Inflammatory sebotropic reaction associated with kava kava ingestion

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Key words: histopathology; kava kava; natural medicine; nutritional supplements; sebotropic.

INTRODUCTION

Kava kava or Piper methysticum is a plant native to the Pacific Islands, traditionally valued for its relaxant properties and role as a ceremonial beverage.1 It has gained worldwide use as an alternative medication for anxiety, stress, and insomnia.1,2 This case report describes an unusual sebotropic eruption believed to be caused by kava ingestion.

CASE REPORT

A female patient in her 30s presented with 9 days of a burning, pruritic skin eruption that began on her dorsal forearms and progressed to include her face, chest, and back. She was febrile and had lymphadenopathy, facial edema, and myalgia. She denied arthralgia, sore throat, difficulty breathing or swallowing or mucosal changes. She had recently started a new brand of kava tea, which doubled her previous daily dosage of kavalactone but discontinued it 4 days into symptoms. Physical examination was notable for facial swelling, postauricular lymphadenopathy and erythematous papules coalescing into plaques on the face, arms, thighs, chest, abdomen, and back (Fig 1, A and B).

Laboratory results were significant for an elevated white blood cell count of 11600 cells/µL (reference range, 4500-11000 cells/µL), eosinophil count of 890 cells/µL (reference range, 0-450 µL), neutrophil count of 8900 cells/µL (reference range, 1800-7800), aspartate aminotransferase of 85 U/L (reference range, 10-30 U/L), and alanine aminotransferase of 222 U/L (reference range, 10-40 U/L). The patient had normal thyroid studies and negative blood cultures. Serology for hepatitis, cytomegalovirus, Epstein-Barr virus, and syphilis were negative.

A punch biopsy of the left shoulder found an unremarkable epidermis overlying mild folliculocentric lymphoid inflammation prominently involving the sebaceous lobules with partial necrosis of the sebaceous lobules. Neutrophils were also present within the sebaceous lobules (Fig 2). The surrounding dermis showed a scant perivascular lymphoid infiltrate. Stains for herpes simplex virus—1, herpes simplex virus—2, and varicella zoster virus were negative.

The patient was given oral prednisone, 60 mg, for 1 day and then increased to 80 mg/d for 2 days with minimal benefit. Cyclosporine, 100 mg twice daily, was added 11 days into symptoms, which led to a rapid improvement of symptoms.

DISCUSSION

The use of kava kava and other nutritional supplements has become more widespread.3 The prevalence of supplement use in the United States is estimated to be between 64% and 69%, with around 50% of those surveyed using supplements regularly.3 Despite the widespread popularity of supplements, lack of knowledge about mechanism of action and differing levels of quality control during production can lead to unintended and potentially dangerous effects. Kava supplements have been associated with elevated liver enzyme levels and questionable hepatotoxicity, along with several other side effects, including a reversible xerotic dermopathy related to heavy regular ingestion.1,4,5 Preliminary evidence from the University of Iowa Carver College of Medicine,a private practice,b and the Departments of Dermatologyc and Pathology,d University of Iowa Hospitals and Clinics.

Funding sources: None.

Conflicts of interest: None declared.

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suggests kava may inhibit multiple cytochrome P450 subtypes, which could affect the metabolism of a patient’s other medications. In addition, kava should not be used concomitantly with central nervous system depressants, such as alcohol or benzodiazepines, because of potentiation of drowsiness and the depression of motor reflexes.

The folliculocentric sebaceous inflammation seen in our patient’s biopsy was striking and was previously reported in conjunction with kava use. Jappe et al reported on 2 patients with pruritic, erythematous plaques present in a sebotropic distribution on the torso and arms that fully resolved upon discontinuation of the supplement. Histopathology in these cases similarly showed lymphocytic infiltration of sebaceous glands and destruction of sebocytes. Likewise, Huynh et al and Guro-Razuman et al also reported on patients with pruritic eruptions of the face and torso after kava supplementation, with the latter’s patient also experiencing dermatomyositis-like symptoms.

The sebotropic distribution of eruptions is thought to be caused by the lipophilic nature of kavapyrones, which make up kava extract. It is thought that the lipophilic kavapyrones concentrate in the lipids in sebaceous glands, leading to a lymphocytic inflammatory response targeting the sebaceous gland and causing a clinically visible reaction. We hope this report raises awareness of this unusual cutaneous reaction to kava and highlights the importance of asking patients about supplement use.

REFERENCES
