Thrombosis of the Main Stem of the Pulmonary Artery Associated with Pulmonary Tuberculosis
Report of a Case

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Savacool and Charr1 in 1941, reviewed the literature for one hundred cases of pulmonary artery thrombosis of the main stem or its main branches. In their series of cases nineteen were reported to have had pulmonary tuberculosis as a primary diagnosis. Perusing the literature through to 1946 failed to disclose any further reports of pulmonary artery thrombosis.

The following case is reported because of its many interesting features and because of the diagnostic problem presented by the course and symptoms:

E.H., a thirty year old white housewife, entered the San Diego County General Hospital, Tuberculosis Division, on October 26, 1946, with pulmonary tuberculosis confirmed by roentgenograms and sputum containing acid fast bacilli. The patient had been essentially well until nine years before this entry when she first recalled noting dyspnea, with slight exertion, occasionally associated with cyanosis of her face and fingers.

At that time she was seen in the Out-Patient Department of the United States Naval Hospital, at San Diego, where a tentative diagnosis of congenital heart disease was made. Specifically, an interatrial septum defect was considered. Roentgenograms revealed an enlarged right auricle and pulmonary conus. There was a systolic murmur at the base of the heart on the left side of the sternum. The systemic blood pressure and physical examination at that time were otherwise within normal limits. The x-ray films taken on these visits are no longer available for re-examination.

The patient attended the clinic intermittently, noting a gradual increase in the severity and frequency of her symptoms. In February 1945, she entered the United States Naval Hospital following a sudden increase in the above symptoms plus an acute onset of fatigue, fever, sweating, cough and vague chest pain anteriorly.

Roentgenogram taken at that time (figure 1) showed an increase in the size of the upper mediastinal shadow with a bulging density in the region of the pulmonary conus on the left. There were scattered areas suggestive of atelectasis in the left lung field with almost complete atelectasis at the apex. There was also some enlargement of the hilar density on the right. The trachea, in the upper portion of the chest, deviated sharply to the right suggesting the effect of pressure from the mediastinum. The electrocardiogram revealed marked right axis deviation.

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The possibility of bronchopneumonia or tuberculosis was considered at that time. Repeated examination of the sputum revealed no acid-fast bacilli and blood cultures on two occasions were negative. The patient was treated with sulfathiazole and required the use of an oxygen tent. Her acute symptoms gradually subsided, over a period of three weeks. The cardiac murmur and a daily afternoon spiking temperature to 101 degrees F. orally disappeared as her symptoms subsided. The patient was discharged one month after entry. The x-ray film taken at the time of her discharge showed a considerable amount of clearing of the process in the left lung field with essentially no other changes in the heart or vascular shadows. There was still some residual atelectasis present in the left apex.

Following her discharge from the hospital, the patient continued to note her previous complaints of occasional dyspnea and cyanosis. On September 26, 1946, she had a re-check roentgenogram taken at the San Diego County Tuberculosis Clinic. This film revealed, on the right side, a fibrotic infiltration extending out from the hilar region into the second interspace with an area simulating cavitation at the end of the second rib anteriorly. The hilar markings showed some increase in their size and density. The left side revealed some increase in the density previously seen in the first and second interspaces with areas suspicious of cavitation. The contour of the heart and mediastinum were not apparently changed.

The sputum examined at that time was found to contain acid-fast bacilli. The patient entered Vauclain Home on October 26, 1946, following the establishment of the diagnosis of tuberculosis.

The past history included a full term normal delivery preceded by elevation of the blood pressure during the last trimester. She denied symptoms of heart trouble before 1937, joint pains or swelling, chorea,
tuberculosis contacts, venereal disease, or allergy. In the family there was no known case of congenital abnormalities or tuberculosis.

The physical examination on admission revealed a well developed, poorly nourished, cyanotic, dyspneic white female appearing older than her age and easily upset emotionally. Her temperature was normal, pulse 90 and regular, and respirations 22. Blood pressure readings were as follows: right arm 118/90, left arm 120/95, leg 110/90.

Positive findings included a lag in the excursion of the left side of the chest. Anteriorly, at the apex on the left, dulness with breath sounds decreased in intensity and bronchial in quality were noted. Posteriorly, at the left apex, dulness with bronchovesicular breath sounds and a few crepitant rales were heard. The heart was not enlarged to percussion and no murmurs, irregularities or thrills were present. P2 was louder than A2. One observer noted an apical, grade one systolic blowing murmur. Her fingers were exhibiting a moderate degree of clubbing.

The laboratory tests on admission were as follows: urine essentially negative; hemoglobin 103 per cent; WBC 6,600; leukocytes 72; lymphocytes 25; monocytes 3 per cent. The Kline and Wassermann tests were negative and the sputum contained acid-fast bacilli.

The roentgenogram, dated November 15, 1946 (figure 2) taken soon after admission, revealed no change on the right side. On the left side the density in the upper mediastinal region appeared somewhat larger and now simulated a tumor. The configuration of the heart was unchanged. The electrocardiogram revealed notching of the QRS waves in leads 2, 3 and 4, with inversion of the T waves in these same leads. The S wave was depressed in lead 1. These findings were interpreted as representing right axis deviation and right ventricular strain.

In January 1947, roentgenogram of the chest with ingestion of barium showed deviation of the esophagus to the right and posteriorly at the level of the fifth, sixth, seventh and eighth thoracic vertebrae, appar-

FIGURE 3

FIGURE 4
ently from extrinsic pressure. Surgery for a possible mediastinal or bronchogenic malignancy was considered at this time but was deferred because of the poor operative risk and prognosis for the patient with active tuberculous lesions in the lung parenchyma.

On February 3, 1947, a Bucky film (figure 3) showed an increase in the size of the density in the right hilar region, with soft infiltration extending out laterally. On the left side there was a fairly well circumscribed density in the upper portion of the hilar region giving the appearance of a tumor mass with a well defined area of atelectasis extending into the lower part of the upper lobe. The interpretation of this increased density was still not clear. During this time, while on bed rest, the patient gradually gained weight. She had an occasional late afternoon spiking temperature to 101 degrees F. orally. In April 1947, blood streaked sputum was noted for the first time. Cyanosis was still present and unchanged from that noted at the time of her admission.

On June 17, 1947, the patient felt a sudden pain in her left lower chest which increased in severity during the following three days. This pain was associated with fever, apprehension, increased pulse and cyanosis and the production of small quantities of dark red blood in the sputum. These symptoms, fluctuating in severity, continued until death. Physical examination at the time of the onset of these more severe symptoms revealed dulness over the left side of the chest with absent breath sounds and medium moist rales throughout the left lung field. An x-ray film taken of the chest on June 20, 1947 (figure 4) showed little change in the findings on the right side. The left side was obscured by a homogeneous density except at the extreme apex where some creation was visible. The heart and mediastinum were shifted to the left. The picture appeared to be that of atelectasis rather than pleural effusion. Attempts at aspiration of the left chest were unsuccessful. Bronchoscopy on July 23, 1947, revealed narrowing of the left main bronchus, apparently due to extrinsic pressure. The orifice of the left upper lobe bronchus could not be visualized. The bronchial mucosa was not remarkable.

In September, re-check laboratory findings included hemoglobin 138 per cent; RBC 7,550,000; WBC 9,750; lymphocytes 19; monocytes 5; eosinophils 1; leukocytes 75 per cent; hematocrit 80 cc./100 cc.; blood urea nitrogen 24.1 mg. per 100 ml. On October 5, 1947, the circulation time from arm to tongue with gluco calcium was twenty-three seconds; from arm to lung with ether was ten seconds.

The patient continued in a gradual down hill course, becoming more dyspneic and less rational until October 7, 1947 when she expired without any apparent acute incident terminally. The temperature remained below 100 degrees F. and the pulse varied between 100 and 110. The respiratory rate was 20 to 25 per minute up to the time of her death. No peripheral edema or enlargement of the liver was noted. A roentgenogram taken just before death revealed no change in the left side of her chest, however, the lesion in the right midlung field appeared to have been clearing and hardening.

The postmortem examination revealed, in the thorax, old pleural adhesions bilaterally. The left lung showed scattered areas of atelectasis, especially in the upper lobe with multiple areas of active tuberculosis. The lower lobe was the site of a massive infarct with thrombotic obstruction of the artery to this lobe. The right lung showed multiple tuberculous lesions, especially at the apex, with a few scattered thromboses of the smaller pulmonary arteries. The heart presented the picture...
of dilatation and hypertrophy of the right side. There was a large laminated antemortem thrombus apparently occluding the pulmonary stem and its two branches extending to the pulmonary semilunar valves. The thrombus was gray in color at the pulmonary artery branches, becoming red in color as it approached the pulmonary valve. The blood clot was firmly attached to the wall of the artery, but when separated there was a clean plane of cleavage with no gross evidence of disease of the large arteries. Unfortunately, no microscopic sections are available.

**Discussion**

At the onset of her symptoms this 20 year old woman sought medical attention for shortness of breath and blue discoloration of her fingertips. Upon examination cor pulmonale and a basal systolic murmur were found. In the absence of any evidence of pulmonary abnormality a tentative diagnosis of interatrial septum defect was made. Cardiac catheterization would have been of great value in ruling out patent foramen ovale and determining the pressure in the pulmonary circulation which was undoubtedly elevated.

According to Brenner, the above picture fulfills the criteria for a diagnosis of primary pulmonary vascular sclerosis. De Navasquez et al question the significance of pulmonary vascular lesions in some cases of cor pulmonale due to pulmonary hypertension without apparent etiology and propose the term idiopathic right ventricular hypertrophy. Taft and Mallory believe that this pulmonary hypertension of unknown etiology probably precedes sclerotic changes in the pulmonary vessels. In the absence of any evidence of left heart failure, the markedly prolonged circulation time from arm to tongue as compared to the only slightly prolonged circulation time from arm to lung, point to some peripheral vascular disease in the pulmonary circulation. Brill and Krygier emphasize the significance of cyanosis that is out of proportion to the amount of dyspnea as being an outstandingly frequent finding in pulmonary vascular disease, particularly pulmonary vascular sclerosis. This picture was an outstanding feature of the patients symptoms, nevertheless that diagnosis remains in the realm of speculation.

In retrospect, therefore, it appears that the patient when first seen had cor pulmonale due to pulmonary hypertension of unknown etiology, possibly due to pulmonary vascular sclerosis.

The acute episode requiring hospitalization in 1945 was considered at that time to be due to a pneumonitis in the left upper lobe complicating a congenital heart disease. Because of the similarity of the roentgenograms taken at the United States Naval Hospital and those taken on admission to Vauclin Home, it is interesting to speculate whether or not the patient had
tuberculosis and/or atelectasis in the left upper lobe in 1945. The former may have been present in spite of repeatedly negative sputa examinations for acid fast bacilli. The latter could have been due to pressure on bronchi by pulmonary vascular thrombi originating at that time.

The time of origin of the pulmonary thrombus raises another point for speculation. The thrombus may have formed in 1945 at the United States Naval Hospital, resulting in atelectasis as pointed out above. On the other hand, this process may not have started until after entry into Vauclain Home where the embarrassed cardio-pulmonary dynamics complicated by tuberculosis in a bed ridden patient may have set the stage for the onset of the process of thrombus formation.

The authors feel that the patient at the time of her first hospitalization, in February 1945, probably had tuberculosis and atelectasis following the onset of the thrombosis. This thrombus continued to grow until the impairment of the blood supply to the left lower lobe was sufficient enough to result in the infarct which occurred in June 1947. Life was maintained until the heart became unable to stand the strain of pumping blood through the pulmonary arterial system so massively thrombosed.

In Dock's explanation of the localization of phtisis he points out that the incidence of apical or subapical lesions of tuberculosis is higher in patients with pulmonary hypotension as compared to the incidence in patients with pulmonary hypertension. This is not inconsistent with the course of events in this case, for the formation of the thrombus in the pulmonary artery may have been expected to have materially decreased the effective pulmonary arterial pressure. This, according to Dock, decreases the oxygenation and impairs function of humoral defenses in the body.

That the thrombosis occurred first in the left pulmonary artery is suggested by the fact that the first changes noted in the lungs were found on the left. Savacool and Charr in their series of 100 cases report only six cases which were interpreted as having thrombosis of the left main branch only or origin of the thrombus in this branch of the pulmonary artery. The relative infrequency of this finding was explained by the longer and more tortuous course of the right as compared to that of the left branch of the pulmonary artery.

**SUMMARY**

An interesting case of thrombosis of the pulmonary artery stem and its main branches in a patient with pulmonary tuberculosis is presented with a discussion of the pertinent findings and observations.
RESUMEN

Se presenta un caso interesante de trombosis de la arteria pulmonar y sus ramas principales en un paciente con tuberculosis pulmonar y se discuten los hallazgos y observaciones pertinentes.

REFERENCIAS