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Development of pneumoperitoneum after CPR

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ABSTRACT

INTRODUCTION: Chest compressions are performed routinely and have several well-known complications, however one of the rare complications is pneumoperitoneum caused by air entry through a perforation of the viscus. The exact cause of the perforation is not always clear. Furthermore, this rarely reported condition does not have clear management guidelines.

PRESENTATION OF CASE: We present an uncommon complication of pneumoperitoneum following successful resuscitation possibly caused by the presence of an orogastric tube at the time of compressions in a 79 year old Hispanic male. Following chest compressions, a distended and tympanic abdomen was noted and air seen under the diaphragm in X-ray imaging.

DISCUSSION: A review of previous case reports along with etiology and evaluation of risk factors is presented.

CONCLUSION: Although the exact cause of pneumoperitoneum cannot be confirmed, emergency personnel should be aware of the risk factors associated with viscus perforation during chest compressions.

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1. Introduction

While many ideas in medicine can trace their lineage back for more than a thousand years, the idea of resuscitation is relatively new. First explored in the mid-1700s as a method to revive drowned victims, the modern closed chest compression technique was first described in the 1960s. New guidelines developed at a series of National Conferences started in 1966 combined chest compressions, external ventilation and defibrillation which resulted in a dramatic rise of successful resuscitations.^{1,2} Given the wide spread use of CPR, it is important to understand the drawbacks. Commonly known complications include rib fracture, sternum fracture, bleeding in the anterior mediastinum, heart contusion, laceration of liver and spleen and pneumothorax.³ Knowing these complications is essential to managing patients who are successfully resuscitated in order to avoid life threatening sequelae of chest compressions.

In this paper, we present a patient who developed pneumoperitoneum after CPR. Compared to the amount of patients who receive CPR, pneumoperitoneum has been reported very rarely. As of 2009, there have only been 67 reports.⁴ As the exact cause and risk factors leading to pneumoperitoneum have yet to be confirmed, we present the details of this case to further bolster the community of

knowledge in the hope that further work may lead to techniques that minimize this risk along with clear clinical guidelines for management.

2. Case report

A 79 year old Hispanic male was brought to the ED by ambulance because of difficulty breathing, altered mental status and mild fever. The patient had a past medical history of diabetes mellitus type II, hypertension, dyslipidemia, multiple duodenal ulcers and decubitus ulcer. Three months prior, the patient was hospitalized for decompensated CHF, pneumonia, ARDS requiring intubation, acute kidney injury and *C. Difficile* infection.

In the ED, the patient was cachectic and using accessory muscles for breathing. Vital signs were measured at: 99.8 °F, SaO₂ of 90% on room air, blood pressure of 111/56 mmHg, heart rate of 96 bpm and respiratory rate of 25 bpm. Further testing in the ICU revealed high anion gap metabolic acidosis, stage 4 chronic kidney disease, coagulopathy with INR 1.25, urinary tract infection with *Staphylococcus aureus* as well as bacteremia with *S. aureus*. An orogastric tube, central line and Foley catheter was placed to aid feeding, medication delivery and input/output monitoring. X-ray showed clear lungs bilaterally.

In the ICU, the patient continued to deteriorate and stopped breathing, SaO₂ dropped to 70% and the patient progressed from severe bradycardia to pulseless electrical activity and ventricular fibrillation. Patient was intubated and started on ACLS protocol. For 12 min the patient received chest compressions, IV push of

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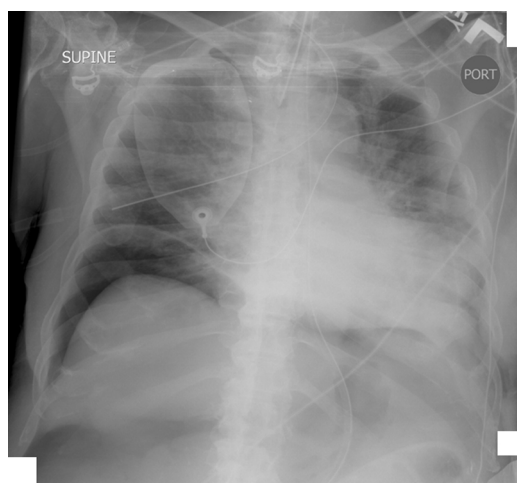


Fig. 1. Large amount of free air is visible under the right hemi-diaphragm.

epinephrine, amiodarone and sodium bicarbonate and two deliveries of 150J defibrillation. Resuscitation procedure was successful and patient returned to normal sinus rhythm.

After compressions, a chest X-ray was performed to check for rib fractures. On the X-ray in Fig. 1, there was a surprise finding. A collection of free air was seen under the right hemi-diaphragm. Surgery was immediately consulted and found no signs of peritonitis. There was no abdominal rigidity, guarding or distention. Furthermore, there were no signs of inferior vena cava compression or diaphragm splinting which may indicate tension pneumoperitoneum. There was however 300 mL of coffee ground fluid aspirated from the orogastric tube indicating bleeding from the stomach.

The patient was monitored by surgery every day and did not manifest any physical signs of peritonitis. The patient's metabolic status continued to deteriorate and the patient eventually passed away due to severe metabolic acidosis and cardiac arrest 4 days later.

3. Discussion

Appropriate treatment for symptomatic tension pneumoperitoneum is reduce pressure via OG/NG tube aspiration or needle decompression. In this case, the patient did not have a distended abdomen and did not have any signs of peritonitis. Definitive treatment of pneumoperitoneum is laparotomy with the goal of finding and repairing the perforated viscus. However, in the absence of any physical signs, the risks of laparotomy outweigh the benefits and should not be done. In such patients, laparotomy can be delayed until the patient is hemodynamically stable and better able to tolerate surgery.

Pneumoperitoneum following CPR is rarely reported. In existing literature, the most common cause of gastric perforation is along the lesser curvature of the stomach caused by esophageal intubation, mouth-to-mouth air delivery, or airway obstruction.^{4–6} Usually the cause of perforation can be determined, however in this case the precipitating factor is not certain. Increased airway resistance due to the patient's pulmonary resistance may have forced air into the stomach since as little as 15 cm H₂O is needed to cause gastric inflation.⁷ This patient's history of peptic ulcers also predisposed him to perforation.^{8,9} Finally the presence of an orogastric tube has been reported of being capable of causing gastric perforation.

Although the soft rubber of a feeding tube is not commonly thought to be capable of perforating the bowel, there have been several reported cases. In one reported case, the insertion of a

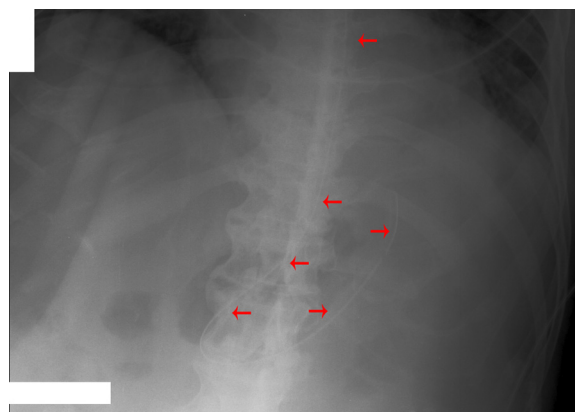


Fig. 2. Orogastric tube placement in stomach before CPR. Note the loop around the stomach.

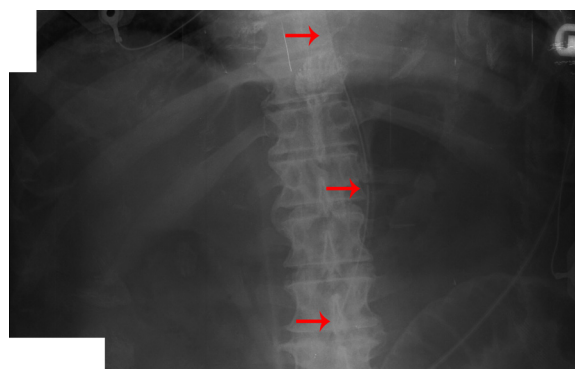


Fig. 3. The position of the orogastric tube is seen after CPR. The tube can be seen to course through the esophagus and under the diaphragm. Note the tube position appears to be different as compared to Figure 2.

nasogastric tube was confirmed to be placed correctly on X-ray but was later shown to be perforating the stomach on CT¹⁰ causing a pneumoperitoneum. In another case, a patient who previously had a Roux-En-Y gastric bypass developed pneumoperitoneum after placement of a nasogastric tube.¹¹ In this patient, 300 mL of coffee ground fluid was aspirated immediately after CPR but never again. Since no obvious cause of perforation was ever found, the orogastric tube is speculated to have caused a small perforation which later healed. This also supports the findings from later X-rays that show the amount of air under the diaphragm diminishing.

The image shown in Fig. 2 shows the orogastric tube placed correctly, overlying the stomach prior to chest compressions. While the tube is not coiled, there is a loop of tube seen within the stomach. After compressions, it can be seen in Fig. 3 that the orogastric tube's tip has changed position. It is possible that chest compressions may have introduced mechanical forces onto the tube causing it to push into an already weakened stomach wall.

4. Conclusion

Chest compressions will continue to be the standard in cardiopulmonary resuscitation and ACLS protocol however medical personnel must be cognizant of the complications which may arise after successful resuscitation. Patients with pneumoperitoneum require emergent surgical evaluation looking for signs of peritonitis or tension pneumoperitoneum. If either sign is present, immediate laparotomy for repair and drainage is the required. In the absence of physical signs, surgery may be delayed with plans for

laparoscopy or laparotomy at a time when the patient is better able to tolerate the procedure. Immediate interventions involve broad spectrum antibiotics and standard post resuscitation care. Clinicians should also be aware of risk factors such as airway disease, incorrect intubation and a history of ulcers. In this case the presence of an orogastric tube may also have contributed to the development of pneumoperitoneum, however this could not be confirmed with CT imaging due to the fact that the patient was too unstable to be moved from the ICU.

The exact cause of pneumoperitoneum is hard to determine since there are multiple causative factors and so few reported cases. For this reason, clinical practice will likely be derived from case reports and series.

Conflict of interest

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Ethical approval

Non-interventional case study.

Author contributions

Vishnu Mani, Laxman Pradhan and Sanjiv Gray were responsible for data analysis and writing the manuscript. Laxman Pradhan was solely responsible for data collection.

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