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Psoriasis as Wolf's isotopic response over BCG scar

Sir,

Wolf's isotopic response, first introduced by Wolf *et al*., refers to the development of a new unrelated dermatosis at the healed site of a previous dermatosis.[1] The commonly reported second diseases are urticaria, lichen planus, psoriasis, infections such as molluscum contagiosum, fungal granuloma, rosacea, morphea and granuloma annulare.[2] Various dermatoses that can develop on a Bacillus Calmette-Guerin (BCG) scar site include, cutaneous tuberculosis, keloids, sarcoidosis and nodular hidradenoma but development of psoriasis over it is very rare. We describe a case of psoriasis developing on a healed BCG scar site in a child.

A 1.5-year-old healthy girl, born out of non-consanguineous marriage, presented to the skin department of MKCG Medical College and Hospital, Berhampur, Odisha, with a single, reddish, elevated, scaly lesion on the left upper arm, noticed by the mother five months after birth. There was a history of BCG vaccination, 24 h after birth, at the site. Subsequently, the injection site became swollen, red and ulcerated in 3–4 weeks for which the mother consulted some local physician and applied topical medication (nature unknown). The lesion healed with scar formation by of 8–10 weeks. After about three months, the mother noticed a grain sized reddish, elevated scaly lesion over the scarred area which gradually increased to attain the present size, associated with itching. Topical antifungals were applied but there was no improvement in the lesion. Family history was noncontributary.

On examination, a single, well-demarcated, erythematous, indurated plaque of size 3 × 3 cm with thick adherent white scales, was present on the left deltoid region on the site of BCG scar with sparing of other areas of the body [Figure 1]. Grattage test was done over the lesion and Auspitz sign was positive. Based on the clinical findings, psoriasis and psoriasiform lupus vulgaris were considered in differential diagnosis. All routine investigations were within normal limits. Tuberculin test was not done, due to its non-specific nature.

Histopathological examination of lesional skin biopsy revealed acanthosis, parakeratosis, hypogranulosis, regular elongation of rete ridges, collection of neutrophils within the parakeratotic layer with vascular proliferation in papillary dermis and mild-to-moderate chronic inflammatory cell infiltrates in superficial dermis, consistent with psoriasis [Figures 2 and 3]. With a final diagnosis of psoriasis vulgaris (possibly due to Wolf's isotopic response) on BCG scar, topical calcipotriol (0.005%) for twice daily application was started. After one month of treatment, the lesion showed improvement.

The various possible hypotheses formulated to explain this unusual phenomenon include viral, neural, immunological and vascular etiologies.[2] However, the most plausible composite hypothesis is "locus minoris resistentiae," or immunocompromised cutaneous district which refers to a focus of dysregulated immunity, caused by alternation

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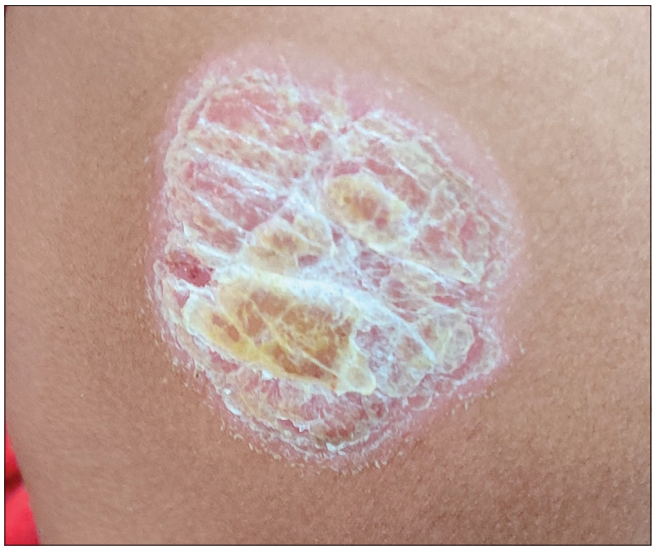

Figure 1: Single, well-demarcated, erythematous, indurated plaque of size 3 × 3 cm with thick adherent white scales on the left deltoid region, on the site of BCG scar

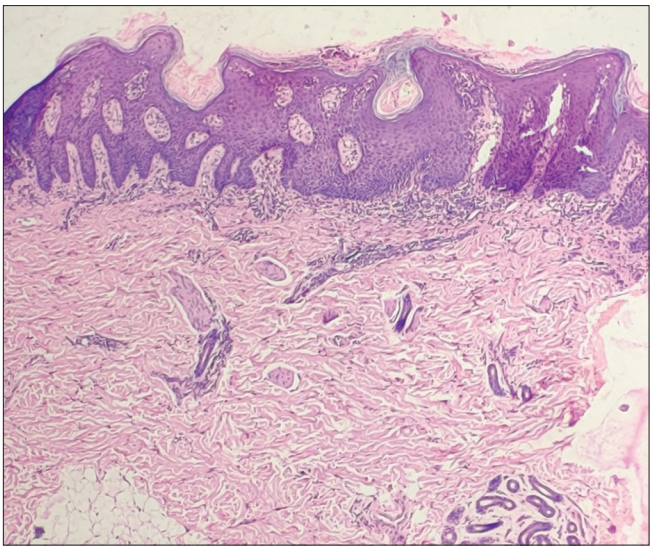

Figure 2: Scanner view showing acanthosis, parakeratosis, elongation of rete ridges, hypogranulosis, collection of neutrophils within the parakeratotic layer with vascular proliferation in papillary dermis and mild-to-moderate chronic inflammatory cell infiltrates in superficial dermis (H&E, 40×)

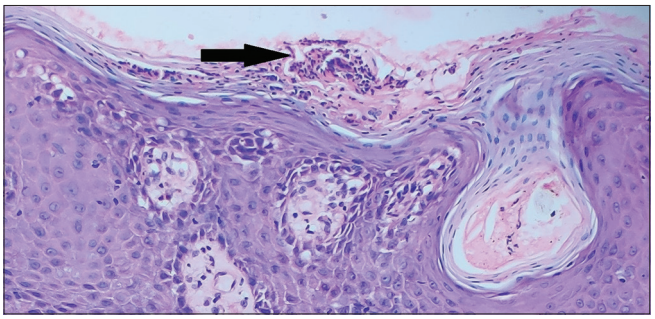

Figure 3: Magnified view showing collection of neutrophils within the parakeratotic layer that is Munro's microabscess (H & E, ×400)

in release of neuropeptides or defect in lymphatic flow, occurring secondary to any local cause such as herpetic infections, radiations, burns, intradermal vaccinations or stasis in lymphatic flow.[3] This local area serves as a site for the development of secondary infections, tumors or immune-mediated reactions.[3]

Host immune cells recognize BCG antigen and stimulate production of pro-inflammatory cytokines such as tumor necrosis factor-alpha, interleukin 1 and 6.[4] Antigen-presenting cells in skin present this antigen to the T-lymphocytes leading to T-cell priming and activation of acquired immunity.[4] This activated adaptive immunity together with dysregulated local immunity can provide a trigger for the development of immune mediated disorders like psoriasis. Takayama *et al*., reported occurrence of tuberculid eruptions with psoriatic dermatitis following BCG vaccination in a six-month-old baby. This was confirmed based on the presence of epithelioid granuloma in dermis, which improved spontaneously without any medications.[5]

Koebner response, a closely related phenomenon, needs to be differentiated here. In Koebner phenomenon, there is a development of similar cutaneous lesions in the setting of an already existing dermatosis, following any trauma; whereas in Wolf's isotopic response, there is a development of a new dermatosis localized to the particular site of a previously healed dermatosis without appearance of those similar lesions elsewhere in the body.[2] Further studies are necessary and reporting of such similar cases will be helpful in differentiating these two responses.

Here, we report a case of psoriasis developing on a BCG scar in a 15-month-old baby, five-month post-vaccination, possibly due to Wolf's isotopic response. This unusual, rare case can be explained by the hypothesis of local immune dysregulation with stimulated innate and acquired immunity following vaccination.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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Pseudodominant inheritance of self-improving collodion ichthyosis with homozygous mutation in the *ALOX12B* gene

Sir,

Collodion baby is a common phenotype for several autosomal recessive congenital ichthyoses, rather than a distinct disease entity. These babies present with a parchment-like membrane at birth, which gradually sheds in two to four weeks revealing the underlying ichthyosis variant. Around 60–80% children eventually develop lamellar ichthyosis or non-bullous ichthyosiform erythroderma. However, in about 10–20% cases, they represent self-improving collodion ichthyosis, previously called self-healing collodion baby.[1,2] It is associated with homozygous or compound heterozygous mutation in *TGM1, ALOX12B, ALOXE3,* NIPAL4, ABCA12and *CYP4F22* genes.[3,4] We describe a case of self-improving collodion ichthyosis with homozygous mutation in *ALOX12B* gene with a pseudodominant inheritance pattern.

We report a two-year-old boy, born to second-degree consanguineous, South Indian parents, Dravidian by origin, following an uneventful pregnancy. At birth, the child had collodion membrane associated with ectropion and eclabium [Figure 1]. The collodion membrane subsequently shed by four weeks, exposing a mildly xerotic skin. However, at the age of one year four months, the child developed fine generalized scaling with well- defined erythematous and scaly areas involving the the angles of mouth, neck, axillae, scalp and groin along with mild palmoplantar keratoderma, consistent with mild ichthyosiform erythroderma [Figures 2a-2d]. Systemic examination was unremarkable. Family history revealed similar dryness of skin in the child's father at birth which gradually improved with age, without any definite history of collodion membrane at birth. Paternal cutaneous examination revealed fine scaling all over his body with mild palmoplantar keratoderma. Skin biopsy was not done in our case.

Figure 1: Child at birth with collodion membrane

Next generation sequencing demonstrated a homozygous, missense variation in exon 12 of the *ALOX12B* gene which resulted in the amino acid substitution of tryptophan for arginine at codon 548, c.1642C>T (p.Arg548Trp;ENST00000319144.4). The observed variation was noted in the lipoxygenase domain of the ALOX12B protein. The *in silico*

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