Intracranial Hodgkin's disease shown by radioisotope scan

S. CURRIE AND G. W. H. JARDINE

From the Departments of Neurology and Radiotherapy, The London Hospital, London

Verified intracranial deposits of Hodgkin's disease are rare. Thus there was only one among 125 cases reported by John and Nabarro (1955). In the monograph by Williams, Diamond, Craver, and Parsons (1959) there were four among 1,992 cases. Todd (1967) reported two among 1,339 cases. The deposits are usually subdural, intracerebral metastases being exceptional. This rarity contrasts with the incidence of spinal extradural deposits, around 5% in most series (Goldman, 1940; Smith and Stenstrom, 1948; Verity, 1968; Currie and Henson, 1969); the absence of a true intracranial extradural space may be a factor, as pointed out for lymphosarcoma by Jernstrom, Crockett, and Bachhuber (1966). In only two of the cases of subdural metastasis recorded in the literature has intravenous gamma-encephalography been carried out (Gaelen and Levitan, 1967; Verity, 1968), in each as an adjunct to contrast studies (arteriography and pneumography). Its use is described in a further case which presented with epilepsy and was confirmed at necropsy. Its place in the investigation and management of similar cases is discussed; this is pertinent as, firstly, the rarity means that a coincidental lesion needs exclusion and, secondly, the relatively benign nature of most deposits, which are solitary and compress rather than invade brain, makes their demonstration more than an academic exercise.

CASE REPORT

The patient (P. McD. London Hospital No. 165416), a 21-year-old woman, first attended the Radiotherapy Department in October 1967 with a history of abdominal pain for one year and of sweating and sciatica for three months. On examination there was cervical adenopathy. Radiographs of the chest showed a mediastinal mass. Supraclavicular lymph node biopsy revealed diffuse fibrous Hodgkin's tissue. A course of 220 kV irradiation was given to the mediastinum, neck, and axillae with a tumour dose of 2,500 r in 15 treatments over 20 days. In April 1968 collapse of the left lung occurred due to obstruction of the left main bronchus; a further course of irradiation relieved her dyspnoea. During an admission in May 1968, with dysphagia due to pharyngeal moniliasis, the patient suffered an episode of focal sensory epilepsy in which tingling occurred in the left hand spreading to the whole limb within seconds; for 10 minutes subsequently there was numbness and for 24 hours, weakness of this limb. In June 1968 a left-sided focal attack occurred, this time motor, affecting the face then upper limb with a Jacksonian march, followed by loss of consciousness. An electroencephalogram suggested a right fronto-parietal lesion. A gamma-encephalogram (GEG) was performed using 10 mC Technetium-99m with automatic continuous brain scanning; this showed a superficial circumscribed fronto-parietal lesion, approximately 3 cm in diameter (Figs. 1a and b). The patient was given phenobarbitone and had no further attacks. In view of the widespread disease, she was treated by antimitotic drugs (procarbazine, cyclophosphamide), but her respiratory condition continued to deteriorate and she died in August 1968.

Necropsy (No. 229/68) showed involvement of the mediastinum, lungs, spleen and bone marrow as well as of lymph nodes. There was a 3 cm subdural deposit resulting in a depression in the right parietal region of the brain (Figs. 2 and 3); macroscopically it resembled a meningioma. Neither bone nor brain was invaded. Histological examination of this deposit showed diffuse fibrous Hodgkin's tissue, as in the earlier biopsy, with small numbers of Reed-Sternberg cells and scanty lymphocytes (Fig. 4).

DISCUSSION

The published reports of intracranial deposits of Hodgkin's disease have used the classification of Jackson and Parker (1944). This comprises paragranuloma (which does not involve the nervous system), granuloma (producing intracranial metastases in less than 0.5% of cases), and sarcoma (which may occur as a primary mesenchymal brain tumour). While the value of this classification is limited by most cases falling into the granuloma group, the more detailed one of Lukes and Butler (1966) is often hard to apply, due to variation within a single lesion. The histology in our case is to some extent non-specific, being found terminally in many. Regardless of which classification is used, intracranial metastasis is rare, the present case being the only verified one in 210 cases attending the London Hospital over a period of 10 years (a further case had
an asymptomatic deposit); this is in accord with the published series including the recent one of Todd (1967) who found two cases in 1,339 seen in Manchester during 18 years. Our case illustrates well the characteristic subdural deposit with compression but not invasion of underlying brain. Fein and Newill (1954) recorded a case in which obliteration of the subdural space and invasion of the brain did occur. Buckley and Warwick (1968) described a case in which a subdural deposit was said to involve brain; whether this meant actual invasion is uncertain from their report. In their review of the literature, Ljungdahl, Strang, and Tovi (1965) suggested that intracerebral and those subdural deposits not attached to bone might be haematogenous, implying that non-viability of most tumour emboli might explain both the rarity of deposits and the occurrence of episodes without necropsy findings. The clinical picture of metastasis is unlike that of the progressive multifocal leucoencephalopathy which may accompany the reticuloses and which is probably due to viral action allowed by reduced immunological reactivity (Richardson, 1961; Howatson, Nagai, and Zu Rhein, 1965); in this mental symptoms with cortical, brain-stem and cerebellar signs occur. Winkelmann and Moore (1941) described a complex case with apparent intracerebral as well as subdural Hodgkin’s disease but their illustrations are of leucoencephalopathy, as pointed out by Aström, Mancall, and Richardson (1958).

The uptake by tissues of radioactive isotopes is dependent on factors such as vascularity and permeability, the latter being the more obscure in tumours, with possibly an active cellular component (Planiol, 1966). The gamma emitter Technetium-99m, as used in this case, has a short half-life; its
Intracranial Hodgkin’s disease shown by radioisotope scan

A case of Hodgkin’s disease is described in which a subdural deposit presented with epilepsy, was demonstrated by gamma-encephalography, and confirmed at necropsy. The place of radioisotope studies in the management of similar cases is discussed.

Our thanks are due to Dr. H. F. Hope-Stone for permission to publish the case, Professor H. Urich for the photographs of the pathology, and the Physics Department for those of the GEG. This work was supported by the British Empire Cancer Campaign through the Neuropathological Research Unit of The London Hospital Medical College; we thank the Director, Dr. R. A. Henson, for this and for his advice.

REFERENCES
