Roentgenographic Aspects of Complete and Incomplete Pulmonary Infarction*

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With the advent of effective medical and surgical therapy for pulmonary and systemic thromboembolic disease, it has become essential for the radiologist to diagnose this disease in the lung as rapidly and accurately as possible. The appearance of pulmonary emboli and thrombi on the x-ray film is varied, and in this paper an attempt will be made to review the roentgenographic appearance in 25 cases (see Table I) in which there were positive findings, and to correlate these with other cases reported in the literature.

Incidence

The high incidence of pulmonary embolism is attested by numerous studies. Barnes1 estimated that 33,748 people die from this cause annually in the United States; if this is applied to the general population, it may be assumed that 3,068,000 people now living in this country will eventually die of pulmonary embolism—and for every fatal attack, there occur two or three instances of non-fatal episodes. Belt2 found pulmonary emboli in about 10 per cent of 567 routine autopsies, causing death in about 6.5 per cent of them. In 3,500 post-mortem examinations, Hampton and Callister12 found pulmonary emboli in 9 per cent; they caused death in 3.5 per cent. These included post-operative, cardiac and medical cases, being respectively 40, 30 and 30 per cent. In another study of 6,548 autopsies, Krause and Chester22 found hemorrhagic infarcts in 5.2 per cent. Pulmonary emboli caused 6 per cent of all post-operative deaths at the Mayo Clinic between 1917 and 1927,14 and 5.8 per cent between 1928 and 1933.1 An actual increase in the incidence of cases of thromboembolism has been reported from Tulane University and Massachusetts General Hospital;28 these statistics make apparent the necessity for early diagnosis of these disorders.

It has been stated that the diagnosis of an infarct by x-ray examination is probably subject to more error than any other lesion of the lungs; the error is almost always a negative one, i.e., failure to consider an infarct as the cause of the abnormal shadow.21,24 Carlotti, Hardy, Linton and White9 said that pulmonary emboli were probably diagnosed in less than half of the medical patients in whom they were found at autopsy. They reported on 108 cases studied between 1936 and 1940 at the Massachusetts General Hospital in which the infarct was observed on the x-ray film in only 36 cases (33 per cent); between 1941 and 1945 an infarct was reported

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on the interpretation in 73 out of 122 cases (60 per cent). Incidentally, the
diagnosis of a "consolidation" dropped from 48.2 to 18.9 per cent in these
two series. Krause and Chester\textsuperscript{22} in a review of 344 aseptic hemorrhagic
infarcts, reported that the correct diagnosis had been made in only 22 per
cent. In a review of 74 autopsied cases of pulmonary emboli, Dehlinger and
Riemenschneider\textsuperscript{9} found that only 28 had chest x-ray films; in 18 of these
the emboli were not recognized because of the presence of other pulmonary
or pleural disease. In the 10 remaining cases, pulmonary emboli with or
without infarcts were the only pathological findings, and in five of these
there were no abnormal radiographic densities; in the other five, the
greatest misinterpretation of shadows cast by infarcts, the only pathologic
process found at autopsy, was with pneumonitis or broncho-pneumonia.
Vander Veer and his co-workers\textsuperscript{31} stated that 42 out of 89 cases of pul-
monary emboli had x-ray film signs of pulmonary infarction, consolidation,
atelectasis or pleural effusion, and that nine more (in review) had local-
ized areas of emphysema.

Thus, the accuracy of the roentgenographic diagnosis of pulmonary
infarction and embolism is low as compared to the recognition of other
pulmonary diseases. In our experience, one of the major causes for this
was the patient's poor condition, so that bedside films were the only kind
available for study. It is conceivable that with technical improvements,
a higher degree of diagnostic accuracy will result. Robbins\textsuperscript{31} recommended
that the following techniques be utilized when possible: fluoroscopy, spot
films, a high kilovoltage (85-95) with a short exposure, PA projections,
lateral and oblique projections if spot films were not available, and finally,
six-foot films. With improvements in techniques, it should be possible to
obtain six-foot films for patients in their beds, in the x-ray rooms.

Rigler\textsuperscript{29} stated that careful attention must be given to the history, in
order to differentiate infarcts from pneumonia, and this may be of more
value than any other technical aid.

\textit{Classification}

In this paper the classification of Hampton and Castleman\textsuperscript{12} will be
used, adding to it a discussion of the appearance of pulmonary emboli
without infarction. In their classic paper, they subdivided pulmonary in-
farcts into two types: the complete and the incomplete. In the former
there is a sequence in the involved lung area consisting of the following:
filling of the alveoli with blood, while larger units, such as the alveolar
sacs and atria remain filled with air, up to 24 hours. (Thus, there may
be difficulty for 12 to 24 hours in visualizing the process on the films.)
At the end of 24 hours necrosis of the alveolar walls and degeneration
of the red cells commences. Organization begins in the second week and
may last for months until a fibrous scar remains; a very large infarct may
remain hemorrhagic indefinitely. The incomplete infarct begins similarly,
with edema, fluid and red and white cells in the alveoli; this does not
progress to alveolar wall destruction and heals by resolution in two to
four days.
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<th>Case</th>
<th>Age</th>
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### TABLE I (Continued)

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**Figure 1, Case 1:** A 42-year old female with multiple septic infarcts following pelvic and abdominal surgery, with good response to protracted antibiotic and anticoagulant therapy—*Figure 1A:* Infarct at right base, impossible to distinguish from pneumonia. History essential for diagnosis—*Figure 1B:* Second large infarct at left base, three months later—*Figure 1C:* Lateral view, on same day, demonstrates the rounded cardiac margin, the pyramidal shape, and the adjacent small interlobar effusion; note, also, that the long diameter of the infarct is parallel to the largest pleural surface. The density is homogeneous and the margins are sharp. This resolved slowly, leaving a linear scar and small pleural adhesions.
Complete Infarcts

There are both primary and secondary manifestations of complete infarcts. The primary signs are those produced by the infarct itself and the secondary signs by the effects of the infarct (see Table I). The primary signs are as follows:

1) An abnormal density is present which is variable in shape, depending entirely on its location to determine its configuration. Thus, it may be pyramidal or triangular, round, oval or irregular. Odd and round shadows may be caused by superimposition of multiple infarcts and should be "dissected" by multiple films, including spot films. The borders are sharp if seen in the degree of obliquity in which its greatest thickness is penetrated. The infarct is always in contact with one or more pleural surfaces; the cardiac margin is rounded or "hump-shaped." The long diameter is always parallel to the largest pleural surface involved.

In the cases recorded in this paper, these criteria have been found; also noted has been the homogeneous character of the density in all cases. Helpful in reaching a diagnosis of infarction has been the occasional odd shape of the lesion—oval, band-like, rectangular or amorphous. Figures 1A and 1B show the usual appearance, that of large basal densities which resemble pneumonias, but can be appreciated as infarcts when additional views are obtained—Figure 1C. Figures 2, 3, 4, 5, 6B, 7B and 8B are other infarcts, some with markedly irregular shapes.

2) The density may be small or large, varying from 0.3 to 10 cm., and averaging 3 x 2 x 1 cm.; one or many may be present. Our series showed larger lesions; the average size was 3.5 x 5 cm.; multiple lesions were common.

**FIGURE 2**
*Figure 2, Case 5:* A 50-year old male who developed a pulmonary infarct five days after a herniorrhaphy. Anticoagulant therapy successful. Film shows an odd almost rectangular density in right lung; it is sharply defined and has no secondary signs of fluid or diaphragmatic elevation. Note abdominal distention.—*Figure 3*

**FIGURE 3**
*Figure 3, Case 8:* A 45-year old female; pulmonary infarct appeared seven days after spontaneous rupture of cul-de-sac abscess; septic pelvic thrombophlebitis; good response to dicoumerol and antibiotics. Film shows well-demarcated, homogeneous density above right costo-phrenic angle; this disappeared without sequelae in one month.
**Figure 4, Case 12:** A 68-year old male with multiple small pulmonary emboli secondary to thrombo-phlebitis of left leg; controlled by dicoumerol. Rectangular, homogeneous, small infarcts seen on the film, in left lung, together with characteristic long linear scars of old infarcts at right base.—**Figure 5, Case 20:** A 60-year old male with hemoptysis and pleural pain in December 1950 and March 1951; arteriosclerotic heart disease and auricular fibrillation. Round, sharply defined homogeneous mass seen on film; this was in anterior segment of right upper lobe. Pre-operative diagnosis was peripheral carcinoma of lung. Lesion excised and found to be characteristic pulmonary infarct.
3) The location is variable. Of 344 hemorrhagic infarcts, Krause and Chester\textsuperscript{22} reported involvement in the right lower lobe as 27 per cent (solitary infarcts) and in the left lower lobe as 15 per cent; in the right lower lobe combined with infarcts in other lobes there were 69 per cent, and in the left lower lobe combined with other lobes there were 53 per cent. Hampton and Castleman\textsuperscript{12} reported 74 per cent in the lower lobes, 43 per cent on the right and 31 per cent on the left. Kirklin and Faust\textsuperscript{20} reported 18 infarcts at the right base, three at the base of the right upper lobe, three in the left lung, and four in both lungs. Thus, there is some predilection for the right lower lobe, and this has been our observation too, the right lung being the site of an infarct two times as often as the left, with the right lower and middle lobes being involved in 44 per cent of cases.

4) Hemorrhagic infarcts heal by organization, from the periphery inward.\textsuperscript{17} Usually, a scar is left consisting of masses of dense, tangled elastic fibrils\textsuperscript{25} which may be visible on the film as an irregular linear band.\textsuperscript{20} Fleischner, Hampton and Castleman\textsuperscript{10} have described this scar as a dense single line which runs in any direction, reaches a pleural surface and ends either as a rounded nodular shadow flat against the pleural surface or directly in a thickened or retracted pleura; multiple lines due to multiple infarcts may be present. These shadows are to be differentiated from areas of plate-like atelectasis and interlobar pleuritis\textsuperscript{10,12,26} (Figure 4). However, an infarct may clear completely.

5) Various complications have been reported from pulmonary infarcts. These have included pneumonia, abscess and bronchopleural fistula with empyema.\textsuperscript{17,20,21,22} We have observed one instance of encapsulated fluid which cleared slowly on medical management alone. Pleural effusion as

**FIGURE 6A**

*Figure 6, Case 23: A 32-year old male with periodic bouts of pulmonary emboli for several years; autopsy showed thrombophlebitis of femoral veins and multiple pulmonary infarcts.*—**FIGURE 6A:** Linear shadows at left base believed to represent incomplete infarcts. Note short length and oblique orientation.—**FIGURE 6B:** 24-hours later, several small infarcts are seen in same location; these are broad densities, oriented in similar manner; complete clearing in a week. A large infarct developed a month later; just before death (eight months later) residual scars and pleural adhesions were present.
Figure 7, Case 24: A 28-year old male with multiple pulmonary emboli and massive cardiac enlargement. Poor response to anticoagulant therapy. Complete recovery after ligation of inferior vena cava (followed for 1½ years).—Figure 7A and 7B: Linear shadows believed to represent incomplete infarcts in lower two-thirds of left lung, varying in position over a six-day interval. These corresponded with clinical evidence of pulmonary emboli. Complete clearing in 10 days.—Figure 7C: Complete infarcts three weeks later, in right lung. Complete resolution in two weeks; no residual scars or pleural changes.
a complication will be discussed later. In general, the presence of complications, in these days of potent antibiotics, makes the diagnosis more difficult and might cause one to question the original diagnosis.

Many secondary signs of pulmonary emboli have been described:

1) Clouding in the region of the costophrenic angle on the side of the infarct has been described as a sign of an infarct.\textsuperscript{17} Wharton and Pierson\textsuperscript{25} reported it is the first sign of an infarct, appearing 24 hours after the onset of symptoms, while the remainder of the lung was still clear. The explanation for this phenomenon is not clear, and one assumes that if this does happen, it represents an impairment of the pleural integrity. In this series, this sign has not been observed.

2) Accentuation of the hilar vessels on the side of the lesion has been reported as a contributory sign of an infarct.\textsuperscript{17} This is assumed to be due to dilatation of the pulmonary vessels on the affected side,\textsuperscript{36} and if present, is probably due to passive hyperemia.\textsuperscript{32} In this series, dilatation has been noted in the hilum, but has been attributed to congestive changes, rather than to the embolus or infarct. Apparently Rigler also attributes this to congestive changes, judging from the description of the lesions in his monograph, "The Chest."\textsuperscript{29}

3) The presence of a prominent pulmonary conus is not a reliable secondary sign of an infarct. This was demonstrated indirectly by Horn, Dack and Friedburg\textsuperscript{16} who, in 42 patients dying after pulmonary embolism, found no definitive cardiac change; the degree of dilatation of the right side of the heart was uncertain; marked dilatation occurred in only four cases, and the evaluation of slight or moderate dilatation was very uncertain and difficult in the post-mortem state. This finding was not observed in these 25 cases.

4) Elevation of the diaphragm on the affected side is not a reliable sign of infarction; it has been observed in seven of our cases, but not in those with scars in the parenchyma, in which a loss of pulmonary volume could be assumed.

5) Pleural effusion may or may not be secondary to an infarct. This is difficult to prove. Hampton and Castleman\textsuperscript{12} found effusions in only 40 per cent of their cases, but in only 13 per cent did it seem likely that the infarct produced the fluid; rather could it be attributed to congestive failure. In the absence of congestive failure it might be assumed that the fluid is on the basis of the infarct.\textsuperscript{21} A large effusion may conceal the infarct entirely.\textsuperscript{13} Figure 1C demonstrates some of the pleural changes.

Moberg\textsuperscript{27} reported that the pleural effusion was of greater roentgenographic dominance than the parenchymal density in about half of 46 cases of pulmonary embolism. This is contrary to our own experience, in which five cases showed small amounts of fluid; only in a sixth case was the amount of fluid large; even in the latter, the density of the infarct was not hidden by the fluid.

In general then, the secondary signs described heretofore seem to be misleading, with the exception of the small pleural effusion in the presence.
Figure 8A: A 55-year-old man with pleural pain and hemoptysis; arteriosclerotic heart disease. Figure 8B: Seven days later, an irregular density is present in the same location, representing a complete infarct. Patient lost to follow-up.
of contiguous parenchymal disease, in a non-cardiac patient; in this situation this may be valuable indirect evidence of an infarct.

Incomplete Infarcts

The incomplete infarct was first described by Wharton and Pierson, and later by Hampton and Castleman, as an abortive or incipient infarct, one that ended by resolution instead of organization, and therefore, disappeared in 24 to 48 hours. Pathologically, there is edema; red and white blood cells appear in the alveoli but no alveolar wall destruction occurs. Several observers came to the conclusion that simple bland embolism of the pulmonary artery produced circulatory changes in the lung area but not a true infarction, unless there was passive congestion. It has been postulated that this condition was caused by multiple small emboli arising from thrombi in an operative field. This can occur in the normal lungs of animals; thus, a transitory x-ray shadow may be produced. It has been thought that this situation may be similar to that found in the early stage of an infarct and that if the conditions are proper (circulation is disturbed), the incomplete infarct will go on to completion; if not, it will disappear by resolution. The x-ray film appearance of the incomplete infarct has not been hitherto described. In three cases described here (Figures 6, 7 and 8) faint, sharp, short (2 to 4 cm.) lines were seen in chest films, coincident with the clinical occurrence of pulmonary emboli; these were situated transversely or obliquely and were preceded by negative roentgenograms; they changed character in two to seven days. In each of two cases, the source of emboli was the inferior vena cava; one recovered after ligation of the inferior vena cava; the second had post-mortem proof of a thrombus in that vessel.

The sharp lines observed in these three cases had to be differentiated from linear atelectasis, pulmonary scars, and interlobar pleuritis. This was done as follows:

1) Linear or plate-like atelectases appear on films as multiple, long, linear shadows which are constantly horizontal, occurring at the bases of the lungs and are associated with elevation of the diaphragm and intrathoracic shadows; they are usually perpendicular to the longitudinal fissures. The shadows believed to represent incomplete infarcts occurred in the middle third of the lungs, as well as the bases, were short, often oblique in the frontal view, often single and not associated with abdominal lesions; slight diaphragmatic elevation was noted in only one instance.

2) Pulmonary scars could be ruled out since previous films were negative and the linear shadows changed character or disappeared quickly.

3) Interlobar pleuritis could not be demonstrated in these three cases; the position of the shadows described was different from that of the lung fissures.

It would seem logical to attribute these abnormal, transient linear shadows to the embolic disease of the lung; in one case, the shadow dis-
appeared after several days; and in the other two cases complete infarcts appeared at the same site, within 24 to 48 hours.

Obviously, the experience in interpreting these linear shadows is limited and adequate explanation of the x-ray appearance is lacking; further studies and observations are necessary to verify the suggestion present, and preferably anatomical proof should be obtained, although this may be very difficult.

The concept of the incomplete infarct probably overlaps that of the pulmonary embolus without infarction. Westermark first described the shadow cast on the film by the latter condition as a sharp, local area of increased radiability (ischemia or anemia) on the peripheral side of the lung embolus. On the central side he noted abrupt cessation of the vascular markings; the ischemic area was wedge-shaped with its apex to the hilum and the base to the lung periphery. His explanation for this observation was that when a pulmonary embolus occurred in a patient without stasis of the pulmonary circulation there would be no infarct; in this situation there would be ischemia of the involved part of the lung producing an anemic, pale and aerated area.

Shapiro and Rigler described this phenomenon in detail in three cases; they pointed out that this appearance can also be found contiguous to an area of hemorrhagic infarction. Vander Veer et al., in a review of 89 cases of pulmonary infarction found nine instances of localized pulmonary "emphysema," but details of these cases were not presented.

There is some evidence, however, that suggests a close parallel between the pathologic findings in the incomplete infarct and in the embolus without an infarct. There is a general lack of proof of the existence of a pale infarct of the lung. Holman and Mathes are quoted as having produced pale infarcts with sterile emboli in dogs, but a review of their experimental data shows that they produced a slight edema and hemorrhagic infiltration in 30 of 32 animals; this is probably the same as an incomplete infarct. Physiologic and anatomic factors also support this hypothesis: the adequacy of the bronchial artery in nourishing the abnormal pulmonary zone and the richness of the anastomoses of the branches of the pulmonary artery in areas of lung less than lobar in size.

In a review of 173 cases of pulmonary embolism in cardiac patients Lenegre and Neel found embolism without infarction in 92 instances, and with infarction in 53; Westermark's sign was not observed. Moberg did not find it in 46 cases, nor has it been found in this series of cases. The paucity of roentgen evidence in situations in which there is good clinical evidence of pulmonary emboli may well lead the radiologist, when confronted by a negative roentgenogram, or one with the above described lines, or with a zone of ischemia, to a diagnosis of pulmonary embolism without infarction or with incomplete infarction.

Acknowledgement: The author is indebted to Dr. L. G. Rigler for invaluable constructive criticism and advice.
SUMMARY

1) There is a very high incidence of pulmonary thromboembolic disease. This appears to be on the increase.

2) Examples of complete infarcts are given, emphasizing their radiographic variability. The odd shape of a homogenous density is sometimes helpful in diagnosis. Multiple x-ray films are important. Secondary signs are usually unreliable.

3) The importance of recognizing the incomplete infarct is stressed. Three examples are described.

4) Embolism without infarction (or, probably with incomplete infarction) occurs very frequently; an area of pulmonary ischemia may be a roentgen sign of this situation, as described by Westermark and others. Also, clinical evidence of a pulmonary embolus associated with negative roentgen evidence may warrant this diagnosis, too.

RESUMEN

1) Existe una elevada frecuencia de enfermedad tromboembólica del pulmón. Esto parece que está aumentando.

2) Se dan ejemplos de infartos completos, haciendo notar su variabilidad radiográfica. La extraña forma de una sombra densa homogénea es útil a veces para el diagnóstico. Son importantes las radiografías múltiples. Los signos secundarios habitualmente no son dignos de crédito.

3) La importancia de reconocer el infarto incompleto se recalca. Se describen tres ejemplos.

4) La embolia sin infarto (o probablemente con infarto incompleto) ocurre muy frecuentemente; un área de isquemia pulmonar puede ser un signo de esta condición tal como ha sido descrita por Westermark y otros. También la evidencia clínica de un émbolo pulmonar sin evidencia a los rayos X puede justificar este diagnóstico.

RESUME

1) Il y à actuellement une très grande quantité d’embolles pulmonaires. Cette affection semble être en augmentation.

2) L’auteur rapporte des exemples d’infarctus complets, en insistant sur la variabilité de l’aspect radiologique. L’aspect atypique de l’ombre homogène est parfois précieux pour le diagnostic. Il est important de pratiquer des radiographies multiples. Les signes secondaires restent habituellement sans valeur absolue.

3) L’auteur insiste sur l’importance qu’il y à reconnaître l’infarctus incomplet. Il en donne trois exemplos.

4) L’embolie sans infarctus, ou probablement avec infarctus incomplet est une éventualité très fréquente. Cet état peut se caractériser radiologiquement par l’existence d’une zone pulmonaire ischémique ainsi que l’ont décrite Westermark et d’autres auteurs. Ainsi, il est possible d’affirmer le diagnostic d’embolie pulmonaire sur des signes cliniques évidents, en l’absence de toute image radiologique.
REFERENCES

30. Rigler, L. G.: Personal communication.