Iatrogenic esophageal perforation in a preterm infant
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A premature male newborn was transferred six hours after birth with a diagnosis of esophageal atresia from another pediatric institution. The baby had been born at 31 weeks of gestation to a 29-year-old G2/P1 by caesarean section due to strong vaginal bleeding and breech presentation. Pregnancy had been uncomplicated. In particular, several prenatal ultrasound examinations had never demonstrated a polyhydramnios.

The infant had adapted well with Apgar scores of 4, 6, and 9 at 1, 5, and 10 minutes, respectively. His birth weight was 1750 g (P50-75), his birth length 46 cm (P90) and his head circumference 30.5 cm (P50-75). Within a few minutes of life, the baby developed respiratory distress and an increasing oxygen requirement. Oral intubation was only successful after several attempts, and no oro- or nasogastric tube could be placed.

Because the pediatricians suspected esophageal atresia, a contrast study of the upper esophageal pouch was requested. Barium sulfate was used as contrast medium. When the study failed to demonstrate a continuous esophagus and the barium projected over the upper thoracic aperture, esophageal atresia with an upper pouch and a tracheo-esophageal fistula was suspected (type I, according to L. Spitz). With the intent to stabilize the baby’s respiratory condition, two doses of surfactant were administered and the infant was prepared for transfer.
On arrival at our department, the baby’s cardiorespiratory status was stable. A babygram revealed a depot of contrast medium in the cervical region and another smaller one within the stomach (Fig. 1). These findings were inconsistent with the presence of esophageal atresia. Therefore another attempt to insert a nasogastric tube was made and successfully performed by an experienced pediatric anesthesiologist (Fig. 2). Thus, esophageal atresia could definitely be ruled out. Close inspection of the posterior pharyngeal wall revealed a fibrin-covered lesion (about 5 mm in diameter) representing a tear (Fig. 3). A diagnosis of iatrogenic perforation of the esophagus was made. The child received broad-spectrum antibiotics, a proton pump inhibitor and was extubated within two days. Feeding via the nasogastric tube was started on day three. On day of life 10, a contrast study using a small amount of water-soluble contrast medium showed free passage and regular peristalsis of the esophagus. As expected, the barium had stayed in place (Fig. 4). Repeat endoscopy demonstrated complete healing of the pharyngeal tear.

The further hospital course of the baby was uneventful. It remains to be seen whether the barium extravasation will have negative consequences.
Babygram revealing a collection of contrast medium in the upper thoracic paravertebral area; another collection can be seen within the stomach.
Babygram after successful insertion of a nasogastric tube (asterisks: tip reaching the distal duodenum or proximal jejunum).
Endoscopic view of the posterior pharyngeal wall showing a fibrin-coated lesion.
Contrast study with a water-soluble contrast medium demonstrates free passage through the esophagus (day ten after birth); barium sulfate (asterisks) from the previous examination has stayed in place.
Iatrogenic esophageal perforation is uncommon in newborn infants and children. In neonates, particularly in preterm babies, repetitive placement of oro- and nasogastric tubes or repetitive pharyngeal suctioning has been reported to cause esophageal perforation (1-5). Rigid, hurried or inexperienced manipulation with the laryngoscope blade or repeat suctioning may cause spasm of the cricopharyngeal muscle and may result in closure of the esophageal lumen (3, 6). In addition, extension of the neck compresses the esophageal wall against the cervical vertebra and thus facilitates formation of a laceration. Finally, digital manipulation by the obstetrician during breech delivery (i.e., Veit-Smellie maneuver) has been reported to result in pharyngeal tears (4, 7). Neonatologists, pediatric anesthesiologists and pediatric surgeons should consider this possibility when confronted with a patient such as the one presented in this case report.

When a nasogastric tube enters the perforation site it cannot be passed into the stomach, but will get stuck at the upper or mid-thoracic level (Fig. 5). This finding can be misinterpreted as being indicative of esophageal atresia (3, 8-10). Nearly all patients reported to have iatrogenic esophageal perforation were referred with the initial diagnosis of esophageal atresia. If contrast medium is applied through the malpositioned nasogastric tube, an irregularly shaped pouch can be visualized in the posterior mediastinum, sometimes extending down to the diaphragm (“double-esophageal structure”) (Fig. 6).
Iatrogenic esophageal perforation: nasogastric tube enters the perforation site but cannot be advanced into the stomach.
Classification of esophageal atresia, according to L. Spitz; the former Type III (H-fistula) has been excluded.
Contrast medium instilled through the misplaced tube outlines an irregular pouch in the posterior mediastinum extending down to the diaphragm ("double-esophageal structure"); image from a different case (see Ref. 10).
Contrast studies are usually unnecessary in suspected esophageal atresia. A chest x-ray and abdominal plain film will usually suffice to establish the diagnosis. Visible air and a coiled tube in the upper pouch, as well as the presence or absence of air within the bowel will normally allow proper classification of the type of esophageal atresia (Fig. 7). Perforation of the upper pouch as a complication of continuous suction has been reported but is probably extremely rare (11).

Mollit et al. described three types of lesions associated with esophageal perforation: 1) pharyngeal pseudodiverticulum created by a local cervical leak, 2) mucosal perforation extending posteriorly in parallel to the esophagus (“double-esophageal structure”) (Fig. 6), and, 3) free intrapleural perforation with free air within the mediastinum and pleural space (10, 12).

When perforation has occurred, signs and symptoms may vary initially; the condition may even be clinically silent (8-10). However, perforation and dissection may also cause edema and obstruction of the upper third of the esophagus. This may result in excessive drooling and difficulties in feeding or vomiting. Coupled with the inability to advance a nasogastric tube into the stomach, these signs and symptoms may lead to a misinterpretation as esophageal atresia (7, 8, 10, 13). Endoscopy will allow the identification of the perforation site, possibly reveal a false lumen and show a patent esophagus; it must be carried out with great caution to avoid further injury (10, 14).
Early diagnosis of esophageal perforation in neonates may allow nonsurgical management. In contrast, esophageal perforation in adults carries a grave prognosis (2, 4, 10, 15, 16). Depending on the initial presentation, most children with iatrogenic esophageal perforation can be treated conservatively. Broad spectrum antibiotics and proton pump inhibitors are given for 10 days and a nasogastric tube inserted under fluoroscopic control can be used for feeding, whereas oral feedings are withheld during this time. We suggest that a repeat esophagography be performed to show free passage and absence of a leak before oral feedings are started.

Generally, treatment of esophageal perforation in neonates should be successful with a good outcome (2, 4, 10). The consequences of the barium extravasate in our patient, however, are still unclear. Barium sulfate in the mediastinum or even within the thoracic cavity may evoke an inflammatory response with granuloma formation and ultimately lead to fibrosis (17).


