Case Report

Gastrostomy tube migration complicated with acute pancreatitis: Two case reports with review of literature

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ABSTRACT

The percutaneous endoscopic gastrostomy (PEG) tube is an important method of providing enteral nutrition to patients with swallowing disorders and those who need long-term enteral nutritional support. The association between PEG tube migration and acute pancreatitis is rare and was previously described in the literature. To the best of our knowledge, only 11 cases have been reported in the literature. In this article, we are describing two cases of acute pancreatitis secondary to PEG tube balloon migration to the duodenum. These two case reports exemplify that PEG tube migration to the duodenum is not uncommon, and it may lead to disturbance of the biliary flow, obstruction of the ampulla of vater, and acute pancreatitis.

Key Words: Gastrostomy, pancreatitis, percutaneous endoscopic gastrostomy tube, percutaneous endoscopic gastrostomy tube complications

INTRODUCTION

Percutaneous endoscopic gastrostomy (PEG) tube is an important method of providing enteral nutrition to patients with swallowing disorders and those who need long-term enteral nutritional support. Because of its ease and safety of placement, the number of patients with PEG tubes continues to rise. Most of the complications associated with PEG tube are minor, but several have the potential to cause significant morbidity if not recognized and treated correctly. These two case reports illustrate one of the problems related to PEG tube migration to the duodenum and its associated complications, including disturbance of the biliary flow and acute pancreatitis.

CASE REPORTS

Case #1

A 76-year-old Hispanic female who is a nursing home resident, with a past medical history of Alzheimer dementia, diabetes mellitus, coronary artery disease, hypothyroidism, and oropharyngeal dysphagia for which she had a PEG tube inserted 1 year before her admission. Her PEG tube was replaced in the nursing home with a balloon gastrostomy tube shortly before her presentation. The patient was sent from the nursing home for abdominal pain of 1 day duration, the pain was associated with nonbloody nonbilious vomiting, without any change in bowel habits, fever, or chills. Her list of medications included tylenol, plavix, diltiazem, insulin lispro, levothyroxine, simvastatin, and esomeprazole. Initial vital signs showed blood pressure of 145/70 mmHg, pulse rate of 110 beat/min, and respiratory rate was 18 breaths/min, and respiratory rate was 18 breaths/min, oxygen saturation was 98% on room air. Physical examination was significant for tachycardia, normal heart sounds with no murmurs or added sounds, normal breath sounds and a soft abdomen with epigastric tenderness and active bowel...
sounds, PEG tube was noted in place. Laboratory studies showed hemoglobin of 12.1 g/dL, white blood cell (WBC) 8900/µL (normal, 4500–11,000/µL), platelets 322,000/µL (normal, 150,000–450,000/µL), creatinine 1.53 mg/dL (normal, 0.6–1.3 mg/dL), urea 60 mg/dL, glucose 381 mg/dL, calcium 9.3 mg/dL, albumin 3.3 g/dL, alkaline phosphatase 248 U/L (normal, 50–136 U/L), total bilirubin 0.5 mg/dL (normal, 0.3–1.2 mg/dL), aspartate aminotransferase 22 U/L (normal, 10–30 U/L), alanine aminotransferase 28 U/L (normal, 10–40 U/L), lipase 918 U/L (normal, 31–210 U/L), serum triglyceride, and serum calcium were within normal limits. Abdominal ultrasound showed distended gallbladder with no gallstones, sludge or biliary dilatation. Computed tomography (CT) scan of the abdomen [Figure 1] revealed the suboptimal position of the PEG tube, with the balloon and tip in the second/third portion of the duodenum, without evidence of pancreatic inflammation. A diagnosis of acute pancreatitis was made based on high lipase and abdominal pain. At bedside, the PEG tube was pulled back to stomach and secured to the abdominal wall. The patient was treated conservatively with intravenous fluid and analgesics. Within the following 2 days, all of her symptoms improved, with normalization of her lipase, alkaline phosphatase, and creatinine. The patient was discharged after 2 days, and she did not have any further episodes of pancreatitis.

Case #2
A 42-year-old male with past medical history of traumatic brain injury and quadriplegia had a gastrostomy tube placement 2 years ago for enteral feeding, he presented to our service with 1 day history of epigastric pain, associated with fever, chills. No vomiting or changes in bowel movements. Also an increased residual volume through gastrostomy tube was noted.

On initial evaluation, the physical examination showed vital signs within normal limits excepts for sinus tachycardia, unremarkable chest examination, abdominal examination showing gastrostomy tube in the mid-epigastric area, tenderness on superficial palpatation in the epigastric area, no guarding, or rigidity. Laboratory studies showed WBC 16,000/µL (normal, 4500–11,000/µL), platelets 165,000/µL (normal, 150,000–450,000/µL), creatinine 1.75 mg/dL (normal, 0.6–1.3 mg/dL), blood urea nitrogen 25 mg/dL (normal, 7–18 mg/dL), glucose 135 mg/dL, calcium 9 mg/dL, albumin 3.6 g/dL (normal, 3.5–5 g/dL), alkaline phosphatase 143 U/L (normal, 50–136 U/L), total bilirubin 3.3 mg/dL (normal, 0.3–1.2 mg/dL), aspartate aminotransferase 205 U/L (normal, 10–30 U/L), alanine aminotransferase 99 U/L (normal, 10–40 U/L), lipase 12,883 U/L (normal, 31–210 U/L), amylase 1996 U/L (normal, 25–115 U/L), serum triglyceride, and serum calcium were within normal limits. Abdominal ultrasound showed mildly distended gallbladder with no gallstones. CT of the abdomen showed the migration of the PEG tube to the second part of the duodenum with compression on ampulla of vater, pancreatic head edema, and peripancreatic inflammatory changes, suggestive of acute pancreatitis [Figure 2].

Although the gastrostomy tube was repositioned and pulled directly after the diagnosis was made, the patient had a complicated course in the hospital, with septic shock, multi-organ failure, third spacing, and capillary leak syndrome. We think that the poor outcome was mostly due to the delay in the diagnosis of acute pancreatitis.

DISCUSSION

PEG tube placement has become the preferred method to provide nutritional support for patients requiring long-term (>4–6 weeks) enteral nutrition, due to its ease and safety of placement. Especially for those with oropharyngeal dysphagia and impaired swallowing due to neurological events.
Since it was first described in 1980, PEG tubes use has increased significantly. It is estimated that 10% of nursing home residents and about 1.7% of Medicare patients over the age of 85 years undergo gastrostomy tube placement. The incidence of short- and long-term complications related to PEG actual insertion is low. The overall complication rate has been reported to be between 4% and 23.8%, with a procedure-related mortality of 0–2%. The most common complications related to established PEG tubes are peristomal infection and leakage, buried bumper syndrome, inadvertent removal, and fistulous tracts formation. More serious complications are infrequent and include peritonitis, necrotizing fasciitis, and aspiration.

Acute pancreatitis secondary to migrated replacement gastrostomy tubes has also been reported in the literature. To the best of our knowledge, a total of 11 cases were reported, four of these cases were reported in the period of 1986 until 2005. Eight cases were additionally reported in the period of 2005 until 2014. Whether this is due to increased prevalence of PEG tube use, increased awareness or even increased reporting of this potential complication is unclear. Brauner et al. described 11 cases of reported acute pancreatitis caused by tube dislodgement in the duodenum, six of them had foley catheter, three had balloon gastrostomy with external disk bumper, and two had PEG with external disk bumper. All mentioned cases were attributed to replacement of PEG tube, none to new insertion. While time from the replacement of the tube to the onset of symptoms varied widely from 1 day to 1 year. Shah et al. emphasized the importance of avoiding the use of foley catheter as permanent replacement options, as it was associated more with migration and subsequent pancreatitis.

In our first case, which is the more frequent of the two, the PEG tube was replaced shortly before pancreatitis, in the setup of a medical facility without any immediate complications and with no radiographic confirmation, then it was secured with an outer bumper and the balloon was inflated. We hypothesize that the tube migrated to the duodenum due to peristalsis or it was mistakenly placed in the duodenum when it was replaced. While difficult to prove causality, the rapid response of symptoms and surrogate endpoints with correction of the PEG tube position, especially with the continuation of all of the patient’s medications, favors PEG tube disturbance of biliary flow as a precipitating factor to acute pancreatitis.

Our two cases represent an addition to the literature and illustrate the potential complications associated with PEG tube migration to the duodenum. Early recognition and management are essential to optimize outcomes. To avoid this complication we recommend periodic examination and documentation of the distance of the balloon from the skin to document the position of the tubes or any inadvertent migration of the tubes. The balloon was inflated with saline in both cases. The PEG tube was secured to the abdominal wall with stitches in the first case only. Furthermore, the use of Foley catheters as permanent replacement tubes should be discouraged as they are more likely to migrate, because they lack the external bumper to secure the tube to the abdominal wall, and they also lack the markings on the surface of the catheter which does not allow measurements of the depth of balloon placement.

CONCLUSION

The exact incidence of PEG tube migration to the duodenum is unclear, and early recognition and treatment of this complication is crucial. The physician should be aware of the symptoms and signs of PEG tube migration and their complications, and use the appropriate diagnostic studies to confirm the diagnosis, especially in the setting of associated acute pancreatitis.

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Conflicts of interest
There are no conflicts of interest.

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