Acute Gastric Dilatation Causing Respiratory Failure and "Tension Pneumothorax" in an Elderly Woman With a Diaphragmatic Hernia*

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The occurrence of respiratory failure as a result of a large diaphragmatic hernia is a well-described entity in infants with congenital hernias. On reviewing the literature, the authors did not find a similar clinical presentation in the adult population. They report the case of an elderly patient with a large hiatus hernia who developed recurrent episodes of life-threatening respiratory failure and hemodynamic compromise due to recurrent gastric dilatation. Decompression with nasogastric suction resulted in dramatic and immediate relief of the respiratory distress. One should keep in mind the possibility of intrathoracic gastric dilatation as a cause of acute respiratory insufficiency in patients with hiatal hernia.

The association between diaphragmatic hernia and respiratory failure in neonates is well established. The major factor causing respiratory failure in these young infants is hypoplasia of the lung due to sustained compression in utero by the herniated abdominal contents. In cases of Bochdalek hernia, without surgical correction, 75 percent of these infants will not survive, and extracorporeal membrane oxygenation may be required until lung function recovers adequately. The association between gastric herniation and pulmonary compromise is less well described in the adult population. We would like to report here the unusual occurrence of respiratory compensation secondary to acute gastric dilatation in a patient with preexisting diaphragmatic hernia.

**CASE REPORT**

An 84-year-old woman, a nonsmoker, was admitted to the intensive care unit of the Hadassah University Hospital because of acute respiratory failure. She had a large diaphragmatic hernia, which had been diagnosed 20 years previously on a routine chest x-ray film. Five years ago, she was hospitalized with unexplained respiratory failure that required mechanical ventilation. Two days prior to her present admission, she developed a mild nonproductive cough with progressive dyspnea but no fever. She had no episodes of vomiting or alteration of consciousness. On admission, she was alert and in severe respiratory distress, with a respiratory rate of 46 breaths per minute, pulse of 110/min, systolic blood pressure of 70 mm Hg, and temperature of 38.0°C. She was not cyanotic and displayed no clubbing. There was rightward deviation of the trachea and marked distension of the neck veins. Examination of the chest

**FIGURE 1.** Chest x-ray film obtained on admission shows massive gastric dilatation with rightward shift of the mediastinum.

**FIGURE 2.** Repeat x-ray film obtained 2 h later, after insertion of a nasogastric tube shows that the mediastinum has returned to midposition.

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revealed severe kyphosis with no air entry over the left chest and normal breath sounds on the right. Heart sounds were heard to the right of the sternum. There was no evidence of deep vein thrombosis in the legs.

Laboratory workup disclosed a white blood cell count of 15,000 mm³; a hemoglobin level of 13.2 g/dl; and normal renal and liver function tests. Arterial blood gas analysis on admission showed the following values: PO₂, 63 mm Hg; PCO₂, 120 mm Hg; pH, 7.19; HCO₃⁻, 46 mmol/L. A chest x-ray film (Fig 1) showed massive gastric dilatation with rightward shift of the mediastinum.

The patient was intubated and mechanically ventilated. A nasogastric tube was inserted, and large amounts of gas and fluid were drained. Once gastric decompression had been achieved, the patient's respiratory and hemodynamic status improved dramatically, and the mediastinum returned to midpoint (Fig 2). Repeat blood gas analysis 4 h after insertion of the nasogastric tube showed the following values: PO₂, 95 mm Hg; PCO₂, 53 mm Hg; pH, 7.39. The patient was treated with intravenous cefuroxime and aminophylline with inhalations of albuterol (albutamol) and ipratropium bromide. She was gradually weaned, and on the seventh day after admission, extubation was performed. That same evening, she again developed severe respiratory distress and CO₂ retention with radiologic evidence of gastric dilatation. She improved immediately after a nasogastric tube was inserted. She had recurrent similar episodes, all of which responded to nasogastric tube insertion. Thereafter, the patient stabilized, and the nasogastric tube was removed without further episodes.

Metabolic, gastrointestinal, and neurologic workup did not reveal an underlying cause for the acute gastric dilatation. Findings from computed tomography of the chest and lung perfusion-ventilation scanning were not contributory to a diagnosis. The patient was discharged on the 23rd day after admission in no respiratory distress.

**DISCUSSION**

In neonates, a large diaphragmatic hernia frequently results in respiratory failure. The most common form giving rise to this complication is the Bochdalek type (posterior), but Morgagni's hernias and congenital eventration of the diaphragm may also impair respiratory function. Respiratory compromise is primarily due to hypoplasia of the lung tissue due to prolonged pressure in utero from the intrathoracic intestinal contents. In these patients, anatomic correction of the hernia does not result in immediate respiratory relief, and extracorporeal membrane oxygenation may be required until the hypoplastic lung becomes functional.

Although diaphragmatic hernia is very frequent in the adult population, it seldom gives rise to respiratory symptoms. If gastroesophageal reflux is also present, aspiration may occur, resulting in lung infection or bronchospasm. Diaphragmatic hernia has not, however, been reported as causing respiratory compromise due to a mechanical pressure effect.

Acute gastric dilatation is not infrequent after trauma and after major abdominal or thoracic surgery. It may occur in patients with autonomic dysfunction and gastroparesis, such as in familial dysautonomia. Gastric dilatation may further compromise respiratory function in patients with preexisting lung disease, but as long as the stomach is below the diaphragm, it is not a major factor in causing respiratory failure.

Our patient presented with a combination of these two entities, namely; massive gastric dilatation in the presence of a herniated intrathoracic stomach. We believe that this was the primary cause of her acute respiratory failure. This contention is supported by the fact that the patient's respiratory distress was immediately relieved on several separate occasions by the insertion of a nasogastric tube. We could find no similar cases in the literature, although a case of transient respiratory distress following air insufflation of a herniated stomach prior to gastroscopy has been reported.11

Acute dilatation of the intrathoracic stomach caused marked rightward shift of the mediastinum (Fig 1), and in our opinion this shift resulted in hemodynamic compromise in a manner analogous to that seen with tension pneumothorax. Decompression resulted in immediate hemodynamic recovery, as happens following insertion of an intercostal drain in patients with tension pneumothorax.

It is not clear what the initial stimulus causing gastric dilatation was. One possibility is that the patient had a mild respiratory infection, as evidenced by the presence of cough, leukocytosis, and mild fever on admission, and that this resulted in tachypnea, apneophasia, and progressive dilatation of the stomach. After insertion of the nasogastric tube and decompression of the stomach, a moderate elevation of PCO₂ persisted, which may have been due to chronic restrictive lung disease due to kyphoscoliosis.

In conclusion, in the presence of a diaphragmatic hernia, acute gastric dilatation may cause respiratory failure and hemodynamic collapse, not only in neonates, but also in the elderly. Insertion of a nasogastric tube is a lifesaving procedure that results in rapid relief of the respiratory and cardiovascular distress.

**REFERENCES**


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Acute Gastric Dilatation Causing Respiratory Failure (Berkman et al)