Sudden 'Stroke-Like' Onset of Hemiparesis Due to Herpetic Encephalitis

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ABSTRACT: ***Background/Objective*:** Herpes simplex encephalitis usually has a progressive cause. Sudden neurological deficits are unusual. ***Method*:** Case study. ***Results*:** A 17-year-old girl presented with an acute onset focal neurological deficit followed one week later by the more classical feature of altered level of consciousness, fever and focal seizures. The diagnosis of hepetic encephalitis was made by magnetic resonance imaging (MRI) and by the significant increase in cerebrospinal fluid titres of antibodies against herpes simplex type I. ***Conclusion*:** Herpetic encephalitis should be considered in the differential diagnosis of acute stroke in young patients even in the absence of encephalitic features, if common etiological factors such as embolization and intracerebral bleed are excluded.

RÉSUMÉ: Hémiparésie à début subit due à une encéphalite herpétique. ***Introduction et objectif:*** Nous décrivons un cas d'encéphalite, due au virus de l'herpès simplex, dont le mode de présentation clinique a été inhabituel par le début subit de l'hémiparésie, à la façon d'un ictus cérébral. ***Méthode:*** Une jeune fille de 17 ans a présenté un déficit neurologique suivi, une semaine plus tard, par les manifestations plus classiques: une altération de la conscience, de l'hyperthermie et des convulsions focales. ***Résultat:*** Le diagnostic reposait sur les manifestations observées à l'imagerie par résonance magnétique (MRI) et sur l'augmentation significative des titres d'anticorps contre le virus de l'herpès simplex I dans le liquide céphalo-rachidien. ***Conclusions:*** L'encéphalite herpétique devrait être incluse dans le diagnostic différentiel de l'ictus cérébral aigu chez le jeune patient, même en l'absence de manifestations d'encéphalite, si les principaux facteurs étiologiques courants, tels l'embolie ou l'hémorragie intracérébrale, ont été exclus.

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Herpes simplex virus (HSV) infections are distributed worldwide.[1] HSV infection of the central nervous system is among the most severe of all human viral infections of the brain,[2] and causes an acute necrotizing encephalitis with edema, necrosis and hemorrhage that selectively affects the temporal and frontal lobes.[3] The usual clinical presentation is with fever, headache, focal neurological deficit and focal seizures, with progression to confusion, disorientation and possibly coma within a few days.[3] In this report, we describe a case presenting like an acute stroke.

Case Report

A 17-year-old girl was admitted to Security Forces Hospital with the complaint of headache and left-sided weakness. The patient was healthy until ten hours prior to admission, when she developed severe right hemicranial throbbing headache, associated with nausea, and followed one hour later by sudden weakness of the left upper and lower limbs with deviation of the mouth to the right side. The headache improved with the onset of the weakness. The patient had no other medical problems and was taking no medication. There was no history of migraine, nor of heart disease, nor any history of recent travel or exposure to toxins. She was newly married, and not on contraceptive medication.

Physical examination revealed a conscious, oriented young lady with normal vital signs. The fundus examination showed sharp disc margins and no vascular changes. The neck was supple. Cranial nerve examination showed an upper motor neuron facial nerve palsy on the left side. Motor system examination revealed weakness on the left side with power 0/5 in the arm and 2/5 in the leg, hypotonia, hyporeflexia and an extensor plantar response on the left. There were no cerebellar signs and no signs of meningeal irritation; the skin and the mucous membranes of the oropharynx were normal. Chest, heart and abdominal examination showed no abnormality.

Laboratory data included hemoglobin of 12.5 g/dL; white blood count of 6,400/mm3 (67% neutrophils + 26% lymphocytes and 5% monocytes); platelet count of 272,000/mm3. Serum sodium (140 mmol/L); potassium (4.2 mmol/L); calcium (2.3 mmol/L); phosphate (1.05 mmol/L); total protein of (76 g/L); creatine kinase (106 u/L); and arterial blood gas studies were normal. VDRL test was non-reactive and thromboplastin time was normal. Protein C, protein S, and antithrombin III were normal; antinuclear antibodies, antiDNA, ESR, C3 and C4 were also normal. Brucella serology was negative. Echocardiography showed a normal heart. A contrast-enhanced brain CT scan was normal. The CSF examination done on admission showed clear colorless fluid with no WBCs but 400 RBCs, protein 0.49 g/L and sugar 3.4 mmol/L. Gram stain and stains for acid fast bacilli were negative. Antibodies against herpes simplex virus were not found in the CSF or the blood (less than 30 ELISA units).

The patient's condition remained stable for five days after admission while she was receiving supportive measures. On the 6th day, she became febrile and confused with a temperature of 38°C; a focal left-sided seizure which became generalized then occurred and recurred three times with deterioration of consciousness and respiratory compromise. She received 10 mg i.v. diazepam and 500 mg phenytoin but despite this, she had two additional seizures resulting in marked hypoxia.

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She was intubated and ventilated, and received an additional 500 mg phenytoin which controlled the seizures. An urgent brain CT scan done on the same day showed an enhancing lesion with a small bleed mainly in the right temporal lobe with marked edema (Figure 1). EEG showed slow waves with occasional sharp waves over the right centro–temporal region suggestive of herpes simplex encephalitis. Although the presentation was atypical, treatment with acyclovir and dexamethasone was initiated. Lumbar puncture was thought to be contraindicated because of the temporal lobe swelling. The patient was extubated after three days after regaining consciousness and being seizure-free. A gadolinium-enhanced MRI (Figure 2) on the tenth day showed the same picture as did the CT scan. A follow-up CT scan showed resolution of the brain edema and the temporal lobe swelling. A second CSF examination showed clear colorless fluid with 22/mm lymphocytes, RBCs, 45/mm, protein 0.55 g/L and glucose 3.9 mmol/L (blood glucose 5.9 mmol/L). Antibodies against herpes simplex type I was elevated in both CSF and serum; 210 ELISA units in CSF and 1986 in the serum, results strongly suggestive of herpes simplex encephalitis.

Subsequently, the patient's neurological status showed progressive improvement. She was discharged from the hospital after 14 days walking unsupported with residual weakness of the left upper limb.

Discussion

Herpes simplex virus is the commonest cause of severe focal encephalitis in man,[4] with an approximate annual incidence, in the USA, Sweden, England and other western countries of 1 in 250,000.[5,6,7] It accounts for up to 10-20% of encephalitic viral infections of the CNS worldwide.[8] Such data are not available from any Arab country.

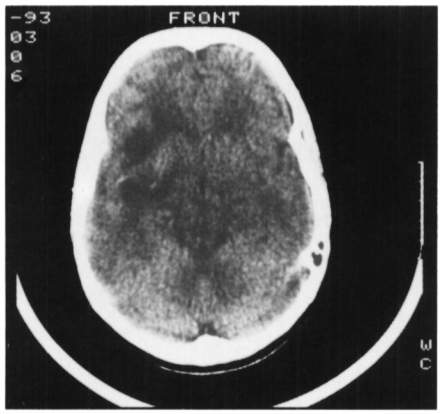

Figure 1: CT brain done 6 days after admission showing enhanced right temporal lobe lesion with oedema and small bleed.

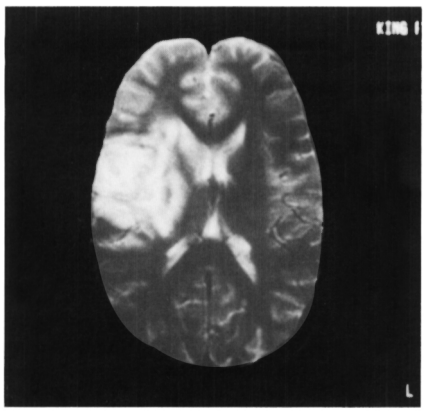

Figure 2: A Gadolinium-enhanced MRI image done on the 10th day showing the same lesion seen in the CT scan.

The clinical findings of herpes simplex encephalitis (HSE) are non-specific and do not allow for empirical diagnosis of the disease based solely on the clinical presentation.[9] HSE is the only viral CNS infection for which therapy has proved useful in rigorously controlled trials;[7,10] successful therapy depends on a high level of suspicion. As noted before, the clinical diagnosis is often presumptive and supported by evidence of focality on physical or radiologic evaluation.[10]

The diagnosis in our case was eventually made by the MRI findings and by the significant increase in CSF antibody titres from 0 to 210 ELISA units over 10 days. MRI is thought to be the most sensitive noninvasive test in the diagnosis of HSE, especially in the early stages.[3] The detection of a rise in CSF antibody titres has been considered as a useful diagnostic test but only retrospectively; the finding has a sensitivity ranging from 70% to 90% and a specificity ranging from 81% to 88% depending on the type and number of serologic tests used.[4] The definitive diagnosis of HSE depends on either a positive brain biopsy result or (increasingly) on polymerase chain reaction amplification of HSB DNA from the CSF. Neither of these studies could be done in our centre. The difficulty in diagnosis in our case was due to the atypical presentation, not previously recorded in the literature. The stroke-like onset, without any encephalitic features appearing for a week is unusual, especially with no evidence of bleeding on the CT scan. Normal CSF findings on admission have been reported previously.[11,12] Early MRI examination could be of great help in delineating the pathology at presentation.

We conclude that HSE should be considered in the differential diagnosis of acute stroke in young patients with no obvious embolic source, evidence of carotid artery dissection, nor evidence of intracerebral bleeding even in the absence of encephalitic features, since effective antiviral therapy improves the outcome and decreases the high mortality and morbidity.[10]

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