A case of lymphangitis carcinomatosa of the lung from carcinoma of the prostate

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Summary of the Clinical Findings (Dr. F. Clifford Rose)

A.P. A male, aged 84 years, (hospital index number K.7330) was admitted as an emergency to Westminster Hospital on 20th September, 1955. He had been poorly and short of breath for the past six years but had become much worse in the last three or four months. About four months ago he noticed that his ankles were swollen and, on direct questioning, he said that the swelling did not diminish in the mornings. During the week previous to his admission he had been rambling and confused, and had persistent vomiting unrelated to meals. He was constipated, his appetite was poor but he was apparently not losing any weight.

On examination he was an old man with a sallow complexion. He was extremely dyspnoeic at rest but was not cyanosed.

Cardio-vascular System. His pulse was regular and of a fair volume. The heart sounds were normal. There was an apical systolic murmur. The blood pressure was 160/80. The neck veins were 6 cms. above the sternal angle, and there was moderate ankle oedema.

Respiratory System. At the right base the percussion note was dull and the air entry was diminished, with reduced vocal fremitus. There were râles at both bases.

Central Nervous System. He was confused and disorientated but there was no evidence of any other abnormality in the central nervous system.

Investigations showed a haemoglobin of 11.8 gms. per cent. and a white blood cell count of 10,000 per cu.mm. The blood urea was 81 mgm. per cent. A portable X-ray of the chest was reported as showing the pulmonary changes of congestive heart failure with basal pulmonary oedema and a small pleural effusion on the right side. Five days later the blood urea had risen to 116 mgm. per cent. but the serum electrolytes were within normal limits. The electro-cardiogram showed no abnormalities. He was treated with Digoxin and Mersalyl, which resulted in some regression of his oedema but his dyspnoea did not improve and he developed progressive laryngeal stridor. He gradually became hoarse and had difficulty in taking fluids. Laryngoscopy nine days after admission showed bilateral oedema of the cords which were covered with a muco-purulent exudate. Eleven days after admission, the temperature rose to 103°F., he became comatose and he died.

The clinical diagnosis was congestive cardiac failure due to coronary insufficiency.

Relevant Autopsy Findings (Dr. George Lumb) (P.M.203/55)

The body was that of an elderly male showing cyanosis of the face. There was a considerable quantity of sticky muco-pus in the upper air passages, and the trachea and main bronchi showed a marked acute inflammation of the mucosa.

The Lungs. There were adhesions between the posterior part of the lower lobe on the right side and the parietal pleura. Throughout both lungs there was a generalized suppurative bronchopneumonia with associated chronic bronchitis. There were numerous small nodules scattered throughout both lungs (Figs. 1 and 2). Over the pleural surfaces there were dilated white vessels forming an irregular tracery under the visceral pleura.

The heart was normal in size and weighed 330 gms. It showed no evidence of hypertrophy of the left ventricle or dilatation of the right ventricle. The aortic valve showed considerable calcification of its cusps but there was no evidence of any other valvular lesion. There was some atheroma round the orifices of the coronary vessels. The remainder of the coronary vessels showed widespread atheromatous changes but there was no evidence of thrombosis, or of obliteration of the lumen. The myo-cardium showed no evidence of fibrosis or other abnormality. In the mediastinum there were numerous enlarged lymph nodes, the largest measuring 1 cm. in diameter. These were hard in consistency and whitish on cut surface. No other abnormal lymph nodes or metastatic deposits were found anywhere else in the body. The thoracic duct was examined carefully but was found to be normal. No enlarged lymph nodes were found elsewhere in the
FIG. 1.—Specimen of lung removed at post mortem.

FIG. 2.—Close-up of surface of lung showing white nodules.

FIG. 3.—Histology of carcinoma in the prostate showing a moderately well differentiated arrangement. Haematoxylin and Eosin x 10.

FIG. 4.—Histology from carcinoma of the prostate showing an anaplastic arrangement. Haematoxylin and Eosin x 70.
body. The urinary bladder was dilated and showed trabeculation of its wall with evidence of chronic cystitis. The distention of the bladder resulted from a gross enlargement of the prostate which, on cut surface, showed the majority of its substance to be soft and fleshy, but posteriorly, on the right side, there was a zone of extreme hardness and grittiness measuring 0.5 cm. in diameter. In the spleen there was a single pale infarct measuring 3 cms. in diameter.

The cause of death was terminal suppurative bronchopneumonia in the presence of lymphangitis carcinomatosa.

Histology from the prostate shows simple hyperplasia throughout most of its substance. In the hard area there is an adenocarcinoma which shows varying appearances in different zones. Some parts show a well differentiated acinar arrangement made up of paley staining cells (Fig. 3). Elsewhere the picture is one of extreme anaplasia with small darkly staining cells showing no characteristic architectural arrangement (Fig. 4). Mitoses are not seen frequently but there are numerous areas of necrosis. There is clear-cut evidence of infiltration of the surrounding muscle.

The lymph nodes from the mediastinum are replaced by adenocarcinoma with the same general appearances as the better differentiated zones in the prostate. The lungs show a widespread purulent bronchopneumonia and in addition a diffuse carcinomatous infiltration. The malignant cells are lying almost entirely in distended lymphatic spaces and are scattered throughout the lung substance showing no greater concentration in the region of the hilum than at the periphery. Perivascular lymphatic blockage is a constant feature (Fig. 5) and there are some zones where malignant cells can be seen bursting out of the lymphatic spaces and infiltrating the alveoli (Fig. 6). The spleen shows an infarct clearly defined from the remainder of the organ. There is thrombosis in some of the adjacent small vessels but no evidence of malignant cells either in the infarcted area or in the remainder of the spleen.

Discussion

Dr. Lumb: Lymphangitis carcinomatosa arising from a carcinoma of the prostate is a well-known, although somewhat unusual, occurrence. The two most common carcinomas responsible for extensive lymphatic invasion in the lung arise in the stomach and the breast. There are examples in the literature of cases in the gall bladder and the colon. I seem to remember that Ewing has described one from a squamous cell carcinoma of the tongue and Willis speaks of one where the primary lesion was in the cervix uteri.

Dr. Rose: In view of the post mortem findings, the X-ray was reconsidered by the radiologist (Fig. 7). His comment was that it was compatible with a lymphangitis carcinomatosa but that it was very difficult to make the diagnosis in the presence of congestive heart failure and it was thought that the mediastinal enlargement might have been due to enlarged lymph nodes.

Dr. R. I. S. Bayliss: What was the diagnosis during life?

Dr. Rose: Cardiac failure.

Dr. Bayliss: Due to?

Dr. Richard Tonkin: It was assumed to be due to diffuse myocardial sclerosis.

Dr. Bayliss: Did the electrocardiogram show evidence of right ventricular preponderance and did you do leads over the right side of the heart?

Dr. Rose: Yes, chest leads were used and there was no evidence of right sided preponderance.

Dr. Bayliss: I know it is easy to be wise after

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FIG. 5.—Clumps of malignant cells in perivascular lymphatics. Haematoxylin and Eosin x 10.

FIG. 6.—Plugged lymphatics shewing some areas where malignant cells are bursting into alveoli. Haematoxylin and Eosin x 12.
the event but, with all due respect, the X-ray does not look very like pulmonary congestion. In the right lung field you can see obvious opacities and there is a generalized streakiness, which extends out to the periphery of the lung. It may be of interest to point out that radiologically you may see oedema of one lung only and we have had several examples of patients with left sided heart failure with unilateral pulmonary oedema.

DR. TONKIN: I agree that it is possible to see on closer scrutiny of this particular X-ray, that the hilar shadow is much less marked on the left side than on the right. One must admit that the diagnosis in this case was preconceived. He was 84 with a history suggestive of myocardial insufficiency. He had all the signs of progressive congestive failure. His response to ordinary measures was inadequate and when he developed disorientation it was assumed that uraemia had developed.

DR. A. D. MORGAN: Was there any evidence of pulmonary hypertension?

DR. ROSE: No.

DR. TONKIN: The heart does appear enlarged in the X-ray but you cannot say more than that because you cannot see the chest on the right side as a result of the congestion.

DR. R. H. B. PROTHEROE: How do you differentiate radiologically between the lung changes of congestive heart failure and uraemia?

DR. BAYLISS: In left sided heart failure there are two main appearances. The first is congestion of the hilar vessels which become more prominent. The second is pulmonary oedema which extends out from the hilum and may be most marked, clinically and radiologically at the lung bases.

Doniach (1947) described a condition which he called uraemic oedema of the lungs. Radiologically the appearance is of centrally located shadows (bat's wings) radiating out from the hila. This oedema may be transitory but, in fatal cases, the histological picture was of an intra-alveolar exudate rich in protein and associated with mononuclear infiltration and organization; Doniach's patients all had hypertension in addition to uraemia, and it is probable that the oedema is related more to hypertensive left ventricular failure than to the uraemia. Thus uraemia per se does not produce lung changes and the condition described by Doniach may be seen in the absence of a raised blood urea.

DR. A. D. MORGAN: The condition Doniach described was a consolidation rather than congestion around the hilum.

DR. ROSE: I have brought along an X-ray of lymphangitis carcinomatosa from the museum. This case is secondary to a carcinoma of breast and shows the typical appearances.

DR. BAYLISS: In the X-ray of the patient under discussion he has more than lymphangitis carcinomatosa, for Dr. Lumb has already told us of the superadded suppurative bronchopneumonia and bronchitis.

DR. LUMB: From the pathological point of view the diagnosis here is perfectly clear-cut and the interest centres on the method of spread from the primary site to the lung. The most commonly accepted theory is that metastatic deposits occur in the hilar and tracheo-bronchial glands which obstruct the normal efflux of lymph from the lungs. Subsequently, there is extension of the tumour towards the periphery by retrograde permeation of the peribronchial and sub-pleural lymphatics. The extent of spread varies. It may be localized to the hilar region or it may extend to the periphery. Distended lymphatics may rupture and invade the lung substance. Tumour occlusions may result in reversal of the lymph flow in the lung itself and then emboli may be carried peripherally and commence separate areas of permeation. As long ago as the middle of the last century Waldeyer suggested that respiratory movements might contribute towards carrying cells out to the periphery once the direction of lymph flow had been reversed. I know that Dr. Morgan has some views of his own on this subject. Perhaps he would be good enough to tell us about them.

DR. MORGAN: In a review of the literature
some years ago, I found that about 15 per cent. of published cases of lymphangitis carcinomatosa were associated with right ventricular hypertrophy. In all of these the pulmonary arterioles were blocked by tumour cells or by thrombosis, recent or organized, and in these cases it seems to me just as likely that the spread through the lung may result from haemagenous metastases with subsequent spread of tumour cells from the small arterioles under the pleura into the perivascular lymphatics. Thus it is possible that the lymphatic spread through the lungs may be from the periphery towards the hilum. It is difficult to see why one should get a retrograde lymphatic spread. Sometimes there are solid columns of tumour growing up the thoracic duct, in which case even if the glands were blocked, there is no particular reason why retrograde extension should occur. It seems to me more likely that the tumour cells from the thoracic duct pass into the venous circulation and reach the pulmonary arterioles via the right side of the heart. There is no histological evidence in these cases of increased lymphatic invasion of the hilar part of the lung as compared with the periphery. If there were retrograde spread due to blockage of lymphatics at the hilum, one would expect the lymphatics throughout the lung to be grossly dilated and, on the whole, they are not.

**Dr. Lumb:** I do not know that I would entirely agree with your statement about dilatation of lymph vessels. Certainly in this case there is evidence of gross dilatation and there is no evidence whatsoever, despite careful examination, of any tumour deposits in arterioles. I have no objection to the idea of retrograde lymphatic permeation for I am sure that it occurs in other sites as well as the lung. A good example is the spread from carcinoma of the stomach into the remainder of the bowel and even as far as the peri-anal skin, where I am sure the mechanism is obstruction of the lymph nodes in the drainage area with retrograde spread back to the bowel wall.

**Dr. Morgan:** There is one point I should like to emphasize in the relationship of cor pulmonale to lymphangitis carcinomatosa. I am quite certain that whatever the mode of spread by lymphatics, the right ventricular hypertrophy results from obliterator changes in the pulmonary vessels, and not from the presence per se of tumour cells in the pulmonary lymphatics.

**Dr. Harold Hewitt:** Is it possible that the carcinoma you have demonstrated in the prostate is, in fact, a carcinoma in situ and that the process in the lung may be unrelated to it?

**Dr. Lumb:** No, I do not think so, because the prostatic carcinoma shows clear-cut evidence of muscle invasion round the tumour and there is a definite appearance relationship between the primary and metastatic growths.

**Dr. Morgan:** I agree with Dr. Lumb’s interpretation of the findings in this case; there is no doubt that the carcinomas remaining confined to the prostate and found by chance at post mortem cannot be regarded in the same light as the truly invasive type of tumour, that is, clinical prostatic cancer.

**Dr. Lumb:** There are several other things that it would have been interesting to discuss, such as the advantages and disadvantages of cytological diagnosis and diagnosis by the method of the open biopsy such as they are trying out in New York. One would have liked to have heard opinions on the relative merits of radical surgery and hormone therapy in the treatment of carcinoma of the prostate but we have managed to keep the discussion confined to the problems of diagnosis and the particular problem of lymphangitis carcinomatosa in this case, and I am afraid we must leave it there for we have come to the end of our time.

**REFERENCE**


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