-up plain abdominal X-ray at 6 months showed no colonic dilatation.



Figure 1 Plain abdomen at presentation supine (A) and upright (B) colonic dilatation with large fecal content and 6 months after treatment (C) no colonic dilatation.



Figure 2 Computed tomography (CT) coronal view (A-C) circumferential wall thickening at the hepatic flexure of the colon and marked dilatation of the transverse colon distal to the lesion.

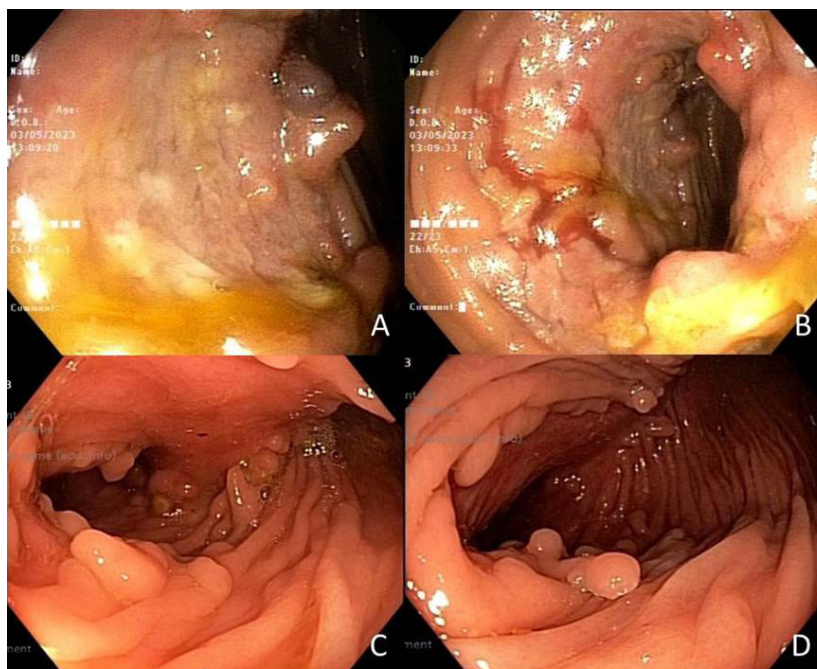


Figure 3 Endoscopic findings at presentation (A-B) circumferential ulcers at hepatic flexure and transverse colon and after treatment 6 months (C-D) healed ulcers with pseudopolyps.

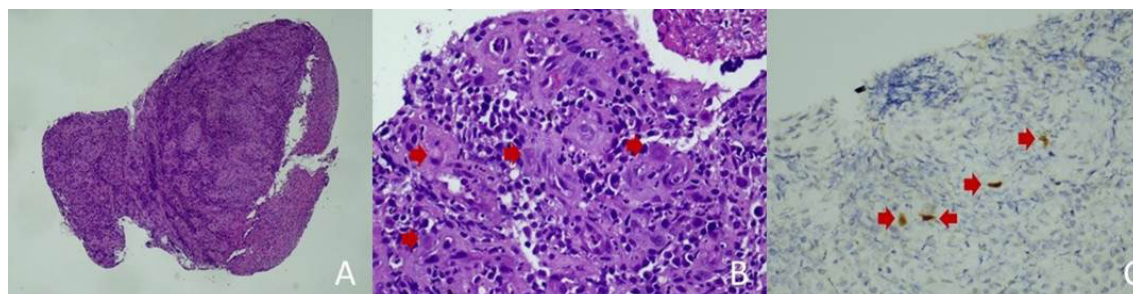


Figure 4 Pathology chronic ulceration with moderate activity (A-B) and positive immunostaining for cytomegalovirus (C).

Discussion

Chronic intestinal pseudo-obstruction (CIPO) is a rare disorder characterized by a dysfunction in the intestinal propulsion mechanism. This dysfunction leads to obstructive symptoms and radiologic evidence of intestinal dilation without any physical blockage. Various conditions, including viral infections, have been linked to CIPO. Patients with CIPO associated with CMV, Varicella-Zoster virus, Epstein-Barr virus, and JC virus have been reported^{3,4}.

CMV infection in immunocompetent individuals is rare, but more commonly seen in elderly patients with comorbidities¹. The colon is a frequent site of CMV involvement⁵. The endoscopic presentation of CMV colitis features a variety of ulcer types, predominantly well-defined and punched-out. Other ulcer types observed may include geographic and longitudinal ulcers. Additionally, findings may encompass a cobblestone appearance, inflamed mucosal surfaces, pseudotumor formations, mucopurulent exudate, spontaneous bleeding, or areas of normal mucosa^{1,6,7}.

CMV can affect various cell types within the gastrointestinal tract, including the vascular endothelium and stromal fibroblasts. In patients with CMV-associated CIPO, viral inclusions have been observed in the myenteric plexus and submucosal ganglia, potentially disrupting the colon's autonomic innervation⁸. CMV has been linked to other motility disorders. Segmental colonic hypoganglionosis associated with CMV colitis has been reported in immunocompetent

hosts⁹. CMV DNA has been detected in the small bowel of a patient with visceral neuropathy, and cases have been reported in patients with Ogilvie's syndrome^{10,11}. Additionally, CMV has been identified as the cause of strictures leading to acute colonic obstruction and toxic megacolon^{12,15}. CMV-induced CIPO can arise from both primary CMV infection and the reactivation of latent virus, particularly in immunocompromised individuals. The pathophysiology is driven by a two-pronged mechanism involving direct viral cytopathic effects and the host's immune response, leading to progressive damage to the enteric nervous system (ENS). Direct viral effect on glial cells and enteric neurons in the myenteric plexus, leading to cell lysis and destruction of ganglia. Immune response resulting in myenteric ganglionitis and vasculitis. In our patient we observed transverse colon dilatation distal to the colonic ulcer at the hepatic flexure, suggesting that CMV-induced bowel dysmotility may be a contributing factor. The standard, first-line antiviral treatment for CMV colonic ulcer involves either Ganciclovir or Valganciclovir. The usual duration of therapy is two to three weeks. Foscarnet is the primary alternative agent and is reserved for patients who experience intolerance to Ganciclovir/Valganciclovir or for cases of documented drug resistance. Approximately 50% of patients achieve healing six weeks after antiviral treatment, with about 90% showing complete healing by 12 weeks¹. The patient received a standard three-week course of Ganciclovir. Follow-up evaluation performed

one month post-treatment demonstrated histologic clearance of CMV, confirming virologic control. Although the infection was eradicated, the colonic ulcers and associated intestinal dilatation only fully resolved by eight weeks following the start of therapy. This delayed clinical recovery is significant. If the CIPO were solely due to direct viral cytopathic effect, prompt resolution would be expected immediately following the elimination of the lytic virus. The observed lag suggests the primary pathology was an immune-mediated enteric neuropathy. This supports the hypothesis that the CIPO was sustained by a robust, persistent host inflammatory response, which continued to cause dysfunction until the inflammation subsided, long after the CMV antigenic drive was eliminated by the antiviral treatment.

Prucalopride, a selective 5-HT₄ receptor agonist known to enhance colonic motility and typically indicated for chronic constipation, was initiated based on a previous randomized controlled trial supporting its use in CIPO¹⁶. However, the patient's symptoms did not improve with this treatment, and the medication was discontinued following the colonoscopy.

Conclusion

In patients with CIPO, secondary causes, including CMV infection, should be investigated, even in immunocompetent hosts. CMV-induced dysmotility may be the underlying pathophysiology.

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