CASE REPORTS

Difficult in clinical assessment of lymph-nodes after endolymphatic radio-iodine

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Endolymphatic radio-iodine is being used prophylactically in the treatment of malignant melanoma and may prove extremely valuable in preventing the development of lymph-node metastases.

However, it poses certain problems in ‘follow-up’ assessment, and these are illustrated by the following case history.

Case report
R.E., a man aged 41, was referred by his doctor in 1963 with a large mole on his back which had increased in size. The appearances at that time were benign, and he was advised to undergo local excision, but was unwilling to have this done.

He was seen again in June 1966, with increase in size of the mole, which showed a raised central non-pigmented area, and satellite formation round the edge. Small soft lymph-nodes were palpable in both axillae. There was no other lymphadenopathy or evidence of metastases.

Investigations: Hb 103%, chest X-ray normal.

Operation (June 1966). The melanoma was excised from his back with a 5 cm margin of skin around and monoblock excision of subcutaneous tissues down to and including the fascia of the underlying muscles. A split skin graft was taken from the back of the right thigh and applied to the defect. Histology of the specimen showed an ulcerating malignant melanoma. The tissues around and deep to the lesion were free of malignant cells.

Following the operation a bilateral therapeutic lymphangiogram was performed using 15 μCi of UF Iodine. Good films were obtained of the lymph-nodes in both axillae and they showed no evidence of metastases.

The patient was seen at regular intervals in the clinic and given physiotherapy for arm movements. The axillary lymph-nodes became slightly tender soon after the lymphangiogram but showed no obvious changes in consistency until in August 1966 a large firm lymph-node was palpable in the left axilla. A X-ray of the area showed no definite evidence of metastases, but it was decided in view of the clinical picture to undertake a block dissection of the left axilla.

Histology of the specimen obtained showed only a lipogranulomatous reaction to the previous lymphangiogram with no evidence of malignancy (Fig. 1).

The patient remained well, but developed a node in the right axilla with similar characteristics to those removed from the opposite side. The chest X-ray was normal and an expectant policy was pursued.

Discussion
Lymphangiography was introduced to this country in 1951 by Kinmonth, and has been used extensively to investigate congenital and acquired abnormalities of the lymphatic system (Gough, Guiney & Kinmonth, 1963; Kinmonth, Rob & Simeone, 1962; Kinmonth, 1952, 1965; Wallace et al., 1961). It has given a great deal of information about these conditions and has led to modifications in the treatment of lymphoedema.

The diagnostic use of lymphangiography in malignant conditions involving lymphatic tissues promised much, but in practice has been subject to certain limitations.

Difficulties of technique and of interpretation of X-rays restrict the procedure to specialist centres. While gross involvement of lymph-nodes by reticulooses and secondary deposits may be
detected and clinical impressions confirmed by lymphangiography, microscopic involvement of nodes may produce no observable changes. A useful aspect may be in the assessment of completeness of surgical clearance of lymph-nodes (Lemon et al., 1966; Shibata et al., 1966).

Endolymphatic therapy with radioactive gold and iodine compounds has been used for the treatment of metastatic deposits in lymph-nodes and vessels (Ariel & Resnick, 1964; Jantet, 1958; Jantet et al., 1964; Wallace et al., 1961). It has also been used prophylactically in combination with surgical treatment for malignant melanoma. In this condition, regional lymph-nodes, although clinically negative have been shown to be involved with microscopic tumour metastases in at least one-third of cases (Fortner, Booher & Pack, 1964).

The histological changes produced by ultrafluid lipiodol used in lymphangiography are well described (Ravel, 1966). They are characterized by hypoplasia and eventual disappearance of germinal centres, the presence of oil droplets in sinuses, follicles and germinal centres, and by sinus histiocytes.

There is an initial acute reaction with infiltration by plasma cells, and polymorphonuclear and eosinophil leucocytes, followed in a few days by a foreign-body giant-cell reaction round the droplets of contrast medium. Giant cells are also present in the lining of lymphatics outside the lymph-nodes. This reaction persists for several months unchanged and eventually disappears without fibrosis.

These microscopical changes may make the task of the pathologist more difficult in a search for malignant deposits, but the clinical changes produced in the lymph-nodes also pose a problem for the surgeon.

Nodes that were previously clinically innocent enlarge and become palpable (Gough et al., 1963). They also become firmer in consistency simulating secondary deposits. Although associated tenderness of the suspicious nodes often occurs to suggest that the changes are due to reaction to the contrast medium, the decision to undertake further surgery or not may be difficult.

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**References**

Case reports


Muscle necrosis and acute renal failure in carbon monoxide poisoning

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Scattered reports have appeared in the literature concerning the occurrence of acute muscle necrosis in cases of carbon monoxide and barbiturate poisoning (Howse & Seddon, 1966). Acute renal failure, presumably due to the release of myoglobin from the necrosed muscle cells, has been reported in such a case by Loughridge, Leader & Bowen (1958). The present case is reported because of the relative rarity of the condition, because of the diagnostic problems involved and because the patient survived the additional complication of acute renal failure.

Case report

A 44-year-old female was admitted on 11 October 1966 having taken an unknown number of capsules of sodium amytal and imipramine, and having then attempted to gas herself. On admission she was comatose and arreflexic; shortly afterwards she developed apnoea and severe hypotension. Carboxyhaemoglobin level was 35%, and serum barbiturate level 1.8 mg/100 ml. She was treated with the usual conservative measures, artificial ventilation and forced diuresis, as described by Linton, Luke & Briggs (1967). She regained consciousness 12 hr after admission, and there were no cerebral or respiratory complications. It was then noted that her urine volume had fallen to 5 ml/hr, although this had been excellent during the 10 hr of forced diuresis. The urine was dark brown in colour, and was initially reported to contain haemoglobin. It was suspected that the patient had sustained an episode of acute intravascular haemolysis probably as a result of drug ingestion. The diagnosis of acute renal failure was made on the basis of continuing severe oliguria (urine volume <300 ml/day), a blood urea of 180 mg/100 ml and a urinary urea concentration of 800 mg/100 ml. The usual conservative regime for acute renal failure was established, but despite severe fluid restriction the patient developed gross oedema of both legs and feet about 5 days after admission. This feature, together with the marked tenderness of the leg muscles, and the fact that the brown pigment disappeared very quickly from the serum despite severe renal impairment raised the possibility that this was a case of muscle necrosis and that the pigment was myoglobin, which has a much higher renal clearance than haemoglobin (Bailie, 1964). That the urinary pigment was in fact myoglobin was then confirmed by spectroscopic examination.

The patient remained severely oliguric for 15 days, and required one peritoneal dialysis and two haemodialyses before diuresis occurred. She then made an uncomplicated recovery from her renal failure, but when mobilization was attempted, she had great difficulty in walking. Examination of her legs revealed that in addition to oedema, her calf muscles were tense,
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