

Abdominal Compartment Syndrome and Percutaneous Needle Decompression in a Woman with Corrosive Injury

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Abstract

Abdominal Compartment Syndrome (ACS) is defined by organ dysfunction caused by intra-abdominal hypertension. Increase in intra-abdominal pressure causes multiple adverse physiologic events and is uniformly fatal if left untreated. We report the case of a 76-year-old woman of pneumoperitoneum due to hydrochloric acid-induced corrosive injury, leading to ACS with pulseless electrical activity. Emergency needle decompression was done and successfully restored circulation. It reminds possible ACS in patients of pneumoperitoneum and use of percutaneous needle decompression before operation in emergent condition.

Keywords: Abdominal compartment syndrome; Pneumoperitoneum; Needle decompression

Introduction

Abdominal Compartment Syndrome (ACS) is defined by organ dysfunction caused by intra-abdominal hypertension [1-3]. Better understanding of the relationship between intra-abdominal hypertension and ACS has been achieved since the recent establishment of a method for accurately measuring intra-abdominal pressure. Increase in intra-abdominal pressure causes multiple adverse physiologic events. ACS is uniformly fatal if left untreated, and the mortality rate is high even in treated patients. ACS caused by pneumoperitoneum from corrosive injury is rare because of early recognition. Here, we report a case of corrosive injury with ACS.

Case Presentation

A 76-year-old obese woman presented to our emergency department with hydrochloric acid-induced corrosive injury due to a suicide attempt with detergent intake 2.5 hours ago. On arrival, the patient was alert and had the following vital signs: heart rate, 59 beats per minute; respiratory rate, 18 breaths per minute; body temperature, 36.8°C; blood pressure, 159/91 mmHg. She had epigastric pain without nausea, vomiting, dyspnea, or chest pain. Physical examination showed epigastric tenderness without rebound pain or muscle guarding. Her laboratory results were as follows: white blood cell count, 17,200/mm³; hemoglobin level, 15.6 g/dL; platelet count, 207,000/mm³; creatinine level, 0.81 mg/dL; sodium level, 135 mEq/L; potassium level, 4.2 mEq/L; arterial blood gas: pH, 7.219; PaO₂, 115.2 mmHg; PaCO₂, 26.4 mmHg; HCO₃⁻, 10.5 mm/L; and oxygen saturation, 97.5%. No subphrenic free air was noted on initial standing chest radiography. A proton-pump inhibitor was used, and esophagogastroduodenoscopy was arranged. A sudden onset of severe abdominal pain and abdominal distention were noted and shortness of breath was complained. Repeated physical examination showed diffuse abdominal tenderness and muscle guarding. The patient presented with shallow and rapid respiration with desaturation (SpO₂ 88%) and drowsy consciousness soon. Emergent rapid sequence intubation was performed. Her blood pressure dropped after intubation, so a fluid supplement and inotropic agent were administered and abdominal Computed Tomography (CT) scan was soon performed. The CT images showed pneumoperitoneum with a large amount of free air and a decreased caliber of vessels including the abdominal aorta and Inferior Vena Cava (IVC) (Figure 1). Sudden Pulseless Electrical Activity (PEA) occurred after the CT scan. Cardio-Pulmonary Cerebral Resuscitation (CPCR) was performed, and the general surgeon was consulted immediately. ACS was highly suspected because of severe pneumo-peritoneum with



Figure 1: A computed tomography image shows a large amount of free air in the abdomen. The inferior vena cava (arrow) almost collapsed and abdominal aorta (arrowhead) were compressed.

compression of the IVC and abdominal aorta, but nasogastric tube was failed to be inserted; therefore, needle decompression was performed using large caliber needles. Two needles for central venous catheter use were punctured and inserted into abdominal cavity from the location below umbilicus. Spontaneous circulation was restored immediately after needle decompression. The general surgeon arrived soon afterwards and suggested emergency operation. However, the patient's family refused operation or further aggressive treatment including resuscitation because of poor prognosis. Septic shock and multi-organ failure occurred, and the patient finally died 3 days later at hospital.

Discussion

ACS can be caused by severe abdominal trauma, ruptured abdominal

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aortic aneurysm, retroperitoneal hemorrhage, pneumoperitoneum, massive ascites, pancreatitis, neoplasm, or abdominal wall burn eschar [3]. ACS caused by hollow organ perforation is rare because of early diagnosis and rapid management [4]. It is usually occurred in bowel perforation during gastrointestinal endoscopy with extravasation of insufflating gas. No previous literature has analyzed risk factors of ACS in patients of pneumoperitoneum. From our case, pre-oxygenation during intubation procedure may relate to ACS because ACS occurred soon after intubation. Besides, severity of gastrointestinal perforation may be another factor contributing to ACS but it cannot be proved in our case since endoscopy was not performed.

ACS affects the pulmonary, cardiovascular, renal, gastrointestinal, and central nervous systems [2,3]. In this case, in which ACS was caused by severe pneumoperitoneum, rapid development of PEA was mainly caused by cardiovascular dysfunction. Elevated intra-abdominal pressure induces cardiac output reduction, which is caused by decreased venous return due to IVC compression [5]. Besides, increased thoracic pressure also decreases superior vena cava flow and causes cardiac compression [3]. Previous literature has mentioned percutaneous needle decompression may improve patients' condition, bridging the time to operation [6]. There were only a few cases before and no regular indication for needle decompression was known. Indication

of percutaneous needle decompression was PEA, probably due to ACS in our case according to clinical judgement. Needle decompression of pneumoperitoneum was soon performed during resuscitation, and spontaneous circulation was restored immediately. We present this case to remind possible ACS in patients of pneumoperitoneum and the use of emergency needle decompression in unstable patients of ACS.

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