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Short Term Memory Deficit in a Patient with Cerebral Sarcoidosis

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Sarcoidosis is defined by Scadding (1967) as "a disease which is characterized by the presence in all of several affected organs or tissues of epithelioid cell tubercles . . . proceeding either to resolution or to . . . (the formation of) fibrous tissue". The organs which are most commonly involved are the lungs, skin, eyes, liver, spleen and joints. Clinical evidence of involvement of the central nervous system may be found in 5 per cent of all cases (Silverstein *et al*, 1965; Wiederholt and Siekert, 1965; Delaney, 1977). Typical lesions are cranial nerve palsies, intracranial space-occupying lesions, pituitary or hypothalamic lesions and, less frequently, lesions of the brainstem or spinal cord (Waldenstrom, 1937; Matthews, 1965; Wiederholt and Siekert, 1965; Scadding, 1967; Delaney, 1977).

Of interest to psychiatrists are several reports of change of personality, 'dementia' and 'poor memory', in patients with cerebral sarcoidosis (Hook, 1951; Maycock *et al*, 1963; Matthews, 1965; Silverstein *et al*, 1965; Hahn, 1971; Delaney, 1977). However none of these reports include a formal examination of cognitive function. We describe a patient with typical features of cerebral and extracerebral sarcoidosis and in whom detailed cognitive testing has revealed a selective defect of short-term memory.

Case Report

R.P. is a Dominican (West Indian) female who has no significant family history. Her sarcoidosis began at the age of 24 as bilateral hilar lymphadenopathy and the diagnosis was confirmed by Kveim test and the findings of epithelioid granulomata in biopsies from liver and paratracheal lymph nodes. Two years later she had a minor seizure, and shortly after this developed headaches, weakness and inco-ordination, and was found to have a sensory level at T4. CAT scan showed mild ventricular dilatation and evidence of a right frontal granuloma with basal meningeal sarcoid. Myelogram showed complete obstruction from T2 to T7. WAIS was 89 (full scale). She was treated with dexamethasone (6 mg/day) and improved markedly over the course of a year. This neurological improvement has been sustained, without relapse.

At the age of 28 she became distressed by an awareness of a growing memory impairment over 3 to 4 weeks. She then suddenly became agitated and doubly incontinent and had delusions, sometimes that she was pregnant, at others that she was dead. She was disorientated in time and place and began to hear voices at night. Her affect was bizarre, with simultaneous laughter and tears. This acute brain syndrome settled after 6 to 8 weeks of treatment with haloperidol and an increased dose of dexamethasone (16 mg/day). After an initial improvement her memory remained mildly impaired, but she was able to live independently using a notebook as a mnemonic aid. At discharge WAIS was 78 (full scale), Wechsler logical memory was 55 per cent (delayed, as a per cent of immediate recall), Rey-Osterreith was 45 per cent. This score is typical of bilateral temporal lobe lesions.

After discharge the steroid was changed to prednisolone and reduced to 5 mg daily. Shortly after this dose was achieved she became aware of a further deterioration in her memory for 2 months, without a change in her neurological status. This was confirmed by several informants. She also complained of insomnia and compulsive eating with weight gain, but these symptoms were not confirmed after admission. Smells seemed very powerful to her and there was a subjective increase in libido but no sexual disinhibition.

She was re-admitted, now aged 30. On examination she was found to be obese, with a mildly ataxic gait, hyperreflexia of the legs and an extensor plantar response bilaterally. There was total anosmia. There was no behavioural disturbance and the mood was mildly depressed. There were no delusions, hallucinations or thought disorder. She was disorientated in time, but not in place, and was completely unable to remember a name and address or to reproduce from immediate memory a series of simple geometrical figures. She could not remember the events of the day or find her way around the ward. There were no other cognitive deficits. Formal psychometry showed a WAIS of 82 (full scale), logical memory 19.9 per cent and Rey-Osterreith of 29.8 per cent. There was therefore a significant reduction in short-term memory without a reduction of general intelligence.

The investigations, including full hypothalamic function tests and ESR were all normal. The CAT scan was unchanged compared to a previous scan.

The diagnosis of further relapse of sarcoidosis was made on the basis of the psychological tests and the absence of other possible causes. Prednisolone was increased to 60 mg a day and then reduced over several weeks to 10 mg a day. An improvement in her orientation began to be seen after 2 to 3 weeks, and she eventually became fully oriented in time, without prompting, and could remember a name and address after 2 minutes. Unfortunately her performance never reached the baseline on a series of objective tests of new learning ability, so these were unable to show the improvement noted clinically. She was eventually discharged home to a semi-independent life in her own home, which she now runs efficiently, with the aid of her diary.

Discussion

Although our patient has typical features of extracerebral sarcoidosis, she is the first in whom a selective defect of short term memory has been reported. 'Dementia' has been described in a number of cases in which detailed cognitive testing was not reported (Matthews, 1965; Silverstein *et al*, 1965; Delaney, 1977). However from what is known of the pathology of cerebral sarcoidosis a selective memory defect is more likely than a global dementia. A granulomatous involvement of the basal meninges was found in every patient in a series of 14 patients with cerebral sarcoidosis who were examined *post mortem* (Delaney, 1977) but a generalized loss of cortical tissue has not been reported. We therefore suggest that in our patient, and probably in others, the limbic structures which are known to be involved in the acquisition of a short-term memory may have been damaged by the basal meningitis which is known to exist in cerebral sarcoidosis. The diagnosis of sarcoidosis should be considered in patients with selective defects of short-term memory, as in some cases cerebral sarcoidosis apparently responds to treatment with steroids (Delaney, 1977).

References

DELANEY, P. (1977) Neurologic manifestations in sarcoidosis. *Annals of Internal Medicine*, **87**, 336–45.

HAHN, R. (1971) Unusual forms of sarcoidosis. *Southern Medical Journal*, **64**, 541–5.

HOOK, O. (1951) Sarcoidosis with involvement of the nervous system. *Archives of Neurology and Psychiatry*, **71**, 554–75.

MATTHEWS, W. B. (1965) Sarcoidosis of the nervous system. *Journal of Neurology Neurosurgery and Psychiatry*, **28**, 23–9.

MAYCOCK, R. L., BERTRAND, P., MORRISON, C. E. & SCOTT, J. H. (1963) Manifestations of sarcoidosis. *American Journal of Medicine*, **35**, 67–89.

SCADDING, J. (1967) Sarcoidosis. London: Eyre and Spottiswoode.

SILVERSTEIN, A., FEUER, M. M. & SILTZBACH, L. E. (1965) Neurologic sarcoidosis. *Archives of Neurology*, **12**, 1–11.

WALDENSTROM, J. (1937) Some observations on uveoparotitis and allied conditions with special reference to symptoms from the nervous system. *Acta Medica Scandinavica*, **91**, 53–68.

WIEDERHOLT, W. C. & SIEKERT, R. G. (1965) Neurological manifestations of sarcoidosis. *Neurology* (Minneap.), **15**, 1147–54.

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