SECONDARY BRONCHIAL CARCINOMA SIMULATING A STRANGULATED FEMORAL HERNIA

BY

E. WILSON HALL, F.R.C.S.Ed.

A. GEOFFREY SHERA, M.A., M.D., B.Ch.

AND

E. OWEN FOX, M.R.C.S., D.M.R.E.

(From the Princess Alice Memorial Hospital Unit of the
Eastbourne Hospital Management Committee)

[WITH PHOTOGRAPHIC PLATE]

Introduction (A. G. S.)

It is well recognized that bronchial carcinoma is a most protean tumour, perhaps the most protean of all in respect of its metastases, which are not infrequently the first manifestation of the disease. A very large literature has grown up on this subject and we hesitate to add to it, but this case was so unusual and so misleading in the manner of its presentation of this fell disease—so apt first to show itself as a metastasis before the primary tumour is known—that we thought it worth recording for the guidance of others who may perhaps meet its fellow. Another point is that the biopsy pointed quite clearly, dramatically, and unexpectedly to the site of the primary tumour.

Our final justification lies in the statement of Professor Willis (1948), who says: "Because of the frequent misdiagnosis of this disease [and later he refers to glandular metastasis more specifically in this respect], only fully proved necropsy records are of any value for the analysis of its properties." These will be duly presented.

Clinical History

A married woman aged 76 had noticed some three weeks previously a tense and increasingly painful lump in the left femoral region, which enlarged rapidly. On August 8, 1949, she was seen by me (E. W. H.) in consultation with her own doctor, Dr. Ethel Downing, of Eastbourne.

She was admitted to the Princess Alice Hospital the same day as a case of femoral hernia, the notes recording that a large reddish lump was found in the left femoral region, very tense and irreducible, and probably of omental origin, as there was no history of vomiting.

Operation was performed the same day, and on incising over the tumour fluid squirted out under great pressure; it was thick, yellow, and haemorrhagic, but odourless. It was found to be a degenerating glandular mass, which was completely excised down to normal tissue. The wound was drained, and then closed with interrupted sutures, sulpha-penicillin powder having been installed. During after-treatment 2,700,000 units of penicillin were given. The specimen was sent to the pathologist for examination.

Pathological Report (A. G. S.).—The specimen consisted of a large portion of fat and a smaller portion of new growth, measuring 2½ by 1½ in. (6.25 by 3.75 cm.). On section the latter was soft in consistence and white in colour. **Histology.**—The tumour seemed to be a lymph gland, heavily invaded by a bronchial carcinoma showing two phases: (1) polymorphic and oat-cell anaplasia, and (2) squamous metaplasia (Plate, Figs. 1 and 2). It was held to be a bronchial carcinoma of Grade 4 malignancy (Broders's classification).

The chest was accordingly x-rayed on August 19 by E. O. F., who reported as follows: "There was uniformly increased density in the right lower lobe consistent with a localized area of atelectasis and probable bronchial carcinoma. Adhesions were present at the left costo-phrenic angle" (Plate, Fig. 3).

Subsequent History

The wound broke down and the edges sloughed. The patient became somewhat "mental" and persisted in playing with the dressing. She recovered well from the operation and her condition gave no cause for alarm until just before death. There were no chest symptoms at any time until then. The patient went slowly downhill, looking unwell, but with no definite symptoms or signs—for example, anorexia or wasting. Atelectasis of the lower lobe was not found clinically.

On the afternoon of the day of death the patient became drowsy and comatose, but with good pulse and no other signs. She died suddenly at 10 p.m. on September 8, exactly one month after admission to hospital. Her pulse and temperature were raised the day before death.

Post-mortem Examination

The body was that of an elderly woman (5 ft. 9 in.—175 cm.) showing good nutrition; rigor mortis and posterior hypostasis were present. Cutaneous cachectic pallor was marked. In the left femoral region, sloping down and outwards, there was an elliptical wound 4 by 2 in. (10 by 5 cm.), which is gaping and at the bottom of which is an ulcerating neoplastic area.

Owing to removal of the heart and lungs *in toto* the former was not examined, but appeared to be unaffected by neoplasm so far as the pericardium was concerned and not obviously enlarged.

Lung.—One and a half inches below the bifurcation of the right main bronchus the mucosa was mottled with new growth. Just below this there was a scirrhus stricture of new growth,

FIG. A.—Right lower lobe of left lung: atelectasis and neoplasm.

below which was a large wedge of white, soft necrotic growth which was breaking down. This was in the right lower lobe, confirming the x-ray report of atelectasis (Fig. A). Microscopy showed an oat-cell carcinoma (Plate, Fig. 4).

Liver.—In the free border of the left lobe there was a whitish tumour about the size of a halfpenny. This was expected to be a secondary deposit, but was in fact an adenoma of the liver.

Brain.—Except for marked atheroma of the circle of Willis, and elsewhere some general atheroma of the aorta, no pathological changes apart from senility were found, nor were there any other metastases—for example, cerebral.

Discussion

Although the very limited size of the primary growth would seem to favour operative procedures such as lobectomy, the patient's age and rapid decline clearly ruled this out.

The precise cause of death is not clear, but the soft necrotic growth in the lung was probably producing considerable toxæmia, which affected the myocardium of an elderly subject. The route of metastasis was probably haematogenous.

We are greatly indebted to Dr. J. E. McCartney, of the Southern Group Laboratory, Park Hospital, for the photomicrographs of the primary and secondary tumours and to the local hospital photographic service of Messrs. Turnham, of Eastbourne, for the photograph of the specimen. We are also indebted to Messrs. Ilford Ltd. for the x-ray photograph.

REFERENCE

Willis, R. A. (1948). *Pathology of Tumours*, pp. 359, 374. Butterworth, London.

SJÖGREN'S DISEASE ASSOCIATED WITH A NUTRITIONAL DEFICIENCY SYNDROME

BY

R. B. RAFFLE, M.B., M.R.C.P.

In 1933 Henrik Sjögren first described the disease which bears his name. The eye changes were first noted, but the condition was later found to involve a large number of other structures. The non-ocular features have been stressed in later papers by Sjögren and by Weber (1945). The present case, which was seen in general practice and which is unassociated with eye changes, shows certain clinical features which seem worthy of record.

Case Report

The patient, a spinster of 50 working as a charwoman, first reported in January, 1949, complaining of depression and vague "rheumatic" pains affecting the occipital, interscapular, and sacral regions. She felt weak, lacked appetite and energy, and was subject to early morning waking. These symptoms had been present for about three months.

Pronounced facial swelling affected both parotid regions, and on inquiry she stated that this had started first on the left side of the face in July, 1947. The initial attack had been diagnosed as mumps, but the swelling had never subsided completely. Instead there had been recurrent exacerbations with incomplete regression, and some weeks later the right side of the face became similarly affected. Later the patient noted swellings under her chin. There had never been any pain. Some weeks after the onset of the swelling she had noticed dryness of her mouth, which became worse and caused difficulty in swallowing and speaking. The dysphagia had become intensified in the last few weeks, and was then associated with a feeling of food sticking behind the larynx. During this time she had persistent soreness of the tongue.

There were no gastro-intestinal symptoms and no paraesthesiae, but for many years she had been subject to a typical Raynaud phenomenon in the fingers during cold weather. She had also had what seems to have been a distal pulp abscess affecting the left middle finger in November, 1948, since when the finger had been persistently cyanotic and subject to attacks of burning pain on warming. It was involved in the "local syncope" of the Raynaud attacks in common with the other fingers, but the attacks lasted longer in this finger and never recovered beyond the stage of cyanosis. The menopause had occurred two years previously. She had never been subject to eye trouble.

Examination on January 19 revealed a spare, pale, nervous woman, looking older than her years and possessing a conspicuous swelling in both parotid regions. Each parotid gland was enlarged to the size of half an orange, and was firm and nodular but not tender. Both submaxillary glands showed a similar plum-sized enlargement. The whole buccal cavity was dry, and the mucosa was dull red in colour and of a matt texture. The tongue was dry, red, and glazed, the papillae not being evident. She also showed an angular stomatitis and cheilosis. The skin was dry, the hair thin, and the eyebrows normal. The left middle finger was cyanosed and the finger-nails showed koilonychia. The heart, lungs, and abdomen revealed no abnormality. The blood pressure was 135/85. The spleen was not palpable.

Blood examination showed: haemoglobin, 70% (Haldane); red cells, 4,200,000; colour index, 0.83; white cells, 5,000 (polymorphs 72%, eosinophils 1%, lymphocytes 15%, monocytes 12%). E.S.R. (Westergren; 200 mm. column): 1 hour reading, 20 mm. The Wassermann reaction was negative. A chest x-ray film revealed no abnormalities. Urine examination showed nothing abnormal.

Progress.—The patient was at first given vitamin A, 150,000 units daily. On February 7 she was not improved in any way, and vitamin A therapy was stopped and ferrous sulphate, 18 gr. (1.2 g.) daily, was administered. On February 23 the haemoglobin was 85% and the red blood cells numbered 4,500,000. There was no improvement in the buccal condition, but she felt subjectively better. "Beplex" capsules, six a day, were given. This provides a daily intake of 6 mg. of thiamin hydrochloride, 4.8 mg. of riboflavin, and 60 mg. of nicotinic acid.

On March 5 the haemoglobin was 105% and the red blood cells numbered 4,800,000. She felt much more "like herself," looked better, was sleeping well, and was no longer fatigued. The soreness of the tongue had gone and also the angular stomatitis and cheilosis, but the xerostomia persisted and the glandular enlargement was unaffected. Her appetite was improved and the dysphagia less troublesome; she attempted to take a more normal diet, and reported that the sensation of food sticking behind the larynx had vanished. Three beplex capsules and 9 gr. (0.6 g.) of ferrous sulphate were continued daily, and the improvement was maintained.

Discussion

The course of the illness seems to fall roughly into two stages—the first, which had started in July, 1947, comprising the gradual increase in size of the salivary glands together with the xerostomia; the second, beginning in October, 1948, and associated with the complaints of fatigue and general ill-health. During this second period the soreness of the tongue and the finger infection had developed.

The salivary gland changes are compatible with the diagnosis of Sjögren's disease, the ocular features of which are sometimes delayed, xerostomia being the presenting symptom. The age, sex, and psychological type of the patient also point to this diagnosis, and the association with Raynaud's phenomenon has been noted before. As Ellman and Weber (1949) point out, it is doubtful how far the condition can be distinguished from Mickulicz's disease.

The glossitis, angular stomatitis, cheilosis, koilonychia, and hypochromic microcytic anaemia of the second stage are clearly the result of nutritional deficiency. In view of the part allegedly played by vitamin deficiency (e.g., hypovitaminosis A) in the aetiology of Sjögren's disease, inquiry was made into the dietary history. In spite of the fact that the patient was a spinster, living alone and doing part-time domestic work, this was good, and there was no evidence to support the existence of any gross primary nutritional error preceding the onset of the glandular swelling, or, indeed, prior to 1948. During that year, however, the

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FIG. 1.—Inguinal gland. Mitoses, vesiculation, and inflammatory cells; remnants of lymphoid tissue. ($\times 220$.)

FIG. 2.—Inguinal gland. Oat cells. The hyper-chromatic nuclei are almost universal. ($\times 290$.)

FIG. 3.—Increased density in right lower lobe; probable bronchial carcinoma. Adhesions are present at the left costophrenic angle.

G. SMITH: EFFECT OF DIATHERMY CURRENTS ON METAL IMPLANTS

FIG. 4.—Primary growth of right lung (lower lobe). ($\times 100$.)

FIG. 1.—Site of implant (tantalum gauze). ($\times 30$.)

FIG. 2.—Tissue reaction to implant (tantalum gauze). ($\times 200$.)

A. M. BOYD AND R. P. JEPSON: EXTERNAL ILIAC ARTERY THROMBOSIS

FIG. 1.—Case 2. Haematoxylin and eosin. ($\times 12$.)

FIG. 2.—Case 2. Haematoxylin and eosin. ($\times 60$.)

FIG. 3.—Case 2. Weigert's elastic stain. ($\times 60$.)