

An Endoscopic Finding of Unknown Etiopathogenesis: Case Report of Pseudomelanosis Duodeni “Black-Pepper-Speckled Duodenal Mucosa”

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ABSTRACT Although pseudomelanosis is usually observed in the duodenum, it is a rare endoscopic finding that can also be observed in the stomach and that is characterized by small, flat, brown-black speckles. Most of our information regarding this endoscopic finding, the etiopathogenesis of which could not be completely exhibited yet, is based on the case reports in literature. We also wanted to share the case of a patient whom we diagnosed with pseudomelanosis duodeni (PD), to contribute to the literature. Although PD is most commonly seen with conditions such as chronic renal failure, diabetes mellitus and iron replacement, sometimes its etiology may not be clearly demonstrated.

Keywords: Pseudomelanosis duodeni; endoscopy; etiology

Accumulation of black pigments in colonic mucosa was first specified in 1829, and its etiopathogenesis was clarified, but pigment accumulation in duodenal mucosa was first specified in 1976 by William M. Bisordi and Martin S. Kleinman, and its etiopathogenesis could not be clarified yet.^{1,2} It is considered that pseudomelanosis duodeni (PD) is associated with gastrointestinal bleeding, chronic renal failure (CRF), hypertension, diabetes mellitus (DM) and various medical treatments such as hydralazine, furosemide, and iron preparations.^{1,3} It is reported that anthraquinone laxatives in the aetiology of melanosis coli do not exist in the etiopathogenesis of PD.¹ Although PD is observed in every age group and both genders, it is predominantly observed more in the sixth-seventh decades and in females.^{1,4,5} In its pathological assessment, black pigments are located in mucosal macrophages in a sub-epithelial manner and

mostly on top of villi. In histochemical studies, it was demonstrated that the pigment consisted of a mixture of iron sulphide, hemosiderin, lipo melanin and ceroid.^{1,5}

CASE REPORT

A 76-year-old female patient was examined due to the complaints of heartburn and distension. In her medical history, the patient stated that she has had complaints of heartburn, gastric acidity and distension for approximately ten years and she needed to use a proton pump inhibitors and anti-acid medication for a long time. According to the history of the patient, she was diagnosed with breast cancer and had surgery sixteen years ago; she received chemotherapy and radiotherapy, and the final cycle of chemotherapy and radiotherapy was seven years ago. The patient had had hypothyroidism, hypertension and hyperlipidemia for fifteen

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years and osteoporosis for ten years, and she had been continuously using levothyroxine sodium, lansoprazole, ibandronic acid, atenolol, pravastatin, and lapatinib, which is a tyrosine kinase inhibitor, for the last year. The patient's body mass index was 28 kg/m² and no pathological findings were observed in her abdomen in the physical examination. No pathological findings were observed in her hemogram and biochemical evaluation as well and her analyzed iron parameters (iron, total iron binding capacity and ferritin), folate and vitamin B12 levels were normal, too.

In the esophagogastroduodenoscopy it was observed that her fundus mucosa was pale and it had lost its brightness, and an erythematous structure was observed in the antrum. In the mucosa of the second section of the duodenum, small, flat, brown-black point speckles that looked like sprinkled black pepper were observed (Figure A, B, C). Antrum and duodenum biopsies were performed. As the patient also had the complaint of distension and she was older than fifty years, a colonoscopy was also performed and it was observed that her colonoscopy findings were normal. In the histopathological evaluation of the patient's duodenal biopsies, it was observed that pigmented macrophages existed in the lamina propria and the results of staining associated with iron were negative. As a result of Giemsa staining of biopsy material collected from the antrum, it was observed that *Helicobacter pylori* was negative.

Informed consent was obtained from the patient.

DISCUSSION

Although PD is typically observed in the duodenal mucosa, it is defined as a benign finding that is observed as small, flat, brown-black point speckles also specified

in gastric mucosa.¹ In literature, PD was first defined in a 42-year-old patient with diabetes and CRF, as a black pepper sprinkled structure in the second section of the duodenum.² Despite the fact that many cases and series of cases were reported up to now, its aetiology is still unclear. There are a few asserted theories regarding its pathogenesis. One of these theories is the assertion stating that accumulation of iron associated with deficiency in the luminal iron transport after intramucosal haemorrhage or oral iron replacement in the duodenal mucosa can cause PD; the fact that most of the iron absorption occurs through the proximal duodenum supports this theory.^{4,6-8} Another theory asserts that the defect in macrophage metabolism can cause accumulation of iron sulphide and therefore, it is reported that medication containing cyclic compounds such as indole phenol and skatole can cause PD together with deteriorated macrophage metabolism.⁸ It was demonstrated that PD can be observed concomitantly with many drugs and diseases and the most commonly observed concomitant medications and conditions are use of ferrous sulphate, hydralazine, propranolol, hydrochlorothiazide, and furosemide, CRF, hypertension, DM and gastrointestinal bleeding.⁷ In many of the reported cases, multiple co-morbid conditions were observed, as in the case of our patient. Our patient also had hypertension and was using atenolol. While deteriorated renal functions and DM were commonly specified in the literature, these conditions were not concomitant in the case of our patient. In addition, different from the previously specified cases, a history of solid organ cancer existed in our case, although remission existed for a long time. Moreover, different from the drugs used by the other specified patients, our patient used a tyrosine kinase inhibitor, and it may be necessary to take this into consideration for the future assessments in terms of its role

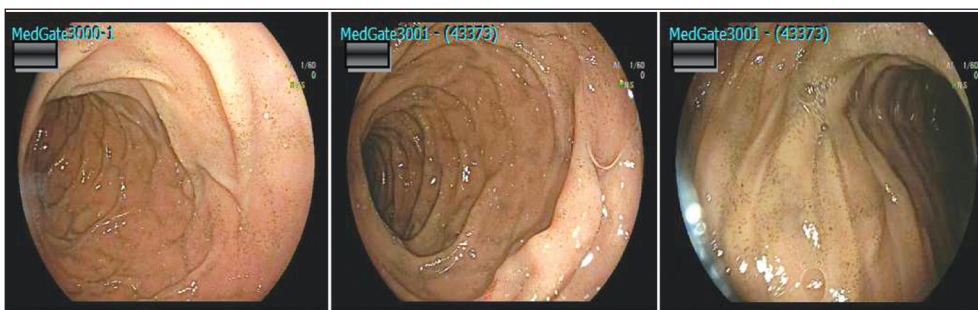


FIGURE 1: A, B, C) Endoscopic findings in duodenum mucosa.

in the pathogenesis. PD is usually observed as an incidental endoscopic finding, and no specific findings that can be considered as its clinical correspondence were specified. On the other hand, epigastric pain, heartburn, gastric acidity and nausea are the clinical complaints that require gastroscopic evaluation in the reported series of cases. Although endoscopic images are descriptive in the differential diagnosis of PD, necrosis, metastatic melanoma, consumption of charcoal, brown bowel syndrome, hemosiderosis and hemochromatosis are included.⁹

The etiopathogenesis of PD, which is significant in terms of endoscopic image, has not been clarified yet and this increases the interest in this endoscopic entity. A more comprehensive series of cases are needed for a better clarification of its etiopathogenesis.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: Bilger Çavuş, Filiz Akyüz; **Design:** Bilger Çavuş, Aslı Çiftçibaş Örmeci; **Control/Supervision:** Sabahattin Kaymakoğlu, Filiz Akyüz; **Data Collection and/or Processing:** Bilger Çavuş, Alp Atasoy; **Analysis and/or Interpretation:** Bilger Çavuş, Aslı Çiftçibaş Örmeci, Sabahattin Kaymakoğlu; **Literature Review:** Bilger Çavuş, Alp Atasoy; **Writing the Article:** Bilger Çavuş, Aslı Çiftçibaş Örmeci, Alp Atasoy; **Critical Review:** Filiz Akyüz, Sabahattin Kaymakoğlu; **Materials:** Filiz Akyüz.

REFERENCES

1. Odze Robert D, Goldblum John R. Surgical Pathology of the GI Tract, Liver, Biliary Tract and Pancreas. 3th ed. Philadelphia: Elsevier Saunders; 2015. [Link]
2. Bisordi WM, Kleinman MS. Melanosis duodeni. Gastrointest Endosc. 1976;23(1):37-8. [Crossref] [PubMed]
3. Thure Caire M, Kalan S, Brady P, Gill J. Pseudomelanosis of the stomach and duodenum: an uncommon endoscopic finding. Endosc Int Open. 2014;2(3):E191-2. Erratum in: Endosc Int Open. 2014;2(3):C1. [Crossref] [PubMed] [PMC]
4. de Magalhães Costa MH, Fernandes Pegado Mda G, Vargas C, Castro ME, Madi K, Nunes T, Zaltman C. Pseudomelanosis duodeni associated with chronic renal failure. World J Gastroenterol. 2012;18(12):1414-6. [Crossref] [PubMed] [PMC]
5. Sathyamurthy A, Chela H, Arif Z, Holly J, Arif M. Pseudomelanosis Duodeni. ACG Case Rep J. 2015;2(2):72-3. [Crossref] [PubMed] [PMC]
6. Schuerle T, Aoun E, Clarke K. Pseudomelanosis duodeni in a postrenal transplant patient. BMJ Case Rep. 2013;2013:bcr 2013 200466. [PubMed] [PMC]
7. Jain SS, Shah DK, Khot AA, T NR, Gharat AR, Rathi PM. Pseudomelanosis duodeni of undetermined etiology. Gastroenterology Res. 2012;5(4):171-3. [PubMed] [PMC]
8. Abumoawad A, Venu M, Huang L, Ding Xi-anzhong. Pseudomelanosis duodeni: a short review. Am J Digest Dis. 2015;2(1):41-5. [Link]
9. Qureshi NUA, Younus MF, Alavi K, Sheikh MY. Gastric and duodenal pseudomelanosis: An extended unusual finding in a patient with end stage kidney disease. Case Reports in Gastrointestinal Medicine. 2016. [Crossref] [PubMed] [PMC]