Henoch-Schönlein purpura associated with infliximab therapy for pediatric Crohn's disease

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To the Editor

Anti-tumor necrosis factor-α (TNF-α) agents, such as infliximab, have been applied for treatment of inflammatory bowel disease for years. In general, anti-TNF-α agents are well tolerated; however, adverse effects associated with anti-TNF-α therapy have been reported, such as infections, psoriasis, vasculitis, vitiligo, granulomatous reactions and neoplasms [1]. In this letter, we present a case of Henoch–Schönlein purpura (HSP) occurring in a child with Crohn's disease attributed to the infliximab therapy.

A 12-year-old boy with Crohn's disease was admitted to our department because of abdominal pain and hematochezia for 2 days. The patient had received two doses of infliximab (5 mg/kg) and achieved a clinical response 3 weeks before this admission. The patient presented with periumbilical pain and hematochezia with fresh blood for 10 times after he ate cold rice noodles from the refrigerator. Blood tests showed an increased leukocyte count of 16.88×10^9/L, with 75% neutrophils and 8.7% lymphocytes. The C-reactive protein was 83 mg/L. Liver function, urea, creatinine clearance and electrolytes were normal. The clinical symptoms were not relieved after 3 days of empirical antimicrobial therapy with cefoperazone and sulbactam for suspected intestinal infection. Multiple, diffuse and palpable purpuric lesions were observed in both his feet (Fig. 1a and b). The patient suffered with knee and ankle joints swelling and pain. Colonoscopy revealed severe inflammation, edema, mucosal congestion and hemorrhage in the intestinal lumen (Fig. 1c). Furthermore, purpura rash and small ulceration in the intestinal were found (Fig. 1d). Immunofluorescence with IgA demonstrated a strong positive staining of blood vessel walls of the intestine (Fig. 1e). Taken together, the diagnosis of HSP was made.

After 3 days of intravenous corticosteroids (2 mg/kg/day) treatment, the clinical symptoms of the patient were improved (Fig. 1c). Then, the intravenous corticosteroids dose was gradually decreased and switched to oral at 1 mg/kg/day. The patient was discharged home on an oral dose (0.5 mg/kg/day) of corticosteroids. The majority of HSP-related symptoms were completely resolved; however, nephritis was observed by repeated proteinuria after 4 weeks of discharge. The infliximab therapy was discontinued. The patient received total enteral nutrition as a replacement therapy for Crohn's disease.

Up to date, HSP induced by anti-TNF-α agents is rarely described. Rahman *et al*. reported a case of HSP that can be attributed to the use of adalimumab in a 19-year-old male with Crohn's disease [2]. Condamina *et al*. described that a 20-year-old female Crohn's disease patient developed severe HSP with neurological involvement followed by adalimumab therapy that led to drug withdrawal [3]. Nobile *et al*. reported a case of herpes zoster infection followed by HSP in a 12-year-old girl receiving infliximab for ulcerative colitis [4].

Studies have suggested that the association between anti-TNF-α agents and HSP could be evidenced by vasculitis onset in a short time from anti-TNF-α agent infusion, vasculitis resolution after drug withdrawal or symptoms reappearance on drug re-exposure [3,5]. Possible pathogenic theories for the development of vasculitis include deposition of anti-TNF/TNF immune complexes in small capillaries, antibody production, direct drug toxicity on vessel walls and shifts in T cell responses [5].

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Conflicts of interest

There are no conflicts of interest.

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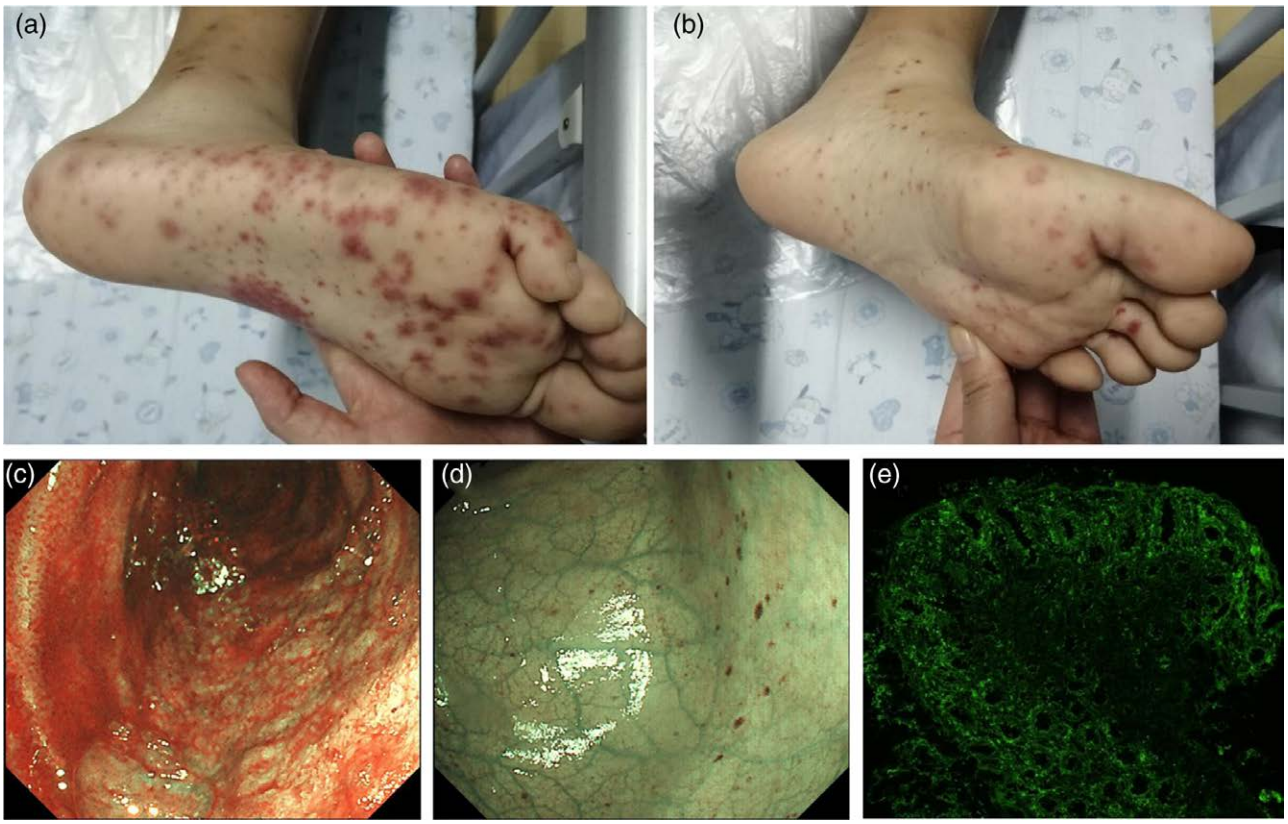

Fig. 1. (a, b) Palpable purpuric lesions on both feet; (c, d) purpuric lesions revealed on colonoscopy and (e) immunofluorescence revealing immunoglobulin A (IgA) staining of vessel walls in the colon.

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