Carpenter's pigmentation mimicking ashy dermatosis associated with African padauk wood dust

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Key words: African padauk; ashy dermatosis; carpenter; patch test; pigmentation.

INTRODUCTION

Various adverse cutaneous reactions may occur as a result of exposure to wood dust or exotic woods. These reactions include allergic contact dermatitis, irritant contact dermatitis, urticaria, photoallergic and phototoxic reactions, and multiformelike reactions. Exposure is also associated with nasal, eye, and respiratory symptoms.

Padauk is a timber that belongs to pantropical genus *Pterocarpus*. African padauk is used for furniture, carvings, and musical instruments. Cases of allergic contact dermatitis caused by padauk have been reported.[1,2] To our knowledge, the dyspigmentation associated with padauk wood dust has never been reported.

CASE REPORT

A 37-year-old male carpenter had generalized melanosis for 11 years. Physical examination found reticulate pigmentation characterized by bilateral, symmetrical, gray-brown patchy or mottled pigmentation on the face, neck, nape, arms, forearms, armpits, breasts, abdomen, and back. The patches were most pronounced on the face and neck (Fig 1, *A* and *B*).There was no atrophy, erythema, or telangiectasia. Palms, nails, teeth, gingiva, mucosae, and areas below the waist were normal. He denied any systematic symptoms.

For the last 11 years, he has been employed as a carpenter, cutting, sanding, and gluing wood. Generally, he worked with African padauk, Russian *Pinus sylvestris*, and latex. The workplace was indoors with limited ventilation. No protective equipment except for gloves were used in the winter. Starting from the left side of the forehead, the pigmentation proceeded to the other sites. The patient denied significant medical history and any events related to the pigmentation. No history of contact or ingestion of heavy metals was obtained. No similar case was noted at the working site.

Results of laboratory investigations including blood and urine examination, hepatic and renal profile, serum electrolytes, cortisol hormone, immunology profile (including IgE), and thyroid profile were normal. Blood glucose testing found increased fasting plasma glucose, postprandial plasma glucose (1 hour, 2 hours, and 3 hours), postprandial insulin (3 hours), and postprandial C-peptide (2 hours). Fasting C-peptide and glycosylated hemoglobin levels were normal. Radiographs of the chest and electrocardiogram were normal.

Skin biopsy of a hyperpigmented patch behind the right ear found scattered dyskeratotic cells in the prickle layer, increased melanocytes in the basal layer, and medium perivascular melanophages and lymphocytic infiltration in the dermis. Liquefaction of basal layer was observed (Fig 2).

Patch tests were performed with the suspected wood dusts and materials from the workplace, including African padauk wood dust, Russian *P sylvestris* wood dust, and latex. The patch tests were applied to the upper left side of the back and were occluded for 2 days. Readings were done on day 2, day 3 (Fig 3), and day 4. Positive patch tests were observed with African padauk wood dust (positive reaction at day 2, day 3, and day 4).

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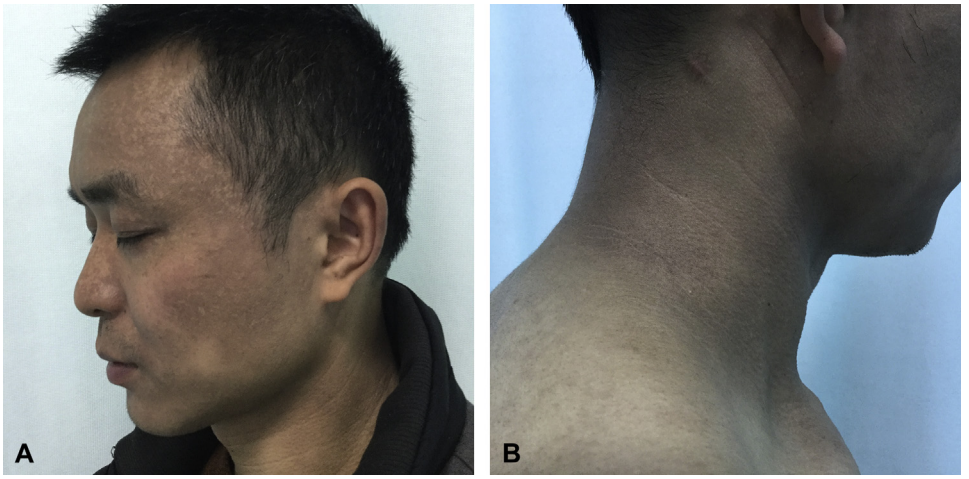

Fig 1. **A** and **B**, Hyperpigmentation on face, neck, nape, and shoulder.

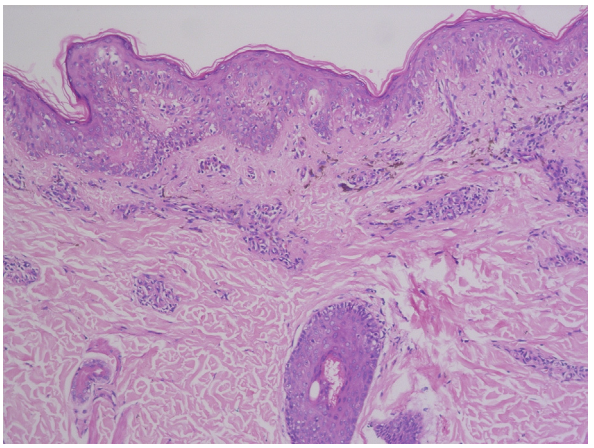

Fig 2. Scattered dyskeratotic cells in prickle layer, increased melanocytes in basal layer, perivascular melanophages and lymphocytic infiltration in the dermis, and liquefaction of basal layer. (Hematoxylin-eosin stain; original magnification: ×100.)

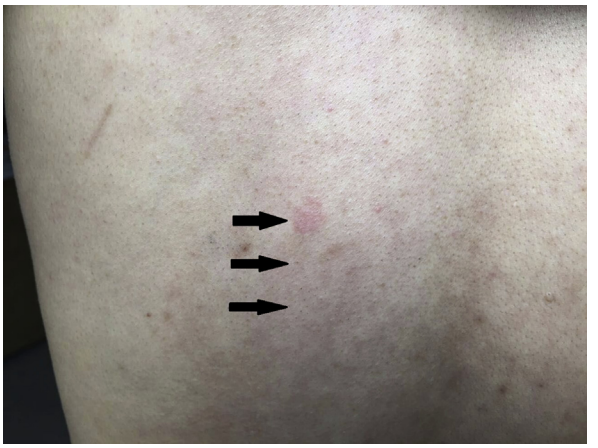

Fig 3. Patch test results, D3. From top to bottom: African padauk wood dust, Russian *Pinus sylvestris* wood dust, latex.

The patient was treated with oral vitamin C, vitamin E, compounded glycyrrhizin, and topical retinoids. The patient was instructed to avoid padauk wood dust and sun exposure. Meanwhile, he was treated with gliclazide and miglitol for 1 month. He then switched to diet control according to the endocrinologist's advice. No significant improvement on the dyspigmentation was noted after the first 6 months. However, a significant improvement was noticed on the face, neck, nape, breasts, abdomen, and back on the patient's last visit. By the last visit, he had been taking vitamin C and vitamin E for 15 months, compound glycyrrhizin for 8 months, and topical retinoid on the face for 12 months. The pigmentation on the arms and forearms did not improve well. The patient did not apply the topical retinoid on the sites other than face and did not avoid African padauk wood dust completely. The regimen was then adapted to oral vitamin C and topical retinoid on all dyspigmented sites for another 6 months.

DISCUSSION

Woods are capable of causing allergic or irritant contact dermatitis, which typically occurs on the exposed areas. The allergens found in woods include quinones, stilbenes, phenols, and terpenes.[3] Woods of tropical origin are more sensitizing, as they contain more quinones. Some cases of asthma and rhinitis have measured IgE to specific species of wood, suggesting a type I hypersensitivity reaction, whereas type IV hypersensitivity has been suggested to play a role in occupational dermatitis. Another mechanism includes individual risk factors such as susceptibility and sensitization to agents. Also, there

might be a role for subclinical injury or inflammation for the development of pigmentation.

For this case, the clinical and pathology correlation was suggestive of 2 conditions, erythema dyschromicum perstans/ashy dermatosis or pigmented contact dermatitis.[4] These 2 conditions do have overlapping clinical features. Both have a potential allergic etiology. Clinically, both have generalized hyperpigmentation at the late stage of the disease. Histologically, both have melanin incontinence, melanophages in the papillary dermis, and lichenoid lymphocytic infiltration. Dermatitis of little or no sign led to hyperpigmentation by repeated contact with very small amounts of the contact sensitizer in the occupational exposure. Fine dust collects on and in clothing, and the settling inside clothing might explain effects on anatomically shaded skin.

The histopathology findings presented could be potentially interpreted as an interface dermatitis with pigment incontinence. Basal cell liquefaction and melanophages in the dermis might be provoked by accumulation of small amounts of allergen producing type IV allergic cytolytic reaction. Basal liquefaction was regarded as a major histologic feature resulting in melanin dropping from cytolysis of epidermal basal cells. Perivascular melanophages and lymphocytic infiltration could be suggestive of a lichenoid allergic reaction rather than a toxic reaction. The positive results of patch tests confirmed the contact sensitization and implied a potential etiology associated with African padauk wood dust.

Cutaneous manifestations of hyperglycemia or diabetes are classified into 4 categories: infections, diseases directly associated with diabetes such as diabetic bullae, manifestations of complications such as microangiopathy, and reactions to diabetic treatment such as insulin.[5] The patient's blood glucose was normal after 1 month of treatment, and we did not consider the patient's melanosis to be associated with the hyperglycemia and insulin/C-peptide profile.

The oral medication and topical retinoids proved to be effective after a long treatment course. Complete avoidance of the suspected contact sensitizer might be beneficial in curing the hyperpigmentation.

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