Buried Bumper Syndrome with a Fatal Outcome, Presenting Early as Gastrointestinal Bleeding after Percutaneous Endoscopic Gastrostomy Placement

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Abstract:
Percutaneous Endoscopic Gastrostomy (PEG) has gained wide acceptance among patients who require prolonged tube-feeding support. A rather unusual complication of PEG placement is migration of the internal bumper through or into the abdominal wall. This was first described in 1988 and is called the buried bumper syndrome (BBS). The syndrome is a late complication of PEG tube placement. The manifestations of the syndrome must be recognised and the patient referred for emergency endoscopy and removal of the bumper. Failure to recognise this syndrome may result in serious complications including gastrointestinal bleeding, perforation of the stomach, peritonitis and death. We describe a case where a patient developed the buried bumper syndrome quite early after PEG placement. The syndrome manifested with gastrointestinal bleeding. Although we removed the buried bumper endoscopically, and placed another PEG tube, the patient developed peritonitis and died 16 hours after the removal of the migrated bumper. (J Postgrad Med 2003;49:325-7)

Key Words: PEG, Buried bumper syndrome, Migration.

Percutaneous endoscopic gastrostomy (PEG) has been widely used since the last few years in order to provide long-term nutritional support to patients unable to maintain an adequate oral intake. Although the incidence of complications related to this technique is low, many problems have been reported since its introduction in 1980. The buried bumper syndrome (BBS), in which the internal bumper migrates from the gastric lumen and lodges in the gastric wall or anywhere along the gastrostomy tract, is an uncommon long-term complication of PEG tubes. We report a case of BBS that occurred quite early after PEG placement and caused upper gastrointestinal bleeding which is an unusual manifestation of the syndrome. Although the bumper was removed and a new tube placed, migration of the bumper resulted in peritonitis and death.

Case History
A 32-year-old woman was admitted to the Neurosurgery unit of our hospital because of severe craniofacial injury following a car accident. Several weeks later we were asked to place a gastrostomy tube in the patient. A 22F MIC PEG system (Medical Innovations Corporation, Milpitas California) was placed without difficulty. After a week the patient was discharged and written instructions were given to her caregivers. Monthly telephone follow-up was scheduled, so as to check the patient's clinical status. The tube was functional 21 days after PEG placement and then the patient was referred to our hospital because her caregivers noticed melaenic stools. According to her caregivers, feeding through the tube was not difficult and no peritubular leakage had been noticed. On admission, the abdomen was mildly distended with hypoactive bowel sounds and diffuse tenderness. Digital rectal examination was positive for melaena. The PEG tube site was unremarkable but the tube was not freely movable. A palpable subcutaneous mass was thought to correspond with the bumper within the tract.

Laboratory results included leukocytes 21,000/cmm, platelets 223,000/mm³, and international normalized ratio (INR) 1.18.

The patient was taken to the intensive care unit and after administration of cefazolin (1g IV over 1 hour) and erythromycin (500 mg IV over 30 min) upper gastrointestinal endoscopy was performed. The PEG tube site was unremarkable but the tube was not freely movable. A palpable subcutaneous mass was thought to correspond with the bumper within the tract.

Buried bumper syndrome is an unusual complication of PEG placement.

Endoscopic examination of the duodenal bulb, and after administration of cefazolin (1g IV over 1 hour) and erythromycin (500 mg IV over 30 min) upper gastrointestinal endoscopy was performed. The internal bumper was not visible on the gastric wall, but at the presumed site the mucosa was ulcerated with a small crevice in the centre, and there was oozing from the margins of the ulcer (Figure 1). Endoscopic examination of the duodenal bulb, and after administration of cefazolin (1g IV over 1 hour) and erythromycin (500 mg IV over 30 min) upper gastrointestinal endoscopy was performed. The migrated bumper was removed using the technique described recently by Venu et al, and a new tube (26 MIC PEG) was placed. The PEG tube was cut 3 cm above the skin level. A needle from a new PEG kit was advanced through the shortened stump into the stomach, and was grasped with a snare. The thread and the endoscope were brought out through the mouth, and looped onto a new
The buried bumper syndrome can result in serious complications, including gastrointestinal bleeding, perforation of the stomach and peritonitis.

Discussion

The buried bumper syndrome is an uncommon but serious long-term complication of PEG tubes and has been reported to occur in 1.6% of patients. The bumper becomes lodged anywhere between the gastric wall and the skin along the original PEG-tube track. It probably occurs as a result of excessive tension between the internal and external bumpers leading to gastric ulceration at the bumper site. Epithelisation occurs in some patients with covering of the gastrostomy stoma with normal gastric mucosa and this can result in complete closure of the orifice. However, early endoscopic evaluation can reveal a small irregular crevice. Fouch et al suggested that alteration in the physical characteristics of the bumper caused by gastric acid may facilitate pressure necrosis of the gastric wall and subsequent migration of the bumper. The inability to infuse feeding solution through the tube, peritubular leakage and abdominal pain are the most common manifestations of BBS. Some patients may be asymptomatic due to impairment of sensation, especially in patients with head injury as in our case.

In reported cases of BBS, several different PEG systems had been used, suggesting that the PEG tube design per se may not account for BBS. However, the introducer PEG devices use a balloon catheter and therefore carry practically no risk of BBS in contrast to the pull/push-through systems. Yet, this method is not widely used in Europe due to technical difficulties, complications and high costs. In our case we had used an MIC tube, which has several features predisposing to tube migration, including the small inner bumper with its sharp tapered flange and hard plastic composition.

A buried bumper should be removed even if the patient is asymptomatic, because the tube may continue to migrate until it is completely impacted in the abdominal wall and may even cause perforation of the stomach. Endoscopic Ultrasound (EUS) of the gastric wall with a catheter US probe can facilitate the localisation of the bumper. US imaging provides valuable additional information in deciding whether a surgical or endoscopic approach should be attempted to remove the PEG. A variety of techniques have been used to manage this syndrome and remove the buried bumper. We managed to remove the bumper using the technique described recently by Venu et al. There must be good communication between the physician and the caregivers regarding PEG care because a buried bumper can result in very serious complications, such as perforation of the stomach, peritonitis and death, as in our patient. The caregivers should be instructed that during daily cleaning of the external PEG site, the PEG should be pushed in approximately 1 cm and rotated prior to repositioning of the external bumper. The length of the tube protruding beyond the abdominal wall should be examined at regular intervals so that migration can be recognised. Avoiding external tube traction must be emphasised, as avoidance of the placement of gauze pads beneath the external bumper. When peritubular leakage, inability to infuse feeding solution, inability to push in and rotate the tube are noticed or when abdominal pain develops, BBS should be considered and the patient should be referred for emergency endoscopy. Although this syndrome is considered a late complication of PEG placement, in our patient it developed just 21 days after PEG placement.

In conclusion, physicians should be aware of this uncommon but serious complication of PEG placement and of the various approaches of removing the buried bumper. Specific instructions should be given to caregivers for preventing BBS. Manifestations of the syndrome, including gastrointestinal bleeding, perforation of the stomach and peritonitis.
bleeding, must be recognised and the patient referred for emergency endoscopy and removal of the bumper. The syndrome is considered a late complication, but it can also occur early after PEG placement. Failure to recognise this syndrome may result in serious complications including gastrointestinal bleeding, perforation of the stomach, peritonitis and death.

References

Diagnosis: Recurrent Blount’s disease
Infantile Blount’s disease results from disordered growth of the proximal medial physis and metaphysis of the tibia, clinically seen as a progressive varus deformity of the knees with associated internal tibial torsion. It is rare (estimated incidence of 0.05 per 1000 live births). It tends to be bilateral and symmetrical and is more common in boys, early walkers, obese children, and those of West African and Scandinavian origin.

Treatment is based on the age of the patient and the severity of the condition. Early infantile Blount’s disease should be kept under close observation as spontaneous improvement may occur below the age of 4 years. If the deformity progresses, a single osteotomy of the tibia and fibula can be performed for the correction of the deformity.

Recurrent infantile Blount’s disease is a difficult problem to treat. Complex reconstruction with elevation of the medial tibial plateau and lateral epiphysiodesis has been used in recurrent cases. Two-stage reconstruction using an Ilizarov external fixator has been advocated for severe cases. This involves gradual hemiplateau elevation in the first stage to correct the varus deformity followed by epiphysiodesis, limb lengthening and rotational correction in the second stage to address the residual deformities.

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References