A 63-year-old man was admitted to our emergency department because of abdominal pain with distension and ischemia of both lower limbs for the past 2 hours. Previously, he had vomited extremely following alcoholic excess and an opulent meal.

In his previous history, distal esophageal stenosis was obvious after surgical therapy of Boerhaave syndrome 3.5 years earlier with primary suturing of the distal esophageal perforation and anterior semifundoplication.

At clinical examination, the patient presented with stable cardiopulmonary function. The abdomen was massively distended and tender, and both legs were blue, revealing signs of prolonged ischemia with absent palpable pulses of the femoral artery in both groins. Palsy of the legs was not yet apparent.

Computed tomography of the chest and abdomen showed huge amounts of free intra-abdominal air and fluid, suggesting perforation (Figure 1A). Consecutively, there was an external abdominal aortic compression with lumen collapse (Figure 1B), and the iliac arteries, as well, were compressed down to the superficial femoral artery as a consequence of severe abdominal compartment syndrome.

Because the patient got progressively unstable during computed tomography examination and shocky, we inserted a trocar (intravenous needle) into the abdominal wall. The abdominal free air could be evacuated immediately (Figure 2A). Computed tomography documented reversal of the aortic compression (Figure 2B), and the lower extremities were reperfused. Clinically, the groin pulses were palpable.

At laparotomy, perforation (8 cm in diameter) of the lesser gastric curvature was found, combined with fibrinous peritonitis of all 4 abdominal quadrants. Moreover, the stomach was rather burst and revealed an abnormal dilatation. This

Figure 1. A, Computed tomography showed huge amounts of free intra-abdominal air and fluid, suggesting a perforation. B, Computed tomography showed external abdominal aortic and iliac arteries compression with lumen collapse.
dilatation with perforation might have been due to previous vomiting against the esophageal stenosis. The rupture of the lesser curvature of the stomach was sutured by linear stapler.

During 2 further relaparotomies (days 1 and 2), resection of gangrenous colon and small bowel was performed. The patient died on day 2 of sepsis with multiorgan failure.

To our knowledge, this is the first report in the literature of massive pneumoperitoneum followed by gastric bursting, causing acute aortic compression syndrome released by immediate abdominal trocar insertion. Although abdominal compartment syndrome is not uncommon in visceral surgery, and can be easily detected by intravesical pressure measurement via a special urinary catheter (pathological values, >12 mm Hg), significant aortic compression is a rare event. Microvascular disturbance without major clinical long-term consequences is often suspected in the case of temporary intra-abdominal compartment syndrome, but profound insights into its pathophysiology are lacking.

Krol and Hallett described a case with transient, marked flattening of the abdominal aorta, iliac arteries, and venous structures during active vomiting while a diagnostic computed tomography examination was being performed. Because the pressure leading to major arterial compression has to exceed at least the diastolic arterial blood pressure, the authors stated that it can possibly peak as high as 290 mm Hg during vomiting episodes. This pressure might have even been elevated in our patient after vomiting and gastric rupture, possibly triggered by a distal esophageal stenosis after surgical therapy of Boerhave syndrome. As concluded by Krol and Hallett, this pressure peak—although transient—might be much higher than abdominal masses tend to reach. In our case, abdominal compartment led to prolonged aortic compression with consecutive irreversible and fatal mesenteric ischemia.

**Disclosures**

None.

**Reference**

Bursting Acute Release by Trocar Insertion

Markus Paschold, Ines Gockel, Katja Oberholzer, Hauke Lang and Christoph Düber

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