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INFECTIVITY OF VARICELLA AND HERPES ZOSTER

To the Editor,

Varicella is transmitted by droplet infection from nasopharynx. Susceptible people may contract varicella from patients of either varicella or herpes zoster (HZ) as vesicular fluid of HZ is also infectious. Importance of vesicular fluid of varicella in transmission is not known although it contains great deal of virus. Herpes zoster usually occurs as sporadic affliction of individual or rarely in clustered or localized epidemics.[1] These clustered epidemics show that herpes zoster is occasionally temporally related to exposure to varicella zoster virus (VZV).

A 29-year-old woman had attack of herpes zoster in relation to trigeminal nerve 20 days back and she was put on laser treatment as pain persisted after clearance of lesions. After 2 days she brought her 4-year-old son who had crop of polymorphic eruptions which were centrepetal in distribution. The child was diagnosed as a case of varicella. There was no history of similar lesions at home or in neighbourhood. One day later his 7-year-old sister also showed similar features.

In another case, an old man of 50 years had been suffering from pain and burning sensation in distribution of C5-6 segments on right side for 3 days which was followed one day later by appearance of grouped papulovesicular lesions. His 3 grandchildren who stay with him had already taken treatment for varicella 12-14 days prior to appearance of herpes zoster symptoms in him.

Events described above clearly show development of varicella following herpes zoster and reciprocally development of herpes zoster following varicella. Illnesses followed appropriate incubation periods. Herpes zoster to varicella is not uncomman and Seiler[2] found the incidence of 15.5% amongst susceptible children who had not previously had varicella. We believe that this mode of transmission is more frequent than observed and is common especially when index case is young and children in same family had not yet suffered from varicella or if grandparents suffer from herpes zoster then grandchildren get varicella form them.

The explanation for second case is that reactivation of latent virus in ganglion may be due to reinfection with VZV as is also evident from appearance of herpes zoster in clusters.[1] Similar cases have also been reported in past.[3] Defences that are responsible for preventing recrudescent VZV infection are reliant on continual boost of immunity consequent upon subclinical reinfectin. It is possilbe that at times reinfection may stimulate humoral immunity which interferes with cell mediated defences and leads to reactivation of VZV with clinical lesions of herpes zoster. Some immunity is present in such cases and therefore they develop segmental herpes zoster rather than disseminated disease which is rare. Thus exposure to VZV may also be considered another factor for reactivating latent virus in herpes zoster in addition to other established precipitation factors such as trauma,

irradiation etc. So herpes zoster should be considered a potentially infectious or contagious disease.

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MALIGNANT ACANTHOSIS NIGRICANS

To the Editor,

Acanthosis nigricans (AN) which is associated with malignancy usually occurs after 35 years of age, is quite extensive with involvement of mucous membranes, palms and soles and has accompanying pruritus. Most commonly associated malignancies are adenocarcinomas of gastrointestinal tract.

A 38-year-old female had generalised pruritic hyperpigmentation and brown velvety, verrucous lesions in flexors for past 4 years. Her oral and genital mucous membranes were velvety and hypertrophic, palm and soles were thickened and pigmented and dorsa of the hands showed fine papular lesions. For the last 3 years she had developed off and on pain in epigastrium with loss of weight and appetite and generalised weakness. For past 8 months, she had also developed two linear rows of hyperpigmented lesions with warty surface besides the verrucous lesions of AN. She was admitted to hospital and during the stay she developed migratory pain and swellings in the right arm and above the left eyebrow. This was diagnosed as migratory thrombophlebitis. Routine investigations, skiagrams of chest and pituitary fossa, barium series and USG of both upper and lower abdomen were normal. Histologically lesions from cubital fossa were acanthosis nigricans and those on breast were seborrhoeic keratosis. USG was repeated during subsequent visit to hospital two months later and it showed a mass 2"x1" involving the body of pancreas.

Acanthosis nigricans and suddenly erupting seborrhoeic keratosis are known markers of internal malignancy and association between the two is known. The presence of both in our patient suggested the presence of some internal malignancy which came out to be carcinoma of the body of pancreas.

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BENIGN MUCOUS MEMBRANE PEMPHIGOID SIMULATING GENITOULCERATIVE DISEASE

To the Editor,

A 45-year-old male patient had recurrent vesiculobullous lesions on the prepuce, coronal sulcus and glans penis for 5 years. There was burning micturition, moderate itching and pain. There was no history of extramarital sexual contact or any constitutional symptoms. Multiple ulcers, tender 3 mm to 3 cm in size, variable in shape with well-defined non-indurated margins and red granulation tissue were seen on the glans penis, shaft of penis and undersurface of prepuce. In addition a few small, 4 mm to 1 cm tense bullae containing clear fluid were also seen. Mucosa around the ulcers and bullae was whitish, wrinkled and firm. Regional lymph nodes were not enlarged. Histopathologically moderate acanthosis, small splits in basement membrane zone and one big bullae extending to full epidermis were seen. Dermal fibrosis,