Bacterial Pneumonia or Pulmonary Infarction?*

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The "textbook" pictures of bacterial pneumonia and pulmonary infarction differ sufficiently to permit accurate diagnosis. Unfortunately, these "textbook" pictures often are absent, particularly in patients with pulmonary infarction. Differentiation in such cases can be a real challenge, because both maladies may give rise to dyspnea, pleuritic pain, tachypnea, tachycardia, fever, cyanosis, hypotension, cough, hemoptysis, jaundice, leukocytosis, and similar roentgenographic abnormalities.

This report emphasizes that bacterial pneumonia and pulmonary infarction frequently mimic each other clinically, indicates that most methods for distinguishing between these illnesses are unsatisfactory, and underscores the few techniques that are helpful in differentiation.

**DISCUSSION**

Several points relating to the early recognition of pulmonary infarction merit attention. The best diagnostic clue may be a concurrent condition frequently predisposing to pulmonary thromboembolism, such as congestive heart failure, trauma (especially to the lower extremities and pelvis), immobility (resulting from medical illnesses, surgical procedures, or prolonged sitting), venous disease of the legs, obesity, or cancer. Even so, pulmonary infarction may strike healthy, active people, including adolescents.1 Sources of the emboli commonly remain obscure. Pleuritic pain, hemoptysis, and thrombophlebitis are absent more often than present. Less than 10 percent of pulmonary thromboemboli lead to infarction of lung; therefore, at the time necrosis develops, viable segments of lung probably harbor thromboemboli, too. Such thromboemboli may cause manifestations (dyspnea, tachypnea, wheezing, syncope) that precede or accompany the infarction.

**Medical History**

Shaking chills (not just chilliness) point strongly to bacterial pneumonia. Additional hints are a preceding upper respiratory tract infection followed by gradually increasing malaise and then cough, usually productive of purulent sputum. Patients with pulmonary infarction more often become ill with dramatic suddenness, seldom have a cough, and experience shaking chills only if the emboli are septic or the infarct becomes infected.

**Physical Examination**

Physical signs are not specific for either malady. Although high fever is more typical of bacterial pneumonia, it occurs with sufficient frequency in pulmonary infarction to be unreliable as a differential diagnostic sign. Patients with pulmonary infarction, as compared to those with bacterial pneumonia, generally are more dyspneic and tachypneic in relation to the extent of their physical and roentgenographic abnormalities and rarely exhibit classic signs of consolidation. They more often manifest hypotension, either transient or recurrent, and more commonly show signs suggesting pulmonary hypertension and right-sided congestive heart failure, e.g., a loud pulmonic component of the second heart sound and elevation of the jugular venous pressure.

A pleural friction rub helps in differentiation only when chest roentgenogram shows no parenchymal disease. Then, infarction is much more likely than infection. Pulmonary infarcts often do not cause roentgenographic changes early in the course of the illness.

**Laboratory Studies**

Sputum examination is one of the best ways of differentiating bacterial pneumonia from pulmonary infarction. In bacterial pneumonia the sputum classically is purulent, occasionally foul smelling, and may contain bright red flecks of blood; Gram's stain typically shows many bacteria
and polymorphonuclear leukocytes. In pulmonary infarction, sputum, when present, usually is frankly bloody with few bacteria or inflammatory cells. Later, its appearance may change to that of dark clots and then dark brown, semiliquid material. If the infarct becomes infected (a rarity in my experience), the sputum may be indistinguishable from that in bacterial pneumonia.

**Blood cultures** often reveal the causative microorganism in patients with bacterial pneumonia but show no growth in cases of bland pulmonary infarction.

**Total leukocyte** count has limited discriminatory value. It usually is normal or slightly elevated in pulmonary infarction, but I have observed counts as high as 40,000 per mm³ in patients with massive, bland necrosis of pulmonary tissue.

Elevated serum lactic dehydrogenase (LDH) activity, normal serum glutamic oxaloacetic transaminase (SGOT) activity, and increased serum bilirubin concentration forms a triad once considered a sensitive indicator of pulmonary embolism and infarction. Subsequent studies have shown, however, that these tests fail to differentiate pulmonary infarction from pneumonia and a host of other disorders.

**Pleural fluid**, when bloody, always should suggest pulmonary infarction, but in most cases of infarcted lung, such fluid is not sanguineous. The effusion characteristically is sterile in pulmonary infarction, but in bacterial pneumonia, it may harbor the causative microorganism. The specific gravity, total and differential leukocyte counts, and protein concentration of the pleural fluid are not distinctive for either disorder.

**Roentgenographic** similarities of bacterial pneumonia and pulmonary infarction are the chief source of diagnostic confusion between the two entities. Each is responsible for parenchymal infiltrates of varied size and shape, with or without pleural effusion, atelectasis, or cavitation. In contrast to bacterial pneumonia, pulmonary infarcts always abut a pleural surface and predominate in the lower lobes, especially the right. They also may appear in concert with dilatation of one or both main pulmonary arteries, decreased peripheral vascular markings in the affected portions of lung (oligemia), or engorged vessels in the nonaffected areas (pleonemia). Further roentgenographic clues to pulmonary infarction are infiltrates appearing first in one lung and then the other, or "pneumonia" unresponsive to chemotherapy.

**Electrocardiographic** abnormalities are of limited aid in the recognition of pulmonary infarction, because they are nonspecific, characteristically fleet-

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**Pulmonary function tests** and arterial blood gas studies provide data that are too variable and nonspecific to differentiate bacterial pneumonia from pulmonary infarction.

**Cardiac catheterization** cannot discriminate between these illnesses but may prove helpful when it reveals pulmonary arterial hypertension in the absence of left-sided congestive heart failure or preexisting pulmonary parenchymal disease. Whereas pulmonary arterial pressure frequently rises in thromboembolic disease of lung, it remains normal in bacterial pneumonia.

**Scintillation scanning of the lungs** has limited usefulness in distinguishing between bacterial pneumonia and pulmonary infarction, because any process responsible for parenchymal abnormalities on chest roentgenogram may result in diminished radioactivity ("cold defect") within the involved region(s). Nevertheless, if "cold defects" also appear in roentgenographically normal portions of lung, they suggest that coexisting parenchymal densities represent pulmonary infarcts.

**Pulmonary arteriography** is the most specific means of differentiating bacterial pneumonia from pulmonary infarction. In bacterial pneumonia the pulmonary arteries proximal to the subsegmental level show neither filling defects nor obstructive lesions (Fig 1, left), whereas in pulmonary infarction they contain filling defects, appear obstructed, or both (Fig 1, right). Because sizeable pulmonary thromboemboli may undergo rapid, spontaneous resolution, the arteriogram may be normal if obtained a week or more after the suspected embolic episode. Arteriographic examination is virtually mandatory before pulmonary embolectomy and highly desired before interruption of blood flow through the inferior vena cava. It obviously is not indicated in every case of suspected pulmonary infarction. Yet, without angiographic demonstration of thromboemboli, an element of uncertainty remains in the clinical diagnosis of pulmonary infarction, no matter how "typical" the other findings may be.

Despite use of the aforementioned techniques, the question of infected vs infarcted lung some-
times will persist. To minimize diagnostic error, the physician should think of both maladies when he considers either, particularly if the process involves the lower lobes, especially the right. Dangers of delaying treatment for pulmonary infarction rival the hazards of withholding specific chemotherapy in bacterial pneumonia. Thus, as long as the diagnosis remains in doubt, treatment for both disorders seems well advised.10

**Conclusions**

When the clinical problem is that of bacterial pneumonia vs pulmonary infarction, the best support for infection is shaking chills, purulent sputum, or bacteremia, whereas the best evidence of infarction is the angiographic demonstration of pulmonary thromboembolism. Important clues to infarction are a concurrent condition frequently predisposing to pulmonary thromboembolism; frankly bloody, nonpurulent sputum; sanguineous pleural effusion; migratory parenchymal infiltrates; and "pneumonia" unresponsive to chemotherapy. Such findings are inconstant, however, and it is unwise to consider them requisites for diagnosis. Moreover, one never should doubt or reject the possibility of pulmonary infarction simply because of high fever, leukocytosis, normal jugular venous pressure, "atypical" pulmonary lesions, nonbloody pleural effusion, failure to detect the source of the emboli, or because the patient is young or appears otherwise healthy.

Clinical differentiation of bacterial pneumonia from pulmonary infarction occasionally is not possible. In that circumstance I recommend treatment for both disorders.

**References**


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A DOCTOR AS A FAMOUS PLAYWRIGHT

Anton Pavlovich Chekhov (1860–1904) studied medicine in Moscow but adopted a literary career. The doctor in Chekhov was never completely driven out by the writer. He catches a great many people when their pretenses and defenses are down. No man is hero to his doctor. Now Doctor Chekhov had a sharp eye, an unflinching sense of truth, much humor, and a tender heart. And what is finally communicated to us by his drama which represents a much greater and more highly original achievement than his short stories, good though they are, because the medium is so much more obstinate and difficult, is an immense brooding tenderness, unequalled by any other modern dramatists, and all the more remarkable because it never lapses into sentimentality. 

Priestley, J. B.: Literature and Western Man, Harper and Brothers, New York, 1960

STRANGE APHORISMS

Those who live in the shadows and those who live in the sun have never been able to get along together. The relationship between parents and children is no less difficult no less fraught with drama than that between lovers. The growing child developing into an independent individual surprises and annoys its parents. Two people may be all in all to one another but habit plays a large part in their daily intimacy. Transplant them, separate them, and very soon they will strike new roots in strange soil. To the friend once we made the confidant of our every thought, we cannot bring ourselves to say a word. Silence covers all.


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