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HERPES ZOSTER MYELITIS: REPORT OF TWO CASES

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SUMMARY

Two male patients aged 40 and 45 years with HIV infection and paraplegia are presented. The two had sub-acute onset paraplegia with a sensory level, which developed 10 days after herpes zoster dermatomal rash. They both had asymmetrically involvement of the lower limbs. Investigation including imaging of the spinal cord did not reveal any other cause of the neurological deficit. The two responded very well to treatment with acyclovir. Herpes zoster myelitis is a condition likely to rise with the upsurge of HIV infection and there is a need to identify the condition early. We also review the literature on the subject.

INTRODUCTION

Herpes zoster (HZ) is caused by varicella-zoster virus and is characterised by a vesicular dermatomal rash. It results from reactivation of latent infection in the sensory ganglion neurons after an earlier infection with chicken pox (varicella). The incidence of herpes zoster has increased since the emergence of HIV infection. It is classically a benign condition, neurological complications are common and include transient pain and paraesthesia associated with the acute rash, post herpetic neuralgia, segmental sensory loss or motor paresis, encephalitis, myelitis, and cerebrovascular occlusion(1-5). Myelopathy is a rare complication of HZ that usually develops in the immunocompromised host. In large series the incidence varies from 0 to 0.8% in the general population or in the immunocompromised patients respectively(6).

We review two cases that presented with this disorder.

CASE REPORTS

Case 1: P.I a forty-year old male known to be HIV positive presented with a herpes zoster rash on T 4 dermatome on the right side of the chest. He was started on acyclovir cream, calamine lotion and analgesics. He was not on any anti-retroviral treatment. Ten days later he presented with sudden onset of generalised convulsion, fever, confusion and inability to use both lower limbs. He also admitted to having constipation and inability to pass urine. Examination revealed a very sick patient, febrile with a temperature of 38°C and generalised lymphadenopathy. Significant neurological findings were confusion, mild neck stiffness, negative Kernigs sign and flaccid paralysis of both lower limbs. The muscle power in both lower limbs were grade 0, deep tendon reflexes were absent with a sensory level at T5. The rest of the systemic examination was normal expect for the healing herpes zoster lesion. The following investigations were performed;

EEG was basically normal. CT scan of the head showed enhancement with contrast in the meninges consistent with an inflammatory lesion, cerebrospinal fluid examination revealed raised proteins, leucocytosis but no organisms was grown. MRI of the thoracic spine showed myelitis in the T4 spinal cord region. He was catheterised and started on intravenous antibiotics, intravenous acyclovir, analgesics and physiotherapy. He made steady progress with the power in the lower limbs improving. He later on developed a pressure sore which required surgical intervention. At discharge the power grade on the right leg was 4 while grade 3 on the left.

Case 2: M. O. a 45-year old man presented with sudden onset of headache and weakness of both lower limbs. About ten days earlier he had developed herpes zoster lesion on the right chest. At admission he was noted to have a herpes zoster lesion on the T6 dermatome on the right side. He was hypertensive with a blood pressure of 180/110 mmHg. Neurological examination revealed flaccid paraplegia with absent deep tendon reflexes and a sensory level of T7. He was also noted to have bilateral sensorineural deafness. Investigations done included: radiculogram which was reported as normal, HIV antibodies were positive by the ELISA method and the haemogram was normal. He was started on intravenous acyclovir and physiotherapy. He was not on antiretroviral at any time in his illness. He made steady progress. The muscle power at discharge was grade 4 in all the limbs.

DISCUSSION

These two cases reveal the classical characteristic of herpes zoster myelitis with the usual delay between the appearance of the rash and the neurological deficit(4,7). The neurological deficit onset may vary from eight days to ten weeks after the rash with a mean of two weeks. Neurological symptoms usually begin unilaterally or if bilaterally are asymmetric with principal involvement of motor and posterior column functions altered ipsilateral to

the rash, and spinothalamic sensory function altered contralaterally(7). Spinal sensory level occurs in more than one third of patients. Usually there is a sub-acute progression of the deficit with the maximum deficit in three weeks of the initial myelopathy.

Laboratory tests are more helpful in excluding other causes rather than specifying varicella zoster. Myelography is usually normal as one of our patients demonstrated Cerebrospinal (CSF) fluid pressure is usually normal. There may be elevated protein with pleocytosis mainly monocytes and lymphocytes(3,8). The CSF glucose is usually normal.

Diagnosis of HZ myelitis is usually not difficult when neurological symptoms develop in temporal proximity to the rash. Initial differential diagnosis may include transverse myelitis, spinal cord compression or inflammatory polyneuropathy. In patients with HIV, other causes of myelopathy and radiculopathies must be considered. This will include HIV induced vacuolar myelopathy, cytomegalovirus (CMV), herpes simplex virus (HSV) myelitis or radiculopathy but the temporal relationship with the rash and localisation of the lesion often gives the diagnosis away(9). The asymmetry in the neurological presentation also helps differentiate this diagnosis from other causes of myelitis. Although no CD4 profile was done in these patients clinically they were not in advanced state of HIV disease.

The major pathological findings in herpes myelitis are posterior column abnormalities, demyelination, and necrotising inflammatory myelopathy with or without vasculitis(7-12). Demyelination is thought to occur secondary to viral infection and destruction of oligodendrocytes since Cowdry type A inclusion are usually seen in cells resembling oligodendrocytes. The pathology suggests four mechanisms of injury: direct infection and/or immune-mediated destruction of oligodendrocytes with resultant demyelination; infarction secondary to vasculitis; leptomeningitis; infection of other components including neurons, astrocytes and ependymal cells.

The virus usually spreads peripherally by axoplasmic transport and presumably exploits those mechanism that are involved in directing normal macromolecular traffic towards the periphery. Devinsky *et al*(7) speculate that central spread from the dorsal root ganglia to cord causes myelitis since most of the cases they saw had intranuclear inclusion bodies in Schwann cells and fibroblasts of posterior roots. The prolonged interval between peripheral spread (shingles)

and central spread (myelopathy) however, suggest cell to cell contact rather than intraaxonal spread. Subsequent viral spread within the cord apparently can occur concentrically within the cord, both laterally and vertically. Once extending beyond the grey matter, infection appears to preferentially involve oligodendrocytes, leading to demyelination.

Various studies have shown the benefit of treating HZ myelitis with antiviral especially acyclovir(13). It is therefore recommended that patients be promptly treated with intravenous acyclovir. The two patients reported here responded quite well to acyclovir.

The neurological outcome is usually fair. Devinsky *et al*(7) found that in 25 patients, three made total recovery, 16 partial recovery while six had little or no improvement.

With the current pandemic of HIV in the country one expects increase in cases with this syndrome.

REFERENCES

1. Portenoy R.K., Duma C. and Folry K.M. Acute herpetic and postherpetic neuralgia: clinical review and current management. *Ann. Neurol.* 1986; **20**:651-664
2. Thomas J.E. and Howard F.M. Segmental zoster paresis - a disease profile. *Neurology, Minneapolis* 1972; **22**:459-466
3. Jemsek J., Greenberg S.B., Taber L., Harvey D., Gershon A. and Couch R.B. Herpes zoster associated encephalitis: clinicopathologic report of 12 cases and review of literature. *Medicine, Baltimore* 1983; **62**:81-97.
4. Hardy M. Du zona Gazzette des Hopitaux Civil et Militaires 1876; **28**:427-447.
5. Eidelberg D., Sotrel A., Horoupian D.S., Neumann P.E., Pumarola-Sune T. and Price R.W. Thrombotic cerebral vasculopathy associated with herpes zoster. *Ann. Neurol.* 1986; **19**:7-14.
6. Ragozzino M.W., Melton L.J., Kurland L.T., Chu C.P. and Perry H.O. Population based study of herpes zoster and its sequelae. *Medicine, Baltimore* 1982; **61**:310-316.
7. Devinsky O., Cho E., Petito C.K. and Price R.W. Herpes Zoster myelitis. *Brain* 1991; **114**:1181-1196.
8. Muder R.L., Lumish R.M. and Corsello G.R. Myelopathy after herpes Zoster. *Archives of Neurology, Chicago*. 1983; **40**:445-446.
9. Price R.W. Neurological complications of HIV infection. *Lancet.* 1996; **348**:445.
10. Ruppenthal M. Changes of the central nervous system in herpes zoster. *Acta Neuropathologica, Berlin* 1980; **52**:59-68.
11. Hogan E.L. Krigman Herpes zoster myelitis: evidence for viral invasion of spinal cord. *Archives of Neurology, Chicago* 1973; **29**:309-313.
12. McAlpine D., Kuriowa Y., Tokokura Y. and Araki S. Acute demyelinating disease complicating herpes zoster. *J. Neurol. Neurosurg. Psychiat.* 1959; **22**:120-123.
13. Yasuda Y., Akiguchi I. and Kameyama M. A case of herpes zoster myelitis improved with acyclovir. *Rinsho Shinkeigaku* 1990; **30**:4452-4454.