

An unusual cause of dysphagia

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A 42-year-old male presented with 2 months history of progressive dysphagia predominantly to solids. Computed tomography of the chest showed a large subcarinal lymph node (Fig. 1A). His PPD skin test was positive (32 mm x 30 mm). He underwent gastroduodenoscopy where a submucosal bulge with normal overlying mucosa causing luminal narrowing was noted in mid-esophagus. Endoscopic ultrasound (EUS) revealed a large (6 cm x 4.5 cm) subcarinal lymph node with few hyperechoic areas (Fig. 1B). Multiple lymph nodes were also seen in the upper mediastinum. EUS-guided fine-needle aspiration was done from the subcarinal lymph node that yielded caseous material. The cytological examination of the aspirate showed granulomatous inflammation and the stain for acid fast bacilli was negative. He was started on four drug antitubercular therapy and responded with resolution of dysphagia and weight gain.

Dysphagia is an uncommon presentation of tuberculosis [1]. Esophageal tuberculosis may mimic esophageal malignancy. It is most commonly recognized as an extrinsic bulge on endos-

copy. Other endoscopic features include ulcers and polypoidal lesions. The most common site of esophageal tuberculosis is mid-esophagus [2]. EUS provides the opportunity to detect and sample these lesions and has emerged as tool of choice to diagnose esophageal tuberculosis. The presence of hyperechoic foci, patchy anechoic or hypoechoic areas in the lymph nodes on EUS suggest tubercular etiology [3]. The response to four-drug combination therapy is rapid and the resolution of dysphagia is usually noted within 3 weeks of initiation of therapy [2].

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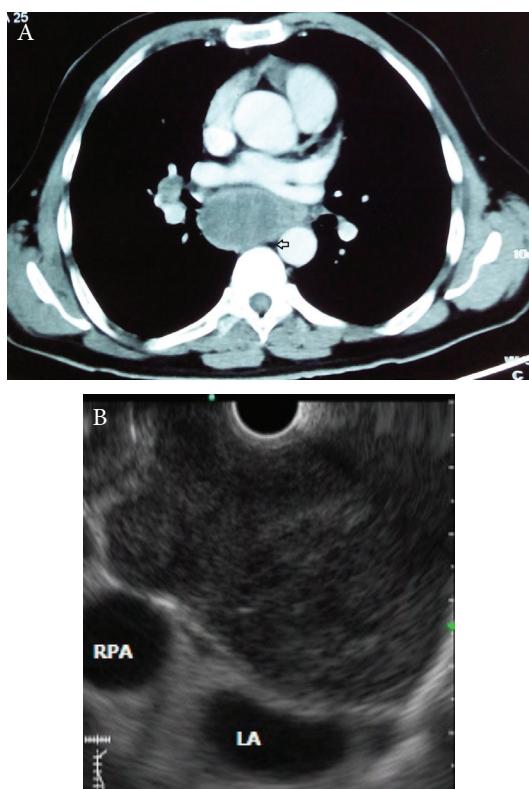


Figure 1 (A) Computed tomography showing enlarged subcarinal lymph node compressing the esophagus (arrow) (B) Endoscopic ultrasound: Large subcarinal lymph node compressing the left atrium LA, left atrium; RPA, right pulmonary artery

Successful en bloc endoscopic submucosal dissection of early gastric cancer and rectal lateral spreading tumor in a Greek hospital

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Basioukas S. and Xinopoulos D. [1] previously reported successful endoscopic submucosal dissection (ESD) of early gastric cancer, type '0-IIa', in a Greek hospital. We would also like to add our experience from gastric and rectal ESD and one-year follow up. Although, narrow band imaging (NBI) magnification endoscopy is a prerequisite, for detailed evaluation of NBI magnifying patterns of mucosal cancer, according to Japanese experts [2], we managed to successfully complete two ESD cases, despite the absence of NBI magnification endoscopy, as was also the case in Basioukas' and Xinopoulos' [1] report. Furthermore, we report sufficient follow up in our cases (no local recurrence at one year), necessary for curable ESD.

We herein report a successful, curable, *en bloc* ESD resection of early gastric cancer, type '0-IIa', 2 cm in diameter, at the middle posterior gastric body along the lesser curvature, in a 77-year-old male (Fig. 1 A, B) and mucosal lateral spreading rectal tumor (LST), >4 cm in diameter, type '0-IIa+IIc' tumor, at <1 cm distance from the dentate line, in a 65-year-old male, (Fig. 2 A, B), performed at our endoscopy department, in September 2012.

In view of severe co-morbidities the first patient was a poor candidate for surgery, and, after detailed explanation, he agreed and signed to undergo ESD. In the second patient, colonoscopy due to hematochezia, revealed an LST, granular non-homogenous rectal tumor, type '0-IIa+IIc', according to [3] the Paris classification, 1 cm from the anal merge, on posterior wall, and more than 4 cm in diameter (Fig. 2A). Due to size, location and morphology, it was decided to resect the tumor by ESD, with the patient's agreement. Both gastric and rectum ESDs were performed at our endoscopy department under conscious sedation. Cup-technique and dual knife was used for marking of the tumor border, circumferential cutting and ESD in both patients (Fig. 1B, 2B). Coagulation spray was used for hemostasis. No short- or long-term complications were reported and the patient was discharged after a three-day in-hospital stay. Procedure time was more than 6 h.

Control endoscopy on the day of discharge revealed a normal ESD ulcer without signs of bleeding or perforation (Fig. 1C, 2C). No blood transfusions were necessary. Histological examination of gastric ESD specimen (46x25x3 mm in dimensions) (Fig. 1D) showed low- and locally high-grade dysplasia (20x20 mm), corresponding to mucosal cancer, type IV according to the revised [4] Vienna classification of gastrointestinal epithelial neoplasia, resected *en bloc* within normal margins (horizontal and vertical margins normal). Histology of the rectal ESD specimen (Fig. 2D) showed vilous rectal adenoma with low-grade dysplasia (46x37x14 mm)

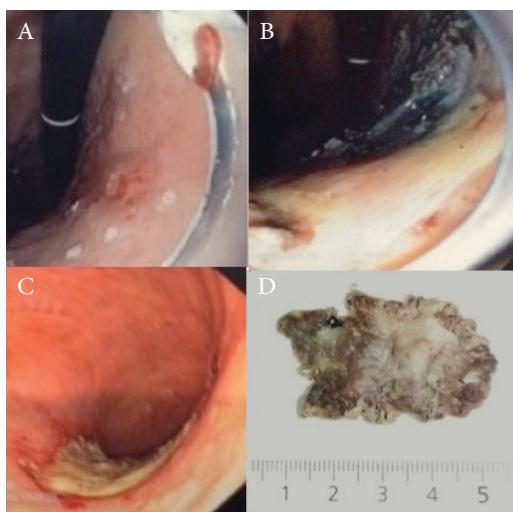


Figure 1 (A) Marking of early gastric cancer at the middle body along lesser curvature type '0-IIa' before endoscopic submucosal dissection (ESD) (B) ESD bedem (C) ESD ulcer (D) Gastric ESD specimen



Figure 2 (A) Rectal lateral spreading rectal tumor granular, non-homogeneous, type '0-IIa+IIc', 1cm from anal merge. (B) Submucosal space during endoscopic submucosal dissection (ESD) (C) ESD ulcer after completion of ESD (D) Rectal ESD specimen

[14] (Vienna classification type III) resected within normal margins. Control endoscopy one year later showed normal scar at the site of ESDs, no residual tumor, while random biopsies from the ESDs scar were normal.

According to our cases, although only two, and in absence of NBI magnification endoscopy, we consider ESD feasible for curable *en bloc* resection of early gastric cancer and rectal LST in a Greek hospital. However, further expertise is necessary.

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Jejunal polyp: a rare cause of intussusception

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We present the case of a 36-year-old male complaining of non-radiating abdominal pain for 15 days, with multiple episodes of green-colored vomitus. Computed tomography (CT) scan revealed jejunoo-jejunal intussusception with a 6×3×3cm homogenously enhanced smoothly marginated polypoidal soft tissue neoplastic lesion at the lead point (Fig. 1). An intestinal segment with intussusception was sent for histopathology. On gross examination, a single sessile polypoidal mass was identified measuring 6×3×3cm. The cut surface was white, firm with focal myxoid appearance. Microscopic examination (Fig. 2) showed submucosal tumor with overlying ulcerated jejunal mucosa. The tumor was moderately cellular containing interlacing fascicles of bland spindle-shaped, smooth muscle cells. The nuclei appeared elongated and cigar-shaped, without any pleomorphism. There was no evidence of necrosis or abnormal mitosis. Immunohistochemistry was positive for desmin and smooth muscle actin but negative for c-KIT (CD117) excluding the possibility of gastrointestinal stromal tumor (GIST) and establishing the diagnosis of jejunal leiomyomatous polyp.

Tumors of the small intestine, benign or malignant, are

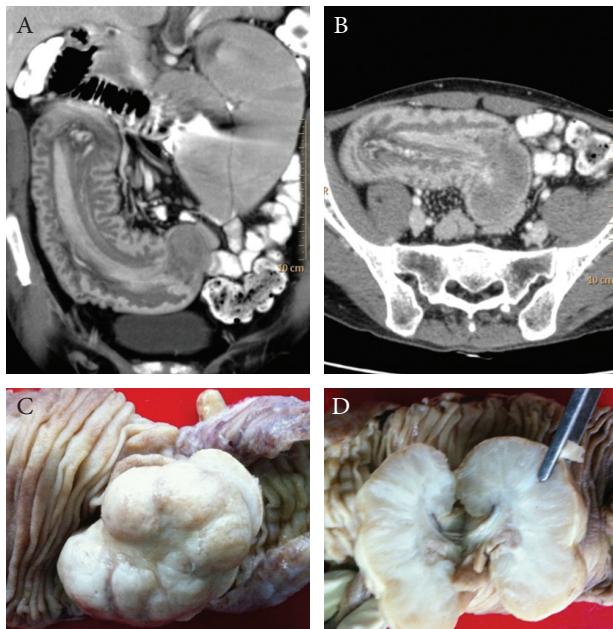


Figure 1 (A, B) Coronal and axial contrast enhanced computed tomography scan images revealing jejunoo-jejunal intussusception with homogenously enhancing polypoidal neoplastic lesion at lead point. (C, D) Small bowel polyp on external and cut surface, respectively

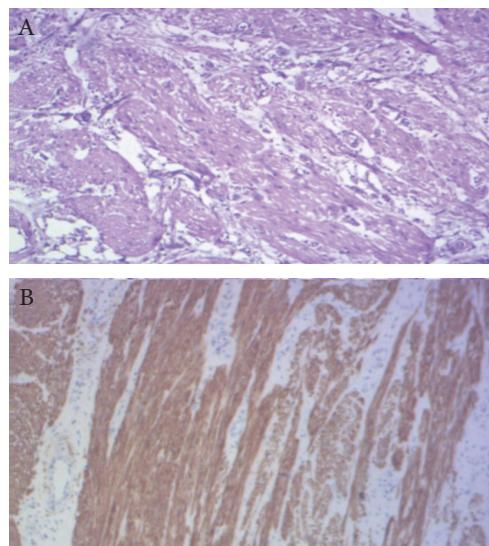


Figure 2 (A) Tumor comprising benign spindle cells arranged in interlacing fascicles (H and E 100 X). (B) Immunohistochemistry desmin showing strong positivity of tumor cells (100 X)

rare [1]. Their clinical manifestations are variable and include abdominal pain, melena, hematochezia and intestinal obstruction, the latter being the most common cause for hospital admission. Benign tumors of the small intestine presenting as acute abdomen requiring immediate intervention are rare. Leiomyomatous polyps in the gastrointestinal tract have been described commonly in the esophagus and rectum. However there are only a few case reports of leiomyoma involving the small intestine [2]. Preoperative diagnosis is difficult to make owing to the absence of specific clinical symptoms and difficulties in radiologic evaluation of small bowel. Ultrasonography and CT scan with or without contrast are of little help since no specific features for small bowel leiomyoma have been described. Leiomyoma in the small bowel can arise either from the muscular layer or small muscles of vasculature. Leiomyomatous polyps can mimic lymphoma, epithelial tumors, neurofibroma or GIST on radiology. Correct diagnosis can be achieved only on routine histopathology or immunohistochemistry [3].

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Unusual polyposis in ulcerative colitis

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A 32-year-old male presented in April 2012 with intermittent pain in the abdomen for 2 years. Frequency of abdominal pain increased over a period of 6 months and was associated with loose stools mixed with blood and mucus. He had lost 6 kg in weight over a period of 6 weeks. There was no history of jaundice, Koch's bacillus, diabetes or hypertension. Physical examination was unremarkable except for pallor. Routine investigation showed hemoglobin 10g/dL, total WBC count 17,030/cmm, platelet 560,000/cmm. Serum alanine aminotransferase (ALT) was 21 IU/L, serum creatinine was 0.8 IU/L, and serum electrolytes were within normal limits. Colonoscopy revealed ulceration, loss of vascularity and friability of mucosa involving the rectum and the entire colon up to cecum (pancolitis) suggestive of ulcerative colitis. Rectal biopsy showed basal plasmacytosis, crypt distortion with cryptitis and loss of goblet cells which was consistent with ulcerative colitis. The patient was treated with oral prednisolon 40 mg at baseline with a tapering dosage and 5 aminosalicylate tablets (5 ASA) 800mg t.i.d. At the end of 2 weeks of treatment the symptoms improved. Steroids were stopped and 5 ASA was continued.

In October 2012 the patient complained of generalized weakness. Bowel frequency was twice daily. There was no history of blood in stool. Colonoscopy done at this time showed extensive colonic polyposis mainly involving the rectosigmoid area. Polyps were pedunculated and sessile ranging from 2 mm to 2 cm. The surface of the polyps on endoscopy was covered with fibrous exudates suggestive of cap polyposis (Fig. 1A). Intervening mucosa appeared to be in remission. Three larger polyps were resected endoscopically and submitted to histopathological examination. On microscopic examination, there was polypoidal configuration of rectal mucosa, covered with a cap of granulation tissue, suggestive of cap polyposis (Fig. 1B). Adjacent rectal mucosa showed elongated and branched crypts. Lamina propria inflammation was mild. There was no evidence of adenomatous change or malignancy.

Cap polyps are rare types of polyps with distinct endoscopic and histological features [1-4]. This lesion was first described by Williams GT [5] in 1985. To our knowledge, no

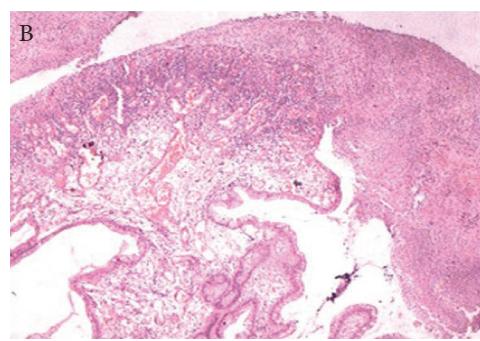


Figure 1 (A) Multiple polyps at endoscopy with fibrinopurulent cap. (B) Polypoidal configuration of colonic mucosa with a cap of granulation tissue on the surface suggestive of cap polyposis (HE 100X)

more than 100 cases have been reported in the literature [1-6]. Cap polyps are identified by the presence of a fibrinopurulent cap on endoscopy and on histology they are characterized by dilated and elongated crypts with a cap of granulation tissue on the surface. Exact etiology is not known. Various possible causes include mucosal prolapse syndrome, ischemia, abnormal bowel motility or inflammation [7-9]. Diarrhea with blood and mucus is the most common presentation of cap polyposis. Clinically they can be misdiagnosed as ulcerative colitis leading to inappropriate treatment [6]. Histopathology can differentiate cap polyposis from inflammatory pseudopolyps of ulcerative colitis by presence of cap of granulation tissue. This was a case of cap polyposis developed in patient of ulcerative colitis, six months later. Patient did not respond to therapy and was subjected to total colectomy. Awareness of this condition and additional case studies are required to evaluate clinical course of cap polyposis.

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Portal hypertension complicating myelofibrosis in a patient without portal or hepatic vein thrombosis

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In the setting of myelofibrosis, portal hypertension often results from portal and/or hepatic vein thrombosis. However, intrahepatic non-cirrhotic portal hypertension without portal or hepatic vein thrombosis is rarely described in these patients [1,2]. Herein, we report a case of myelofibrosis presenting with variceal bleeding in the absence of portal vein or hepatic vein thrombosis.

A 23-year-old female patient presented with massive hematemesis and melena at the emergency department. She had no history of viral hepatitis, alcohol abuse, or any other liver diseases, but she had been diagnosed with JAK^{V617F}

mutation (i.e., the V617F mutation of the Janus kinase 2 gene) negative myelofibrosis by bone marrow biopsy three years ago. Physical examination revealed that the spleen was palpable about 20 cm below the left costal margin. Laboratory tests showed a decreased platelet count of $17 \times 10^9/L$ and hemoglobin concentration of 70 g/L, a relatively normal liver function (alanine aminotransferase: 8 U/L, total bilirubin: 18.7 μmol/L, albumin: 30.4 g/L) and prothrombin time of 15.5 sec. Hepatitis B virus surface antigen and anti-hepatitis C virus antibody were negative. Abdominal color Doppler ultrasound and contrast-enhanced computed tomography demonstrated the patent portal vein and hepatic veins, massive splenomegaly, and no ascites. Upper gastrointestinal endoscopy confirmed that upper gastrointestinal bleeding was caused by large esophageal varices. Liver biopsy was not performed due to a significantly low platelet count. Thus, she was diagnosed with portal hypertension secondary to myelofibrosis. Blood transfusion, octreotide, and prophylactic antibiotics were performed at the emergency department. After that, bleeding was controlled. This patient was transferred to our department of Digestive Interventional Radiology. At this time, laboratory tests demonstrated that hemoglobin concentration was elevated to 83 g/L with a relatively normal liver function (alanine aminotransferase: 16 U/L, total bilirubin: 11.3 μmol/L, albumin: 33.2 g/L) and prothrombin time of 14.7 sec. A transjugular intrahepatic portosystemic shunt was planned, but the patient did not consent. Thus, propranolol was prescribed for the prevention of variceal rebleeding. She was doing well without any episodes of variceal rebleeding about one year after discharge.

Comparably with previous case reports, our case suggests the possibility of intrahepatic non-cirrhotic portal hypertension without portal or hepatic vein thrombosis in myelofibrosis. In clinical practice, myelofibrosis should be excluded in patients with symptomatic portal hypertension of unknown cause. However, due to the absence of liver biopsy, we are uncertain about the presence of liver infiltration from myeloid cells.

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