REFERENCES


Elephantiasis of External Ears: A Rare Manifestation of Pediculosis capitis

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A case of elephantiasis of the external ears associated with pediculosis capitis is reported. Hypersensitivity to the louse bite and Melkersson-Rosenthal syndrome are discussed.

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Elephantiasis and elephantiasiform enlargement of the ears have rarely been described, following eczema, frostbite, erysipelas and piercing of the ears (3). Elephantiasis of the ears associated with pediculosis does not previously appear to have been reported.

CASE REPORT

A 33-year-old woman with enlarged and mildly pruritic ears of 3 years’ duration had been heavily infested with pediculus capitis for several years. Both ears were erythematous and markedly swollen: the right one (Fig. 1) was more severely affected. A few post-auricular subcutaneous nodules (5-10 mm) were present bilaterally. The skin over the nape of the neck and upper back was indurated and hyperpigmented. Right cervical lymph nodes were enlarged. She had a persistent right peripheral facial palsy, thought to be Bell’s palsy. The white cell count ranged 8.2-11.3 x 10^9/l. with 8-11% eosinophils. The erythrocyte sedimentation rate was 45 mm/hour (normal 20 mm/hour).

Other tests including serum protein electrophoresis, liver function tests and antinuclear antibodies were normal. Scrapings of ear lobes and nasal mucosa showed ano acid-fast bacilli. Chest X-ray, and bone survey were normal. Patch tests with the whole battery of European standard allergens proved negative. Cervical lymph node biopsy showed non-specific reactive changes. Skin biopsies of the right ear (Fig. 2) showed hyperkeratosis, follicular plugging, focal spongiosis, elongated rete ridges and numerous branching hair follicles. The dermis was infiltrated by chronic inflammatory cells composed predominantly of lymphocytes together with eosinophils, plasma cells and a few melanin-laden macrophages. The cellular infiltrate formed aggregates with no germinal centres around dilated capillary branches. Numerous dilated and conspicuous lymphatic spaces were present, especially in the superficial and mid-dermis. Mast cells were apparently increased in number. No acid-fast bacilli, epithelioid or foreign-body type granuloma were seen. There was no iron pigment or amyloid deposition. Biopsies of post-auricular nodules and the skin of the back showed similar changes. However, the lymphatics were not dilated or conspicuous in the biopsies of the back lesions.

Her pediculosis was treated with DDT. Intralessional injections of 10 mg/ml triamcinolone acetonide (kenalog) resulted in a decrease in the size and induration of the ears, with almost complete resolution of subcutaneous nodules.
COMMENT

Repeated exposures to the body louse will result in a hypersensitivity reaction in most individuals (7). Experimentally sensitized humans to sandflies (9) and to mosquito bites (6) and guinea pigs to flea bites (1), regardless of species of arthropod, manifest a definite sequency of types of reactivity.  

Histological studies on human skin (2) and on guinea pigs (4) in different stages of reactivity to arthropod bite suggest that histological changes conform with those of immediate and delayed hypersensitivity reactions. Cutaneous manifestations in this patient appear to be the result of long-standing exposure to the louse bite in a sensitized individual. Elephantiasis of the ears may be due to a continued hypersensitivity reaction in this particular anatomical location. Obstruction of lymphatic vessels by granulomas has been proposed as a factor in cheilitis granulomatosa (8). In the absence of typical clinical features and absence of epithelioid granulomata, cheilitis granulomatosa appears unlikely in our case. Chronic hypertrophic vulvulitis with similarities to cheilitis granulomatosa have, however, been described (5).

REFERENCES


Mutilating Palmo-Plantar Keratoderma

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A 36-year-old male, with no history of hereditary palmo-plantar keratoderma, showed at the age of 2–3 years a typical keratoderma of the Unna Thost variety on palms and soles. At the age of 16 years he developed mutilating symptoms localized to the fingers. Radiologic examination showed abnormally pointed end phalanges. (Received December 21, 1982.)

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In the northernmost county of Sweden (Norrbotten) the incidence of hereditary palmo-plantar keratoderma Unna Thost was found to be 0.55% (1); one case of mutilating palmo-plantar keratoderma was found in this material (5, 2). Two types of mutilating palmo-plantar keratoderma have been described, the first a syndrome associated with the name of Vohwinkel, determined by an autosomal dominant gene with or without associated anomalies; the second an autosomal recessive type connected with ectodermal dysplasias (3, 6).

Palmo-plantar keratoderma is present from infancy and, though diffuse, is honeycombed by small depressions. The hyperkeratotic areas are generally surrounded by an intensive erythroderma on both hands and feet. Constricting hyperkeratotic bands lead to progressive strangulation of the digits from the age of 4 or 5 years, though this development can be delayed for 25–30 years. Hyperhidrosis is invariably present, and warty papules on the backs of hands and feet are usually to be found. In the autosomal dominant type of mutilating keratoderma the following associated symptoms have been described: linear keratotic lesions on elbows and knees, reticular skin pigmentation, dystrophia of the cornea, lipomatosis and osteopetrosis (Albers-Schönberg disease). The autosomal recessive type is connected with loss of hearing, patchy cicatricial alopecia and keratosis of the groins and perianal skin.