

## A Case of Gastric Emphysema Accidentally Found By Computed Tomography for the Evaluation of Trauma

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### Article History

Received: 13.12.2017

Accepted: 17.12.2017

Published: 30.12.2017

### DOI:

10.36347/sjmcr.2017.v05i12.012



**Abstract:** The patient was a 55-year-old man who had fallen from a height of 1.5 m when cutting a tree branch. He complained of face, chest, back, right femur and leg pain. He had hypertension and gastritis. He was diagnosed with cerebral concussion, right 8 and 9<sup>th</sup> rib fractures, right hemopneumothorax, liver injury with ascites and pelvic and leg fractures by whole computed tomography (CT). He underwent right thoracotomy, and received oxygen and a fentanyl infusion. On the same day, follow-up CT revealed gastric emphysema (GE), air in the gastric-omental vein and the portal vein of the liver with enlargement of the stomach. As he had no abdominal pain and signs of peritoneal stimulation, conservative therapy was selected. He survived and was discharged. A possible mechanism of GE was that massive amount of air associated with the enlargement of stomach due to paralysis, entered the site of an underlying gastric lesion and resulted in the gastric wall being filled with air. This unique case adds one more cause to the list of documented etiologies of GE.

**Keywords:** Gastric emphysema; pneumothorax; CT.

### INTRODUCTION

The radiographic finding of gastric emphysema (GE) with portal venous gas is classically an ominous finding that is associated with a high rate of mortality [1]. Although this is classically the case, this imaging finding must be quickly correlated with the overall clinical picture, allowing for the essential differentiation between highly lethal emphysematous gastritis (EG) and the much more benign GE, each of which has drastically different management strategies [1]. We herein report a case of GE that was accidentally detected by computed tomography (CT), which was performed for the evaluation of trauma.

### CASE PRESENTATION

The patient was a 55-year-old man who had fallen from a height of 1.5 m while cutting a tree branch. He complained of face, chest, back, right femur and leg pain. He had hypertension and gastritis. On arrival, his Glasgow Coma Scale score was E4V4M6. He had a blood pressure of 88/66 mmHg, a heart rate of 80 beats per minute, an SpO<sub>2</sub> of 100% under oxygen inhalation (10 L per minute by mask), and a body temperature of 36.4°C. The main results of a biochemical analysis of the patient's blood were as follows: white blood cell count, 19,300/ $\mu$ l; hemoglobin, 13.9 g/dl; platelet count, 22.6  $\times$  10<sup>4</sup>/ $\mu$ l; aspartate aminotransferase, 243 IU/L; alanine aminotransferase, 174 IU/L; blood urea nitrogen, 23.8 mg/dl; creatinine, 0.95 mg/dl; creatinine phosphokinase, 992 IU/l; c-reactive protein, 0.3 mg/dl, activated partial thromboplastin time, 27.0 (27.0) seconds; international normalized ratio of prothrombin time, 1.16, and fibrin degradation products, 387 $\mu$ g/m L. He was diagnosed with cerebral concussion, right 8 and 9<sup>th</sup> rib fractures,

right hemopneumothorax, liver injury with ascites, pelvic and leg fractures based on the whole-body CT findings. Right thoracotomy was performed, and he received oxygen, and an infusion of fentanyl, and was admitted to the intensive care unit. On the same day, follow-up CT for the evaluation of thoracic and abdominal traumatic lesions showed GE, air in the gastric-omental vein and portal vein in the liver with enlargement of the stomach (Figure 1). As he had no abdominal pain or sign of peritoneal stimulation, conservative therapy with an indwelling nasogastric tube for decompression was selected. Follow-up CT on the 2<sup>nd</sup> hospital day showed the disappearance all signs of the GE, and air in the gastric-omental and portal veins. He had also passed gas. He started to drink liquids first, followed by the per os intake of food without any remarkable events. Gastroscopy revealed superficial gastritis at the fornix and diffuse atrophic gastritis. He was discharged on foot on the 20<sup>th</sup> hospital day.

CT shows gastric emphysema (triangle), air in gastric-omental vein (white arrow) and portal vein in

the liver (black arrow) with enlargement of the stomach

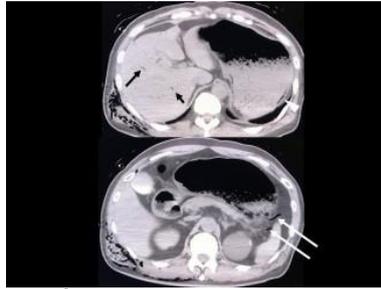


Fig-1: The 2<sup>nd</sup> truncal computed tomography scan

## DISCUSSIONS

Matsushima *et al.* conducted a comprehensive literature review of the adult cases of EG and GE reported in MEDLINE [2]. A total of 75 cases were included in their review. There were no differences between the two groups with regard to sex, age, or the most frequent complaint (abdominal pain). In contrast the GE group included greater numbers of patients with diabetes, fever, ischemic findings on gastroscopy, surgical removal of the stomach, and a higher rate of mortality in comparison to EG. The frequency of vomiting and malignancy in the GE group were significantly greater than those in the EG group. According to their theory, secondary to mechanical injury of the stomach mucosa as a result of direct injury to the mucosa or mucosal tear due to increased intraluminal pressure within the stomach, in addition to gastric outlet obstruction, is most likely main mechanism underlying the formation of the GE. In addition, previous studies have reported various mechanisms underlying the formation of GE, including a mechanical hypothesis (due to the presence of air, which is increased by mural pressure due to factors such as vomiting), a mucosal damage hypothesis (the mucosa is disrupted by factors such as an indwelling nasogastric tube or gastroscopy), and the pulmonary disease hypothesis (alveolar air dissecting down the mediastinum in cases involving severe asthma or chronic obstructive pulmonary disease) [3-7]. In the present case, there was no record of vomiting, indwelling nasogastric tube, gastroscopy and pneumomediastinum was not observed on the second CT scan. Instead, the existence of a past history of gastritis and gastric enlargement maybe due to the transient paralysis induced by traumatic impact or the use of fentanyl might have led to the formation of GE. In other words, a massive amount of air in the enlarged stomach (due to paralysis) entered the site of an underlying gastric lesion and resulted in the gastric wall being filled with air. This is the first report of this mechanism being associated with the formation of GE and this unique case adds one more cause to the list of documented etiologies of GE.

## Conflict of interest

The authors declare no conflicts of interest in association with this study.

## ACKNOWLEDGEMENTS

This manuscript received financial support from the Ministry of Education, Culture, Sports, Science and Technology (MEXT)-Supported Program for the Strategic Research Foundation at Private Universities, 2015-2019, concerning (The constitution of a total research system for comprehensive disaster and medical management, corresponding to a wide-scale disaster).

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