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**Stevens-Johnson Syndrome Induced by Clofibrate**

Sir,

Clofibrate and other fibrin acid derivatives are commonly pre-
scribed for the treatment of hyperlipidaemia. Skin rashes have been
reported in about 2% of patients (1), including severe
generalized erythema (2), urticaria (3), purpura (4) and vesiculobul-
ous eruptions (5). Clofibrate-induced erythema multiforme has
previously been reported in one patient 12 months after taking the drug (6). The Committee on Safety of Medicine in the United Kingdom has had a report of one other case of
erythema multiforme associated with the use of the drug (per-
sonal communication). This is the first report of a patient who
developed Stevens-Johnson syndrome.

A 33-year-old man was found to have hypertriglyceridaemia
of 5.23 mmol/l (normal <1.86) on routine testing. He was
commenced on 500 mg three times daily of clofibrate by his
general practitioner. Ten days later, he developed a high fever,
generalized malaise and severe pain on swallowing. He also had
a severe generalized erythematous rash, affecting the entire skin
and mucous membranes. There was no past medical or other
drug history of note.

Examination revealed an ill-looking patient with a tempera-
ture of 38°C. He had bilateral conjunctivitis and marked ulcerative
lesions on the lips, oral and nasal mucosa as well as the
genitalia. There were tense erythematous patches of varying
sizes in the entire trunk and limbs. Vesicles, bullae and target
lesions were noted in the extremities.

Apart from a leucocytosis of 15 x 109/l, blood, urine and viral
cultures were all negative. There was no rise in the titre for
mycoplasma.

A skin biopsy of a typical lesion on the right thigh showed
epidermal cell necrosis and desquamation, basal cell hydropic
degeneration with numerous cytophils and marked oedema,
telangiectasia and perivascular infiltrates of polymorphs and
cosinophils in the upper and mid dermis. The diagnosis of
Stevens-Johnson syndrome was established and clofibrate was
discontinued on admission to hospital.

The patient was treated with intravenous fluids initially, and
40 mg of oral prednisolone daily was given subsequently 48 h
after admission because of increasing painful dysphagia. Seven
days later, he could tolerate oral fluids and there was gradual
improvement in the skin and mucous membranes. Prednisolone
was tailed off over 10 days and the skin lesions gradually and
completely resolved over 20 days. There was no recurrence of
any rash at follow-up after 3 months.

The lack of other etiological factors, especially infections,
the close temporal relationship between the onset of the syndrome
and the commencement of clofibrate as well as the improvement
on drug withdrawal strongly suggest drug-induced Stevens-
Johnson syndrome. The patient was not rechallenged with
the drug because of the severity of the syndrome.

Though clofibrate-induced serious skin diseases are rare, clo-
fibrate should be included in the list of drugs capable of ind-
ucing Stevens-Johnson syndrome.

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