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CAVITATION IN BLAND INFARCTS OF THE LUNG*

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Misfortunes always come in by a door that has been left open.—(Czech proverb)

PULMONARY INFARCTION usually occurs in patients who are already ill from cardiac failure, and these people are not in good shape to resist any further weighting of the scales against them, such as could be brought about by another infarction or by the degeneration and infection of an existing one. They desperately require the support of corrective therapy, which must be kept up for an adequate length of time.

Although little attention has been given in the literature to the occurrence of abscess formation and cavitation in bland lung infarcts, we believe that this complication is an important one, for its supervention makes the proper treatment of lung infarction even more imperative. If the possibility of its presence is not borne in mind, it may well be overlooked and the worsening of the clinical picture be ascribed to causes less easily combated.

In two years we have seen five cases in which bland pulmonary infarction progressed to cavity formation. We report them because we believe that they show the necessity of persisting with the prime essential of treatment, anticoagulants, and also to draw attention to a lesion which cannot be as uncommon as has been suggested in the literature.

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CASE 1

A 56-year-old farmer, under treatment at home for congestive cardiac failure secondary to chronic hypertension, suddenly noticed the onset of dyspnœa at rest and hæmoptysis, and became greatly apprehensive. A friction rub appeared at his right base; a right basal pneumonia was reported by the radiologist and penicillin was prescribed.

His congestive failure did not improve and three weeks later he was sent to hospital. On admission, fluoroscopy revealed a greatly enlarged heart and infarction in the right lower lobe. Dicoumarol was given and a therapeutic level of prothrombin in the blood maintained for the next six weeks. At the end of that time he left our service, although he was still in congestive failure, and aureomycin was administered instead of the anticoagulant, with the addition of Digoxin and Salyrgan.

Only three days after the cessation of dicoumarol another lung infarction occurred, as shown by the sudden onset of pain in the right chest, with increase of dyspnœa. Pleural friction recurred, and increased density at the right lung base with elevation of the diaphragm was seen in the radiograph.

During the next three weeks his pulse rate tended to increase in spite of full digitalization; he had sweating attacks and a persistently raised sedimentation rate, but his white cell count never exceeded 8,000. Twenty days after the occurrence of the second clinical infarct he brought up a large amount of foul sputum containing Friedländer's bacillus. A cavity at the base of the right lung was reported by the radiologist. For the next few days he continued to expectorate two potfuls of foul sputum daily. Two attempts at surgical exploration and drainage of the cavity were made but were unsuccessful; no anticoagulants were given. The patient's condition rapidly became worse and he died. At autopsy numerous adherent thrombi were found in the pulmonary vessels of the lower right lobe, which was firm with areas of recent and old infarction. There was an abscess cavity 3 cm. in diameter within one of these areas. The left lung also showed a large hæmorrhagic infarct along the sulcus of the lower lobe. The heart weighed 640 gm. and the left ventricular wall was 20 mm. in thickness. There was a healed myocardial infarct anterolaterally.

CASE 2

A business man aged 48, a known hypertensive, was admitted to hospital with auricular fibrillation and congestive failure. With Digoxin, salt restriction and Salyrgan the œdema cleared rapidly. On his 16th hospital day he was awakened by severe pain across the front of his chest radiating to his right shoulder, and he had tenderness of the left calf, while his right arm became swollen. On x-ray examination, cloudiness of the right base was seen. Pulmonary infarction and right axillary thrombosis was diagnosed.

Canad. M. A. J.
June 15, 1955, vol. 72

Therapeutic prothrombin levels were established and maintained in the blood with dicoumarol in spite of repeated hæmoptyses. The patient remained desperately ill and the congestive failure increased, so that daily injections of Salyrgan became necessary. He had no fever, but his pulse rate showed a steady rise: his erythrocyte sedimentation rate, which was normal on admission, increased to 68 mm. in the first hour and his white cell count from 10,000 to 14,000, with 76% neutrophils.

Seventy-four days after admission, he expectorated large amounts of brownish sputum which on culture grew pneumococci and *Strep. viridans*. As these organisms were sensitive to chloramphenicol 2 gm. of this was given daily in addition to dicoumarol and he began to recover from his heart failure. Dicoumarol was continued for another two months and the sputum gradually diminished. Seven months later the cavities had disappeared and the patient was ambulant.

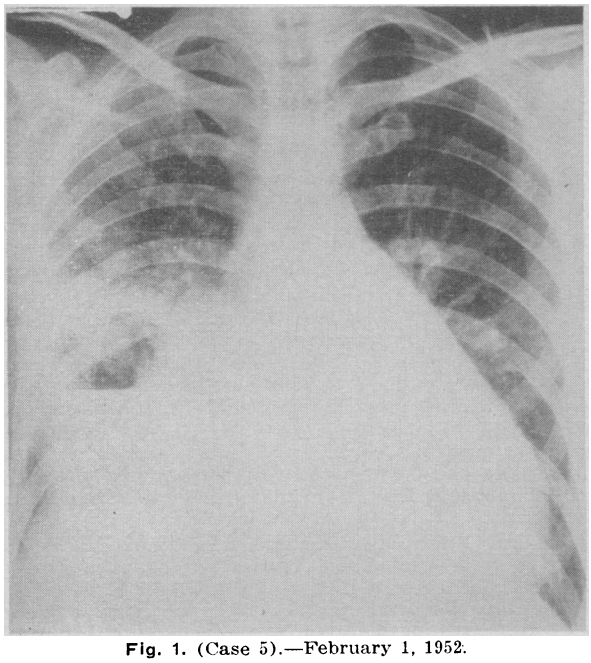

Fig. 1. (Case 5).—February 1, 1952.

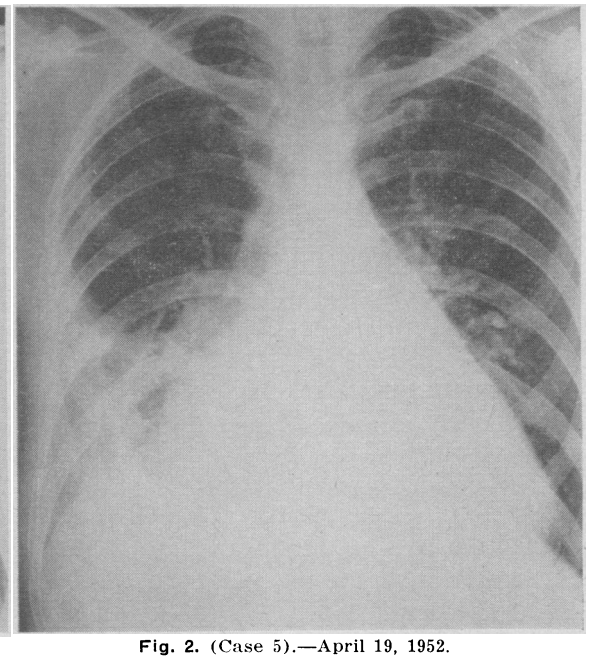

Fig. 2. (Case 5).—April 19, 1952.

CASE 3

A 69-year-old farmer was admitted with hypertensive failure and auricular fibrillation. The response to digitalis and bed rest was good but on his 4th hospital day he complained of substernal pain with increase of dyspnœa and became very frightened. His pulse rate went up to 140; there was some fever and his peripheral œdema got worse. The pain extended to the right side during the next two days and was exacerbated by coughing or breathing; Homans's sign was positive in the right calf. The radiograph showed consolidation in the middle zone of the right lower lobe with a little pleural effusion.

Pulmonary infarction was diagnosed and adequate dicoumarol given. The fluid at the right base, aspirated on three occasions, was thin and blood-stained. He continued to run a temperature of 99-101° F.; his white cell count rose from 9,000 on admission with 74% neutrophils to 16,500 with 85% neutrophils; and his erythrocyte sedimentation rate increased from normal to 87 mm. in the first hour. His general condition remained poor.

On the 23rd hospital day he began to spit blood; large amounts of chocolate-coloured sputum appeared during the following week and on the 28th hospital day cavitation of the right lower lobe was seen in the radiograph. Sputum culture yielded hæmolytic streptococci, and penicillin was prescribed. With the release of the pus from the pulmonary abscess his cardiac failure began to respond once more to therapy, but unfortunately the dicoumarol was discontinued before the patient had got up and about. Two weeks later another attack of substernal pain heralded further lung infarction, this time in the left lung where new crepitations were heard. X-ray examination showed cloudiness of the lower lung field with elevation of the diaphragm.

Dicoumarol medication was started again and this time was continued for two months, until the patient was well enough to walk and the cavity had almost disappeared. In the spring of the following year the cavity could no longer be seen.

CASE 4

An 80-year-old man, who three years earlier had suffered a coronary thrombosis, was admitted with auricular fibrillation and congestive heart failure. He made slow progress on routine treatment with digitalis and Salyrgan. On his 32nd hospital day he complained of severe substernal pain and breathlessness; cyanosis was observed, with impaired movement and crepitations at the right base. No anticoagulants were prescribed, and recovery from this episode was protracted.

Four months after admission, he had a second attack of right-sided chest pain, with hæmoptysis, fever and breathlessness. Considerable cloudiness of the lower right lung field was seen in a chest film, with marked left ventricular enlargement. Although it seemed clear that further lung infarction had taken place, no dicoumarol was given.

During the next few days his temperature rise persisted; his white cell count rose to 23,000 with 96% of neutrophils; he continued to spit blood and one morning his bed linen was found badly blood-stained from his expectoration during the night. A radiograph that day, eight days after the second lung infarction, revealed cavitation with a fluid level at the right lung base; and sputum culture yielded hæmolytic staphylococci and streptococci.

He died two days later. At autopsy a right lower lobe cavity measuring 8 cm. by 5 cm. containing greenish pus was found, which extended to the pleura. The pul-

monary artery to the right lower lobe was completely occluded by ante-mortem thrombus. There was a fresh infarct besides the older one in which the abscess was located. There were several small infarcts in the left lung. Both coronary vessels showed atherosclerosis.

CASE 5

A 50-year-old man with rheumatic mitral disease who was under treatment at home for congestive failure, was suddenly attacked by right-sided chest pain, with increased breathlessness, and hæmoptysis; his wife noticed deepened cyanosis and distension of his neck veins. His temperature rose to 100° F., and consolidation of the right middle lobe with a small effusion was observed on x-ray examination. Pulmonary infarction was diagnosed.

He was admitted to hospital and immediately given dicoumarol. Although therapeutic prothrombin levels were maintained, his general condition remained poor. His temperature returned to normal, but he had sweating attacks and his pulse rate tended to rise; there was marked anorexia. His white cell count was within normal limits, but his erythrocyte sedimentation rate rose to 107 mm. in the first hour and he was very anxious and apprehensive.

One morning he vomited eight ounces of foul-smelling sputum and during the next few days expectorated further large quantities, which on culture grew terramycin-sensitive pneumococci. His general condition took a marked turn for the better; his pulse rate settled and his appetite returned. A chest film now showed a large cavity in the right lower lobe with a fluid level (Fig. 1) and an enlarged left ventricle.

Surgical drainage was advised by a consultant, but as this would have meant interrupting the dicoumarol therapy, it was decided to continue with the anticoagulant plus terramycin. He slowly improved, without radical measures; and four months later the cavity appeared closed on x-ray examination (Fig. 2). The patient left hospital and returned to work.

DISCUSSION

The incidence of cavitation in bland lung infarcts is not settled. Levin, Kernohan and Moersch[8] consider that infection together with cavitation is liable to happen only in very large infarcts in which collapse of the segment has developed, and in which the blood supply has become so precarious that avirulent organisms normally present in the bronchioles invade the degenerating cells. They believe that in most infarcts the blood supply is so abundant that infection does not occur. Schwedel[10] holds similar views.

Gsell,[3] on the other hand, thinks that infarction with secondary infection is a common cause of lung abscesses. He suggests that the lesion is often misinterpreted by pathologists who, finding polymorphonuclear infiltration at the margin of an infarct, fail to observe the thrombus within the artery and conclude that the abscess has formed within a segmental pneumonia.

The condition cannot be an extreme rarity, because we have seen five cases over a period of two years, and in a recent report Harvey and Sabiston[5] describe five patients who developed empyemata following abscess formation in lung infarcts; all were treated in one hospital over a period of three years. There is no doubt that failure to recognize the abscesses makes them appear more uncommon than they really are; a review of post-mortem findings published since 1930 gives an incidence of 2.7% of cavities in the combined series; a clinical diagnosis was made in less than half of the cases.[1 to 4, 6 to 14]

Even the antecedent infarction can easily be missed. In drawing attention to the difficulties, Schwedel points out that in the so-called asymptomatic cases there may be a change in the clinical course of the patient, or a failure to respond to diuretic or other drug therapy which had previously been effective, as a result of infarction. It is so frequent in cardiac-failure patients treated in bed that it should always be sought when the condition of the patient changes for the worse.

The radiological diagnosis has been discussed by Smith.[12] There may be vague clouding of the base of one lung, obscuring the costo-phrenic angle and suggesting an influenzal pneumonitis, or shadows indicating an effusion. More localized shadows may be not unlike those of lung abscess, and may develop appearances indicating cavity formation. There may be elevation of the diaphragm, pointing to partial basal collapse. Linear shadows may represent scars of past infarcts or present thrombus formation. Triangular or wedge-shaped shadows are only occasionally observed.

The onset of abscess formation after infarction has been described by Chester and Krause.[2] The distinguishing clinical feature is the appearance of a foul, purulent sputum days or weeks after the onset of the infarct. It is usually accompanied by a secondary rise in temperature and an elevation of the leucocyte count; and in patients with heart disease by an increase in the severity of the cardiac failure. Levin *et al.*[8] indicate the clinical course with more clarity. They describe seven patients in whom pulmonary infarction had been followed by a quiescent period which in turn had been followed by a period of fever with leucocytosis, and had resolved after the production of purulent sputum. Autopsy revealed cavitation but x-ray evidence of this during life had sometimes been lacking. In each of the cases described here the occurrence of lung infarction was shown by characteristic signs and symptoms;

the x-ray appearances were those of cloudiness at one base, effusion, consolidation, or elevation of the diaphragm, as described by Smith.

Cavitation was first seen in three of the cases about 20 days after infarction, in one 58 days after, and in the fifth 8 days after the second of two clinical infarctions. In all five, good evidence that trouble was developing in the lung infarctions became available; the important points observed were the intractability of the cardiac failure; the persistence of fever, or of increased pulse rate, or both; the occurrence of a leucocytosis, or a raised erythrocyte sedimentation rate, or both; and less well-defined symptoms of a continuing malaise such as anxiety, apprehension, anorexia, and sweating.

The value of anticoagulant therapy, which must be continued while the patient is bedridden, is suggested by our experience with the five patients. In the first, dicoumarol therapy was interrupted because it was thought that he had recovered from one infarction; another followed almost immediately and progressed to abscess formation. The second and third patients received anticoagulants throughout their illness, in spite of the repeated hæmoptyses which were seen in the second case; both eventually made a good recovery. The fourth had no anticoagulants; he died after attempts had been made to drain his lung abscess by surgery. The fifth case gave rise to some discussion; one consultant advocated surgical drainage of the lung abscess, but as this would have meant a cessation of treatment with dicoumarol his advice was not followed and therapeutic prothrombin levels were maintained in the blood until the patient became ambulant.

Antibiotics were given as adjuvants, but we believe the cardinal principle of treatment to be the maintenance of impaired clotting power in the blood throughout the period of bed care. That very large abscesses may heal with this regimen is well shown in Figs. 1 and 2.

Probably all people with cardiac failure bad enough to require bed treatment should be given anticoagulants.

SUMMARY

Five cases in which cavitation occurred in bland lung infarcts are described. The importance of adequate and persistent anticoagulant therapy in patients with cardiac failure is stressed.

We wish to thank Dr. A. J. Glazebrook, Director of the Department of Medical Research, St. Boniface Hospital, for his assistance.

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PSEUDOCYSTS OF THE PANCREAS: REPORT OF FIVE CASES*

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THE INCIDENCE of pseudocysts of the pancreas is low. The admission of five cases to this hospital within one year (16,089 admissions) prompted this report.

At the Mayo Clinic, 88 cases were found in 700,000 admissions, and at the Henry Ford Hospital 21 cases in 650,000 admissions.

By definition, pseudocysts are cystic collections of fluid without epithelial lining in the peripancreatic tissues of the lesser peritoneal sac. Confusion exists in differentiation from true cystic lesions, such as retention cysts, if pressure necrosis or autodigestion of the epithelial lining occurs. Precise pathological diagnosis cannot always be attained, as excision of all or part of these cysts is difficult.

Pseudocysts arise from the liberation of pancreatic ferments, lipase and trypsinogen, into the

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