March 1963

MARSDEN: Carcinoma of the Male Breast

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Treatment

It is generally agreed that surgery is the first line of treatment. In practice, however, 20 to 35% are inoperable due to metastases, fixity to the chest wall, ulceration or emaciation (Somerville, 1952; Treves and Holleb, 1955; Mohardt, 1956). Radical mastectomy is performed if possible, otherwise a simple mastectomy or local excision. Pre- or post-operative radiotherapy plays an important part but the prognosis for those treated by radiotherapy alone is poor (Mohardt, 1956). Orchidectomy is usually indicated by the appearance of metastases, but it is sometimes combined with simple mastectomy. Estrogens may be given for recurrences following orchidectomy and some remarkable results have been reported—e.g. Ogilvie, 1961—but other reports suggest that tumour activity can be increased by both estrogens and androgens (Schofield, 1957; Pyrah, 1956) combined orchidectomy and adrenalectomy with success, but medical adrenalectomy is more widely used.

Prognosis

The poor prognosis of carcinoma of the male breast is undoubtedly due to the high inoperability rate which is in turn due to the delay in diagnosis. Thus the overall five-year survival for men in most reported series (Somerville, 1952; Mohardt, 1956; Sandison, 1956) is only 30%, but for the operable the figure is 42% (Sandison, 1956) and this compares favourably with the 34 to 48% quoted by Haagensen (1956) for women.

Summary

The case history of a 54-year-old man with simultaneous bilateral carcinoma of the breast with Paget's disease of one nipple is presented. The main features of carcinoma of the male breast are briefly reviewed.

It is a pleasure to acknowledge the help of Miss M. D. Snelling and Mr. R. S. Handley under whose care the patient was admitted.

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__REVERSIBLE PSYCHOTIC SYNDROME AFTER POTASSIUM PERCHLORATE TREATMENT__

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Potassium perchlorate has been extensively used in the treatment of thyrotoxicosis since it has been shown to be an effective and relatively safe therapeutic agent (Godley and Stanbury, 1954; Morgans and Trotter, 1954, 1960; Crooks and Wayne, 1959, 1960). Nevertheless side-effects may occur and up to date the following reactions to this drug have been reported: gastrointestinal disturbances,
skin rashes, pyrexia, proteinuria, thrombocytopenia, marked neutropenia, reversible agranulocytosis, lymphadenopathy and rarer, but more serious, the nephrotic syndrome (Lee, Vernier and Ulstrom, 1961) and fatal aplastic anemia (Brit. med. J., 1961).

Neurological or psychiatric complications have not yet been described and this prompted the present case which is reported to draw attention to a reversible psychotic syndrome developing after treatment of thyrotoxicosis with potassium perchlorate.

Case Report

A 20-year-old lawyer was admitted to the Evangelismos Hospital in October 1961 with a severe psychotic reaction of 15 days' duration for further investigation.

The family and past medical history, with regard to both physical and mental health, were non-contributory and the patient had been working efficiently until 15 days before admission.

Thyrotoxicosis was first diagnosed in 1955 and since then his condition was fluctuating continuously because of intermittent treatment with carbimazole and propylthiouracil. Periods of clinical and laboratory euthyroidism were repeatedly followed by periods of typical thyrotoxicosis, subsiding each time with anti-thyroid treatment.

In March 1961 he developed an exacerbation of moderate severity, lasting until June 1961 when the BMR was +50% and the body weight 50 kg. The patient was put on potassium perchlorate, 300 mg three times daily, with a rapid improvement of thyrotoxic features. A month later the BMR was +11% and the body weight 56 kg. The same dosage of potassium perchlorate was continued for a further three months when he first felt mild aches in the muscles. The patient became quite anxious and stopped taking the drug. The BMR was +19%. Three days later he began to be bothered by a hollow noise originating from a nearby factory that in the past had not bothered him, although it was of the same intensity. Because of this, he was unable to sleep and felt the need to have another person close by. Four days before admission he went to confession and was afraid to eat, drink or sleep. He blamed this condition on the hypofunctioning of the thyroid gland, which in turn he thought was due to the small dosage of the drug he was taking. That night he became lethargic and the next day he acted as a moribund patient, giving directions for the problems facing him and asking for a priest and Holy Communion. Two days before admission the routine laboratory tests were within normal limits and the BMR +27%, but he again asked for Holy Communion and had visual hallucinations having to do with the 'Kingdom of Heaven' to which he was going. He believed also that his blood had been poisoned.

On admission the patient was restless, constantly expressing paranoid ideas, ideas of reference and persecution, and having visual hallucinations. Neurological examination revealed no organic abnormality.

The thyroid gland was diffusely enlarged two to three times the normal size. The skin was moist on palms and slightly dry on the dorsal surface of the hands. There was no exophthalmos, no tremor. Pulse rate 72 per min., body weight 56 kg. Blood pressure and temperature normal. No other abnormality was noted on physical examination.

Investigations after admission showed: 131I uptake 72.7% in 2 hrs., 65.4% in 4 hrs., 58.4% in 10 hrs., 44% in 24 hrs.; Blondal ratio 6.64; PB 19I in 48 hrs., 6.23% per litre; BMR +22%. Thyroid scanning showed a moderate homogeneous enlargement of the thyroid gland. ECG, EEG and the remainder of the laboratory examinations were negative.

The patient was hospitalized for 15 days under purely symptomatic therapy and without any specific treatment for the psychotic syndrome or the thyroid condition. The mental state remained severely upset during the first week, rapidly improving thereafter.

For a period of three weeks after discharge the patient was feeling well and he started working slightly. But consequently thyrotoxic features reappeared and the BMR increased to +38%. In December 1961, after pre-operative Lugol's therapy, a subtotal thyroidectomy was performed and the histologic examination (Dr. Vlachos) showed diffused parenchymatic hyperplasia consistent with thyrotoxicosis.

When last seen, 16 months after thyroidectomy and 18 months after the psychotic syndrome, the patient felt very well without any treatment.

Comment

Until recently, and according to experimental and clinical data, potassium perchlorate was considered relatively non-toxic and was therefore increasingly used in the treatment of thyrotoxicosis. (Brit. med. J., 1960). However, during the last two years five cases of fatal aplastic anemia following therapeutic administration of potassium perchlorate have been separately described (Hobson, 1961; Johnson and Moore, 1961; Fawcett and Clarke, 1961; Rienhoff, 1961; Krevans, Asper and Rienhoff, 1962) and caution was recommended in the use of this drug. (Brit. med. J., 1961).

The case reported above represents another severe side-effect not previously described, characterized by delusions, paranoid ideas and hallucinations. The psychotic state developed in the fifth month of treatment and subsided on withdrawal of the drug without any specific treatment and any recurrence during a year's follow-up. Perchlorate therapy may therefore be correlated to the psychotic reaction since no underlying electrolyte, metabolic, infective or circulatory disorders were observed.

The patient's negative family and past history and the absolutely normal mental condition following the subsidence of the syndrome without any therapy make the possibility of a schizophrenic reaction unlikely. Hormonal imbalance is also an unlikely etiologic factor. Hypothyroid or hyperthyroid states are associated with various psychiatric syndromes, but the patient was already clinically euthyroid for a long time when this developed. A small increase of the B.M.R. observed during the first days is not infrequent in patients under stress. The increase in radiiodine uptake is also a usual laboratory finding in euthyroid individuals when the determination is done shortly after stopping the administration of potassium perchlorate, perhaps because of an induced thyroid gland iodine deficiency. Moreover the fact that the syndrome was cured without any antithyroid therapy but on the contrary after the interruption of the drug and that the psychological state remained
unaffected when subsequently thyrotoxicosis reappeared are all strong evidence against the etiologic significance of the latter.

By excluding therefore other potential causes, the possible relationship of this syndrome to the potassium perchlorate, and particularly by a mechanism of toxic reaction, becomes more likely since allergic manifestations were not observed.

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EISENMENGER SYNDROME WITH RADIOLOGICAL CALCIFICATION OF MAIN PULMONARY ARTERIES

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Calcification of the main pulmonary artery or of its left and right branches gross enough to be visualized radiologically and diagnosed during life has not been described, as far as we could find from the literature. Atherosclerosis of the pulmonary arteries is of course common in long-standing pulmonary hypertension, and these atheromatous patches would naturally get calcified, although not large enough to be seen on X-ray examination. Thrombosis-in-situ of the large pulmonary arteries has been described by Brenner (1931), Dimond and Jones (1954), Magidson and Jacobson (1955), Ring and Bakke (1955) and by others. Magidson and Jacobson (1955), describing a series of these cases with autopsy findings did not mention any gross calcification of the pulmonary arteries. Closed chest injury has been mentioned as a cause of pulmonary artery thrombosis (Dimond and Jones, 1954; Don Michael, 1962, etc.)

Aortic valve cusp damage following non-penetrating injury of the chest and causing aortic incompetence has been described (Wood, 1956).

The case we describe below is of a young female who had pulmonary incompetence, with the clinical picture of Eisenmenger's syndrome and radiological calcification of the main pulmonary arteries, following a history of trauma to the chest.

Case Report

A 27-year-old unmarried female patient was admitted to the General Hospital, Colombo, in October 1962 with a history of dyspnea on ordinary exertion, such as walking on the flat, since the age of ten years.

At the age of eight years the patient had fallen while playing and had struck the front of her chest on the edge of a stone step, with another child's weight falling on her back. For one month following this she had a pain in the precordial region. Two years after the chest injury she woke up one morning with a sense of dyspnea and pain in the chest, and these symptoms increased as she was made to walk to school. She had a syncopal attack on the way to school and was cyanosed during the attack.

There had been no syncopal attacks since then, but anginal pain on walking had been experienced off and on.