High-altitude Pulmonary Edema with Pulmonary Thromboembolism*

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High-altitude pulmonary edema (HAPE) is a form of noncardiogenic pulmonary edema. The pathophysiology of HAPE remains unclear. A case of HAPE was associated with pulmonary thromboembolism of a left upper pulmonary artery. Pulmonary thromboembolism was an important factor in development of HAPE in this case.

(Chest 1993; 103:948-50)

HAPE = high-altitude pulmonary edema.

High-altitude pulmonary edema (HAPE), a severe form of acute mountain sickness, is observed in Japan, especially at an altitude of 2,600 to 3,000 m in the "Japanese Alps." It seems that pulmonary hypertension and increased pulmonary endothelial permeability are mainly involved. Hackett et al. reported four patients without a right pulmonary artery, all of whom suffered from HAPE at altitudes of 2,000 to 3,000 m in Colorado. Pulmonary edema occurred in the left lung which received the entire right ventricular output. Their case report suggests that the hemodynamic alterations of high pressure and flow in the pulmonary artery are important in the development of HAPE.

A patient with HAPE associated with pulmonary thromboembolism showed remarkable pulmonary hypertension. The role of pulmonary thromboembolism in development of HAPE is examined.

CASE REPORT

A 15-year-old high school student arrived at 2,700 m in the Japanese Alps from the Kanto area (almost sea level) on Aug 25, 1990. He was healthy before climbing. When he climbed up to 2,900 m the next day, he noted headache and complained of cough, sputum, and shortness of breath. On the morning of Aug 28, he became progressively cyanotic, dyspneic, and comatose. He was rescued by helicopter and admitted to the Shinshu University Hospital (altitude 660 m) the evening of Aug 28. On admission, his condition had improved to a certain extent. He was confused but not comatose. He was febrile (37.3°C). Respiration were 46 per minute, pulse rate was 117 beats per minute, and blood pressure was 110/70 mm Hg. Coarse crackles were audible over both sides of the chest. The main laboratory test results disclosed the following values: hemoglobin, 15.2 g/dl; red blood cells, 496 x 10^6/cu mm; hematocrit, 43.2 percent; platelets, 16.8 x 10^9/cu mm; white blood cells, 22.700/cu mm, with band cells of 23 percent; ESR 14 mm/1 h; CRF 22.1 mg/dl; BUN, 14 mg/dl; creatinine, 0.5 mg/dl; Na, 135 mEq/L; K, 4.0 mEq/L; Cl, 99 mEq/L; total protein, 6.0 g/dl; Alb, 3.3 g/dl; total bilirubin, 1.2 mg/dl; SGOT, 33 KU; SCPT, 15 KU; LDH, 365 mIU; CK, 639 mIU; and glucose, 114 mg/dl. A sample of arterial blood at room air revealed that the PaO2 was 28.3 mm Hg, the PaCO2 was 28.3 mm Hg, and the pH was 7.457. A chest x-ray film showed a dilated main pulmonary artery and scattered infiltrates all over the lung fields, which were improved on the seventh hospital day. He was discharged on the 13th hospital day.

DISCUSSION

Our patient was diagnosed with HAPE from our criteria, including clinical symptoms, physical findings, and chest roentgenographic infiltrates. On admission, he showed pulmonary hypertension of 56 mm Hg. His pulmonary artery pressure was remarkably elevated compared with our previous Japanese patients with HAPE, since our ten cases of HAPE, of whom four patients were previously reported, revealed moderate pulmonary hypertension of 28.5 ± 3.9 (SD) mm Hg on admission. In spite of improved symptoms and PaO2, pulmonary hypertension still persisted. Thereafter, the patient was treated with high-flow oxygen and antibiotics. The PaO2 improved to 74.9 mm Hg in FIO2 of 0.5 the next day. All symptoms cleared on the third hospital day, but high Ppa persisted (mean pressure of 40 mm Hg). Infiltrates seen on the chest x-ray film disappeared on September 3. We performed a 99m-Tc-MAA perfusion lung scan and a 133Xe ventilation lung scan. Perfusion scan (Fig 2A, left) showed a defect mainly in the left upper lung field, in contrast with normal ventilation scan (Fig 2B, right). Digital subtraction pulmonary angiography revealed inadequate visualization of the area of the left upper lobe artery. Because of the appearance of thrombocytopenia (9.7 x 10^9/cu mm), the Swan-Ganz catheter was removed on the fourth hospital day. His arterial blood gas analysis, laboratory findings, and thrombocytes became normal on the seventh hospital day.

![Figure 1. A chest x-ray film on admission showed scattered infiltrates in bilateral lung fields.](image-url)
fore, we guessed the abnormality to be the pulmonary circulation. We confirmed a diagnosis of pulmonary thromboembolism because there was a normal ventilation lung scan, and defects in both perfusion lung scan and pulmonary angiography.

The pathophysiology of HAPE remains unclear. The accumulation of protein-rich fluid in the bronchoalveolar space implies an increased permeability of the pulmonary vascular endothelium. The increase in permeability is probably caused by hypoxia, hypoxia plus inflammatory mediators, high intravascular pressure, or some combination of these factors. Pulmonary hypertension also plays an important role in the development of HAPE. It is well known that pulmonary hypertension with normal wedge or left arterial pressure and normal cardiac output is present in patients with HAPE. Moderate pulmonary hypertension (mean pressure of 28.5 mm Hg) existed on admission in our Japanese cases of HAPE as mentioned earlier. Furthermore, we have previously described that there is the accentuated pulmonary vascular response to hypoxia and exercise in persons susceptible to HAPE. It seems likely that the hyperresponse of pulmonary artery pressure and pulmonary vascular resistance to hypoxia and exercise is an important factor in the development of HAPE which occurred even at modest altitudes (2,600 to 3,000 m) in places such as the Japan Alps. The pulmonary capillaries may rupture when their internal pressure is greatly elevated.

The case report of Hackett et al has implicated pulmonary thromboembolism in the development of HAPE in our present case. The alterations of the pulmonary circulation in thromboembolism mimic those in the absence of the right pulmonary artery. Therefore, it seems that the incidence of HAPE in persons with pulmonary thromboembolism is very high.

We assumed that the genesis of pulmonary thromboembolism in our present case was not due to the complication of the Swan-Ganz catheterization, but related to the development of HAPE. Several studies have suggested the presence of hypercoagulation in HAPE, since there is a decrease in platelet counts and prolonged prothrombin time during an early stage, and microvascular thrombi in the pulmonary vessels postmortem. To our knowledge, however, no other case of HAPE associated with pulmonary thromboembolism has shown a distinct defect in the lung perfusion scan or pulmonary angiogram such as documented in the present case.

A patient is described with HAPE associated with pulmonary thromboembolism. Pulmonary thromboembolism results in pulmonary hypertension and high flow into the opened vessels. These factors are important in the development of HAPE in our present case, occurring at modest altitudes of 2,900 m in the Japan Alps.

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Tension Fecal Pneumothorax in a Postpartum Patient

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A 20-year-old woman developed severe shortness of breath 4 h after a cesarean section. Chest roentgenogram showed a pleural effusion and tension pneumothorax; insertion of a chest tube drained liquid stool. At surgery she was found to have a left diaphragmatic defect with herniation, strangulation, and perforation of the transverse colon into the pleural cavity. (Chest 1993; 103:950-51)

Although traumatic diaphragmatic hernia complicating pregnancy has been recognized, to our knowledge, this is the first case reported presenting as a tension fecal pneumothorax.

CASE REPORT

The Medical Service was consulted regarding a 20-year-old postpartum woman because of anuria, fever, and persistent tachycardia.

The patient was a gravida 1 para 1 woman with an uneventful 41-week pregnancy up until 72 h prior to our consult when she presented to the hospital with an 8-h history of recurrent bilious vomiting, nausea, generalized constant crampy abdominal pain, and constipation. She was given IV fluids, antิemetics, and soap suds enema with resolution of her symptoms.

Forty-two hours prior to our consult she was readmitted to the hospital for induction of labor and given oxytocin (Pitocin) as well as prostaglandin. A cesarean section was performed 40 h later after failure of the fetus to descend.

Nine hours prior to consult, she was noticed to have a temperature of 38.6°C, her urine was cloudy, and she was given cefoxitin. At that time her only complaint was left-sided chest pain that worsened with breathing; she admitted this had been present for two days.

In the immediate postpartum period, she was noticed to be persistently tachycardic and in mild respiratory distress. She remained febrile and she had no urine output despite receiving 4.5 L of IV fluids during the previous 12 h.

Her medical history was remarkable for heroin and cocaine abuse until three years prior to hospital admission. She had sustained a left lower hemithorax stab wound two years prior to admission.

Physical examination revealed a 20-year-old moderately obese woman in moderate respiratory distress. Blood pressure was 105/45 mm Hg, heart rate was 170 beats/min, respiratory rate was 38, and temperature was 38.6°C. She had dry oral mucosa and poor skin turgor. Her trachea was deviated to the right. There were decreased breath sounds in the left hemithorax with hyperresonance to percussion in that area. Abdominal examination revealed a distended abdomen with a fresh Pfannenstiel sutured cesarean incision and absent bowel sounds; the abdomen was soft and nontender but the patient was under the effects of epidural anesthesia. There was no peripheral cyanosis or edema.

Laboratory data were as follows: WBC count, 17.8 with 87 percent polymorphonuclear leukocytes; hemoglobin and hematocrit, 13.7 g and 40.3 percent; serum urea nitrogen 20 mg/dl; creatinine, 0.8 mg/dl; Po2, 79 mm Hg, Pco2, 24.2; and pH, 7.47 on room air. After a chest roentgenogram revealed a hydropneumothorax with shift of the mediastinum to the right (see Fig 1), a chest tube was inserted into the sixth intercostal space with drainage of foul-smelling, turbid, greenish material. Examination of the fluid revealed a lactate dehydrogenase (LDH) value of 8,020 U, glucose level of 21 mg/dl, and WBC count of 3,600/cu mm. Gram stain showed many Gram-negative rods, Gram-positive rods, and Gram-positive cocci in pairs: the amylase level in the pleural fluid was 1,290 U/l. Culture of the fluid grew Escherichia coli, Enterococcus faecalis, and diphtheroids.

After IV hydration and chest tube placement, the patient's urine output increased and her respiratory distress improved.

Initially an esophageal rupture was suspected but a diatrizoate meglumine (Gastrografin) swallow failed to demonstrate its presence. Because of continued drainage of fecal material through the chest tube and persistence of her hydropneumothorax despite the insertion of a second chest tube, she was taken to surgery 24 h after the first chest tube insertion. A defect was found at the apex of the left hemidiaphragm with strangulation and perforation of the midtransverse colon. An omentectomy and colon resection encompassing the perforation was performed with primary Anastomosis. The bowel was reduced into the abdominal cavity and the diaphragmatic defect was repaired.

DISCUSSION

While traumatic diaphragmatic hernia is a well-recognized complication of blunt and penetrating injuries to the abdomen and thorax, strangulation of the large bowel that migrates through that hernia into the thorax with subsequent rupture and the development of fecal pneumothorax is most unusual. Indeed, only three prior cases of tension pneumothorax associated with strangulation and perforation of the colon through a traumatic diaphragmatic hernia have been reported. Moreover, although traumatic diaphragmatic hernia complicating pregnancy has also been recognized, to our knowledge, this is the first case reported in

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Figure 1. Left tension pneumothorax with a left pleural effusion.