of the undifferentiated nature of the metastases and unwillingness of patient to continue treatment, the primary site of malignancy could not be ascertained. But it is likely that these metastases could have arisen from prostrate because bone involvement in prostatic carcinoma is not uncommon.

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Koebner response in psoriasis

To the Editor

In 1872, Dr. Heinrich Koebner spoke on the cause of psoriasis, presenting a case in which, 5 or 6 years after the appearance of an isolated plaque of psoriasis, various traumatic events in remote parts of the body (excoriation from horseback riding, suppuration from lymphadenitis, horse bite, and tattoos), evoked outbreaks of psoriasis in the patient at exactly the same site, in the shape of the injured skin.[1] This phenomenon is known as the isomorphic or Koebner response to injury.

Koebner believed that the skin of a patient with psoriasis has a peculiar predisposition to injury that may remain latent for many years. At variable intervals, local irritation will result in psoriaisis.[2] Patients with Koebner response are a "unique group who have distinctive epidermal or dermal response. Koebner response may be a marker for a subset of psoriatic patients. For these patients injury is a pathway to psoriasis. This pathway may result in an early onset or in an early flare of psoriasis.[3]

Psoriatic lesions have appeared following trauma due to gunshot wounds, lacerations, operative incisions, tattooing, burns, ultraviolet light, primary irritation from chrysarobin, iodine application, or in association with infections and furunculosis, pressure from wrist watch and even grasping of a pencil. There is usually a 10 to 14 day latent period between injury and development of lesions, but onset may be delayed as long as 2 years.[4]

My first case is a 30-year-old electrician who developed an isolated psoriatic plaque exactly at the site of electric shock injury on the tip of right middle finger approximately 3 weeks after trauma. The lesions subsequently spread to involve both palms and soles, and developed into a classical case of palmo-plantar psoriasis within 3 months of electric shock injury.

The second case is a 20-year-old polio affected boy who developed psoriasis over his left sole, lateral border of left foot and left shin at the pressure bearing points. His right lower limb was polio affected since childhood, leading to increased weight bearing and subsequent friction over his left lower limb.

To the best of our knowledge, this is the first case of development of psoriasis following electric shock injury in Indian literature. The second case amply points to psoriasis developing in areas of friction and increased weight bearing.

In different studies, 25% of all patients with psoriasis relate that at some time or other they have developed psoriatic lesions following trauma.[3,4] The Koebner response had been considered to be an expression of severe and/or labile psoriasis. However, it does not appear to be associated with the type of psoriasis, extent of skin involvement, resistance to therapy, worsening of psoriasis or duration of disease.[3] Etiologically, the predominance of infiltrating cytotoxic T-cells found in the epidermis and dermis in Koebner positive skin are activated by heat shock proteins and directly induce lytic changes in keratinocytes. Alternative explanations include degranulation of mast cells and release of proteases by macrophages.[5]

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Allergic reaction to phorate :an organophosphorus compound

To the Editor

Phorate ($C_7H_{17}O_2PS_3$) is a non-biocumulative organophosphorus compound marketed as 90% pure emulsifiable liquid or granules for the killing of mites, ophids, corn root worms, nematodes and other pests inhabiting crops, vegetables, ornamental or fruit plants. It is extremely toxic to mammals. Improper handling of the chemical or its container without adequate safety measures, may contaminate, drinking water, edibles, clothing, utensils and the human body.[1] It can be absorbed by lungs, skin and gastrointestinal tract. It is metabolized to phorate sulphoxide, phoratesulphone and oxygenated analogues in the liver and excreted as diethyl phosphoric acid, o-o diethyl phosphorothioic and o-o diethyl phosphorodithioic acids in urine(35%) and faces (3-5%).[2] It is seen that although the