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Case Study

CEREBROPROTEIN HYDROLYSATE INDUCED SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS): A CASE REPORT

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ABSTRACT

Cerebroprotein hydrolysate is a newer pharmacological neurotropic agent and considered as a promising therapeutic agent for dementia, Alzheimer's disease, traumatic brain injury and acute ischaemic stroke. Studies revealed that most of the side effects are minor. Here, we reported a case of Systemic inflammatory response syndrome (SIRS) probably due to use of Cerebroprotein hydrolysate in a patient with acute ischaemic stroke.

Keywords: Cerebroprotein hydrolysate, Dementia, ischaemic stroke, Systemic inflammatory response syndrome

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INTRODUCTION

Cerebroprotein hydrolysate is a newer pharmacological agent which is reported to be neurotropic in nature and manufactured synthetically by the standardised enzymatic breakdown of lipid-free porcine brain proteins. It has been reported that cerebroprotein via certain mechanism enhances neurogenesis, neuronal survival and neuronal plasticity and also has neuro immunotropic mechanism of action [1]. By improving the metabolism of neuron and protecting nerve damage studies have shown cerebroprotein hydrolysate to be useful in vascular dementia, Alzheimer's disease, traumatic brain injury, acute ischaemic stroke and extrapontine myelin lysis [2-4]. Cerebroprotein hydrolysate augmented proliferation, differentiation and migration of adult subventricular zone (SVZ) neural progenitor cells resulting in increased number of neural progenitor cells and neuroblasts to contribute to neurogenesis. This may be the mechanism for beneficial effect in acute ischaemic stroke and traumatic brain injury [5]. Studies have revealed that most of the side effects are minor. Most commonly headache, nausea, vertigo, increased sweating, agitation, fever, flu-like syndrome, hallucination, confusion, etc have been reported. It is contraindicated in hypersensitivity, epilepsy and severe renal impairment and used with caution in pregnancy and lactation [6].

CASE REPORT

A 52 y old female patient, known hypertensive and diabetic was hospitalised for the sudden weakness of the right side of the body for last 2 d and was diagnosed to be an acute ischaemic stroke. Plain MRI of the brain revealed acute ischemic infarct in (L) corona radiate with the affection of left frontal lobe white matter. All other routine investigations including routine blood, blood biochemistry, routine urine,chest x-ray, ECG, USG abdomen are within normal range. She was clinically stable without having any evidence of infection. She was administered with standard medications (injection mannitol, pantoprazole, piracetam, methylcobalamin, insulin and telmisartan). Next day injection Cerebroprotein hydrolysate 60 mg intravenously once daily was added. On 2nd day patient was getting better with stable vitals. Her blood pressure was around 160-170 mmHg systolic and 90–100 mmHg diastolic. After 2nd dose of cerebroprotein hydrolysate patient suddenly developed a fever with chill and rigor and hypotension (systolic BP lowered to 80 mmHg). Injection Cerebroprotein hydrolysate and all other relevant medications were immediately stopped and the patient was managed in ICUwith IV fluids, inotropes and dexamethasone injection and antibiotic-pipericillin tazobactam. On the day of admission WBC count was 8800/cumm and ESR 35 mmAEFH; after a patient had developed fever and hypotension WBC count raised to 28100/cumm and ESR 95 mmAEFH, CRP 123.2 mg/l, Procalcitonin level was 91.09 ng/ml. Following treatment of shock on the very 2nd-day patient was absolutely normal with normal vitals. BP raised up to baseline value and laboratory parameters returned to normal values (WBC count–5500/cumm, ESR 35 mmAEFH and CRP 27.1 mg/l)

DISCUSSION

Neurodegenerative disorders are one of the leading causes of death and disability in both developed and developing countries. Many neurotrophic drugs are developed and used. Cerebroprotein hydrolysate is the latest neurotrophic drug launched. Its superiority over other neurotrophic agents is because of its different mode of action which helps in faster and more complete nerve repair and growth. It consists of short biological peptides which act like endogenous neurotrophic factors. It is given in a dose of 60–180 mg once daily for 10–20 d by slow intravenous infusion in 250 ml saline over 60–120 min. Neurotrophic activity can be detected up to 24 h* after a single injection [7].

Several studies have revealed minor adverse effects. Guo Wei Yan *et al.* confirmed allergic manifestation with the use of preproprotein hydrolysate injection [8]. Our patient developed sudden symptoms of SIRS after 2 d of drug administration which immediately improved after discontinuation of the drug and proper management. Dramatic development of shock and dramatic improvement after discontinuation of the drug within 48 h* made us think that it might be due to the adverse reaction of cerebroprotein hydrolysate.

The casualty assessment by World Health Organisation Uppsala Monitoring Center scale [9] and Naranjo's algorithm [10] score 6) showed a probable relationship between the drug and the reaction.

Though rare, clinicians must be aware of such reaction in order to ensure timely diagnosis and treatment. Cerebroprotein hydrolysate is still in its nascent stage and will need rigorous randomised controlled trials before its efficacy and safety is well established.

CONFLICT OF INTERESTS

Declared none

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