HERPES ZOSTER — A CLINICAL STUDY

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Herpes Zoster or shingles is not a rare disease and is frequently seen in the out-patient department of a hospital. It is an acute infectious disease caused by a virus and characterised by unilateral, segmental inflammation of the posterior root ganglions or extramedullary ganglions of cranial nerves and by a painful vesicular eruption of the skin along the peripheral distribution of the involved nerve.

The disease was called zona ("girdle") by the Greek, because of the band like distribution of the eruption about the trunk. Bokay (1888) suggested a possible etiologic relationship between zoster and varicella. Paschen (1919) demonstrated virus in a smear from vesicle fluid and Rake et al (1948) reported the quadrangular shape of virus with approximate dimension 210 x 240 mμ which was also reported by Evans and Melnick (1949). Lipsechutz (1921) defined the specific histopathology of the skin lesion.

The cultivation of varicella zoster virus in human tissue was first reported by Willer and Stoddard (1952). Willer and Coons (1954) used the fluorescent antibody technique for serologic identity of virus from varicella and herpes zoster and demonstrated that antibody from patients combined with virus or virus antigen in the cells infected with virus from either disease.

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Epidemiological Observations:

Herpes zoster occurs sporadically throughout the year while chickenpox or varicella is an epidemic disease with a definite seasonal incidence. Observations of 53 cases, who attended the out-patient department of District Hospital, Hamirpur, U.P. for treatment during the period of January, 1971 to December, 1971, showed that the incidence increased during March, April, August, September and December as shown in Table I. Mathur et al (1967) observed the increased incidence in March, August and December in their study of 62 cases.

This increase in incidence is related to the incidence of chickenpox in the city which has been observed by us and the same was also reported by Mathur et al (1967). The previous history of varicella (35 or 66%) led to the theory that adult herpes zoster is the result of provocation of dormant varicella virus in a partially immune host. This provocation can occur during an outbreak of epidemic in the surroundings. Simpson (1954) recorded high figure of herpes zoster among chickenpox contacts. Rake et al mentioned an instance where 10 out of 22 susceptible contacts of a case of zoster in a children's hospital developed chickenpox 15–19 days after appearance of eruption.

Infection is rare in children but increases in frequency, severity and duration with age and is more frequent in males than females (Lewis L. Coriell 1966). Mathur et al (1967) has given

TABLE I

Showing age and sex incidence with monthwise incidence of herpes zoster in 1971.

Age group & Sex	Jan	Feb	March	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Total	Percentage
Upto 10 years	—	1	—	1	—	—	—	2	—	—	—	1	5	9.4
11-20 ,,	—	—	2	2	—	1	—	2	3	—	1	2	13	24.5
21-30 ,,	1	—	1	2	1	—	1	1	1	—	1	2	11	20.7
31-40 ,,	—	1	—	1	—	—	—	2	2	—	—	2	8	15.2
41-50 ,,	—	—	1	2	—	1	—	1	1	—	—	1	7	13.2
51-60 ,,	1	—	1	1	—	—	—	2	—	—	—	1	6	11 3
61 & above	—	—	1	1	—	—	—	—	1	—	—	—	3	5.7
Total	2	2	6	10	1	2	1	10	8	—	2	9	53	100
Male	2	1	4	7	1	1	1	6	6	—	2	6	37	69.8
Female	—	1	2	3	—	1	—	4	2	—	—	3	16	30.2

male female sex ratio of 3 : 1. In our series we also observed that zoster infection is less commonly seen in children (5 or 9.4%), commonly seen in young adults of second, third and fourth decades of life with male female ratio of 2.3 : 1.

CLINICAL OBSERVATIONS

1. Pre eruptive stage :

It was observed that persons usually come to the hospital after 3 or 4 days when multiple vesicles have appeared on the path of nerve etc. The pre-eruptive stage consists of fever and constitutional symptoms, with pain, paresthesias or hyperaesthesia over the distribution of the involved nerve for 2–4 days. Fever usually varied from 38° c to 39° c as shown in Table II.

TABLE II

Symptoms and signs in pre-eruptive stage

Symptoms and signs	No. of cases	Percentage
Pyrexia (38° - 39.6°-c)	48	90.6
Malaise	50	94.3
Pain over affected part with paresthesias or hyperaesthesia	52	98.1

2. Eruptive phase :

Following the pre-eruptive phase there appears erythematous dermatitis which quickly becomes papular and vesiculates with large or small grouped vesicles on an erythematous base. The vesicles at first clear, later appear in bunch on a nerve distribution, affecting only half side of body and stops abruptly at midline.

Out of the 53 patients about 50% were having lesions on the chest involving intercostal nerves and other involved sites shown in table III.

TABLE III

Showing the site of lesion.

Site of lesion	No of cases	Percentage
Face and neck	4	7.4
Upper extremity	7	13.3
Chest	27	50.9
Abdomen	12	22.7
Lower extremity	3	5.7

In some cases (10 or 18.9%) the eruptions appear first near the spinal column with successive crops over the distal distribution of the nerve, which later on in four cases became bilateral which was unusual presentation. The pain and vesicular band, following radicular lines run transversely around the trunk or vertically over arm and leg, but it is rarely seen below the elbow or knee, which was also observed by Bargaon and Lewis L. Coriell (1966). Meningismus and headache was also observed mainly in 4 or 7.4% of cases

where face and neck were involved. Pain is much less or even absent in children (5 or 9.4%) as in table IV.

TABLE IV

Symptoms and signs of eruptive phase.

Symptoms and signs	No. of cases	Per-centage
Eruptions	53	100
Pain of burning type	44	83
Meningismus	4	7.4
Headache	11	20.9
Regional Lymphadenopathy	41	76.4
Secondary infection of vesicles	21	39.6

3. Clinical Investigations :

Leucocytosis was only seen in 22 or 41.5% of cases who had secondary infection of ruptured vesicles as shown in table V. Lumbar puncture was done only in 21 cases.

TABLE V

Showing results of clinical investigations.

Clinical investigations	No. of cases	Per-centage
1. Leucocytosis-max. 22000/cu.mm	22	41.5
2. Cerebro-spinal fluid (21 cases) increased protein-max-50mg%	5	23.8
mononuclear pleocytosis max. 150/cu.mm	7	33.4
Increased pressure	5	23.8
3. Erythocytic Sedimentation rate max-36 mm	23	43.4

Lewis L. Coriell (1966) Bailey (1952) Simpson (1954) observed that fluid from unruptured vesicles is sterile, C.S.F. in 40% of cases showed increased pressure, pleocytosis upto 300 mononuclear cells. They have also observed raised E. S. R. which is mainly due to secondary infection.

4. Complications :

The following complications were observed.

(a) Post herpetic neuritis 6 or 11.3%

(b) Perceptive deafness 1 or 1.9%

(c) Arthritis 1 or 1.9%

(d) Radiculitis 1 or 1.9%

Post-herpetic neuritis, a common complication, was characterised by severe pain along the distribution of the nerve involved at the time of appearance of herpes zoster. The same was also described by Mathur et al (1967) and Lewis L. Coriell (1966).

Conclusions :

The following conclusions have been made by the study of 53 cases :—

1. Definite relationship was observed in Herpes Zoster and Varicella.
2. Commonly seen in young adults on 2nd, 3rd and 4th decades of life.
3. Females are less commonly affected with sex ratio to male is 1 : 2.3.
4. Lesions are almost unilateral and common site involved is intercostal nerves.
5. Erythematous dermatitis followed by vesicular bands following radicular lines. Eruptions have erythematous bases and secondary infection may take place in ruptured vesicles.
6. Increased pressure, raised protein and pleocytosis in C.S.F.
7. Most common complication is post herpetic neuritis.

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False

Studies on blood flow with tissue clearance technique using iodoantipyrine I 131 (I.A.P.) shows that there is no statistical difference between increased blood flow of the normal and atopic individual. Vessels are actually dilated in areas of blanching but the visible evidence of hyperemia is obscured by the local accumulation of edema fluid.

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