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Acute Disseminated Encephalomyelitis Following Pneumococcal Meningitis Infection

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Introduction: Acute disseminated encephalomyelitis (ADEM) is an acute inflammatory and demyelinating disease of the central nervous system, resulting in various neurological symptoms. Usually, the disease appears following vaccination or systemic viral infections. In rare cases, the disease appears following pneumococcal infections.

Case Presentation: The patient was a 27 year-old man who was referred to the clinic following a few days of fever and cold with consciousness deficit and right hemiplegia. Based on the analysis of cerebrospinal fluid (CSF) and diagnosis of pneumococcal meningitis, he received suitable antibiotic treatment. Despite complete return of consciousness, good general condition, and negative smear and culture of CSF, fever continued and no considerable improvement was observed in the hemiplegia. Therefore, brain magnetic resonance imaging (MRI) was performed and according to the findings, treatment was started with the diagnosis of acute disseminated encephalomyelitis. Treatment with prednisolone at first obviated the fever and after a month brought about a complete hemiplegia cure. Following the status of the patient after three months, his MRI clearly showed considerable reduction in lesions.

Discussion: There is possible occurrence of ADEM following pneumococcal meningitis. Regarding the occurrence of neurological symptoms such as visual disturbance, hemiparesis or hemiplegia following bacterial meningitis, ADEM can be considered as one of the differential diagnoses to be accompanied by MRI. Acute disseminated encephalomyelitis should be treated using suitable dose of corticosteroids.

Keywords: Encephalomyelitis; Inflammatory; Demyelinating Autoimmune Disorders CNS; Meningitis; *Streptococcus pneumoniae*

1. Introduction

Acute disseminated encephalomyelitis (ADEM) is a rare disease, which follows variousviral infections such as measles, rubella, chickenpox and mumps, and in rare cases it follows vaccinations including measles, polio, Japanese encephalitis, tetanus toxoid, influenza and hepatitis B. In some recent studies, this disease has been reported to occur after some viral infections such as influenza A and B, hepatitis viruses, as well as non-viral agents such as *Mycoplasma*, *Salmonella typhi*, *Leptospirosis*, *Legionella*, *Borrelia* and *Rickettsia*. A history of infection or vaccination is found in about two thirds of pediatric cases and half of adult cases. This disease appears to be acute and is accompanied by neurological symptoms, fever and loss of consciousness (1). Differential diagnosis includes acute attack of multiple sclerosis and encephalitis. Prognosis of these patients is dependent on the etiologic agent, and the worst prognosis follows measles. Treatment of ADEM with a high dose of corticosteroid leads to improvement of neurological complaint after one to six months of initiation (2). Patients that do not respond to corticosteroid can respond positively to IVIG and plasmapheresis. In the absence of proper treatment, mortality from ADEM is about 30% (3). This is a case report on a patient with ADEM, which is an unusual complication of pneumococcal meningitis.

2. Case Presentation

The patient was a 27 year-old man who was taken to the emergency ward of the hospital due to fever and loss of consciousness. The patient suffered from cold, mild fever and neck pain for the three previous days. In the physical examination of the patient the consciousness level was at stupor and the patient's vital signs were: temperature, 38.8°C; respiratory rate, 30 breaths/minute; pulse rate, 102 beats/minute; blood pressure, 110/70 mm Hg; and neurological examinations showed neck rigidity and positive brudzinski's sign, right pupil dilation with response to light, lack of movement responses of upper extremities to painful irritations, generalized areflexia, and bilateral extensor of plantar reflex. After hospitalization, diagnostic procedures and treatment were performed with the primary diagnosis of bacterial meningitis. Treatment

was immediately started with vancomycin 1g, twice daily, ceftriaxone 2g, twice daily, and dexamethasone; however, lumbar puncture was performed after brain computer tomography (CT). Generalized edema along with bilateral temporal horns was observed in the CT scan. Cerebrospinal fluid (CSF) had a completely cloudy appearance and the results of CSF analysis and other examinations were as follows: in complete blood count (CBC); white blood cells (WBC): 14200 cells/mm^3; polymorph nuclear (PMN): 90%; hemoglobin (Hb): 14; platelet (PLT): 122000 (cells/mm^3); blood sugar (BS): 142 mg/dL; CSF: WBC: 610 cells/mm^3; PMN: 60%, red blood cells (RBC): 50 cells/mm^3; glucose: 0; protein: 350 mg/dL; and many gram-positive diplococcic were seen by Gram stain of CSF fluid. Due to the poor general condition of the patient, the patient was transferred to the intensive care unit (ICU) under respiratory support. After 48 hours, *Streptococcus pneumoniae* was detected in the culture of blood and CSF samples. During the fourth day of treatment, the general condition and consciousness of the patient was somewhat improved and his pupils' size became normal. On the eighth day of treatment, the patient became fully conscious and was disconnected from the ventilator due to good respiratory condition, but continued to have fever and showed no remarkable improvement in the movement of right extremities (with the exception of slight movement of fingers). To investigate the cause of the continued fever and hemiparesis, examinations were carried out to check the probable complication or improper treatment of meningitis, existence of brain spacious lesion and the possibility of conditional disease such as collagen vascular disease. The results of the blood, urine and tracheal cultures were negative, and all of the collagen vascular tests (RF, ANA, C-ANCA, P-ANCA, ds DNA, C_3, C_4, CH_{50}…) were normal. Renewed lumbar puncture and CSF analysis showed proper treatment response.

The results of examinations at this point were as follows; WBC: 210 cells/mm^3 by 60% PMN; RBC: 260 cells/mm^3; protein: 39 mg/dL; glucose: 38 mg/dL; and smear and culture were negative. For further investigation, electroencephalograph (EEG) and MRI were carried out. The slowness of diffuse along with theta and delta waves were observed in the EEG. Bilateral plague-like lesions as well as hyper-signals were observed in T2 images from the MRI, with and without contrast enhancement. These lesions were often observed in symmetric patterns in subcortical, periventricular, and pons regions; some were with contrast enhancement (Figure 1). Given the possibility of multiple sclerosis, CSF was studied for oligoclonal bands, and the result was negative.

Based on the findings of the brain MRI and diagnosis of ADEM, the patient was given prednisolone 60 mg/day. The fever diminished after four days and there was strength improvement in the right upper and lower extremities. After nine days of high dose corticosteroid therapy, the strength and muscle tone of the extremities reached four-fifths of its normal level, so that the patient could

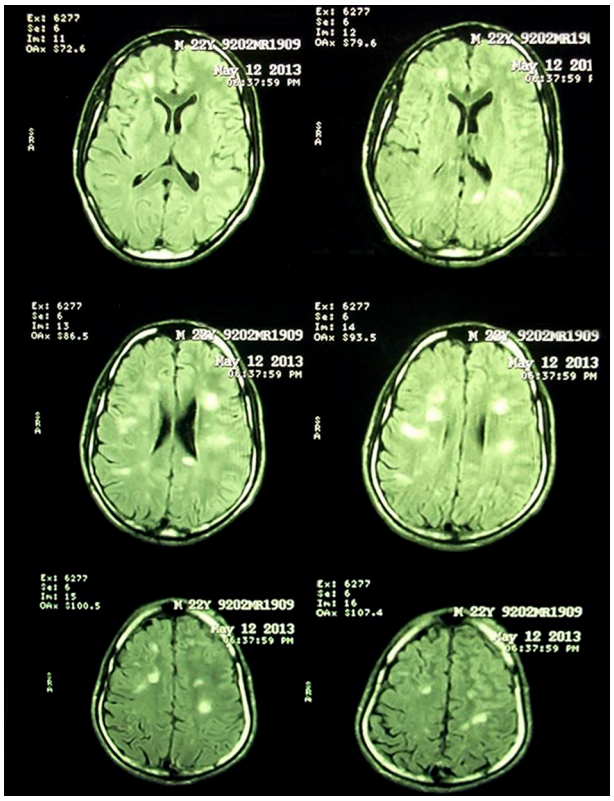

Figure 1. Brain MRI After ten Days of Admit. Bilateral Plague-Like Lesions Were Observed

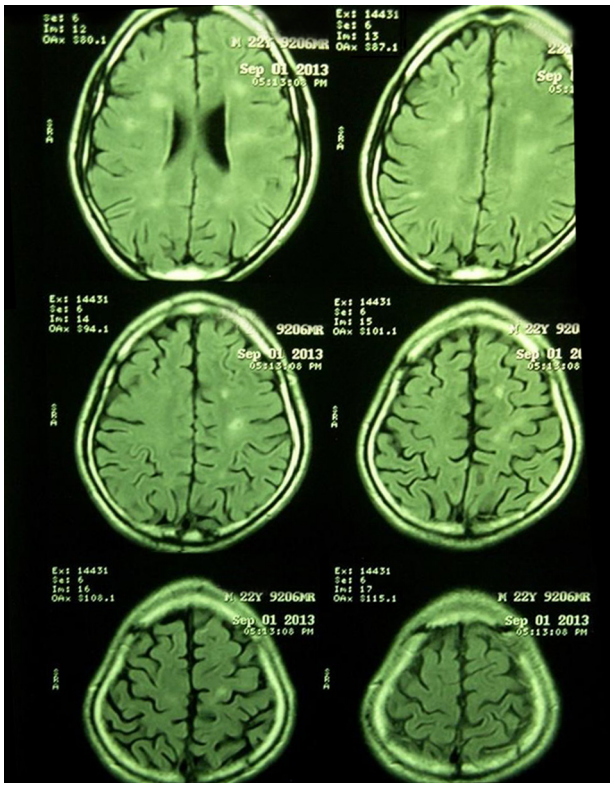

Figure 2. Brain MRI After Three Months of Follow up. The Lesions Had Been Reduced Remarkably

walk without any aid. Finally, the patient was discharged with oral prednisolone alone and without any antibiotics. Prednisolone was tapered within one month and his paresis fully recovered within the three months of follow up. In the last MRI, the lesions had been reduced remarkably and there was no uptake of contrast material, which confirmed the diagnosis of ADEM (Figure 2).

3. Discussion

ADEM is a single episodic inflammatory demyelination disease, which results in rapid focal or multifocal neurological disorders. The disease mostly occurs following vaccination or systemic viral infection, and in a few cases, by bacterial infections including pneumococcus (1-3). The management of ADEM is based on the treatment of the primary disease and a high dose of corticosteroid. Patients will achieve full recovery if proper treatment is provided. However, in the absence of proper treatment, mortality rate can be as high as 30% (2, 3). In 2004, Ohnishi et al. reported a 39 year-old man, hospitalized with the diagnosis of pneumococcal meningitis and sepsis, whose consciousness level worsened despite four days of treatment. An MRI was taken and ADEM was suspected, and he was treated with corticosteroid and discharged under good conditions after 40 days (4). In 2009, Ueda et al. introduced a 58 year-old man who underwent treatment after having been diagnosed with pneumococcal meningoencephalitis. Eleven days after treatment, the patient developed left hand and ipsilateral facial nerve plegia. After MRI and CSF analysis, the patient was diagnosed with ADEM and treated with a steroid pulse. He recovered 40 days after the treatment initiation (5). In 2012, Thomas Williams presented a 50 year-old woman, who had referred to their hospital with complaints of acute myelopathy, three days after complete pneumococcal meningitis treatment, and discharged from hospital. After thorough examination, the patient was diagnosed with ADEM and recovered after corticosteroid treatment (6). In 2013, Yu et al. introduced a four year-old girl who suffered from loss of consciousness. The patient was hospitalized and underwent suitable antibiotics having been diagnosed with pneumococcal meningoencephalitis. After eight days, patient's CSF became normal but fever and loss of consciousness did not improve. After brain MRI and investigation of CSF, the patient was diagnosed with ADEM and underwent high dose methylprednisolone and intravenous immunoglobulin; the outcome was satisfactory (7). The disease has a variety of symptoms and signs such as consciousness deficit ranging from drowsiness to coma, meningeal irritation signs, fever, generalized reflex reduction and extensor of plantar reflex, pyramidal involvement, acute hemiparesis, cerebellar ataxia and cranial neuropathy including optic neuritis and spinal cord dysfunction (transverse myelitis) (2, 3). The disease initiates rapidly and progresses in a few days. Brain MRI, the most useful imaging tool, demonstrates multifocal lesions in the early stages, which are undistinguishable from multiple sclerosis lesions. These lesions recover-fully or partially after treatment, and new lesions will not be created, which is damaging for multiple sclerosis (MS). In Brain MRI, most of these lesions are seen as contrast enhancement at the acute stage and sometimes remain permanent. The existence of fever at the onset of disease, recent antecedent of vaccination or viral infection and different levels of consciousness deficit, seen with ADEM, help differentiate it from MS. In patients with lumbar puncture and CSF examination, pressure is usually above normal, WBC is from low to medium, protein is increased, sugar becomes lowered or normal, oligoclonal bands can be less than MS and IgG does not increase. The CSF changes mentioned in these patients are likely to be due to the fact that the disease has a viral or bacterial origin (2, 3). All the evidence provided by MRI for ADEM was observed in our patient, however the increase in cells was greater than medium and sugar was very low in CSF, which can be due to the bacterial factor. Regarding the case in the present study and other similar case reports, ADEM can be seen following bacterial infections and pneumococcal meningitis. If neurological signs such as vision disorder, hemiplegia, or hemiparesis are observed following bacterial meningitis, ADEM should be considered. Moreover, after confirmation of ADEM, treatment with corticosteroid should be implemented.

Authors' Contributions

All authors contributed in study concept and design, drafting of the manuscript and critical revision of the manuscript for important intellectual content.

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