LETTERS TO THE EDITOR

KERATOACANTHOMA MASQUERADING AS SQUAMOUS CELL CARCINOMA

To the Editor,

Keratocanthoma is a rapidly evolving tumour of the skin composed of keratinising squamous cells originating from pilosebaceous follicles. It can closely simulate squamous cell carcinoma both clinically and histologically.[1] While solitary keratoacanthoms are not uncommon, the multiple variety is rare.[2]

A 55-year-old male tailor presented with complaints of recurrent horny lesions over both lower limbs of 30 years duration. Lesions initially grew rapidly for 1-2 months and then stopped growing. Some of the lesions resolved spontaneously, while surgical excision of the remaining lesions was done in 1965. The lesion recurred again over the angle of the mouth on the right side. It was excised and reported as squamous cell carcinoma. For the past 6 months he had a fungating mass, 13x10 cm, over left foot extending from ankle posteriorly to the middle of the foot exteriorly (Fig. 1). A horny lesion was seen adjacent to this. Multiple horny lesions were present over both limbs with involvement of nail fold sulci. 5-6 inguinal lymph nodes were palpable, which were discrete, firm, mobile and non-tender.

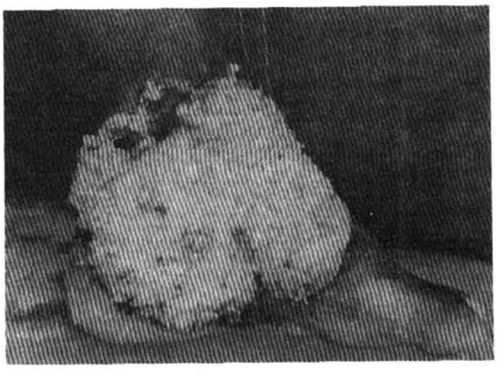

Fig. 1. Large fungating growth over the medical aspect of the left foot.

Systemic examination was non contributory. Indirect laryngoscopy, ultrasonogram of abdomen and upper GI endoscopy did not reveal any abnormality. Multiple biopsies from the edges of the lesion showed an irregular large central crater filled with keratin material. Epidermis extended on either side as a lip. There were no bizzare mitotic figures or hyperchromatic nuclei to suggest squamous cell carcinoma. A diagnosis of keratoacanthoma was made. Histopathology of the inguinal lymph nodes did not show any metastasis. Total excision of fungating mass was done with split skin grafting. Rest of the lesions were untouched. Two of patient's sibling also had similar lesions while rest of the family members were normal.

Points which may help on histological differentiation between early keratoacanthoma and squamous cell carcinoma include the following.[3] In keratoacanthoma, organisation of epithelial growth is not disturbed and cells are attached to one another with well formed prickles. Mitotic figures when present are of natural appearance and the ratio of nucleus to nucleoli is unaltered. Basal cell membrane is sharply limited and well preserved and dermal inflammatory infiltrate permeates the epithelial growth. These are not seen in squamous cell carcinoma. Inspite of these differences it may be difficult to differentiate the two conditions as had happened in our case.

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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,