

Research Paper

Medical Science

Acute Disseminated Encephalomyelitis A Manifestation of Parenteral Placentrex; A Rare case Report

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ABSTRACT

A 27year old female patient with acute disseminated encephalomyelitis after repeated injection of placentrex who was treated for infertility is described. There was no evidence of prior infection or vaccination. The patient recovered rapidly after treatment with methylprednisolone. Acute disseminated encephalomyelitis should be considered a rare complication of parenteral therapy with placentrex.

KEYWORDS: placentrex, ADEM, Infertility

INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is an inflammatory demyelinating disease of the CNS that often follows a viral illness or vaccination. It is clinically characterized by the acute or sub acute onset of multifocal neurologic disturbances that

typically follow a monophasic course. [3]. Nevertheless relapses have been reported making it difficult to distinguish from Multiple sclerosis. ADEM affects predominantly children and young adults. Although the pathophysiology of ADEM is largely unknown, an autoimmune response to myelin basic protein triggered by infection or immunization is strongly suspected to be the main etiological factor [1] the disease typically occurs after infections or vaccinations [4]. However, in many patients with ADEM no evidence of prior infection or vaccination can be found. We report one patient with ADEM after repeated injection of placentrex. This case report of successful recovery from fulminant ADEM following early treatment with pulse intravenous high-dose methylprednisolone, suggests that this agent should be studied in other cases.

CASE REPORT

A 27 yr old female married for last 5 yr without any issue was treated for infertility with injection placentrex. Patient received injection placentrex for 5 days. The next day after the last dose she developed slurring of speech and difficulty in walking. Her speech became unintelligible and uncleared. However her comprehension was intact. On the day of initiation of symptoms she developed difficulty in walking. She walked with dragging of left foot. Twelve hours later she became disoriented and stopped recognizing family members. There was no history of fever, vomiting, headache & convulsion. Past and family history was not remarkable except history of infertility.

At the time of hospitalization the patient's body temperature was 98.6°F, her pulse was 78 beats per min and regular, and her blood pressure was 110/70 mm Hg. General physical assessment found no abnormality. The patient was irritable, opened her eyes spontaneously but did not obey commands and only produced unintelligible words. Her pupils were equal and reactive to light. Corneal reflexes and oculocephalic responses were normal. The Patient was moving her all limbs with normal muscle tone, with brisk tendon reflexes, and bilateral Babinski signs.

Patient was subjected for neuroimaging (3Tesla MRI). MRI brain (T2 weighted) showed multiple patchy discrete white matter hyperintensity lesions in both cerebral hemisphere and were enhancing

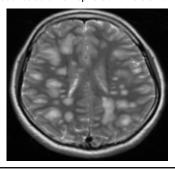


Figure- 1 T2 weighted axial MRI (3 Tesla) sequences of brain showed multiple confluent white matter hyper intensity in both cerebral hemi-

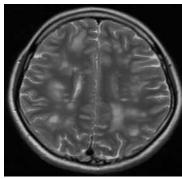


Figure- 2 Repeat MRI of brain after one week showed regression of the lesions in T2 weighted images.

with gadolinium contrast (Figure 1). MR spectroscopy revealed no abnormality. The result from magnetic-resonance venography was normal. A chest radiograph showed no abnormalities. Antibody tests for HIV, cysticercoids, and toxoplasma were negative. Patient was evaluated for syphilis, tuberculosis, hepatitis. However infective pathology could not be proven. An MRI of the cervical and thoracic spine showed no abnormality. CSF study, Visual evoked potential, Brain auditory evoked response, was non contributory. The results of blood, chemical, and hematological tests were normal.

Patient was treated with intravenous injection methyl prednisolone 1 gram for 5 days. The next day she was clinically stable and started symptomatic recovery. After another two days the patient became anxious and agitated, and complaining of headache and was given acetazolamide 250 mg three times daily, analgesics and zolpidem 5 mg at bed time. The patient clinically recovered completely during the next seven days. Patient was reviewed with MRI of brain. Repeat MRI of brain after one week showed regression of the lesions in T2 weighted images (Figure 2).

The clinical features of this case, along with the extensive white matter changes on cranial MRI without evidence of active brain infection, were consistent with the diagnosis of ADEM. The disease is mostly monophasic, with the highest mortality rate during the first week of the illness [3]. Prolonged disturbances in consciousness have a poor prognosis and the level of consciousness correlates most closely with the overall morbidity and mortality. The diagnosis of ADEM is often made by exclusion and there may be an important delay between onset of neurologic symptoms and initiation of effective therapy. Very few drugs like gold [1], infliximab [5] and some herbal products [2] may cause ADEM. However to our knowledge, no case has been reported about placentrex a cause for ADEM. As injection placentrex is used for the purpose of pelvic inflammatory disease, sub mucosal fibrosis and sometimes in treatment for infertility, this serious side effect should be a future consideration. There was a close temporal relation between parenteral

treatment and onset of neurological symptoms, which is compatible with the time interval of a few days up to 4 weeks usually found in patients with ADEM. Similar to immunizations, the placental extracts were given repeatedly, which may have boosted the immune response.

Placental extracts contain potentially immunogenic proteins, glycoproteins, and phospholipids. All these ingredients are able to provoke a response in the immune system, ranging from fever to classic anaphylactic allergic reactions as well as delayed immunological cellular reactions such as vasculitis. Potential cross reactivity between placental extracts and brain protein could be a possible mechanism for inducing demyelination. Possible interactions between different placental extracts have only rarely been subjected to scientific evaluation.

CONCLUSION

To conclude, our case of ADEM as a rare adverse event after parenteral therapy with placental extracts. However it is exquisitely steroid responsive. It should be more scientifically evaluated for safety and efficacy of parenteral placentrex. These rare adverse effects to be kept in mind while using this medication by physician as well as obstetrician. Post marketing surveillance studies for detecting safety problems should be mandatory just as for any other drug.

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