SACRAL HERPES ZOSTER AND URINARY RETENTION

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Acute retention of urine as a presenting symptom of manifestation of herpes zoster is known, but a 35-year-old male with herpetic involvement of third posterior sacral root presented to us with acute retention of urine, followed next day with herpes zoster.

Key words : Herpes zoster, Urinary retention.

Most of the cases of herpes zoster show uneventful recovery. Sometimes such cases are complicated by secondary infection and post herpetic neuralgia. Rarely some other complications such as superficial gangrene, ocular involvement, haematogenous dissemination, myelitis, or encephalitis may also occur. The direct extension of infection from the sensory ganglia to adjacent parts of the nervous system may result in motor paralysis in 1 to 5 percent of cases.[1] From time to time, paralysis of the limb,[2,3] diaphragmatic paralysis,[4] and dysfunction of bladder,[5-7] and anus[6] have also been reported in association with herpes zoster. We herein record a patient who developed retention of the urine preceding the cutaneous manifestation of herpes zoster of the third posterior sacral root.

Case Report

A 35-year-old male was admitted for retention of urine and pain in lower abdomen of two days duration. It was associated with pain and burning sensation on the genitalia and right buttock. History of colicy pain, haematuria, dysuria, frequency, burning of micturition and abnormal bowel habits was denied. On examination, the bladder was found to be full. He was put on continuous catheter drainage by means of an in-dwelling catheter. Routine investigations and special investigations pertaining to urinary retention were within normal limits. Next day, he developed the lesions of herpes zoster along the distribution of the third sacral segment on the right buttock and right half of genitalia. The cutaneous lesions started healing on the tenth day. The catheter was removed on the twelfth day of catheterisation and the bladder function was normal thereafter.

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Comments

The parasympathetic efferent fibres to bladder come from the second, third and fourth sacral segments of spinal cord via the inferior hypogastric plexus and vesical plexus of nerves. These fibres are motor to detrusor muscle and inhibitory to sphincter vesicae. The urinary retention in this case could have been due to direct neural extension of the virus from the sensory ganglion to the parasympathetic efferent fibres of third sacral segment resulting in detrusor muscle paralysis and constriction of the sphincter vesicae.

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LETTERS TO THE EDITOR

CURE IN PEMPHIGUS–A POSSIBILITY

Recently,[1] we proposed pulse therapy with dexamethasone and cyclophosphamide for pemphigus and observed the following two advantages over the conventional treatment schedules :

(1) The hospital stay of the patients was drastically reduced to 4-5 days in a month, enabling the patient to continue with his job or other social responsibilities, and (2) the side effects of prolonged treatment with corticosteroids and/or immunosuppressive drugs, including osteoporosis, obesity, diabetes etc were virtually absent. Of the 10 patients reported earlier,[1] cases 1, 3, 4 and 9 have been lost to follow up, while the remaining 6 have been followed up for another 2 years or so, the results of which are as follows :

Case 2 had received 10 dexamethasone cyclophosphamide pulses (DCP) till February 1984, and was free from pemphigus lesions since March 1983. During the next 9 months, he continued to take only 50 mg cyclophosphamide orally and remained free of the lesions. In November 1984, he developed a mild relapse and was given DCP again. Since then, he has received 12 monthly DCP and 5 more DCP at approximately 2-month intervals. All through this period, he has been free from pemphigus lesions, till last seen in April 1986.

Case 5 had received 7 DCP till November 1983, and was free from the lesions. He continued to take 50 mg cyclophosphamide orally daily and remained free from the lesions till June 1984. Since then, the disease has got reactivated and till February 1986, he had received 17 more DCP with continued recurrences of the disease. This patient however, has been quite irregular in taking the treatment.

Case 6 had received 14 DCP till March 1984 and had been free from pemphigus lesions since December 1982. In March 1984, she had a mild relapse treated with 20 mg prednisolone a day for a few days. Till February 1985, she received another 8 DCP, but had remained free of pemphigus lesions. Subsequently, she stopped all treatment and has remained free from pemphigus lesions.

Case 7 had received 23 DCP till April 1984, and had been free from pemphigus lesions since August 1983. He continued to receive DCP till November 1984 (29 DCP) after which he continued only 50 mg cyclophosphamide a day orally. In June 1985, he stopped even oral cyclophosphamide and has still been free from pemphigus lesions.

Case 8 had received 9 DCP till April 1984 and was free from pemphigus lesions since January 1983. Till April 1985, he received 5 more DCP (total 14), after which he continued to take only 50 mg cyclophosphamide a day orally till Febraury 1986. He has not had any pemphigus lesions ever since.

Case 10 had received 20 DCP till April 1984, but was continuing to develop recurrences in between the DCP. This continued till November 1984, though the relapses were progressively milder. Between November 1984 and June 1985, he stopped treatment. DCP was started again in June 1985 and after 2 more DCP, he stopped having further lesions. Once again he stopped having further DCP, though 50 mg cyclophosphamide a day was continued. In February and March 1986, he has been given 2 more DCP, but he continues to be free from pemphigus lesions since August 1985.