An Incidental Finding of Gastric and Duodenal Pseudomelanosis: A Case Report

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Abstract
Pseudomelanosis of the upper gastrointestinal tract is a rare diagnosis of undetermined significance, most commonly affecting the duodenum. Endoscopically, it is characterized by dark spickled pigmentation. Its development has been linked to certain conditions and medications. Involvement of the stomach is extremely rare with very few cases reported in the literature to date. We report an 85-year-old male who is known to have type 2 diabetes mellitus, dyslipidemia, iron deficiency anemia, and chronic kidney disease who underwent an esophagogastroduodenoscopy for evaluation of upper gastrointestinal bleeding and was found to have gastric and duodenal pseudomelanosis confirmed by biopsy. It is an extremely rare benign condition, but metastatic melanoma has to be ruled out, as was done in this case.

Introduction
Pseudomelanosis of the upper gastrointestinal tract is a rare endoscopic finding, most commonly found in the duodenum, and is known as pseudomelanosis duodeni [1]. Pseudomelanosis duodeni has been reported on multiple occasions, but to the best of our knowledge,
only 7 cases of gastric pseudomelanosis have been previously reported in the literature [2]. Pseudomelanosis is characterized by the detection of speckled dark pigmentations on endoscopy [3]. There is no specific etiology, but a number of factors has been linked to pseudomelanosis including diabetes mellitus, hypertension, chronic kidney disease, hemochromatosis, gastrointestinal bleeding, and the use of certain medications [2].

**Case Presentation**

An 85-year-old male with type 2 diabetes mellitus, hypertension (on furosemide, hydralazine, and β-blocker), dyslipidemia, iron deficiency anemia (on ferrous sulphate), chronic kidney disease, and under follow-up for dementia, presented with a 1-week history of reduced oral intake, recurrent vomiting, and decreased level of consciousness. On examination, the patient looked severely dehydrated, was hypotensive (92/58), mildly tachypneic (20) with a heart rate of 87 and Glasgow coma scale of 11, and was found to have melena on examination. Laboratory results demonstrated leukocytosis (12 × 10^9), low hemoglobin (12.3 g/dL; baseline 11.2 g/dL), high C-reactive protein (106 mg/L), high urea (53 mmol/L; baseline 27 mmol/L), acute kidney injury on top of his chronic kidney disease (500 µmol/L; baseline 269 µmol/L), elevated lactate (4.6 mmol/L), elevated procalcitonin (4.88 ng/mL), and evidence of urinary tract infection on urine analysis. The patient was admitted to the intensive care unit with a diagnosis of severe sepsis secondary to urinary tract infection, acute on top of chronic kidney injury and upper gastrointestinal bleeding.

The hemoglobin dropped on day two of admission to 9.7 g/dL. An esophagogastroduodenoscopy showed a hiatus hernia with Hill’s classification grade 4, a 7-mm sessile polyp with a Paris classification of 0-Is which was removed by snaring, extensive gray to black speckles on the posterior wall of the gastric corpus, as well as the duodenum indicating possible gastric and duodenal pseudomelanosis, respectively (Fig. 1).

Histological examination of the gastric mucosa revealed intact lining epithelium with overall preserved architecture. The lamina propria had brownish-black pigment-laden macrophages (Fig. 2) and Perl’s Prussian blue stain tested positive, confirming iron deposition (Fig. 3). This is consistent with the diagnosis of pseudomelanosis. The duodenal polyp was found to be hyperplastic.

The patient’s urosepsis was managed with antibiotics guided by the culture results while the kidney injury was managed conservatively with intravenous fluids and supportive measures. He improved clinically and was transferred to the wards on day 5 of admission. The renal function was back to baseline, septic markers improved, and the patient was finally discharged home after 12 days of hospitalization with full recovery.
Discussion

Pseudomelanosis of the upper gastrointestinal tract, specifically the stomach, is an exceptionally rare endoscopic finding. The first case ever reported traces back to 1383 [4]. It is a benign condition that is typically linked to diseases such as diabetes mellitus, hypertension, chronic kidney disease, hemochromatosis, gastrointestinal bleeding, and medications such as iron sulfate, propranolol, hydralazine, furosemide, and hydrochlorothiazide [2]. On the contrary, pseudomelanosis of the lower gastrointestinal tract, also known as melanosis coli, is a common finding on colonoscopy [5]. Patients with a history of chronic constipation and long-term use of laxatives have been linked to melanosis coli [6]. The possible contributing factors to pseudomelanosis in our case include chronic kidney disease, diabetes mellitus, hypertension, and his medications which included ferrous sulphate, hydralazine, furosemide, and β-blockers.

Gastric and duodenal pseudomelanosis are often unforeseen on endoscopic or pathologic assessment. Nevertheless, due to the rarity of this condition, there are no long-term sequelae to gastric and duodenal pseudomelanosis, as they are clinically insignificant. With regards to our patient, he was critically ill and with the findings suggestive of upper gastrointestinal bleeding including melena and the drop in hemoglobin, esophagogastroduodenoscopy was indicated. We believe that pseudomelanosis was an incidental finding and is not related to his presentation of upper gastrointestinal bleeding.

Of the 7 cases reported in literature of gastric pseudomelanosis, our patient demonstrated similar characteristics [2]. The classical endoscopic features of pseudomelanosis were present in our patient, typically showing as speckled dark pigmentation in the stomach as a result of deposition of iron in the lamina propria macrophages, which can be visualized microscopically with Perl’s stain [3]. In fact, charcoal ingestion, ferrous sulfide, hemosiderin, lipofuscin, and pseudomelanin deposition can also result in pseudomelanosis. Moreover, parts of the deposited pigment can contain calcium, magnesium, and other minerals [1]. The differential diagnosis of pseudomelanosis duodeni include metastatic malignant melanoma, brown bowel syndrome, hemosiderosis, and hemochromatosis [7]. The melanosis associated with malignant melanoma does not stain with Perl’s stain but rather would be stained with the Masson-Fontana stain [8]. Table 1 illustrates the differential diagnosis of dark mucosal staining including their
Table 1. Differential diagnosis of dark mucosal staining including their characteristics, organs involved, and the histopathological findings.

| Differential diagnosis | Associated conditions | Organs involved | Histopathology findings |
|------------------------|-----------------------|-----------------|-------------------------|
| Metastatic melanoma    | Primary skin melanoma | Can affect any organ. When it affects GI tract, it might present with dark-blackish discoloration of the mucosa | Usually seen as multiple flat or polypoid lesions. Cytological atypia with increased nuclear cytoplasmic ratio, enlarged hyperchromic nuclei, prominent nucleoli, and frequent mitoses. Melan-A, S100, HMB45, and SOX-10 immunohistochemical stains |
| Gastric siderosis       | Repeated blood transfusion Hemochromatosis Alcohol related liver disease Iron supplementation | Mainly affect the stomach. Detailed history is important to identify the primary condition | Yellow-brown hemosiderin pigment which stains positive for iron |
| Brown bowel syndrome    | Associated with malabsorption syndromes including celiac disease, Crohn’s disease, post-gastric bypass, and alcohol abuse as a result of vitamin E deficiency | GI tract | Lipofuscin-like pigment deposits in smooth muscle cells in the muscularis mucosa and muscularis propria |
characteristics, organs involved and the histopathological findings [9–11]. In conclusion, gastric pseudomelanosis is an extremely rare and benign condition, but histopathological examination is necessary for confirmation of the diagnosis and to exclude metastatic melanoma.

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**Statement of Ethics**

Written informed consent from the next of kin was obtained to publish this case and accompanying images. Ethical approval as obtained by the IRB Research Committee at King Hamad University Hospital (reference No. 19-294) to publish this case.

**Conflict of Interest Statement**

The authors declare that there is no conflict of interest.

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**Author Contribution**

Mohamed Wael Mohamed and Rawan Althahabi: drafting the case report and literature review. Faisal Abubaker: critical revision and editing. Omar Sharif: critical revision and final approval.

**Data Availability Statement**

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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