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Paradoxical Reaction to Antitubercular Treatment Causing Colonic Obstruction

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A 41-year-old man presented with abdominal distention, right lower quadrant pain, and 10 kg of weight loss for 3 months. His medical history was unremarkable, and he was taking no medications. On examination, there was right lower quadrant tenderness without rebound. Laboratory examination showed white blood cells of $9.0\times10^3/\mu L$, hemoglobin of 12.3 g/dL, and C-reactive protein of 6.2 mg/dL (range, <0.14 mg/dL). An abdominal computed tomography (CT) scan disclosed circumferential mural thickening involving the ascending and transverse colon (Fig. 1A). The ascending colon was shortened with deformed ileocecal region. Chest CT scans were negative. Gastrographin enema radiograph disclosed shortening of the ascending colon and marked stricture at the hepatic flexure (Fig. 1B). Colonoscopy showed an ulceration surrounded by inflammatory polyps that presented stenotic behavior at the hepatic flexure (Fig. 2A). The colonoscope could not pass through the stricture. Although tuberculous culture and polymerase chain reaction examination for *Mycobacterium tuberculosis* of biopsies from the ulceration were negative, histopathological examination showed epithelioid cell granulomas with Langhans giant cells (Fig. 2B) and an acid-fast bacillus (Fig. 2C), making the diagnosis of colonic tuberculosis without concomitant immunocompromised condition. Antitubercular treatment with isoniazid, rifampicin, ethambutol, and pyrazinamide began. However, four weeks later, the patient had abdominal pain with distention and colonoscopy confirmed complete colonic obstruction despite the healing of the pre-existing tubercular ulcer (Fig. 2D). He then underwent right hemicolectomy with uneventful recovery. The histopathology of the resected specimen confirmed tuberculous obstructing lesions excluding neoplasms. Antitubercular treatment followed, and he remains well.

Complications associated with intestinal tuberculosis include obstruction, perforation, and bleeding. Paradoxical reaction was defined as clinical worsening of pre-existing tuberculous lesions after the initiation of appropriate antitubercular treatment.[1] This reaction affects the cutaneous, respiratory, and abdominal organs. Intestinal obstruction and perforation on this situation can be paradoxical reaction.[2,3] The pathological mechanism of this reaction remains unknown; however, it may include delayed hypersensitivity by increased exposure to killed mycobacterial antigens and fibrotic scar formation.[1,2] Progression of stenosis to obstruction has been regarded as clinical dilemma in the treatment of intestinal tuberculosis and several prophylactic agents have been applied.[4] In conclusion, al-

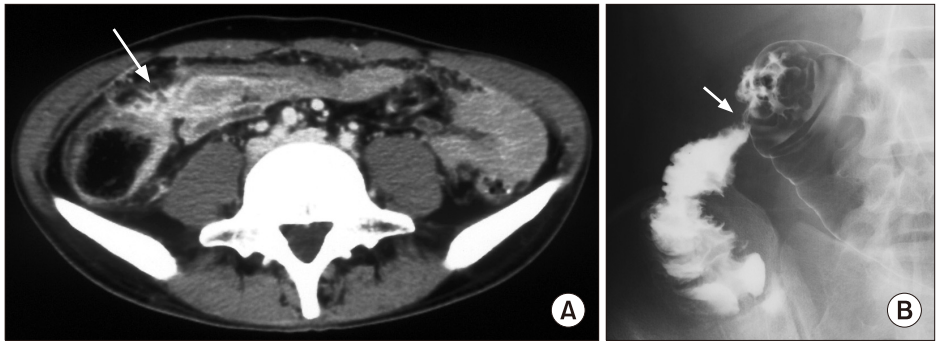

Fig. 1. (A) An abdominal computed tomography scan disclosed circumferential mural thickening involving the ascending and transverse colon. Note the stricture at the hepatic flexure (arrow) and mesenteric inflammation. (B) Gastrographin enema radiograph disclosed shortening of the ascending colon with surface irregularity and marked stricture at the hepatic flexure (arrow).

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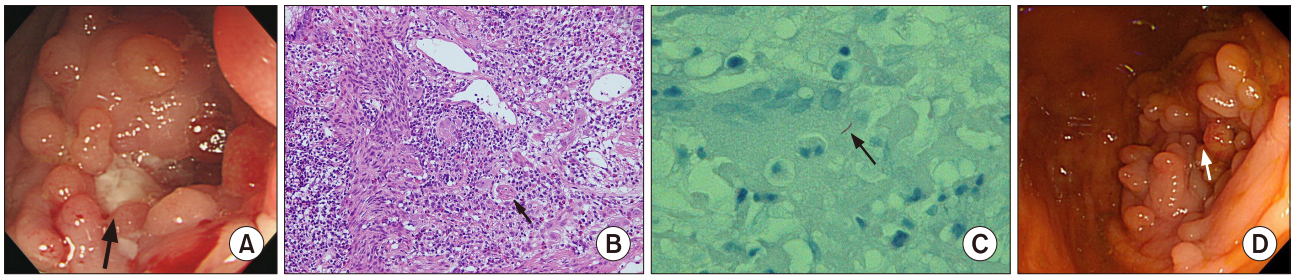

Fig. 2. (A) Colonoscopy showed an ulceration (arrow) surrounded by inflammatory polyps that presented stenotic behavior at the hepatic flexure. (B) Histopathological examination of the biopsy showed epithelioid cell granulomas with Langhans giant cells (arrow) (Hematoxylin and eosin, ×40). (C) An acid-fast bacillus was identified inside the granuloma (Ziehl-Neelsen, ×200). (D) Colonoscopy confirmed the complete colonic obstruction (arrow) despite the healing of the pre-existing tubercular ulcer at the hepatic flexure.

though rare, pre-existing intestinal strictures should be monitored carefully for paradoxical reaction to avoid obstruction.

CONFLICT OF INTEREST STATEMENT

None declared.

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