Ramsay Hunt syndrome (RMS) is the herpes zoster infection of the geniculate ganglion of seventh cranial nerve. This is the common form. We saw a case who presented with cranial polyneuropathy. A forty-four-year-old male named AK presented with pain and vesicular eruption in the left mastoid region and over the left pinna. Two days after this he felt difficulty in swallowing food and water and simultaneously noticed facial asymmetry. He also felt vertigo and tinnitus. There was no diplopia, ptosis or facial pain, headache, vomiting, convulsions, or loss of consciousness. There is no history of weakness of limbs or bladder and bowel involvement. He was not a diabetic or a known case of tuberculosis. There was no family history of similar problems. His general examination did not reveal any abnormality. His higher functions were normal. However he had lower motor neurone (LMN) type of facial nerve lesion on the left side. There were also LMN type of ninth and tenth cranial nerve on the same side. He had neural type of deafness on the left side. No other cranial nerves were affected and he had no motor or sensory deficit. There were no cerebellar signs or signs of meningeal irritation. Skull and spine were normal. CSF study did not reveal any abnormality. CT scan of the brain was also normal. Routine haematological tests, blood glucose and X-ray chest were normal.

As there were no evidences of any other cause of multiple cranial nerve lesions in this case like basal meningitis, infiltration of the base of the brain by malignancy or evidence of brain stem vascular or neoplastic lesion; it was diagnosed as a case of multiple cranial nerve involvement (polyneuritis cranialis) due to Vericella zoster, variant of R H Syndrome. The case was treated with Acyclovir 750mg every 8hourly by intravenous route for 10days. The patient improved remarkably but continued to have impaired hearing after three months of follow up.

Our case was a case of Vericella zoster involvement of facial nerve as the illness started with facial weakness with vesicular eruption over pinna and mastoid area. This case fits into the diagnosis of Ramsay Hunt syndrome. This syndrome is more commonly seen in 5th and 6th decade as happened to our case. However, this case had added features to the classical description of RMS. There were ipsilateral other cranial nerve lesions like 8th, 9th and 10th cranial nerves. Few others have also noticed other cranial nerve involvement. The other cranial nerves that are affected are VIII, IX, V, and X in that order. Facial numbness, tongue numbness, corneal anaesthesia may be associated with R H Syndrome. But other cranial nerve involvement is quite rare. Most of the patients recover to some extent, but it may be incomplete. Our case had almost complete recovery except some hearing impairment on the affected ear after three months of follow up.

**REFERENCES**


**Hypoglycemia and Catatonia**

Sir,

Middle aged male with history of diabetes mellitus on oral hypoglycemic presented with acute onset of catatonia. Investigation revealed low blood glucose level. He responded promptly to intravenous dextrose.

Catatonia is a state of apparent unresponsiveness to external stimuli in a person who is apparently awake. Other than primary psychiatric disorder (e.g. Schizophrenia, substance abuse or mood disorders), various metabolic and neurological disorder also may present with catatonic symptoms.

Fourty two years defence civilian was brought by his friends to the causality of Naval Hospital at Port Blair on 22 June 2006, with history of not talking and not performing his routine works since morning. There was no history of convulsion, loss of consciousness or fall. The individual is staying with his friends in a rented house leaving his family behind at Kerala. According to one of his friends he was taking some Ayurvedic or allopathic medicines prescribed by a medical practitioner at Kerala for diabetes. They denied any history of abnormal behavior in the past or history substance abuse.

On examination, he was awake but mute, sitting on the bed. Temp. 98.4 F. Pulse – 84/min, regular. BP – 116/84 mmHg. There was no pallor, icterus or lymphadenopathy. He was spontaneously moving all 4 limbs but not following any verbal command. Resistance to passive movements was present in all muscle groups. DTJ – Normal. Plantars were flexor. There was no abnormal posture or movement. Meningeal signs were negative. Examination of cardiovascular system, respiratory system and abdomen were essentially normal.

Random capillary blood glucose was 36mg/dl by glucometer. Venous blood glucose – 40 mg/dl. Hemogram/Urine analysis/LFT – WNLECG – WNL. 50 ml of 25% Dextrose was administered. He showed prompt response, started talking and following command. The individual revealed that he was on Glibenclamide 5 mg BD. He was not feeling well in the previous day and did not take his meal properly.

He was admitted in the ward for hypoglycemia
monitoring. There was no recurrence of hypoglycemia. He was discharged after 48 hours with an advice to come for follow in medical OPD after a week.

Extreme negativism and mutism in this patient fulfills the American Psychiatric Association diagnostic criteria for catatonia in the absence of primary psychiatric disorder or substance abuse. Catatonia in the presence of low blood glucose level and its prompt response to intravenous dextrose (Whipple’s triad) convincingly document the relationship between hypoglycemia and catatonia in this patient. Neuroglycopenic symptoms in the form of behavioral abnormality, confusion, altered sensorium, seizure or coma is well known manifestations of hypoglycemia. But association between catatonia and hypoglycemia could not be found after extensive search in the internet using the key words Catatonia, Hypoglycemia and Neuropsychiatric manifestations in various combinations. This case emphasizes that blood glucose should routinely be tested in all patients presenting with catatonia.

Surg Lt Cdr S Ghosh
Graded Specialist (Medicine), INHS Dhanvantari, Port Blair, A and N Islands.
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