Severe mesangiolysis in a patient exposed to glycol ether solvents

Sir,

Toxic agents are known causes of mesangiolysis and the best examples are Habu snake venom and mitomycin C [1]. We describe a patient with mesangiolysis occurring during a period of glycol ether exposure.

Case. A 53-year-old man complained of peripheral paraesthesia. His history consisted of an 130/85 mmHg hypertension, and a proteinuria, discovered 1 year ago. Laboratory investigations showed 65 ml/min creatinine clearance, 67 g/l haemoglobin, 200 UI/l lactic dehydrogenase and 12 µmol/l total bilirubin. Repeated glycaemia was normal. Hepatitis B–C serologies, cryoglobulins and antinuclear antibodies were negative. Urinalysis showed 1.5 g/day proteinuria without haemosiderin. No immunoglobulin deposits were detected. Nine had a mesangiolysis pattern. Seven had microangiopathy. Tubules had heterogeneous vacuolization of cells with altered mesangial matrix. This case suggests that glycol ether exposure is an unrecognized cause of mesangiolysis.

Comment. Glycol ethers are widely used solvents in surface coatings [2]. Our case is consistent with a mesangiolysis due to a glycol ether according to the work survey, the neurological symptoms and the absence of primitive glomerulopathy. Massive exposure to glycol ethers is followed by central nervous depression, paraesthesia, vomiting and, finally, acute renal failure [3]. Renal biopsies were not performed and mesangiolysis should be overlooked.

Animal studies were conducted to examine the distribution of N-butoxy-ethanol, but without morphological investigation. They showed that percutaneous absorption lead to both a urinary excretion and biological abnormalities, such as renal failure and moderate haemolysis. N-butoxy-ethanol penetrates the skin and toxic action from skin exposure is more likely than from inhalation. Here, skin absorption should be favoured by the non-ionic surfactant. Human erythrocytes are less sensitive to the haemolytic effects of N-butoxy-ethanol than rat erythrocytes. This fact should explain that haematological changes in humans occur slightly during chronic exposure. Similarly, repeated ingestion of 2-amino-ethanol caused kidney damage in dogs, namely tubular necrosis without mesangiolysis, but with haemolysis [4].

The mechanisms of mesangiolysis after exposure of glycol ethers could be explained by a direct toxicity on mesangial cells or an indirect toxicity leading to haemolysis and, possibly, microangiopathy. Indeed, in a series of patients treated with mitomycin, a mesangiolysis on biopsies has already been described, associated with microangiopathy and haemolysis in the absence of overt haematological changes [5]. Moreover, mesangiolysis was also observed in haemoglobinopathies without microangiopathy. The tubular toxicity of haemosiderin deposits is known, but haemosiderin has been also isolated within the glomerular cells with altered mesangial matrix. This case suggests that glycol ether exposure is an unrecognized cause of mesangiolysis.

Conflict of interest statement. None declared.

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Fig. 1. Renal biopsy showing numerous features of segmental mesangiolysis with large microaneurysm formation. Jones silver stain. Magnification: ×400.

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