Two-Step Approach In The Treatment Of Buried Bumper Syndrome As A Late Complication Of Percutaneous Endoscopic Gastrostomy

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INTRODUCTION
Percutaneous endoscopic gastrostomy (PEG) has become the method of choice for long-term, mostly longer than 4 weeks, enteral nutrition in patients who are unable to take food orally due to various reasons despite having a functioning gastrointestinal tract (1). Buried bumper syndrome, defined as the migration of internal bumper of PEG tube into the gastric or abdominal wall, may be rarely encountered in the long-term follow-up of patients with PEG (2). Excessive traction continuously applied to the tube for an extended period causes this complication with following steps: necrosis develops as a result of pressure in the gastric mucosa where the internal bumper located, the internal bumper starts to migrate from gastric lumen into the gastric mucosa, if and when the traction is continued, it migrates into the abdominal wall, and finally the gastric mucosa is totally covers the previous opening and it results a complete block between the tube and the gastric lumen (2). The most favored solution of this complication is to remove the PEG tube by external traction, usually without the need of abdominal incision, followed by positioning a replacement tube through the same site (3). However, in patients as presented in this report, there may be an abscess in the abdominal wall as a result of delayed diagnosis of this complication due to various reasons and therefore it could be impossible to perform such procedure in the same session.

CASE REPORT
An 84-year-old woman (N.G., # 03-5687) who had a PEG tube was referred to our institution with the complaint of tube non-functioning. The medical records of patient showed that the indication of PEG was the absence of swallowing reflex following a cerebrovascular accident and the presence of aspiration pneumonia during that period. At the initial evaluation, the patient was in-cooperated, hypotensive (arterial blood pressure= 90/50 mm Hg), and tachycardic (heart rate=124/m). There was a loss of turgor and tonus indicating a remarkable fluid deficiency as a result of patient not receiving any fluid or nutritional support for the last 6 days.

On the physical examination, the internal bumper of the tube was measured 0.5 cm away from the skin level which indicated a probability of tube dislocating. There were marked tenderness, edema, erythema, and swelling indicating a cellulitis at the skin around the tube entering. A remarkable fluctuation under this overlying skin was noticed. The results of blood analysis out of normal limits
were as follows: white blood cells=17,200/mm³, potassium=3.1 meq/L, sodium=131 meq/L, chloride=88 meq/L, blood urea nitrogen=56 mg/dL, creatinine=2.3 mg/dL. Ultrasound revealed a subcutaneous abscess cavity superficial to the abdominal wall around the PEG tube which was in contact with the internal bumper (Figure 1).

Figure 1
Figure 1: A. The view of the buried bumper syndrome by ultrasound; note the internal bumper which was superficial to the abdominal muscle wall (black arrows) and the tract between the abscess cavity and the skin (white arrow), B. The buried internal bumper of PEG tube (yellow arrow) within the abscess cavity was easily shown by ultrasound; note the acoustic window which indicates the tube's lumen.

Under local anesthesia, the tube was removed with a skin incision and the abscess was drained followed by debridement of all necrotic tissues (Figure 2).

Figure 2
Figure 2: External view of the wound following removal of PEG tube and drainage of abscess by classic surgical approach under local anesthesia.

Careful probing of the wound revealed that the abdominal wall was completely The wound was left open and daily wound care was recommended to the patient-caring nurse at this rest house. Insertion of a replacement tube trough the same site seemed as a wrong strategy in this case since the presence of local infection may facilitate the development of more serious soft tissue infections such as necrotizing fasciitis. Instead, a broad-spectrum systemic antibiotherapy consisting of a first generation cephalosporin and metronidazole was initiated and the nutritional support was provided by a naso-gastric tube insertion for one-week period. Once the infection regressed, a PEG tube was re-inserted by pull method at another site rather than at the previous site (Figure 3).

Figure 3
Figure 3: Endoscopic view of the internal bumper of re-inserted PEG tube; note the complete mucosal covering of previous tube opening superior to the present gastrostomy site.

The patient has been symptom-free during 2 months of follow-up.

DISCUSSION
Buried bumper syndrome may develop in patients with PEG with reported incidence of 1.5 to 1.9% (⁵,⁶). A certain interval, usually reported around 4 months, is required for complete mucosal healing of the opening after the internal bumper migrate into the gastric or abdominal wall as a result of excess pressure applied to the tube. This interval may be as short as 2 months and as long as 7 years depends on the level of traction-force applied to the tube and the quality of patient-care (¹,⁴).

The most common symptoms of the syndrome are immobilization and clogging of the PEG tube (¹). If mucosal covering of internal bumper is complete, even fluids cannot be given through the tube. Insisting on to use the PEG tube in this circumstance almost always results in an abscess
formation in the superficial or deeper locations to the abdominal muscle wall depends on the burying level of the tube. Presence of any secretion around the tube, any difficulty to administer liquids through the tube, or any pain in the abdominal wall should alert the clinician for the possibility of buried bumper syndrome (1-3). Although a carefully taken patient history and a detailed physical examination in which the migrated internal bumper can be palpated in most cases usually indicate the syndrome, the exact diagnosis of buried bumper syndrome is established by endoscopy. Radiologic examinations such as ultrasound, computerized abdominal tomography, or even magnetic resonance imaging may be helpful for definite diagnosis; however, they are not needed in all cases.

Once the diagnosis is established, the solution is universal: (1) to remove the clogged and buried tube either by simple external traction or with the help of surgery under local anesthesia, (2) to re-insert a PEG tube, preferably a replacement tube, through the same site. We had two patients with buried bumper syndrome in our previously reported series of 115 cases (4). They did not have any abscess in the abdominal wall; therefore, a replacement tube could be inserted successfully in each patient at the same session (4-10). In the reported case, the presence of an abscess under the PEG tube placement-site and worse clinical conditions of the patient at the time of diagnosis forced us to follow the strategy that was to drain the abscess first, then to correct the patient’s status by nutritional support as well as fluid and electrolytes corrections, thereafter to control the infection at the tube site either by a broad-spectrum antibiotherapy and local wound care, and finally to re-insert a PEG tube at a different site. This type of treatment may be defined as two-step approach in the management of buried bumper syndrome since many other surgical methods to solve similar conditions like this situation are named with this terminology.

Regardless of whether the tube re-placement is performed at the same session of the removal of older buried tube or after a certain interval, the development of this bothering complication can be prevented basically by allowing for an additional 1.5 cm between the external bumper of the PEG tube and the skin in order to minimize the risk of pressure necrosis and exchanging it with a replacement tube after 3 to 6 months in patients who need long-term enteral feeding

(1-10).

CONCLUSION

In conclusion, in the treatment of buried bumper syndrome in patients with associated abdominal wall abscesses, we advocate two-step approach consisting of removing the PEG tube and draining the abscess in the first step and re-positioning a PEG tube at a different site following a certain period of antibiotherapy and daily local wound care in the second step to solve the problem. Doubtless, all measures should be taken in order to prevent the development of this complication.

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References

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