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Case Report

Ramsay hunt syndrome: a case report

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ABSTRACT

Ramsay hunt syndrome (RHS) is a neurological condition comprising of complications with *varicella zoster* virus (VZV) which results in inflammation of geniculate ganglion. In the days to weeks following zoster, various cranial nerve palsies can develop because the varicella-zoster virus is latent in the cranial nerve ganglia. The trigeminal and facial nerves were the most commonly affected by zoster, based on the cranial nerve involvement. Another common impacted nerve was the vestibulocochlear nerve. As VZV is exclusively human pathogen, research on VZV latency is limited to only human ganglia examination. This case report describes about Ramsay Hunt Syndrome induced or also called as Herpes Zoster Oticus caused by *Varicella zoster* virus. Diagnosis mainly involved laboratory investigations, patient history, treatment data and other relevant information co-related from various literature. A 46-year-old female patient was diagnosed Ramsay hunt syndrome on the basis of clinical findings as right facial palsy, blisters over right pinna, otalgia and right ear erythematous lesions. Patient also exhibited deviation of angle of mouth to left side and unable to close right eye. This was accompanied by elevated erythrocyte sedimentation rate (ESR) exhibiting acute inflammation. Hemoglobin was low and total leukocyte count was high. Following that, corticosteroids and antivirals were prescribed to assist her medical condition. Remission is slow for RHS, hence patient was also advised for rehabilitation therapy to improvise her lower motor neuron facial palsy. Recurrence is possible in long term so it is imperative to initiate early treatment within 72 hours as soon as the symptoms are observed for better patient care.

Keywords: Ramsay hunt syndrome, *Varicella zoster* virus, Corticosteroids, Anti-viral

INTRODUCTION

Herpes zoster oticus (HZO) is an infectious disease exhibiting clinical feature of erythematous vesicular eruptions or rashes in the pinna and external auditory canal along with otalgia. If accompanied by ipsilateral facial nerve paralysis, it is called Ramsay Hunt syndrome (RHS).[1] It occurs by reactivation of *Varicella-Zoster* virus (VZV) which remains dormant in the sensory root ganglia of the facial nerve and can cause ipsilateral lower motor neuron facial nerve paralysis.[3] The risk for reactivation of latent VZV is due to a persons decreased cell mediated immunity.[2] 2% and 10% of all cases of acute peripheral facial nerve palsy are caused by RHS. Diagnosis is based on patient history, physical and clinical findings.[1] Some other investigational tests include, direct Immunofluorescence to detect VZV antigen having a sensitivity of 90% and specificity of 99%. House-Brackmann's grading scale to assess severity of facial nerve paralysis. Audiometry to identify hearing loss by pure tone. Facial nerve electroneuronography (ENoG) an electrophysiological test to measure nerve degeneration. Remission is rare. As few as 20% of patients may recover completely. Corticosteroid and antiviral therapy is recommended.[4] Antibiotics can be given for treating secondary infection to relieve pain, medications like anticonvulsants, opioids, tricyclic antidepressants and lidocaine patches. To minimize the outbreak and post-herpetic neuralgia the zoster (shingles) vaccine is effective in decreasing HZO incidence.[1] For this reason, all patients with RHS should receive an early diagnosis and therapy in order to stop

chronic facial nerve degeneration. Hence, this case report reflects on the typical clinical features of RHS and its diagnostic criteria.

CASE REPORT

A 46-year-old female patient was admitted in general medicine department of Yashoda Hospital Hyderabad with complaints of giddiness for 3 days, deviation of mouth to left side from 2 days and history of right ear discharge from one week. She also suffered multiple episodes of vomiting in the last 3 days. No social history or past medical history was noted. The laboratory diagnostic tests given in Table 1, following the diagnosis the treatment was started and medications prescribed shown in Table 2, the discharge treatment regimen prescribed was mentioned in Table 3.

Table 1: Laboratory tests.

Lab tests	Normal values	Test values
Haemoglobin		
Hb	11-16.5 g/dl	8.30g/dl ↓
PCV	35-50%	29.60% ↓
TLC	3500-10000/cumm	12930/cumm↑
ESR (female)	0-20 mm/hr	61.00 mm/hr↑
Blood gas values		
pH	7.350-7.450	7.543 ↑
pCo2	32.02-48.0 mmHg	28.3 mmHg↓
Oximetry values		
ctHb	11.4-17.5g/dl	9.1g/dl ↓
spO2	94.0-98.0%	84.8% ↓
Metabolic values		
Blood glucose	65-95 mg/dl	109 mg/dl ↑
Lactate	0.4-0.8 mmol/l	1.5 mmol/l ↑

Table 2: Medications prescribed.

S. no.	Medicationsprescribed	Dose	Freq.
1	Inj. Ceftriaxone	2 gm	BD
2	Inj. Pantoprazole	40 mg	OD
3	Inj. Thiamine+Pyridoxine	1 amp	OD
4	Inj. Paracetamol	1 gm	OD
5	T. Cinnarizine +Dimenhydrinate	20 and 40 mg	OD
6	T. Prednisolone	60 mg	OD
7	T. Valacyclovir	1000 mg	OD
8	T. Ferrous ascorbate+Folic acid+Zinc sulphate	200 mg	OD
9	Syp. Citric acid+Potassium citrate	10 mg	TID
10	Inj. Amoxycillin/Clavulanate potassium	1.2 gm	BD
11	Inj. Diclofenac	1 amp	BD
12	Acyclovir ointment	5%	TID

Table 3: Discharge medications.

S.no.	Medication prescribed	Dose	ROA	Frequency
1	T.Valacyclovir	1000 mg	PO	OD
2	T. Cefuroxime	500 mg	PO	BD
3	T.Cinnarizine and dimenhydrinate	20, 40mg	PO	OD
4	T. Nuhenz forte	1tab	PO	OD
5	Inj. Pantoprazole	40 mg	PO	OD
6	T. B-rich Q 10	1tab	PO	0D

HIV 1 and2 AG/AB (Antigen-Antibody) test: 0.06. Biological reference >1 is reactive, 0.9-1 is susceptible & retested, <0.9 is negative.

Patient demonstrated no immuno-compromised condition as HIV values was negative. Hematological analysis revealed significant findings with decreased hemoglobin (8.30g/dl), packed cell volume (29.60%), total leukocyte count (12,930/cum) suggesting a viral infection. Blood gas values show increased pH (7.543) and decreased partial carbon dioxide (28.3 mmHg). Oximetry values show decreased concentration of total hemoglobin (9.1 g/dl) and saturation of peripheral oxygen (84.8%). Metabolic values show increased Blood glucose levels (109 mg/dl) and lactate (1.5 mmol/l) observed.

The following clinical findings were noted on day-wise assessment

Day 1 observations were patient was conscious and coherent. She was suffering from right ear pain, ear-discharge accompanied by watery eyes and general weakness. Vitals were normal.

Day 2 right ear erythematous lesions along with pus discharge was identified. This involved presence of blisters over right pinna and pain. Physical examination showed deviation of angle of mouth to left side and unable to close right eye. For this Doppler Ultrasound for cardiac functioning was tested and reported to be normal. Patient observed to follow commands. Vitals normal.

The House Brackmann Scale was utilized for the clinical assessment of facial nerve function. The scale is based up on functional impairment consisting of gradings between I (normal) and VI (no movement). According to the House Brackmann scale the patient was categorised with grade IV (moderate severe dysfunction) which includes incomplete closure of eye and asymmetric mouth. Therefore, based on the sign's, symptoms and physical examination of facial characteristics the patient was diagnosed with right facial LMN (lower motor neuron)

palsy being an etiological factor for Herpes zoster oticus or RHS. The diagnosis was majorly made based on patient characteristic features and from clinical point of view.

DISCUSSION

Treatment was promptly initiated with injection ceftriaxone at a dosage of 2 gm, administered 2 times daily via IV route and indicated to treat gram negative aerobic bacteria (shingles). Injection pantoprazole at a dosage of 40 mg, IV, administered once daily, and was prescribed as a prophylactic treatment to prevent acidity caused by antibiotics. To treat low hemoglobin levels injection thiamine with pyridoxine was given. Injection paracetamol and diclofenac were helpful to treat moderate to severe pain associated with ear which the patient experienced. Tablet cinnarizine and Dimenhydrinate given to improve watery eyes and giddiness of patient complaints and given once daily.

To treat blisters formed over right pinna Prednisolone suggested in treatment. To treat herpes zoster (shingles) Tablet valacyclovir with dosage of 1000 mg orally given for once daily, it prevents nerve damage in long term. Tablet ferrous ascorbate along with folic acid and zinc sulphate in combination as a supplement to improve immunity. As high levels of lactate reported it is needed to maintain acid base balance in order to prevent metabolic acidosis, for which syrup Citric acid along with Potassium citrate prescribed. Amoxycillin/Clavulanate potassium given to treat ear infection caused by discharge and pain. Additionally, Acyclovir ointment of 5% applied topically as an add on to treat Herpes viral infection.

On day 3 symptomatic improvements seen. The ear pain decreased. Vitals normal. Patient was then advised for discharge. Patient was instructed to adhere to the medicines prescribed. Follow up appointments were scheduled to aid in RHS recovery. Patient was counseled for physiotherapy and diet. Advised to avoid cold items and exposure to cold environments, as it may aggrevate the symptoms.

Discharge medications included anti-viral valacyclovir. Tablet cefuroxime to arrest infection by gram negative aerobic bacteria causing shingles. Tablet cinnarizine and dimenhydrinate given to improve watery eyes and giddiness. To treat iron deficiencies Tablet nuhenz forte which consist of the composition of alpha lipoic acid (200 mg)+benfotiamine (200 mg)+chromium (200 mcg) +folic acid (1.5mg)+methyl cobalamin (1500 mcg)+Myo Inositol (100 mg)+pyridoxine (3mg), which was given orally for once daily. Pantoprazole as a prophylactic treatment to prevent acidity caused by antibiotic. Tablet B-rich Q 10 composition of: benfotiamine (100 mg)+methyl cobalamin (1000 mcg)+alpha lipoic acid (200 mg)+pyridoxine Hcl (3 mg)+inositol (100 mg)+folic acid (1.5 mg) given as nutritional multivitamin supplement. RHS has incidence of 5 per 1,00,000. Seen in approximately 12% of facial nerve palsy cases. It is marked by high occurrence of side effects even after therapy and poor prognosis.[5] Patient's complaint of giddiness, deviation of mouth to left side, otalgia can be similarly co-related to another case report.[6] Apart from skin lesions certain neurological disturbances like tinnitus, vomiting, vertigo was similar to this case reporting vertigo and vomiting symptoms of patient.[7]

Vesicular eruptions on conchae and external auditory meatus of left side were clinical features reported in a case which was in co-relation to this case report.[8] As the patient unable to close right eye it is indicative of facial weakness experienced as mentioned in other case report as well. The patient followed commands and was able to hear, he did not experience any hearing loss which was a known clinical feature in RHS.[6] Patient was categorized with grade IV in House Brackmann scale whereas some other case reports showed grade V symptoms.[5] However, in this case diagnosis was based mostly on clinical symptoms that applied to a small number of additional cases.[8] Antiviral drugs have been reported to exhibit a reduction in the degree of rash and the intensity of pain when given within 72 hours of onset of rash.[9] This may be connected to our case study, in which the patient showed improvement after receiving both valacyclovir and acyclovir.

Recommended treatment for RHS mainly comprises of steroids with acyclovir alongside rehabilitation programs. Rehabilitation program like electrical stimulation, infrared radiation, and facial neuromuscular exercises including auto massage, relaxation exercises are frequently suggested for the enhancement of motor function in RHS patients.[8] In the present case, the same course of treatment was observed to be delivered.

However, as patients may become resistant to acyclovir, valacyclovir, a more recent medication, was used in our instance finding the same line of treatment in another study.[8] Steroids seen to alleviate facial paralysis, reduce inflammation and brings down the House Brackmann grade to I in 52% patients.[6] However to prevent increase in severity of medical condition treatment needs to be started within 3 days of symptoms onset. In this case the medicines were initiated within 24 hours of onset of symptoms. This time period was considered appropriate to start treating RHS. Therefore, this prevented aggravation of patient condition and was able to discharge her as the symptoms were observed to be subsided. Remission of facial palsy was slow which is in accordance with other cases.[10]

CONCLUSION

A relatively rare neurological condition is RHS and is constituted by the varicella-zoster virus progressing throughout the nervous system. Misdiagnosis can happen because of the variety of its clinical presentations.

Therefore, for management to be effective, a precise diagnosis and prompt therapeutic action are essential.

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