



ADVERSE DRUG REACTIONS, INCLUDING SJS, TEN

PITYRIASIS ROSEA-LIKE ERUPTION FOLLOWING INFLUENZA (H1N1) VACCINATION: ABOUT A CASE REPORT

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Background: Pityriasis rosea (PR) is a common, acute, and self-limited inflammatory skin disease. Its cause is still not completely understood. However, viral agents, autoimmunity, psychogenic status, and numerous drugs have been proposed as possible factors to PR. Up to date, 29 cases of PR-Like Eruption have been reported after vaccinations for smallpox, tuberculosis, poliomyelitis, influenza, papillomaviruses, diphtheria, tetanus, hepatitis B, pneumococcus, diphtheria-pertussis-tetanus (DTP) and yellow Fever. We report a case of PR-Like eruption induced by influenza vaccine.

Observation: A 28-year-old male presented to our dermatology clinic with a non-itchy skin rash, which had been present for three weeks. The patient described that a herald skin lesion was spotted initially followed several days later by the onset of many scaly lesions on his trunk and proximal extremities. The first herald patch developed two days after he underwent H1N1 vaccination (vaxigrip). An examination revealed a widespread eruption mainly involving the trunk and proximal upper limbs. There were numerous erythematous plaques, many of which had peripheral scales. Routine laboratory analyses showed eosinophilia. Skin biopsy demonstrated a slightly acanthotic epidermis with mild spongiosis. The superficial dermis revealed slight papillary edema, perivascular and superficial dermal infiltrate of lymphocytes and histiocytes. This aspect of eczematous dermatitis with lesions of keratinocyte necrosis was in favor of a drug eruption. Total regression of all lesions was noted within two months.

Key message: In our case, the skin rash developed within 2 days of vaccination: a causal relationship was therefore highly suggested in our patient. The precise mechanism leading to PR after vaccination is unknown. However, the reported cases have driven the hypothesis that vaccination induced immune stimulation may trigger the reactivation of latent infectious agents such as HHV-6 or HHV-7 and subsequently the PR develops owing to the reactivation of viruses.

