



Gastric Emphysema and Its Possible Causes: Diagnosis and Management

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Introduction

Gastric Emphysema (GE) is not an isolated entity but is always a sign of an underlying disease [1]. It refers to the presence of air within the wall of the stomach. The presence of air within the G-I tract wall can appear at any site, from esophagus to the rectum [2]. The stomach is the least frequent site of presentation, accounting hardly for 9% of all cases reported [3]. It is not life threatening [4] and can be caused by a variety of iatrogenic and non iatrogenic events [5-9]. Gastric emphysema in the context of gastric pneumatosis, including emphysematous gastritis as well, was described for the first time more than one century ago by Fraenkel. Its pathogenesis and etiology is not yet fully understood and still debated. Evidences from single case reports and series have attempted to explain its pathogenesis focusing mainly on mechanical, bacterial or mucosal damage theory [10-12]. Many of the possible causes described in episodic retrospective cases might overlap and increase the chances of developing GE. We present the case of a 64-year-old woman with main diagnosis of pancreatic neuroendocrine tumor, that went through many clinical complications leading finally to GE.

DISCUSSION:

Gastric pneumatosis (GP) was classified as idiopathic (15% of cases) or secondary (85% of cases), ranging clinically from a mild condition to a fulminant disease. The clinician's challenge with this entity is to define the time we are in front of gastroautopsy cases). Cases have been reported simply accusing the malignancy itself of being a possible cause of EG, although rarely documented. Adding to this evidence, in our case, we also assume that the mucosal damage was caused by cytotoxic agents administered 3 weeks before presentation. There are many reports of conventional chemotherapy and EG with agents such as cyclophosphamide, methotrexate, vincristine, doxorubicin, daunorubicin, doxetacel, cytarabine, 5 -fu, paclitaxel, etoposide , irinotecan, cisplatin and also recently new target drugs like erlotinib, geflon antibodies like bevacizumab [38]. The onset of symptoms is 3 weeks to 3 months after chemotherapy and longer with targeted therapy or antibodies (3 -14 months). In our case, our patient underwent an initial cycle with carboplatin-etoposide 3 weeks before the onset of symptoms. Reported chemotherapy cases are almost always associated with neutropenia, describing neutropenia as an important factor for the development of EG. In our case, the patient had grade 4 neutropenia after 2 weeks of the first cycle of chemotherapy and 1 week before the onset of symptoms of GE. Other extrinsic factors

such as alcohol abuse, long consumption of NSAIDs or extra-gastric processes such as pulmonary GE caused by alveolar rupture and air leakage through the mediastinum and dissecting down to reach stomach wall, chronic obstructive pulmonary disease, polymyositis, recent abdominal surgery, perforated appendicitis, intestinal infarction and ischemia, small intestinal volvulus, following increased use of modern computed tomography, this entity is diagnosed more often, posing a difficult clinical dilemma, as the clinical significance of GP ranges from mild to catastrophic. Gastric emphysema presents a hypodense, linear or curved fringe on the gastric wall with distension, without sign of thickening of the gastric wall, the latter being typical of emphysematous gastritis and helping in the diagnosis of differentiation.

The main explanations for GP are that it originates by disruption of the gastric mucosa leading to air dissecting into the gastric wall. A combination of alterations in mucosal integrity, intraluminal pressure, bacterial flora, and intraluminal gas, each of them or together play an interactive role in GP formation .

Traumatic EG caused by transmural air diffusion after a mucosal injury, such as a biliary stent, has been reported and in our case, ERCP or gastrostomy could have been contributory. The placement of the nasogastric tube was also charged, but in our case it helped relieve the symptoms. Another described mechanism of genesis is obstruction of gastric exit due to peptic ulcer, pyloric stenosis, carcinoma of the antrum or gastric volvulus. In our case, the patient had a direct gastric obstruction due to the neoplastic progression of the head of the pancreas and gastroparesis, causing high intramural pressure and facilitating superficial mucosal tears during vomiting, as was the case in our patient . Vomiting has often been described as a very common cause, but in our case it is not clear whether this is a cause or a response to previously established gastroparesis and GE.

Conclusion:

The presence of air within the mural wall of the stomach may be associated with a wide range of conditions, ranging from benign to fatal, and combination of clinical presentation, imaging, and biochemical parameters should guide diagnosis, focusing mainly on early differential diagnose of GE and emphysematous gastritis. These two entities have different etiopathogenesis and impact, intraabdominal findings and different prognosis. Considering the chronologic precipitation of the G-I complications we should be aware of this complication leading us to a rapid decision on treatment approach and efficient management of these cases.