Subcutaneous Emphysema Due to Perforation of the Stomach

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INTRODUCTION

Since Abeille (1853) first described subcutaneous emphysema of the face and neck, secondary to gastrointestinal tract perforation, several articles have been published dealing with the location of perforation and with the mechanisms of gas spread, by different authors.

Because of the uncommon occurrence of subcutaneous emphysema following perforation of the gastrointestinal tract, other than the esophagus, the exact cause and mechanism still seems debatable.

Recently we had one case of massive subcutaneous emphysema of the face and neck associated with pneumomediastinum and tension pneumoperitoneum, which developed secondary to perforation of the stomach.

The intention of this report is to stress that patients with perforation of the stomach can occasionally develop subcutaneous emphysema. This information may be helpful to those who treat subcutaneous emphysema cases. For this paper the literature has been reviewed and a new case reported which is the first report from Korea.

CASE SUMMARY

The patient was an 18 year old male and admitted on Jan. 7, 1971, through the emergency room, with marked abdominal distention, vomiting and dyspnea of one day's duration.

His past history indicated that he had had pulmonary tuberculosis and Pott’s disease for more than 10 years. One month ago he felt occasional dizziness, headache and anorexia, and 24 hours prior to admission he had abdominal pain and bouts of vomiting after meals. The patient denied any history of peptic ulcer.

He was slender, acutely ill looking and semistuporous. B. P. 120/100, P. R. 140/min, R. R. 40/min. Subcutaneous emphysema over the face, neck and anterior chest wall was immediately noticed. Heart sounds were slightly remote, but without any murmur or arrhythmia. Breath sounds were markedly diminished over both lower lung fields and Hamman’s sign was positive. The abdomen was markedly distended, especially in the epigastrium, with tenderness and rebound tenderness.

Hb. 17.2 gm. %, Hct. 54%, WBC 17600/mm3, segmented neutrophiles 76%, stab neutrophiles 4%, urinary sugar: negative, urinary protein: one positive, serum Na: 133 mEq/L, K : 4.4m Eq/L, Cl : 96mEq/L, Co2 comb. power : 16m Eq/L. Chest X-ray showed mediastinal emphysema as well as emphysema in the neck and face (Fig. 1). Old pleural scar and a fibrocalcific lesion in the left lung field were found.
An upright abdominal X-ray showed an extensive pneumoperitoneum with a fluid level in abdominal cavity (Fig. II).

The patient was then appropriately prepared for an exploratory operation with the presumptive diagnosis of perforation of infra-diaphragmatic portion of the distal esophagus, along with peritonitis. Vital signs of the patient became suddenly worse, during transfer of the patient to the operating room. On arrival at the operating room, no blood pressure was detected and the patient was really in a dire situation. A large (17G) bore needle was introduced into the abdominal cavity, which released tension pneumoperitoneum and there was some improvement of respiration and vital signs. The patient soon had to be explored under local anesthesia with endotracheal intubation. The patient’s vital status improved soon after opening the peritoneum but required 2500 c.c. of whole blood transfusion. The patient was then given a general anesthesia.

Opening the peritoneum, there was a perforation of the anterior wall of the fundus of the stomach, the size of which was about 2.0 cm. in diameter, and the abdominal cavity was soiled with gastric contents and inflammatory exudate. The lower esophagus was explored, which showed no evidence of perforation or disease.

A partial fundectomy of the anterior wall, excising the area of perforation, was performed and closure was made in double layers. A gastrostomy tube was inserted. After the operation, he did not fully recover from the anesthetic. Respiration was shallow and rapid, and the pulse rate was 150/min.

Therefore, he placed under controlled respiration with a Bennett respirator. Urine output was 20 c.c./hour, and the blood pressure fluctuated. In spite of all the appropriate measures taken vital signs did not improve and, finally, the patient expired 4 hours after the operation in the recovery room. Autopsy was denied. Excised specimen of the stomach disclosed an acute focal necrosis of the gastric wall, with recent hemorrhage, and many bacterial colonies. No evidence of ulcer was found.

DISCUSSION

It is well known that subcutaneous emphys-
ema of the face and neck, along with mediastinal emphysema, usually comes from perforation of the esophagus or from injury to the tracheobronchial tree. It is also known to occur in patients following violent straining as in cases of severe vomiting (Macklin), diabetic patients while vomiting (Beigelman), and complicated labor cases (Brill), without any grossly demonstrable perforation of the esophagus.

The first description of subcutaneous emphysema of the face and neck from bowel perforation (multiple perforation of the cecum) in 1853 by Abeille has been followed by several reports in which subcutaneous emphysema, due to perforation of the gastrointestinal tract, is well documented (Brown 1944, Woodruff 1952, Eadie 1955, Crown 1959).

Newman (1868) is credited with the first report of subcutaneous emphysema following perforation of the stomach, and several reports were published thereafter (Poensgen 1873, Faber 1885, Podlaha 1926, Korach 1927, Vainio 1954, Ebrie 1956).

There have been many reports on the mechanisms of development of subcutaneous emphysema in patients with bowel perforation. Colonic perforations infrequently produce emphysema of the anterior abdominal wall, scrotum, perineum and thigh, while high gastric perforations usually produce supravacuicular emphysema (Oetting et al. 1955). In the cases of colonic perforation, direct escape of gas will produce either pneumoperitoneum or collect in pararectal tissue (Ainsworth 1959), but in the case of high intestinal perforation the paraesophageal ligament and retroperitoneal paravertebral tissue may be the main routes of air extending to the posterior mediastinum, face and neck (Oetting 1955).

Podlaha found in his experiment that hydrogen peroxide injected through the subserosa in dogs and cadavers, diffuse into the mediastinum through the pleuroesophageal ligament and aortic hiatus and concluded that there must be some mechanical condition to do such a diffusion. However Vainio suggests in his literary review that tension pneumoperitoneum spreads apart the loose peritoneal coverings of the diaphragm to allow the gas to reach the mediastinum. McCorkle and Stevenson theorized that gas diffuses directly through the parietal peritoneum into the mediastinum, secondary to tension pneumoperitoneum.

Welty postulated that air passes to the mediastinum, via the perivascular sheath of vessels supplying the region of the perforation, to eventually reach the aortic hiatus.

It is deemed in our case that a mediastinal dissection occurred through the paraesophageal hiatus and periaortic tissue due to extreme tension pneumoperitoneum, secondary to the perforation of the stomach.

Vigyazo talked about the case of a duodenal ulcer perforation with subcutaneous emphysema and postulated that serosa overlying the perforation acted as a valve to keep air out of the peritoneal cavity and forced it, instead, into the subserosal tissue. Stablegren and Tabbit emphasized that subcutaneous emphysema is an important sign of an intraabdominal abscess containing gas forming bacteria, but an infection caused by a gas forming organism and producing subcutaneous gas, is debatable. Anyway, there are many postulations for the route of gas diffusion from the abdomen into the mediastinum and subcutaneous tissue of the face and neck. Particularly in the cases of perforation of the stomach, paraesophageal ligament, aortic hiatus and the parietal peritoneum seem to be the main plausible routes for it.

As to the cause of perforation of the stomach in our case, it can be said that the perforation was not from a gastric ulcer or any...
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The tissue slide of the excised specimen showed only focal necrosis with recent hemorrhage. It is hard to say, in our case, whether the bacterial colonies found in the tissue caused focal necrosis of the stomach wall or developed secondary to the tissue necrosis of the stomach wall.

Several different causes have been entertained as to the mechanism of rupture of the stomach such as visceral ischemia, congenital defect, neurogenic vascular causes or intraluminal pressure. However, rupture probably occurs primarily due to increased intraluminal pressure (Aired 1937). What makes the intraluminal pressure high enough to cause rupture of the stomach?

Maddock et al (1949) described the air sucking mechanism, which probably plays an important role in the pathogenesis, and can be summarized as follows: The normal stomach, in a cadaver, can tolerate volumes of fluid as high as 4000 cc. Normally the cricopharyngeal muscle maintains a tonic contration, which prevents the intake of abnormal quantities of air into the esophagus and stomach, but under various situations such as pain, surgical procedures or anesthesia, the superior esophageal sphincter relaxes. Thus, with increased negative pressure in the esophagus, during inspiration, the air is aspirated directly into the stomach causing dilatation. One thing to be additionally considered is that there is probably a moderate impairment of the passage of air at the level of the pylorus owing to a reflex ileus. If the increase of the intragastric pressure occurs abruptly the patient also loses the capacity to vomit or eructate, as a result of the occlusion of the gastroesophageal junction caused by the increased angulation of the esophagus against the fixed fibers of the right crus of diaphragm.

This produces a one-way valve at the gastroesophageal junction and the intragastric pressure increases with the ultimate rupture of the wall. (Albo). It is hard to find what exactly made the stomach perforate in our case, but at least, it is evident that increased intraluminal pressure of the stomach cavity played an important role.

SUMMARY

A case of subcutaneous emphysema of the face and neck associated with mediastinal emphysema and tension pneumoperitoneum due to perforation of the stomach, is reported. The review of literature is on the history and mechanism of subcutaneous emphysema of gastrointestinal tract origin, and we emphasize the fact that there is a possibility of subcutaneous emphysema, secondary to bowel perforation.

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REFERENCES

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