


Shin MS, Ho KJ. Diffuse thymic hyperplasia following chemotherapy for nodular sclerosing Hodgkin's disease: an immunologic rebound phenomenon? Cancer 1983; 51:30-3

**Left Lung Asthma***

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The chest radiograph of a woman with asthma showed signs of obstructive emphysema of the left lung. Fiberoptic bronchoscopy excluded obstruction of a large bronchus. The signs disappeared after antiasthmatic treatment, suggesting that they were caused by airway closure. In usual asthma, airway closure affects both lungs; the reason for the unilateral predominance we observed is unknown.

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Bronchial asthma is characterized by widespread airway narrowing that may result in complete airway closure. Usually, airway narrowing and closure are heterogeneously distributed, but both lungs are affected. We report the case of an asthmatic patient who suffered from airway closure predominating in the left lung.

**Case Report**

A 32-year-old white woman with a 17-year history of allergic asthma due to house dust mites was hospitalized because of an exacerbation. She reported daily attacks for the 2 weeks preceding admission and complained of severe dyspnea. Rectal temperature was 38°C; respiratory rate was 18/min; heart rate was 90 beats per minute; and systolic blood pressure was 120 mm Hg, with a 13-mm Hg paradox. Accessory muscles of inspiration were permanently contracted. Auscultation revealed wheezing in the right hemithorax only; the left hemithorax was silent and hyperresonant to percussion. Pneumothorax was suspected, and chest radiographs were obtained at total lung capacity (TLC) and residual volume (RV). There was no pneumothorax, but signs of obstructive emphysema were present (Fig. 1). Obstruction of a large bronchus was suspected, and fiberoptic bronchoscopy (Olympus, 6-mm external diameter) was performed. All visible airways were patent; there was no mucous plug, foreign body, or tumor. Values for peak expiratory flow rate (PEFR) were 150 L/min and 190 L/min before and after nebulization of albuterol (5 mg), respectively. Further treatment included nebulized albuterol (5 mg four times daily), theophylline (10 mg/kg of body weight per day orally), and prednisolone (40 mg/day orally). Twenty-four hours after admission, PEFR values were 300 L/min before albuterol nebulization and 380 L/min after. Auscultation and radiographs at TLC and RV showed that the signs of obstructive emphysema had disappeared.

**DISCUSSION**

Chest radiographic abnormalities are uncommon in asthma, and when present, they differ from the one we observed. In our patient the radiographic appearance (obstructive emphysema) suggested obstruction of a large bronchus; however, endoscopic examination showed that bronchi within the reach of the fiberoptic bronchoscope were patent.

Obstructive emphysema is often explained by a check-valve (or ball-valve) mechanism. Air would then enter the lung during inspiration but would not flow out during expiration. As a result, alveoli would become more and more distended and possibly rupture; also, the lung territory corresponding to the airway with the check valve would be expected to be overdistended at both RV and TLC. In our case, inflation of the two lungs differed at RV but was similar at TLC (Fig 1). Thus, rather than a check-valve mechanism, it is likely that the radiographic aspects were caused by airways of the left lung opening and closing at a given lung volume. Air could enter the left lung during inspiration when the opening volume was reached and flow out during expiration until the patient had returned to the closing volume; the airways would then close, preventing the left lung from deflating to its RV with subsequent overdistention and mediastinal shift visible on the radiograph at RV.

Airway caliber depends on the transmural pressure, which, in static or near static (no or low airflow) conditions, is the transpulmonary pressure (P<sub>PR</sub>). The P<sub>PR</sub> is related to pulmonary volume, increasing as volume increases. In normal subjects, airway closure occurs in small airways situated in the dependent regions of the lung, at a volume below functional residual capacity, when P<sub>PR</sub> becomes negative (pressure inside the pleural space exceeds pressure within the lung). In asthmatic patients, airway closure can occur in larger airways, at much higher lung volume, in nondependent regions and at positive P<sub>PR</sub>. In our patient the site of airway closure must have been in the airways not seen through the fiberoptic bronchoscope, ie, airways less than 6 mm in internal diameter. Since the whole left lung appeared overinflated, a great number of airways must have closed during expiration. The absence of breathing sounds at left lung auscultation indicates that the left lung airways...
were closed during tidal breathing. The critical closing-opening volume must therefore have been situated above
the end-tidal inspiratory volume but below TLC. Indeed, if
airways had remained closed at TLC, as occurs in some
asthmatic attacks, opening briefly only when the strong
inspiratory efforts make inspiratory pleural pressure during
tidal breathing greater than static P1, at TLC, a true check-
valve mechanism would have been created, with subsequent
left lung overdistention at TLC.

Usually, airway obstruction due to narrowing or closure
affects both lungs.1 Unilateral bronchospasm has been ob-
served in one patient undergoing pleurodesis for recurrent
pneumothorax;2 a bronchoconstrictor reflex triggered by
pleural stimulation was hypothesized. In our patient, unilat-
eral airway closure was probably caused by the pathologic
processes (bronchospasm; airway wall inflammation; secre-
tions) which provoke asthmatic symptoms in usual patients,
since usual therapy (bronchodilators; corticosteroids) cor-
rected the disorder. Why did these abnormalities predomi-
nate within the left lung? Predominant distribution of
airborne allergens into the left lung may be speculated.
Because inspired air is preferentially distributed into the
dependent lung during tidal breathing and with the double
assumption that our patient was lying preferentially on her
left side when asleep and that she inhaled house dust mite
allergens (present in large quantities in mattresses3), one
can explain a side difference in allergen penetration. It was
not possible to ask the patient about her sleeping habits
because she had been discharged when this hypothesis was
raised. Alternatively, the left lung airways may have been
more reactive to inflammatory mediators than the right
ones. With the mechanisms of airway hyperreactivity re-
mainingly largely unknown, so are the possible reasons for
such a difference.

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