CLINICAL VIGNETTE

Intractable Nausea and Vomiting in an Elderly Patient

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Case Report

A 75-year-old female with seronegative rheumatoid arthritis and frequent falls was admitted to a skilled nursing facility (SNF) two weeks after an elective hospital admission for spinal surgery. She had fallen one month prior to her back surgery and sustained a burst fracture of her fourth lumbar vertebra, resulting in spinal stenosis. Surgery consisted of an L3-L4 laminectomy and L3-L5 fusion. Surgery was complicated by epidural hemorrhage, resulting in right leg weakness.

At the time of admission to the SNF, she had 5/10 pain in both her legs, and persistent weakness of her right leg. Although oxycodone was ordered as-needed for pain, it was seldom used. Her other medications included sertraline, bupropion, methotrexate and gabapentin.

About three weeks into her SNF stay, she developed an episode of nausea and vomiting without other abdominal symptoms, which initially responded to ondansetron. She had transient constipation three weeks prior which had resolved. One week after her episode initial onset of nausea, her nausea and vomiting recurred - this time with a bilious appearance. Diagnostic studies were notable for an unremarkable metabolic panel, and a plain abdominal radiograph showing minimal adynamic ileus. Due to ongoing nausea and poor oral intake, she remained on maintenance intravenous fluids, with scheduled ondansetron and as-needed prochlorperazine to treat her nausea. After brief improvement in her nausea and a gradual tolerance of a regular diet, her intractable nausea and vomiting returned, and she was re-admitted.

Upon hospital admission, she had stable vital signs. Labs, including complete blood count, basic metabolic panel, liver function tests, and lipase were unremarkable except for mild hypoalbuminemia. Computed tomography (CT) scan of the abdomen and pelvis with contrast showed copious stool throughout the colon. Her nausea and vomiting were initially attributed to constipation and prolonged post-operative ileus. She was treated with a potent bowel regimen and a nasogastric tube placement for bowel decompression. Her ileus and constipation eventually resolved but, her nausea and vomiting persisted. Patient was observed to have episodes of food regurgitation soon after eating - both in the presence and absence of nausea. Gastroenterology was consulted and she underwent and an esophagogastroduodenoscopy (EGD), which showed a normal appearing esophagus without stricture, ring, diverticulum, or esophagitis. EGD did show scattered gastric erosions, but helicobacter pylori test returned negative. She was treated with a proton-pump inhibitor and sucralfate but they did not alleviate her symptoms.

Due to her ongoing nausea and vomiting, the patient underwent a barium esophagram which demonstrated severe esophageal dysmotility. She was started on a trial of IV erythromycin to increase GI motility. However, after 6 days of IV erythromycin, she still had no significant improvement in her ability to tolerate food. Due to concern for worsening protein-calorie malnutrition, gastric tube placement was discussed with the patient and her son. After much deliberation by both patient and her family, she underwent successful gastric tube placement and tolerated tube feeds well. Her hospital course was also notable for refeeding syndrome requiring IV phosphorous and thiamine. The gastroenterologist recommended outpatient follow-up for further evaluation of esophageal dysmotility and consideration for possible interventions.

After a prolonged four-week hospitalization the patient was transferred to a different SNF, receiving nutrition exclusively through tube feeds, with nothing being given by mouth. She remained free of nausea after admission, and about one month after her discharge from the hospital, she cautiously initiated oral feedings and eventually advance to a mechanical soft diet. With increasing oral intake, her tube feedings were reduced to half the original amount.

Discussion

This case illustrates a unique presentation of severe esophageal dysmotility. Esophageal dysmotility describes a disturbance in the structure, musculature, swallow and contractile mechanisms that involved the esophagus. Symptoms of esophageal dysmotility may present as dysphagia, non-cardiac chest pain, reflux, nausea and vomiting or regurgitation.

Esophageal motility disorders can result from physiologic changes and are affected by age-related disease burden and medications. Signs, symptoms, and typical features of the disorder may present differently in older adults. In 1964, the term “presbyesophagus” described age-related decrease in contractile amplitude, polyphasic waves in the esophageal body, incomplete sphincter relaxation, and esophageal dilation).[1] Other studies have described changes in upper esophageal sphincter function that may be related to reduction

in neurons in the myenteric plexus.[2] Upper esophageal sphincter (UES) pressures also decline and contraction velocity and duration decrease with age.[3]

Autoimmune connective tissue diseases, especially rheumatologic conditions can also cause damage to vital organs, including the gastrointestinal tract. The direct action of antibodies against organs and also pharmacological therapies can increase risks for delayed motility. In addition to dysmotility, fibrosis, stricture and ulcerations can occur.[4] Prompt diagnosis and treatment may reduce risk of complications, including malnutrition, dehydration and increased mortality.[5]

Clinical observations of gastroesophageal reflux disease (GERD) found a age factor affecting esophageal contractile strength and peristaltic function.[6] These changes in upper esophageal function may affect esophageal motility and account for subsequent complaints of reflux, nausea, regurgitation or dysphagia. The significance of these changes may not be initially obvious and presentations may be clinically silent or minimally symptomatic.

One study examined aging impact on degeneration of esophageal motility and whether this was affected by GERD.[7] Patients with GERD underwent diagnostic manometry and were analyzed by age. Esophageal contraction waves above the lower esophageal sphincter and peristaltic contractions were reviewed. Esophageal peristalsis was affected by age independent of reflux, whereas GERD had an impact on esophageal contraction wave amplitudes, but not on peristalsis.

Another review examined impact of age on manometric esophageal characteristics and function. Contraction amplitudes and maximum active tension decreased with age. Aging also caused increased stiffness in the esophageal wall along with decreased peristalsis. These changes may increase the risk for reflux and dysmotility in the elderly.[6] Also, diminished esophageal myenteric neurons result in reduced peristaltic function, a phenomenon that contributes to motility disorders such as achalasia.[2] Overall, older adults may be at greater risk for motility disturbance and have decreased ability to compensate for additional stress or caustic injury.

In addition to a thorough clinical history and exam, appropriate radiologic, endoscopic and manometric diagnostics are important in evaluating motility disorders. High resolution manometry with esophageal pressure topography is used to classify motility disorders based on the Chicago Classification.[8] Manometry evaluates relaxation pressure in the esophagogastric junction (EGJ) as well as peristalsis. The pattern of dysmotility ranges from complete absence of contractility, disordered and spastic, to hypercontractile which can help classify the type of motility disorder. Achalasia is a disorder of the lower esophageal sphincter where the esophageal sphincter fails to relax and body fails to undergo peristalsis. Pseudoachalasia mimics achalasia, but can be due to infectious causes or malignancy. Other conditions include spastic disorders such as diffuse esophageal spasm and nutcracker esophagus, and other nonspecific motility disorders which are benign and nonprogressive. Non-specific motility disorder describes presentation of dysphagia, reflux, nausea, regurgitation with abnormal findings of motility and peristalsis, without fulfilling the criteria for defined diagnoses. In this case, EGD was overall unrevealing, however esophagram demonstrated severe esophageal dysmotility. High resolution esophageal manometry was advised to isolate the location of dysfunction, and this study is still pending.

The goal of treatment is aimed at symptom management, maintaining nutritional needs and improving quality of life. Treatment options include pharmacologic or interventional modalities. Medications would include anti-emetics, promotility agents and proton pump inhibitors to control nausea, gastroparesis and reflux. For persistent symptoms, calcium channel blockers, nitrates and low dose tricyclic antidepressants may be considered. There are limited data to support the use of other therapies such as non-TCA antidepressants and SSRIs which can lower esophageal sensitivity.[9] Botulinum toxin may offer effective but temporary relief for those with hypercontractile esophageal peristalsis and persistent symptoms.[10,11] Peroral endoscopic myotomy (POEM) may also provide short term relief, however, evidence is limited and the procedure has higher risk for complications such as postoperative dysphagia.[12]

Conclusion

This vignette reviews the development and persistence of intractable nausea, vomiting, and regurgitation in an older individual with severe esophageal dysmotility. Older adults experience age-related physiologic changes that affect critical organ systems. They may manifest symptoms atypically, and disturbance in gastrointestinal functions may predispose them to higher risks for motility disorders. Other, multifactorial contributors include comorbidities and polypharmacy can affect symptoms. Although there is no clear gender association for GI motility disorders, females have increased incidence for autoimmune conditions which can affect dysmotility. This patient had underlying rheumatoid arthritis, pain after her spinal surgery requiring multimodal management, and a protracted SNF and hospital course, which cumulatively may have contributed to the severity of her presentation. Increased vigilance to atypical indicators is crucial when caring for the older population and should warrant prompt evaluation to avoid delays in diagnosis and management.

REFERENCES

1. **Soergel KH, Zboralske FF, Amberg JR**. Presbyesophagus: esophageal motility in nonagenarians. *J Clin Invest*. 1964 Jul;43(7):1472-9. doi: 10.1172/JCI105023. PMID: 14192528; PMCID: PMC289623.
2. **Eckardt VF, LeCompte PM**. Esophageal ganglia and smooth muscle in the elderly. *Am J Dig Dis*. 1978

May;23(5):443-8. doi: 10.1007/BF01072928. PMID: 677096.

3. **Castell DO**. Esophageal disorders in the elderly. *Gastroenterol Clin North Am*. 1990 Jun;19(2):235-54. PMID: 2194944.
4. **Kato R, Nakajima K, Takahashi T, Miyazaki Y, Makino T, Kurokawa Y, Yamasaki M, Takiguchi S, Mori M, Doki Y**. A case of advanced systemic sclerosis with severe GERD successfully treated with acotiamide. *Surg Case Rep*. 2016 Dec;2(1):36. doi: 10.1186/s40792-016-0162-5. Epub 2016 Apr 13. PMID: 27072944; PMCID: PMC4829569.
5. **Cabre M, Serra-Prat M, Palomera E, Almirall J, Pallares R, Clavé P.** Prevalence and prognostic implications of dysphagia in elderly patients with pneumonia. *Age Ageing*. 2010 Jan;39(1):39-45. doi: 10.1093/ageing/afp100. Epub 2009 Jun 26. PMID: 19561160.
6. **Gregersen H, Pedersen J, Drewes AM.** Deterioration of muscle function in the human esophagus with age. *Dig Dis Sci*. 2008 Dec;53(12):3065-70. doi: 10.1007/s10620-008-0278-y. Epub 2008 May 8. PMID: 18461452.
7. **Gutschow CA, Leers JM, Schröder W, Prenzel KL, Fuchs H, Bollschweiler E, Bludau M, Hölscher AH**. Effect of aging on esophageal motility in patients with and without GERD. *Ger Med Sci*. 2011;9:Doc22. doi: 10.3205/000145. Epub 2011 Aug 18. PMID: 21863136; PMCID: PMC3159207.
8. **Kahrilas PJ, Ghosh SK, Pandolfino JE**. Esophageal motility disorders in terms of pressure topography: the Chicago Classification. *J Clin Gastroenterol*. 2008 May-Jun;42(5):627-35. doi: 10.1097/MCG.0b013e31815ea291. PMID: 18364587; PMCID: PMC2895002.
9. **Nguyen TM, Eslick GD**. Systematic review: the treatment of noncardiac chest pain with antidepressants. *Aliment Pharmacol Ther*. 2012 Mar;35(5):493-500. doi: 10.1111/j.1365-2036.2011.04978.x. Epub 2012 Jan 13. PMID: 22239853.
10. **Marjoux S, Brochard C, Roman S, Gincul R, Pagenault M, Ponchon T, Ropert A, Mion F**. Botulinum toxin injection for hypercontractile or spastic esophageal motility disorders: may high-resolution manometry help to select cases? *Dis Esophagus*. 2015 Nov-Dec;28(8):735-41. doi: 10.1111/dote.12282. Epub 2014 Sep 12. PMID: 25212219.
11. **Vanuytsel T, Bisschops R, Farré R, Pauwels A, Holvoet L, Arts J, Caenepeel P, De Wulf D, Mimidis K, Rommel N, Tack J**. Botulinum toxin reduces Dysphagia in patients with nonachalasia primary esophageal motility disorders. *Clin Gastroenterol Hepatol*. 2013 Sep;11(9):1115-1121.e2. doi: 10.1016/j.cgh.2013.03.021. Epub 2013 Apr 13. PMID: 23591282.
12. **Khashab MA, Saxena P, Kumbhari V, Nandwani M, Roland BC, Stein E, Clarke JO, Stavropoulos S, Inoue H, Pasricha PJ**. Peroral endoscopic myotomy as a platform for the treatment of spastic esophageal disorders refractory to medical therapy (with video). *Gastrointest Endosc*. 2014 Jan;79(1):136-9. doi: 10.1016/j.gie.2013.08.021. PMID: 24342590.