Spontaneous Lung Hernia*

Antonio T. Donato, M.D., F.C.C.P.,** Florencio A. Hipona, M.D.,† and Shito Navani, M.D.‡

An unusual case of spontaneous herniation of the lung is presented. The mechanism of intercostal muscle disruption, etiology, and treatment of lung hernias are discussed.

*From the Departments of Cardiothoracic Surgery and Radiology, The Boston City Hospital, Boston.
**Presently Staff Surgeon, Veterans Administration Hospital, Salem, Va., and Assistant Professor of Surgery, University of Virginia Medical School.
†Associate Director of Radiology, The Boston City Hospital and Professor of Radiology, Harvard Medical School.
‡Chief, Outpatient Department of Radiology, The Boston City Hospital and Associate Professor, Boston University School of Medicine.
Reprint requests: Dr. Hipona, Boston City Hospital, 818 Harrison, Boston 02118

Herniation of the lung, a relatively uncommon clinical entity, is defined as the protrusion of lung tissue beyond the confines of the thoracic cavity through an abnormal opening in the chest wall, diaphragm or mediastinum lined by pleura.

To date only 266 cases of lung hernia have been published. Munuell recently gave comprehensive reviews on the subject.

Morel-Lavellée classified lung hernia according to anatomic location: cervical, thoracic and diaphragmatic; and according to etiology: congenital or acquired. He further differentiated acquired lung hernia as: traumatic, consecutive, spontaneous and pathologic. The term 'consecutive hernia,' previously referred to as 'hernia of delayed onset after injury,' is now classified under 'traumatic hernia.'

Current survey of the literature indicates that 82 percent of lung hernias are acquired, while 18 percent are congenital: 65 percent thoracic, 35 percent cervical, with only one reported case of diaphragmatic hernia by Beale in 1882. Of the acquired type, 52 percent are secondary to penetrating and nonpenetrating trauma to the chest. Of the remaining acquired lung hernias, 29 percent are spontaneous and only 1 percent are pathologic which are secondary to tuberculosis, nontuberculous inflammation or neoplastic disease of the chest wall and pleura.

Spontaneous hernia results from prolonged continuous or excessive increase in intrathoracic pressure and strain which forces a portion of the lung through a relatively weak area in the chest wall. Our case exemplifies an unusual mechanism of intercostal muscle disruption, evolution of lung herniation and entration of the diaphragm.

CASE REPORT

A 58-year-old foundry man felt sudden sharp exacerbating pain in the right side of the chest following clockwise rotation of his body. He subsequently developed right upper abdominal pain and coughing spells.

Physical examination revealed a very obese, 250 lb white man with a blood pressure of 120/80 mm Hg and pulse rate of 100/min. Ecchymosis and erythema were noted in the right lower chest and upper abdomen, anteriorly. A crepitant mass 8 × 6 cm was palpable at the right seventh intercostal space, with marked separation of the seventh and eighth ribs and costochondral junction of the eighth rib.

A posteroanterior chest roentgenogram was unremarkable. Chest fluoroscopy demonstrated herniation of the anterior basal segment of the right lower lobe through the separated seventh and eighth ribs. Spot films taken in the oblique view confirmed this finding (Fig 1).

The patient was given analgesics for pain and advised to reduce weight. He lost 20 lb, but pain persisted.

He subsequently underwent right thoracotomy, with repair of the lung hernia. The seventh intercostal space was widely separated, anteriorly and laterally, with disruption of the intercostal muscles up to the eighth costochondral junction. An oval defect measuring 8 × 5 cm contained the anterior basal segment of the right lower lobe, which gaped back and forth with respiration. The hernial sac was covered with...
pleura. It was also noted that the anterior attachment of the muscular diaphragm to the seventh and eighth ribs was disrupted, and contiguous localized diaphragmatic eventration measuring 10 × 8 cm was present.

The diaphragm was repaired by imbricating the diaphragm with silk sutures and attaching this to the anterior ribs. The herniated lung was reduced and the sac excised. Periosteal flaps developed from the seventh and eighth ribs were then approximated with Tevdek sutures to cover the defect. Marlex mesh was used to reinforce the periosteal flaps. In addition, the seventh and eighth ribs were approximated with three Teflon bands. The muscle, subcutaneous tissue and skin were closed in layers after a chest tube and subcutaneous Hemovac drains were in position.

The postoperative course was uneventful. One year follow-up showed no recurrence of the hernia as shown by a comparative spot roentgenogram of the right chest taken during fluoroscopy (Fig 2).

**COMMENT**

The development of the spontaneous lung hernia involves two factors: firstly, a weakness in the thoracic wall or boundaries of the chest cavity usually occurs in the chest wall anteriorly from the costochondral junction to the sternum because of the absence of the external intercostal muscle, posteriorly from the costal angle to the vertebralca because of the absence of the internal intercostal muscle and superiorly in the cervical area from deficiency of Sibson's fascia, parietal pleura or neck muscles. Secondly, the intrathoracic pressure is abnormally increased as in chronic bronchitis, whooping cough, blowing a musical instrument or straining.

Our case represents spontaneous lung herniation in the right side of the chest following an apparently normal body motion such as clockwise rotation of the trunk. However, in this patient, the maneuver dislocated the eighth costochondral junction with further separation of the seventh and eighth ribs and widening of the interspace, thereby disrupting the intercostal muscles. The anterior diaphragm was similarly subjected to excessive tension, disrupting the muscle from its attachment. The weakness in the intercostal space, together with the increased intrathoracic pressure produced by coughing allowed the lung to protrude beyond its normal confines. Furthermore, the disrupted muscular attachment of the diaphragm, coupled with the negative intrathoracic and increased intra-abdominal pressure produced the eventration of the diaphragm.

Chest wall hernias have been described following costochondral and rib separation and spontaneous rupture of intercostal muscle, and spontaneous rupture of intercostal muscle ascribed to old age, myositis, acute infection,
syphilis, tuberculosis, neoplasm and excessive fatigue rendering the muscle fibers less likely to resist sudden tension.

The diagnosis of lung hernia is evident by the chest wall deformity, palpable orifice, through which a smooth, soft crepitant reducible mass appears under the skin, which changes in size during inspiration and expiration.

The standard posteroanterior roentgenogram of the chest usually fails to demonstrate a pulmonary hernia. Fluoroscopy in the oblique views usually confirms the diagnosis. As in this case, the diagnosis was confirmed only after fluoroscopic examination, visualizing the separated seventh and eighth ribs and the lung gliding back and forth with respiration through a defect at the level of the seventh interspace.

Lung hernia, when asymptomatic, does not need surgical intervention. Lung hernia should be repaired, if it produces constant pain, recurrent infection and, particularly, if the patient’s occupation involves heavy exertional activity.

The current practice of surgical repair is done utilizing available contiguous tissue combined with synthetic plastic material. After the hernial sac was excised, we utilized periosteal flaps developed from the ribs above and below the margins of the hernial orifice as described by Goodman, and Marlex mesh was used according to Grunwald and Knox. Furthermore, we applied reinforcing Teflon bands to approximate the ribs to give more stability to the repair.

REFERENCES
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Cold Injury and Clubbing: A Possible Relationship*

Vijay Soman, M.D.,** and Melvin Hershkowitz, M.D.†

A 40-year-old Negro truck driver with pneumonia showed advanced clubbing of the fingers and toes, unrelated to his acute infection. No reliable hereditary basis could be documented. The chronology of the observed changes suggested that an episode of exposure to severe cold in Korea at age 21 might have been the crucial initiating factor in the development of his clubbing. Rabbits exposed to severe cold develop productive bone changes, and a possible unidentified mechanism could produce similar changes in man. However, the rarity of this response to cold injury remains unexplained in man.

Hippocrates first documented clubbing of the fingers in the fifth century BC, but not until 1890 did Marie¹ and Bamberger² describe its significance as an overt manifestation of underlying systemic disease. Clubbing of the fingers and toes is now known to exist in both hereditary and acquired forms. Fischer et al.,³ in their review, concluded that the fundamental cause, or causes, of clubbing, acquired or hereditary, remains unknown. They suggested that in acquired cases a previously inapparent or dormant genetic factor may become activated under stress to produce overt clubbing.

In this report we describe a patient who might have developed striking clubbing of the fingers and toes on this basis. The possible stressful stimulus, cold injury, has not been described in the literature to date as a factor in the genesis of clubbing.

CASE REPORT

A 40-year-old Negro truck driver came to the Jersey City Medical Center at 10 a.m. on Dec. 2, 1971, complaining of pain in the left side of the chest, chills, fever, and cough for four days. The cough was nonproductive. The pain was confined to the left lower posterior region of the chest, and was aggravated by breathing and coughing. There was no history of weight loss, night sweats, previous illnesses or surgical operations. The patient had not been hospitalized during his civilian life. He had used alcohol moderately for 15 years, usually one or two ounces of Scotch whisky daily. For 20 years he had smoked and inhaled two packs of cigarettes daily, but no cigars or pipe. He took no medications or drugs.

Physical Examination

Physical examination on admission showed an acutely ill man. His temperature was 102°F rectally, the pulse rate 100/min and regular, the blood pressure 140/70 mm Hg, and

*From the Department of Medicine, Jersey City Medical Center and the New Jersey Medical School, Jersey City.
**Chief Resident, Department of Medicine.
†Associate Director, Department of Medicine.

Reprint requests: Dr. Hershkowitz, Jersey City Medical Center, Jersey City 07304

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