Massive Pulmonary Infarction Simulating Carcinoma of the Lung*

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Many inflammatory and other benign lesions of the lung may imitate carcinoma of the lung, both clinically and radiologically. Often thoracotomy, and even pulmonary resection, is necessary to rule out the presence of a malignant lesion. One of the rarer lesions that has been mistaken for carcinoma of the lung is a localized infarct of the lung. Neville and Munz¹ have reported two cases in which a segmental resection and a lobectomy were performed respectively for lung infarcts thought to be carcinoma grossly. Perkins and Bradshaw² also cite two cases in which limited pulmonary resections were performed for localized lesions that were later found to be infarcts of the lung. A similar case has been reported by Smith.³ In such cases as these, resection was deemed necessary only to rule out a more serious pathologic entity, for localized infarcts of the lung can be expected to heal spontaneously.

We are unaware, however, of any reported case in which a massive infarct simulated carcinoma of the lung clinically. We wish to report such a case which was treated successfully by lobectomy.

Case Report

D. F., a 51 year-old portrait painter, entered Stanford University Hospital with a history of a five-week illness beginning with coryza, sore throat, and cough, accompanied by fever ranging between 100°F and 103°F. Two weeks after the onset of this illness, he experienced a sharp pain in his right anterior chest and transient aching in his left calf. In spite of antibiotics, the fever continued, and a chest film demonstrated right pleural effusion. Bronchoscopy and right thoracentesis had been performed at another hospital where 650 cc. of serosanguineous pleural fluid had been

FIGURE 1A: Preoperative posteroanterior chest film showing right pleural effusion and density of right lower lobe. FIGURE 1B: Preoperative lateral chest film showing extent of density in right lower lobe.

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obtained, while bronchoscopy revealed no abnormality. No tumor cells or acid-fast bacilli were found in this effusion. A culture of the pleural effusion grew out Strep-
tococcus fecalis. On the basis of the appearance of the chest films (Fig. 1) and the
serosanguineous nature of the effusion, the referring physician felt that the most
likely diagnosis was carcinoma of the lung with pleural metastases.

On admission to our hospital, physical examination revealed the following: temperature 100° F., pulse 90, and blood pressure 120/74 mm.Hg. Abnormal findings were
limited to the right hemithorax, where a respiratory lag was noted. There was dull-
ness to percussion at the right base posteriorly, with coarse rales and a friction rub.
The legs were not swollen and there were no areas of tenderness. Hematocrit was 43,
and the white cell count was 13,800 with 81 segmented polys. The tuberculin test was
positive and the histoplasmin and coccidioidin skin tests were negative. The serology
was negative.

Four days after entry his temperature rose to 102°F. and the rales in the right
lung increased. The white cell count was 18,000 with 86 polys.

The x-ray film appearance of collapse of the right lower lobe, the bloody pleural
effusion, and the recurrent pneumonitis suggested to us also that the most likely
diagnosis was carcinoma of the lung with extension to the pleura. Thoracotomy was
advised to obtain a tissue diagnosis.

One week after admission, thoracotomy was performed through the bed of the fifth
rib. Heavy, fibrous pleural adhesions were noted, especially around the lower lobe.
One thousand cc. of yellowish, serosanguineous fluid were removed. The lobes were
separated and it was noted that the lower lobe was uniformly firm and liver-like in
consistency. Frozen section of an area of thickened pleura was reported as “chronic
inflammation.” Several hilar nodes were enlarged and appeared inflammatory. The
lower lobe appeared to be diseased, and therefore lobectomy was performed. As the
main lobar artery was divided, it was found to be completely occluded by a thrombus.

The pathologist’s report was as follows: “Gross specimen: greatly thickened pleura.
The most striking feature is the complete occlusion of the main artery of the lobe by
thrombus; the bronchi are clear. Most of the lobe feels diffusely more firm than normal.
Histological examination: the pulmonary artery to this lobe is completely occluded
by a fairly recent thrombus which shows only beginning fibrosis at its periphery. The
lung parenchyma is necrosed over large areas. In others, there is partial to almost
complete collapse with macrophages in most of the alveoli. The pleura is thickened
with fibrosis and heavily infiltrated with acute inflammatory cells. On its surface are
thick strands of fibrin. Diagnosis: embolism, pulmonary artery, (right lower lobe);
infarct, lung (right lower lobe); collapse, lung (right lower lobe).” Cultures of the
pleural fluid were sterile. Cultures of the lung tissue grew only a few Staphylococcus
albus; there was no growth of fungi or tubercle bacilli.

Following surgery a gradual decrease in his fever occurred. On the eighth post-
operative day, he developed thrombophlebitis in the calf. This responded to conserv-
ative measures and he was discharged home on the 13th postoperative day (Fig. 2).

FIGURE 2A

FIGURE 2B

FIGURE 2A: Postoperative chest film showing expansion of remaining right lung
and small amount of residual fluid. FIGURE 2B: Postoperative lateral film. Note the
absence of the previous posterior density.
Three weeks later he was readmitted with recurrent thrombophlebitis in the left leg. Again he was treated conservatively for 10 days, and was discharged as improved. During the next month he was seen in the outpatient clinic, when it was felt that he had recurrent thrombophlebitis. A program of rest, elevation, and support was advised. Subsequently, he was in good health until 18 months later when he committed suicide for causes unrelated to his previous illness.

Discussion

The disregarded history of transient aching in the calf previous to the pleuritic pain, and the protracted and recurrent thrombophlebitis postoperatively make it extremely likely that the leg veins were the source of the embolus. This is in keeping with reports of large series of pulmonary emboli, stating that the veins of the lower extremities are the source of the emboli in from 63% to 86% per cent.

It has been shown both experimentally and clinically that pulmonary embolism in an otherwise healthy lung does not produce an infarct. This has been explained as a result of the complex interrelationship of the three pulmonary vascular systems, namely the pulmonary arteries, the bronchial arteries, and pulmonary veins. Mathes and her co-workers feel that the bronchial arteries in an otherwise healthy lung can nourish pulmonary vasculature beyond an embolus through capillary anastomoses. Ellis et al., however, concluded that the pulmonary artery itself was capable of supplying blood to an area distal to an embolus by capillary anastomoses. This was true, however, only if some part of the pulmonary artery supply to a lobe remained. If emboli were present in a given lobe, then there was no possible way for unoxygenated blood to go around the block. The case reported by Woesner et al. of thrombi occluding the pulmonary arteries to the middle and lower lobes without infarction supports the idea that the bronchial arteries alone can nourish large areas of pulmonary tissue. Ligation of the main pulmonary artery did lead to massive infarction in one reported case, but the author postulated that early onset of infection in the lung was a predisposing factor.

In the absence of heart failure, the factors which predispose to pulmonary infarction are pleural effusion, bronchial obstruction, atelecasis, pneumonia, multiple emboli, and shock. Parin states that the sudden occlusion of a portion of the pulmonary vascular tree may cause peripheral hypotension by a reflex mechanism, and in turn, decrease the bronchial circulation. However, the experimental studies of Hara and Smith have not confirmed this.

One might ask why infarction occurred in our patient. The size of the embolus, occluding all of the pulmonary arterial blood to the right lower lobe would be sufficient explanation if one agreed with Ellis' that the bronchial arteries were probably of little or no importance in the etiology. On the other hand, this does not explain Woesner's case where two lobes were apparently nourished satisfactorily by the bronchial arteries. It has been stated that pleural effusion may predispose to infarction by interference with pulmonary venous drainage. However, pleural effusion often follows infarction, and this was probably the sequence in this case. Bronchial obstruction and atelecasis were not present and thus need not be considered here as etiologic agents. Perhaps the most likely explanation of the infarction is the presence of previous infection in the involved lobe. There is no conclusive evidence, but the onset of pleuritic pain occurred after three weeks of respiratory symptoms. This patient's initial illness was quite likely pneumonitis which, when combined with a pulmonary embolus, resulted in an infarction.

This case demonstrates the often reiterated principle that no matter how hopeless the situation may appear clinically, every effort, including thoracotomy if necessary, should be expended to obtain a tissue diagnosis.

REFERENCES


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EFFECTS OF SODIUM ANTIMONY TARTRATE ON THE MYOCARDIUM

Cardiograms of 59 patients receiving S. A. T. treatment were recorded before and after treatment. They showed reduction in amplitude of T, characteristic upward sloping S-T segments with terminal dipping and later frank T wave inversion, most frequently in the precordial leads V3 to V6 and prolongation of the Q-T duration. These changes are divided into 4 grades of severity: one patient alone showed no change, one showed grade I change, 22 grade II, 18 grade III, and 17 grade IV changes.

The changes are attributed to cumulative antimony in the myocardium producing metabolic and functional changes and possibly actual necrosis. They are regarded as being a sign of impending clinical myocardial damage with potentially serious or lethal consequences.

It is suggested that until S. A. T. is supplanted by a safer but equally effective drug, a wise precaution is to record a cardiogram after the administration of 15-20 grains (0.9-1.2 g.) and modify the course of treatment of severe changes are seen.


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THERAPEUTIC EMPLOYMENT OF PNEUMOPERITONEUM IN CERTAIN NONTUBERCULOUS DISEASES OF THE LUNGS

In clinical studies, the author shows the convincing advantage of utilizing pneumoperitoneum in suppurative diseases and traumatic affections of the lung, complicated by pulmonary hemorrhage. This treatment not only exerted a prompt hemostatic effect, but also was conductive to a clinical and anatomic recovery. A good curative effect of pneumoperitoneum was also observed in suppurative affections of the lungs coupled with tuberculosis.