CASE REPORT

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Melanosis Ilei Associated with Chronic Ingestion of Oral Iron

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Melanosis can affect various parts of the gastrointestinal tract. Melanosis of the colon is not uncommon, while melanosis of the ileum is extremely rare. We report a case of melanosis ilei associated with chronic ingestion of oral iron (256 mg of ferrous sulfate once or twice daily for approximately 5 years) in a 32-year-old woman with end-stage renal disease. The findings of a colonoscopy, which was performed as a part of her medical checkup, were normal up to the cecum; however, numerous brownish-black punctuate pigmentations of the ileal mucosa were observed. Microscopic examination revealed hemosiderosis in the lamina propria of the ileal mucosa, particularly at the tips of villi. The diagnosis of melanosis (hemosiderosis) ilei was made based on the endoscopic and histological findings. **(Gut and Liver 2009;3:315-317)**

Key Words: Hemosiderin; Melanosis; Ileum

INTRODUCTION

Melanosis includes a variety of conditions associated with abnormal grayish-black or brownish-black pigmentation of an organ and can affect various parts of the gastrointestinal tract. Melanosis of the colon is not uncommon, while melanosis of the ileum is extremely rare.[1] Here, we report the endoscopic and histopathological findings of a melanosis ilei with a review of the literature.

CASE REPORT

A 32-year-old woman was transferred to our hospital for a renal transplantation workup and underwent endoscopy as a part of her medical checkup. She was diagnosed chronic renal failure due to glomerulonephritis 11 years ago, and had been on hemodialysis since 10 months. She did not complain of any other symptoms including constipation. She neither smoked nor used laxatives containing anthranoid or herbs. Her medications consisted of multivitamins, calcium carbonate, nifedipine, candesartan cilexetil, and ferrous sulfate. She had taken ferrous sulfate 256 mg twice daily (occasionally once daily) for approximately 5 years. Her white cell count was 4,000/mm^3, hemoglobin 10.9 g/dL, and platelets 142,000/mm^3. Laboratory investigations for anemia, which were performed while she was taking ferrous sulfate, showed a serum iron of 30 μL/dL (normal range, 65-157), unsaturated iron-binding capacity of 231 μL/dL (normal range, 191-269), and serum ferritin of 184.3 ng/mL (normal range, 20-300). The hyperkalemia and azotemia were attributed to her end stage renal disease. Esophagogastroduodenoscopy findings were unremarkable with normal duodenal mucosa. Colonoscopic examination was performed using a standard video colonoscope (EC-590ZW/L; Fujinon Inc., Saitama, Japan), which provides the computed virtual chromoendoscopy (CVC). At colonoscopy, the entire colon appeared normal up to the cecum, however, diffuse numerous brownish-black punctuate pigmentations were detected in the terminal ileal mucosa up to the about 15 cm proximal part from the ileocecal valve (Fig. 1A). With the observation of CVC mode, its characteristic brownish-black pigmentations in contrast to the yellowish background of the normal mucosa were more accentuated than those with the conventional white-light images (Fig. 1B). On microscopic examination, many pigment-containing macrophages were seen in the lamina propria of the

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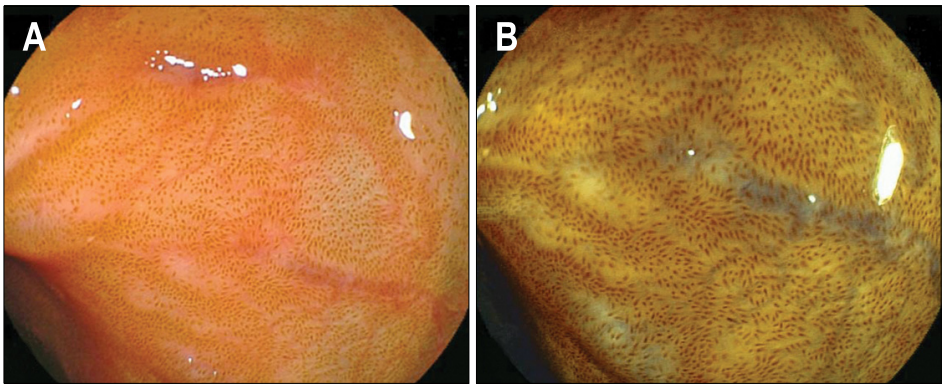

Fig. 1. Colonoscopic view of melanosis ilei. (A) Diffuse, dark-brown punctuate pigmentations are evident in the terminal ileal mucosa. (B) The characteristic endoscopic features are accentuated in a corresponding computed virtual chromoendoscopy image.

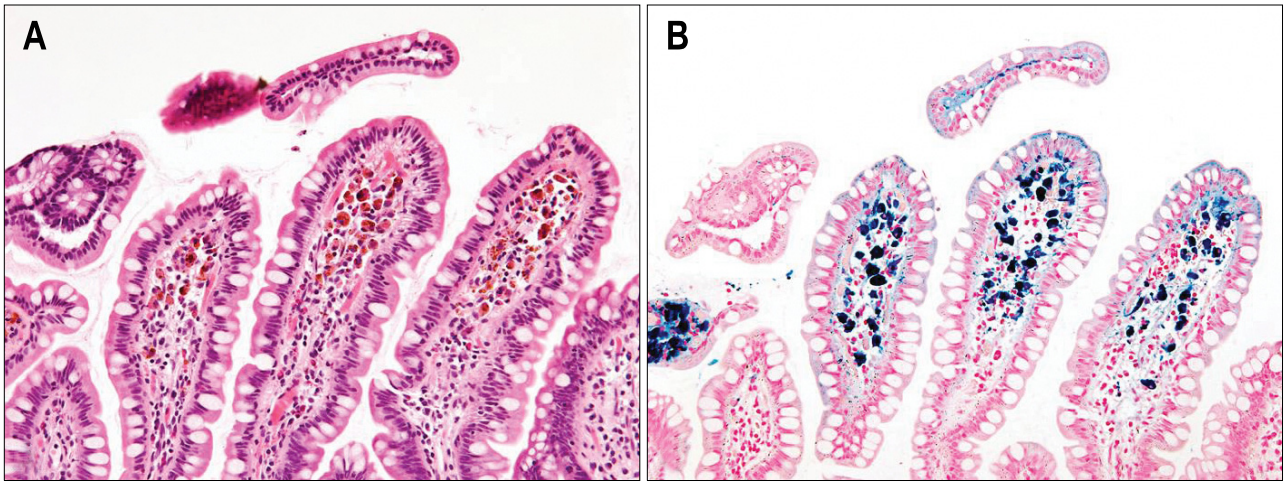

Fig. 2. Microscopic findings of melanosis ilei. (A) Conspicuous pigment-containing macrophages are evident in the lamina propria, particularly at the tips of the villi (H&E stain, ×400). (B) Prussian blue staining for iron was strongly positive (×400).

ileal biopsies, particularly at the tips of villi (Fig. 2A). Prussian blue stain was positive (Fig. 2B), indicating that the pigments were hemosiderin. Based on the endoscopic and histopathological findings, the diagnosis of melanosis ilei was made.

DISCUSSION

The term melanosis may be confused with melanin pigmentation. However, a variety of compounds may cause dark pigmentation of the intestine. Melanosis coli is not uncommon, however, very few cases of melanosis ilei are described in the literature.[2-5] Melanosis ilei may be recognized with or without melanosis coli.[2] Melanosis ilei combined with melanosis coli should be differentiated because it cannot be ascertained that the terminal ileum and other parts of the small intestine are never affected in melanosis coli.[1,6] Melanosis ilei combined with melanosis coli usually results from the chronic use of laxatives.[6] In contrast, melanosis ilei without melanosis coli is not associated with the use of laxatives, as in our case. Melanosis ilei without melanosis coli, endoscopically, shows grayish-black or brownish-black punctuate pigmentations in the ileal mucosa, which does not extend beyond the ileocecal valve like our case.[2-6]

The reported pigments of melanosis in the gastrointestinal tract are variable, and include lipofuscin in melanosis coli,[7] iron sulfide in melanosis duodeni,[8,9] and a combination of hemosiderin and lipofuscin in melanosis jejuni.[10] Although iron sulfide may be detected as melanosis duodeni, melanosis duodeni was not observed in our case. The reported pigments in melanosis ilei include silicates of aluminum and magnesium, hemosiderin, and charcoal.[3,11,12] Ghadially *et al.*[11] showed that pigment granules in melanosis ilei are heterolysosomes which are similar to those seen in pulmonary macrophages by electronmicroscopical studies. Hemosiderin may produce gross pigmentation of the ileal mucosa in patients associated with episodes of gastrointestinal bleeding due to the long-term use of salicylates or in patients with he-

reditary hemochromatosis.[3,12] Recently, Lee *et al.*[4] presented a case of melanosis ilei associated with the chronic ingestion of edible charcoal. In our case, conspicuous pigment-containing macrophages were seen in the lamina propria, and the strong positive result of Prussian blue staining for iron indicated that the pigment was hemosiderin. Since our patient had consumed oral iron supplementation for a long time, ingested irons may have attributed to the production of melanosis ilei. The possible mechanism of developing melanosis ilei in a patient with chronic ingestion of iron could be that following digestion, the released materials may be taken up by macrophages in the gut, however, why in only rare cases it presents as the gross pigmentation we call "melanosis ilei" is not known.

Observation with CVC mode, which is based on the selection of spectral transmittance with a dedicated wavelength, may accentuate the contrast of the mucosal surface and facilitate the assessment of the nature of gastrointestinal mucosal lesion.[13] Therefore, CVC image may accentuate endoscopic findings of melanosis ilei and may be helpful for the detection of subtle changes of melanosis ilei. However, the clinical feasibility of CVC image for the detection of melanosis ilei should be investigated in the future studies.

In conclusion, we report a rare case of melanosis ilei, which was hemosiderosis in the lamina propria of the ileal mucosa, associated with chronic ingestion of oral iron in a patient with end stage renal disease.

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