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Hemiconvulsion-hemiplegia-epilepsy syndrome

A clinical, electroencephalographic and neuroradiological study

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Abstract Six patients (4 boys and 2 girls) with hemiconvulsion-hemiplegia-epilepsy (HHE) syndrome are described. They had prolonged seizures, lasting from 30 min to 12 h, at ages 1–4 years. These took the form of hemiconvulsion in three of the children and generalized tonic–clonic seizures in the others, being preceded by hemifacial twitching or head and eye deviation in two. They were followed by hemiplegia, which cleared with time in five patients, apart from subtle pyramidal tract signs. One child had spastic quadriparesis, choreiform movements, contracture deformities and severe mental retardation following repeated status epilepticus. Subsequent epilepsy developed in five patients and was satisfactorily controlled with carbamazepine and/or phenobarbitone. Cerebral hemiatrophy was documented in all patients by cranial computed tomography and/or magnetic resonance imaging. Single photon emission computed tomography (done in 4 patients) showed ipsilateral hypoperfusion (of the damaged hemisphere). Electroencephalography showed ipsilateral slowing and low voltage of background activity. Epileptiform discharges were found on the ipsilateral side in two cases and the contralateral side (the undamaged hemisphere) in one.

Key words Hemiconvulsion-hemiplegia-epilepsy syndrome · Child · Neuroradiology

Introduction

Hemiconvulsion-hemiplegia-epilepsy (HHE) syndrome is one of the recognized sequelae of convulsive status epilepticus [8]. It is characterized by the occurrence, in the course of a febrile illness in a child younger than 4 years, of prolonged clonic seizures with unilateral predominance, followed by the development of hemiplegia. Neuroradiological studies show oedematous swelling of one hemisphere at the time of the initial convulsive status, followed a few weeks or months later by characteristic global cerebral hemiatrophy independent of any vascular territory [2, 10]. Subsequently epilepsy supervenes, with seizures usually originating in the hemisphere contralateral to the hemiplegia.

The incidence of the syndrome has declined considerably in the industrialized countries over the past 15 years [1, 18], probably because effective management of status epilepticus (and especially the introduction of intravenous diazepam in the early 1960s) has become more widely available [15].

We describe the clinical and electroencephalographic (EEG) features of five Saudi children and one Yemeni girl

who had HHE syndrome. The neuroradiological findings, including those obtained with cranial computed tomography (CT), magnetic resonance imaging (MRI) and single photon emission computed tomography (SPECT), are also outlined. Hitherto, the syndrome has not been reported from the Middle East and we are unaware of any report of SPECT features in patients with HHE syndrome.

Methods

Electroencephalographic evaluation included scalp recordings using the international 10/20 system with 16–21 channel recordings [9]. Regional cerebral blood flow was assesed using technetium-99m (^{99m}Tc) hexamethylpropyleneaminoxime (^{99m}Tc-HmPAO) or ^{99m}Tc ethylcysteinate diamer (^{99m}Tc-ECD) SPECT. This investigation was done, interictally, for four patients following the utilization of SPECT for paediatric patients at King Khalid University Hospital (KKUH), Riyadh, in 1993.

Case reports

A summary of the clinical features of six patients with HHE syndrome is shown in Table 1.

Case 1

A 12-year-old Saudi boy who lived in a desert in the northern part of Saudi Arabia was transferred (age 10 years) from a regional hospital for the investigation of left-sided weakness associated with abnormal movements. The condition started with a left-sided hemiconvulsion, the duration of which could not be ascertained by the parents, followed by a generalized tonic-clonic (GTC) seizure. Within minutes of his arrival at the hospital, this responded to clonazepam and phenobarbitone. However, on the following day, the boy was noted to have left hemiparesis associated with left choreiform movements. The abnormal movements subsided after 12 days, and his speech slowly recovered whereas, the hemiparesis remained the same.

He had had his first seizure when aged 1 year, in the form of a ‘prolonged’ left hemiconvulsion involving the face and the limbs. He was taken to hospital and was discharged with residual left hemiparesis, which resolved with time. Thereafter, the parents noted that he was slow to learn compared with his siblings, but he was later able to go to a regular school.

When examined on arrival, the boy looked well, alert and fully oriented. He had dysphasia but showed no involuntary movements. Examination of the central nervous system (CNS) revealed left-sided hypertonia, muscle weakness (grade 3 power in upper limb extensors and lower limb flexors, according to the MRC scale [12]) exaggerated deep tendon jerks (with no clonus) and positive Babinski sign.

Routine laboratory investigations were unremarkable, the sickling test and urine test for neurometabolic screening being negative. Electrocardiography and echocardiography were normal. Electroencephalography (EEG) revealed an excessive amount of bilateral theta-delta activity with predominance on the right side. There was gross voltage asymmetry, with lower voltage on the right seen predominantly in the fronto-temporal regions. A CT brain scan showed right cerebral hemiatrophy. Bilateral carotid angiography revealed a normal appearance of both internal carotid arteries and their main branches. There was no evidence of vascular displacement, abnormal narrowing or tumour blush. Hypoperfusion of the right hemisphere was evident on SPECT scan (Fig. 1).

The patient’s condition improved during his stay in hospital; he remained seizure-free and gained some speech. He was discharged on phenobarbitone (3.5 mg/kg per day). At follow-up examinations, he showed progressive improvement in his motor power, but he did not go back to school owing to residual speech and memory handi-

Table 1 Summary of the clinical features of six patients with hemiconvulsion-hemiplegia-epilepsy syndrome (*GTC* generalized tonic-clonic, *IQ* intelligence quotient, *L* left, *R* right)

	Patient 1, M, 12 years	Patient 2, F, 12 years	Patient 3, M, 12 years	Patient 4, M, 9 years	Patient 5, M, 9 years	Patient 6, F, 5 years
Longest initial convulsion						
Age at onset (years)	1	2	3½	3	1	4
Duration	Prolonged	12 h	30 min	1 h	Prolonged	30 min
Type	Hemiconvulsion (L)	Hemiconvulsion (R)	GTC	Hemiconvulsion (L)	GTC	GTC
Sequelae	Hemiparesis (L)	Hemiparesis (R)	Hemiparesis (R)	Hemiparesis (L)	Hemiparesis (L) + dysarthria	Hemiparesis (L) + dysphasia
Residual epilepsy						
Age at onset (years)	10	2	9	3	1	None as yet
Type	Focal first ± GTC	Hemiconvulsion (R) + GTC	GTC	Hemiconvulsion (L)	GTC	–
Sequelae	Hemiparesis (L), Choreiform movements (L) dysphasia (resolved), low IQ	Spastic quadriparesis, contracture deformities, choreioathetosis, dystonia, mental retardation	Hemiparesis (R), mental retardation	Low IQ	Hemiparesis (L), mental retardation, truncal ataxia	

caps. At the age of 12 years, he was still free of seizures but had an intelligence quotient (IQ) of only 75 on the Stanford-Binet test. Neurological assessment 6 months later revealed normal power, tone and coordination on both sides. Abnormal signs were confined to brisk deep tendon jerks on the left side, associated with a positive Babinski sign. He also demonstrated abnormal co-movements of the left hand when walking on his heels and on Fog's test [7] (Fig. 2).

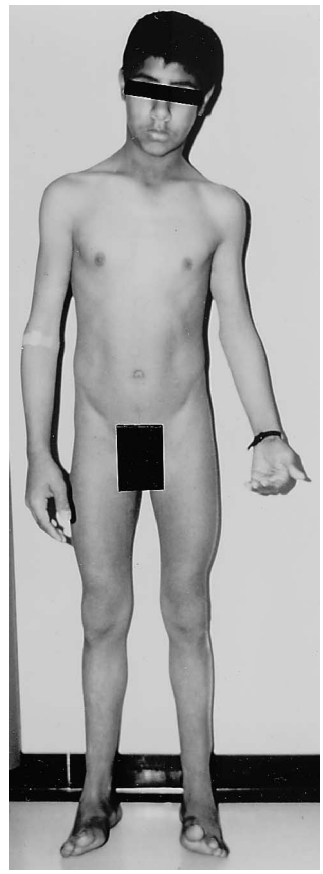

Fig. 2 Patient 1 with residual left-sided hemiparesis at the age of 12 years. Abnormal co-movements of the left hand are seen on Fog's test [7]

Case 2

A 12-year-old Saudi girl was admitted electively for work-up because of psychomotor retardation and uncontrolled seizures.

She had been well up to the age of 3 months, when she received the second dose of oral polio and DPT vaccines. This was followed by a febrile GTC seizure lasting for about 10 min and aborted in hospital. Thereafter, she remained well apart from recurrent febrile convulsions, each lasting for <20 min. At 2 years, and while travelling, she had a succession of convulsive episodes lasting for about 12 h, between which she did not regain consciousness. Each started with hemiconvulsion involving the right side, followed by GTC seizures with deviation of the mouth and eyes to the right side. No anticonvulsant therapy was given. Following this episode, the girl was noted to have right-sided weakness and could neither walk, crawl nor feed herself. She was taken to the hospital and was started on phenytoin but the seizures were not controlled (about 3/month). At 5 years of age, she developed another prolonged fit, similar to the previous one, that lasted for about 2.5 h. Thereafter, she could no longer either sit independently or speak. The frequency of the seizures remained about the same despite therapy with phenytoin and phenobarbitone. She bacame totally dependent and needed continuous care at home.

On examination, she was conscious, cooperative and able to obey simple commands (e.g. opening her mouth). However, she was aphasic and microcephalic (head circumference 47.5 cm <2 SD). She showed facial grimacing and choreoathetoid movements associated with dystonic posturing of the upper limbs. She also had spasticity affecting all four limbs with multiple contracture deformities associated with scissoring of the legs. Tendon jerks were brisk on the left side and could not be elicited on the right side because of the contractures.

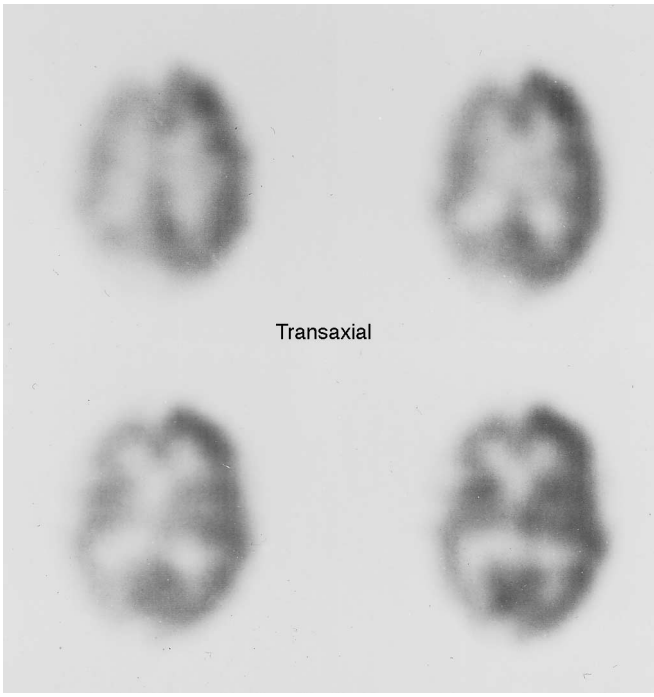

Fig. 1 Single photon emission computed tomography (SPECT) brain scan in case 1, showing hypoperfusion of the right cerebral hemisphere

Routine laboratory investigations were unremarkable. The phenobarbitone level was 9 μg/ml (N=15–40 μg/ml) and the phenytoin level, 0.2 μg/ml (N=10–20 μg/ml). Electroencephalography (EEG) and video EEG revealed asymmetric background with low-voltage delta-theta activity on the left side. During sleep, there were frequent bursts of spikes with phase reversing in the left centrotemporal area. Brain auditory evoked responses (BAER) and electroretinogram (ERG) were normal, whereas visual evoked potentials (VEP) showed bilaterally delayed P100 latencies. Magnetic resonance imaging (MRI) revealed left-sided cerebral hemiatrophy (Fig. 3A). X-ray of the pelvis showed subluxation of the left femoral head. The patient underwent open bilateral adductor tenotomy and anterior obturator neurectomy to reduce scissoring, improve hygiene and also keep the left hip in place. She was enrolled in a comprehensive rehabilitation programme and her condition was successfully controlled with carbamazepine following gradual withdrawal of phenobarbitone and phenytoin.

Case 3

A Saudi boy was referred at the age of 12 years for the evaluation of uncontrolled epilepsy. He had had normal early development, remaining well apart from an attack of measles at 3 years of age. At

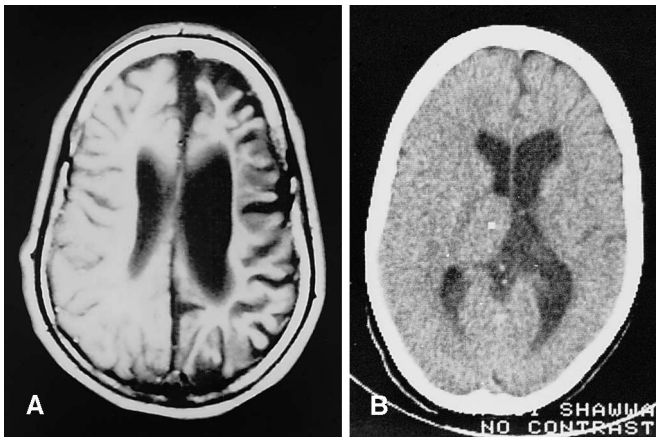

Fig. 3 **A** Global left-sided cerebral hemiatrophy on T1-weighted MRI image in case 2. **B** Cranial CT in case 4, showing atrophy of the right hemisphere and dilatation of the right ventricle. Note the associated atrophy of the right caudate nucleus and thalamus

the age of 3½ years, he had a prolonged seizure following a febrile illness. This was described by the parents as starting first with deviation of the head and eyes to the right side followed by GTC convulsions that lasted for 30 min. He recovered with residual deterioration in speech and an abnormal limping gait with paucity of movement on the right side. At 4 years, he had a similar attack lasting for about 1 h and was put on sodium valproate (30 mg/kg per day). However, he had another four episodes of shorter febrile seizures (<15 min) and he was admitted following each of these for a short stay. At 9 years of age, he was admitted to hospital following three episodes of GTC seizures each lasting for about 30 min. He was recorded as mentally retarded, and neurological examination was reported to be normal. The dose of Na valproate was raised to 40 mg/kg per day. An EEG 2 weeks after discharge showed an asymmetric background with slower and lower voltage in the left than the right side of the brain. There were also frequent bursts of isolated spikes focalized in the right mid-temporal region (Fig. 4).

Subsequently, this patient was irregular in attending for follow-up. When first seen at the paediatric neurology clinic, he still had one attack /month of GTC seizures lasting for 5 –10 min. Neurological examination showed normal findings apart from abnormal co-movements of the right hand on Fog's test and during walking on the heels. Deep tendon jerks were brisk on the right side and were associated with a positive Babinski sign. Brain CT scan showed mild left-sided cerebral hemiatrophy. Gradual replacement of Na valproate by carbamazepine resulted in better control, and seizure activity was confined to short GTC lasting for few seconds associated with lateral deviation of the eyes. These used to happen only during febrile illnesses.

Case 4

A Saudi boy was referred at the age of 9 years because of uncontrolled epilepsy. He had been well up to the age of 2 years, when he had a febrile seizure consisting of tonic-clonic movements of the left arm and leg with turning of the head to the left side and generalized stiffening of the rest of the body. The family could not recall its duration. Two months later, he had another febrile seizure lasting about 15 min. At the age of 3 years, he developed a prolonged left-sided hemiconvulsion lasting for 1 h, which subsided without treatment but was followed by a limping gait. During the following week, he had several similar daily attacks (for about 5–10 min) and received Na valproate. However, the seizure frequency remained the same although they started to occur only during sleep.

Examination revealed no abnormality apart from a difference in the size of his thumbnails, the left one being smaller. He was shy, and the Stanford–Binet test revealed an IQ of 65. Abnormal neurological findings were confined to brisk deep tendon reflexes on the left, associated with a positive Babinski sign, and co-movements of the left hand observed when he was asked to walk on his heels and on Fog's test. Investigations included an EEG, which showed gross asymmetry with continuous runs of low-voltage delta and theta waves on the right side. Well-organized alpha rhythms (at 9 Hz) were seen on the left but these were absent on the right. There were also frequent discharges of spikes and slow waves (S–SW) on the right posterior temporal and middle parietal areas. No photic following was observed on the right side. Brain CT scan (Fig. 3B) and MRI revealed right cerebral hemiatrophy, whereas a SPECT brain scan showed hypoperfusion of the right cerebrum.

His anticonvulsive medication was replaced by carbamazepine and he has remained seizure-free for the last 15 months.

Case 5

A 9-year-old boy, who lives in a remote area in the northern part of Saudi Arabia, was admitted for the evaluation of uncontrolled epilepsy. He had been developing normally until the age of 1 year, when he had GTC convulsions associated with a febrile illness. The parents did not recall its approximate duration, although they stated that he remained unconscious for about 2 h. Following this episode, he developed an abnormal staggering and limping gait and his cognitive behaviour was noticeably slower than that of his siblings. Subsequent to phenobarbitone medication the frequency of convulsions decreased to about one every 3 months. The seizures were still GTC, febrile and afebrile and lasting under 10 min. Six months before the boy's admission, they increased in frequency to about one/week, and he developed nocturnal enuresis. Neurological examination revealed dull mentation and brisk tendon reflexes on the left side, associated with a positive Babinski sign. He had mild truncal ataxia and was unable to walk along a straight line, but he had no intension tremor. He could neither walk on his heels nor demonstrate Fog's test.

On admission, this patient had a low level of phenobarbitone (0.6 μg/ml, N = 15–40 μg/ml). Routine biochemical and neurometabolic work-up were negative. Nerve conduction studies and electromyography were normal. Electroencephalography (done during sleep/sedated state) showed a diffusely slow background, which was slower and of lower voltage in the right side. The usual sleep phenomena (vertex sharp waves, sleep spindles and K-complexes) were lacking. No epileptiform discharges were seen. Cranial CT and MRI scans showed mild right-sided cerebral hemiatrophy. No abnormal signal intensities were revealed by MRI, but there was mild cerebellar vermis atrophy. A SPECT scan of the brain showed hypoperfusion of the right hemisphere, mainly involving the parietal and occipital regions.

During the boy's hospital stay, he continued to have convulsions despite adequate phenobarbitone therapy and drug levels. The addition of carbamazepine resulted in substantial control. On subsequent clinic visits (which were irregularly kept by the patient) the frequency of seizures was noted to be about two short (< 2 min) GTC convulsions within a period of 4 months.

Case 6

A previously healthy, developmentally normal 4-year-old Yemeni girl had a convulsion shortly after running frightened to her mother. The seizure was characterized initially by deviation of her mouth to the right, right-sided hemifacial twitching, lip smacking and uprol-

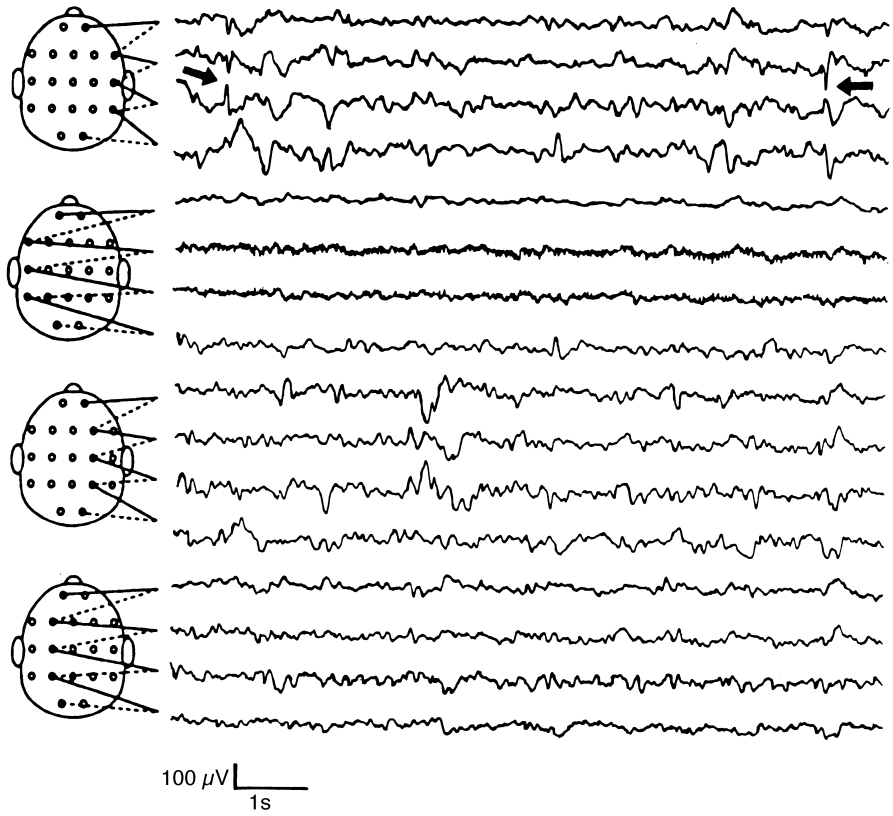

Fig. 4 EEG during sleep/sedation in case 3, showing asymmetric background with slower and lower voltage on the left than on the right side. Bursts of isolated spikes are seen in the right mid-temporal region (*arrows*)

ling of the eyes. This was followed by a GTC convulsion lasting for a period of 30 min. On the following day, the mother noticed that she had a limping gait and could not use her left hand properly, and that her speech had also become slow. Her power recovered with time, although she tended to use the left hand less efficiently. Mentation and speech were back to normal and she remained seizure free. No anticonvulsants were prescribed.

On examination at the neurology clinic (aged 5 years), she had normal speech and it was noted that she interacted in an intelligent way. She had no facial weakness but walked with a limp on the left side, with decreased associated movements. Tone was increased in upper and lower limbs. There was demonstrable weakness on the left side (grade 4 power on the upper limb extensors and lower limb flexors according to MRC scale). Tendon jerks were brisk on the left and the plantar reflex was upgoing. Because of the left-sided weakness, she could neither walk on her heels nor demonstrate Fog's test.

Routine laboratory tests revealed no abnormalities. An EEG (done during sleep/sedated state) showed a background dominated by theta-delta activity, with superimposed drug-induced beta waves. It was mildly asymmetric, with more slowing and less beta activity on the right. No epileptiform discharges were observed. MRI showed mild right-sided hemispheric atrophy.

Discussion

HHE syndrome, as described by Gastaut et al. in 1960 [8], starts with an initial phase of convulsive seizures. These are usually clonic in type, as seen in our patients, and often prolonged in the form of status epilepticus that may extend over several hours. The ascertained duration of the initial status in this cohort ranged between 30 min and 12 h. In a group of 73 patients reported by Aicardi et al. [4], it lasted longer than 24 h in 31 cases and for more than 6 h in another 20. The jerks are usually unilateral, as was observed in three of our six patients [1], but they may start on one side and cross to the other or may be initially generalized [1, 4]. The initial seizure was associated with fever in three of the six patients, compared with 75% in large series [3], and in two of them status epilepticus had been preceded by episodes of shorter convulsions, as opposed to 18/89 reported in the series of Aicardi et al. [4]. The presenting seizures of HHE syndrome (which are unilateral or predominantly unilateral) are generally associated with coma and are immediately followed by the appearance of hemiplegia involving the side of the body where the convulsions were localized [1]. When the convulsions start on one side and cross to the other, the side involved last is the one that remains hemiplegic [4]. The hemiplegia, initially flaccid, eventually becomes spastic, although the intensity often tends to decrease [3]. It disappeared within 12 months in 20% of the cases reported by Gastaut et al. [8]. However, Aicardi [1] noted that even when the paralysis cleared, some degree of spasticity, increased deep tendon reflexes and pyramidal tract signs persisted. We observed

similar findings in four of our patients and found associated co-movements of the hand (on the affected side) when walking on the heels and on Fog's test [7] to be additional helpful physical signs. One of the remaining two patients had demonstrable weakness 1 year after the onset of hemiplegia, whereas the other remained quadriparetic following two periods of status epilepticus that lasted for 2.5 h and 12 h, respectively. Spastic quadriplegia following repeated status epilepticus has been reported in patients with HHE syndrome [4].

Two patients developed choreiform movements, which were unilateral, involved the side of the hemiconvulsion and resolved with time in one and were generalized and permanent in another. Movement disorders (predominantly choreiform) have been observed following status epilepticus [6]. Four patients had aphasia or an observable language deficit at the onset of hemiplegia, which cleared with time in all but one girl who experienced recurrent status epilepticus. Aphasia was reported to follow hemiplegia in 22 of 89 patients with HHE syndrome and was usually transitory, lasting for under 2 months [4]. On the other hand, all of the five children who were evaluated 1 year after the onset of hemiplegia were noted to have residual mental impairment. Mental retardation has been reported to be associated with hemiplegia in 80% of HHE patients [4].

Hemiconvulsion–hemiplegia syndrome will evolve to the secondary appearance of partial seizure in 56–70% of patients [4, 8, 16], with 85% of the epilepsies having started within 3 years of the initial hemiconvulsion following an average interval of 1–2 years [4]. All of the five children in this study who were followed up for longer than 1 year after the onset of hemiplegia had complex partial seizures, with secondary generalization in three of them. Complex partial seizures, account for about two-thirds of the late seizures in HHE syndrome [4, 18]. Approximately one-third of HHE patients develop simple partial seizures; 20%, secondarily generalized seizures; and 10%, repeated episodes of status epilepticus [4].

The abnormalities seen on EEG tracings of the six patients consisted of slow waves that were either unilateral or predominantly unilateral, as reported previously [4, 10]. Epileptiform discharges were found on the ipsilateral side (the damaged hemisphere) in two cases and the contralateral side (the undamaged hemisphere) in one. In a similar study in 25 children [10], epileptiform discharges were ipsilateral (in the damaged hemisphere) in 13, contralateral in 9 and on both sides in 3.

Neuroradiological studies revealed cerebral hemiatrophy on CT and/or MRI scan in all patients. The involved hemisphere was uniformly atrophic with ventricular dilatation and cortical atrophy; a different picture from the more limited atrophies observed with ischaemic lesions of vascular origin [1]. Cerebral angiography was done in the first patient seen in this cohort (case 1) and was found to be normal; Aicardi's experience was similar in a large series of 31 patients who had been subjected to this investigation [4]. On the other hand, interictal SPECT brain scans were helpful in delineating the hypoperfused atrophic hemisphere in the four children investigated. This procedure may prove to be a valuable additional diagnostic procedure in the evaluation of patients with HHE syndrome.

In the present cohort, epilepsia partialis continua (Rasmussen syndrome) – which may superficially simulate HHE syndrome – was excluded on clinical grounds. The former condition is characterized by the occurrence of *continuous* myoclonic jerks, localized to a limited area on one side of the body [1]. It evolves into *progressive* hemiplegia with mental deterioration and may end fatally or leave severe neurological sequelae after an active period of months or years.

In many cases no obvious cause is found for the status hemiconvulsion that results in brain damage, although it may herald an acute encephalopathy (e.g. meningoencephalitis) [3]. Patient 5, who developed status epilepticus at the age of 1 year, recovered with residual hemiparesis and truncal ataxia. His CT scan revealed cerebral hemiatrophy and also mild cerebellar vermis atrophy, which could have been the sequela of an acute infectious/parainfectious cerebellar ataxia [5]. Conversely, prolonged seizure activity may cause brain injury independent of ischaemia, hypoxia or other metabolic factors [11, 19], a complication that has been supported by both neuroradiological [2, 8, 10] and neuropathological studies [20]. These seizure activities can occur in a structurally intact brain in the case of cryptogenic status epilepticus, or result from an asymptomatic hemispheric lesion of prenatal or perinatal origin that initiates or localizes the seizures [1, 3]. Yet, irrespective of the cause, early and adequate treatment of prolonged infantile seizures, especially of febrile convulsions, would reduce the incidence of HHE syndrome. This has been reflected in the rapid decline of its incidence in the developed world and has been ascribed to the better general health of children and to the availability of more effective drugs for terminating prolonged seizures [14]. It is also reflected in the relatively older age of children in the present report (mean=9.8 years), whose infancy fell at the start of the rapid developments in social welfare and health care in Saudi Arabia [13].

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