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

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ABSTRACT
Cerebroprotein hydrolysate is a newer pharmacological neurotropic 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].

INTRODUCTION
Cerebroprotein hydrolysate is a newer pharmacological neurotropic 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 (insulin and telmisartan). N ext day injection Cerebroprotein hydrolysate 60 mg intravenously once daily was added. On 2nd day patient was absolutely normal with normal vitals. BP raised up to 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 mm Hg). Injection Cerebroprotein hydrolysate and all other relevant medications were immediately stopped and the patient was managed in ICU with IV fluids, inotropes and dexamethasone injection and antibiotic-piperlicillin-tazoctactum. On the day of admission WBC count was 9800/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 preporeprotein 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

CONFLICT OF INTERESTS
Declared none

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