Herpes Zoster Oticus (Ramsay-Hunt Syndrome) In Pregnancy

R Ekore, S Adeyanju, I AJAYI, A Arije

Citation

R Ekore, S Adeyanju, I AJAYI, A Arije. *Herpes Zoster Oticus (Ramsay-Hunt Syndrome) In Pregnancy*. The Internet Journal of Otorhinolaryngology. 2008 Volume 8 Number 2.

Abstract

Herpes zoster oticus is caused by varicella-zoster virus, which causes chicken pox and shingles1, 2. Shingles results from a reactivation of a latent infection by the varicella zoster virus. The cause of the reactivation is unclear but it is common in patients with neoplasm or after trauma), AIDS and other immunocompromised states3 . It is a delayed expression of the virus1,3. It is thought to be a cranial polyneuropathy4 and is said to be the second most common cause of atraumatic peripheral facial paralysis5. Here, we report a case of herpes zoster oticus (Ramsay-Hunt Syndrome) in mid-trimester of pregnancy in a young nulliparous woman, with multiple dermatomal involvement.

CASE REPORT

Mrs Y.S.A., an un-booked 28 year-old nulliparous muslim trader, presented at our hospital at a gestational age of 23 weeks and five days, with a six-day history of pain on the left side of her face, ear, neck, shoulder and upper chest, and a four-day history of rashes in the painful areas, reduced hearing in the left ear and inability to close the left eye. The pain was peppery in nature and was persistent. There was associated intermittent fever and vomiting. The patient could not ascertain a history of chicken pox infection during her childhood.

Examination revealed a young lady in painful distress with vesicular rashes extending from the left side of her face and ear to her jaw, neck, shoulder and upper chest. The rashes has crusted exudates on them (topical local herbs and concoctions had been applied) with erythematous surroundings. There was slight deviation of her mouth to the right and other features of left facial nerve palsy, and Bell's sign was positive. She was febrile, not pale, and anicteric with good hydration status. Pulse rate was 124/minute, regular and bounding. Blood pressure was 130/70 mmHg, heart sounds were normal. Respiratory rate was 20/minute and her chest was clinically clear. Her abdomen was gravid, with a symphysio-fundal height of 24 cm.

An Assessment of Herpes Zoster Oticus (Ramsay-Hunt Syndrome) in Pregnancy was made and investigations done revealed a packed cell volume (PCV) of 33%, total white blood cell count of 4,500/mm $_3$ (Neutrophils:35%, Lymphocytes: 62%, Monocytes 1%, Eosinophils: 2%), retroviral screening test was negative, malaria parasite test was positive. Obstetric scan revealed a normal live foetus at a gestational age of 25 weeks with adequate liquor and minor placenta praevia. Urinalysis revealed trace proteinuria, yeast cell: ++, leucocytes: 4-6/hpf and epithelial cells: ++

She was subsequently given i.m. tetanus toxoid injection 0.5 ml stat, and placed on Tabs prednisolone 10mg dly, tegretol 50 mg b.d., i.v. pentazocine 30 mg 6 hrly, tab dzp 10 mg b.d and topical gentian violet and fluconazole pessaries. Anti-viral drugs were not readily available. She was reviewed by an Internist and an Obstetrician while on admission, responded relatively well to the above line of management, booked for ante-natal care and was discharged home on the 13 th day on admission.

Follow-up was regular during which she complained of persistent pain which subsided gradually. A later obstetric scan revealed a normally located placenta. She eventually had spontaneous vaginal delivery of a live female neonate at term, with a birth weight of 3.6 kg. She was given anti-D (Rhogam) injection shortly after delivery.

DISCUSSION

Herpes zoster oticus (Ramsay-Hunt Syndrome) results from herpetic involvement of the facial (geniculate), vestibulocochlear or trigeminal ganglia $_6$. The dormant virus resides in sensory nerve ganglia for a variable time, until reversion of latent virus to an active and infective stage

overcomes the immune reaction. The virus then multiplies within the ganglion, with subsequent neuritis and neuralgia. Viral particles are then released into the skin via nerve endings and characteristic clusters of vesicles form. A syndrome of radicular pain without cutaneous lesions has been reported when immune mechanisms are able to recover in the middle of the process [3] . The CN VIII features are due to close proximity of the geniculate ganglion to the vestibulocochlear nerve within the bony facial canal [7] .

There is segmental pain and paraesthesia, followed within hours or days by the appearance of grouped vesicles on an erythematous base about the auricle and external auditory canal, face, mouth, neck and scalp. Sometimes, vesicles appear only in the pharynx or hard palate [3,6,8] . The rash is typically unilateral and does not cross the midline. Distribution of the rashes is dermatomal, the rashes are vesicular and the vesicle which contain varicella zoster virus heals without scar formation. Crusting occurs as the vesicles resolve [3] .

In some patients, pain may precede the rash. In immunocompromised patients, the rash may be extensive and haemorrhagic and infection may spread to internal organs [1] .

There is hearing loss, tinnitus and persistent vertigo in 20 – 30% of the patients. . Facial palsy (not considered to be Bell's) can also occur and prompt initiation of oral steroids and acyclovir should begin when this happens. This can lessen vertigo and improve recovery of facial nerve function. Hearing loss does not seem to response as well to this treatment [6] .

Other symptoms include fever, ear pain, nausea, vomiting, vertigo, tinnitus, nystagmus, hearing loss and facial paralysis [8,9] . Loss of taste in the tongue and dry mouth and eyes may also occur [8] .

Diagnosis is made on clinical grounds but confirmation may be done by culture or cytologic examination of materials obtained from the lesions. This usually reveals giant cells (large, multi-nucleated protoplasmic masses) with intranuclear inclusions with a clear halo and marginated chromatin [1] .

Treatment is mainly supportive and is the same for pregnant and non-pregnant individuals [10] . Oral acyclovir and famcyclovir in uncomplicated cases may shorten course and reduce severity. Early treatment of all patients with 7 – 10 day course of famciclovir (500 mg t.d.s) or acyclovir (800 mg five times daily, with oral prednisolone 60 mg dly for 3 – 5 days). Oral glucocorticoids are given to reduce acute symptoms and possibly reduce the incidence of post-herpetic neuralgia. Oral prednisolone given in tapering course over 3 weeks has also been shown to successfully improve outcome in most patients. Forscanet is given for acyclovir-resistant infections in immunocompromised patients. The rashes are very painful and may require the use of narcotic analgesics [1,3] . Adjunct forms of treatment include sublesional corticosteroid injections, neurosurgery occasionally for intractable pain and tricyclic antidepressants [3] .

Complications include post-herpetic neuralgia, localized hypoesthesia, secondary bacterial infections, ophthalmic zoster and disseminated zoster. Complications are worse in immune compromised patients [3] .

Prognosis is generally good and determined by how early steroid therapy is commenced as this is more likely to prevent irreversible complications [8,11] . Hearing loss may be permanent in some cases. Vertigo may last for days or weeks. Facial paralysis may be temporary or permanent [8] . Compared with Bell's palsy, patients with herpes zoster oticus often have more severe paralysis at onset and are less likely to recover completely [5] .

ACKNOWLEDGEMENT

We would like to thank the management and staff of St. Mary's Catholic General Hospital, Eleta, Ibadan, Nigeria.

References

1. Leventhal W.D., Hueston W.J. Virella G. Infectious Diseases (Viral Infections). In: Textbook of family Practice. 6th Edition. Rakel R.E. (ed). W.B. Saunders Co. Philadelphia, Pennsylvania. 2002. Pgs 358 - 359.
2. Labuguen R.A. Initial Evaluation of Vertigo. Amer Fam Phy. 2006; Vol 73(2).
3. Roaten S.P., Chaker B., Pandya A.G. Herpes Zoster (Dermatology). In: Textbook of family Practice. 6th Edition. Rakel R.E. (ed). W.B. Saunders Co. Philadelphia, Pennsylvania. 2002. Pg 1009.
4. De S., Pfleiderer A.G. An Extreme and Unusual Variant of Ramsay-Hunt Syndrome. J Laryngol Otol. 1999 Jul; 113(7): 670-1. [Medline]
5. Martinez O.A., Lahoz Z.M.T., Uroz d. H.J.J. Ramsay-Hunt Syndrome. An Med Interna. 2007 Jan; 24(1):31-4. [Medline]
6. O'Handley J.G., Tobin E, Tagge B. Ramsay-Hunt Syndrome (Otorhinolaryngology). In: Textbook of Family Practice. 6th Edition. Rakel R.E. (ed). W.B. Saunders Co. Philadelphia, Pennsylvania. 2002. Pg 434.
7. Sweeney C.J., Gilden D.H. Ramsay Hunt Syndrome. J Neurol Neurosurg Psychiatry. 2001 Aug; 71(2):149-54. [Medline]
8. National Institute of Neurological Disorders and Stroke

(NINDS) Herpes Zoster Oticus Information Page. Retrieved on 11/2/2008 from www.ninds.nih.gov/disorders/ramsay2/ramsay2.htm.
9. Ear Institute of Chicago: Medical Info. Herpes Zoster Oticus (Ramsay Hunt Syndrome). Retrieved on 11/2/2008 from www.chicagoear.com/med_info/herpes_zoster.htm.
10. Bloem C. Herpes Zoster Oticus. Emedicine (The Medscape Journal). Retrieved on 23/6/2008 from www.emedicine.com/emerg/topic250.htm.
11. Aframian D., Ben-Oliel R., Sharav Y. Ramsay Hunt Syndrome - Differential Diagnosis, Pathogenesis and Therapy. Harefuah. 1999 Feb; 136(4):278-80, 339.

Author Information

RI Ekore, FWACS
Principal Medical Officer/Consultant Family Physician, University Health Services (Jaja Clinic), University of Ibadan

SA Adeyanju, FWACP
Consultant Obstetrician/Gynaecologist, Adeoyo Maternity Hospital

IO AJAYI, FWACP, Ph.D.
Senior Lecturer/Consultant Family Physician, Dept of Epidemiology, Medical Statistics and Environmental Health,, University of Ibadan

A. Arije, FMCP
Reader, Dept. of Medicine, College of Medicine, University of Ibadan