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Letter to the Editor

A CASE OF SUB-ACUTE SCLEROSING PANENCEPHALITIS PRESENTING WITH NONSPECIFIC PSYCHIATRIC SYMPTOMS

Dear Editor,

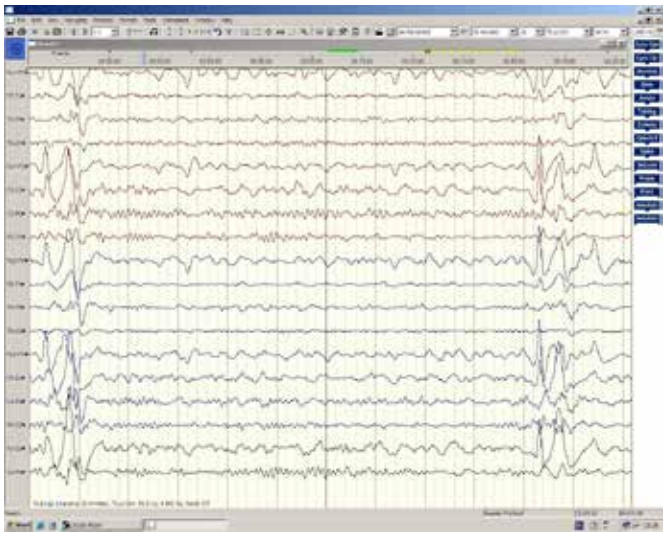
Subacute Sclerosing Panencephalitis (SSPE) was first defined by Dawson in 1933 (Tushar et al. 2012). It is a rare, late term complication of measles infection (Schönberger et al. 2013). Although the disease may be seen in patients between 1 month to 27 years of age, it is most commonly seen between the ages of 4 and 15 years (Chakor and Santosh 2013, Schönberger et al. 2013). Approximately 50% of patients evaluated clinically had measles disease or were vaccinated before 2 years of age (Garg 2002). The course of the illness is rapid and death occurs within 1-3 years after the start of symptoms (Praskanth et al. 2006). In the literature, remission occurs in only 5% of all cases (Garg 2002). Illness generally starts with intellectual dysfunction, personality changes, myoclonus and epileptic seizures (Mahendra et al. 2014). Myoclonus, unilateral movement disorders, grand-mal seizures, and visual disturbances appear later (Kandadai et al. 2014). Diagnosis is made by detection of rubeola antibodies in plasma or cerebrospinal fluid (CSF), and the demonstration of virus in brain tissue (Garg 2002, Kartal et al. 2015).

CASE

A 14-year-old male was brought to the neurology clinic due to decreased participation during lessons, increased reaction time to questions, and decreased communication with friends, all of which wasere observed by his teacher. His parents reported that they first went to a psychiatry clinic but the psychiatrist sent them to neurology to exclude an organic etiology due to the sudden onset of symptoms and the absence of any previous psychiatric history. His parents reported that he had measles infection at the age of 4 months and he was fully vaccinated. His family history was unremarkable. His physical examination was within normal limits. During the neurological examination he was conscious, cooperative, and his attention was within normal limits and he had normal orientation to space, time, and person. During his speech he exhibited mild difficulty in finding appropriate words and his reaction time was delayed during questioning. Other neurological examination findings were normal.

Biochemical work up (glucose, urea, creatinine, aspartate amino transferase, alanine aminotransferase, sodium, potassium, calcium, chloride, thyroid function tests, vitamin B12, hemogram), and cranial magnetic resonance imaging sequences (axial T1, T2, fluid attenuated inversion recovery) were normal. Electroencephalography (EEG) was performed to exclude encephalopathy, revealing periodic slow wave paroxysms (Figure 1). This EEG finding suggested SSPE, and lumbar puncture was performed as a result. The opening pressure was 160 mmH_2O, closing pressure was 110 mmH_2O, CSF glucose concentration was 92 mg/dl, protein concentration was 30 mg/dl, and CSF rubeola antibody index was 2.41. A diagnosis of SSPE was made based on the

Figure 1. Electroencephalography (EEG) record with recurrent slow wave activity.



EEG findings and significantly elevated CSF rubellarubeola IgG levels. Isoprinosine treatment was started after the diagnosis. During follow up the patient exhibited epileptic seizures, impairment in balance and motor function, dysarthric speech, disturbances in orientation, loss of cooperation, decreased level of consciousness, and difficulty with respiration. He was followed for approximately 9 months in our pediatric intensive care unit and then discharged to home with a mechanical ventilator.

DISCUSSION

As previously reported in the literature, childhood SSPE cases typically present as impairment in intellectual functions and behavior changes and later myoclonus, generalized seizures and disturbance in motor functions (Mahendra et al. 2014, Ravi et al. 2015). Although the initial symptoms in our case were nonspecific, progression of the neurological findings and clinical deterioration occurred later. However, in adult-onset cases so called atypical symptoms like visual impairment and catatonia occur more frequently at the onset of disease (Dayal and Balhara 2014). In the present case, the initial symptom was decreased social contact. The patient was initially brought to the psychiatry clinic because his teachers and parents thought these symptoms were due to a depressive episode.

CSF analysis and neuro-imaging are very important in SSPE cases that present with atypical clinical features. In EEG, decreased baseline activity, especially prominent rhythmic delta activity at the frontal region and spikes have been described in SSPE cases (Kartal et al. 2015). In our case, CSF analysis was performed just 10-15 days after the start of symptoms due to detection of rhythmic delta activity by EEG.

In conclusion, SSPE may begin with variable and atypical clinical figures. In patients who present to the psychiatry clinic with nonspecific symptoms such as decreased social contact, SSPE should considered. EEG is a readily applicable diagnostic method for cases of suspected SSPE.

Mustafa Çelik

e-mail: mustacelik@yahoo.com

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