Tension Pneumoperitoneum: A Rare Complication of Upper Gastrointestinal Endoscopy

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ABSTRACT

Introduction: Tension pneumoperitoneum is defined as the massive accumulation of air in the peritoneal cavity, which results in a sudden increase in intraabdominal pressure. Various iatrogenic procedures are responsible for this complication. We herein report a case of tension pneumoperitoneum resulting from upper gastrointestinal endoscopy.

Case Description: A 45-y-old man was referred to our department because of melena. Esophagogastroduodenoscopy was unsuccessful because of failure to inflate the stomach, and sudden abdominal distension was noted immediately after the procedure. The hemodynamic status of the patient was compromised, and the imminent collapse was prevented with emergent needle paracentesis. Decompression of the abdominal cavity restored vital signs to normal allowing definitive surgical treatment.

Discussion: Upper gastrointestinal endoscopy is a rare cause of this life-threatening complication. The combination of acute abdominal distension with hemodynamic instability following endoscopic procedures should raise suspicion of tension pneumoperitoneum. Needle decompression is life-saving and should be performed instantly upon recognition of this condition.

Key Words: Tension pneumoperitoneum, Upper gastrointestinal endoscopy, Needle decompression.

INTRODUCTION

The presence of free intraperitoneal gas, known as pneumoperitoneum, usually indicates an underlying disease process such as hollow viscus perforation but does not pose by itself a threat to the patient. On the contrary, the massive accumulation of intraabdominal air under pressure known as tension pneumoperitoneum (TPP) is potentially life threatening, because it can lead to the development of acute abdominal compartment syndrome. Various causes of TPP include positive-pressure ventilation, gastric rupture due to improper cardiopulmonary resuscitation, gastric or duodenal ulcer perforation, and endoscopy.1 Upper gastrointestinal (GI) endoscopy is an extremely rare cause of TPP with <10 cases reported in international literature.1–8

CASE REPORT

A 45-y-old man was transferred from a regional hospital to the gastroenterology department for melena and low hematocrit. On admission, the patient was alert with normal vital signs, and his abdomen was soft with no signs of peritoneal irritation. Erect X-rays showed no free intrabdominal air, and white blood cell count was only mildly elevated (11.000/mm³, 78% neutrophils). He had a history of chronic alcohol abuse and was on nonsteroidal anti-inflammatory drugs for the past 15 d due to a fractured left humerus. Upper GI endoscopy was attempted without success, because of poor visibility due to failure to adequately inflate the stomach. After the procedure, the patient’s abdomen became overly distended, and he was rushed to the radiology department for an abdominal computed tomography scan, which revealed massive accumulation of free intraperitoneal air and fluid (Figure 1). The patient’s condition deteriorated, as he became confused and pale with rapid weak pulse and hypotension. His abdomen became barrel-shaped, tender, and tympanic to percussion. Despite rapid infusion of intravenous fluids, after a few minutes, shock was established with undetectable pulsation and blood pressure. To prevent the imminent circulatory arrest, immediate needle decompression of the peritoneal cavity was decided. A 14-gauge needle was inserted subumbicularly at the midline of the abdomen, and a sudden gush of air was noted. Blood
pressure returned to normal, and peripheral pulse became palpable as abdominal distension subsided. Emergency laparotomy revealed a large perforated ulcer (3cm) on the anterior wall of the duodenal bulb, a bleeding ulcer on the posterior wall of the duodenal bulb, cirrhosis of the liver, and findings of portal hypertension. Ligation of the bleeding vessel, antrectomy, gastrojejunal Billroth II anastomosis, and truncal vagotomy were performed following meticulous irrigation of the peritoneal cavity. The patient recovered without major postoperative events and was discharged 15 d later.

DISCUSSION

The increased intraabdominal pressure caused by tension pneumoperitoneum exerts its deleterious effects mainly on the pulmonary and cardiovascular systems.2 Massive air accumulation causes elevation of the diaphragm, which restricts lung volumes, decreases venous return, decreases cardiac output, and may even lead to cardiopulmonary arrest depending on the magnitude of the intraabdominal pressure.9 In the case of TPP after upper GI endoscopy, the air leak can be attributed to either direct iatrogenic injury of the gastric wall during submucosal dissection of a gastric lesion7,8 or to the pneumatic perforation of a gastric or duodenal ulcer. In our case, shortly after insufflation of air into the stomach, the intraluminal pressure exceeded the duodenal wall tension limit on the already fragile ulcer bed causing it to burst. Sealed perforation could not have taken place, because in that case, abdominal tenderness would probably have been found on admission, free subphrenic air would have been found, and white blood cell count would have been much more elevated.

Timely diagnosis is essential in order to achieve a favorable outcome. Similarly to tension pneumothorax, diagnosis must be based mainly on clinical findings; radiographic studies should be considered only in uncertain cases. TPP should be suspected when gastrointestinal endoscopy is followed by sudden distention of the abdomen, which becomes barrel-shaped, although this finding can be overlooked in obese patients. Percussion on all areas of the abdomen reveals tympanicity, and bowel sounds are usually present but distant. Abdominal tenderness can also be found when inflammation of peritoneal cavity is present.10 Dyspnea, signs of venous congestion (edema, skin mottling), or even acute aortic occlusion (loss of pulses and sensation) on the lower extremities can be noted during physical examination.11 If TPP progresses, it leads to hypotension, tachycardia, and even cardiorespiratory arrest.12 If diagnosis cannot be established with history and physical examination, erect or decubitus abdominal films can be helpful. Erect films show upper displacement of the diaphragm and medial displacement of the liver (saddlebag sign),13 and air juxtaposed in the interfaces of the viscera makes the outline of structures like gallbladder and spleen more distinct.14 Decubitus films are performed in cases for which erect films cannot be performed and demonstrate a large volume of intraabdominal air that shifts to the nondependent side.14 Urgent abdominal computed tomography, provided that it can be undertaken without undue loss of time, is helpful in that it demonstrates massive pneumoperitoneum with bowel loop compression and centralization of the abdominal organs.5

Once TPP is recognized, urgent abdominal paracentesis should be performed to release the pressure on the vena cava and aorta, and improve blood circulation and respiratory function. A large-bore venous catheter should be percutaneously inserted in the midline superiorly or inferiorly to the umbilicus. Restoration of hemodynamic instability following decompression of the abdominal cavity prevents circulatory arrest and allows definitive treatment. To prevent pneumatic perforation during upper GI endoscopy, patients should be well prepared and fasted prior to the examination. Endoscopists should be very cautious with air insufflation, especially when dealing with a poorly prepared patient, and should also be alert to identify the complication of TPP.1
CONCLUSION

A high level of suspicion is required for the early recognition of TPP, a rare, life-threatening but potentially reversible complication of the upper GI endoscopy. Surgeons and endoscopists should be alarmed when failure to inflate the stomach during endoscopy is followed by sudden abdominal distension and circulatory decompensation. Once diagnosis of TPP is established, immediate needle decompression is imperative in order to prevent circulatory arrest.

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