

Tension pneumoperitoneum after cardiopulmonary resuscitation

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A 61-year-old chronic schizophrenic patient developed sudden cardiac arrest and ventricular fibrillation. Tension pneumoperitoneum was noted after cardiopulmonary resuscitation. The patient had hypotension, lower limbs cyanosis and priapism. The tension was relieved by insertion of a large bore IV catheter through the abdominal wall. Immediately after decompression, the blood pressure improved with the lower limbs cyanosis and priapism resolved. (*Hong Kong j.emerg.med.* 2000;7:110-113)

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Case report

A 61-year-old chronic schizophrenic patient developed sudden cardiac arrest in a psychiatric institute. He was found to have distended abdomen and deterioration of his general condition for one day before the arrest. Immediate cardiac rhythm after the arrest revealed ventricular fibrillation. A prompt defibrillation was successful in restoring a spontaneous circulation. The patient required ventilation initially by bag-valve-mask followed by oro-tracheal intubation. The blood pressure was maintained at 90/60 mmHg and the patient was transferred to our emergency department.

On arrival at the emergency department of our hospital, the patient was unconscious with no spontaneous respiration and his carotid pulse was thready on palpation. His systolic blood pressure was 60 mmHg and the diastolic component was not measurable. Surgical emphysema was noted on the neck and the abdomen was distended and very tense. The oro-tracheal tube was found to have slipped out to the oropharynx. Reintubation was performed with application of cricoid pressure preceded by preoxygenation with bag-valve-mask.

On thorough examination, the patient was afebrile

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and with a Glasgow coma scale of 3. The neck veins were distended and the trachea was central. On auscultation, the lung fields were clear and air entry was equal. The tense and distended abdomen was tympanitic on percussion with no audible bowel sound. There was deep cyanosis of his lower limbs as well as a penile erection. The correct position of the endotracheal tube was verified and a nasogastric tube was inserted. However, there was no relief of the abdominal distension. His haemoglobin level was 3.8 g/dl and there was no evident source of active haemorrhage.

Radiographs of the chest and the abdomen revealed free gas under the diaphragm. (Figures 1 & 2) A tension pneumoperitoneum was diagnosed and needle decompression was performed using a 14G catheter. There was a gush of air heard with relief of the abdominal distension. The priapism and the lower limbs cyanosis resolved and the systolic blood pressure raised to 85 mmHg.

The patient was sent for emergency laparotomy immediately. There was 3.5 litre of pus and food residue in the peritoneal cavity and no intra-abdominal bleeding was found. A 5 cm long perforation extending from the lesser curvature to the anterior wall was identified. The small and large intestines appeared ischaemic. A partial gastrectomy followed by peritoneal lavage was performed. Post-operatively the patient required ventilatory and high dose of inotropic support. The patient succumbed 24 hours after the operation.

Pathology report of the partial gastrectomy



Figure 1. Free air under the diaphragm.



Figure 2. Free air under the diaphragm and in the peritoneal cavity.

specimen revealed extensive haemorrhagic gastritis with a perforated lesser curvature ulcer. The microscopy examination also showed chronic gastritis with no evidence of malignancy or *Helicobacter pylori*.

Discussion

Tension pneumoperitoneum (TPP) is a rare phenomenon that can become rapidly symptomatic. The mortality rate can be up to 80%.¹ The leading cause of tension pneumoperitoneum is ruptured abdominal viscera which accounts for 90%.² Other causes may be due to iatrogenic perforations caused by laparoscopic surgery or barotrauma.²⁻⁴ Some even believe that gas-forming organisms caused the phenomena of TPP.⁴ The most common site of perforations are the lesser curvature of the stomach and the caecum.^{3,5,6} Gastric rupture along the lesser curvature is favoured by the lesser mucosal fold, low elasticity and fixation by the hepatogastric ligament.¹

Our postulation is that this man has had a prolonged perforation of stomach wall before the cardiac arrest as suggested by severe anaemia and the large amount of pus inside the peritoneum. The gas in the peritoneal space was a result of perforated viscera and gas formed by the bacteria. In the process of active resuscitation, however, the continuing bag-valve-mask ventilation together with the external cardiac massage might result in a pumping action pushing more gas into the peritoneal space. This process would be accentuated if the omentum and liver overlying the rupture site act as a one-way valve.⁷ This can be explained by the failure of the nasogastric tube in decompressing the distended abdomen.

Another possible hypothesis of the pneumoperitoneum is pulmonary barotrauma subsequent to over-aggressive ventilation during resuscitation.⁸ Air leaks from ruptured alveoli to the mediastinum which moves inferiorly into the intraperitoneal space either directly through the pleuro-peritoneal defect or indirectly through the retroperitoneal space.⁸ However, in this patient, this hypothesis is less likely because of the intraoperative finding of ruptured stomach and the lack of pneumomediastinum in chest X-ray.

In this patient, the very high intra-peritoneal pressure was indicated by the clinical features of hypotension, lower limbs cyanosis and priapism. The hypotension was markedly corrected by the needle decompression of the intra-abdominal tension. In TPP, cardiac index decreases because of the reduction of stroke volume secondary to decreased venous return, especially from the inferior vena cava,⁷ or due to compression of aorta and increase in systemic arterial resistance.

The cyanosis was probably secondary to reduced venous return of the lower limbs whereas the priapism might be due to the effect of raised pressure on the pelvic veins or the spinal cord.⁷

Tension pneumoperitoneum will also impede respiration by reducing tidal volume and lung compliance because of the splinted diaphragm. In addition, decreased venous return can worsen any ventilation perfusion mismatch.

Since the TPP in this patient was probably due to pre-existing pneumoperitoneum aggravated by bag ventilation and external chest compression, some measures can be used to prevent the development of TPP. Attention to the airway position and avoidance of over-zealous bag ventilation should be emphasised. Also, early endotracheal intubation is vital and application of cricoid pressure can prevent excessive air entry into the peritoneal cavity.⁷

Tension pneumoperitoneum is an emergency and requires prompt treatment. Therapeutic decompression has been reported with a small trocar through the abdominal wall,^{2,4,7} by transabdominal chest tube or by an emergency mini-laparotomy.^{1,6} Mini-laparotomy permits faster evacuation of large volume of air as compared to needle decompression. The former method uses the open approach under direct vision to enter the peritoneal cavity and hence reduces the risk of visceral injury especially when distension mechanism is unclear.⁴ However, the main drawback is that this is operator dependent and relatively time consuming.

Another local case was reported by Siu⁹ in 1997. The patient developed tension pneumoperitoneum after cardiopulmonary resuscitation. A minilaparotomy relieved the tension and improved

the ventilation but the patient could not be revived. Post-mortem examination was waived and hence the source of leakage was not documented.

Just like tension pneumothorax, prompt recognition and decompression of this condition is important and can sometimes be life-saving.

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