to *E. histolytica* [6–9]. Nevertheless in chronic alcoholics some authors [10, 11] have observed a considerable modification in the regulation process of the T and B lymphocyte function. In addition, in alcoholics the skin presents atrophy of the epidermis and of the sebaceous glands. Such modifications can cause a notable weakening of the cutaneous barrier layer which, together with the depression of the cell-mediated immunity, facilitates the implant of parasites on the epidermic surface and subsequent colonization. In conclusion, we think this case of cutaneous amebiasis in an immunodeficient alcoholic should be considered as an opportunistic infection.

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Dermatology 1997;194:371

Serum Antibodies to Parvovirus B19 in Patients with Pityriasis rosea

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Parvovirus B19 is a DNA virus of the Parvoviridae family, of which it is the only one considered to be a human pathogen [1]. It has been shown in recent years to be the causative agent of erythema infectiosum [2]. More recently, it has also been implicated as a cause of papular-purpuric (petechial) gloves-and-socks syndrome [3, 4].

The cause of pityriasis rosea is still unknown although a viral etiology is strongly suspected [5]. To the best of our knowledge, the role of parvovirus B19 as a possible causative agent of pityriasis rosea has not been examined yet. We report herein the results of serological tests for this virus in 13 patients with pityriasis rosea.

Thirteen patients with classical pityriasis rosea, 7 females and 8 males, aged 17–25 years (mean 23.3), were included in the study. Blood samples were collected 7–30 days (mean 13 days) after the appearance of the rash (including the herald patch). The presence of IgG and IgM antibodies directed against parvovirus B19 was studied using a standard IBL ELISA kit (Kurzarbeitsanleitung, Hamburg, Germany). IgG antibodies were found in 5 (38%) patients. IgM antibodies were not detected in any of the 13 patients.

The percentage of pityriasis rosea patients with positive IgG antibodies against parvovirus B19 is close to the reported prevalence of 40–60% of this antibody in the general population [1]. IgM antibodies to parvovirus B19 appear a few days after infection with this virus [1]. Therefore, the absence of IgM antibodies, which reflect recent infection, in all of our patients does not lend support to a possible role of parvovirus B19 in the pathogenesis of pityriasis rosea.

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Dermatology 1997;194:371–373

The Petrified Ear – A Manifestation of Dystrophic Calcification

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Key Words
Petrified ear · Dystrophic calcification · Diabetes mellitus

The petrified ear (PE) is a rare condition in which the ears become stone-hard [1–4]. The real aetiology is still unknown, but many pathogenetic factors are reported in the literature (table 1). We report a case in which the only demonstrable cause was diabetes. Dystrophic damage of the cartilage following diabetic micro-angiopathy might be one of the pathogenetic stimuli of this rare entity.

A 75-year-old male who worked as a guard until he was 60, presented with an ulcerated plaque localized on his left ear. The lesion

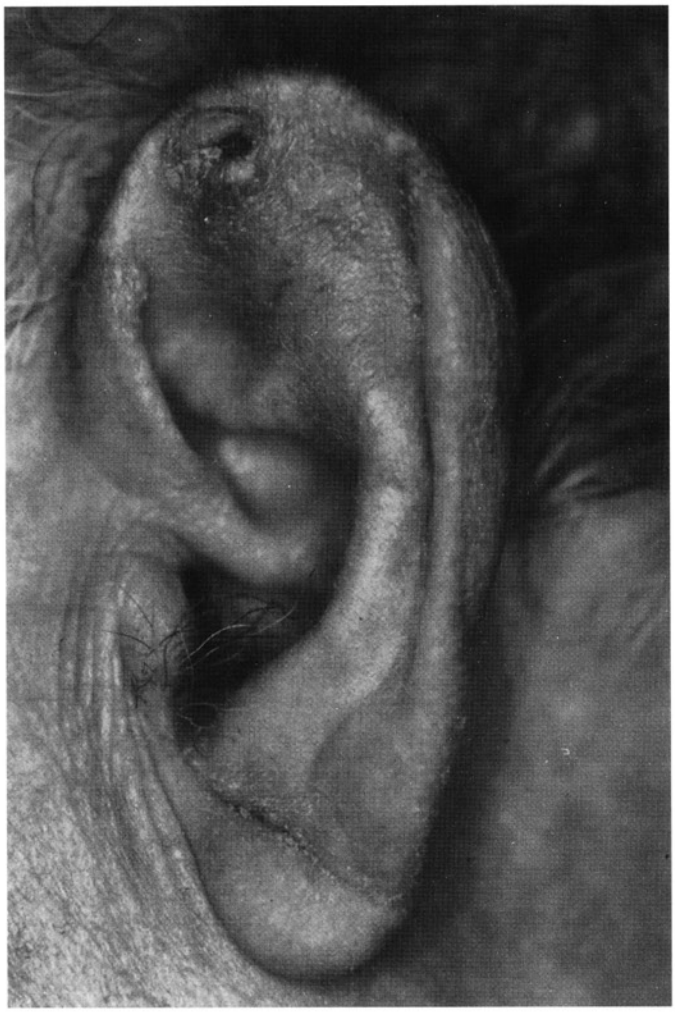

Fig. 1. Ulcerated plaque localized on the patient's left ear. The lesion was about 1 cm large and asymptomatic but, on pressure, emitted a whitish creamy material.

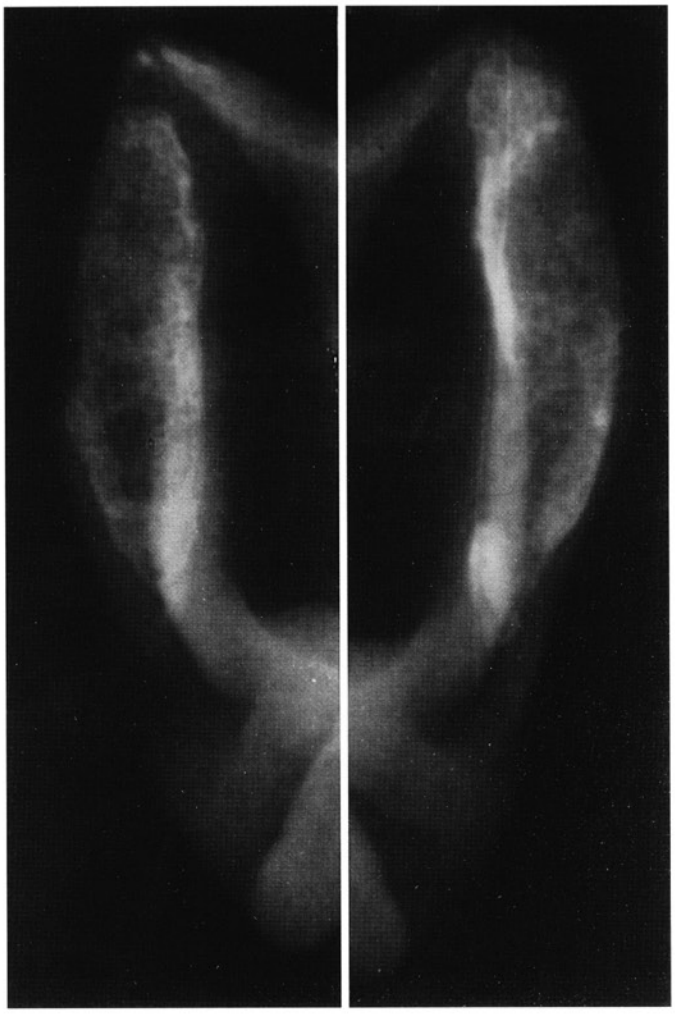

Fig. 2. Bilateral track-like calcification of the cartilage of the pinna.

was about 1 cm large and asymptomatic but, on pressure, emitted a whitish creamy material. Over the last 2 years the patient had noticed a slow progressive induration of his auricles. On examination, the cartilaginous portions of both external ears were stone-hard to touch, but the general configuration of the auricle was more or less normal (fig. 1). The patient was otherwise in apparent good health and his history was negative for local and systemic diseases. He had no recollection of episodes of frostbite, actinic burns and other trauma to his auricles. Laboratory studies revealed an elevated blood glucose level to 248 mg/dl (normal 70–120 mg/dl and an elevated urine glucose level to 1,000 mg/dl (normal 0.0–15 mg/dl); serum calcium, serum phosphorus, parathormone and vitamin D_3 were within the normal limits. Ophthalmoscopic examination showed a micro-aneurysm and angiosclerosis. A biopsy specimen of the right ear revealed calcification of the cartilaginous tissue. A radiograph of the external ears showed bilateral track-like calcification of the cartilage of the pinna (fig. 2). Vascular calcifications were detected in the radial artery in its tract crossing the anatomical snuff-box. Calcifications of the synovial membrane and the articular second metacarpal capsule were also present.

Clinical apparent calcification of the auricular cartilage is a rare event. The incidence in the general population is unknown; in a series of 300 patients, Gordon [4] found 3% with radiological evidence of calcification of ear cartilage. Most patients are asymptomatic and the real prevalence of this entity may be underestimated.

Inflexibility of the ear during manipulation is the most common symptom. If the ear canal is involved, external otalgia is, at times, a common complaint. Ulceration is infrequent. In our patient, an ulcerated plaque which emitted a whitish creamy material was the earliest symptom. The calcification of PE is usually considered to be a dystrophic calcification as a consequence of a tissue injury; calcium and phosphate metabolism and serum levels are normal.

Many pathogenetic hypotheses have been elaborated in order to explain the development of PE. Some local factors such as actinic damage, frostbite, mechanical trauma, inflammatory diseases or radiation therapy may induce trophic damage to cartilage and a resulting dystrophic calcification. The history of our patient allowed us to exclude almost all the causes of PE reported in table 1 with the exception of diabetes. Diabetes mellitus is in fact included among the systemic causes of PE, but the

Table 1. Aetiology of auricular calcifications as reported in the literature

	Local factors	Systemic disorders
Clinical appearance	more localized calcification	more extensive and more often bilateral calcification
Aetiology	frostbite trauma inflammatory diseases radiation therapy actinic damage	Addison disease hypopituitarism ochronosis diabetes mellitus hyperthyroidism pseudohypoparathyroidism sarcoidosis gout collagen vascular diseases perichondritis syphilitic chondritis familial cold hypersensitivity senility acromegaly

exact mechanism by which the disease can induce calcification is unknown. We believe that diabetic micro-angiopathy may be responsible for a dystrophic damage of the cartilage. In our patient, micro-angiopathy was demonstrated by ophthalmoscopic examination. Moreover, ring-like vascular calcifications were present in the radial artery in its tract crossing the anatomical snuff-box and in the synovial membranes. It is well known that a correlation exists between calcifications and long-term diabetes. Ring-like arterial calcifications are characteristic of diabetic diseases and are due to neuropathy which induces degenerative alterations of smooth muscle fibres of the vessel walls with consequent calcification.

Therefore, we believe that in our patient diabetic micro-angiopathy could have represented the main cause of dystrophic calcification of the auricular cartilage. However, we believe that PE cannot be considered a reliable marker of diabetes because we cannot exclude with certainty some other causes such as chronic actinic damage, frostbite or slight trauma; moreover, diabetes is a rather common disease while PE is a rare manifestation. The association of PE with diabetes could therefore be a coincidence. PE is an asymptomatic condition and it could be more frequent in diabetics than is generally believed, particularly in people with important micro-angiopathy and neuropathy with vessel calcifications. More studies in large populations of diabetics are required to establish the prevalence of PE.

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Dermatology 1997;194:373–374

Acquired Acromelanosis due to Phenytoin

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Key Words
Acromelanosis, acquired · Phenytoin

A 19-year-old girl presented with complaints of asymptomatic progressive darkening of fingers and toes of 3 years' duration. She was a known patient with grand mal epilepsy and had been on a combination therapy of phenobarbitone 60 mg b.d. and phenytoin 100 mg b.d. for 5 years. Phenobarbitone was however stopped after 1 year and phenytoin was continued for the next 2 years. At the completion of 2 years of therapy, she noticed gradual and progressive darkening of all fingers and toes. The pigmentation was diffuse, greyish in colour, extending up to the dorsum of the proximal phalanges, and it was more prominent over the dorsum of the interphalangeal joints and distal phalanges (fig. 1). As the patient did not have any seizures, the anticonvulsant was stopped at the end of 3 years. This was followed by gradual but incomplete regression of pigmentation without any treatment. After 1 year of discontinuation of therapy, seizures recurred and she was again put on phenytoin at the same dose, i.e. 100 mg b.d. Pigmentation re-appeared within 3 months of therapy. No history of any other drug intake was available and no other member of the family had a similar pigmentation. The nails were normal and showed no pigmentation. Examination of mucosae, hair and other parts of the skin revealed no hyperpigmentation. No other side effects of phenytoin were observed. She was advised to discontinue phenytoin, and alternative treatment with carbamazepine 100 mg t.i.d was started. In 2 months of follow-up, pigmentation was noted to be regressing without any specific treatment.

Discussion

The acromelanosis in the present patient was considered to be acquired due to phenytoin therapy. Although she had received pheno-

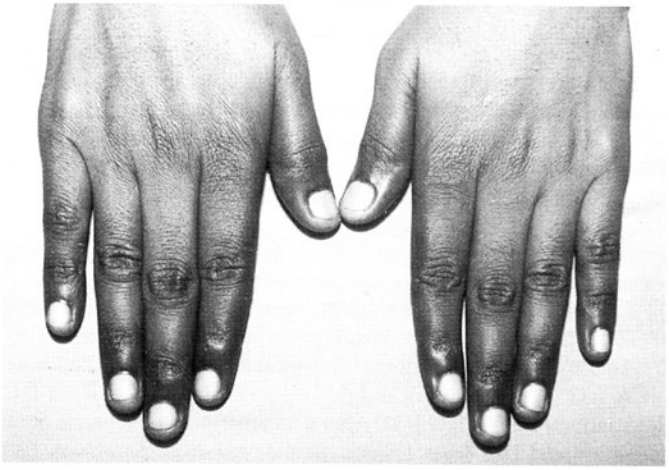

Fig. 1. Diffuse pigmentation over the dorsum of fingers.