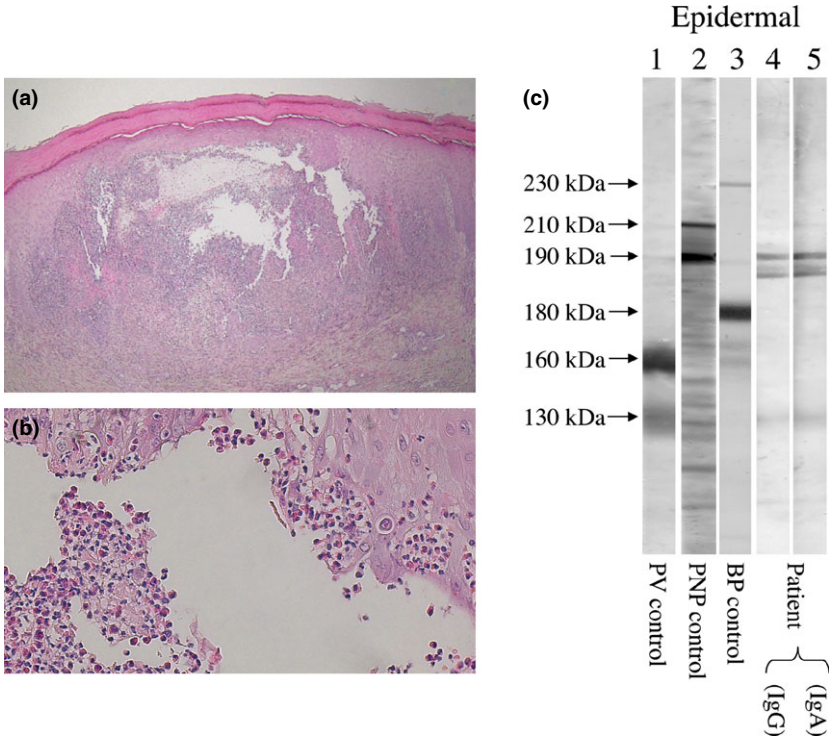

Figure 2 Histopathological findings and the result of immunoblotting. (a, b) Skin biopsy from the pustules on the patient's left foot, showing an intra-epidermal eosinophilic abscess and eosinophilic infiltration of the upper dermis and perivascular areas, with few infiltrating neutrophils [original magnifications: ×40 (a), ×200 (b)]. (c) Immunoblotting of normal human epidermal extract. A pemphigus vulgaris (PV) control serum reacted with the 160 kDa Dsg1 and 130 kDa Dsg3 (lane 1), a paraneoplastic pemphigus (PNP) control serum reacted with the 210 kDa envoplakin and the 190 kDa periplakin (lane 2), and a bullous pemphigoid (BP) control serum reacted with the 230 kDa BP230 and the 180 kDa BP180 (lane 3). IgG and IgA antibodies reacted with the 130 kDa Dsg3 as well as the 190 kDa periplakin (lanes 4 and 5 respectively).

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Dermatitis by Tropical Rat Mite, *Ornithonyssus bacoti* (Mesostigmata, Macronyssidae) in Italian city-dwellers: a diagnostic challenge

Editor

Mesostigmata (Gamasida) is a suborder of mites within the order Parasitiformes.[1] Some species can bite humans, typically causing pruritic, non-specific skin lesions. The mesostigmatic mites most commonly associated with cases of dermatitis are from the Macronyssidae and Dermanyssidae families.[1] All are haematophagous non-burrowing mites of very similar size and shape, and can be seen with the naked eyed as they range from 0.5 to 1.5 mm in size.[1] Adults are able to survive several weeks without a blood meal.[1] The tropical rat mite, *Ornithonyssus bacoti*, Hirst (Macronyssidae) is one of these species. It can occur worldwide mostly as an ectoparasite of wild rats and mice,[2,3] but also pet and laboratory rodents may occasionally be the source of the mite.[2,4–6] As a nest parasite, it only visits its host to feed, mainly during the night; and then returns to its nearby hiding place during the day. In the absence of its natural host, human beings may become subject to mite infestation. Outbreaks of tropical rat mite (TRM) dermatitis have been reported in humans in urban areas in Europe, because of wild/pet rodent infestation of flats[2–4] but also due to occupational exposure,[5]

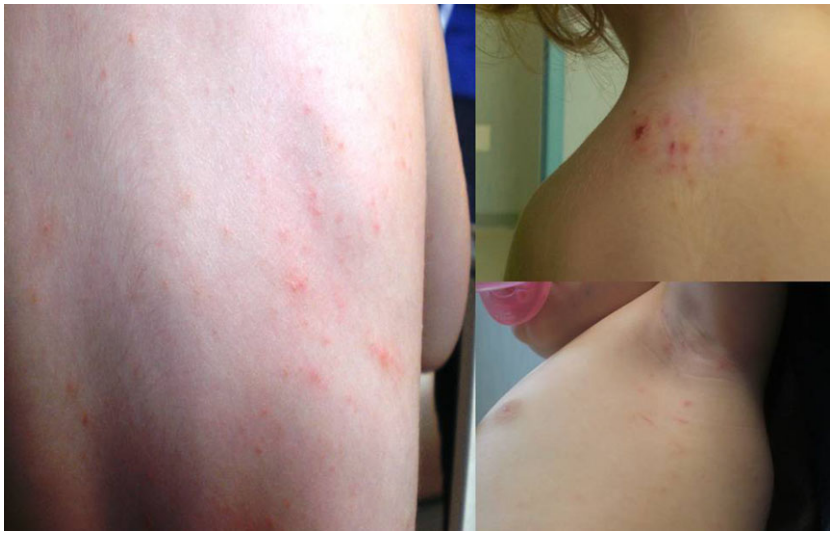

Figure 1 2-year-old girl: skin injuries caused by bites of the Tropical Rat Mite, *Ornithonyssus bacoti.*

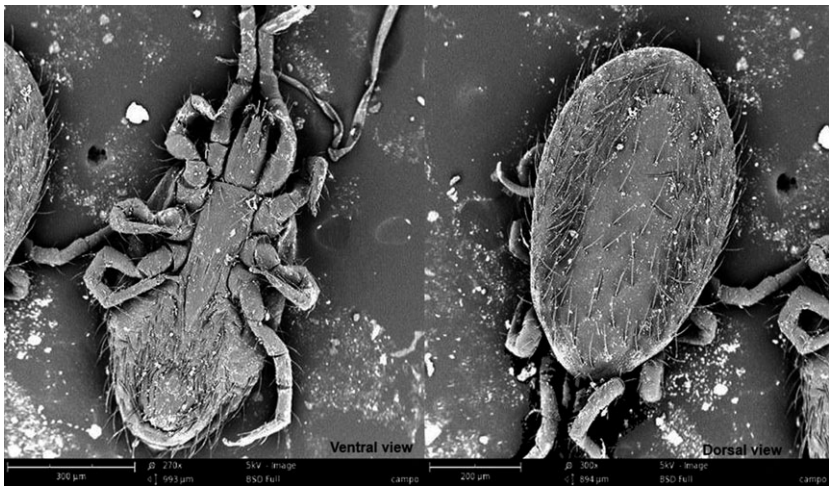

Figure 2 *Ornithonyssus bacoti* (tropical rat mite), female: ventral and dorsal view (Scanning Electronic Microscope, Phenom World).

including a single record from Italy.[6] These scattered cases show that parasitization by the TRM is not obvious and skin lesions are usually misdiagnosed, unless the mite is discovered and correctly identified. To confirm this, we report an urban outbreak of TRM dermatitis diagnosed by identifying the parasite. In November 2013, veterinary entomologists from the Zooprofilactic Institute (IZSPB), Foggia were contacted to inspect a house for mites. Owner referred that her 2-year-old daughter suffered from multiple erythematous papules over a 9-month period. Cutaneous lesions were generalized but predominantly located in groups on the neck and body areas covered by clothes (trunk, upper extremities and abdomen) (Fig. 1). Her 71-year-old grandmother had also experienced the same symptoms after spending a single night in the child's home. Bites by arthropods/non-burrowing mites/wood-mite and *Streptococcus spp.* infection respectively, were diagnosed after two paediatric and two dermatological consultations. Symptomatic relief was also unsuccessful. Mites were detected in dust samples and in child's bedding, after careful investigation of the patient's living quarters by IZSPB staff. All of the parasites were identified as *Ornithonyss bacoti* based on morphological keys[1] (Fig. 2). The source of the mites was a previously eliminated rats' nest in the living-room. Medical texts and parasitology manuals rarely mention mesostigmatic mites in association with human parasitization; detection and correct identification of these ectoparasites is also difficult. This makes it a challenge for physicians, including dermatologists, to recognize epizoonotic mite-associated dermatitis, including skin disorders caused by *O. bacoti*. This inevitably leads to failures in treatment. In addition to *O. bacoti,* mestostigmatic avian mites such as *Ornithonyssus sylviarum* and *Ornithonyssus bursa* (Macronyssidae), and more frequently *Dermanyssus gallinae* (Dermanyssidae) can be associated with urban

dermatitis in Europe,[2,7] including Italy.[8,9] When unexplained pseudoscabious eruptions occur on patients in urban areas, investigation for mesostigmatic mites should always be undertaken. If small rodents are held as pets, animals and their bedding should be carefully examined by a veterinarian. Diagnosis and treatment of these infestations require accurate anamnesis, detection and identification of the parasite, removal of the source and disinfestation. As studies suggest that the rat mite may be a reservoir of various zoonotic pathogens and a vector of *Bartonella henselae,*[10] its early diagnosis remains crucial. Dermatitis by mesostigmatic mites, including the TRM dermatitis, are without doubt more frequent than one might expect. Increased physicians awareness of the possible role of these ectoparasites in urban infestations of humans, and applying the One Health approach will be essential for diagnosis, treatment and prevention.

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A novel mutation in the FERMT1 gene in Turkish siblings with Kindler syndrome

Editor

Kindler syndrome (KS) is a rare autosomal recessive genodermatosis, was first reported in 1954 by Theresa Kindler.[1] It is clinically characterized by the presence of minor trauma-induced blisters usually arising within the first days of life. Photosensitivity, poikiloderma and diffuse cutaneous atrophy with the characteristic cigarette paper-like wrinkled skin tend to appear.

Kindliness are evolutionarily conserved FERM domain-containing proteins; which have recently emerged as key regulators of integrin activation.[2] Among them, Kindlin-1 is expressed in epithelial cells, predominantly in the skin, the intestine and the kidney and loss-of-function mutations in the gene coding for kindlin, *FERMT1* cause the Kindler syndrome.

A 2-year-old Turkish boy was admitted to our outpatient clinic with repeatedly trauma-induced blisters on the hands and knees. His mother also noticed photosensitivity. This condition started in the first year after birth. His parents were not consanguineous. He had four siblings, of which only one sister had similar symptoms. Physical examination revealed hypo-hyperpigmentation on the face and severe skin atrophy and poikiloderma on his hands and knees (Fig. 1a,b). Acral features included parchment-like skin atrophy, onycolysis, subungual hyperkeratosis. There was no involvement of the oral mucosa. Ocular examination revealed hypertelorism.

The second patient is a 7-year-old girl, the sister of the first case. She had blisters on her face and neck since she was 1 year old. She also suffered from photosensitivity. Skin examination showed xerosis, skin atrophy on her hands, poikiloderma on her face (Fig. 1c,d) and reticular pigmentation on the neck. On her ocular examination she had also hypertelorism. Examination of the oral cavity showed poor preservation of teeth and severe periodontitis, with pseudomembranous necrotic and bleeding areas.

A skin biopsy and blood tests were obtained from both patients. Histopathological examinations of cutaneous biopsies showed minimal epidermal atrophy, dilatation of blood vessels in the upper dermis with mononuclear infiltration, focal vacuolar degeneration of basal layer with subepidermal cleft, and presence of pigmentary incontinence.

The genetic analysis for our patient was performed by the Institute of Human Genetics of Freiburg. Mutational analysis of all coding regions and exon–intron boundaries of the *FERMT1* gene was performed as described previously.[3] DNA sequences were compared to the reference sequences from the NCBI Entrez Nucleotide database (NM_017671.4; NC_000020.11). We detected the mutation c.1140-2A>T in a homozygous state (Fig. 2). We diagnosed the patients as having KS.