
Pseudomelanosis Duodeni : Association with Hypertension and Chronic Renal Failure : Case Report

**SOMBAT TREEPRASERTSUK, M.D.*,
PONGSEPEERA SUWANGOOL, M.D.***,**

DUANGPORN THONG-NGAM, M.D.,
PINIT KULLAVANIJAYA, M.D.***

Abstract

We present the first reported case with typical endoscopic and histological findings from Thailand. An 80-year-old man presented with chronic periumbilical abdominal pain for 3 months and melena for one week. He had had hypertension for 17 years, chronic renal failure for 4 years and gouty arthritis for 3 years. Panendoscopy was done and showed diffusely scattered small black and brown pigmentation over the stomach and duodenum. Tissue biopsies from the black pigmented lesions were taken for further microscopic and histochemical evaluation. Histological finding and special histochemical stains, Fontana stain, revealed mild chronic inflammation with accumulation of hemosiderin pigment in the lamina propria of the stomach and duodenal villi. This condition is called Pseudomelanosis duodeni. The literature of this condition was also reviewed.

Key word : Pseudomelanosis Duodeni, Iron, Hypertension, Chronic Renal Failure

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Pseudomelanosis duodeni is a scattered, speckled black pigmentation, usually smaller than 2 mm in diameter stained throughout the duodenal mucosa. This was first described in 1976 by Bisordi

and Kleinman⁽¹⁾. Only 31 cases of this condition have been reported to date⁽¹⁻¹³⁾. Most of the reported cases were the elderly with the history of chronic diseases such as hypertension, chronic renal

* Division of Gastroenterology, Department of Medicine,

** Department of Physiology,

*** Department of Pathology, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand.

failure, diabetes mellitus and some relevant drugs including hydralazine and propranolol. Histochemical studies are useful for determination of the nature of the pigments which are variable such as melanin, iron and lipofuscin. However, its etiology and clinical significance remains obscure. We reported the first case of Pseudomelanosis duodeni in a Thai patient with clinical, endoscopic, pathologic findings and reviewed the literature.

CASE REPORT

An 80-year-old man was referred from a general practitioner with chronic periumbilical abdominal pain for 3 months and melena for one week. He had had hypertension for 17 years, chronic renal failure for 4 years and gouty arthritis for 3 years. There was no history of alcohol intake or smoking during the past 7 years. He denied constipation and laxative abuse. He was taking hydralazine, metoprolol, aspirin, ferrous-sulfate, furosemide and calcium carbonate for underlying diseases. Physical examination revealed anemia, dry skin and pulsatile abdominal mass 3 cm in diameter with bruit. No cutaneous nor oral mucous membrane pigmentation was found. The rest was unremarkable. Laboratory test showed hemoglobin of 9.24 g/dl, hematocrit of 26.5 per cent with MCV of 87.9, blood urea nitrogen of 84 mg/dl and creatinine of 7.2 mg/dl. Serum lipase and amylase as well as liver function test were normal. He was admitted for evaluation of the cause of chronic abdominal pain and melena. Abdominal ultrasonogram with doppler showed focal dilation of superior mesenteric artery, 3.1 x 2.7 cm in diameter. Panendoscopy showed normal esophagus but diffusely scattered small black and brown pigmentation over the stomach and duodenum (Fig. 1). Tissue biopsies from the black pigmented lesions were taken for further microscopic and histochemical evaluation. Histological finding and special histochemical stains, Fontana stain, revealed mild chronic inflammation with accumulation of hemosiderin pigment in the lamina propria of the stomach and duodenal villi (Fig. 2-3).

The laboratory test for serum iron and ferritin were performed later and were showed two times higher than the upper limit of normal: serum iron = 55 microgram/dl (normal 100-170), serum ferritin = 601 ng/ml (normal 15-400). So this is the first case report of Pseudomelanosis duodeni of Thailand.



Fig. 1. Duodenal bulb shows linear-shaped black pigmentation, scattered and discrete.



Fig. 2. Duodenal biopsy showing brown-black amorphous pigment in the lamina propria. H & E stain x 400.

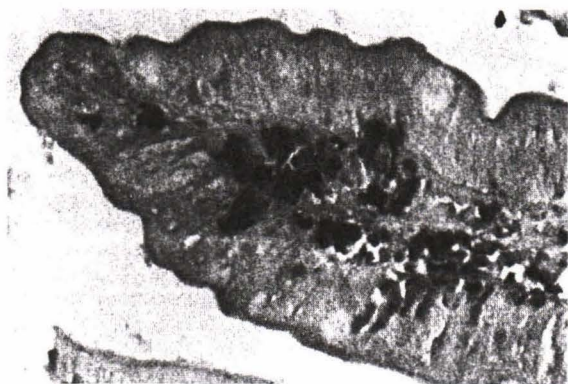


Fig. 3. Duodenal villus showing dark brown amorphous pigment in macrophages in the lamina propria. Fontana stain x 400.

DISCUSSION

Melanosis duodeni was named in 1976 by Bisordi and Kleinman⁽²⁾. Later Cowen, et al used the term, Pseudomelanosis duodeni which is more appropriate than melanosis duodeni in 1980⁽³⁾. The clinical significance of Pseudomelanosis duodeni was not known, but Sharp and colleagues found that the pigment disappeared with correction of the folate deficiency and healing of the gastric ulcer⁽³⁾. There have been only 31 cases of this condition reported to date in Medline⁽¹⁻¹³⁾. In early case reports, there was some confusion with its definition but later it was defined by means of the gastroendoscopic finding, histochemical stain or electron microscopy⁽⁴⁻¹³⁾.

The granules in Pseudomelanosis duodeni were more variable in size and shape with a distinctive crystalline appearance⁽⁶⁾. The melanin granules were elongated and ovoid shape within the lamina propria. In some reports, the negative reaction from the histochemical stain for iron might be false negative because the histochemical stain for iron is known to give a positive reaction with iron oxide but not with iron sulfide^(4,6).

Clinical correlation with this finding is unclear but this condition is more common in the sixth and seventh decade of life and associated with the medical illnesses especially chronic renal fail-

ure, hypertension or some medications listed in Table 1 such as hydralazine, propranolol, thiazide, ferrous sulfate and methyldopa⁽⁴⁻¹⁴⁾.

Kang and colleagues postulated that iron staining in the duodenal mucosa may be due to intramucosal hemorrhage or increased luminal absorption of iron⁽⁸⁾. The latter hypothesis was more likely if the impairment of subsequent transport of iron occurred and resulted in the iron-deposition. Kang also found that iron was shown in the duodenal biopsy of 71 per cent of 48 chronic renal failure patients on maintenance hemodialysis with macroscopically normal gastroduodenoscopic finding. This percentage was statistically significant when compared to those found in the other patients undergoing gastroduodenoscopy. In contrast to the melanosis coli, the Pseudomelanosis duodeni has not been found in association with anthracine laxative abuse⁽⁴⁻¹³⁾.

However, this condition appeared to be acquired rather than congenital because no pigment was seen upon previous gastroduodenoscopy in some reported cases^(7,11). The clinical significance of Pseudomelanosis duodeni awaits further studies. The spectrum of pigment compounds and their relation to iron metabolism as well as circumstances in which it occurs needs to be defined.

Table 1. Clinical data of 32 patients with pseudomelanosis duodeni (histologically confirmed) during 1980-1999.

Reported by/Year	Age	Sex (Number of cases)	Symptoms	Concomitant medical Problems	Medications	Stain with
Cown ML, et al (1) 1980	62	F(1)	No data	HT, Erythema multiforme, Nontoxic goiter, Diverticulosis coli	Propranolol, Thiazide Hydralazine, Methyldopa Prednisolone	Not melanin
Pounder DJ (4) 1983	60	F(1)	Chronic gastric ulcer	No data	No data	Ferrous sulfide
Steckman M, et al (5) 1983 North Carolina	65	M(1)	Dysphagia	CHF, HT, Anemia	Ferrous sulfate, Bisacodyl, Amitriptyline	Iron (Hemosiderosis)
Yamase H, et al (6) 1985 Connecticut	66	F(1)	UGI. Bleeding	CRF, HT, Anemia Arthritis	Hydralazine, Propranolol Sulindac, Acetaminophen	Iron
Gupta TP, et al (7) 1985 Michigan	66, 68, 69	F(3)	Hematemesis (2) Anemia (1)	HT, DM CRF, CHF, Stroke	Hydralazine, Colchicine Furosemide, Methyldopa Folic acid, Al(OH ₃) Prednisolone, Ferrous sulfate	Iron
Kang JY, et al (8) 1987 Singapore	14-79	M(4) F(5)	Dyspepsia (2) Pretransplanted evaluation (7)	CRF, Anemia	Ferrous sulfate Propranolol, Thiazide Methyldopa, Hydralazine	Iron
Castellano G, et al (9) 1988 Spain	65	F(1)	UGI. Bleeding	HT	Propranolol Hydralazine Thiazide, Amiloride	Ferrous sulfide
Lin HJ, et al (10) 1988 China	64	M(1)	Periumbilical pain	HT, DM, Chronic liver disease (Hepatitis B)	No data	Iron and Lipofuscin
Rex DK, et al (11) 1988 Indianapolis	58, 69, 72	F(3)	Epigastric pain (3)	HT, Diverticulosis Degenerative joint disease Angina	Prazosin, Aspirin, Indomethacin Thiazide, Digoxin Hydralazine, Metoprolol	Iron sulfide
El-Newihi HM, et al (12) 1995 Mississippi	58, 71	M (2)	UGI. Bleeding	Alcohol abuse, HT CRF, Gout	Hydralazine, Furosemide Propranolol, Thiazide	Iron, sulfur and Calcium
Wang K, et al (13) 1995 Taipei	means age 64.6 ± 9.1	M(6) F(2)	Abdominal pain (4) Check up (3) Hiccup (1)	HT, DM Common bile duct stone, Renal stone	Hydralazine, Propranolol Thiazide, Methyldopa, Glibenclamide	Iron ; strongly positive 3 from 8 cases
Present case 1999 Thailand	80	M(1)	Periumbilical pain and melena	HT, CRF, Gout	Hydralazine, Metoprolol Aspirin, Ferrous sulfate, Furosemide	Iron

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รายงานผู้ป่วยที่มีภาวะเยื่อลำไส้เล็กมีสีดำ ซึ่งเกี่ยวข้องกับโรคความดันโลหิตสูง และภาวะไตวายเรื้อรัง

สมบัติ ตรีประเสริฐสุข, พ.บ.*, ดวงพร ทองงาม, พ.บ.**,
พงษ์พีระ สุวรรณกุล, พ.บ.***, พินิจ กุลละวณิชย์, พ.บ.*

รายงานผู้ป่วยชาย อายุ 80 ปี ที่มีอาการปวดท้องรอบ ๆ สะดือ เรื้อรังมานาน 3 เดือน และมีถ่ายดำ 1 สัปดาห์ โดยที่ผู้ป่วยมีโรคประจำตัว คือความดันโลหิตสูงและภาวะไตวาย ผู้ป่วยได้รับการตรวจโดยการส่องกล้องทางเดินอาหารส่วนต้น พบว่ามีลักษณะผิดปกติ คือเยื่อลำไส้เล็กส่วนต้น มีสีดำ น้ำตาล ลักษณะเป็นจุดเล็ก ๆ กระจายทั่วกระเพาะอาหารส่วนปลาย และลำไส้เล็กส่วนต้น ได้ทำการตัดชิ้นเนื้อบริเวณดังกล่าวไปตรวจทางพยาธิวิทยา พบว่ามีลักษณะเยื่อลำไส้เล็กเสปร่วมกับมีเหล็กสะสมอยู่เป็นจำนวนมาก ซึ่งเรียกภาวะนี้ว่า ซูโด เมลานโนซิส ดูโอเดโน และได้พบทวนวรรณกรรมของภาวะนี้ไว้ด้วย

คำสำคัญ : ซูโดเมลานโนซิส ดูโอเดโน, ธาตุเหล็ก, ความดันโลหิตสูง, ภาวะไตวายเรื้อรัง

สมบัติ ตรีประเสริฐสุข และคณะ

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* สาขาวิชาโรคทางเดินอาหาร, ภาควิชาอายุรศาสตร์,

** ภาควิชาสรีรวิทยา,

*** ภาควิชาพยาธิวิทยา, คณะแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย, กรุงเทพฯ ๙ 10330