The Case of the Mercury Heart

Sir,

We were interested in the letter entitled “The Case of the Mercury Heart” by Muzio, Guerra and Rongioletti (1). One of us, JT, reported a patient with a particularly severe rash in the groin and vulvar area after dental amalgam work (2). It transpired that the patient had been sensitised to phenyl mercuric salt in a spermicidal cream.

In Scotland there are still a large number of silver amalgam fillings inserted annually. For the year 1993, 787,000 fillings were performed. This compares with 1,772,993 in 1968 (Scottish Dental Estimates Board). Thus, as a very crude estimate, assuming on average a million fillings a year for 25 years with a mean mercury content of 1.5 g, then 37.5 metric tons of mercury has been used for this purpose in a population of about 5.5 million.

There have been recent UK media suggestions of possible systemic dangers of mercury amalgam fillings, and the public is being encouraged to consider removal and replacement by other materials despite the fact that there is little scientific evidence for this (3). Replacement may go ahead, and thus a considerable number of people will be subjected to a sudden increase in mercury exposure. There may therefore be an increase in cases presenting with mercury toxicity. The flexural nature of the eruption has been commented upon by other authors (4,5).

Spermicidal creams containing mercury are no longer available in the United Kingdom or, to the best of our knowledge, in Europe. They may, however, have been previously used by those now middle-aged. This is the age group with a large preponderance of mercury amalgam fillings. It may be worth enquiring along appropriate lines in such patients presenting with flexural, or in particular, a genital rash.

REFERENCES

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Antithrombin-III Plasma Levels in Patients with Venous Leg Ulcer Disease

Sir,

In the development of venous leg ulcers, insufficiency of the deep leg veins is of primary pathogenic importance. The insufficiency often results from previous episodes of deep venous thrombosis, which phlebographically is demonstrable in over 80% of venous leg ulcer patients (1). Inherited deficiencies of anticoagulant proteins of the coagulation cascade, such as the protein C–protein S system, are among the risk factors which significantly predispose the affected individual to develop deep venous thrombosis and subsequently venous ulcers (2–5).

The significance of the anticoagulant protein antithrombin-III has not been investigated before in venous leg ulcer patients. Deficiency of antithrombin-III could be of primary importance, since antithrombin-III is a very potent physiological inhibitor of activated clotting factors IIa, Xa, IXa, Xla and XIIIa, an action which is significantly amplified when heparin or other heparan sulphates are present (2–4).

During a 6-month inclusion period, we collected plasma samples from 46 unselected, consecutively admitted venous leg ulcer patients. Plasma concentrations of antithrombin-III were determined using a functional assay (3, 4), and concentrations were expressed in percentages of pooled, normal plasma. The mean plasma antithrombin-III concentration was 98% (SD 17%) in the 46 patients. None of the patients had antithrombin-III concentrations below 50%, which is indicative of inherited antithrombin-III deficiency (3, 4). Antithrombin deficiency does thus not appear to be a common risk factor for the development of venous leg ulcer disease.

REFERENCES

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