the right iliac fossa; part of it hung down into the pelvis (Fig. 2). It was removed after separating the rather vascular adhesions binding it to the ileum and the posterior abdominal wall. With the exception of slight post-operative chest complications, the patient progressed satisfactorily and was discharged on December 19. Histology of the specimen removed showed necrotic omental fat tissue with vascular thrombosis. In some areas there was evidence of organization and recanalization of the vessels.

FIG. 2.—Case 3. Partially necrotic great omentum adherent to the antimesenteric border of the ileum.

The operation in this case was performed about a month after the onset of symptoms. It is reasonable to assume that torsion was the cause of complete separation of omentum from its normal attachment; partial revascularization had taken place following its further attachment to the small bowel and postero-lateral abdominal wall.

Comment

The pre-operative diagnosis of torsion is seldom made; about 80% of cases are diagnosed as appendicitis, but, considering the duration of symptoms, the patients look less toxic than do cases of appendicitis. The age incidence varies between 35 and 55 years, and the condition is twice as common in males. The three cases reported here were all those of men between 45 and 50.

Cases 2 and 3 are examples of secondary torsion, the third case being associated with intraperitoneal adhesions, and the second with a right inguinal hernia, though there was no evidence of adhesions between the hernial sac and the omentum. No definite cause was found for torsion in Case 1, which can therefore be grouped as an example of primary or idiopathic torsion.

I wish to thank Mr. T. G. I. James for his advice and for permitting me to publish these cases, which were admitted under his care. My thanks are also due to Miss D. M. Barber and Miss E. D. Hewland, the medical artists, for the drawings.

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SPONTANEOUS PNEUMOTHORAX FROM SECONDARY SARCOMA OF LUNG

BY

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The association of pulmonary metastases with spontaneous pneumothorax is rare, and is not described in such standard textbooks of radiology as those of Twining (1938) and Golden (1941). No recorded case of pneumothorax from secondary carcinoma of the lungs has been found in the recent literature. Thornton and Bigelow (1944) described two cases of spontaneous pneumothorax due to secondary sarcoma of the lung, and Lodmell and Capps (1949) added three further cases. In view of the apparent rarity of the condition, the following case is of interest. It showed bilateral spontaneous pneumothoraces following pulmonary metastases from a leiomyosarcoma of the uterus.

Case Report

A woman aged 51 was admitted to Brompton Hospital on March 19, 1949, for investigation of her chest. She was nulliparous, and her husband had died 15 years earlier from pneumonia. She had spent the past four years in Johannesburg. She had had no relevant previous illnesses, except an operation in May, 1948, when a subtotal hysterectomy had been performed for fibroids. No further details of this operation are available. She had since remained in good health until January, 1949, when she fell ill with an influenza-like illness. A radiograph of the chest revealed a left-sided pneumothorax and scattered rounded opacities in both lung fields; there were also bilateral pleural effusions. A provisional diagnosis of multiple secondary deposits was made and the differential diagnosis of hydatid disease was considered. As her relatives were in England, she was flown home on March 18.

On admission to Brompton Hospital next day she was orthopnoeic and had a temperature of 101° F. (38.3° C.). She had lost a stone (6.4 kg.) in weight since January, 1949, and had coughed up flecks of blood on two recent occasions. Bowel habits and micturition were normal, and there had been no recent vaginal discharge or bleeding.

On examination she was pale and showed signs of loss of weight. She had a remittent fever up to 102° F. (38.9° C.) while in hospital. No enlarged lymph nodes were felt. Air entry was diminished over both lungs, and there was dullness with scattered rales in the left upper and right lower zones. The liver was not palpable, and there was a right lower paramedian scar; vaginal examination revealed a fixed cervix and a firm mass in the right fornix.

Investigations.—The urine contained nothing abnormal. A blood count showed: Hb, 75%; red cells, 3,800,000 per c.mm.; white cells, 12,000 (polymorphs 76%, lymphocytes 8%, monocytes 13%, eosinophils 2%, basophils 1%). E.S.R. (Westergren) 76 mm. in one hour. Repeated examinations of the sputum showed no malignant cells, though in one examination a few clumps of "suspicious cells" were revealed. No tubercle bacilli were seen. The Casoni test (intracutaneous injection of hydatid fluid) was negative.

A chest radiograph on March 22 (see Illustration) showed bilateral pneumothoraces, that on the left side being now localized to the apex. In this region there was a large rounded opacity situated anteriorly, as seen in the lateral view, and a localized hydropneumothorax. Both lung fields showed several rounded shadows. Intravenous pyelography revealed no abnormality.

A punch biopsy was performed on April 6. A Silverman needle (kindly lent by Professor D. W. Smithers) was intro-

Radiograph of chest on March 22, showing bilateral pneumothoraces, that on the left side being localized to the apex. There are multiple rounded opacities in both lung fields, and fluid is present at both bases.

duced into the first left interspace $1\frac{1}{4}$ in. (4.4 cm.) lateral to the margin of the sternum, and the specimen reported upon as follows: "The material provided is that of a cellular malignant tumour. The tumour cells are very pleomorphic anaplastic cells with large deeply staining nuclei. The material is insufficient for making a definite diagnosis."

The patient was considered unsuitable for radiotherapy, and was therefore given symptomatic treatment only. Her condition slowly deteriorated, with a remittent fever, until her death on April 25.

Necropsy Report (extracted from Brompton Hospital post-mortem register).—The right pleural cavity contained a large pneumothorax with collapse of the upper lobe. The right lung was extensively infiltrated by deposits of tumour, some of which had caused ulceration of the visceral pleura. The left pleural cavity was obliterated except at the apex, where there was a small pneumothorax. Multiple deposits were present throughout the left lung, and a large deposit in the apex of the left upper lobe contained necrotic blood-stained material. The left main bronchus contained a mass of growth, 4 in. (10 cm.) long, lying in the bronchus and arising from a tumour mass in the lingula. In the pelvis the body of the uterus had been removed. The right ovary was replaced by a hard, white, irregular tumour, approximately 3 in. (7.5 cm.) in diameter. The left ovary was cystic and its contents were blood and necrotic tissue. The cervix, vagina, and bladder were normal. There was no macroscopic evidence of secondary deposits in the liver or other organs.

Histology.—"The mass replacing the right ovary is a well-differentiated leiomyosarcoma. Even in this lesion, however, there are areas where the tumour is highly pleomorphic, and other areas show extensive necrosis. The wall of the left ovarian cyst is lined by a zone of pleomorphic tumour. The frond-like mass in the left main bronchus is pleomorphic, contains many giant cells, and shows extensive necrosis. The degree of differentiation of the leiomyosarcoma in the various lung metastases is extremely variable. Some are anaplastic with numerous giant cells, others show almost normal differentiation. Necrosis is widespread."

Comment

The clinical findings and radiological appearances suggested the diagnosis of multiple pulmonary metastases. The differential diagnosis of hydatid disease had been considered in South Africa, for hydatid cysts may produce multiple rounded shadows in the lung fields in association with a pneumothorax. However, the finding of a mass in the pelvis favoured the diagnosis of metastases, and this was confirmed by punch biopsy. The necropsy showed that the pneumothoraces were almost certainly caused by broncho-pleural fistulae arising from necrotic pleural metastases. Partial re-expansion of the left lung had occurred during life, from the growth of the large mass in the left upper lobe.

The histological appearances of the tumours in the ovaries and lungs were those of leiomyosarcoma. It is thought probable that these deposits were secondary to the uterine tumour which had been removed 11 months before the patient's death, and that this was a leiomyosarcoma and not a benign fibroid. Willis (1948) has cited a case in which the interval between hysterectomy for supposedly simple "fibroids" and the development of leiomyosarcomatous metastases in the lungs was almost two years. An adequate histological study will usually reveal whether a uterine fibroid is benign or malignant, and it is a well-recognized experience to find suspiciously sarcomatous areas in a fibroid which is clinically benign (Willis, 1948). Less frequently a tumour may appear benign after careful study, but its malignant nature is proved by its subsequent clinical behaviour (Steiner, 1939). It has not been possible to examine the tumour removed by hysterectomy in the present case, but its malignancy seems to have been proved by the subsequent development of metastases.

Spontaneous pneumothorax may occur in association with a primary bronchial carcinoma, especially in the later stages, when there is secondary abscess formation and tissue gangrene (Young, 1936; *Brit. Ency. med. Pract.*, 1949). A pneumothorax may also result from the local spread of a malignant tumour—for example, oesophagus or heart (Perry, 1939). However, the association of a spontaneous pneumothorax with secondary pulmonary metastases has been recorded only in recent years. Of the two cases described by Thornton and Bigelow (1944) one was due to spread from a fibrosarcoma of the hand that had been amputated five years earlier, and the second followed an amputation of the leg for an osteogenic sarcoma of the femur. In both instances the pneumothoraces were bilateral. Lodmell and Capps (1949) described three further cases. In the first, pulmonary metastases and bilateral pneumothoraces developed one month after a mid-thigh amputation for an osteogenic sarcoma of the tibia, and in the second case the primary growth was an inoperable sarcoma of the neck. The third case was an angiosarcoma of the retroperitoneal space; multiple pulmonary metastases and a spontaneous pneumothorax developed five months after laparotomy.

The mechanism of production of a pneumothorax due to secondary metastases has been discussed by Lodmell and Capps. The most likely explanation is that the pneumothorax is caused by a broncho-pleural fistula following necrosis of a subpleural nodule, and this was probably the method in the present case. Lodmell and Capps, however, failed to demonstrate such a fistula in two of their three cases. They suggested that in these cases the tumour nodules had produced a ball-valve

obstruction of the bronchi, with distension of the peripheral alveoli. These distended alveoli might subsequently rupture direct through the visceral pleura or into the interstitial spaces. In the latter case the air may then track along intrapulmonary tissue planes to the mediastinum, or peripherally to the subpleural surface, when blebs may form and subsequently rupture.

The rare occurrence of spontaneous pneumothorax in association with metastatic tumours of the lung is somewhat surprising, as lung necrosis is a conspicuous feature in such cases, and pleural perforation might be expected more commonly. Certainly few such cases have been recorded.

I wish to thank Dr. Neville Oswald for permission to publish this case. For the reports on the pathological findings I am indebted to Dr. J. W. Clegg.

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HAEMOLYTIC TRANSFUSION REACTION DUE TO AN ANTI-LEWIS^a AGGLUTININ

BY

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AND

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In April, 1950, a blood sample was sent to our laboratory from a woman aged 25. She had been admitted to the surgical department of this hospital, where a cholecystectomy was performed by Professor Dr. I. Boerema. Two hours after the operation she developed a severe shock, which was treated with blood transfusions. The total amount of blood given, from three different donors, was 1,500 ml. During the infusion of the second bottle there was a mild transfusion reaction with chill and a rise of the temperature to 38.7° C. The transfusion, however, was not discontinued, and blood from the third donor was endured without further

reactions. Nevertheless this patient showed the typical signs of a haemolytic transfusion reaction—that is, oliguria with haemoglobinuria—but made an uneventful recovery on being treated in the usual way.

A recheck of the patient's and donors' blood was made. Tables I and II show the results of the blood grouping and cross-matching of the patient's serum with

TABLE I.—Blood Groups

Patient	..	..	..	A ₁	..	MN	..	cde
Donor I	..	..	..	A ₂	..	MN	..	cde
Donor II	..	..	..	A ₁	..	MM	..	cde

cell suspensions of donors I and II. An Rh or M or N antagonism could be excluded. The patient's serum contained a cold agglutinin which was still active at body temperature. Incomplete antibodies could not be detected by the indirect Coombs test. We could as well exclude an A₁ and A₂ antagonism, the patient's serum reacting with both A₁ and A₂ cell suspensions at different temperatures. Out of 27 cell suspensions six were agglutinated at 37° C. and five at 44° C.—that is, in about 20%.

On the basis of this frequency we excluded the possibility of an antagonism of the systems H, Jobbins, Kell, and Cellano, and thought a Lewis antagonism most probable, for the following reasons: (1) we were able to prove the connexion between the secreting properties of the patient and the intergroup reaction under discussion, the patient being a secretor, the first donor a non-secretor, and the second donor a secretor; and (2) no agglutination occurred if the patient's serum was mixed with saliva of a secretor and red cells of the first donor (saliva inhibition test).

On these observations we felt justified in concluding that it was a haemolytic transfusion reaction due to the presence of an anti-Lewis^a agglutinin. It is therefore the blood of the first donor that must have been responsible for the haemolytic reaction.

Dr. van Loghem kindly controlled the serological examination in his laboratory. He could exclude the factors C^w, S, P, and Duffy as eventual causes of the transfusion complication (Table III). He also compared our serum with a known anti-Lewis^a serum and found identical figures. Hence he could confirm our results.

The occurrence of anti-Lewis^a agglutinins in human sera has been described several times already. However, it seems to be the first time that this antibody has given rise to a haemolytic transfusion reaction.* Fortunately the consequences of this discovery do not seem to be serious from the clinician's point of view. We may easily believe that the frequency of these reactions will be extremely low. Yet the occurrence of even such rare reactions compels us to take the necessary precautionary measures.

*It was learnt from a personal communication that both Mr. I. Dunsford (Sheffield) and Dr. M. M. Pickles (Oxford) saw similar transfusion reactions. Neither case, however, has so far been published.

TABLE II.—Cross-matching

		37° C.			Room			3° C.			
		1:1	1:2	1:4	1:1	1:2	1:4	1:1	1:2	1:4	1:8
Serum patient	Saline	++	+	±	++	+	±	+++	+++	+	±
Cells donor I	Albumin	+	—	—	—	—	—	+	±	—	—
Serum patient	Saline	—	—	—	—	—	—	+	±	—	—
Cells donor II	Albumin	—	—	—	—	—	—	+	±	—	—