

negative as were the serum *Chlamydia trachomatis* antigen by ELISA, rheumatoid factor and antinuclear antibodies. HLA B27 was positive. A bone scintigraphy revealed an abnormal uptake in the left knee and metatarsophalangeal joints. The synovial fluid of the left knee revealed: normal glucose protein 72 g/l, many red and white cells with 79% neutrophils.

The urine culture was negative. Gram stain of the urethral discharge disclosed Gram-positive cocci, but the culture was negative, perhaps because the patient had been previously treated with doxycycline. The patient, with a clinical diagnosis of Reiter syndrome, was put on indomethacin with some improvement and was discharged home. Two days later, the patient was newly admitted with swelling of his left leg below the knee. A venography revealed several defects on the femoral venous system indicative of a deep venous thrombosis.

Parameters of coagulation and fibrinolysis were normal, including the number of platelets, prothrombin, partial thromboplastin and thrombin times, antithrombin III functional antigen, functional plasminogen levels, functional plasminogen activator inhibitor, and the protein S and C activity. Anticardiolipin antibodies were negative. The patient was anticoagulated with rapid resolution of the picture. Nine months later he remains well.

Hypercoagulable state defines a concept which has been related to several rheumatic diseases as well as different conditions.⁴ In our patient we could not find any abnormality in the coagulation system, so that other unknown mechanisms could have taken place.

Although Csonka described the occurrence of thrombophlebitis in Reiter's syndrome many years ago,³ we have been unable to find reports from other authors in a review of the literature using MEDLINE files back through 1966. Moreover, in no reported case has the patient undergone a complete coagulation study to exclude underlying coagulation abnormalities. Although infrequent, thrombophlebitis should be considered as another complication of Reiter's syndrome. However, the initial coagulation abnormality remains unknown.

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References

1. Willkens, R.F., Arnett, F.C., Bitter, T. *et al.* Reiter's syndrome. Evaluation of preliminary criteria for definite disease. *Arthritis Rheum* 1981, **24**: 844-849.
2. Sairanen, E., Paronen, I. & Mahonen, H. Reiter's syndrome: a follow-up study. *Acta Med Scand* 1969, **185**: 57-63.
3. Csonka, G.W. Features and prognosis of Reiter's syndrome. Clinical aspects of Reiter's syndrome. *Ann Rheum Dis* 1979, **38**: 4-7.
4. Schafer, A.I. The hypercoagulable states. *Ann Intern Med* 1985, **102**: 814-828.
5. Csonka, G. Thrombophlebitis in Reiter's syndrome. *Br J Vener Dis* 1966, **42**: 93-95.

Poisoning from topical salicylic acid

Sir,
Treatments applied to the skin may have unwanted systemic effects. We report a patient who suffered poisoning from topical salicylic acid.

A 42 year old woman who suffered from psoriasis, was prescribed 10% salicylic acid in white soft paraffin by her general practitioner. This was intended for application to localized areas of hyperkeratosis but the patient used it widely on her trunk and limbs. We estimate that she applied 50 g of the ointment daily over a 10 day period. She was not taking any oral medication; her body weight was 45 kg. Her psoriasis deteriorated and she was admitted to hospital. Two days before admission she had developed deafness and nausea.

On admission almost her entire skin was affected by psoriasis. She was agitated and had a pyrexia and a tachycardia. Serum salicylate (measured 14 hours after the last application of the ointment) was 2.6 mmol/l (therapeutic range up to 2.2 mmol/l). Serum bicarbonate was 13 mmol/l (normal range: 22-28 mmol/l); renal and hepatic function were normal. The topical salicylic acid was discontinued and supportive treatment was given. Twenty-four hours later her symptoms had resolved and her vital signs were stable; serum bicarbonate was normal and serum salicylate had fallen to 1.5 mmol/l.

The patient suffered poisoning primarily because a high concentration of salicylic acid was applied to a wide area. Her low body mass made her more susceptible to toxicity. Absorption of the drug was increased because of the inflamed condition of the skin. Chronic administration of salicylic acid may produce intoxication at serum levels lower than those seen in acute poisoning.¹

Most previous reports of salicylate poisoning have arisen during intensive treatment of ichthyotic conditions in hospital.²⁻⁴ Our report illustrates that salicylate poisoning may also occur in ambulant patients. It is often difficult to ensure that treatment is applied according to instructions in such patients. We advise caution in the use of high concentrations of salicylic acid.

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References

1. Temple, A.R. Acute and chronic effects of aspirin toxicity and their treatment. *Arch Intern Med* 1981, **141**: 364-369.
2. Davies, M.G., Briffa, D.V. & Greaves, M.W. Systemic toxicity from topically applied salicylic acid. *Br J Med* 1979, **1** (6164): 661.
3. Anderson, J.A.R. & Ead, R.D. Percutaneous salicylate poisoning. *Clin Exp Dermatol* 1979, **4**: 349-351.
4. Galea, P. & Goel, K.M. Salicylate poisoning in dermatological treatment. *Arch Dis Child* 1990, **65**: 335.