

Letters to the Editor are welcomed. They may report new clinical or laboratory observations and new developments in medical care or may contain comments on recent contents of the Journal. They will be published, if found suitable, as space permits. Like other material submitted for publication, letters must be typewritten, double-spaced, and must not exceed **two** typewritten pages in length. No more than five references and one figure or table may be used. See "Information for Authors" for format of references, tables, and figures. Editing, possible abridgment, and acceptance remain the prerogative of the Editors.

Acute Hepatitis in a Patient Given Propofol During Colonoscopy

To the Editor: Colonoscopy is an effective colon cancer screening procedure and is becoming increasingly common with our aging population. It is important for physicians to be aware of any potential complications of the procedure. We present a case of acute hepatitis in a patient given propofol during colonoscopy.

A 62-year-old female presented to our emergency room with a one-week history of nausea, vomiting, and epigastric pain. She had a colonoscopy 14 days prior during which multiple small polyps were removed and diverticulosis was noted. Two-hundred and fifty milligrams of propofol was used for sedation during the colonoscopy. Her history was only significant for peptic ulcer disease. She drank two alcoholic drinks per week, smoked two packs of cigarettes per day, denied illicit drug use, and denied taking supplements or herbals. Her vital signs were stable on presentation and the physical exam was remarkable for scleral icterus and a nontender, nondistended abdomen without hepatosplenomegaly. Initial laboratory information showed markedly elevated aspartate aminotransferase (AST) of 2309 U/L and alanine aminotransferase (ALT) of 1313 U/L, alkaline phosphatase of 322 U/L, and total bilirubin of 4.8 mg/dL. She was admitted with a diagnosis of acute hepatitis.

Further evaluation revealed negative markers for hepatitis A, hepatitis B, human immunodeficiency virus, herpes simplex virus, Epstein-Barr virus, cytomegalovirus, acetaminophen, anti-nuclear antibody, antismooth muscle

antibody, iron studies, and urine drug screening; hepatitis C antibody was positive. Abdominal CT showed heterogeneous attenuation of hepatic parenchyma with associated periportal edema consistent with acute hepatitis. Her transaminase peak was on hospital day 3 (Table) with findings consistent with acute hepatocellular injury.

Liver biopsy performed on hospital day 7 showed hepatitis with severe activity and mild to focally moderate fibrosis (Metavir grade A3/stage F2). The severe activity seen on the biopsy was not typical of chronic hepatitis C and raised concern for toxin or drug reaction. After the biopsy, the transaminases trended downward spontaneously. Her symptoms resolved and she was discharged on hospital day 10 on a proton pump inhibitor. Outpatient lab testing showed normalization of her transaminases.

As the liver biopsy was concerning for drug or toxin injury, new drug exposures were sought. The only new exposure that she had in the two weeks prior to admission was the propofol used in the colonoscopy, which was suspected to be the causative agent for her acute hepatitis.

Table. Figure-relevant laboratory information

	October 1, 2007	March 11, 2008	March 13, 2008	March 14, 2008	March 19, 2008	March 21, 2008	March 27, 2008	April 3, 2008
PT (ref 12.3–15.0 s)	—	12.4	—	13.7	15.0	14.0	10.6	10.4
INR	—	0.96	—	1.14	1.34	1.19	1.0	1.0
PTT (ref 24.3–37.2 s)	—	30.3	—	31.1	33.9	32.1	29.4	29.9
Total protein (ref 6.0–8.0 g/dL)	7.5	6.6	6.9	6.7	5.5	6.1	7.6	7.6
Albumin (ref 3.5–5.0 g/dL)	4.1	3.6	3.9	3.7	2.7	3.1	3.9	4.0
Total bilirubin (ref 0.2–1.3 mg/dL)	0.2	4.8	6.7	7.2	10.0	10.5	7.1	4.4
Direct bilirubin (ref 0.0–0.3 mg/dL)	—	—	4.7	4.8	6.8	6.6	—	—
Indirect bilirubin (ref 0.0–1.1 mg/dL)	—	—	0.8	0.9	1.3	1.5	—	—
AST (ref 15–50 U/L)	20	2309	3750	3851	2644	965	98	56
ALT (ref 10–50 U/L)	11	1313	2219	2008	1504	914	197	62
Alkaline phosphatase (ref 40–125 U/L)	94	322	311	289	225	232	196	162

PT, prothrombin time; INR, international normalized ratio; PTT, partial thromboplastin time; AST, aspartate aminotransferase; ALT, alanine aminotransferase; s, seconds.

Propofol (diisopropylphenol) is a lipid-soluble sedative used in anesthesia. It is commonly used as a sedating agent in colonoscopies with monitored anesthesia care.^{1,2} While pancreatitis has been documented in patients receiving propofol,³ there is limited information on the potential hepatotoxic effects of this agent.^{1,4} However, the disturbance of hepatocellular integrity has been associated with propofol anesthesia in surgical patients.⁵ Levels of glutathione transferase alpha (GSTA), a very sensitive marker of hepatocellular integrity, were elevated after propofol infusion in these patients over that of controls. Transaminases were not elevated and no clinical evidence of hepatocellular injury was noted. The investigators did not propose a mechanism for the elevated GSTA levels, but noted that propofol can be a significant metabolic burden to the liver.⁵

There are only two previous cases in the literature of isolated acute hepatitis following propofol administration. One case describes a 66-year-old male who developed hepatitis 48 hours after therapeutic endoscopic retrograde cholangiopancreatography.¹ The patient had slightly elevated alkaline phosphatase and AST and ALT greater than 50 times the normal level, which is consistent with hepatocellular injury. The second case describes a 17-year-old female who developed hepatocellular injury one day after femoral hernia repair.⁴ Liver-associated enzymes in this case showed normal alkaline phosphatase and an AST of 1423 U/L with an ALT of 1567 U/L. In both cases, the hepatitis resolved spontaneously with supportive measures.^{1,4} Unfortunately, neither case had liver biopsy confirmation. Our patient, like the other two case studies, had spontaneous resolution of the hepatitis. The liver biopsy results and the rapid normalization of the transaminases are consistent with hepatitis from drug or toxin injury. Using the validated Roussel Unidad de Coordinación de la Lucha Anti-Fraude Causality Assessment Method, the patient

had a drug causality score of +6 (scale -9 to +15), indicating a probable relationship.⁶

This is an unusual case of acute hepatitis resulting from propofol. As propofol is commonly used in endoscopic procedures, it is important that physicians are aware of this potential adverse reaction, especially when encountering postendoscopy patients.

Huy D. Nguyen, MD

Marie L. Borum, MD, EdD, MPH

Division of Gastroenterology and Liver Diseases
George Washington University Medical Center
Washington, DC

References

1. Polo-Romero FJ, Paricio P, Tovar A, et al. Propofol-induced acute toxic hepatitis after brief sedation for endoscopic retrograde cholangiopancreatography. *Endoscopy* 2008; 40(suppl 2):E49.
2. Aisenberg J, Cohen LB. Sedation in endoscopic practice. *Gastrointest Endoscopy Clin N Am* 2006;16:695-708.
3. Kumar AN, Schwartz DE, Lim KG. Propofol-induced pancreatitis: recurrence of pancreatitis after rechallenge. *Chest* 1999;115: 1198-1199.
4. Anand K, Ramsay MA, Crippin JS. Hepatocellular injury following the administration of propofol. *Anesthesiology* 2001;95: 1523-1524.
5. Tiainen P, Lindgren L, Rosenberg PH. Disturbance of hepatocellular injury associated with propofol anaesthesia in surgical patients. *Acta Anaesthesiol Scand* 1995;39: 840-844.
6. Danan G, Benichou C. Causality assessment of adverse reactions to drugs—I. A novel method based on the conclusions of international consensus meetings: application to drug-induced liver injuries. *J Clin Epidemiol* 1993;46:1323-1330.

Erythroderma as the First Manifestation of Colon Cancer

To the Editor: Skin disorders are common and associations with systemic illnesses, such as end stage renal failure, diabetes mellitus, and thyroid diseases are well recognized. Associations with

underlying nondermatological malignancies have been reported, but are extremely rare.^{1,2}

A previously healthy 76-year-old man presented to the dermatologist with a 1-month history of worsening generalized pruritic rash. Relevant past medical history included hypertension controlled with indapamide for the previous few years. He denied being exposed to any substances that may have caused the skin reaction and was a nonsmoker. On examination, the only positive finding was a generalized exfoliating skin rash consistent with erythroderma. Skin biopsies were negative for underlying skin malignancies. Blood investigations (complete blood count, renal function, blood sugar, liver function test, thyroid function test, and erythrocyte sedimentation rate) were all normal. Chest radiography was normal. Tumor markers showed mildly elevated carcinoembryonic antigen (CEA, 9.5 IU/L, normal range <3.5 IU/L) and carbohydrate antigen (CA 19-9, 177 IU/L, NR <37 IU/L). Alpha fetoprotein and prostate-specific antigen were normal. Gastrointestinal (GI) evaluation and upper GI endoscopy only showed mild gastritis and duodenitis. Colonoscopy showed a concentric tumor at the hepatic flexure. Biopsies confirmed moderately differentiated adenocarcinoma. A computed tomography staging scan showed two small lesions in segments six and seven of the liver. In view of the good functional status, the patient was offered treatment that included chemotherapy with or without attempted resections of both the primary and secondary hepatic lesions. However, the patient declined any further investigations or treatment. He was given topical steroid treatment with some improvement of the skin disorder.

Erythroderma is an uncommon but serious inflammatory skin disorder that typically affects the elderly. Presentations may be acute or insidious. Common associations include pre-existing skin dermatoses (psoriasis, atopic dermatitis, seborrheic dermatitis, and contact dermatitis), medications, and ma-

lignancies. Up to 11% of patients have associations with underlying malignancies and a large proportion are associated with cutaneous T-cell lymphoma.³ Among GI malignancies, associations with esophageal, gastric, and gallbladder cancers have been reported.⁴⁻⁶ To our knowledge, association with underlying colorectal cancer has been previously reported. The underlying pathogenesis of this association is not unknown, but probably represents a paraneoplastic phenomenon. Treatments of the underlying condition have been shown to cause regression of the skin disorders. Therefore, it is important to evaluate for underlying malignancies in patients without any previous dermatological disorders presenting with generalized erythroderma.

Vui Heng Chong, MRCP, FAMS
Chee Chian Lim, MRCP, FAMS,
FRCP

Gastroenterology Unit
Department of Medicine
Raja Isteri Pengiran Anak Saleha (RIPAS)
Hospital
Bandar Seri Begawan BA, Brunei 1710
Darussalam

References

1. Akhyani M, Ghodsi ZS, Toosi S, et al. Erythroderma: a clinical study of 97 cases. *BMC Dermatol* 2005;5:5.
2. Rosen T, Chappell R, Drucker C. Exfoliative dermatitis: presenting sign of internal malignancy. *South Med J* 1979;72:652-653.
3. Sehgal VN, Srivastava G, Sardana K. Erythroderma/exfoliative dermatitis: a synopsis. *Int J Dermatol* 2004;43:39-47.
4. Harper TG, Latuska RF, Sperling HV. An unusual association between erythroderma and an occult gastric carcinoma. *Am J Gastroenterol* 1984;79:921-923.
5. Kameyama H, Shirai Y, Date K, et al. Gallbladder carcinoma presenting as exfoliative dermatitis (erythroderma). *Int J Gastrointest Cancer* 2005;35:153-155.
6. Deffer TA, Overton-Keary PP, Goette DK. Erythroderma secondary to esophageal carcinoma. *J Am Acad Dermatol* 1985;13:311-313.

The Effect of Severe Iron Deficiency Anemia on Primary Photophobia

To the Editor: A 73-year-old physician presented with an exacerbation

of eye pain. Past history included mild hypothyroidism and mild diabetes, both easily treated with oral medications. Past surgeries included fusion of lumbar disk and fractured femur. The patient's symptoms began after six weeks of at least sixteen hour days on the computer preparing manuscripts. The patient had no symptoms until 2001, when he developed dyspnea and severe eye pain. These gradually worsened over a one-month period before he saw his physician. The pain was a 3 on a scale of 1 to 10, with 10 being the worst. Examination by four different ophthalmologists revealed normal sclera, retina, intraocular pressure, etc. Electroretinography at Emory University showed marked constriction of retinal vessels following light exposure.

In February 2004, the patient was hospitalized because of a one-month duration of exertional dyspnea gradually increasing in severity. Physical examination was negative except for pallor. Laboratory findings included a hypochromic, microcytic anemia with hemoglobin of 4.1% and a reticulocyte count of 0.1% with a normal total white blood cell count and differential. A host of radiographic studies including barium x-rays of the upper and lower gastrointestinal tract, gastroscopy, colonoscopy, and MRI of the small bowel revealed no lesions. The guaiacum stool test was negative. Radioisotope studies showed normal vitamin B₁₂ absorption and impaired iron absorption. He was given 6 units of blood and the dyspnea disappeared and the eye pain returned to the usual level of severity. Following discharge from the hospital, he was placed on oral iron, 1 gm daily. During the following three years, the severity of the eye pain did not vary much, although it became more intense during the brightest days of the summer months, and during the 2 to 3 weeks prior to Christmas when most houses in the city had electric light displays. His hemoglobin varied from 12 to 14 gm%. In early March 2007, the pa-

tient noticed the appearance of exertional dyspnea and a progressive increase in the severity of his eye pain. Two weeks later, he consulted his physician to check his hemoglobin; it was 7.1 gm%. Injections of Imferon restored his hemoglobin to 12.1 gm% and his symptoms abated.

Herman Hugh Fudenberg, MD,
DDG, IOM

NIT Research Foundation
Inman, SC

Lansoprazole-Induced Acute Interstitial Nephritis

To the Editor: A 70-year-old Caucasian male presented to his primary care provider with a history of fatigue and nausea of two weeks' duration. The patient denied fever, vomiting, or other constitutional symptoms. Three weeks prior to presentation, the patient took lansoprazole for his worsening reflux symptoms. Four days after taking the lansoprazole, he experienced nausea and two loose stools for a day. He then discontinued the medication. The patient did not report reduced oral intake despite the nausea. He had an 8-year history of hypertension and took lisinopril 5 mg once daily and intermittently took omeprazole for gastroesophageal reflux disease. The last dose of omeprazole was four weeks prior to taking the lansoprazole. No abnormalities were found during his annual screening three months prior to his presentation.

On examination, the patient was afebrile, with a pulse rate of 62/minute and blood pressure of 138/88, and without change in orthostatic blood pressures. He did not have a rash, and the rest of the examination was unremarkable. The complete blood count with differential was normal; but the patient's blood urea nitrogen (BUN) was elevated to 45 mg/dL, and his creatinine was 3.8 mg/dL. The rest of his metabolic parameters were normal. The urinalysis was normal and failed to show eosinophiluria. The renal ultrasound was also normal.

After admission to the hospital, lisinopril was discontinued and the patient received intravenous crystalloids for the next 48 hours. BUN and creatinine decreased to 38 mg/dL and 3.1 mg/dL, respectively, but remained at those values. Antineutrophil cytoplasmic antibody (ANCA) and complement levels were normal. Since there was no improvement in the kidney function, the patient underwent a kidney biopsy. It revealed diffuse infiltration with lymphocytes, monocytes, and occasional eosinophils, normal glomeruli, and mild tubulitis suggestive of acute interstitial nephritis. During his last visit to the clinic, the patient's creatinine was 2.8 mg/dL.

To our knowledge, this is only the fourth case reported suggesting that lansoprazole can cause acute interstitial nephritis (AIN).¹ Although our patient was exposed to omeprazole, he did not take proton pump inhibitors (PPI) for approximately four weeks prior to taking lansoprazole. Due to the temporal relation of the patient's symptoms, renal failure, and exposure to lansoprazole, the drug can be classified as a "certain" medication causing AIN.² The prompt recognition of the offending agent is the cornerstone in managing drug-induced AIN; especially as the clinical presentation of PPI-induced AIN is unusual.³ We conducted a survey (n = 23/70) at our hospital to investigate the awareness among physicians of potential nephrotoxicity of PPIs. Ninety-six percent of physicians did not recognize the potential nephrotoxicity of PPIs. Physicians need to be aware of this side effect, because patients are at risk of permanent loss of kidney function.³

Joshua Eken, Medical Student
University of North Dakota School
of Medicine
Grand Forks, ND

Gautam Phadke, MD
PGY 3, Department of Internal Medicine
University of North Dakota School
of Medicine
Grand Forks, ND

Sumera Ahmed, MD
Internal Medicine
MeritCare Health System
Fargo, ND

Adit Mahale, MD, FACP

Department of Internal Medicine, Division
of Nephrology
University of North Dakota School
of Medicine
MeritCare Health System
Grand Forks, ND

References

1. Jose J, Saravu K, Khera K, et al. Acute interstitial nephritis related to lansoprazole administration. *J Pak Med Assoc* 2008;58:206–207.
2. Edwards IR, Aronson JK. Adverse drug reactions: definitions, diagnosis, and management. *Lancet* 2000;356:1255–1259.
3. Sierra F, Suarez M, Rey M, et al. Systematic review: proton pump inhibitor-associated acute interstitial nephritis. *Aliment Pharmacol Ther* 2007;26:545–553.

Dieulafoy Lesion: A Rare Cause of Gastrointestinal Bleeding

To the Editor: Dieulafoy lesion is an uncommon cause of major gastrointestinal bleeding and may be difficult to recognize. It consists of a large caliber artery that protrudes through a mucosal defect in the stomach causing significant and often recurrent hemorrhaging from a pinpoint nonulcerated arterial lesion. It has been identified more frequently in recent years due to increased awareness. Dieulafoy lesion is thought to be the cause of acute and chronic upper gastrointestinal bleeding in approximately 1–2% of these cases.¹ The incidence, however, might vary from 0.5% to 14%, depending upon selection criteria.² Because a history of nonsteroidal anti-inflammatory drug (NSAID) intake, peptic ulcer symptoms, or alcohol abuse is usually absent, the condition is difficult to recognize.

A 19-year-old woman with a non-significant past medical history who recently emigrated from Thailand presented to the hospital with dizziness, lightheadedness, headache, progressive dyspnea, bilateral leg swelling, and a fever for two days. She also had three episodes of coffee ground vomiting and melena on the day of admission. She was using acetaminophen and Thai herbal medications for dysmenorrhea.

She was a nonsmoker, nondrinker, and denied any drug abuse. Her blood pressure was 101/50, pulse rate was 116, and temperature was 38°C. She was lethargic and dyspneic, and her skin and conjunctiva were pale. Lung examination was normal, but the cardiovascular exam revealed tachycardia. While the abdominal examination was unremarkable, she had tarry stools on rectal exam. Extremity examination revealed moderate bilateral ankle edema. Her blood test showed a hemoglobin of 2.8 g/dL, albumin of 2.8, and a total protein of 4.5. Her electrolytes and chemistry were within normal limits. She was admitted to the intensive care unit with a diagnosis of upper gastrointestinal bleeding and severe anemia. Treatment with intravenous fluids and packed red blood cells was started. Her symptoms improved markedly in a few days and she became afebrile. Her stay was complicated by a modest elevation in troponin with normal creatine phosphokinase. Echocardiography revealed a normal ejection fraction with no wall motion abnormalities. This was attributed to supply ischemia. Upper endoscopy revealed a normal esophagus and a Dieulafoy lesion in the fundus with a stigmata of recent bleeding. The lesion was coagulated with a bipolar probe. The antrum was hyperemic but there were no ulcers identified in the stomach or duodenum. A biopsy for *Helicobacter pylori* was negative. Colonoscopy was unremarkable. The patient remained symptom-free and was discharged from the hospital with proton pump inhibitor therapy.

Dieulafoy lesion should be considered when evaluating any acute and recurrent major gastrointestinal bleeding. If unrecognized, it may cause a life-threatening hemorrhage. Usually, the mean hemoglobin level on admission has been reported to be between 8.4–9.2 g/dL in various studies.^{1,3} Awareness of the condition is a key to accurate diagnosis. It can be easily overlooked at endoscopy as concomitant lesions such as ulcers or varices may wrongly be considered responsible for the bleeding episode. Treatment is by endoscopic modalities

like electrocoagulation and successfully achieves permanent hemostasis in 85% of cases.⁴ This case illustrates a rare and inherently difficult lesion to recognize, because it presents with very low hemoglobin, which is usually uncommon in Dieulafoy lesion, and did not have any risk factor for gastrointestinal bleeding. In practice, we have to consider unusual causes of common diseases to decrease their mortality and morbidity.

Musa Njeru, MD

Ali Seifi, MD

Zia Salam, MD, FACE

Lawrence Ognibene, DO, FACOI

Department of Internal Medicine
AtlantiCare Regional Medical Center
Atlantic City, NJ

References

1. al-Mishlab T, Amin AM, Ellul JP. Dieulafoy's lesion: an obscure cause of GI bleeding. *J R Coll Surg Edinb* 1999;44:222-225.
2. Baettig B, Haecki W, Lammer F, et al. Dieulafoy's disease: endoscopic treatment and follow up. *Gut* 1993;34:1418-1421.
3. Dy NM, Gostout CJ, Balm RK. Bleeding from the endoscopically-identified Dieulafoy lesion of the proximal small intestine and colon. *Am J Gastroenterol* 1995;90:108-111.
4. Ho KM. Use of Sengstaken-Blakemore tube to stop massive upper gastrointestinal bleeding from Dieulafoy's lesion in the lower oesophagus. *Anaesth Intensive care* 2004;32:711-714.

Endobronchial Ultrasound-Guided Transbronchial Needle Aspiration for Diagnosis of a Mediastinum Mass with Severe Central Airway Obstruction After Stenting

To the Editor: Severe central airway obstruction (CAO) is sometimes complicated by various benign and malignant mediastinum lesions.¹ Clinical signs of airway obstruction, the extent of the disease, or comorbidities sometimes make these patients poor candidates for surgery, especially for elderly patients. We report a case of

an 80-year-old male patient with a past history of thyroid papillary carcinoma who received bilateral total thyroidectomy. He was admitted to the hospital complaining of severe dyspnea and stridor. His pretreatment spirometric data were: FEV1/FVC ratio of 56.1%; FEV1 of 1.41 L (60.8% predicted); and peak expiratory flow (PEF) of 1.63 L/min (23.8% predicted). Chest computed tomography (CT) revealed a low-density mass shadow in the right upper mediastinum adjacent to the trachea extending from the right lobe of the thyroid, which compressed the trachea with high-grade tracheal stenosis. The narrowest diameter of the trachea was 4.2 mm. Because his high-grade CAO could have become completely obstructed and life threatening, the complications of surgery and general anesthesia could not be ignored. Therefore, we inserted a flexible bronchoscope through the patient's oral cavity and released the Ultraflex™ (Boston Scientific, Natick, MA) stent to the obstructed area. After the stent had been implanted, his dyspnea and stridor improved. His post-treatment spirometric data were: FEV1/FVC 81%; FEV1 1.78 L (76.8% predicted); and PEF 5.68 L/min (82.5% predicted). Clinical and radiographic findings improved subsequently after we had restored his tracheal patency by the tracheal stent. In order to diagnose the mediastinal tumor, we performed endobronchial ultrasound-guided transbronchial needle aspiration (EBUS-TBNA) with a convex probe equipped with a 7.5 MHz linear probe on its tip (BF-UC260F-OL8, Olympus, Ltd., Tokyo, Japan) to puncture the tumor with real-time guidance. The convex probe was placed below the uncovered stent and a 22-gauge, 4 cm cytology needle (BF-UC260F-OL8, Olympus, Ltd., Tokyo, Japan) was used to obtain specimens. The TBNA needle was inserted into the patient's mediastinal tumor through the pore of the stent. The histology from the EBUS-TBNA core biopsy specimens confirmed a spindle cell tumor.

The patient tolerated the procedures well and without complications.

Patients with CAO from both malignant and benign airway conditions present with worrisome symptoms such as dyspnea and cough and the condition may be life threatening. In some of these patients, the extent of the disease or comorbidities precludes curative surgery. At this time, tracheal stent implantation can immediately relieve the symptoms and provide an alternative palliative modality for patients with mediastinal lesion-related CAO who are poor surgical candidates.²

EBUS-TBNA is a new modality that allows real-time transbronchial needle aspiration of mediastinal and hilar lesions. EBUS-TBNA is minimally invasive, can be done quite safely, and can be performed under local anesthesia in an outpatient setting.³ In addition to its ease of use, EBUS-TBNA provided histological cores that were adequate for immunohistochemistry and a definite diagnosis. Moreover, the convex-probe EBUS is also equipped with a Doppler mode and can evaluate the hypervascularity of the tumor.

To our knowledge, there have been no reports of EBUS-TBNA for tissue sampling through the pore of an uncovered tracheal stent. The 22-gauge, 4 cm cytology needle can be inserted through the pore of the uncovered metallic stent without damaging its structure. After EBUS-TBNA puncture, the shape of the stent was still viable.

In conclusion, if a patient with a mediastinal tumor causing CAO precludes curative surgery due to the extent of the disease or comorbidities, bronchoscopy with Ultraflex™ stent implantation can serve as a palliative management to restore airway patency. After that, EBUS-TBNA can be performed and the puncture needle can be inserted for tissue sampling through the pore of the uncovered stent successfully and without complications.

Chia-Hung Chen, MD

Ho-Yiu Wong, MD

Yi-Heng Liu, MD

Wei-Chun Chen, MD

Yu-Sheng Lin, MD

Chih-Yen Tu, MD

Division of Pulmonary and Critical Care
Medicine
Department of Internal Medicine
China Medical University Hospital
Taichung, Taiwan

References

1. Ernst A, Feller-Kopman D, Becker HD, et al. Central airway obstruction. *Am J Respir Crit Care Med* 2004;169:1278–1297.
2. Noppen M, Poppe K, D’Haese J, et al. Interventional bronchoscopy for treatment of tracheal obstruction secondary to benign or malignant thyroid disease. *Chest* 2004;125:723–730.
3. Herth FJ, Becker HD, Ernst A. Ultrasound-guided transbronchial needle aspiration: an experience in 242 patients. *Chest* 2003;123:604–607.

Ingestion and Endoscopic Retrieval of Tweezers in a 23-Year-Old Patient

To the Editor: A 23-year-old male of Puerto Rican descent presented to our institution requesting the surgical removal of a pair of tweezers, which he reportedly swallowed by mistake the evening prior. He denied having suicidal ideation, but psychiatric history included lacerating his arms and abdomen at the age of 8 after witnessing physical abuse by his father towards his mother. Family history was remarkable for a paternal grandfather with bipolar disorder. On presentation, he denied dysphagia, odynophagia, nausea, vomiting, chest pain, dyspnea, abdominal pain, hematochezia, or melena.

The patient appeared youthful and comfortable. He was hemodynamically

stable. Cardiac and respiratory examinations were benign. His abdomen was soft, nondistended, and nontender with the presence of normoactive bowel sounds. Laboratory evaluations, including electrolyte panel, complete blood count, coagulation profile, and alcohol level were within normal limits.

An abdominal radiograph revealed a 10 cm metallic object in the stomach (Figure). No free intra-abdominal air, obstruction, or dilated bowel loops were seen. Chest radiograph was unremarkable for esophageal emphysema or pneumomediastinum.

Given the patient’s history of trauma, appendectomy, and a gunshot wound to the abdomen, our surgical team cautioned that a laparotomy would result in multiple adhesions. The patient agreed to an endoscopic retrieval of the foreign object. Under conscious sedation, an endoscope

was advanced into the stomach, revealing a broken tweezer freely floating in the pylorus. No tissue damage or implantation of the foreign body into the stomach mucosa was seen. An overtube was subsequently inserted which successfully allowed for the retrieval of the object without damaging the esophagus on return.

Given his involvement with dangerous behaviors and his history of self-mutilation, the patient was medically cleared afterwards and sent to the psychiatric care unit.

Charles P. Koczka, MD

Department of Medicine
SUNY Downstate Medical Center
Brooklyn, NY

Min Win, MD

Adam Goodman, MD
Department of Gastroenterology
SUNY Downstate Medical Center
Brooklyn, NY



Fig. Abdominal x-ray revealing a 10 cm metallic object seen in the stomach. No free intra-abdominal air, obstruction, or dilated loops appreciated.

CME Answers

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| 1. B | 4. C |
| 2. C | 5. A |
| 3. C | 6. C |