SIMULTANEOUS OCCURRENCE OF HERPES ZOSTER AND VARICELLA IN A PATIENT OF TUBERCULOUS LYMPHADENITIS

(A case report)

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Summary

A rare case of concurrent herpes zoster and varicella probably precipitated by tuberculous lymphadenitis has been reported and relevant literature is reviewed.

Bokay¹ was the first, three quarters of a century ago, to claim purely on clinical grounds that varicella and herpes zoster were caused apparently by the same agent. Since then, this view has been supported by many authors. 2 8 4. Both the diseases present with very different clinical mani-festations and age distribution. However, the causative agents of the two syndromes have been shown to be indistinguishable morphologically, antigenically and biologically. 5 6 7.

Varicella has a seasonal peak, usually in the spring; most patients give a history of contact with another infectious patient; it is usual response to primary infection and is seen generally in children. Herpes zoster, in contrast, is independent of any seasonal influence and it is an endemic infection; many patients have not known exposure to either of these infections; but it is the manifestation of a recrudescent infection with residual antibody persisting from previous varicella and occurs

commonly in adults. There have been many reports of the increased severity of varicella and herpes zoster infection in persons without exposure following steroid therapy, 8 9 10 11 12 18 14. X-irradiation, 14 15 in the presence of lymphoproliferative diseases, 4 18 14 16 spinal tumor, 18 malignant process, 18 14 arsenic poisoning or therapy 12 18 19. or in patients in whom immunologic factors of resistance were subnormal because of generalized illness. 14 20 Almeyda has reported associated miliary tuberculosis in one of his 3 cases of varicella and herpes zoster.

Simultaneous occurrence of herpes zoster and varicella in same patient is uncommon. The review of literature shows very few reports uptil now. We had one patient having varicella and herpes zoster simultaneously. He had tuberculous lymphadenopathy and cold abscess in association. Because of the rarity of this association, we have been prompted to report about this patient.

Case Report

A 29-year-old Hindu male was admitted to the Irwin Group of Hospitals, Jamnagar, on 23rd November 1971, for

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M. P. Shah Medical College and Irwin Group of Hospitals, Jamnagar Received for Publication on 2-4-1973 a severe attack of herpes zoster involving the sensory distribution of the ophtalmic branch of the left trigeminal nerve. The attack was preceded for three days by malaise, intermittent moderate fever and an acute neuritic pain on the left side of the scalp, eye and some part of the nose. He was taking antituberculous drugs (Injection streptomycin and tablet isonex) for tuberculous cervical lymphadenopathy since last one month. He did not remember having had any disease in childhood and had no recent known contacts with varicella or zoster.

On examination, patient looked ill. He had 101°F, temperature; pulse rate was 104/min. and blood pressure was 110/80 mm of Hg. Vasiculobullous skin eruptions involving the ophthalmic branch of left trigeminal nerve were present (Fig. 1) Several vesicles over the nose were haemorrhagic. Marked swelling of left eye lids and conjunctival chemosis were observed and it was difficult to open the left eye. Bilateral



Fig. 1
Showing Herpes Zoster involving the first branch of left trigeminal nerve

cervical lymph glands were palpable. Respiratory, cardiovascular, alimentary and central nervous systems were clinically normal.

He was treated with oral analgesics, antihistaminics and local application of lotio calamine over the herpetic eruptions; tetracycline ophthalmic ointment was applied in the eyes. Antituber-culous drugs were continued.

Two days later, while he was still febrile, he developed generalized scattered macular eruptions turned into papules and vesicles all over, giving the appearance of 'true' varicella. (Fig 2) The lesions were noticed in all their stages, mainly, papules, vesicles, pustules and crusts. The temperature settled down after six days of admission. The varicelliform rash had cleared in 12 days, whereas, herpatic lesions had not completely disappeared and took further three weeks to clear. He developed cold abscess in the cervical region on the 12th day of admission; thick pus was aspirated from that.



Fig. 2

Showing rashes of varicella in the back and tuberculous cold abscess on the right side of the neck

Total leukocyte count was 8,200/cmm with polymorphs 60%, lymphocytes 35%, eosiniphils 4% and monocytes 1%. ESR was 54mm. at the end of 1st hour (Westergreen Method). Hb. was 10.4g%. Peripheral smear showed microcytic hypochromic anaemia. Urine analysis was normal. X-ray chest and fundus examination were normal. Aspirated pus from cold abscess was sterile. Serological tests for syphilis were negative. Culture for VZ viruses, fluorescent antibody or neutralization tests could not be carried out because of lack of facilities.

Recovery was uneventful and he was discharged with the advice to continue anti koch's treatment.

Discussion

Though the etiological agent of herpes zoster and varicella is one and the same, simultaneous occurrence of herpes zoster and varicella is very rare. Ferriman²¹, Campbell²² and Almeyda² have reported such cases. Varicelliform eruption followed unilateral herpetic attacks within the five-day interval in all the three cases of Almeyda². In our case also varicelliform eruption followed unilateral herpetic lesion on the fifth day.

As mentioned previously attack of herpes zoster can be precipitated by various factors such as, spinal tumor, neoplasms. X-irradiation. leukemia, steroid therapy, syphilis, arsenic or lead¹³ or any generalized illness which alters immunological responses14,20. Miliary tuberculosis was associated with herpes 'zoster and varicella in one of the three cases of Almeyda². addition to herpes zoster and varicella, our patient had tuberculous cervical lymphadlnitis with cold abscess formation. We presumed that tuberculosis which leads to alteration in immunologic response resulting in diminution of cellular resistance or defective production of the antibodies, might be the ultimate cause for the appearance of herpes zoster and varicella.

The virus of herpes zoster and: varicella is same, but possibly assumes a dual role, a neurogenic role when it is confined to the posterior root ganglia, its: fibres, its peripheral nerves, in the perivascular spaces, the cerebrospinal fluid, and a haematogenous role when it finds its way into the general bloodstream. It explains the three clinical manifestations of this virus infection, namely, herpes zoster, varicella and the joint diseases. Soon after the initial attack of varicella, most of the sensory ganglia in the body for the rest of their lives begin harbouring a harmless component of varicella such as provirus fifty or more foci in incomplete virus in a latent form. It may revert to normal infectiousness in favourable conditions some of which have already been mentioned previously. The frequency of varicella outbreaks originating from herpes zoster can be explained by the facts that the neurotrophic variant can more easily, if given the right host, alter its nidus and become a haemotrophic one. The neurotrophic virus takes time to force a break-through from the perivascular spaces into the bloodstream, to assume the role of a haemototrophic virus.

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REFERENCES

- Von Bokay J: Uber den atiologischen Zusammenhang der Narizellen mit gewissen Follen von Herpes Zoster, Wien Klin Wschr 22:1323, 1909.
- Almeyda J: Simultaneous occurrence of Herpes Zoster and Varicella, Postgrad Med J 18: 175, 1942.

- Taylor, Robinson D: Herpes Zoster in patients with Chicken pox. Brit Med J 1:1713, 1960.
- Mosonyi L, Csiky Tand Kucsera P: Varicella developing from Herpes Zoster in Course of Steroid Treatment, Rheum Balneol Allerg, 4:101, 1963.
- Weller TH, Witton HM and Bell JE: Etiological Agents of Varicella and Herpes Zoster: Isolation, Propagation and Cultural characteristics in Vitro, J Exp Med 108: 843. 1958.
- Taylor-Robinson D: Chicken Pox and Herpes Zoster, II-III, Brit J Exp Path, 40:517, 1959.
- Gold E: Characteristics of Herpes Zoster and Varicella Viruses Propagated in Vitro J Immunol, 95:683, 1965.
- Shee JC and Fehrsen P: Reactivation of Varicella Virus by Cortisone Therapy, Brit Med J, 2:82, 1953.
- 944 Haggerty RV and Eley RC: Varicella and Cortisone, Pediatrics, 18:160, 1956.
- Finkel KC: Mortality from Varicella in children receiving adrenocorticosteroids and adrenocorticotropin, Pediatrics, 28: 436, 1961.
- 11. Dux, E et al: Cited by 12.
- Rado JP, Tako J, Geder L et al: Herpes Zoster House Epidemic in Steroid-treated Patients, Arch Interp Med 116: 329, 1965.

- 13. Hope-Simpson RE: The Nature of Herpes Zoster: A Long-Term Study and a New Hypothesis, Proc Roy/Soc Med 58:9, 1965.
- Gold E: Serologic and Virus-Isolation Studies of Patients with Varicella or Herpes-Zoster infection, New Eng J Mcd 274:181, 1966.
- Pendergrass EP and Kirsh D: Role of Irradiation in Management of Carcinoma of Breast, Radiology, 51: 767, 1948.
- Jaffee HS and Greenberg MS: Herpes Zoster resembling acute Varicella associated with Multiple Mycloma, JAMA 175:1008, 1961.
- Kaim HK, Feldman CA and Cohn LH: Herpes Zoster Generalsatus Pneumonia, Arch Intern Med, 110:98, 1962.
- Kamman GR: Herpes Zoster as Early Symptom of Spinal Cord Tumor, JAMA 91:320, 1928.
- Jacob FM: Bilateral Herpes Zoster following Acute Arsenic Poisoning, Arch, Derm Syph, 24: 280, 1931.
- Berlin BS and Campbell T: Hospital-Acquired Herpes Zoster Following Exposure to Chicken Pox, JAMA 211: 831, 1970.
- 21. Ferrman, DG: Cited by 2.
- 22. Campbell RM: Cited by 2.

TRUE or FALSE?

Ultrastructural change of thickening of BM of cutaneous vessels is not a significant finding in Diabetes Mellitus and cannot be considered a cutaneous marker of Diabetes Mellitus.

(Answer page No. 257)