Obstructive Emphysema in Pneumonia Simulating Cavity*

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Introduction

The occurrence of obstructive emphysema complicating various forms of pneumonia has been known for many years. Prior to the era of the sulfonamides and the antibiotics large intra-pulmonary bullae and subpleural blebs were frequently noted by pathologists in children dying of pneumonia. Snow and Casassa1 presented the radiological and clinical picture of this condition in 1937. The occurrence of ring shadows in resolving lobar pneumonia simulating single or multiple lung abscess has also been recorded (Kerley,4 Pierce and Dirkse,3 Graeser, Wu and Robertson2). A number of authors reported diffuse obstructive emphysema as a prominent pathological finding in influenzal pneumonia during the pandemic of 1918-19 (Wolbach,6 Torrey and Grosh6). For the most part, however, references in the literature to this condition in adults are brief and its importance from the diagnostic and therapeutic standpoint is not amplified. Accordingly it seemed worthwhile to report two cases of localized acute obstructive emphysema or pneumatocele occurring in association with pneumonia and simulating cavitary lung disease.

Case 1: A 59 year old white male garment cutter, was admitted on July 14, 1951, because of hemoptyis. In 1938, 13 years before entry, he had an episode of hemoptyis which lasted for about two days. Two weeks prior to admission he developed a chest cold with cough and purulent expectoration. A chest x-ray film taken then was negative. Symptoms persisted and fluoroscopy by his physician one week before admission is said to have shown a hazy infiltrate in the left infra-clavicular area. He was placed on bed rest at home and two or three days later he suddenly coughed up between a quarter and a half cup of bright red blood. Cough productive of purulent and brownish sputum continued, and a chest x-ray film taken three days before admission, revealed a giant ring shadow surrounded by a zone of hazy infiltration in the left apex and infra-clavicular area. He denied chest pain, fever, chills, weight loss, dyspnea or anorexia.

His family stated that he had been hospitalized at age 18 for pulmonary tuberculosis but he denied this. In 1938, he had an episode of hemoptyis which lasted for about two days. A chest x-ray film was negative at that time.

Physical examination disclosed a patient who was apprehensive, but did not appear to be ill. He appeared older than his stated age. There was impairment of resonance over the left chest posteriorly with a definite area of tubular breathing with bronchophony and increased pectoriloquy approximately six cm. in diameter below and medial to the angle to the scapula. A few post-tussive rales were heard in this area. A grade one aortic systolic murmur was audible over the aortic area, and the aortic second sound was louder than the pulmonic second sound. The blood pressure was 180 systolic, 100 diastolic, pulse rate 100 per minute, respiratory rate 20 per minute and temperature was 98.6° F. The remainder of the physical examination was non-contributory.

Urinalysis revealed a pH of 5.0, specific gravity 1.025, 2+ albumin 1+ sugar and no acetone. There were 2-5 white blood cells, an occasional red-blood cell and no casts in the urinary sediment. Examination of the blood revealed a hemoglobin of 14.2

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grams, red blood cell count 4,500,000, per cubic mm., hematocrit 45 per cent. The sedimentation rate (Wintrobe) ranged between 24 and 32 mm., in one hour. White blood cell count was 13,400 per cubic mm. on admission with 80 per cent polymorphonuclear leucocytes. Four days after admission the white blood cell count was 13,900 per cubic mm., with 70 per cent polymorphonuclear leucocytes and 11 per cent non-segmented polymorphonuclear neutrophils. The white blood cell count was within normal limits after the 13th hospital day. Fasting blood glucose was 192 mgm., urea nitrogen 23.8 mgm., creatinine 1.4 mgm., per 100 cc. Total protein was 7.0 gm., with albumin of 2.3 grams and globulin of 4.7 gm. per 100 cc. Creatinine clearance was within normal limits. Five sputa and one bronchoscopic specimen were negative for tumor cells on examination by the Papanicolaou method. One bronchoscopic smear, two gastric lavage cultures, and three sputum smears and cultures were negative for tubercle bacilli. An electrocardiogram revealed non-specific changes. Skin tests with blastomycin, coccidioidin and histoplasmin were negative. The tuberculin test was positive using purified protein derivative second strength.

X-ray film inspection of the chest on admission revealed a ring shadow in the left upper lobe measuring 12 cm. in maximum diameter. It extended from the extreme apex to the third anterior interspace (fig. 2). There was a thin zone of infiltration around this ring shadow. A lateral film showed the lesion to lie in the axillary portions of the anterior and apical posterior segments of the upper lobe. There was no evidence of fluid level. Chest x-ray films taken June 28, 1956, and July 2, 1961, 13 years and 12 days (figure 1) before entry respectively revealed normal lung fields.

He was placed on modified bed rest. The diabetes was readily controlled by means of a diet of carbohydrates 200 gm., protein 150 grams, and fat 100 grams, and thirty units of NPH insulin daily.

On the third hospital day the patient developed pain in the left popliteal region. This was followed in 48 hours by slight swelling and bluish discoloration of the left calf. The lesser saphenous vein was palpable as a cord just below the popliteal space.

A diagnosis of thrombophlebitis was made. Signs and symptoms subsided in several days on treatment with bed rest and an elastic bandage. Bronchoscopy on the fifth hospital day revealed only moderate hyperemia of the left upper lobe bronchus. A biopsy taken from deep in the left upper lobe bronchus revealed chronic inflammation in the respiratory mucosa with no evidence of tumor.

On the ninth hospital day he was started on 400,000 units of procaine penicillin G intramuscularly daily. All physical findings had disappeared by the 10th hospital day. Serial x-ray film (fig. 3) revealed progressive decrease in the size of the ring shadow in the left upper lobe and development first of single and then of multiple fluid levels. He was discharged on the 25th hospital day, but continued aerosol penicillin at home under the direction of his private physician. Chest x-ray films taken six weeks after discharge from the hospital (fig. 4) showed complete resolution of the lesion with only some residual pleural thickening and a linear density in the left aperture.

**Figure 1** (Case 1): Chest roentgenogram taken July 2, 1951, twelve days prior to admission to hospital. There is no evidence of pulmonary infiltration. **Figure 2** (Case 1): Chest roentgenogram taken July 16, 1951, two days after admission to hospital. There is a ring shadow extending from the left apex to the 3rd left anterior interspace. A moderate amount of parenchymal infiltration and pleural reaction surrounds the lesion.
first anterior interspace. Continued follow-up through October 1953, showed no change in these shadows.

Case 2: A 70 year old white male physician was admitted on August 27, 1951, because of a pulmonary lesion in the right upper lobe. He had been in excellent health until two weeks prior to admission when he developed slight discomfort in the right lateral chest, aggravated slightly by deep inspiration. This was associated with low grade fever. The chest pain subsided within four to five days but the fever persisted. A chest x-ray film was therefore taken and disclosed a ring shadow in the right upper lobe. He was accordingly referred for admission. He denied cough, production of sputum or hemoptysis, and stated that there was no weight loss.

Fifteen years before entry, he had an episode of right sided pleuritis associated with cough and lasting about two weeks. The remainder of the history was non-contributory.

Physical examination revealed him to be in no distress. The chest moved symmetrically and was normally resonant on percussion. Transient crepitant rales and medium and high pitched rhonchi were audible over the right apex posteriorly. The heart was not enlarged on percussion and the aortic second sound was equal to the pulmonic second sound. The rhythm was regular with occasional extrasystoles.

Blood pressure was 150 systolic, 80 diastolic, pulse rate 86, respiratory rate 20 and temperature 99.8°F. rectally.

Urinalysis showed specific gravity of 1.011; no albumin or sugar and 1-3 hyaline and 0-2 finely granular casts per high power field. Examination of the blood showed hemoglobin of 13.5 gm. per 100 cc., red cell count 4,380,000 per cubic mm. hematocrit 45 per cent, 65 per cent polymorphonuclear leucocytes, 10 per cent non-segmenting polymorphonuclears, 2 per cent eosinophiles, 17 per cent lymphocytes and 6 per cent monocytes. The sedimentation rate was 36 mm. in one hour (Wintrobe). Liver profile was within normal limits except for a total protein of 7.65 gms., and albumin of 3.2 gms., and globulin of 4.45 gms. per 100 cc. Kahn test was negative, blood urea nitrogen was 24.7 mgm. and blood glucose was 72 mgm. per 100 cc. Three gastric lavage cultures and one bronchial lavage culture were negative for tubercle bacilli. A bronchoscopic aspiration specimen was negative for secondary organisms, acid-fast bacilli and fungi. Two sputum specimens examined by the Papanicolaou technique were negative for tumor cells. Skin tests with blastomycin, histoplasmin and coccidioidin were negative; tuberculin test was positive to second strength PPD. An electrocardiogram was interpreted as showing non-specific changes.

A chest x-ray film taken three months before admission showed no abnormality of the lungs (fig. 6). One taken a few days before admission (fig. 6) disclosed a 5.5 x 4.5 cm. ring shadow in the periphery of the right infraclavicular area. This shadow

Figure 3 (Case 1): Detail of chest roentgenogram taken on August 2, 1951, the 19th hospital day. The ring shadow is smaller and shows an air-fluid level.—Figure 4 (Case 1): Detail of chest roentgenogram taken on September 20, 1951. A nodular density in the left 1st interspace is all that remains of the inflammatory process.
was surrounded by a 1 cm. zone of infiltration continuous above with a homogeneous ground glass density in the outer half of the right first anterior interspace. The aorta was slightly tortuous. Fluoroscopic and roentgenographic examination of the chest on admission showed a decrease in the size of the ring shadow to 2 x 4 cm. and resolution of the pulmonary infiltration. The ring shadow was in the posterior segment of the upper lobe. Because of rectal bleeding, barium and air contrast enemas were done and reported negative.

Bronchoscopy one week after admission revealed narrowing of the right lower lobe bronchus to about 5 mm. in diameter. The right upper lobe orifice showed hyperemia most marked on its lateral wall. A biopsy from the right upper lobe bronchus was reported as showing acute and chronic inflammation of respiratory mucosa.

During the period of investigation the only therapy was modified bed rest. On the 13th hospital day he was started on aerosol therapy consisting of 0.3 cc. of 0.25 per cent Neo-synephrine in saline, followed by 50,000 units of penicillin dissolved in 1 cc. of saline given four times daily. This medication was continued until discharge on the 30th hospital day. The patient remained afebrile throughout the entire hospital course except during the third week of hospitalization. During the third week the patient developed lowgrade fever in association with superficial thrombophlebitis of the left leg. This followed sigmoidoscopic fulguration of an incidentally discovered benign rectal polyp. He was given anticoagulant therapy with heparin and dicoumarol and recovered uneventfully. Serial fluoroscopy and chest x-ray films (fig. 7) revealed progressive decrease in the size of the ring shadow in the right upper lobe, and clearing of the surrounding pneumonitis. At the time of discharge from the hospital a 2 x 1 cm., ring shadow was still present. An x-ray film taken on November 11, 1951, (fig. 8) five weeks after discharge from hospital revealed complete disappearance of the ring shadow with a residual nodular density in the right infraclavicular area. This remained unchanged in serial x-ray film studies through October 1953.

Discussion

These two patients present a markedly similar clinical picture. Both had an antecedent history of pulmonary disease, one of hemoptysis 13 years before entry, the other of pleurisy 15 years before. Both had negative chest x-ray films taken two weeks and three months, respectively, prior to hospitalization. In both there was a history of acute lower respiratory infection of two weeks' duration with subsidence of most of the symptoms despite the appearance and persistence of a ring shadow in the chest roentgenogram. In both a diagnosis of tuberculosis or lung abscess distal to a

**FIGURE 5**

*Figure 5 (Case 2):* Chest roentgenogram taken July 2, 1951, three months prior to hospital admission. There is no evidence of pulmonary infiltration.  

**FIGURE 6**

*Figure 6 (Case 2):* Chest roentgenogram taken August 21, 1951, three days prior to hospital admission. There is a 5 cm. ring shadow in the right infraclavicular area surrounded by a moderate amount of parenchymal and pleural inflammatory reaction.
bronchiogenic carcinoma was seriously considered. These were effectively ruled out. In both there was bronchoscopic and histological evidence of acute and chronic inflammation in the mucous membrane of the bronchus draining the diseased portion of the lung. In case two there was evidence of old bronchial disease in the form of bronchial stenosis of the right lower lobe bronchus. In both, changes in the size of the ring shadows were rapid, although healing of the lesions was not complete for about two months.

Many of the early authors believed that emphysema occurring with pneumonia was on a compensatory basis. Holt, Torrey and Grosh⁴ and MacCallum⁵ called attention to emphysema and atelectasis in influenza pneumonia and gave his opinion that they were due to partial or complete bronchial obstruction. It was not, however, until the studies of Jackson, Manges and Spencer⁹ in the early 1920's on the changes brought about by aspirated foreign bodies, that a clear understanding of the mechanisms involved was elucidated. Pierce and Dirkse,³ in 1937, clearly described the development of pulmonary pneumatoceles as a complication of lobar or lobular pneumonia. They ascribed it to a persistent check valve effect in the bronchus due either to non-resolution of the initial inflammation of the bronchus or a subsequent distortion by the dilated airspaces. In only one of the four cases they presented did the pseudocyst disappear, although the surrounding parenchymal infiltration cleared almost completely. They, too, noted the lack of symptoms following the resolution of the initial infection.

It has been our experience that in lobar pneumonias, the bronchial tree is not commonly involved in the infectious process. However, in broncho-

**FIGURE 7**

*Figure 7 (Case 2):* Detail of chest roentgenogram taken September 17, 1951, on the 20th hospital day. The ring shadow is now much smaller.

**FIGURE 8**

*Figure 8 (Case 2):* Detail of chest roentgenogram taken November 11, 1951. A linear density at the level of the right 2nd anterior rib is all that remains to mark the site of the original process.
pneumonias of bacterial origin we have been impressed with the high incidence of bronchial inflammation. This bronchitis may be a primary process or may be engrafted on an old bronchial abnormality. Studies on viral pneumonia have also indicated a high incidence of bronchial involvement. The bronchial factor in pneumonia may affect the course and resolution of the infectious process resulting in atelectasis, delayed resolution, pulmonary carnification or obstructive emphysema.

The exact etiology of the pneumonia in our cases is not clear; symptoms subsided in both cases prior to admission, without antibiotic therapy. The absence of any predominant pathogen in the sputum might be taken as pointing to viral pneumonia. Suffice it to say that in both cases there was inflammatory involvement of the bronchial mucosa with congestion and edema. This either alone, or in combination with secretion, produced a check valve effect with entrapment of air beyond the point of obstruction and formation of a pseudocyst. One can only speculate as to why the check valve was formed in the given site. The localization may have been due to old pulmonary disease causing anatomical distortion of the bronchus beyond the limits of bronchoscopic visualization. Or, there may have been a functional abnormality or bronchial cleansing, such as abnormal ciliary action, excessive secretion of rigidity of the bronchial wall. In these cases secondary deformity of the bronchus from the pseudocyst appeared to play little or no part.

There is little exact information available as to the age incidence of this condition. While cases have been reported at all ages, most of them have been in children. This is as might be expected, because of the narrower caliber of the airways in children and the consequent greater ease with which partial obstruction is produced. The occurrence of this condition in these two adult patients may have been related to the known decrease in effectiveness of cough in elderly individuals as well as the possible abnormalities of bronchial function described above.

While the possibility that these lesions were non-putrid lung abscesses cannot be entirely excluded, the early disappearance of symptoms, the small quantity of sputum, the rapid decrease in size of the cyst-like lesions and bronchoscopic findings all militate against it. A similar picture could have resulted from inflammation of the wall of a previously roentgenologically invisible thin walled cyst. Even if this were true, bronchogenic infection and a check valve mechanism must surely have played an important role.

The occurrence of venous thrombosis in the legs of both of these patients raises the question of whether we were dealing with excavation of bland pulmonary infarcts. A review of the literature on this subject discloses differing opinions on the incidence of this complication of pulmonary infarction. Chester and Krause report on 11 instances of abscess formation in 174 subjects with infarcts studied at autopsy. Levin et al report 23 cases of abscess secondary to infarct in 550 necropsies in which pulmonary infarction was a finding. Soucheray and O’Loughlin report the finding of 4 cases of apparent cavitation occurring in serial roentgenograms in a clinical study of 99 cases of pulmonary infarction. None of
their cases were proven pathologically. Cocchi\textsuperscript{13} reports infarct cavities occurring 51 times in 385 cases of pulmonary infarction studied at autopsy. The latter author states that the upper portions of the lungs are preferred sites for this complication. The other authors quoted all state that the abscesses have the same distribution in the lungs as uncomplicated infarcts and most commonly involve the lower lung fields. Secondary bronchogenic infection is stressed by all of these authors as the usual cases of secondary infection and subsequent excavation.

In contrast to these reports, Fishberg\textsuperscript{14} states that cavity formation due to aseptic necrosis or secondary infection is a very rare complication of pulmonary infarction, Castlemen\textsuperscript{15} has stated that secondary infection of a bland pulmonary infarct is a rare coincidence.

Against the diagnosis of cavity in a pulmonary infarct in our cases is the fact that venous thrombosis did not manifest itself until well after hospitalization in both patients. In Case 1 the thrombosis followed a minor surgical procedure on the rectum; in Case 2 the thrombosis was in a superficial vein which only rarely gives rise to pulmonary emboli. The lesions were in the upper lobes in both cases and there was no hemoptysis in Case 2. Finally, on reviewing the literature, we are impressed that this complication of pulmonary infarction has occurred primarily in patients with advanced cardiac disease.

Tuberculosis was dismissed early as the likely diagnosis in the cases here reported, because of the failure to demonstrate acid fast bacilli in the concentrated sputum or in the bronchoscopic smears in the presence of large ring shadows; negative cultures eventually confirmed this approach. Carcinoma was excluded by many negative cytologic examinations of the sputum and bronchoscopic biopsy.

While many of these pneumatoceles will disappear spontaneously with resolution of the bronchial lesion, the course can be hastened and the development of occasional permanent cysts can be prevented by vigorous therapy of the bronchial inflammation. In addition to systemically administered antibiotics, they should be given by inhalation as an aerosol. This is a much more effective way of reaching superficial infection engrafted upon edematous engorged bronchial mucosa.\textsuperscript{16} Bronchodilator and vasoconstrictor drugs such as phenylephrine hydrochloride or nor-isopropyl epinephrine should also be used by the inhalation route. In some instances aminophylline or other bronchodilator drugs should be given systemically. Expectorants, carbon dioxide and steam inhalations for thinning secretion also have a place in selected cases.

**SUMMARY**

Localized obstructive emphysema complicating pneumonia and resulting in pseudocysts or pneumatoceles is reported in two male adults. The literature is reviewed and the differential diagnosis discussed. The role of partial bronchial obstruction in producing the condition and the importance of directing therapy to the diseased bronchus are emphasized.
SUMARIO

Se reportan dos casos de neumonía en hombres adultos complicada con enfisema localizado obstructivo que evolucionaron a pseudoquistes o pneumatocélulas. Se revisa la literatura y se discute el diagnóstico diferencial. Se hace énfasis en el papel de la obstrucción bronquial parcial como causa del padecimiento y la importancia de dirigir la terapéutica al bronquio enfermo.

RESUME

Les auteurs rapportent deux observations d'adultes du sexe masculin qui, à la suite d'une pneumonie, furent atteints d'emphysème obstructif localisé, ce qui amena la formation de pseudokystes ou de pneumatocèles. Ils donnent à cette occasion une revue de la littérature et discutent le diagnostic différentiel. Ils insistent sur l'action d'une obstruction bronchique partielle dans de tels cas, et sur l'importance d'un traitement directement appliqué sur la bronche atteinte.

REFERENCES