

Chronic palpable purpura mediated by Kiwi antigen Act c 1-induced immune complex vasculitis

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For 3 months, a 61 years old female developed recurrent palpable purpura with multiple erythematous and hyperpigmented papules and macules on the dorsum of the feet, lower- and upper legs (Fig. 1A/B). The medical history revealed autoimmune thyroiditis, treated with levothyroxin over 9 years, arterial hypertension (bisoprolol, 6 years), hyperlipidemia (pravastatin since 2 years), and coronary heart disease (acetylsalicylic acid for the last 9 years after coronary stent implantation). No history of infections, neoplasia, autoimmunity or allergic diseases.

White and red blood cell count, coagulation parameters, routine chemistry and hepatitis B/C serology were inconspicuous. Serum electrophoresis, screening for ANA and ANCA auto-antibodies and cryoglobulinaemia were negative. Also, urine and stool examination, including parasitology and guaiac, showed no pathological findings. Thus,

based on the normal clinical-, serological- and pathobiochemistry findings, a systemic vasculitis was excluded. Also, common trigger factors of allergic leucocytoclastic vasculitis, such as plasmocytoma, Hodgkins disease, rheumatoid arthritis/rheumatoid factor or complement defects were absent.

During the patients stay in our department she developed an active relapse of her palpable purpura. Detailed history was taken concerning the circumstances under which new purpuric lesions occurred and the patient reported the ingestion of fruit salads preceding the active rashes. Subsequent analysis of the ingested food preceding her last rash revealed a fruit salad containing apple, pear, banana and kiwi. To verify or rule out foodstuff as elicitor of

vasculitis, the patient was put on elimination diet and then orally challenged to 40 g of fresh fruits that she consumes regularly, including apple, banana, kiwi and pineapple in an unblinded manner. Six to ten hours after consumption of kiwi she reproducibly developed an itchy rash consisting of confluent 3–5 mm purpuric macules and papules on the legs, lower trunk and forearms with consecutive bleeding in the central part of the lesions. Oral provocations with apple, banana and pineapple were negative. Western Blot analysis of a kiwi fruit extract with the patient's serum showed IgG-, but no IgE-reactivity, corresponding to the major kiwi fruit antigen Act c 1 (Fig. 1C).

HE staining of a fresh lesion, that was taken soon after appearance,

We report on IgG-immune complex mediated vasculitis in response to Kiwi antigen Act c 1.

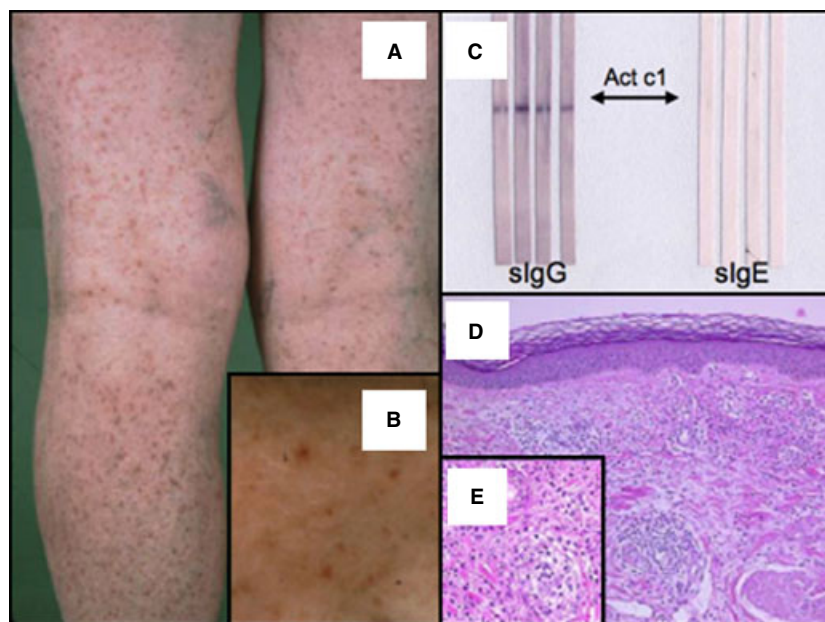


Figure 1 (A/B) Multiple erythematous macules and papules on the lower extremities. (C) Western blot of kiwi extract with patient serum. (D/E) HE staining: neutrophilic infiltration in and around cutaneous vessels with leucocytoclasia.

showed neutrophilic infiltration in and around cutaneous vessels with leucocytoclasia consistent with leucocytoclastic (allergic) vasculitis (Fig. 1D/E).

In the light of the patient's history, diagnostic findings and the reproducible induction of symptoms by oral provocation with kiwi, we diagnosed 'kiwi-induced allergic leucocytoclastic vasculitis'.

Elimination diet with avoidance of the consumption of kiwi lead to slow resolution of the purpuric lesions and hyperpigmentations over time. No recurrences of vasculitis were observed over a period of 3 years.

Using repeated oral food challenges we were able to identify kiwi fruits as a causal elicitor of allergic vasculitis. The presence of Act c 1-specific IgG strongly supports an antigen-specific process, but food-specific IgG antibodies are also frequent finding in healthy individuals (1). Foodstuff is rarely considered as a cause for allergic vasculitis in clinical practice and is reported only anecdot-

ally in current literature (rye, carrot, cow's milk, hen's egg, cocoa products and additives) (2–4). Strikingly, in our patient mainly dependent body regions like legs and feet were affected by vasculitis and therefore, the formation of IgG immune complexes with kiwi antigen and their deposition in dermal postcapillary venules can be assumed as pathogenic process (5).

The authors report no conflict of interest.

J.G., S.K., and J.R. cared for the patient. J.G. and S.K. wrote the manuscript. M.O., M.M. and J.R. reviewed the manuscript. M.O. performed the western blot.

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