Radiographic signs of pulmonary embolism and pulmonary infarction

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A comparative analysis of the radiographic features in a group of patients dying with pulmonary embolism and/or pulmonary infarction, and a group without but suspected of having the conditions before death, showed that two radiologists could diagnose pulmonary infarction correctly in 70% of cases. The most useful radiographic sign was elevation of the diaphragm often with slight atelectasis and a small unilateral effusion. The plain chest radiograph was of little value in diagnosing pulmonary embolism without infarction.

Many radiographic signs of pulmonary embolism and infarction have been described in clinical cases (Fleischner, 1959; Westermark, 1938). However, pulmonary embolism and infarction are frequently wrongly diagnosed, often being missed and sometimes diagnosed when pulmonary infection or carcinoma are present. Marshall (1965) stressed three radiological signs of infarction, namely, the shadow produced by the lesion, evidence of a pleural effusion, and a raised diaphragm. Less frequent features associated with embolism are increased radiotranslucency, pulmonary oedema, and prominent main pulmonary arteries. These may, however, occur in other conditions, and it was therefore decided to take a control group of patients to find if any of these changes are usually diagnostic of infarction, and also to show whether radiologists can diagnose pulmonary embolism and infarction from routine plain radiographs. To be certain of the diagnosis, only patients in whom necropsy evidence was available were studied.

PATIENTS AND METHODS

From the hospital necropsy records of 1967 to 1970 inclusive, cases of pulmonary embolism and infarction were selected if they had a chest radiograph. From the case records the date of the radiograph in relation to death was determined, and radiographs showing evidence of infarction (>3 cm in diameter) or embolism which was later proved at necropsy were selected. This usually meant that only cases in whom infarction had been present for 48 hours or cases with necropsy evidence of fresh infarction, who had had a radiograph performed from 12 to 24 hours before death, were included. Of the 66 cases with emboli/infarction (Table 1), 57 had radiographs within one week of death (mean 1.96 days, SD 1.2), and six, of whom three had fibroed infarcts, within three weeks. Three cases of sudden massive embolism had radiographs taken shortly after symptoms occurred and within four hours of death.

<table>
<thead>
<tr>
<th>Pathological Diagnosis</th>
<th>No. of Necropsies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>No emboli or infarcts</td>
<td>57</td>
</tr>
<tr>
<td>Pulmonary emboli only</td>
<td>17</td>
</tr>
<tr>
<td>Pulmonary infarction only</td>
<td>3</td>
</tr>
<tr>
<td>Pulmonary embolism and infarction</td>
<td>46</td>
</tr>
</tbody>
</table>

Twenty-three per cent of these cases had fractured femurs, and 69% died following abdominal surgery. Although it was difficult to identify the time of onset of embolism or infarction this was usually a few days from death (4–8 days, SD 2.9 days). Clinical features which in retrospect were typical of pulmonary embolism/infarction were recorded for 39 patients. These were haemoptysis (10 patients), sudden dyspnoea (21 patients), pleuritic pain (9 patients), angina (3 patients), and syncope (5 patients). Often there was gradual deterioration with confusion and coma. Signs of consolidation were noted in 15 patients and signs of a 'pleural effusion' in five patients. Often the radiographs were performed to elucidate coincidental infections or cardiac failure.

The pathological examination included examination of the major, lobar, and segmental vessels. If thrombus was firmly adherent it was assumed that embolism had occurred at least 48 hours before death, and if lightly adherent at least 24 hours before death. Additional fresh embolism was not considered. There was one case with an organized fibroed pulmonary arterial occlusion, which was probably embolic.
At necropsy only 16 cases had unilateral emboli: of the 63 cases with emboli, 21 had major pulmonary artery emboli (± other emboli), 20 had lobar emboli (± segmental emboli), and 22 had segmental emboli only. Of the 49 cases with infarcts, massive lobar infarcts were found in 7, infarction (>3 cm) in 35, and multiple small infarcts (<3 cm) were found in 7.

At the time of the radiograph, in 31 cases there was right lower lobe infarction and in 23 cases left lower lobe infarction: eight were bilateral. There were two cases with right upper lobe infarction alone, and of three left upper lobe infarctions two also had infarction elsewhere. There was one case with an abscess secondary to a right lower lobe infarction.

A control series consisted of 57 radiographs from 1969 to 1970 on patients in whom the diagnosis of pulmonary embolism or infarction was suspected at the time of their radiographs, but who did not have these conditions at necropsy; 24-5% were surgical cases. The causes of death in this control group were usually pneumonia (29 cases), left ventricular failure (10 cases), myocardial infarction (7 cases), and carcinoma (6 cases).

The combined series of radiographs were then shown to two radiologists, A and B, without the patient’s name or the pathological features being known and they were asked to comment on the radiological features and whether they thought pulmonary infarction was present. They also commented on the visibility of the pulmonary vessels and whether they were normal or compatible with embolism. (If the lobar vessels were obscured by consolidation, they were considered abnormal.) They commented on any other features suggestive of embolism, and particularly cardiomegaly, pleural effusions, pulmonary oedema, and pulmonary venous engorgement. Elevation of the diaphragm was noted if the right side was more than 1 inch higher than the left, or if the left side was higher than the right.

All available radiographs were examined, but portable supine radiographs were included only when they were part of a series of examinations. Table II shows the type of radiographs performed for both controls and patients with pulmonary emboli/infarction.

### TABLE II

<table>
<thead>
<tr>
<th>Radiographic Examination</th>
<th>No. of Necropsies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>Multiple examinations</td>
<td>12</td>
</tr>
<tr>
<td>PA and lateral</td>
<td>9</td>
</tr>
<tr>
<td>PA only</td>
<td>26</td>
</tr>
<tr>
<td>AP only</td>
<td>10</td>
</tr>
</tbody>
</table>

### RESULTS

An analysis of the correct and incorrect diagnoses is shown in Table III. Below this are details of the patients on whom the radiologists made the correct diagnosis, either by diagnosing infarction or embolization when they were present or by not doing so when they were absent. Below this the incorrect diagnoses are analysed by a similar method.

### TABLE III

<table>
<thead>
<tr>
<th>Radiological Diagnosis</th>
<th>Radiologist A</th>
<th>Radiologist B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Emboli</td>
<td>Infarcts</td>
</tr>
<tr>
<td>Correct diagnosis</td>
<td>66</td>
<td>92</td>
</tr>
<tr>
<td>Incorrect diagnosis</td>
<td>57</td>
<td>31</td>
</tr>
<tr>
<td>Correct positive</td>
<td>19</td>
<td>24</td>
</tr>
<tr>
<td>Correct negative</td>
<td>47</td>
<td>68</td>
</tr>
<tr>
<td>Incorrect positive</td>
<td>13</td>
<td>6</td>
</tr>
<tr>
<td>Incorrect negative</td>
<td>44</td>
<td>25</td>
</tr>
</tbody>
</table>

* A positive radiological diagnosis was made if there were radiological features of infarction or emboli.

The percentage correct diagnosis of pulmonary infarction was 74.7% for A and 65.8% for B. The percentage correct diagnosis of pulmonary embolism was 53.6% for A and 57.7% for B. Thus the diagnosis was correct in 70% of patients with pulmonary infarction and in 55% of patients with pulmonary embolism.

The probability of these figures being due to chance for radiologist A was <0.01 for infarction but not significant for embolization, $X^2$ being 24.6 and 0.75 respectively. For radiologist B, $X^2=6.54$ ($p<0.01$) for infarction and $X^2=2.86$ (p<0.10) for embolism.

### TABLE IV

<table>
<thead>
<tr>
<th>Pulmonary Emboli/Infarcts</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(66 cases)</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>41</td>
</tr>
<tr>
<td>No cardiomegaly</td>
<td>24</td>
</tr>
<tr>
<td>Elevated diaphragm</td>
<td>26</td>
</tr>
<tr>
<td>Bilateral pleural</td>
<td>5</td>
</tr>
<tr>
<td>Effusions</td>
<td>4</td>
</tr>
<tr>
<td>Unilateral large</td>
<td>12</td>
</tr>
<tr>
<td>Effusion</td>
<td>11</td>
</tr>
<tr>
<td>Unilateral small</td>
<td>8</td>
</tr>
<tr>
<td>Effusion</td>
<td>3</td>
</tr>
</tbody>
</table>

The figures refer to the numbers of patients in each group with this radiological feature.

Table IV shows some radiological features that are usually associated with infarction and embolism. The only two that had significant association were elevation of the diaphragm (Fig. 1) and cardiomegaly (one radiograph was not assessed for
Correlation of an elevated diaphragm and pulmonary infarction was most significant ($\chi^2=33.3$, $P<0.01$). This, therefore, seems to be a significant sign of pulmonary infarction, although it did occur in one case of subphrenic abscess, in one case of basal atelectasis, and in three cases of pneumonia. Obese patients or patients having supine radiographs usually had bilateral elevation of the diaphragm, which was difficult to assess.

Basal collapse could often be seen on the radiograph with crowding of vascular markings; linear atelectasis was noted in three cases but in none of the controls (Fig. 2).

There was no difference in the diagnostic accuracy of right-sided (41) or left-sided (38) emboli and infarcts, but only one out of five upper lobe infarctions was detected, presumably due to the greater distance of such infarcts from the diaphragm.

The diagnostic accuracy was not improved if pulmonary infarction and embolism were combined in the pathological and radiographic analysis, as in Table V (for radiologist A, $\chi^2=6.93$, $P<0.01$, and for radiologist B, $\chi^2=3.24$, $P<0.10$). Agreement between radiologists is expressed in Table VI ($\chi^2=42.7$, $P<0.01$ for infarction; $\chi^2=10.4$, $P<0.01$ for emboli). The time of the radiograph in relation to the age of the infarction made no difference to diagnostic accuracy.

<table>
<thead>
<tr>
<th>Pathology</th>
<th>No Emboli or Infarction</th>
<th>Abnormality of Infarction/Emboli</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Radiologist A</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary emboli/infarction</td>
<td>32</td>
<td>34</td>
</tr>
<tr>
<td>Control pathology</td>
<td>44</td>
<td>13</td>
</tr>
<tr>
<td><strong>Radiologist B</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary emboli/infarction</td>
<td>36</td>
<td>30</td>
</tr>
<tr>
<td>Control pathology</td>
<td>41</td>
<td>16</td>
</tr>
</tbody>
</table>

FIG. 1. Radiograph of a patient who subsequently died with pulmonary embolism/infarction: note high right diaphragm and cardiomegaly.
Radiographic signs of pulmonary embolism and pulmonary infarction

FIG. 2. Radiograph of a patient who subsequently died with pulmonary embolism/infarction: note basal lung collapse with a linear atelectasis at the right base.

| TABLE VI |
|------------------|------------------|------------------|
| AGREEMENT AND DISAGREEMENT BETWEEN RADIOLOGISTS AND THEIR CORRELATION WITH PATHOLOGICAL DIAGNOSIS |
| | Agreement among Radiologists | |
| | Correct | Incorrect | No. of Cases |
| Emboli Infarction | 48 | 34 | 82 |
| 76 | 26 | 102 |
| Disagreement among Radiologists | | |
| Correct (A) | Correct (B) | No. of Cases |
| Emboli Infarction | 18 | 23 | 41 |
| 16 | 5 | 21 |

DISCUSSION

From this retrospective controlled necropsy study, two radiologists were able to diagnose pulmonary infarction correctly in 70% of cases.

Smith (1938), Short (1951), Macleod and Grant (1954), Smith (1953), and Stein et al. (1959) have all described radiological abnormalities in pulmonary infarction but all these series have been predominantly of clinical cases without necropsy confirmation. Although these abnormalities may occur in infarction they do not all appear to be of great diagnostic value.

Short (1951) found pleural involvement in 56% of patients with pulmonary infarction, but pleural effusion was found in only 33%. In the present study 26-1% of patients with pulmonary infarcts had small unilateral effusions, and 12-3% of patients with pneumonic consolidation. Chang (1967) and Chang and Davis (1965) suggested that dilatation of one or other pulmonary artery was an important sign of pulmonary infarction but their observations were uncontrolled and few necropsies were reported. Dilatation, which later regresses, in conjunction with other changes is useful supportive evidence of pulmonary infarction. However, an isolated observation (which is usually all there is) of a left pulmonary artery of 17 mm or more in width, or a right pulmonary artery of more than 16 mm, with measurements made at the widest point of the arteries (usually between the eighth and ninth ribs posteriorly in deep inspiration) does not seem of any value in the diagnosis of infarction or embolism. In this series, measurements were made when the vessels were visible on such radiographs and no difference
was observed between the controls and patients with pulmonary emboli or infarction. Oblitera-
tion of pulmonary vessels and areas of radio-
translucency were also inadequate signs of emboli.

Radiological evidence of embolism without in-
farction was stressed by Westermark (1938). He
found absence of pulmonary vascular shadows,
sometimes with enlargement of the hilar shadow
after large emboli, and others have also noted
this (Teplick, Haskin, and Steinberg, 1964;
Arendt and Rosenberg, 1959; Shapiro and Rigler,
1948; Torrance, 1963; Woesner et al., 1953).
Stein et al. (1959) did not observe it and in the
present study only one case satisfactorily showed
it. In animal experiments it was impossible to
produce an area of radiological oligaemia (Mar-
shall et al., 1963). This sign of Westermark has
also been reported in chronic pulmonary artery
thrombosis (Hollister and Cull, 1956) but it is
often found in areas of emphysema.

Laur (1963) thought that visualization of a
defect in a vessel was a direct sign of embolism,
but this was not seen in this study. Hyperaemia
on the opposite side to an embolism (Fleischner,
1959) and pulmonary oedema due to an embolism
(Fleischner, 1959) were not observed in this study.
Left ventricular failure was often found in both
groups in the present study but there was no evi-
dence of an association between this and embolism
and infarction. Fleischner (1962) considered areas
of radiotranslucency or abrupt termination of
blood vessels as important radiographic signs of
emboli but these signs seem rare and not easy to
distinguish from areas of emphysema. Areas of
pulmonary shadowing or cardiomegaly may ob-
scure the pulmonary vessels and only serial films
will elucidate such changes.

The failure of the radiologists to diagnose pul-
monary embolism was not due to the method of
analysis. They rarely noted radiological features
of emboli. Therefore any other method of analy-

sis would have resulted in a larger number of
false negative diagnoses and would not have im-
proved the diagnostic accuracy. The standard of
the radiographs in the control and embolic groups
was similar, in view of the comparability of their
clinical condition, and an error in diagnosis of
pulmonary embolism/infarction was as often
false positive as false negative in both control
and embolic groups.

There were equal proportions of surgical cases
in both control and embolic groups although
there were few traumatic cases in the control
group. Therefore elevation of the diaphragm
after laparotomy could not explain the high inci-
dence of this finding in the embolic group, and
the inequality of such elevation on the two sides
was often of diagnostic value even in supine radi-
ographs. However, the radiologists’ diagnostic
accuracy could have been increased by the exclu-
sion of infarcts that were too recent for the
analysis. There is doubt as to the interval between
the onset of embolism and symptoms and signs
of infarction (and sometimes of embolism) occu-
ring, and although such features had often occur-
red they could not be used to date the time of
infarction. Thus smaller and earlier infarcts, if
included, might have been missed by the radio-
logists.

The usual criticism of a retrospective necropsy
study is that emboli may be missed. Since in all
the control patients the diagnosis of pulmonary
embolism was under consideration it is unlikely
that emboli in the segmental vessels or above
were missed. Smaller emboli are of unknown sig-
ificance. In the embolic group all the emboli may
not have been documented but it is unlikely that
such omissions have decreased the accuracy of
the radiologists’ diagnosis of embolism.

The only other necropsy study of pulmonary
embolism and infarction was that of Hampton
and Castleman (1940) in which necropsy findings
were correlated with chest radiographs performed
post mortem in the erect position. This may be
why they did not detect elevation of the dia-
aphragm. An infarct could be of any shape in
the PA projection but often appeared like a
cushion. They stressed that in the PA or AP
radiographs the shadow may be masked by the
heart or diaphragm, and that it was missed in
25% of cases. It was easily confused with areas of
pulmonary oedema, infection, and atelectasis.

Linear atelectasis are signs of old infarction
(Fleischner, 1962). Simon (1970) felt that such
lines could be due to secondary pulmonary
venous thrombosis and then the line was directed
to the hilum. In retrospect only one case could
be considered to have this sign and only one
other had a small linear shadow which was due
to an old infarct. Good lateral radiographs would
probably have helped in detecting such lesions
but are usually unobtainable.

Although by no means diagnostic (Laur, 1963;
Barritt and Jordan, 1961), the most useful sign
of pulmonary infarction in PA radiographs in
the present study was an elevated diaphragm.
However, this sign was present in less than 50%
of cases and its absence should not make the
clinician exclude pulmonary embolism and
infarction.
We are grateful to the Departments of Radiography and Photography for their assistance, and also to the Department of Pathology for necropsy details.

REFERENCES
