

Recurrent Percutaneous Endoscopic Gastrostomy induced Pancreatitis

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Keywords

Pancreatitis, Epigastric abdominal pain, Nausea, PEG.

Introduction

Acute and recurrent pancreatitis is often categorized by etiology of pathogenesis namely, obstructive, toxic, infectious, and idiopathic. Mechanical ampullary obstruction by gallstones, biliary sludge, ascariasis, periampullary diverticulum, pancreatic or periampullary cancer, ampullary stenosis, duodenal stricture or obstruction are well described causes of acute and chronic pancreatitis [1]. However, the literature on iatrogenic mechanical ampullary obstruction such as by percutaneous endoscopic gastrostomy tube (PEG) migration is much sparser.

The incidence of major complications associated with PEG tube placement is low and fatality is very rare. However minor complications such as the formation of insertion site granulation tissue, tube breakage and leakage and stomal site infection are quite common with rates up to 70% in certain series [2-6]. PEG tube migration has been described as a cause of gastric outlet obstruction [7], duodenal obstruction [8], and rarely as the etiology of obstructive pancreatitis and cholangitis [9,10].

In this report we describe a patient who was diagnosed with PEG migration induced pancreatitis. The diagnosis was supported by clinical presentation, blood tests, imaging, gastroscopy and the recurrence of the episode two weeks following the initial presentation.

Case Report

We report a case of recurrent PEG induced pancreatitis in a 78-year-old woman who initially presented with three days of epigastric abdominal pain, nausea and vomiting. The patient had been PEG-fed since suffering a right middle cerebral artery cerebrovascular accident (CVA) 5 year earlier. The PEG had been

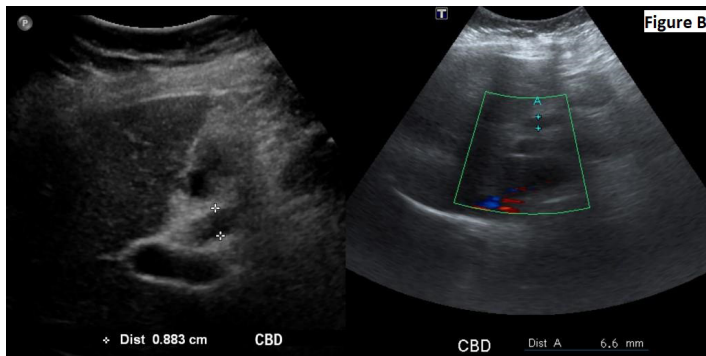
changed to 20F balloon-type device (Bard access systems, Salt Lake City, UT, USA) nine months prior to presentation. Other past medical history included: post-CVA seizure disorder and increased intracranial pressure treated with VP shunt placement, type 2 diabetes mellitus, hypertension and dyslipidemia. She was treated with metformin, atorvastatin, phenytoin and pantoprazole.

Physical examination at presentation demonstrated mild tenderness in the epigastric area on palpation, without peritoneal signs. A 20F PEG in the LUQ was noted with evidence of tube migration secondary to loosened bolster. The distance between the balloon and external disc bumper was 11 cm measured by a scale indicated on the PEG-tube. The patient was afebrile, and had no skin rashes, palpable adenopathy, or hepatosplenomegaly. Vitals signs were within normal range and stable.



Initial laboratory testing was significant for an elevated pancreatic amylase of 1116 U/L (normal range: 13-53 U/L), normal liver

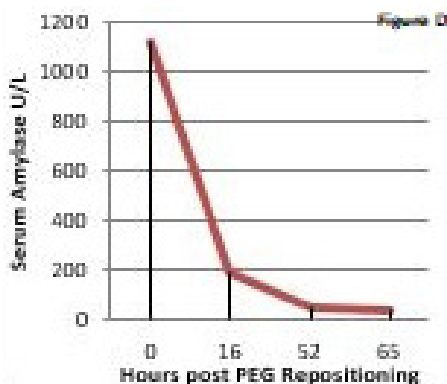
enzymes, lactate, lipid profile and calcium. An abdominal computed tomography (CT) scan performed was significant for PEG balloon migration and positioning in the second stage of the duodenum (white arrowhead on figure A). An abdominal ultrasound (US) ruled out presence of biliary stones and was significant for common bile duct (CBD) dilation to 9 mm (Figure B, left frame).



The PEG was pulled back and secured to the abdominal wall. Gastric lavage post PEG repositioning exhibited a fresh bloody aspirate, thus a gastroscopy was performed. A front view scope revealed an area of ulceration and erosion with bowel wall edema caused by the malpositioned PEG balloon eroding into the duodenal mucosa adjacent to the major duodenal papilla (Figure C).



Post repositioning of the PEG patient's nausea, vomiting and abdominal pain resolved and amylase levels rapidly normalized (Figure D) providing support for migrated PEG induced pancreatitis. Follow up US performed 30 hours post PEG repositioning revealed CBD decompression with normal CBD diameter of 6 mm (Figure B, right frame). Subsequently the patient was discharged to her residence.



Two weeks post discharge the patient re-presented to ER with a similar if not identical symptom complex of nausea, vomiting and epigastric abdominal pain. Her physical exam was significant for PEG external bolster loosening and balloon migration, and mild epigastric tenderness. Her pancreatic amylase was elevated to 350 U/L (normal range: 13-53 U/L) and a repeat CT scan again failed to demonstrate cholelithiasis and there was redemonstration of PEG balloon migration to the second part of the duodenum. During the second admission the PEG was removed and exchanged. This led to prompt resolution of patient's symptoms as well as correction of her laboratory abnormalities back to baseline. She was discharged to her residence and has remained asymptomatic with no recurrence of pancreatitis since.

Discussion

PEG tubes are used for enteral feeding in patients who are likely to have prolonged inadequate or absent oral intake. We describe here the development of recurrent acute pancreatitis in a 78-year-old woman who was fed by a PEG for 5 years following a CVA. Abdominal imaging including CT and US as well as gastroscopy revealed migration of the tube balloon into the second part of the duodenum leading to acute obstructive pancreatitis.

The reported rates of minor complications following PEG tube placement vary from 16 to 70% (2-6). Association with timing of PEG insertion may be used to categorize these complications, although many can be seen at any time following tube placement. These include infection, bleeding, peristomal leakage, and inadvertent tube removal. Some complications are seen immediately following PEG tube placement and include pneumoperitoneum, ileus, perforation of the esophagus or stomach (at a site other than the gastrostomy), or damage to other intra-abdominal organs. Late complications occur after the gastrostomy tract has matured and include deterioration of the gastrostomy site, buried bumper syndrome, and colcutaneous fistula formation [4,5,9].

There are rare reported cases of obstructive jaundice, pancreatitis and cholangitis induced by misplaced or migrated PEG tubes [10,11]. This potentially serious complication can take place at any time following PEG placement. In our literature search we encountered eleven case reports describing pancreatitis associated with gastrostomy tube migration [11-20]. A summary of our literature search is presented in table 1.

Our report is unique in providing the most conclusive evidence for PEG induced pancreatitis with combination of supporting blood work, CT scan, ultrasound, and endoscopic studies. In the presented case we hypothesize spontaneous loosening of the external bolster led to PEG balloon migration and apposition near the major duodenal papilla. Balloon erosion into the mucosa and bowel wall edema adjacent to the papilla (Figure C) led to ampullary obstruction, backflow of pancreaticobiliary secretions and CBD dilation. The rapid resolution of CBD dilation with normalization of pancreatic amylase as well as endoscopic evidence of the PEG balloon eroding into duodenal wall adjacent to the duodenal papilla strongly support our hypothesis. The recurrence of similar

Author	Patient	Etiology	Factors supportive of PEG induced pancreatitis
Bui & Dang 1986	37 M developmental delay, Quadriplegic	Migration of 16F Foley catheter	-Amylase: 1247 U/L (N: 50-110) normalized over 7 days post adjustment -Confirmed on barium contrast radiography
Paniccek et al, 1988	11 M Severe mental retardation	Foley catheter inserted directly into duodenum and balloon inflated.	-Amylase: 1536 U/L remained elevated over 11 days post adjustment -Confirmed on abdominal radiograph with injection of soluble contrast through gastrostomy tube and CT
Barthel & Magnum 1991	32 F CVA induced dementia, Quadriplegic	Migration of 22F Foley catheter	-Amylase: 686 U/L (N:<115) normalized 11 days post repositioning -Confirmed on ERCP: Pancreas divisum; Foley catheter feeding tube tightly applied to the left anterior wall of the duodenum compressing the minor papilla
Duerksen 2001	26 F Cerebral palsy	Migration of 16F balloon gastrostomy tube	-Lipase 920 IU/L (N:30-180) normal value noted 4 weeks post adjustment. -Confirmed on CT: gastrostomy tube present in the second part of the duodenum and on barium contrast radiography: migrated tube in the descending duodenum with the inflated bulb in the region of the major duodenal papilla
Miele & Nigam 2005	44 F anoxic brain injury	Migration of Foley catheter	-Amylase elevated (value not specified) normalized rapidly -Confirmed on US and CT: Dilated intra and extrahepatic biliary tree and ERCP: The major papilla was completely compressed
Periselnieris et al, 2006	5 F CVA	16 F Corflo balloon Gastrostomy tube	-Amylase: 703 IU/L (N: <110) normalized 6 days post adjustment. -Confirmed on MRCP (before): balloon in second stage of duodenum and -Ultrasound (before): Dilated biliary tree, and forward viewing gastroscopy and side viewing duodenoscopy
Imamura et al, 2007	86 F CVA	Migration of gastrostomy-tube, Bard Access Systems 18F	-Amylase: 1191 U/L (N: 32-112) normalized 2 days post adjustment. -Confirmed on abdominal roentgenography and CT (before): the tip of PEG-tube with the inflated balloon was migrated into the second portion of the duodenum, no stones
Shah et al, 2012	79 M CVA on prednisone	Migration of Foley catheter	-Amylase: 1448 U/L (N: 0-100) -Confirmed on ultrasound (before): No stones, sludge or biliary dilatation CT (before): Foley catheter balloon was identified in the second portion of the duodenum
Shah et al, 2012	38 F mental handicap	Migration of Foley catheter	-Amylase: 1,602 U/L (N: 0-100). -Confirmed on ultrasound (before): No stones, sludge or biliary dilatation and -CT (before): Foley catheter balloon was identified in the second portion of the duodenum
Brauner & Kluger 2014	92 F Alzheimer's dementia, CVA	Migration of PEG with external disc bumper	-Urinary amylase: 11,460 U/L normalized 2 days post adjustment -Confirmed on US (before) enlarged gallbladder with no gallstones or sludge. There were no signs of cholecystitis CBD dilated to 16 mm and CT (before): balloon located in the second part of the duodenum and peripancreatic fat stranding with an edematous pancreatic head -US (one day post adjustment): CBD 11 mm

sequence of events two weeks post patient's discharge further substantiates PEG migration induced pancreatitis.

This case highlights the importance of monitoring proper PEG positioning post-insertion and during maintenance care. Ensuring that the external gastrostomy tube bolster is positioned such that 1 to 2 cm of in and out movement is permitted will prevent migration. This will reduce the risk of gastric outlet or small bowel obstruction as well as mechanical ampullary blockage potentially leading to pancreatitis. We suggest confirming proper PEG positioning whenever a patient with a feeding gastrostomy is diagnosed with pancreatitis or obstructive jaundice. As seen in this case and others described in the literature, although rare, PEG migration induced pancreatitis is easily reversible by simple PEG repositioning.

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