Perforation of the Gastrointestinal Tract and Pneumoperitoneum in Newborns Treated with Continuous Lung Distending Pressures

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Abstract. In newborns with respiratory distress being treated with continuous lung distending pressures, the development of pneumoperitoneum could be a manifestation of an air-block phenomenon but it can also be due to a real perforation of the bowel. When this clinical situation arises upright films or cross-table lateral films will be helpful to demonstrate air fluid levels and some consideration should be given to inserting aqueous contrast material into the stomach and take sequential films in order to demonstrate the presence or absence of a perforation. Six illustrative cases are reported.

Key words: Pneumothorax, pneumoperitoneum.

Introduction

Six patients are reported (Table 1) in whom pneumoperitoneum developed while they were being maintained on continuous lung distending pressures [3] and oxygen for a variety of pulmonary and cardiac diseases. In each case there was gastrointestinal perforation, the sites ranging from stomach to colon; the etiology was presumably ischemic necrosis of the gastrointestinal tract since all patients were in shock.

These cases are in contrast to recent reports [1, 2, 4] of patients (with similar basic diseases and therapy with continuous distending pressures) whose pneumoperitoneum was in the absence of gastrointestinal perforation, the air presumably reaching the abdomen from an alveolar leak.

It cannot be assumed that the appearance of pneumoperitoneum in such critically ill patients represents a non-surgical condition; radiologic distinction between "medical" and "surgical" pneumoperitoneum may be impossible, although the presence of air fluid levels [1] in the peritoneum points to a gastrointestinal perforation. The introduction of water soluble contrast agents [1] into the stomach may help in identifying a site of perforation.

Case Reports (Table 1)

Case I: Gastric perforation. An 1130 gram male infant was born after spontaneous vaginal delivery. Although he was depressed, he responded to bag administration of oxygen. Severe respiratory distress then developed, with retractions, grunting and cyanosis. He was intubated and given higher concentrations of oxygen and initially intermittent positive pressure respirations. The condition did not improve, and at 24 hours of age, pneumoperitoneum was noted. He was taken to the operating room and a perforation of the stomach was noted. He died immediately thereafter.

Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>Birth Weight</th>
<th>dx.</th>
<th>P-Med., P-Thorax</th>
<th>P-Peritoneum</th>
<th>Site of Perf.</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. I</td>
<td>1180 gm</td>
<td>RDS b</td>
<td>Yes</td>
<td>Yes</td>
<td>Stomach</td>
</tr>
<tr>
<td>No. II</td>
<td>Premature</td>
<td>RDS a &amp; RHD b</td>
<td>No</td>
<td>Yes with A/F level</td>
<td>Stomach</td>
</tr>
<tr>
<td>No. III</td>
<td>Premature</td>
<td>RDS a</td>
<td>No</td>
<td>Yes</td>
<td>Ileum</td>
</tr>
<tr>
<td>No. IV</td>
<td>755 gm</td>
<td>RDS a</td>
<td>No</td>
<td>Yes</td>
<td>Jejunum</td>
</tr>
<tr>
<td>No. V</td>
<td>2200 gm</td>
<td>RDS a</td>
<td>No</td>
<td>Yes</td>
<td>Stomach</td>
</tr>
<tr>
<td>No. VI</td>
<td>3500 gm</td>
<td>CHF 20 to CNS AV to Vein of Galen</td>
<td>No</td>
<td>Yes</td>
<td>Colon</td>
</tr>
</tbody>
</table>

a RDS Respiratory distress syndrome (i.e. Hyaline membrane disease)
b DIC Disseminated intravascular clotting.
pressure breathing. Chest film showed typical changes of the respiratory distress syndrome (i.e. hyaline membrane disease). He continued to do poorly and repeat radiographic examination the next day showed a right tension pneumothorax, a pneumomediastinum and cervical subcutaneous emphysema. Seizures developed as well as bleeding from the endotracheal tube; a ventricular tap showed gross blood. A second roentgenogram showed the presence of a pneumoperitoneum (Fig. 1) in addition to the previously noted lung changes. He soon died.

At autopsy there was severe hyaline membrane disease and bilateral pulmonary hemorrhage. Bullous emphysematous changes were present in the right lung with a tension right pneumothorax. A gastric perforation of 4 mm length was present in the mid portion of the lesser curvature of the stomach. Focal gastric mucosal necrosis was present about the tear; the muscle coats were disrupted.

Comment: The gastric perforation was a terminal, presumably ischemic event in the shocky downhill course of this patient. His lung disease was accompanied by central nervous system bleeding and pulmonary hemorrhage.

Case II: Gastric perforation. A male infant was born after a 32 week gestation and had immediate problems and low Apgar scores. Severe jaundice developed on the basis of Rh incompatibility. Severe respiratory distress was present and chest films showed typical changes of respiratory distress syndrome. He was treated with oxygen and required ventilatory assistance, initially by intermittent positive pressure breathing and later by endexpiratory pressure methods [3]. An exchange transfusion was done via the umbilical vein.

Abdominal distension developed the next day. Films showed hyaline membrane disease, right upper lobe collapse, and both air and fluid in the abdomen (Fig. 2). Laparotomy showed gastric perforation of the anterior wall; the tear was oversewn. The patient died soon afterwards.

Autopsy showed, in addition, pulmonary hemorrhage, severe hyaline membrane disease, and a left subdural hematoma.

Comment: The finding of an air-fluid level in the abdominal films was helpful [1] indicating the presence of a perforated viscus. In addition, there was no evidence of "airblock" in the form of pulmonary interstitial emphysema, pneumomediastinum or pneumothorax.

Case III: Ileal perforation. This premature infant was born after a 32 week pregnancy with low Apgar score at birth. He rapidly developed respiratory distress and was placed on oxygen given by mask with continuous positive airway pressure. Severe respiratory distress syndrome developed and there were also changes of disseminated intravascular clotting. On the third day, free intra-abdominal air was noted on the chest film. To see if a site of gastrointestinal leak could be detected (since there was no sign of "airblock" as discussed in the previous case) 10cc of 50% sodium diatrizoate was instilled into the stomach via a nasogastric tube. Serial films

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Fig. 1. Case I. (Children’s Mercy Hospital), Gastric perforation. 1180 gm male with R. D. S., right pneumothorax and pneumomediastinum. Gastric perforation was cause of pneumoperitoneum.

Fig. 2. Case II. (Children’s Mercy Hospital), Gastric perforation. Premature male with R. D. S. and Rh. incompatibility. Large abdominal free air-fluid level caused by gastric perforation.