DISCUSSION

Most of the reported cases of gastric rupture associated with oxygen administration have occurred in patients whose nasopharyngeal catheters have slipped beyond the glottis allowing the direct insufflation of oxygen into the gastrointestinal tract. In such cases, the rate of gas entry into the stomach is greater than its egress through the pylorus so that unusually high intra-gastric pressures arise. In infant cadavers the pressure causing rupture has varied from 55 to 320 (mean 117) mm Hg, while in adults 120 to 150 mm Hg is required. This corresponds to a 4L volume in the adult. The majority of adult ruptures have occurred on the lesser curvature, presumably because the decreased number of mucosal folds in this location limits distensibility. Neonatal gastric rupture occurs in a more widespread distribution (being most numerous on the greater curvature) and is probably multi-factorial in origin with gastric mucosal ischemia the primary event with gaseous distension, acidity, and direct trauma contributing to produce the end result.

Gastric rupture may also occur in association with oxygen administration in the absence of a direct route of entry into the gastrointestinal tract. This has been described with both CPAP delivered by face-mask and mouth-to-mouth resuscitation. In such cases, sufficient gaseous pressure develops at the posterior pharynx to be deflected into the esophagus and stomach. While normally more than 25 cm water pressure is required to cause gastric distension, lesser pressures may suffice in the presence of airway obstruction. These pressures can be developed with either mouth-to-mouth resuscitation or manual resuscitation bags. When applied in the presence of severe laryngeal edema, as in our case, this could easily lead to simultaneous but preferential gastric inflation and subsequent rupture. Closed cardiac massage would increase the likelihood of rupture by increasing intra-gastric pressure from without, and by predisposing to gastric lacerations through which rupture could occur.

Gastric rupture remains a rare complication of the administration of oxygen. When using nasopharyngeal catheters, extreme caution in their placement is mandatory. Likewise, adequate gastrointestinal decompression must be insured in the presence of any form of positive-pressure ventilation. Chest wall excursions should also be monitored to ensure adequate air exchange. Gastric rupture must be differentiated from pneumoperitoneum resulting from barotrauma which is usually accompanied by and follows mediastinal and/or subcutaneous emphysema and/or pneumothorax. Contrast studies and peritoneal lavage, with or without instillation of methylene blue, may at times be necessary to assist in the differential diagnosis since gastric rupture is a surgical emergency with an 80 percent mortality rate.

REFERENCES


Unilateral Hyperlucent Lung*

Patent Ductus Arteriosus Coexisting with Bronchial Carcinoid

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We report an unusual case of patent ductus arteriosus (PDA) co-existing with bronchial carcinoid. The initial radiographic presentation was hyperlucent lung. Causes and possible mechanisms of unilateral hypoperfusion of the lung are reviewed briefly. Useful diagnostic modalities in evaluating a hyperlucent lung are also discussed.

Garfunkel and Kirkpatrick described unilateral hyperlucent left lung as one of the specific radiographic signs of PDA in 1963. However, Whitley et al observed similar findings in their patients with

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ventricular or atrial septal defects. None of their patients was found to have an occlusive endobronchial lesion as a contributing cause of unilateral hyperlucent left lung. We report a case of PDA and coexisting endobronchial carcinoid who presented with a hyperlucent left lung.

**Case Reports**

A 48-year-old white man was referred to St. Luke's Hospital Center for further evaluation of the presence of a heart murmur known since he was six years old. Eight months prior to admission, he was seen by his family physician because of episodes of dyspnea on exertion, cough and hemoptysis. A chest roentgenogram and a perfusion lung scan showed a hyperlucency and marked hypoperfusion of the left lung. He was diagnosed as having congestive heart failure secondary to congenital heart disease. The question of pulmonary emboli of the left lung was raised. He was given diuretics and digitalis and experienced improvement. Physical examination on admission at our institution revealed blood pressure 120/80 mm Hg, pulse rate 84 and regular, temperature 99°F (37.2°C) and a respiration rate of 20. Positive findings included strikingly decreased breath sounds over the entire left thorax, a grade 2/6 systolic murmur audible in second left interspace, and a grade 2/6 early diastolic murmur. An ECG showed left ventricular hypertrophy. A chest roentgenogram (Fig 1) showed diffuse hyperlucency without overinflation of the left lung and moderately increased pulmonary vascularity of the right lung. The heart size was normal. All laboratory data were within normal limits. Supravalvular thoracic aortography was performed. It confirmed the presence of a patent ductus arteriosus. Right heart catheterization showed normal pulmonary arterial pressures and no pressure gradients. Pulmonary angiogram showed marked dilatation of the main and central right pulmonary arteries and only minimal perfusion of the left lung. A selective left pulmonary angiogram (Fig 2) showed paucity and decrease in size of pulmonary arteries. Surgical ligation of
the ductus was recommended, but was postponed by the patient for personal reasons. He was readmitted to our hospital approximately nine months later because of recurrence of cough and chest pain. A chest roentgenogram (Fig 3) demonstrated complete collapse of the left lung with marked narrowing of the distal left mainstem bronchus. Bronchoscopic examination and biopsies of the left mainstem bronchus noted a lobulated mass which proved to be a carcinoid. At thoracotomy, repair of the ductus and a left pneumonectomy were performed. At surgery, a coarctation of the left pulmonary artery was also noted. The patient’s postoperative course was uneventful.

**DISCUSSION**

Mechanisms that may have contributed to unilateral hypoperfusion of the lung in this patient include bronchial obstruction by neoplasm, congenital heart disease, and coarctation of the left pulmonary artery. Unilateral hypoperfusion of the lung occurs infrequently. During a one-year period White et al. found only 13 cases of unilateral hypoperfusion from their study of 607 lung scans. Bronchogenic carcinoma is the most common cause of unilateral hypoperfusion. Less common causes are thromboembolic disease, severe parenchymal or pleural disease, congenital heart disease and after pneumonectomy. Rare causes include Swoyer-James syndrome, congenital vascular lesions, pulmonary veno-occlusive disease, endobronchial foreign body and bronchial adenoma.

The pathogenesis of unilateral hypoperfusion of a lung in PDA and in other forms of congenital heart disease is unclear. Since it was of only mild severity in this case, the coarctation probably contributed little to the observed hemodynamic abnormalities.

Endobronchial obstruction can cause unilateral hyperlucency by two mechanisms. Overdistention of the lung parenchyma distal to endobronchial tumors has been attributed to partial obstruction by a check-valve mechanism. In our case this mechanism is unlikely because the volume of the affected lung was decreased (Fig 1). With total bronchial occlusion unilateral hypoperfusion may be due to reflex pulmonary vasoconstriction secondary to alveolar hypoxia. Chaudhuri et al. and McGuinness et al. reported two cases of bronchial adenoma presenting with hypoperfusion by lung scan. None of their patients had associated congenital heart disease. To our knowledge, this is the first case of PDA coexisting with endobronchial carcinoid who presented with a unilateral hyperlucent lung on a chest roentgenogram.

Bronchial adenomas have been termed benign, potentially malignant tumors, and represent 5 to 10 percent of all pulmonary neoplasms. Carcinoid tumors comprise 84 to 91 percent of bronchial adenomas of the lung. In six histologically-proved cases of bronchial carcinoids seen over a ten-year period at our institution, protracted cough, hemoptysis and chest pain were the chief but nonspecific clinical symptoms. They presented as a peripheral nodule or central hilar mass with or without calcification or complete lobar collapse on a chest x-ray film. However, none of our previous six patients manifested as a unilateral hyperlucent lung. Our experience is similar to that of other large series of patients. Conventional linear mediastinal and hilar tomography is a useful and noninvasive modality in evaluating a central endobronchial obstructive lesion. It can delineate a sharply demarcated mass within an air-containing bronchus or demonstrate a narrowed bronchus. Templeton et al. stressed the important role of bronchography in detecting bronchial adenomas, and described the classic sign of outward flaring of the involved bronchus just proximal to the point of occlusion by adenoma. Since bronchial adenoma is a vascular tumor, the unique information provided by bronchography or tomography is invaluable prior to the transbronchial biopsy, in that appropriate precautions and preparations could be taken to avoid or treat possible life-threatening massive bleeding during biopsy.

The prognosis for bronchial adenoma is usually good following surgical resection if the diagnosis can be made early. We would recommend inspiration and expiration chest x-ray films, tomography, lung scans, bronchography, bronchoscopy and pulmonary angiography in evaluating a hyperlucent lung besides physical examination.

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**REFERENCES**

1 Garfunkel JM, Kirkpatrick JA: Decreased vascularity of left lung and unequal aeration of the lungs as a manifestation of patent ductus arteriosus. Am J Roentgen 89:1012-1016, 1963
2 Whitley JE, Rudhe UFL, Herzenberg H: Decreased left lung vascularity in congenital left to right shunts. ACTA Radiologica 1:1125-1131, 1963
6 Fraser RG: Chest refresher course, N.Y. Spring Conference, New York Roentgen Society, New York, April 28, 1978