Seizure 2001; **10**: 125–129
doi:10.1053/seiz.2000.0471, available online at http://www.idealibrary.com on IDEAL®

CASE REPORT

Cognitive dysfunction of right hemisphere-like Todd's paralysis after status epilepticus: a case report

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We describe a case with symptoms of transient diffuse right hemisphere dysfunction (hemispatial neglect, dyscalculia, and disturbance of both spatial construction and visuospatial perception) occurring after status epilepticus. The clinical picture of this case suggested to us that these features could be understood as a variant of Todd's paralysis.

Key words: Todd's paralysis; cognitive dysfunction.

INTRODUCTION

A transient localized neurological deficit is a well-known post-ictal phenomenon. In 1854, Todd described a case with hemiplegia that occurred after an epileptic seizure and named this condition as 'epileptic hemiplegia'. Later on, Jackson widened the limits of this definition[1]. Several neurological states, which include transient cognitive phenomena, have also been described as Todd's paralysis[2–10].

In this report, we present a case with the symptoms of transient diffuse right hemisphere dysfunction (hemispatial neglect, dyscalculia, and disturbance of both spatial construction and visuospatial perception) occurring after status epilepticus. The clinical picture of this case suggested to us that these features could be understood as a form of Todd's paralysis. To our knowledge, there is only one case of hemineglect related to Todd's paralysis in the literature[11].

CASE REPORT

A 28-year-old right-handed woman was admitted with a history of versive seizures heralded by illusions evolving to sudden loss of postural tone 2–3 times a week since the age of 10. Her history of birth, motor and mental development period was unremarkable. She had had one secondarily generalized tonic–clonic seizure (TC) when she was 24. On admission, she had had multiple TC seizures for 4 days. Initially during the seizures, she had perceptual abnormalities such as seeing her hands and limbs smaller than real size, feelings of jamais-vu, and well-formed visual hallucinations. Within 2 days, these seizures had evolved into a tonic–clonic status epilepticus. On admission, the patient was mildly comatose with moderate reacting limbs to painful stimuli. The pupils were mydriatic and reactive to light. Brain stem reflexes were intact. Tendon reflexes were brisk, and plantar reflexes were flexor.

The patient was treated with carbamazepine, 600 mg per day. She became seizure free and regained consciousness the next day. Conjugated gaze to the left was impaired and she had a left hemiparesis and left hemihypesthesis and left-sided neglect. Cranial magnetic resonance imaging revealed neuronal migration abnormalities of pachygyria and polymicrogyria in the right parietal cortex (Fig. 1).

On the second day of her hospitalization, the patient was fully awake. Her hemiparesis and hemihypes-

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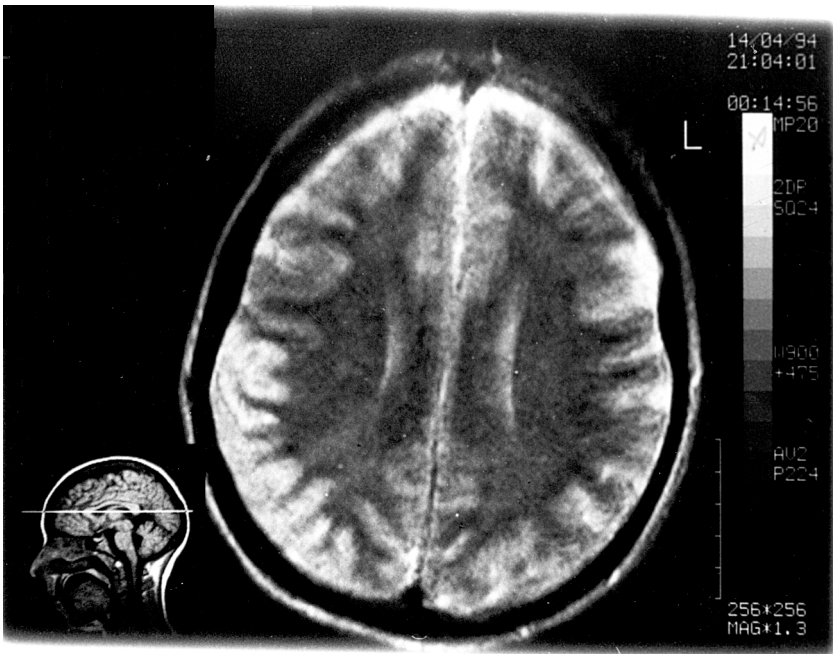

Fig. 1: Pachygyria and polymicrogyria in the right parietal cortex.

thesis had improved but neuropsychometric examination revealed left-sided hemineglect, dyscalculia, impairment of visuospatial perception, and construction. EEG showed diffuse amplitude depression in the right hemisphere (Fig. 2(a) and (b)).

On the sixth day, neuropsychometric test results improved significantly. A second EEG showed localized theta and delta waves in the right fronto-temporo-parietal region (Fig. 3(a) and (b)).

After 1 month, the patient remained seizure free. Her neuropsychometric test results were almost normal except for residual visuospatial perception impairment. The EEG findings were the same as those of the sixth day.

DISCUSSION

The most likely hypothesis to explain the characteristics of post-ictal deficits is that a post-ictal neurological deficit correlates with the localization of the epileptic focus and/or the lesion. The case reports on this subject mostly focus on post-ictal aphasia. It has been reported that post-ictal aphasia is a phenomenon which occurs mostly after seizures originating in the left temporal region[3, 12, 13]. Morrell[4] observed that bilateral hippocampal dysfunction occurred after epileptic discharges originating from bilateral medial temporal regions. Helmstaedter *et al.*[14] stated that post-ictal amnesia was related to the localization of the epileptic focus, and that there was no amnesia after frontal seizures but only after temporal seizures. This amnesia was non-verbal after a right temporal lobe seizure and was verbal after a left temporal lobe seizure. Bergen *et al.*[2] concluded that supplementary motor cortex involvement caused bilateral Todd's palsy in his two cases.

In our case, the features of the seizures suggested that a right parietal cortical lesion was the origin of the seizures. Additionally, post-ictal cognitive dysfunction of right hemisphere type appeared in very close relation with the lesion and the epileptic focus. We think that this case supports the hypothesis that post-ictal dysfunction is associated with the lesion and epileptic focus.

Rolak *et al.*[1] and Biton *et al.*[9] proposed that the great majority of the patients having Todd's palsy also have a structural lesion. The findings of our case correlates with this proposal. The presence of a structural lesion probably contributes to the prolongation of Todd's palsy duration. Todd's palsy is expected to last no longer than 24 hours. Rolak *et al.*[1] reported that the duration of Todd's palsy does not exceed 36 hours. However Biton *et al.*[9] showed that it can be as long as 10 days. The Todd's palsy cases of Helmchen *et al.*[11] improved within 70 hours. Savard *et al.*[10] reported nine cases of post-ictal psychosis lasting 3 hours to months after bouts of complex partial seizures in patients who were otherwise psychiatrically stable. In our patient, hemiparesis and hemihypesthesia resolved completely within 48 hours but cognitive symptoms related to right hemispheric dysfunction took 30 days to resolve enough to permit almost normal functioning. On EEG recordings taken just after the status period, there was diffuse amplitude depression on the right hemisphere. One week

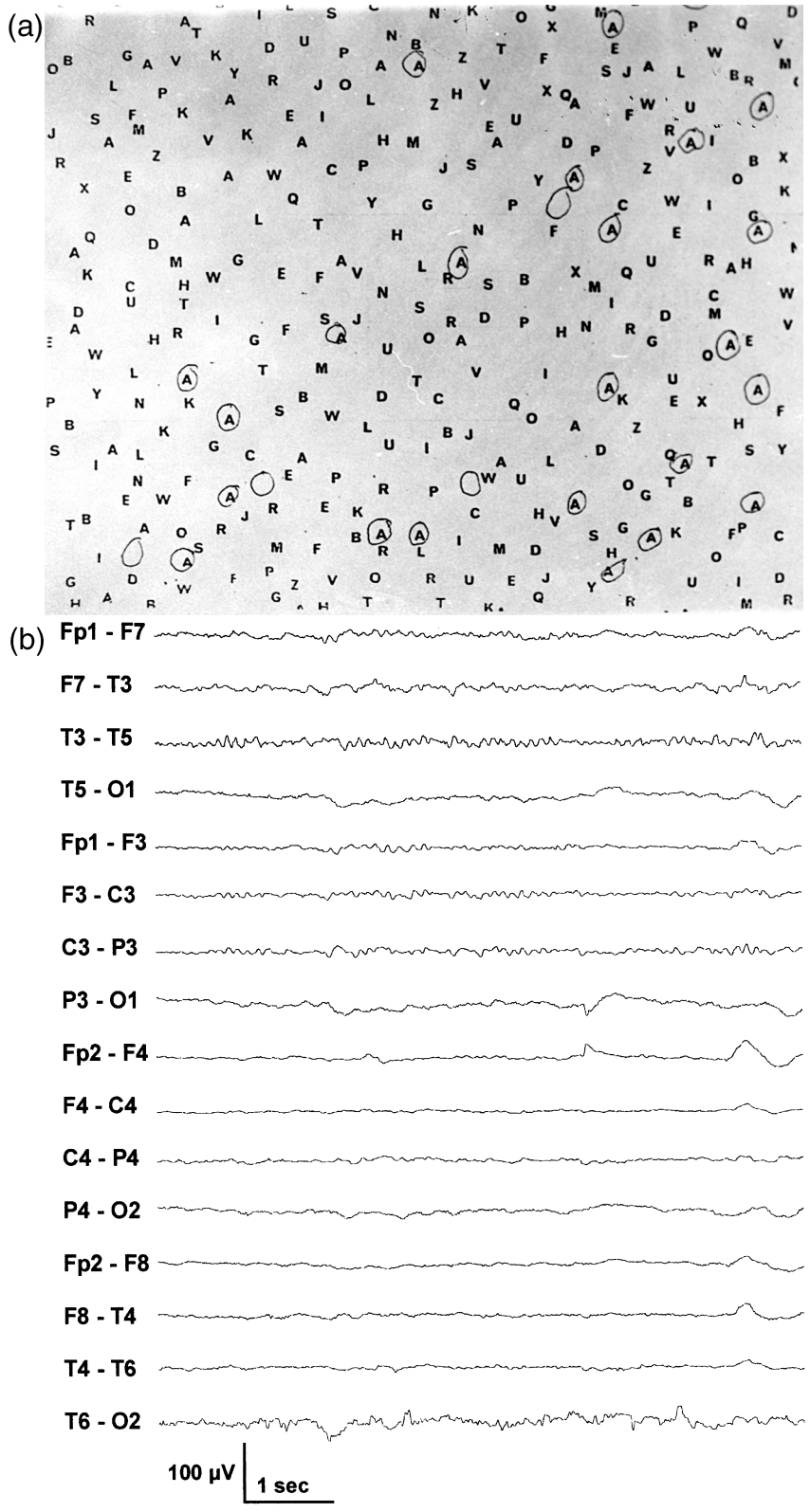

Fig. 2: (a) Left hemispatial neglect shown in a cancellation test on the second day of the patient's hospitalization after status epilepticus. Editor's note: This figure might be interpreted as due to an upper quadrantic field or visual defect. (b) Diffuse amplitude depression in the right hemisphere on the second day of the patient's hospitalization after status epilepticus.

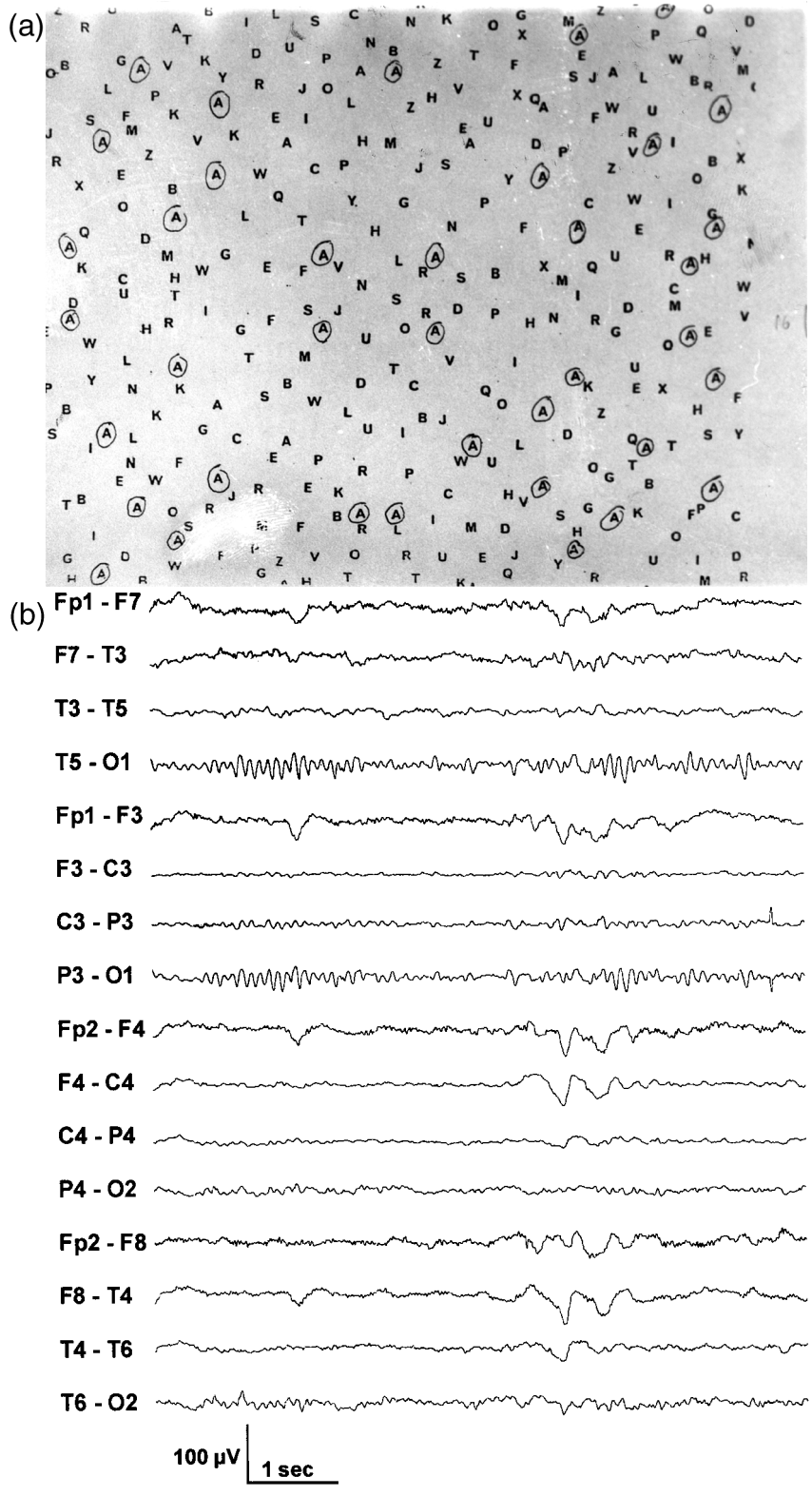

Fig. 3: (a) Improvement of the hemispatial neglect shown in a cancellation test on the sixth day of the patient's hospitalization. (b) Localized slow waves in the right fronto-temporo-parietal region on the sixth day of the patient's hospitalization.

later, amplitude depression on EEG and the cognitive dysfunction had partially improved but right fronto-temporo-parietal slowing persisted on repeated EEG recordings.

These findings suggest to us that: (i) the late bioelectrical slowing on right hemisphere and slight cognitive defects were due to the structural lesion identified on MRI; (ii) the hemispatial neglect with marked visuospatial-constructive impairment which improved after 6 days could be interpreted as a Todd's palsy phenomenon on a background of increased cerebral tissue sensitivity; and also (iii) the concurrently recorded EEG low amplitudes from the right hemisphere showing a parallel improvement also reflect this condition.

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