Cardiac Arrest Due to Tension Pneumoperitoneum Caused by Esophagogastric Perforation and Pyloric Stenosis : A Case Report

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Tension pneumothorax is one of the causes of sudden cardiac arrest with evidence of obstructive shock and subcutaneous emphysema. Emergency chest decompression is a treatment of choice in such a situation. Herein, we report a case of an out-of-hospital cardiac arrest due to tension pneumoperitoneum caused by esophagogastric perforation. A 40-year-old man with a history of duodenal ulcer and pyloric stenosis complained of sudden-onset abdominal pain and developed cardiac arrest during transportation to our hospital. He had jugular venous distention ; subcutaneous emphysema in the upper body trunk, arms, and neck ; and a markedly distended abdomen. Immediate needle-chest decompression was not effective, but after volume resuscitation, adrenaline administration, and abdominal decompression by nasogastric tube, spontaneous circulation was resumed. Radiological findings revealed tension pneumoperitoneum due to esophagogastric perforation. Emergency laparotomy was performed, and the perforation of the esophagogastric junction was detected. The patient was discharged from the hospital without any disability. Notably, in the treatment of a patient with cardiac arrest having subcutaneous emphysema, the cause of obstructive shock could exist not only in the chest, but also in the abdomen. *Shinshu Med J 67 : 113—119, 2019*

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Key words : cardiac arrest, esophagogastric perforation, tension pneumoperitoneum

I Introduction

Tension pneumothorax is one of the causes of sudden cardiac arrest with evidence of obstructive shock and subcutaneous emphysema. Emergency chest decompression is a treatment of choice in such a situation.

Herein, we report a patient with esophagogastric perforation due to pyloric stenosis and who had obstructive shock, subcutaneous emphysema, and tension pneumoperitoneum. The patient developed cardiac arrest during transfer to the emergency department. Nasogastric tube insertion, but not chest decompression, successfully improved his circulation.

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II Case Report

A 40-year-old man in shock was transferred to the emergency department of our hospital. He had a history of duodenal ulcer and *Helicobacter pylori* infection. Endoscopic examination more than 1 year ago revealed stenosis of the duodenal bulb from the stomach pylorus and stomach distension. He had been taking proton-pump inhibitors orally for the previous 5 months. On the day of admission, he suddenly complained of abdominal pain and collapsed at home. At the time of contact with emergency medical services, he was found to have Japan coma scale of 20, heart rate of 120 beats/min, respiratory rate of 60 breaths/min, and 85 % oxygen saturation of arterial blood measured by pulse oximeter at room air. His blood pressure could not be measured, but the

Table 1 Laboratory data upon admission

ALB	5.4	g/dl	CK	164	U/l	WBC	6.23	$\times 10^3$/ul	venous blood gas		
UN	15.1	mg/dl	AMY	123	U/l	RBC	555	$\times 10^4$/ul	pH	7.019	
Cre	1.23	mg/dl	Na	156	mEq/l	Hb	17.3	g/dl	pCO2	120	mmHg
AST	44	U/l	K	4.2	mEq/l	HCT	54.9	%	pO2	8.7	mmHg
ALT	12	U/l	Cl	104	mEq/l	PLT	37.7	$\times 10^4$/ul	HCO3	29.6	mmol/l
γGT	27	U/l	Na/Cl	1.5		PT	12.6	sec	BE	−7.9	mmol/l
T-bil	1.01	mg/dl	CRP	0.32	mg/dl	APTT	19.8	sec	SAT	5.6	%
ALP	257	U/l	eGFRcre	54	ml/min/1.73m^2	PT-INR	1.03		G-Lac	113	mg/dl
LDH	299	U/l	Procalcitonin	0.17	ng/ml	D-dimer	7.5	μg/ml			

ALB : albumin, UN : Urea nitrogen, Cre : creatinine, AST : aspartate aminotransferase, ALT : alanine aminotransferase
γGT : gamma glutamyl transpeptitase, T-bil : Total bilirubin, ALP : alkaline phosphatase, LD : lactate dehydrogenase, CK : creatinine kinase,
AMY : amylase, Na : serum sodium, K serum potassium, Cl : serum chloride, eGFR : estimated glomerular filtration rate, CRP : c-reactive protein
WBC : white blood cell, RBC : red blood cell, Hb : hemoglobin, Hct : hematocrit, PLT : platelet
PT : Prothrombin time, APTT : activated partial thromboplastin time
BE : Base excess, SAT : saturation, G-Lac : Lactate in blood gas, P/F : pO2/FiO2

radial pulse was palpable. His abdomen was distended. During transportation to our hospital, ventilation was assisted with a bag valve mask. However, the patient developed cardiac arrest.

On arrival at the hospital, electrocardiography showed pulseless electrical activity. Physical examination showed jugular venous distention, subcutaneous emphysema in the upper body trunk, arms, and neck, and markedly distended abdomen. Immediate decompression of the bilateral chest with needle was tried considering the possibility of obstructive shock due to tension pneumothorax. However, there was no rush of air, and the patient did not recover after the procedure. Tracheal intubation, volume resuscitation, and intravenous administration of adrenaline were simultaneously performed. Spontaneous circulation was resumed 10 min after arrival at the emergency room. However, his blood pressure was still low. Tympanic sound was heard by percussion of the abdomen. When the 14 Fr nasogastric tube was inserted after return of spontaneous circulation, massive air and approximately 3 l of stomach content were aspirated. Subsequently, the tightness of the abdominal wall was improved, and his blood pressure increased.

Immediate blood examination after arrival at the hospital showed marked elevation of the partial pressure of carbon dioxide and lactic acid (**Table 1**).

Chest X-ray images at the time of admission showed subcutaneous emphysema on both sides of the chest. X-ray image of the abdomen showed free air and dilatation of the intestinal tract (**Fig. 1**). Chest computed tomography (CT) scan showed subcutaneous and mediastinal emphysema (**Fig. 2**). Abdominal CT scan showed wall irregularities, wall thickening and stenosis of the pylorus, marked dilatation of the stomach, and dilatation of the small intestine (**Fig. 3**).

Emergency laparotomy was performed for gastrointestinal perforation. The operation demonstrated perforation of the abdominal esophagus on the small curvature side (**Fig. 4**) and necrosis due to strangulation at two sites in the small intestine. The leakage of a large amount of stomach contents like coffee residue was observed from the perforation site to the peritoneal cavity. Proximal gastrectomy and reconstruction with double tract using the jejunum were performed. In addition, the ileum was partially excised in two places and the stump end, and end-to-end anastomosis was performed.

Meropenem and noradrenaline were administered,

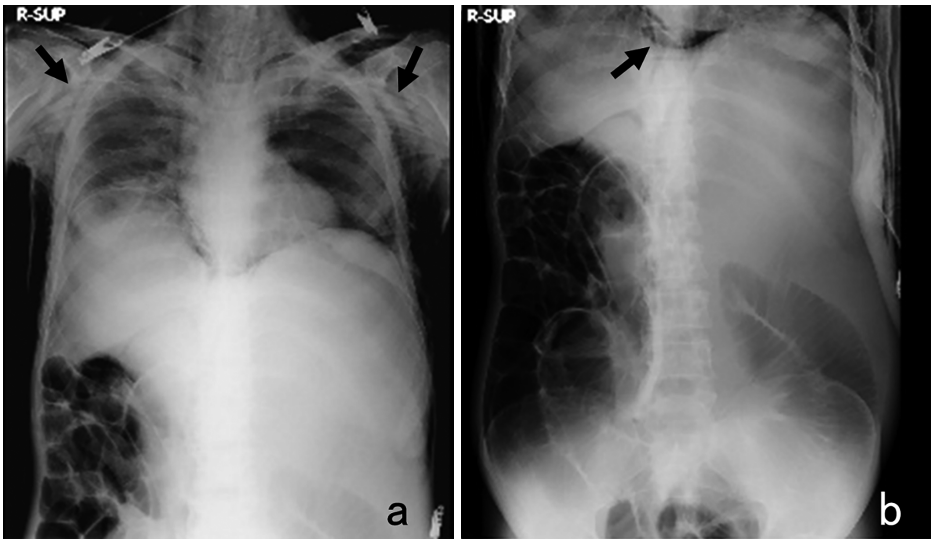

Fig. 1　Chest and abdominal X-ray images on admission (supine position)
a : Chest radiograph showing subcutaneous emphysema on both sides (arrows).
b : Abdominal X-ray image before placing the nasogastric tube showing free air under the diaphragm (arrow) and dilation of the intestinal tract.

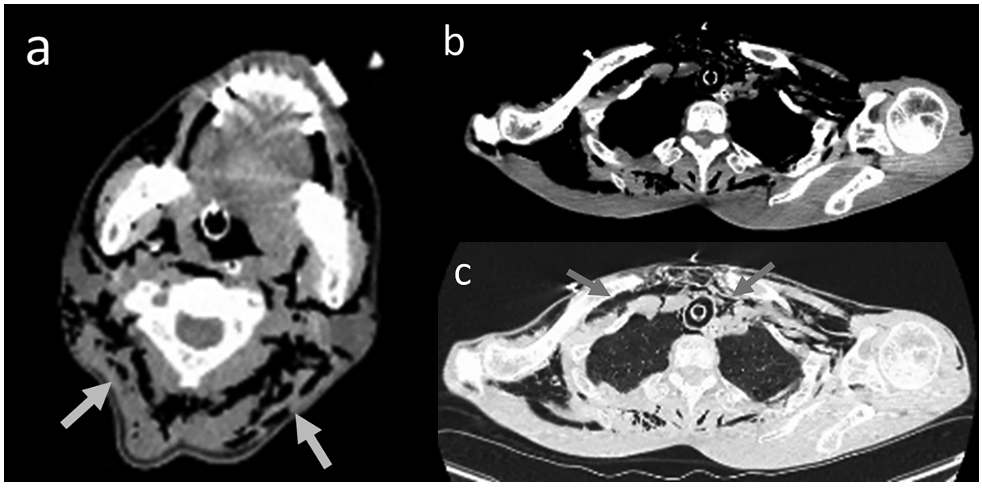

Fig. 2　Computed tomography (CT) images from neck to chest after emergency transport
a : Prominent subcutaneous emphysema in the posterior cervical region (arrows).
b, c : Chest CT images showing subcutaneous emphysema from the bilateral anterior thorax to the lateral thorax around the trachea (arrows).

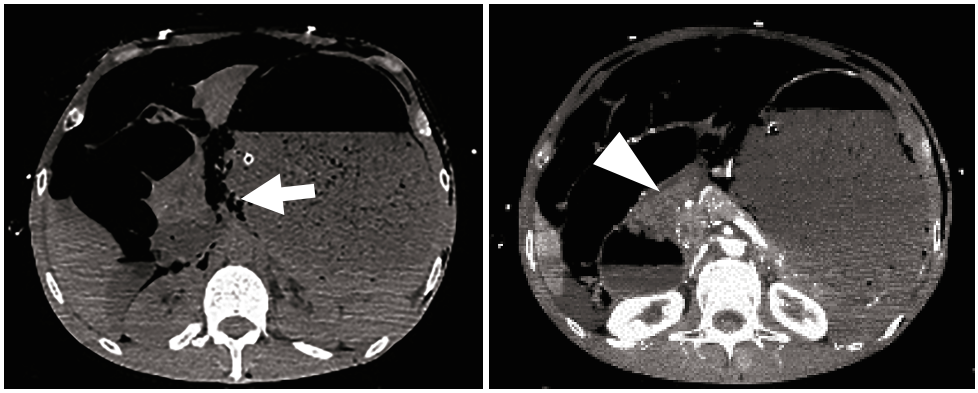

Fig. 3　Abdominal computed tomography confirmed wall irregularity (arrow) on the dilated stomach's minor curvature, pyloric thickening (arrowhead), and dilated small intestine.

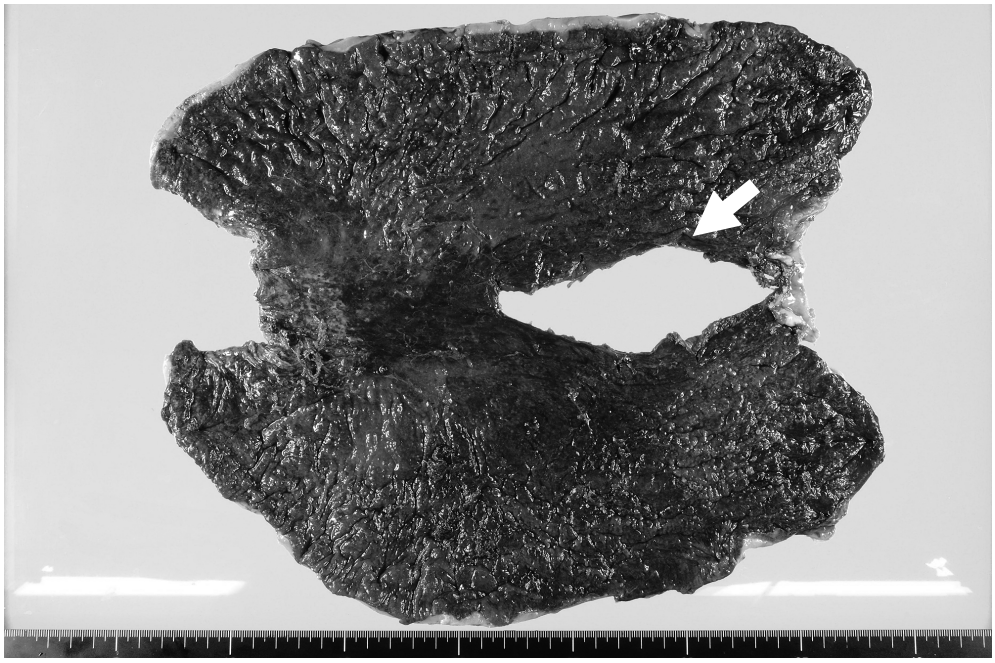

Fig. 4　Macroscopic finding of the resected specimen. The large perforation of the abdominal esophagus on the small curvature side was detected (arrow).

and endotoxin adsorption therapy was done for septic shock after the operation. Recombinant thrombomodulin was administered for disseminated intravascular coagulation. The trachea was extubated on the day after surgery. Noradrenaline administration was completed on the second day after surgery. The patient could drink water about a week after surgery. He had ileus after starting oral ingestion, but recovered with fasting. Pathological examination showed the black color change which is thought to be necrosis in the full-thickness layer of the stomach wall, but did not show any malignant lesion and severe inflammation around the perforation area. He was discharged on the 35th day after surgery, and proton-pump inhibitor has been prescribed (**Fig. 5**). He has had no recurrence and has returned to daily life.

Ⅲ　Discussion

This is a rare case of an out-of-hospital cardiac arrest due to tension pneumoperitoneum. Several factors should be considered in the presented case, such as predisposing factors of esophagogastric perforation, the cause of tension pneumoperitoneum, the cause of the cardiac arrest, and the decision-making in the emergency department.

First, as a predisposition to esophagogastric perforation, there was severe stenosis of the pyloric part of the stomach due to chronic and recurrent duodenal ulcer. As a result, chronic increase in luminal pressure and dilatation of the stomach had occurred[1)2)]. Previous studies reported that a portion of the stomach wall became ischemic due to chronic gastric distension or became perforated due to partial necrosis[3)4)]. For this reason, the esophagogastric junction was possibly easily perforated by a stronger stimulus, as a continuous pressure load was applied to the stomach wall in our patient. Moreover, gastrointestinal perforation is triggered by alcohol, overeating, vomiting, convulsions, and labored breathing[2)]. Esophageal perforation that spreads into the thoracic cavity is called Boerhaave syndrome[5)]. Occasionally, the perforation spreads into both the thoracic and abdominal cavities[3)4)6)7)]. In a previous report, preventive gastrojejunostomy was performed on duodenal stenosis accompanied by high gastric dilatation to prevent rupture[1)].

The second point is the cause of tension pneumoperitoneum. Generally, free air found in the gastric or duodenal perforation is often small. However, in our patient, as a result of pyloric stenosis, there was

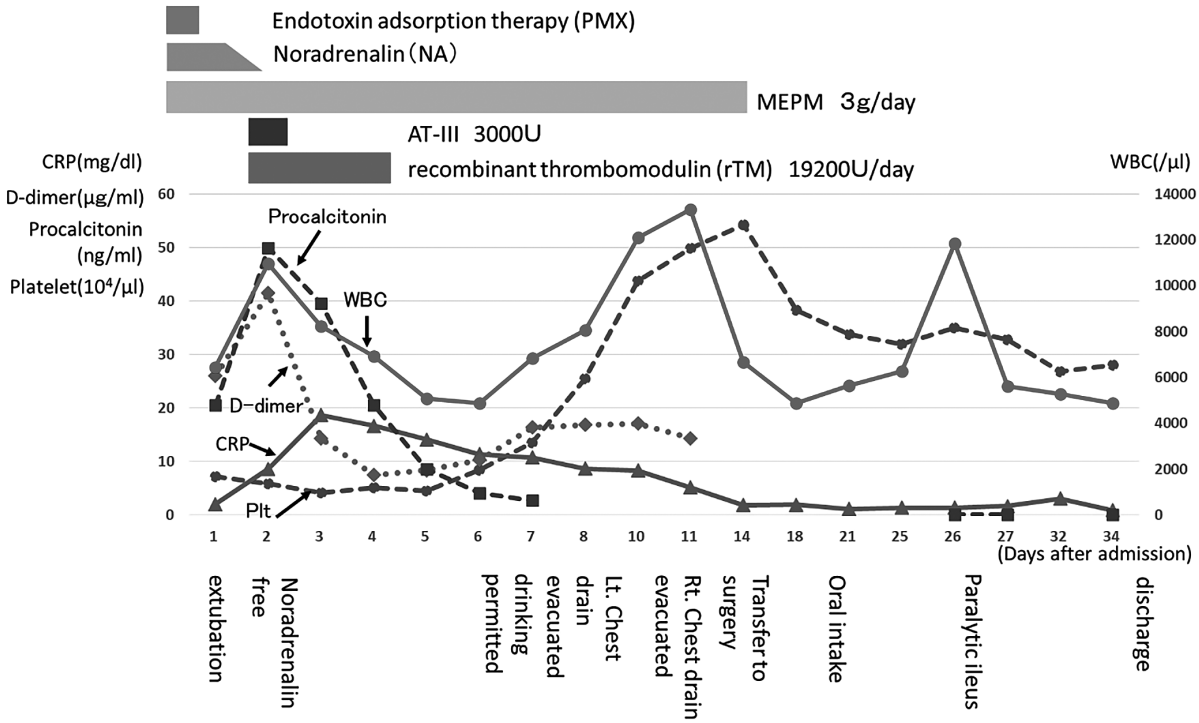

Fig. 5 Hospital course
CRP, C-reactive protein ; Plt, platelet ; WBC, white blood cell

a chronic increase in the luminal pressure and dilatation of the stomach. It was thought that the pressure in the abdominal cavity suddenly increased because a large amount of stomach contents and air entered into the abdominal cavity at high pressure just after the esophagogastric perforation. The increase in intraperitoneal pressure likely caused the decrease in the venous return from the inferior vena cava, resulting in an obstructive shock[7)-9)]. In the reported cases of tension pneumoperitoneum, perforation of the hollow organ has been the most frequent cause at about 80 % to 90 % [10)]. Moreover, tension pneumoperitoneum was reported as a complication of medical procedures such as endoscopic examination[8)11)-14)], laparoscopic mass feeding[15)], and inappropriate cardiopulmonary resuscitation[7)16)-18)].

In some cases, patients with tension pneumoperitoneum develop cardiac arrest. When the cardiac output severely decreases due to the decrease in venous return and increase in afterload by severe pneumoperitoneum, cardiac arrest can occur. In the present case, it appears that a large amount of air entered into the peritoneal cavity and caused further increase in intraperitoneal pressure because of the assisted ventilation with a bag valve mask. Thus, the pre-hospital-assisted ventilation exacerbated the shock state in this case.

This patient had jugular venous dilatation and subcutaneous emphysema in the neck, upper arm, and anterior chest just after arrival at the hospital. Thus, tension pneumothorax was suspected. Needle decompression of the chest was tried, but it was not effective. Spontaneous circulation was achieved after volume resuscitation and intravenous administration of adrenaline. Ideally, the most effective treatment for obstructive shock in this situation might be percutaneous needle decompression of the abdominal cavity[12)15)]. Puncture from the front side of the abdominal wall is not difficult because internal organs are pushed to the dorsal side in the abdominal cavity[11)14)]. Furthermore, there have been case reports in which tension pneumoperitoneum was improved by insertion of a nasogastric tube and reduction of intraperitoneal pressure via a stomach tube similarly to our case[8)]. Usually when we treat a patient with subcutaneous and mediastinal emphysema with ob-

structive shock, we first suspect tension pneumothorax. In previous reports, subcutaneous and mediastinal emphysema were detected in lower intestinal tract perforation into the retroperitoneal space[19)-21)]. Therefore, we should consider that the cause of obstructive shock exists occasionally not only in the chest, but also in the abdomen.

Generally, there is a risk of incorrect insertion into the trachea, with esophageal perforation occurring during placement of the nasogastric tube. With the perforation of the upper gastrointestinal tract, an increased risk of expansion of the perforation is suspected and misdirected insertion into the abdominal cavity will occur. To confirm accurate indwelling of the nasogastric tube, several methods have been performed, such as considering the inserted length from the nostrils, aspiration of stomach contents, and confirmation of X-ray images. In this case, these methods were carried out for safe and accurate indwelling of the nasogastric tube.

In conclusion, this patient with chronic severe gastric distension due to pyloric stenosis had tension pneumoperitoneum and cardiac arrest after perforation of the esophagogastric junction. The patient successfully recovered after appropriate volume resuscitation, decompression of intraperitoneal pressure by nasogastric tube, and laparotomy. Physicians should note that in case of obstructive shock and subcutaneous emphysema in the chest, the cause can exist even in the abdomen.

Conflict interest : The authors declare no conflict of interest.

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