You're the Flight Surgeon

This article was prepared by Matthew H. Ramage, M.D., M.S.

It is Monday morning and you have just finished your Flight Medicine clinic daily brief. You drink your last gulp of coffee and start off to clinic. Your first patient of the day is an emergency department (ED) follow-up from the weekend. He is a 24-yr-old male air traffic control student who presented for evaluation of excruciating right upper quadrant/mid-epigastric pain that radiated to his back, coupled with nausea. The pain began at rest with no known precipitating factors. The pain was reduced by 25% following a "GI cocktail," but did not resolve completely until 2 h later. He was discharged from the ED with no further treatment following 6 h of observation and told to follow up with his primary care physician. He denies any pain at the present.

1. What should be your next step in management?

A. So that he does not miss any class time, return him to controlling status. He is asymptomatic.
B. Start him on a trial of proton pump inhibitor (PPI) and return him to controlling status after the 72-h ground trial.
C. Obtain ED treatment records.
D. Keep him off of controlling status and refer to gastroenterology for complete work-up.

ANSWER/DISCUSSION

1. C. It is common that airmen will be treated at the ED and not have an understanding of what actions were taken. It is always important to get a thorough history, integrate your independent history with that of the ED, and perform an independent interpretation of the various studies that have been performed. While it is important with the trainee population to maximize effective class time on controlling status, proper medical care of the member takes precedence over other duties. The PPI may be a valid option, but only after a complete evaluation. Gastroenterology referral may be needed for further evaluation; however, you have not exhausted your own clinical capabilities.

After asking your clinic staff to obtain any recent ED records, you continue to interview your patient. He reveals that he had had sudden-onset right upper quadrant/mid-epigastric pain that radiated to his back and woke him from sleep. The pain was aggravated by movement and alleviated by bending forward and the aforementioned "GI cocktail." He denies any chest pain, shortness of breath, wheezing, or changes in stooling. He states that he had eaten a large plate of spaghetti with tomato sauce 1-2 h prior to symptom onset. He denies ever having an episode of this severity, but he has had three to four previous episodes of intermittent upper abdominal pain that began 5 mo previously. The episodes were nonlimiting and he did not attribute them to any specific cause. He takes no medication currently, but he has unsuccessfully treated himself with TUMS®, spoons full of baking soda, and Prilosec©. His abdominal pain does not acutely change with food intake. His pain symptoms seem to resolve 3-4 h after onset. He admits to intermittent and progressive difficulty with swallowing solid food over the past few months. He states that he enjoys his training as an air traffic controller. He describes himself as a "high strung worrier," but denies any depressive symptoms. He states that he will lie awake briefly each night and "make lists" in his head for the next day for about 5 min. He does not smoke and he drinks two to three beers per week on the weekend. He is physically active and serves as a CrossFit® instructor. He denies any significant family history.

Your aeromedical technician knocks on the office door and hands you the ED report. Complete metabolic panel, complete blood count, cardiac enzymes, amylase, lipase, urine drug screen, coagulation studies, electrocardiogram, and chest X-ray were all normal and unremarkable. He was afebrile on presentation with normal vital signs as he is currently. The treating physician gave him 1 L of normal saline and a "GI cocktail" that consisted of 30 ml of a 1:1:1 Mylanta®, viscous lidocaine, and Donnatal®. He was told that he had a "stomach ulcer" and was discharged. Your examination reveals no abnormal findings. His abdomen is soft, nontender, nondistended, no masses, no hepatosplenomegally, and he has normal bowel sounds.

2. Given the above information, what is the most likely diagnosis to explain these symptoms?

A. Acute viral gastroenteritis.
B. *Helicobacter pylori*-associated gastritis.
C. Gastroesophageal reflux (GERD).
D. Zenker's diverticulitis.

DOI: 10.3357/AMHP.4058.2015

E. Acute pancreatitis.
F. Anaphylactic food allergy.

ANSWER/DISCUSSION

2. C. With the provided information, GERD is the most likely diagnosis. GERD can elicit substernal chest pain, especially in the postprandial period. Additionally, long standing GERD can result in an esophageal stricture that can lead to dysphagia.

The term "acute viral gastroenteritis" is often used as a catch-all term to describe any form of acute abdominal discomfort not readily attributable to another source. While acute viral gastroenteritis can cause nausea, abdominal pain, and emesis, in this case there was no diarrhea or change in stool.[12]

Helicobacter pylori-associated gastritis should be considered; however, the symptoms generally do not last for more than a few days. The diagnosis can be made in the acute phase with a positive biopsy with negative anti *H. pylori* IgG. In some individuals, the infection can later develop into painful peptic ulcers.[3]

A pharyngoesophageal diverticula (Zenker's diverticula) is a protrusion of the pharyngeal mucosa through an area of muscular weakness. When larger, neck pain can occur as well as halitosis and regurgitation of undigested food.[8]

Acute pancreatitis is a painful inflammatory process of the pancreas that results in mid-epigastric abdominal pain that can radiate to the back and typically without colic. Pancreatitis can stem from biliary obstruction or systemic etiologies such as elevated triglycerides or toxins such as alcohol. In an acute exacerbation, serum amylase and lipase levels will rise within 2-12 h, with lipase remaining elevated for a period after the acute pain has subsided.[9] Pancreatitis is a concern, but the serum amylase and lipase were negative and the pain resolved spontaneously.

Anaphylaxis is an IgE-mediated immune response to a sensitized allergen that causes mast cell activation. This process is less likely with oral administration of an allergen and typically occurs via parenteral routes such as insect envenomation or inhalation of allergens. The symptoms of anaphylaxis vary among individuals, but typically include hypotension, tachycardia, urticaria, bronchospasm, laryngeal edema, diarrhea, and onset within minutes of the offending allergen exposure.[11]

The unknown etiology of the abdominal pain was discussed with the patient, and a comprehensive investigation was begun, including anti *H. pylori* IgG and a detailed food/symptoms journal with a follow-up visit in 7-10 d. An abdominal ultrasound was considered, but was deferred until a later date. Due to the stressful nature of air traffic controller training and reported feeling of worry, the possibility of a psychosomatic etiology was not excluded. He was kept off of controlling status until further clarification of his symptoms.

The patient returned to clinic a week later with a detailed food journal that correlated epigastric/substernal pain with nearly every meal. He denied any symptoms to the degree that previously sent him to the ED; however, he had constant mid-epigastic pain that persisted for a few hours after eating and then resolved spontaneously. He found no predictable food associations, nor specific exacerbation with fatty meals. On further questioning, he admits that in addition to the pain, he has been having intermittent difficulty swallowing solid food. He also states that he has had to eat slowly since he was a child due to painful swallowing of larger food boluses. The *H. pylori* antibody test result returned with a negative result. A barium swallow was performed that was negative for any signs of constriction; however, there was equivocal peristaltic function.

3. What should be the next step in evaluation?

A. *H. pylori* stool antigen testing.
B. Abdominal ultrasound.
C. PPI trial.
D. Esophagogastroduodenoscopy (EGD).
E. Calcium channel blocker trial.

ANSWER/DISCUSSION

3. D. Due to the patient's development of dysphagia, it is indicated that he undergo EGD evaluation. The barium swallow study that was performed prior was inconclusive. In light of the previous negative *H. pylori* anti-IgG results, the *H. pylori* antigen testing will likely not provide useful information. For completeness of evaluation, the abdominal ultrasound would be beneficial; however, the mid-epigastric pain concurrently with dysphagia points away from a biliary cause. PPI and/or calcium channel blocker initiation might be beneficial in the treatment of his symptoms, but the cause of the dysphagia must be addressed.

Over the course of 2 wk while awaiting gastroenterology consultation and subsequent EGD, the patient had a negative abdominal ultrasound, a negative celiac panel, and failed trial of a lactose/gluten free diet. He continued to have substernal/mid-epigastric pain with increasing frequency of three to four times daily, albeit not to the level that caused him to present to the ED; however, he required two to four Percocet tablets per day to cope with the pain. The patient was seen by the local gastroenterologist; an upper endoscopy grossly revealed a diffusely erythematous lumen with scattered white papules. There was trachealization of the esophagus, becoming more pronounced on the distal aspect, resulting in a stricture superior to the gastroesophageal junction. Histologically there was eosinophilic proliferation in the tissue, and the white papules were found to be eosinophilic microabscesses. The patient was given oral fluticasone and Nexium© for treatment of the eosinophilic esophagitis (EE). He underwent extensive allergen testing that can only be described as "pan-positive." The patient showed significant reaction to all tested foods except melon and white meat chicken. The patient initiated a drastic elimination diet, which only consisted of white meat chicken, melon, and water.

4. According to the American College of Gastroenterology, which of the following is not a diagnostic criterion for EE?

A. Esophageal biopsy with ≥ 15 eosinophils/hpf.
B. Symptoms of esophageal dysfunction (dysphagia).
C. Esophageal eosinophilia persistent after PPI trial.
D. A response to dietary elimination.
E. Exclusion of secondary causes of eosinophilia.

ANSWER/DISCUSSION

4. D. Response to treatment or elimination therapy is supportive for the diagnosis, but not compulsory. These are the following diagnostic

criteria for EE as outlined by the American College of Gastroenterology in 2013 (adapted from UptoDateOnline, November 2013, "Pathogenesis, clinical manifestations, and diagnosis of eosinophilic esophagitis," Table 2):

1. Esophageal eosinophilia is a clinicopathological disorder diagnosed by clinicians taking into consideration both clinical and pathological information without either of these parameters interpreted in isolation, and defined by the following criteria:
   - Symptoms related to esophageal dysfunction;
   - Eosinophil-predominant inflammation on esophageal biopsy, characteristically consisting of a peak value of ≥ 15 eos/hpf;
   - Mucosal eosinophilia is isolated to the esophagus and persists after a PPI trial;
   - Secondary causes of esophageal eosinophilia are excluded; and
   - A response to treatment (dietary elimination, topical corticosteroids) supports, but is not required for, diagnosis.
2. Esophageal biopsies are required to diagnose EE. Two to four biopsies should be obtained from both the proximal and distal esophagus to maximize the likelihood of detecting esophageal eosinophilia in all patients in whom EE is being considered.

The pathogenesis of EE is incompletely understood at this time. One model suggests that various individuals have a genetic predisposition to enhanced eosinophil recruitment as a response to environmental or food-borne allergies. The chronic presence of eosinophils in the local tissue yields an inflammatory response that invokes remodeling with hypertrophy and hyperplasia.[4] It is thought that priming of the immune system with mucocutaneous allergens can contribute to a more brisk eosinophil response in the gastrointestinal tract, thus making symptoms worse during times of higher allergen exposure.[10]

Eosinophilic esophagitis has a significant association with other immunologic disease entities such as food allergies and environmental allergies, asthma, and atopic dermatitis.[7] Eosinophilic esophagitis can mimic GERD, but the key distinction is that the eosinophilia evident on esophageal biopsy persists in EE after a 2-mo trial of PPI.[2]

In civil aviation, air traffic control specialists can either be Federal Aviation Administration employees and fall under series 2152 rules or they can be contractors and required to comply with Federal Aviation Administration second class physical standards. While there is no specific prohibition for EE, in accordance with 14 CFR 67.213 (b)(c), there is limitation in qualification for duty if a general medical condition or medication causes impairment in function.[5] Esophagitis that is defined as severe or persistent is grounds for determination of retention in the U.S. Air Force as well as disqualification as a ground-based controller.* In the U.S. Navy, the diagnosis of EE is disqualifying for general naval service as well as air traffic controlling.[1,6] In the U.S. Army, air traffic controllers require a Class 4 physical. In accordance with Army Regulation 40-501, chronic esophagitis is disqualifying for duty.[13]

The patient attained maximal medical improvement 3 mo after diagnosis with oral fluticasone, 40 mg Nexium®, and strict food elimination. His symptoms drastically improved, yet he still required occasional opiate analgesia for one to two pain attacks per month. He underwent a Medical Evaluation Board, which ruled that he should be medically discharged from active duty. This decision was based on the safety-sensitive nature of air traffic controlling and the likelihood that he could be incapacitated by a sudden substernal pain exacerbation. He has since returned to his home of origin and works happily as an emergency medical technician.

Ramage MH. *You're the flight surgeon: eosinophilic esophagitis.* Aerosp Med Hum Perform. 2015; 86(4):418–421.

ACKNOWLEDGMENTS

The author would like to thank Col. Patrick Storms, USAF, MC, SFS, Program Director for the Residency in Aerospace Medicine, U.S. Air Force School of Aerospace Medicine, Wright-Patterson Air Force Base, OH, for his professional review of this article. The views expressed in this article are those of the author and do not necessarily reflect the official policy or position of the Air Force, the Department of Defense, or the U.S. Government.

REFERENCES

1. Bureau of Medicine and Surgery. Manual of the medical department. Washington, DC: Department of the Navy; 2013:15-42(4), 15-44(1). NAVMED P-117. Retrieved 15 December 2013 from http://www.med.navy.mil/directives/Pages/NAVMEDP-MANMED.aspx.
2. Dellon ES, Gonsalves N, Hirano I, Furuta GT, Liacouras CA, Katzka DA. ACG clinical guideline: evidenced based approach to the diagnosis and management of esophageal eosinophilia and eosinophilic esophagitis (EoE). Am J Gastroenterol 2013; 108:679–92.
3. Graham DY, Genta RM. Gastritis and helicobacter pylori. In: Goldman L, Ausiello D, eds. Cecil textbook of medicine, 22nd ed. Philadelphia: Saunders; 2004:823-4.
4. Lucendo AJ, Arias A, De Rezende LC, Yagüe-Compadre JL, Mota-Huertas T, et al. Subepithelial collagen deposition, profibrogenic cytokine gene expression, and changes after prolonged fluticasone propionate treatment in adult eosinophilic esophagitis: a prospective study. J Allergy Clin Immunol 2011; 128:1037–46.
5. National Archives and Records Administration. General medical condition. In: Code of Federal Regulations. Washington, DC: Government Printing Office; 2011. 14 CFR 67.213 (b)(c). Retrieved 15 December 2013 from http://www.gpo.gov/fdsys/granule/CFR-2011-title14-vol2/CFR-2011-title14-vol2-sec67-213/content-detail.html.
6. Naval Aerospace Medical Institute. 7.12. Eosinophilic esophagitis. In: U.S. Navy aeromedical reference and waiver guide. Pensacola, FL: Naval Aerospace Medical Institute; 2013. Retrieved 15 December 2013 from http://www.med.navy.mil/sites/nmotc/nami/arwg/Pages/AeromedicalReferenceandWaiverGuide.aspx.
7. Noel RJ, Putnam PE, Rothenberg ME. Eosinophilic esophagitis. N Engl J Med 2004; 351:940–1.
8. Orlando RC. Diseases of the esophagus. In: Goldman L, Ausiello D, eds. Cecil textbook of medicine, 22nd ed. Philadelphia: Saunders; 2004: 821.

* U.S. Air Force. Medical standards directory; 2013:G3/12. Retrieved 15 December 2013 from https://kx2.afms.mil/_layouts/login/Privacy.htm?ReturnUrl=%2fkj%2fkx4%2fFlightMedicine%2f_layouts%2fAuthenticate.aspx%3fSource%3d%252Fkj%252Fkx4%252FFlightMedicine%252FDocuments%252FMedical%2520Standards%2520Directory%2520%2528MSD%2529%252FMSD%25202013%252DDec%252D2%252Epdf&Source=%2Fkj%2Fkx4%2FFlightMedicine%2FDocuments%2FMedical%20Standards%20Directory%20%28MSD%29%2FMSD%202013%2DDec%2D2%2Epdf. Available to those with access.

9. Owyang C. Pancreatitis. In: Goldman L, Ausiello D, eds. Cecil textbook of medicine, 22nd ed. Philadelphia: Saunders; 2004:880-1.
10. Prasad GA, Alexander JA, Schleck CD, Zinsmeister AR, Smyrk TC, et al. Epidemiology of eosinophilic esophagitis over three decades in Olmsted County, Minnesota. Clin Gastroenterol Hepatol 2009; 7:1055–61.
11. Schwartz LB. Systemic anaphylaxis. In: Goldman L, Ausiello D, eds. Cecil textbook of medicine, 22nd ed. Philadelphia: Saunders; 2004:1616.
12. Semrad CE, Powell DW. Approach to the patient with diarrhea and malabsorption. In: Goldman L, Ausiello D, eds. Cecil textbook of medicine, 22nd ed. Philadelphia: Saunders; 2004:844.
13. U.S. Army. 4-4. Abdomen and gastrointestinal system; 4-18. Lung and chest wall. In: Standards of medical fitness. Washington, DC: Department of the Army; 2011:41,45. Army Regulation 40-501. Retrieved 19 March 2014 from http://armypubs.army.mil/epubs/pdf/r40_501.pdf.

This article was prepared by Jeffrey Lee Moore, D.O., Cody R. Jackson, M.D., Jeffrey C. Ellis, PA-C, and Corrianne Norrid, NC.

You are the flight surgeon in an overseas clinic on your way out to lunch when the clinic's physician assistant (PA) asks you about a patient. A 36-yr-old male patient presents with left-sided upper back pain just below the angle of the scapula that radiates to the front at the costal margin. The pain woke him up from sleep the night prior, but he was able to fall back asleep. Vital signs show a body mass index of 32, blood pressure of 138/92, and pain 8/10, and are otherwise normal. The electrocardiogram and review of systems are unremarkable. His physical exam is normal except he is very tender to palpation in the left upper quadrant with a palpable abnormality. The patient is very vocal and guards on exam. Radiology and laboratory are closed for lunch.

As the supervisory physician, you examine the patient, based on the concern expressed by the PA. In the 15 min it took to discuss the case, the abdomen is now nontender, which is a surprise to both the patient and the PA. In the left upper quadrant, at the border of the rectus muscle and ribs, you feel a small, round 1-cm lump. It is rubbery, mobile, and nontender, and determined to be a noncontributory lipoma. The patient was given a gastrointestinal cocktail and discharged with instructions to go to the emergency room for worsening symptoms or to follow up the next day.

Later that evening, the patient reported to the emergency room with increased abdominal pain, nausea, vomiting, and radiating flank pain to the groin. He had no fever, dysuria, hematemesis, or gross hematuria. A computed tomography (CT) showed a large, heterogeneous, left suprarenal, nonenhancing, well-encapsulated mass with mixed fatty and soft tissue attenuation without calcification measuring 17 cm × 14 cm × 12 cm (**Fig. 1**). No left adrenal gland was seen. This mass did not appear to invade the adjacent structures. No adenopathy was identified. There was mass effect on the left kidney, spleen, pancreas, and bowel in the left upper quadrant. The scan was otherwise unremarkable.

1. Based on the radiologic finding, what would your treatment plan entail?

A. Pain control.
B. Discharge home.
C. Urology referral.
D. Medical evacuation.
E. All of the above.

ANSWER/DISCUSSION

1. E. Taking into consideration that you are in a small overseas hospital with only orthopedic, obstetric, and general surgeons, all of the above answers are correct. Pain control and discharge home with close primary care manager follow-up are essential for monitoring the patient's status while awaiting medical evacuation. If oral pain control fails or his presentation worsens, hospital admission can always be considered. Getting the patient to a urologic specialist is imperative to the patient's care. Contact was made with the regional medical center and it was advised that the patient be sent to a higher level of care near his family. Tumors of this size in a young individual have a poor prognosis.[7] Medical evacuation is the best way to get the patient home for workup, removal, and pathological diagnosis.

Before you can narrow down the diagnoses, it is helpful to determine the severity of the lesion from the CT imaging. When dealing with cystic renal masses, the Bosniak Classification is helpful in diagnosing and managing these lesions through CT evaluation (6). CT imaging allows us to see certain morphologic features along with enhancement characteristics that guide the need for further evaluation and determine malignant risks that likely require further follow-up. The Bosniak Classification is categorized into five different groups:

- Category I – A benign simple renal cyst
  Features: Thin walled, no septa, no calcifications, and no solid components
  Attenuation: Low, like that of water
  Enhancement: None
  Workup: None
  Malignancy: ~0%
- Category II – Benign cystic lesions that are < 3 cm in diameter
  Features: Thin walled, few thin septa, thin calcification, well marginated
  Attenuation: High (due to proteinaceous or hemorrhagic fluid)
  Enhancement: None
  Workup: None
  Malignancy: ~0%

DOI: 10.3357/AMHP.4026.2015