Unorthodox Clinical and Roentgenological Features of Pulmonary Embolism

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The subject of pulmonary embolism is certainly not occult. No longer is every postoperative pulmonary complication labeled pneumonia, pleurisy, or heart disease. Its frequency is fully appreciated and the surgeon is quick to institute measures to combat its occurrence. From a preventive standpoint, with the recent tendency to ambulate patients early, he is observing how the incidence of embolism decreases.

In the field of medicine, pulmonary embolism is known to occur none too rarely. Autopsy, time and time again, reveals emboli in the pulmonary circuit, even when none was suspected clinically. This is especially true of cardiacs.

In 1929 I had the opportunity to assist at the necropsies performed in the pathology laboratory of Erdheim at the Spital der Stadt Wien. The number of pulmonary emboli routinely observed was considerable. It was the rule in those cases first to incise the calves of the cadaver transversely and explore the lumina of the deep veins. Notwithstanding the absence of any external evidence of venous obstruction in the limb, loose thrombi in these vessels were frequently present. This was the most frequent site of origin of pulmonary emboli. It is unnecessary to point out that thrombi may also be found in the larger and smaller pelvic veins, and also attached to the endocardium of the heart.

Pulmonary embolism is more common in the bed ridden patient. In various articles on abuse of bed rest, it has been stated that there are few conditions which require absolute confinement to bed for any protracted period of time. Even in coronary thrombosis the patient is advised to move his toes and legs as soon as feasible. Comatose patients are not allowed to remain rigidly immobile in any one position. Senile patients are compelled to get out of bed as soon as they can.

This paper will only emphasize the possibility of embolism in any patient. Three cases admitted to the medical service of Metropolitan Hospital and a private patient are presented. Their unorthodox clinical and roentgenological features are worthy of comment. One had become an invalid by many episodes of embolism over several years and had been treated by various doctors for diverse illnesses. The second had been discharged recently from the armed forces and was sent in as a case of pneumonia. Only when this disease did not resolve under antibiotic therapy was the correct diagnosis perceived. The third presented right sided and then left sided pleurisy on subsequent admissions to the hospital. During the first admission, cough with blood expectoration, swelling of the legs, and tenderness of the calves failed to appear, and no one suspected pulmonary embolism. The private patient had had two attacks of so called

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bronchopneumonia, and at the onset of his most recent illness had experienced excruciating precordial pain resembling that of coronary thrombosis.

Review of these cases would suggest that if one expects to find the classical features of embolism, many episodes will be wrongly diagnosed. Since the patient's life may be in jeopardy, this error can be more than of academic interest.

Case 1. J. S. 36 yr. old white male, admitted to hospital on May 4, 1945, with a diagnosis of "toxic" pneumonia. Prior to admission temperature had been 105° F. and he received six gm. of sulfadiazine in 24 hours without favorable response. There was history of pain in the lower left chest aggravated by respiration, cough with expectoration of blood tinged sputum, fever, chills, dyspnea, and severe prostration of one to two days duration. In 1935 there was hospitalization for severe right flank pain and gross hematuria, diagnosed as probable renal calculus. In 1937 he had right sided pleurisy, and this past winter, severe colds. Several days after admission, he also recalled having received injections for varicose veins a few years previously. The history obtained was quite meager because of the extreme toxicity of the patient.

When he was well on the road to recovery, he was recognized on the ward by a physician who had treated him for pleurisy in 1937. Since that incident is relevant, it will be gone into later.

X-ray film of the chest showed mottling of both lungs interpreted as bilateral pneumonia (fig. 1). The heart was enlarged and the pulmonic conus prominent. Electrocardiograph revealed right ventricular strain. The liver was palpable. On auscultation P2 was louder than A2. There was a soft systolic pulmonic murmur. A cardiologist thought that a patent interatrial septum defect was a possibility. Sulfadiazine and penicillin were both administered. On May 8, 1945, sulfas was discontinued. The temperature had ranged from 103-105° F. on admission, and on May 17, 1945, had fallen to 99-100° F. He improved generally, but was still slightly febrile. Penicillin was discontinued on May 17, 1945.

On May 23, 1945, his condition deteriorated with gradual rise of temperature. Cardiac decompensation with bilateral hydrothorax was diagnosed. Digitalis was administered and 250 cc. of sanguinous fluid was removed from the right pleural cavity. For the first time the diagnosis of pulmonary infarct appeared on his chart. On May 29, 1945, cough was productive of bloody sputum. Cyanosis and jaundice with a larger, more tender liver were obvious. Penicillin was resumed. On May 31, 1945, bilateral ligation of the common femoral veins was performed. During the operation his condition became grave and exploration of the lumina of the veins was not attempted. Immediately after the operation there was a spectacular change. Temperature dropped to normal and never rose again. On June 6, 1945, penicillin was discontinued. With the exception of moderate edema of the legs, complete recovery quickly took place. The pathological report of the veins revealed chronic phlebitis.

He left the hospital on July 6, 1945. X-ray film on January 4, 1946 (fig. 2) and fluoroscopy on June 12, 1946 were negative except for lower right pleural adhesions. The heart was somewhat enlarged and the conus prominent. The soft systolic pulmonic murmur remained. He stated that he had been particularly healthy. When seen last, one year ago, his health was excellent.
It is of great interest to review the past history of pleurisy in 1937. At that time he had had slight cough for two weeks with pain in the lower right chest aggravated by respiration. Temperature was 101° F. X-ray film revealed obliteration of the right costophrenic angle by a small amount of fluid (fig. 3). The same soft systolic pulmonic murmur heard in 1945 was present. Fluid rapidly accumulated and almost the entire thoracic cavity was filled. Unfortunately the x-ray films taken at the hospital were destroyed. On the first occasion 1000 cc. of clear yellow fluid was withdrawn, and a few days later 2,200 cc. Specific gravity was 1016, protein 1 per cent, 230 cells per c.mm., most of which were lymphocytes. Culture was negative. Examination for tubercle bacilli, including guinea pig inoculation, was negative. Following paracenteses, fluid did not reaccumulate, and x-ray film studies concluded: "Pleural effusion diminished in extent due to aspiration. Marked thickening of the hilus shadow is now visible. The possibility of a primary bronchogenic carcinoma of the right lower main bronchus is suggested." The pathological examination of the fluid was just as misleading. The diagnosis was, "transudate, anaplastic carcinoma, grade IV." He made a rapid recovery and was followed after he left the hospital. Except for pleural thickening at the right base, x-ray film was negative. There was slight pain in this region. In October 1937 slight enlargement of the heart was detected. On April 29, 1938, x-ray film (fig. 4) indicated complete clearing of the right lung field. On May 19, 1938, there was right axillary pain low down and a rub was heard. Slight temperature of 100° F. was found. On March 20, 1939, there was pain in the left axilla. On January 13, 1940, he stated that a month previously a left inguinal hernia had been repaired. The day after leaving the hospital the same pain appeared in the right chest. X-ray film on January 13, 1940 (fig. 5) revealed clouting at the right base. A pleural rub was heard. Temperature was 100° F. In two weeks complete recovery had taken place. Except for frequency of upper respiratory infections, this man remained fairly well until his illness of 1945.

He continued to be employed as a chauffeur as he had been for many years. Whether this sedentary occupation played any role in this disease may be considered. One other point which may be quickly commented on is that since the cardiac findings suggested a congenital lesion with a small atrial septum defect as the most likely possibility, is it too far fetched to assume that the "renal calculus" in 1935 was an embolus from a leg vein which accidentally traversed the hole to lodge in the right kidney?

Case 2. I. S. 38 yr. old Puerto Rican male, admitted from another hospital on January 10, 1946, with a diagnosis of tuberculous pleural effusion. Temperature was 103° F. The chief complaint was pain in the left chest. The history was subsequently obtained through an interpreter. The patient had been in the air force three years previously for one and a half years, and had been discharged because of a heart murmur. While in the service he had been hospitalized for two months because of swelling and numbness of the feet. Following his discharge he was well for a few months. After taking a job as a superintendent his feet commenced to swell. No dyspnea was present. He remained in bed for two months prior to admission. On a few occasions blood was expectorated. A few days prior to admission he was acutely ill with chest pain, hemoptysis, and fever.
At the hospital there were signs of left pleural effusion. A faint systolic murmur was heard at the apex. The heart was enlarged. The liver was enlarged and tender. There was ascites, edema of both legs, and palpable indurated femoral veins. The various diagnoses considered were tuberculous effusion, pulmonary neoplasm, congestive heart failure, pneumonia, and pulmonary embolism with cardiac decompensation. X-ray films on January 11, 1946 (fig. 6) showed left pleural effusion and enlargement of the heart. Sputum was negative for tubercle bacilli. Electrocardiograph showed right ventricular strain. B. P. was 110/80. The temperature was not normal after the 32nd day, notwithstanding sulfonamide and penicillin therapy. On January 18, 1946, thoracentesis produced 1000 cc. of pinkish fluid. The surgical consultants accepted the case and bilateral femoral venous ligations were performed. X-ray films on January 30, 1946 (fig. 7) showed less effusion and on July 2, 1946 (fig. 8) complete resorption. The heart was enlarged and its left border straight. He made an uneventful recovery.

**Case 3.** A. V. 46 yr. old white chauffeur. Admitted on March 1, 1946, for pain in the right upper quadrant, aggravated by respiration. Temperature was 102° F. X-ray film on March 2, 1946 (fig. 9) showed clouding of the right costophrenic angle. Treatment was with penicillin parenterally. There was clearing on March 8, 1946 (fig. 10). Patient left the hospital the next day. He returned on April 2, 1946, with pain in the left leg. Varicosities of the left leg of many years standing were noted. There had been phlebitis in the past. He signed out April 3, 1946, but returned May 5, 1946, with a temperature of 105° F. He had been having pain in the right chest anteriorly with dyspnea for a few weeks. On May 7, 1946, he experienced pain in the left calf. Electrocardiograph showed S1, inverted T2 and T3, Q3, and right axis deviation, pointing to pulmonary embolus. X-ray film on May 9, 1946 (fig. 11) showed clouding in the region of the right apex. On May 16, 1946, there was pain in the left chest with congestive signs. Bilateral superficial and deep vein ligations were performed. Film of June 3, 1946 (fig. 12) showed clearing, except for residual involvement of the left costo-phrenic sinus. Recovery was uneventful.

**Case 4.** A. J. 45 yr. old white male who suddenly developed pneumonia with classical physical signs in the right upper lobe in May 1940. He was treated at home with sulfapyridine. Seen at my office two weeks later, fluoroscopy revealed a small shadow in the right upper lung field. Six weeks following, x-ray film showed complete clearing. Sputa were negative for tubercle bacilli.

In March 1950 there was recurrence of this pneumatic episode, and on March 3, 1950, x-ray film (fig. 13) showed infiltration at the base of the right upper lobe. Sputa were again negative.

On May 3, 1950, after being completely well, he was seized with pain below the left nipple while going to work. It grew progressively worse and dyspnea appeared. The physician at his place of work administered morphine, and referred him to Park East Hospital with a diagnosis of coronary thrombosis. There pneumonic signs were found anteriorly to the left of the heart. Electrocardiographs taken then and several times since were normal. Posterior anterior and lateral films of the chest (fig. 14 and fig. 15) were taken on May 4, 1950. X-ray film findings were, "peribronchial pneumatic infiltration in the lower half of the left lung adjacent to the cardiac border. The possibility that the pneumonic change may be due to an infarction should also be borne in mind." This is best exemplified by the lateral view (fig. 15) which demonstrates a wedge shaped shadow in the lingular segment. He left the hospital in a few days and has remained completely well. He is a polisher by trade and stands on his feet a good deal. There has never been any evidence of thrombi in the lower extremity.

In reviewing the histories of patients with embolism, one does not necessarily find the text book symptoms of chest pain, cough with bloody expectoration, dyspnea, jaundice, and the local lower extremity findings of venous thrombi. There are many possibilities when an embolus lodges in a pulmonary vessel. Not all emboli cause infarcts since hemorrhagic consolidation will not take place when there is adequate collateral circulation. With large multiple emboli occluding vessels, death from shock can be instantaneous, but should the patient survive, pulmonary pathology still might not be evident in the face of shock and prostration. Then, too, pulmonary hypertension could develop and death supervene from dilatation and failure of the right ventricle. (In cases 1 and 2 cor pulmonale was present.) Therefore, if the pulmonary pathology has not expressed itself,
treatment solely with digitalis for heart failure might erroneously be administered.

Even if one's attention is directed toward obvious lung involvement, the absence of the classical syndrome should not rule out the diagnosis of embolism. Case 1 gave a history of chronic invalidism over many years from right sided pleurisy. The final episode of bilateral "toxic pneumonia" could not be glossed over with dilatory and devotional tactics since the patient could have died. Of course, without necropsy, the diagnosis of pneumonia on the death certificate would have been acceptable to the medical examiner. Case 3 also had several episodes of pleurisy, and embolism was not immediately considered since pleurisy without apparent etiology suggested tuberculosis. When reading of "pneumonia that kills in a few hours," in reports of liberated World War II prisoners who had been subjected to rigid confinement, it is understandable to see how a true diagnosis had been overlooked, but surely in modern hospitals this should not happen.

The well known signs of venous thrombi of the lower extremities cannot always be elicited. Homan's sign of pain on dorsi-flexion of the foot, swelling of the leg, dilated superficial veins, and tenderness over the leg veins may not be demonstrable even if thrombi are present. At this point it may be well to mention that even when the diagnosis of embolism is definite, should one of the extremities be edematosus and painful, ligation of the corresponding vein may not prevent recurrence of embolism. A thrombus sufficiently adherent and organized to cause circulatory stasis and obstruction would be far less apt to be the source of embolism than an asymptomatic thrombus of the other leg, only partially attached to the side of the vessel. A dangerous thrombus may give little sign of vessel closure and inflammation. In foreboding types of thrombophlebitis, the clot becomes organized and tenaciously adherent to the intima and is therefore less dangerous. Embolism does not occur often in phlegmasia alba dolens.

FIGURE 11

FIGURE 12
The x-ray film offers little aid in arriving at a definite diagnosis of embolism. Although pulmonary infarcts are usually sharply delimited and wedge shaped at autopsy, the roentgen film will only produce these pathognomonic features if the central ray happens to be made parallel to the side of the infarcts. Fig. 1 through fig. 13 show nothing characteristic of the pulmonary infarcts present, and while this is also true of fig. 14, only its corresponding lateral view (fig. 15) demonstrates the typical wedge shaped shadow of the infarct. When embolism has occurred without infarction, the situation is certainly even more obscure.

**SUMMARY**

Contrary to what the text books would have us believe, in reviewing cases of patients with embolism, one does not always find the symptoms of chest pain, cough with bloody expectoration, dyspnea, jaundice, and the local lower extremity findings of venous thrombi. Pulmonary embolism takes on many guises, and may masquerade as what appears to be dry or wet pleurisy, bronchitis, bronchopneumonia, lobar pneumonia, pulmonary tuberculosis, cor pulmonale, or unexplained shock. The physician should not treat pulmonary embolism by exorcism. Whether bilateral venous ligation or possibly the administration of heparin or dicumarol is undertaken, the patient will require the most assiduous attention. Long delay has resulted in unnecessary death. We are derelict of duty if the post mortem establishes the true cause of death as a disease that might have been curable.

**RESUMEN**

Contrariamente a lo que nos pensar los libros de texto, al revisar los casos de enfermos con embolia no siempre encuentra uno los síntomas de dolor torácico, tos con expectoración hemoptica, disnea, ictericia y los hallazgos locales en las extremidades inferiores de la flebotrombosis. La embolia pulmonar adquiere muchos aspectos y se disfraza con lo que parece ser pleuresía seca o húmeda, bronquitis, bronconeumonía, neumonía lobar, tuberculosis pulmonar, cor pulmonale o choque inexplicado. El médico no debe tratar la embolia pulmonar por exorcismo. Ya sea que se lleve a cabo la ligadura bilateral venosa o el uso de heparina o dicumarol, el enfermo requiere la atención más asidua. El largo retardo ha dado como resultado una muerte innecesaria. Nos sentiremos responsables si el examen postmortem revela la verdadera causa de la muerte si se descubre una enfermedad que podría haber sido curable.

**RESUME**

Contrairement à ce que les textes voudraient nous faire croire, en faisant la revue des observations de malades atteints d’embolie, on ne trouve pas toujours les symptômes de douleur thoracique, la toux avec expectoration sanglante, la dyspnée, l’ictère et les manifestations de thrombose veineuse localisée aux membres inférieurs.

L’embolie pulmonaire apparaît sous des aspects divers et peut prendre le masque d’une pleurésie sèche ou avec épanchement, d’une bronchite, d’une bronchopneumonie, d’une pneumonie lobaire, d’une tuberculose pul-
monaire, d’un coeur pulmonaire ou d’un shock inexpliqué. Le praticien ne devrait pas traiter l’embolie pulmonaire par l’exorcisme. Même si une ligature veineuse bilatérale ou peut-être l’administration d’héparine ou de “dicoumarol” est entreprise, le malade demande l’attention la plus assidue. Une perte de temps a entrainé un décès qui aurait pu être évité. La responsabilité du médecin est en cause si l’examen post-mortem montre que le décès est du à une affection qui aurait pu être curable.

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