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CASE REPORT

Isolated retrograde amnesia for autobiographical material associated with acute left temporal lobe encephalitis

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SYNOPSIS Retrograde amnesia for autobiographical material in the absence of anterograde amnesia or other memory disturbances was found in a patient with acute viral encephalitis. Memory loss showed a temporal gradient, but new learning was spared. Both brain perfusion imaging with ^{99m}Tc-HMPAO SPECT, and EEG localized the lesion in the left temporal lobe while CT and MRI were normal. This observation supports the anatomical differentiation between the different memory functions. The uncommon combination of isolated retrograde amnesia without other neuropsychological findings may raise the doubt of psychogenic aetiology, which in this case was refuted.

INTRODUCTION

Retrograde amnesia is one of the well-known consequences of an acute encephalitis (Rose & Symonds, 1960). In previously reported cases a marked anterograde amnesia has accompanied the deficit. The pattern of remote memory loss together with recent memory deficit is found also in other organic disorders e.g. Wernicke–Korsakoff syndrome, thalamic infarctions and temporal lobe lesions (Parkin, 1984). Often the severity of anterograde amnesia correlates with the severity of retrograde amnesia. However, recent studies have shown that there may be only a low degree of shared variance between anterograde and retrograde memory performance (Kopelman, 1989) and in some cases the latter may dominate the syndrome (Roman-Campos *et al.* 1980; Goldberg *et al.* 1981). Even in cases with so-called isolated amnesias, thorough neuropsychological examination usually reveals other cognitive deficits such as impaired naming (Kapur *et al.* 1992) or agnosia (O'Connor *et al.* 1992; Ogden, 1993). Patients with retrograde amnesia completely without recent memory loss or other deficits are rare and offer a chance to learn more about the different aspects of memory. We present here a case where apart from a clear retrograde amnesia for autobiographical material, no other neuropsychological deficits were found. To the best of our knowledge this is the first report of such a case with a known diagnosis and functional localization of the defect.

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CASE REPORT

Clinical history

The patient was a 33-year-old right-handed woman with no relevant medical, psychiatric or family history. She was married and had three children. Ten days before admission she had been febrile for 2 days. Thereafter she had general malaise but no focal neurological or infectious symptoms. On the day of admission she had two focal and two generalized epileptic seizures. Clinical neurological examination was normal except for a disturbance in memory. She was unable to remember the events of the previous 4 days. The CSF leukocyte count was 4×10^6/ml. Computed tomography of the head (CT) was normal. EEG on the next day demonstrated a diffuse disturbance of background activity. Carbamazepine was administered to control seizures. On day 4 the amnestic symptoms progressed. She was unable to remember the age of her children or the reason for her hospitalization. An EEG follow-up showed general-

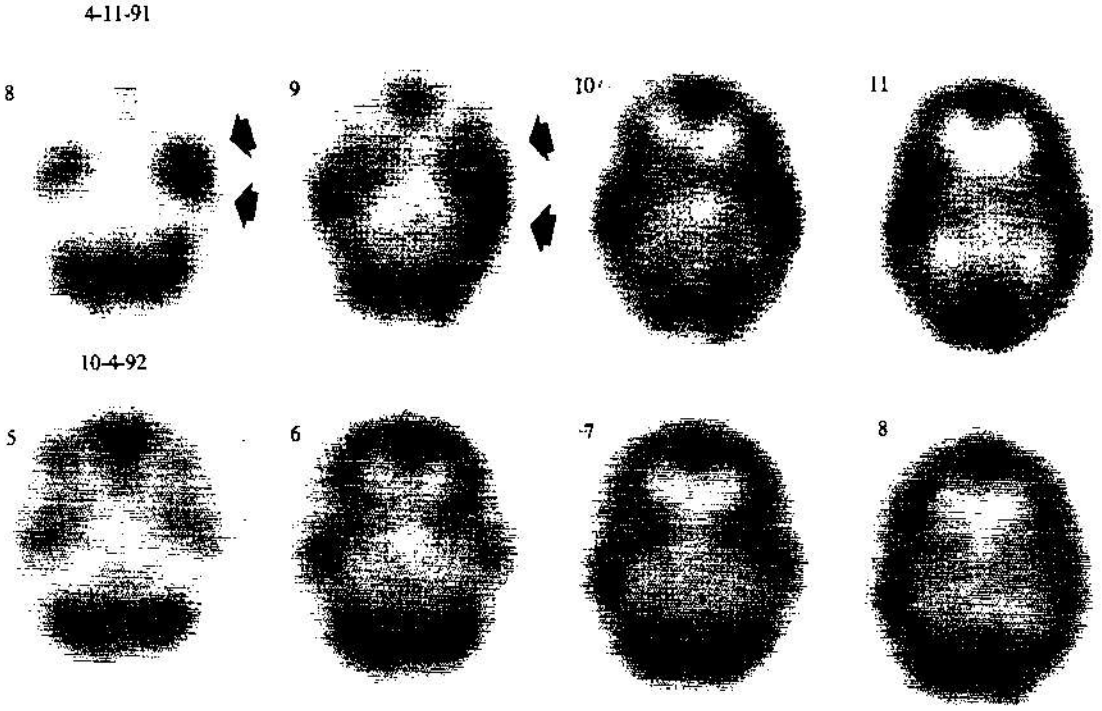

FIG. 1. ^{99m}Tc-HMPAO-SPECT showed increased perfusion in the anterior and middle part of the left temporal lobe (top), which had normalized 5 months later (bottom).

ized slowing of the background and a slow-wave focus with no periodic complexes in the left temporal area. In a control CSF the leukocyte count was slightly elevated (9×10^6/ml). Due to the progressing symptoms, focal EEG, and CSF pleocytosis, Herpes simplex encephalitis (HSE) was suspected and intravenous acyclovir 500 mg t.i.d. was administered. On day 7 the patient became febrile again and had five absence seizures. No focal clinical neurological signs or symptoms developed, but she was completely disorientated and unable to recognize her family. The left temporal slow-wave EEG focus progressed. No seizures appeared after day 7 and ^{99m}Tc-HMPAO single photon emission computed tomography (SPECT) on day 8 revealed hyperperfusion in the left temporal lobe (Fig. 1).

Thereafter the recovery was rapid. On day 13 the patient was orientated suffering only from retrograde amnesia. Three months later Magnetic Resonance Imaging (MRI) using a 1T Siemens Magnetom Scanner was normal (Fig. 2) and only minimal diffuse EEG abnormalities were seen. Brain perfusion in SPECT had normalized in a follow-up 5 months later (Fig. 1). Neither Herpes simplex nor any other diagnostic virus antibody-titre elevations were found in both CSF and serum antibody screening tests. HIV, *Borrelia burgdorferii* and *Treponema pallidum* antibody tests were also negative.

Neuropsychological evaluation

The patient's education consisted of 13 years of school followed by university studies. Pre-morbid intellectual capacity was accordingly estimated to be higher than average. Neuropsychological evaluation was first carried out 17 days after the admission. The patient was able to give a full description of the events in the hospital except for the days of disorientation and seizures. The test battery included tests of intellectual functioning (six subtests of the Wechsler Adult Intelligence Scale, the WAIS, Wechsler, 1955), recent verbal memory (the Wechsler Memory Scale,

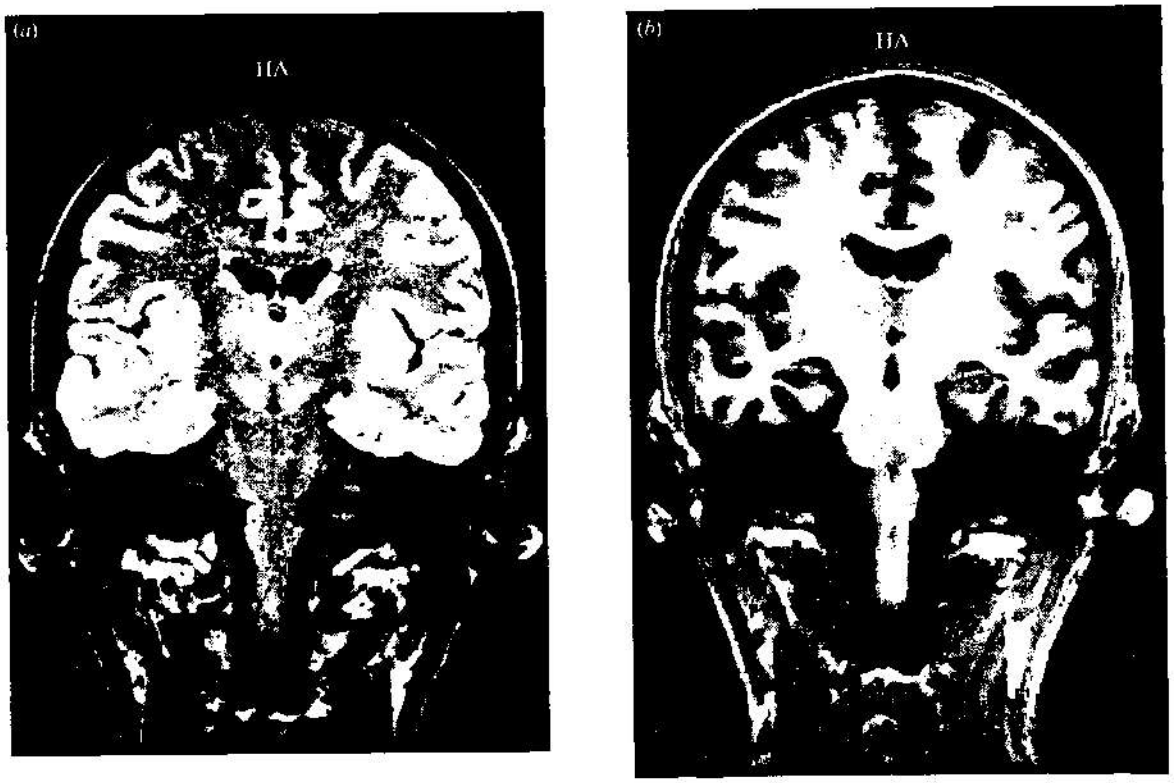

FIG. 2. Normal proton density (a), and T1-weighted (b) MRI scans 3 months after onset.

the WMS, Wechsler, 1945), delayed memory (the WMS stories and associative word pairs after one hour's delay), visual memory (the Benton visual memory test, Benton, 1974), the Stroop test of cognitive flexibility and naming (Lezak, 1983), word-fluency (oral production of words beginning with a given letter) as well as Luria-based tests for visuo-perceptual, motor and language functions (Christensen, 1975). The psychometric profile of the patient is given in Table 1. The patient's intellectual abilities were above average level as was her overall memory quotient and performance in both verbal and visual memory tests. Only in the Logical Memory subtest of the WMS the performance was slightly below the expected high level. No confabulation was observed. The patient had a good learning ability, she was not distracted by the temporal delay of the retrieval and was completely orientated in time and place. No circumscribed neuropsychological defects, such as aphasia, agnosia or apraxia, were found. No perseverative tendencies or signs of reduced mental flexibility appeared and word-fluency was normal. The patient was co-operative, motivated and showed perfect insight to her condition. Her score in the Beck Depression Inventory (Beck *et al.* 1961) was 11 indicating no clinical depression.

Evaluation of the remote memory defect

In the acute stage the patient had been unable to recall giving birth to her youngest son 9 months previously. In the neuropsychological assessment approximately 3 weeks later, individual incidents concerning her youngest son had re-appeared, but retrograde amnesia covered the events of the previous 6 months. Most of the personal facts (name, place and time of birth, address, school history, etc.) were retained.

Three months after the acute phase the Autobiographical Memory Interview (Kopelman *et al.* 1989) was used to estimate the severity of the amnesia (Table 1). In the recent information sections the patient was constrained to recall facts and incidents only from before the onset of the illness. The patient completed the section concerning background information without difficulty. In the Personal Semantic Memory Schedule her childhood and early adult life memories were intact but the retrieval of more recent information was impaired. In the Autobiographical

Table 1. *Neuropsychological profile of the patient. Except for the impairment of autobiographical memory, no cognitive defects were found*

WAIS (scaled scores)		
Information	11	
Arithmetics	14	
Vocabulary	13	
Digit Symbols	14	
Picture Completion	11	
Block Design	12	
VIQ	113	
PIQ	118	
WMS (raw scores)		
Logical Memory, immediate	8	max 23
Logical Memory, delayed	6	max 23
Digit span forward	8	
Digit span backward	5	
Associative Learning	19·5	max 21
Word pairs, delayed	8	max 10
Visual Reproduction	14	max 14
MQ	119	
Benton test, correct	8	max 10
Stroop naming, time(s)	47 (all correct)	
Stroop interference, time(s)	80 (all correct)	
Autobiographical Memory Interview		
Personal semantic memory		
Background information	22·0	max 23
Childhood	20·5	max 21
Early adult life	20·0	max 21
Recent information	10·5	max 21
Total	51	max 63
Autobiographical incidents		
Childhood	8	max 9
Early adult life	5	max 9
Recent information	0	max 9
Total	13	max 27

Incidents Schedule the patient's memory for adulthood and recent times was abnormal. Amnesia was total for the previous 6 months and patchy for several years. The patient's husband confirmed the problems relating to the remote memory loss in everyday situations. He also confirmed the absence of prior psychiatric disorders.

Five months after the acute phase the patient still experienced problems in situations that required memory of remote incidents. She had, however, been able to learn the relevant information about her past and had returned to her normal daily life. Informed consent for this case report was obtained from the patient.

DISCUSSION

The diagnosis of encephalitis was based on clinical follow-up, EEG and CSF. CT and MRI did not reveal pathology but ^{99m}Tc-HMPAO-SPECT showed an area of increased perfusion corresponding to the focal EEG abnormalities. Hyperperfusion correlates with HSE (Launes *et al.* 1988). The clinical history together with initial hyperperfusion in SPECT and cognitive changes resemble those seen in HSE. Cases of such mild forms of HSE have previously been reported (Klapper *et al.* 1984). Especially after the introduction of acyclovir-treatment HSE no longer invariably leads to wide-spread bilateral damage or severe cognitive disorders. However, in our case the viral agent could not be specified.

In his discussion of the nature of amnesia in alcoholic Wernicke–Korsakoff syndrome Parkin (1991) called forth cases with functional double dissociation between anterograde and retrograde amnesias. Such cases are valuable also for the analysis of the functional localization. Usually, retrograde amnesia is only one of multiple neuropsychological deficits. Even the reported cases of isolated retrograde amnesia, where a clear diagnosis has been reached, have included anterograde amnesia in the early stage (Goldberg *et al.* 1981). Completely isolated retrograde amnesia has been claimed to occur after attacks of transient global amnesia (Roman-Campos *et al.* 1980; Kapur *et al.* 1989) but no evidence of structural lesion has been demonstrated in these cases. There have also been reports of well-studied cases where the disease has affected wide areas of brain tissue (Damasio *et al.* 1985). Considering the complexity of cerebral connections it is, in such cases, difficult to relate one particular deficit to a particular location. Damasio and co-workers (1985) stated that the lesions of the lateral temporal lobes and insula could have been the principal source of the retrograde amnesia in their patient. In the HSE case with predominant retrograde amnesia reported by O'Connor *et al.* (1992) MRI revealed wide-spread damage mainly to the right temporal lobe extending parietally, and also to the parahippocampal gyrus and insula in the left hemisphere, as well as bilaterally in the frontal lobes. The case of Goldberg *et al.* (1981) had lesions in the medial and lateral surfaces of the temporal lobe and in the ventral tegmental area of the upper mesencephalon. However, temporal lobes may not be the only crucial structures in producing a retrograde memory deficit. Some studies stress the role of frontal lobes in the autobiographical recollection (Kopelman, 1991;

Della Sala *et al.* 1993) and autobiographical amnesia has also been reported to follow bilateral occipital lesion (Ogden, 1993). We were able to study a patient who had a limited area of brain tissue damage and a clear isolated retrograde memory deficit. In our case the EEG and SPECT localized the lesion into the left temporal lobe. The exact location within the temporal lobe could not be determined since CT and MRI were non-informative.

Validated methods for the evaluation of memory for public events or famous faces do not exist for the Finnish population. Therefore, the retrograde amnesia was assessed using the Autobiographical Memory Interview which has been proved useful in the evaluation of amnesic patients (Kopelman *et al.* 1989). The assessment was done 3 months after the acute stage thus reflecting a stable situation. Epileptic seizures cannot explain the late stage amnesia since no seizures appeared after the first week.

In clinical practice discrete amnesia may raise the doubt of psychogenic aetiology. New learning is often considered to be spared in psychogenic but affected in organic amnesias (Kopelman, 1987; Ericson, 1990). Our case demonstrates an exception to this rule. It has also been suggested that the appearance of a temporal gradient in the amnesia could be in favour of organic instead of psychogenic aetiology (Schachter *et al.* 1982). Our case agrees with this; recent events were completely unretrievable whereas more remote events were easier to recall. However, Sanders & Warrington (1971) found no relative sparing of remote events in their study of 200 normal subjects and 5 amnesic patients. This controversy clearly emphasizes the need for well-standardized and validated tests to control for the difficulty of the questions about incidents of the various decades.

De Renzi *et al.* (1987) reported a patient whose post-encephalitic damage was confined to the anterio-medial part of the left temporal lobe with minimal involvement of the right temporal lobe. The patient showed marked anterograde and retrograde amnesia as well as anomic features, but intact autobiographical memory. In our case the damage to brain tissue was not as wide-spread. Possibly also the location of the lesion within the temporal lobe was different, which might explain the different symptomatology in these two cases. Autobiographical memory does not always correlate with other aspects of retrograde memory, such as memory for famous faces, past events or personal semantic facts (Kopelman, 1989). Our patient's good performance in questions that concerned personal background information and semantic knowledge in contrast to the difficulties in Autobiographical Incidents Schedule may also be due to our patient's well preserved learning ability. Between the time of her discharge from the hospital and the time of testing she had been actively looking at photographs and asking her family and friends questions, thus re-learning her past. Unique episodes cannot be re-learned similarly. Such observations and an explanation for the dissociation of the semantic and episodic memory have also previously been reported (O'Connor *et al.* 1992).

Thus, as the retrograde amnesia had a temporal gradient in our patient, the neuropsychological profile of this case resembles those previously reported. Additionally, we discovered that no other neuropsychological symptom necessarily accompanies the deficit in a case with organic aetiology. The appearance of retrograde amnesia in the absence of anterograde amnesia or any other memory disturbance, which in this case was caused by a lesion in the left temporal lobe, supports the anatomical distinction between different types of memory.

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