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Mallory–Weiss syndrome secondary to vomiting caused by wheat-induced anaphylaxis

Recently, we have treated a case of Mallory–Weiss syndrome (MWS) secondary to vomiting caused by wheat-induced anaphylaxis. Food-induced anaphylaxis (FIA) associated with MWS has not yet been reported in the literature.

A 72-year-old Japanese man experienced several episodes of clouding of consciousness and urticaria after eating wheat products, such as noodles, pasta, bread, and pizza since 2000. The symptoms developed after eating wheat products and exercise. On July 2003, the patient ate Chinese noodles for lunch and walked home. Soon thereafter he felt discomfort, followed by vertigo, nausea, shivering, chills, general itching, wheals, dyspnea, and impaired consciousness. His blood pressure was 100 mmHg during ambulance transport to hospital. On admission, he had a generalized slight erythema, oedema, and impaired consciousness. His temperature was 34.1 °C and his blood pressure was 119/68 mmHg. Electrocardiography showed atrial fibrillation. Corticosteroid and fluid therapy was started on admission. The patient quickly regained consciousness, and his blood pressure increased to 164/82 mmHg. He suddenly vomited food debris several times. The steroid infusion was continued and the patient was given several antihistamines orally. On day 2, the patient had no further gastrointestinal symptoms, his erythema had disappeared, and his pulse rate had normalized. However, laboratory investigations showed an isochromic anaemia and increased blood urea nitrogen. Thus, we assumed that the patient had developed a gastrointestinal haemorrhage. Emergency gastrointestinal endoscopy showed two teardrop-shaped fissures near the cardia of the stomach, and these were diagnosed as Mallory–Weiss tears (Fig. 1). We prescribed lansoprazole and polaprezine orally, and the patient was kept nil by mouth. On day 5, endoscopy confirmed epithelialization of the stomach ulcer. Radioallergosorbent testing (RAST) for specific IgE antibody to wheat was 0.65 Ua/mL (normal, < 0.34).

A positive diagnosis of wheat-induced anaphylaxis was made from the patient's past history of severe reactions, the positive RAST findings, and the systemic symptoms.^{1–3} Given the diagnosis of wheat-induced anaphylaxis and the endoscopic findings, our patient was diagnosed as having MWS secondary to vomiting caused by wheat-induced

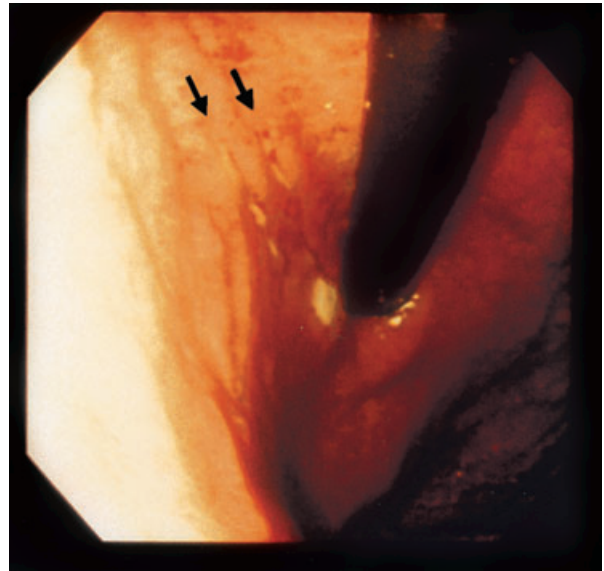


Figure 1 The emergency gastrointestinal endoscopy showed two teardrop-shaped fissures near the cardia of the stomach (arrows).

anaphylaxis. MWS is one of the causes of upper gastrointestinal haemorrhage, which results from an abrupt rise in abdominal pressure due to nausea or vomiting that induces a tear near the oesophago-gastric mucosal junction.⁴ MWS represents 3–15% of all cases of upper gastrointestinal haemorrhage. Mallory–Weiss tears are located mainly in the cardia part of the stomach, and they span across the oesophago-gastric mucosal junction; tears involving only the oesophageal side are rarely seen.

FIA is a form of IgE-mediated hypersensitivity manifested by an abrupt onset of symptoms within minutes to hours of ingesting food.^{1–3} Wheat has been increasingly recognized as a cause of food-dependent, exercise-induced anaphylaxis.^{3,5} In our case, walking was presumed to be the responsible cofactor. Clinicians should be aware of this very rare complication in patients with FIA, especially in patients with excessive vomiting who develop a sudden isochromic anaemia.

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Progressive pigmented purpura (Schamberg's disease) responding to TL01 ultraviolet B therapy

Schamberg's disease¹ is an uncommon form of pigmented purpuric dermatosis. It can occur at any age and is characterized by asymptomatic irregular areas of orange brown pigmentation, with spots like cayenne pepper appearing within and at the edge of the old lesions. It is usually idiopathic but similar appearances can result from contact allergy to clothing dyes, food additive sensitivity and hyperglobulinaemia. Histological features include dermal perivascular chronic inflammatory infiltrate, extravasation of red blood cells and macrophages containing haemosiderin. The course of disease can last several months.² We report a patient with Schamberg's disease who responded to narrow band ultraviolet therapy (Phillips TL01 lamp at 311 nm).

A 22-year-old Caucasian female patient developed an erythematous nonblanching rash on her abdomen shortly after her return from holiday in Greece. Over the next few weeks, her rash gradually spread to involve her lower limbs. She denied itch, arthralgia or mouth ulcers in association with her rash. Systematic examination showed no abnormal features. Examination of her skin showed a widespread purpuric rash along with several areas of postinflammatory and haemosiderin hyperpigmentation. It was particularly obvious on her lower limbs and lower abdominal wall (Fig. 1a). A few similar patches were noted on her upper limbs and there was sparing of her face, neck and upper chest. Her rash was not palpable or ulcerated at any stage. Full blood count, erythrocyte sedimentation rate, blood film, clotting profile, hepatitis serology, Epstein–Barr virus serology, anticardiolipin antibody, lupus anticoagulant, cryoglobulins, immunoglobulins and auto-antibodies were either normal or negative. Histological examination of affected skin showed a mild perivascular mixed cell infiltrate in the upper dermis, with red blood cells in the interstitium. There were no features to support

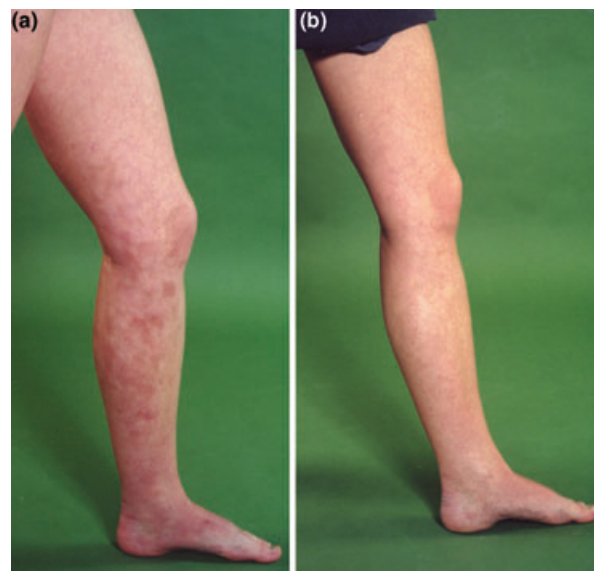


Figure 1 (a) Schamberg's disease just prior to starting TL01 UVB. (b) One year after completing TL01 UVB therapy.

mycosis fungoides. Patch testing to BCDG standard series, clothing dyes, textile series and benzoates was negative. An additive-free diet did not help.

TL01 ultraviolet therapy was given twice weekly for a total of 24 treatments. The cumulative dose of UVB was 11 J/cm² (Fitzpatrick skin type I). Her rash improved considerably at the end of treatment period and has remained clear for the last 1 year (Fig. 1b).

Phototherapy with PUVA in various forms of pigmented purpuric dermatoses has been reported in literature previously.^{3–5} However, there are no reports of narrow band UVB therapy being effective in pigmented purpuric dermatoses. Ghersetich *et al.*⁶ have shown that cell mediated mechanisms may be important in the pathogenesis of Schamberg's disease as CD4-positive T lymphocytes and CD1a-positive dendritic cells predominate in early lesions. In two patients, they have demonstrated that this cellular infiltrate clears with potent topical steroid therapy and PUVA which is associated with clinical clearance.

Ozawa *et al.*⁷ have demonstrated that 312 nm ultraviolet B light induces apoptosis in both epidermal and dermal T cells of psoriatic lesions. It is possible that this form of UVB acts in a similar way in Schamberg's disease and thus suppresses disease activity by immune suppression in skin. We suggest that narrow band UVB be considered in the treatment of progressive pigmented dermatosis.

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