An unusual psychiatric emergency: herpes simplex encephalitis

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A case of fatal herpes simplex virus (HSV) encephalitis, presenting as a psychiatric emergency, is reported. The possibility of HSV encephalitis presenting mainly or solely with psychiatric symptoms is highlighted. HSV can cause a severe form of encephalitis which may present with mainly psychiatric symptoms in some cases. Early treatment with anti-viral agents can reduce mortality and morbidity, but accurate early diagnosis may be very difficult. HSV encephalopathy may mimic psychiatric illness and has been likened to syphilis as the great imitator. The case presented here should serve to raise awareness of the psychiatric features and the need to consider this diagnosis in patients with atypical behavioural disturbance.

Keywords: Herpes simplex encephalitis – Psychiatric symptoms – Behavioural disturbance

INTRODUCTION

Herpes simplex virus (HSV) Type 1 is one of the commonest causes of acute sporadic necrotizing encephalitis in temperate climates (Greenwood et al., 1983). Commonly it presents as a severe illness of rapid onset with pyrexia, headache, meningeal irritation, drowsiness, confusion and fits. This may proceed to delirium, coma and death. Focal neurological signs are common. Psychiatric symptoms can be a feature both in the acute phase and as a major sequel (Lishman, 1987). Patients presenting with psychiatric symptoms may appear initially to be only mildly unwell systemically. Mortality is high, ranging from 55% to 70%, though mortality rates have been reduced in recent years by anti-viral chemotherapy, provided this is given early in the course of the illness.

HSV encephalitis can mimic varied physical conditions including meningitis, subdural haematoma, head injury, tumour or brain abscess. Its psychiatric importance lies in its ability to present with puzzling psychiatric symptoms before overt evidence of CNS involvement is apparent (Wilson, 1976). In this respect it has been likened to syphilis as the great imitator (Hollander et al., 1965). When psychological symptoms are prominent then an acute psychosis or delirium tremens may be misdiagnosed, especially in the early stages when systemic symptoms are few. In specificity of diagnosis a computed tomography (CT) scan can be useful, as can an EEG, but the latter is not always diagnostic. Examination of cerebrospinal fluid (CSF) may reveal the presence of the virus or a fourfold or more rise in antibody titres. High antibody titres in blood indicates recent infection with the virus. Brain biopsy may show the characteristic inclusion bodies, which are diagnostic. The case presented here emphasizes the difficulty in the diagnosis of organic encephalopathies when the presentation is of a psychiatric nature.

CASE REPORT

The patient was a 24 year old, married female, who was referred for urgent psychiatric assessment. She had no significant past medical or psychiatric history and no known history of drug or alcohol abuse. Moroccan-born, she had lived for the past 20 years in the UK and worked as an auxiliary nurse in a psychiatric hospital. She was married, for the past two and a half years, to an Englishman and had no children. It was reported that she had been “well until 2 days earlier. She had gone to work as usual, but became distressed and agitated there, saying she ‘could not remember anything’. On returning home, she shouted at her husband that she was “sick of everything” and wanted to leave him. The GP was called because of her restlessness and agitation. She had been under some pressure at work recently, had had little time off for several months and had also been fasting for the Muslim feast of Ramadan. There
was also a history of marital friction, particularly in the past few weeks. Because of all these factors, and her disturbed condition, admission to a psychiatric hospital for observation and further assessment was suggested.

The husband contacted her parents to inform them of what was happening, but they were against her admission and came and took her to their home instead. The next day her father took her to a "faith healer", who reportedly stated that her husband had caused an "evil spirit" to affect her. The father subsequently prayed over her to rid her of this "spirit".

The patient continued to have periods of agitation, interspersed with periods of drowsiness, and made repeated references to the difficulties of the marital situation. In the early hours of the next day, the parents' GP had to be called because she had become very disturbed and had rushed out into the street. Chlorpromazine and haloperidol intramuscularly were given to sedate her and thioridazine 25 mg 6 hourly was also prescribed. She was referred to the community mental health team and was visited at home that same day by a community psychiatric nurse (CPN) and social worker. They found her to be too drowsy to assess properly and assumed this was due to the medication. The next day, the CPN again visited, this time accompanied by a psychiatrist.

At this time she still seemed drowsy though no other abnormalities were noted. She and the family attributed her behaviour as being due to the difficulties in the marital situation in addition to the other stresses previously mentioned. Admission to a psychiatric hospital was advised for further assessment, but the parents would not agree.

It was therefore suggested that the thioridazine be stopped and that her condition be reviewed the next day, or sooner, if there should be any further deterioration. Later on that day, her husband contacted the team to say that he had seen her and she seemed much better, was fully alert and they appeared to be attempting to resolve their marital problems. However, she had a grand mal fit the next morning and was admitted to a general hospital on the advice of the psychiatrist. She became unconscious and was transferred to a teaching hospital, where she was placed on a ventilator in the intensive care unit.

At this stage, the diagnosis was still uncertain; a drug overdose was regarded as a possibility, as was rabies, because of the patient's trip to Morocco a few months earlier. Investigations included an EEG, performed after the patient had lost consciousness, which revealed an abnormal record with generalized, irregular slow activity sometimes showing a burst suppression pattern. Magnetic resonance imaging (MRI) of the brain showed sinusitis but nothing else. CSF was reported as normal pressure, clear and colourless, with a protein of 210 mg/l, glucose of 3.9 mmol/l, white cell count 270/µl with 99% lymphocytes, red cell count 50/µl, no organisms seen on Gram stain, and no growth after culture. Oligoclonal band studies of CSF revealed a local synthesis of IgG, i.e. oligoclonal bands were present in the CSF but absent in serum, and there was an elevated number of free light chains. Antigen-specific IgG studies were positive for herpes simplex encephalitis. Cytopathology of CSF revealed a moderately cellular material including a few red blood cells together with numerous mature lymphocytes and occasional polymorphs and macrophages. Immunostaining demonstrated a mixture of B and T cells indicating a polyclonal population of lymphocytes. No malignant cells or organisms were seen. Complement fixation tests revealed a high titre specifically for HSV with a result of CSF 4 (other viruses less than 4), serum 32 (other viruses 16 or less). Given the reference range of the laboratory and in conjunction with the other evidence this was considered diagnostic of herpes simplex encephalomyelitis. Brain biopsy was not carried out. Acyclovir treatment was instituted. However, despite this and following a brief period of apparent improvement, the patient unfortunately died about 3 weeks later. A post-mortem examination was not carried out.

DISCUSSION

This case illustrates the potentially grave consequences of HSV encephalitis.

The patient's recent emotional stress, together with some rather complex sociocultural factors in her background, may have made a psychogenic explanation for her personality change and behavioural problems plausible. Raskin and Frank (1974) and Wilson (1976) describe bizarre or schizophrenia-like symptoms in HSV encephalitis and Koehler and Guth (1979) report a case of acute mania in "benign" HSV encephalitis, the patient at no time exhibiting any significantly neurological signs or symptoms. In addition Weinstein et al. (1955) described six cases of encephalitis which were consistent in initial presentation with schizophrenia, and in five of these emotional stress had preceded the condition.

Duncalf et al. (1989) reported a case of subacute sclerosing panencephalitis presenting as schizophreniform psychosis. The case reported here shows how HSV encephalitis can, in the early stages, resemble an acute functional psychosis and, though rare (about 50 cases of severe herpes encephalitis are reported annually in the UK), it should be borne in mind in
patients presenting with acute onset of behavioural disturbance or personality change, particularly in the absence of a previous psychiatric history or drug or alcohol abuse. Torrey (1986) has given guidelines which may help to indicate when an acute psychosis is encephalitic in origin. These are: (a) signs of influenza or headache immediately before or during presentation; (b) extremely disinhibited behaviour; (c) rapid onset of psychosis or seizure-like activity, vague neurological symptoms or neurological signs such as ataxia, nystagmus, or pupillary changes. If treatment with acyclovir is commenced prior to loss of consciousness, mortality rates can be greatly reduced (17% compared with 70% without therapy; Hanley et al., 1987). The severe cognitive sequelae of HSV encephalitis may also be reduced by early antiviral therapy.

However, the lack of a reliable non-invasive technique makes diagnosis in the early stages very difficult (Gordon et al., 1990). Hierons et al., (1978) exemplified this in 10 case reports of necrotizing encephalitis, where herpes simplex was inferred as the causal agent from the distribution of the pathological process, yet they failed to identify the virus in any of the cases, despite extensive investigation.

The prominent psychological symptoms at presentation, exemplified in this case, may be explained by the predilection of HSV for the temporal lobes and orbito-frontal cortex (Davis and Johnson, 1979) as opposed to other viruses which act more diffusely.

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REFERENCES

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