

Indoor dermatitis due to *Aeroglyphus robustus*

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DEAR EDITOR, *Aeroglyphus robustus* is an astigmatid mite described initially by Banks in 1906 and called *Glycyphagus robustus*.¹ In 1941 Zachvatkin described and called the subfamily *Aeroglyphinae* and the genus *Aeroglyphus*. He considered this mite of Canadian origin to be the same as the European species *A. peregrinans* (Berlese, 1892),² and he upgraded the subfamily *Aeroglyphinae* to family *Aeroglyphidae*. It was only in 1959 that Cooreman described *A. robustus* in detail, distinguishing it morphologically from *A. peregrinans*.³ At present, in the Italian acarofauna, *A. robustus* is not present in the family *Aeroglyphidae*,⁴ and its pathogenic role in humans has never been described before. The taxonomy of *A. robustus* is order Astigmata, suborder Acaridia, superfamily Glycyphagoidea, family Aeroglyphidae.

A 57-year-old nonatopic healthy man was referred for an itchy rash with small and isolated erythematous papules, some with pinhead-shaped central tense vesicles and some excoriated and covered by small crusts. The lesions initially involved the extensor surface of the forearms and the sides of the trunk (Fig. 1); after 1 week they spread to the upper and lower limbs. The patient's wife showed similar skin lesions. They had no other systemic symptoms, and physical examination was otherwise unremarkable. The patient was a farmer, and 1.5 months earlier he had brought home two large bags of grain, cramming them in at the entrance. The grain, of unknown origin, had been collected from a container located near the patient's house.

The patient and his wife were treated with oral cetirizine (10 mg per day) and desoximetason ointment, with partial reduction of itching and skin lesions within 10 days. Domestic ectoparasitosis was suspected. In order to verify it, sawdust collected from all of the rooms and the grain bags was exam-

ined with E.D.P.A.[®] (Esame Diretto Polveri Ambientali), patented by one of the authors.⁵ Briefly, dust samples were collected by broom and dustpan from the floor of the home's rooms and labelled in small containers. After microsieving, sedimentation or flotation, pathogen arthropods and their specific traces (fragments, exuviae, bristles, faeces) were observed with a stereomicroscope and prepared and mounted on slides in warm Berlese solution for direct observation by optical microscope.

The dust sample collected from the grain bags was twice as rich in weeds/pests as those collected from the rooms of the house. However, in both samples *A. robustus* was identified, with several adults (Fig. 2a) and immature forms (proto-nymphs, nymphs, larvae). *Aeroglyphus robustus* is morphologically characterized by dorsally tuberculate idiosoma covered by numerous idiosomal multibranching bristles shaped like wheat plugs and, in particular, by two very long opisthosomal bristles in the female (Fig. 2b).^{3,6,7} The so-called hypopial deutonymphs of *A. robustus* were also identified (Fig. 2c); they are typical forms of resistance to untoward environmental conditions. Inspection of the house showed the presence of moulds of the genera *Penicillium* and *Aspergillus*, in a high relative humidity (85%), on some north-facing walls.

The patient tried to eliminate the house infestation using an aerosol automatic spray insecticide based on cyfluthrin. This approach turned out to be ineffective because the mites hide where the insecticide does not reach them and because of the presence of the resistant form (hypopial deutonymph). In fact, a new E.D.P.A. examination showed vital adults and immature forms of *A. robustus*. Intervention by a pest-control company was then required and the domestic environment was fumigated with cyphenothrin. Immediately the patients' skin lesions improved, but after 2 weeks similar new skin lesions in the farmer and his wife appeared again. Probably, hypopial

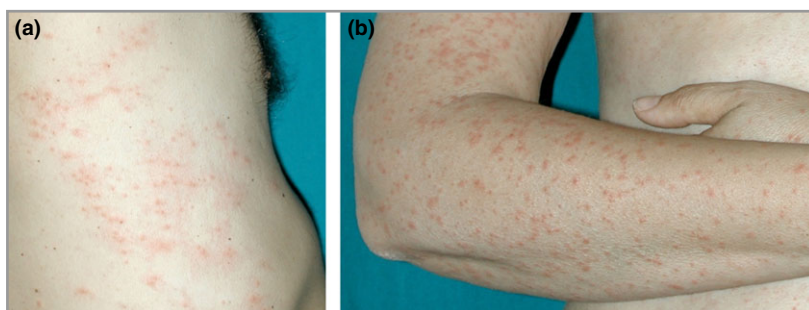


Fig 1. Erythematous papular dermatitis with some central tense vesicles and crusts on the side of the trunk (a) and the extensor surface of the forearm (b).

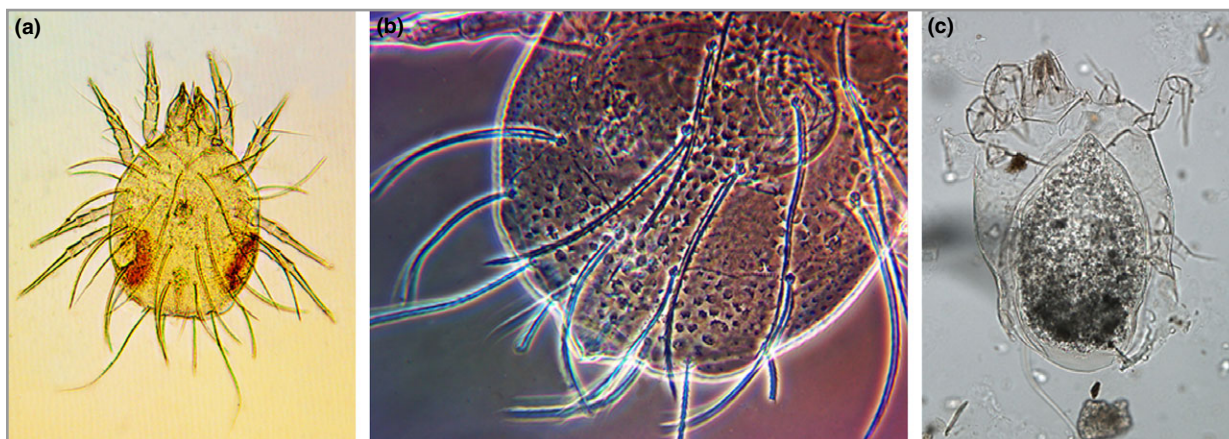


Fig 2. *Aeroglyphus robustus*. (a) Adult male (original magnification $\times 100$), (b) opisthosoma with typical tubercles and multibranch bristles (contrast phase, original magnification $\times 400$) and (c) hypopial deutonymphs with hypopus (original magnification $\times 100$).

deutonymphs restored the mite population, resulting in a new infestation. A second fumigation with cyphenothrin resolved the domestic infestation, as shown by the resolution of skin lesions and by the negativity of a further E.D.P.A. study.

Once the arthropod responsible for the dermatitis was identified through E.D.P.A., it was necessary to clean up the moulds on the house walls with sodium hypochlorite, as these mites are mycophages, and to reduce the environmental relative humidity with an electric dehumidifier.

Aeroglyphus robustus ($330 \times 220 \mu\text{m}$) is a mycophagous astigmatid mite living predominantly on battered and poorly preserved flour products, where moulds develop. It is called 'warty grain mite' because it is known as a pest of stored grain.¹ The life cycle comprises the stages of egg, larva, protonymph, deutonymph and adult, and lasts about 20 days. Environmental conditions that favour its development in the domestic environment are high temperatures ($28\text{--}30^\circ\text{C}$) and high relative humidity ($85\text{--}90\%$);⁸ moulds on the walls are the primary trophic site of *A. robustus*. Currently we cannot assume its role as a vector, but only as a bacterial or fungal reservoir.

Aeroglyphus robustus is an uncommon mite, never recorded before in Italy, probably because of the difficulty in identification of this species. The pathogenetic mechanism underlying skin lesions due to *A. robustus* is not yet known. It is probably related to the multibranch bristles that pierce the skin as foreign bodies and contain irritants, similarly to the better-known *Glycyphagus domesticus* and *Lepidoglyphus destructor*.⁹ However, the pathogenicity of *A. robustus* to humans is probably due not only to these bristles, but also to the irritant contact with exuvial liquids and faeces issued mainly by the larvae and protonymphs.

Exposure to pyrethroid insecticides by fumigation, eradication of wall moulds and restoration of normal domestic humidity are the three key points to eradicate *A. robustus* from the domestic environment. After these measures are taken, thorough cleaning of the house by careful and repeated dust vacuuming is crucial to remove the numerous bristles and other organic fragments still present in the domestic environ-

ment. In fact these continue to be skin irritants even after the mites are killed.

This report is the first case of human dermatitis caused by *A. robustus* and the first evidence of this mite in Italy, although it is possible that dermatitis due to this mite may go undiagnosed or be ascribed to other mites.

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Acneiform eruptions caused by various second-generation tyrosine kinase inhibitors in patients with chronic myeloid leukaemia

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DEAR EDITOR, Tyrosine kinase inhibitors (TKIs) are a novel, well-tolerated and promising class of anticancer drugs, commonly used to treat chronic myeloid leukaemia (CML). Imatinib was the first commercially available TKI, approved for the treatment of CML and gastrointestinal stromal tumours. The development of polyclonal resistance to imatinib, due to point mutations in the bcr-abl kinase domain, yielded second-generation TKIs including dasatinib, radotinib and nilotinib.¹ These second-generation TKIs have shown higher rates of early optimal responses and fewer side-effects than imatinib.¹

These new agents are indeed less toxic, but their range of activity is still not directed at tumour cells only. As a result, their use is associated with various systemic adverse events that go beyond the scope of the present review, but cutaneous side-effects are the most commonly reported.^{1,2} Nevertheless, there have been only a few reports of cutaneous adverse events caused by TKIs in patients with CML. To the best of our knowledge, this is the first report directly focused on acneiform eruptions due to second-generation TKIs in patients with CML. Herein, we report 10 cases of acneiform eruptions with or without rosacea-like eruptions caused by various second-generation TKIs in patients with CML.

We studied 10 patients with CML treated with second-generation TKIs who were seen between November 2013 and May 2015. Patients with CML seen at the haematology department who presented with acneiform eruptions or rosacea-like eruptions were referred to our dermatology clinic (Fig. 1). Second-generation TKIs used for treatment included radotinib ($n = 4$), dasatinib ($n = 4$) and nilotinib ($n = 2$). No other drug was suspected to initiate these eruptions. Patients were interviewed and examined by a single dermatologist, and all diagnoses were made clinically. The characteristics of the patients with CML with skin lesions, and their treatment outcomes are described in Table 1.

The pathophysiology of cutaneous eruptions caused by TKIs is unclear. It has been reported that the TKI-induced rosacea-like eruptions may be associated with a dermal matrix abnor-

malty and T-cell suppression caused by TKIs.^{2,3} TKIs inhibit the tyrosine kinase activities of bcr-abl and platelet-derived growth factor receptor, blocking collagen synthesis and dermal matrix formation.^{2,3} Loosened connective tissue support for cutaneous vessels may result in the recruitment of inflammatory mediators and metabolites, causing symptoms of rosacea such as telangiectasia, erythema, flushing and oedema. In addition, although we could not determine the factors that triggered the acneiform eruptions in our patients, it is suggested by recent studies that TKIs, like epidermal growth factor receptor (EGFR) inhibitors, induce alterations in follicular and epidermal cell homeostasis.^{2,4} However, in our experience there was a difference in the clinical presentation of acneiform eruptions depending on whether they were caused by TKIs or EGFR inhibitors.

In EGFR inhibitor-induced acneiform eruptions, the lesions tend to be more follicular and the distribution of the lesions tends to be along the lateral borderline of the face and on the scalp, rather than on the face itself. In contrast, the lesions of TKI-induced acneiform eruptions are more consistent with the

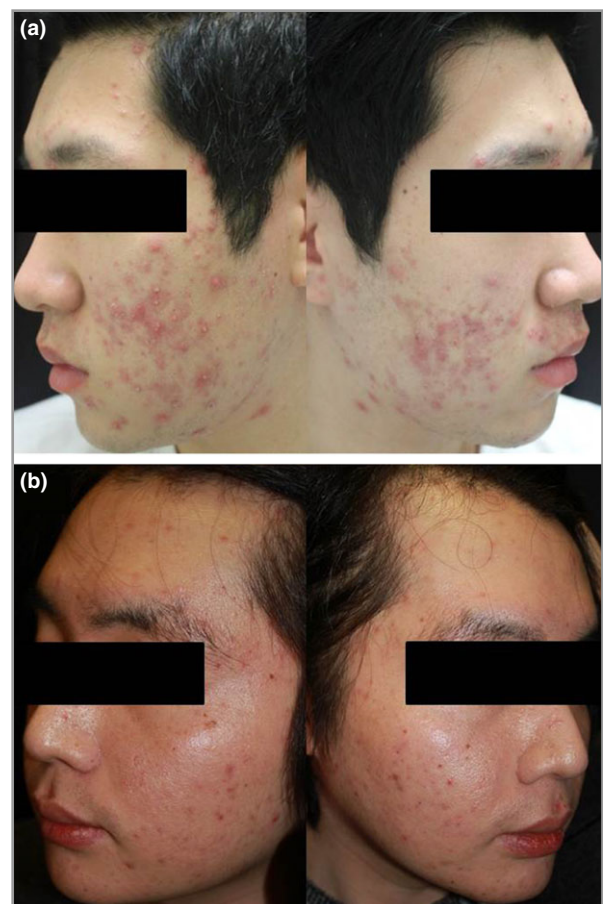


Fig 1. Clinical pictures of two patients enrolled in the study. (a) Patient no. 5 with nodular, pustular lesions of acneiform eruption (grade 6). (b) Patient no. 9 with papular, pustular lesions of acneiform eruption (grade 3) concomitant with a rosacea-like eruption.