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LETTERS TO THE EDITOR

Polymyositis in HTLV-I infected patient

DEAR SIR, an increased prevalence of polymyositis in human T-cell lymphotropic virus type I (HTLV-I)-infected patients has been described [1]. However, the outcome and the management of these kinds of polymyositis cases are not clearly established.

A 36-year-old woman was admitted on September 1993 because of severe myalgia. Two weeks before admission, asthenia and progressive muscle weakness developed. Her temperature was 37.6°C, her general medical examination was normal except for severe proximal muscle weakness, and her neurological examination was otherwise normal. The standard laboratory values were normal except for serum creatine phosphokinase and serum aspartate transaminases, slight inflammatory syndrome, polyclonal hypergammaglobulinaemia, positive circulating immune complex and increased C3 and C4 components of complement. Cryoglobulinaemia was negative. The electromyogram disclosed only myopathic changes. Deltoid biopsy revealed endomysium mildly infiltrated by lymphocytes and atrophic and necrotic muscles fibres, some of which had undergone phagocytosis. There was no vasculitis of the blood vessels or inclusion bodies. Serum HTLV-I-antibody tests were positive by ELISA and western blotting. Drug-induced myopathies, systemic diseases, neoplasm and infectious diseases other than HTLV-I (HIV, Influenzae, Parainfluenzae, Coxsakievirus, Lyme, Trichinosis, and Toxoplasmosis) were excluded. The diagnosis of primary idiopathic polymyositis was made. All symptoms resolved within three weeks without any corticosteroid treatment, and the patient was found to remain free of any such symptoms after 18 months of follow-up.

The pathogenesis of polymyositis in HTLV-I-infected patients is not clearly known and seems to be related to an immunological process and/or direct infection of the muscle fibres by virus [2, 3]. The clinical history of this patient suggests that in polymyositis associated with HTLV-I infection, the prognosis should not be very different from the other idiopathic polymyositis. Spontaneous clinical remission is especially possible. Hence, in such cases the use of corticosteroids, prescribed usually as a first-line drug therapy, may be delayed and reserved for relapses. This observation opens the discussion about the benefits of early corticosteroid therapy in some cases of polymyositis. Prospective studies are needed to determine the prognostic markers of these categories of polymyositis.

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References

1 Mora CA, Garruto RM, Brown P, *et al.* Seroprevalence of antibodies to HTLV-I in patients with chronic neurological disorders other than tropical spastic paraparesis. *Ann Neurol* 1988; **23** (Suppl.): S192–5.
2 Dalakas MC. Polymyositis, dermatomyositis and inclusion-body myositis. *N Engl J Med* 1991; **325:** 1487–98.
3 Dickoff DJ, Simpson DM, Wiley CA, *et al.* HTLV-I in acquired adult myopathy. *Muscle & Nerve* 1993; **16:** 162–5.

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Angio-oedema induced by ACE inhibitors

DEAR SIR, I read with interest the case report by Forslund *et al.* entitled 'Angio-oedema induced by enalapril' [1]. This report clearly emphasized the potentially life-threatening adverse effect of angio-oedema of the upper airway that all clinicians should consider when evaluating patients who use angio-

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tensin-converting enzyme (ACE) inhibitors. I wish to point out another potentially serious adverse effect of ACE inhibitors that also may be secondary to localized angio-oedema: acute pancreatitis.

There has been a growing number of reported cases of acute pancreatitis associated with the use of enalapril [2, 3], lisinopril [4, 5], and captopril [5]. Also, the number of cases of pancreatitis owing to various ACE inhibitors reported to the Food and Drug Administration and various pharmaceutical companies totals over 100 [5]. The inhibition of ACE may lead to elevated bradykinin levels in various tissues with ensuing local vasodilatation and increased vascular permeability, thus leading to angio-oedema [1, 4, 5].

Angio-oedema of pancreatic tissue may result in ductal obstruction leading to acute pancreatitis [4, 5]. Cases of both self-limited and fatal pancreatitis have been reported in association with ACE inhibitor use [4, 5]. Measurement of ACE levels in the setting of pancreatitis or upper-airway angio-oedema may be helpful in the diagnosis of ACE inhibitor-induced angio-oedema [1]. Clinicians should consider the diagnosis of acute pancreatitis when a patient taking an ACE inhibitor presents with abdominal pain that is not explained by another process.

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References

1 Forslund T, Tohmo H, Weckstrom G, Stenborg M, Jarvinen S. Angio-oedema induced by enalapril. *J Intern Med* 1995; **238:** 179–81.
2 Tilkemeier P, Thompson PD. Acute pancreatitis possibly related to enalapril. *N Engl J Med* 1988; **318:** 1275–6.
3 Roush MK, McNutt RA, Gray TF. The adverse effect dilemma: quest for accessible information. *Ann Intern Med* 1991; **114:** 298–9.
4 Marinella MA, Billi JE. Lisinopril therapy associated with acute pancreatitis. *West J Med* 1995; **163:** 77–8.
5 Standridge JB. Fulminant pancreatitis associated with lisinopril therapy. *South Med J* 1994; **87:** 179–81.

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