

Case Report on Lambert-Eaton Myasthenic Syndrome Secondary to Snake Bite (Cobra)

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ABSTRACT

BACKGROUND: Lambert-Eaton Myasthenic Syndrome (LEMS) is a rare presynaptic disorder of neuromuscular transmission in which quantel release of acetylcholine is impaired by autoantibodies for voltage gated calcium channels(VGCC).

CASE REPORT: this case report describes about a 35 year old Indian male presents a history of snake bite(Cobra) two weeks back.His clinical findings and observations later diagnosed to Lambert-Eaton Myasthenic Syndrome (LEMS) secondary to snake bite(Cobra).

CONCLUSION: LEMS secondary to snake bite(Cobra)can be managed effectively withT.Pyridostigmine. Early stage of diagnosis can prevent further complications.

KEY WORDS:LEMS, Snakebite(Cobra),VGCC, T.Pyridostigmine.

I. INTRODUCTION

Lambert-Eaton Myasthenic Syndrome (LEMS) is a rare presynaptic disorder of neuromuscular transmission in which quantel release of acetylcholine is impaired hv autoantibodies for voltage gated calcium channels(VGCC)^[1].Itsmost often associated with small cell lung cancer, although idiopathic presentations comprise approximately 40% of the cases^[2].It causes unique set of clinical characteristics ,which include proximal muscle weakness, depressed tendon reflexes, post tetanic potentiation and autonomic changes^[3].

In cases with a compatible clinical constellation, the diagnosis is confirmed with electrophysiological and serological tests^[4]. The management for the LEMS include Cholinesterase inhibitors :Pyridostigmine, Pottasium channel blockers : 3,4-diaminopyridine, Amifampridine, Immune therapy : Prednisone, Azathioprine, Plasma exchange and Intravenous Immunoglobulin^[5].

Here we present the case of Lambert-Eaton Myasthenic Syndrome (LEMS) secondary to snake bite(Cobra). This report serves to highlight the role of healthcare practitioners in diagnosing and starting its early treatment.

II. CASE REPORT

A 35 year old Indian male presents a history of snake bite (cobra) two weeks ago and had taken treatment from nearby hospital. He has hypertension since one year and is not on any treatment. He was admitted to the neurology department with complaints of slurring of speech for one week. On evaluation he was observed with dysarthria, non fluent aphasia and a blood pressure of 140/90mmhg.TheMRI of the brain with angiogram was taken which shows chronic small ischemic vessel changes. Hematological investigations were performed and he was found to be polycythemic with Hb count of 16.5 gm%. Blood pressure was monitored during the hospital stay and there was an accelerated hypertension.

Table 1. laboratory investigations during the time of admission		
Parameters	Patient Value	Normal Value
TSH(uIU/ml)	0.88	0.35-4.94
Hb(gm%)	16.5	12.5-18
TLC(cells/cumm)	8100	4000-10000
Neutrophils (%)	64	40-75
Lymphocytes(%)	30	20-50

Table 1: laboratory investigations during the time of admission

| Impact Factor value 7.429 | ISO 9001: 2008 Certified Journal Page 70



Eosinophils(%)	03	1-6
Monocytes(%)	03	2-10
ESR(mm/hr)	26	1-20
PCV(%)	48.9	40-54
Platelets(lakhs/cumm)	2.65	1-4.5
Uric Acid(mg/dl)	4.2	3.5-7

In view of voice change, absent deep tendon reflexes, proximal muscle weakness and autonomic symptoms patient was suspected to have Lambert-Eaton Myasthenic Syndrome secondary to Cobra bite. With the prompt diagnosis, the treatment was initiated with antihypertensives such as T.Losartan 50mg, T.Nebivolol Hydrochloride 5mg, T.Chlorthalidone 12.5mg, T.Cilnidipine 10mg and to avoid the platelet aggregation combination of T.Aspirin and T.Atorvastatin 75/10mg was prescribed. To prevent anxiety and depression the patient was given combination of T.Clonazepam 0.5mg and T.Escitalopram 5mg.Based on the MRI report, to treat the multiple givencombination infarction he was of T.Alphaglycerylphosphorylcholine 250mg and T.Piracetam 800mg along with it syrup of Citicoline 500mg/ml and Piracetam 400mg/5ml was administered. Finally, the symptoms of LEMS likedysarthria, non fluentaphasia was treated with T.Pyridostigmine 60mg.

III. DISCUSSION

This case report has ruled out that, early diagnosis and further management of LEMS secondary to snake bite (cobra) helps to prevent complications to a certain extent. In LEMS, antibodies against VGCC decrease the amount of calcium that can enter the nerve ending, hence less Ach can be released from the neuromuscular junction.Cobra bite usually produces post synaptic neurotoxicity, venom binds to the Ach receptors preventing the interaction between Ach and receptors in the post synaptic membrane. LEMS is a presynaptic disorder in which there is decreased Ach release and the blockade thereafter, can be reversed by Acetyl choline esterase inhibitors such as Edrophonium, Neostigmine and Pyridostigmine which act by increasing the available Ach at the synaptic cleft^[6]. The bulbar symptoms including the dysathria and dysphagia was seen in patients with LEMS^[7].Here the envenomation was mild to cause only bulbar symptoms and the patient got better on treatment with T.Pyridostigmine.

IV. CONCLUSION

To conclude, we report a case of LEMS secondary to snake bite(Cobra) that is successfully treated with T.Pyridostigmine. Our study shows that early diagnosis and proper management of LEMS results in improvement in the condition of the patient by preventing the complicatons of LEMS like trouble breathing and swallowing, infections, injuries due to falling or problems with coordination when compared to those left untreated.

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