Henoch-Schoenlein Purpura and Angiotensin-Converting Enzyme Inhibitors

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Dear Sir,

Henoch-Schoenlein Purpura (HSP) is a disease preferably observed in younger individuals and usually associated with infections of the upper respiratory tract or intestine and with food allergies [1]. Recently, it turned out that HSP is also relatively common in adults [2, 3]. Although the cause of the disease is vastly unknown, several triggering factors in the adult have been suspected. Among those, ethanol abuse [3] and drugs, e.g., antibiotics and angiotensin-converting enzyme (ACE) inhibitors have been incriminated. The true causal relationship is putative at least in some reports because the patients were taking several therapeutic agents at the same time. We would like to report the case of a 56-year-old man with HSP following treatment with captopril. A causal relationship between HSP and captopril is highly probable in this case because a positive in vitro lymphocyte transformation test was obtained in the presence of this compound.

A 56-year-old male with a history of myocardial infarction was admitted to a district hospital in September 1991. He had progressive coronary heart disease and was hospitalized due to severe cardiac failure (NYHA class III-IV) and polytopic ventricular arrhythmia (Lown IVb). His medication primarily consisted of amiodarone, pire-tanide, clorazepate and theophylline. Captopril 12.5 mg once daily was added. Two weeks later, the patient was referred to our hospital. On admission, extensive cutaneous purpuric lesions on the lower extremities and a nephritic sediment were noted. The patient complained of severe arthralgia. Skin biopsy revealed a leukocytoclastic vasculitis with deposition of IgA, C3 and fibrin. Renal biopsy was not performed because of multiple cortical cysts detected on sonography. Tests for ANA, ds-DNA, ANCA and rheumatoid factor were negative. C3 and C4 were found normal. Total leukocyte count was normal, the number of eosinophils was not increased. Hepatitis B and C was not detectable; the test for streptococcal infection (antistreptolysin) was negative. After a short improvement, the cutaneous lesions became aggravated. The patient's lymphocytes were examined in the lymphocyte transformation test for drug-specific sensibilization [10]. Strongly positive reaction with a stimulation index of 10 was demonstrated in the presence of captopril at a concentration of 1,000 µg/ml in the culture but not with amiodarone, a substance also known to cause allergic vasculitis [9]. Enalap-

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- pril was substituted for captopril. The patient died of cardiac failure 12 days later without a notable change of HSP.
- Several cases of cutaneous vasculitis due to captopril were reported [3–6] but immuno-histology was performed only in a few patients. Therefore, classification of the underlying vasculitis was not possible. The recent reports of HSP in patients during treatment with ACE inhibitors [3, 7, 8] should draw increased attention to this possible adverse effect. The ability of ACE inhibitors to induce HSP appears to be linked to the chemical structure common to the whole group of ACE inhibitors and is not restricted to compounds with a certain side chain [7, 8].
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