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Calciphylaxis: Passive Transfer

Abstract. Rats were sensitized by mouth with dihydrotachysterol and subsequently challenged by a subcutaneous injection of ferric dextran. Mineralization at the injection site is barely detectable 17 hours after challenge. If, at this time, the challenged skin is transplanted onto a normal recipient, mineralization continues in the nonsensitized host. Sensitization is indispensable only for the initiation of the calciphylactic response and, once "triggered," the mechanism for this type of mineralization is transferable.

Calciphylaxis is a biologic mechanism through which mineralization can be elicited selectively in limited areas of the body by the administration of "challengers" (for example, iron compounds) during a "critical period" after "sensitization" (for example, with parathyroid hormone or vitamin-D derivatives) (1). To gain more insight into the mechanism of this response, experiments were

designed to determine whether a calciphylactically challenged skin region will continue to undergo progressive mineralization once transferred from a sensitized donor to a nonsensitized host.

Ninety female Sprague-Dawley rats (2) with a mean initial body weight of 100 g (range, 95 to 105 g) were subdivided into nine equal groups (40 donors, 40 recipients, and 10 controls) and treated as indicated in Table 1. As a calciphylactic sensitizer, 1 mg of dihydrotachysterol (DHT) (3) in 0.5 ml of corn oil was administered by stomach tube on the 1st day. Ferric dextran, "Fe-Dex" (4) was diluted to contain 500 μ g of elementary iron in 3 ml of water. Twenty-four hours after administering DHT, the subcutaneous tissue under the shaved back of each rat in groups 2 to 5 was infiltrated with this amount, over an area approximately 3 cm in diameter. Seventeen hours later, the Fe-Dex-treated area was excised, a small portion of it was taken for histologic study, and the rest was transplanted either into untreated recipients or into hosts which had been sensitized with DHT 24 hours prior to transplantation, in the same manner as the donors. In order to minimize the possibility of infection and external trauma, the skin flaps were introduced through a small incision underneath the dorsal skin of the host and deposited flat against the back. The operation was performed essentially according to a previously described technique (5), but only the cutaneous wound of the host was sutured.

The animals were killed with chloroform on the 6th day. The biopsy specimens taken at the time of transplantation, and the transplants removed at autopsy were inspected with a dissecting loupe and fixed in alcohol-formol (four parts of absolute alcohol and one part of 10 percent formalin) for subsequent staining of paraffin-embedded sections by the von Kóssa technique for the demonstration of calcium phosphate. The intensity of calcification was expressed in terms of an arbitrary scale of 0 to 3(1).

As indicated in Table 1, skin transplants from untreated (group 1) or merely from Fe-Dex treated (group 2) donors onto untreated recipients, underwent virtually no calcification except for occasional traces of "dystrophic" mineralization of presumably traumatized cutaneous muscle fibers. The Fe-Dex-treated skin of DHT-sensitized

Table 1. Passive transfer of calciphylaxis.

Treatment		Calcifi- cation
Donor	Recipient	in dermis
Untreated	Group 1	
	Untreated	U
	Group 2	
Fe-Dex	Untreated	0
	Group 3	
DHT + Fe-Dex	Untreated	1.8
	Group 4	
DHT + Fe-Dex	DHT	2.8
	Group 5	
DHT + Fe-Dex	(Skin left in situ)	3.0

donors (group 3) showed either no trace or only histologically visible traces of calcification in the challenged derma at the time of transplantation (as judged by the biopsy specimens); however, these same skin flaps exhibited even macroscopically conspicuous calcium deposits after transplantation into unsensitized recipients. Mineralization was most intense along the cut edges of the



Fig. 1. (Left) Biopsy specimen of skin challenged with Fe-Dex, taken from a DHT-sensitized donor just prior to transplantation. No calcification is visible in dermis. (Right) Specimen from same skin flap removed from an untreated host at autopsy. Calcification (black areas) visible throughout the dermis, but most conspicuous at cut margin. A few fibers in the cutaneous muscle are also calcified (both sections by the von Kóssa method, × 35).

transplants where fluid exchange with the host was not impeded either by the surface epithelium or by the deep cutaneous musculature of the skin grafts. This dermal calcification was still more pronounced when skin flaps of similarly treated donors were transplanted into DHT-sensitized recipients (group 4). Here the calcification was almost as severe as in similarly treated rats in which the skin was left in situ (group 5). It is especially noteworthy that in groups 3, 4, and 5 the calcium deposition occurred mainly throughout the thickness of the dermal collagen (as is characteristic for this type of calciphylactic response); even the adjacent donor tissue was often calcified, while in groups 1 and 2 only occasional damaged muscle fibers underwent mineralization.

Presumably, once calciphylaxis is initiated by a challenging substance (Fe-Dex) in an animal suitably treated with a sensitizer (DHT), mineralization continues even if the challenged region is transplanted into an untreated recipient. Apparently, we are dealing with a trigger reaction which, once actively acquired, can proceed passively in the absence of such blood chemical changes (hypercalcemia, hyperphosphatemia, liberation of calcifiable matrix from the bones) as are induced by calciphylactic sensitizers.

> Hans Selye Giulio Gabbiani Beatriz Tuchweber

Institut de Medécine et de Chirurgie Expérimentales, Université de Montréal, Montreal, Canada

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Microsporum nanum: First Recorded Isolation from Animals in the United States

Abstract. The first cases in the United States of swine infections caused by the fungus Microsporum nanum are reported. The infections occurred in a herd of Yorkshire swine in Pennsylvania. The dermatophyte, which readily produced experimental infections, was identified by isolation and studies in pure cultures.

Microsporum nanum is a rare dermatophyte first reported from Cuba by Fuentes (1). The fungus, isolated from a human case of tinea capitis, was



Fig. 1. Microsporum nanum. Hyphae present in skin scrapings. $(\times 400)$

first considered to be a dwarf form of M. gypseum. Subsequent study of new isolates from humans led to a redescription of the fungus as a new species (2). Dawson and Gentles (3) noted that they had received cultures of M. nanum isolated from a group of pigs in Kenya. Recently Brock (4) reported the isolation of this dermatophyte from a case of tinea capitis in man in Louisiana.

Since there is only one published record of the occurrence of M. nanum in the United States and the African pig infections were not described, it was considered worthwhile to report on an outbreak of M. nanum in a herd of swine in Centre County, Pennsylvania.

A ringworm-like lesion was first noted on a 3-year-old Yorkshire sow. The lesion, measuring 13 cm in diameter, was located just caudal to the left shoulder. It had a red cast and was covered with many superficial brown crusts. The crusts were especially prominent at the periphery of the lesion, and they formed a prominent band 2.5 cm in width. The skin was somewhat rough in the infected area. There was no apparent alopecia or pruritus. Examination of other swine in the herd has revealed a number of similar lesions.

On 16 August 1963, skin scrapings were taken for mycological examination from the periphery of the lesion on the 3-year-old sow. A small portion of the scrapings was treated with 15 percent KOH and examined microscopically. Pectinate, highly branched, septate hyphae were present. These hyphae were approximately 2.5 μ wide and of various lengths (Fig. 1). A tentative diagnosis of a dermatophyte infection was made. To confirm this diagnosis, studies in culture media were initiated.

To inhibit bacterial contaminants, scrapings were incubated overnight in 1 ml of distilled water with 1000 units of penicillin and 0.1 g of streptomycin. The specimen treated with antibiotic was then placed on veal-infusion agar and incubated at room temperature for 3 days.

Ten white, rapidly growing floccose colonies appeared. Each colony was 2 mm in diameter. The undersides of the colonies were tannish-orange. During the 1st week of growth, each colony retained its floccose appearance. As the colonies aged, they became granular. The centers were cream-colored, with a buff colored periphery. The reverse side was rose-colored.

Specimens were taken from the colonies and examined microscopically. Numerous macroconidia of *M. nanum* were found. They were all thin-walled,



Fig. 2. Microsporum nanum. Specimens from veal-infusion agar showing macroconidia. $(\times 350)$