Membranous nephropathy caused by mercury-containing skin lightening cream

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Summary: A 46 year old woman developed membranous nephropathy following the use of a mercury-containing skin lightening cream. This association has not been reported in the literature for over a decade and apparently never from this country. It is important that clinicians are aware of this usually eminently treatable cause of the nephrotic syndrome as it is likely to be missed unless specifically enquired for.

Introduction

It is well known that the nephrotic syndrome may be caused by a variety of drugs, but less widely recognized that it may also be caused by cosmetic preparations. The association between the use of mercury-containing skin lightening creams and the nephrotic syndrome was described some time ago, but has not been mentioned in the literature for over a decade and, to our knowledge, has never been reported from this country. We wish to emphasize the importance of this reversible cause of the nephrotic syndrome as it is likely to be overlooked unless a careful history is taken. We have recently seen such a case.

Case report

A 46 year old Pakistani woman, at present resident in Saudi Arabia, presented to the Hammersmith Hospital in April 1986 with a known diagnosis of the nephrotic syndrome. She had been well until March 1985, when she developed peripheral oedema. This persisted despite diuretic treatment and investigations in July 1985 showed three plus proteinuria with a serum albumin of 24 g/l. There was no immediate response to a reducing course of prednisolone, but subsequently there was some reduction in her proteinuria. Because the protein loss did not remit completely, she was referred for another opinion.

The only other significant factor in her history was use of a face cream since August 1984. This had been given to her by a friend in Saudi Arabia, who had found that it improved and lightened her complexion. The instructions were that the cream should be used weekly, but our patient was using it daily. The cream was labelled as being made by the Standard Pharmaceutical Company, London; this company does not appear to exist.

On examination, she was overweight, but there were no other abnormalities. Investigations showed: serum sodium 142 mmol/l, potassium 4.4 mmol/l, urea 3.2 mmol/l, creatinine 71 µmol/l, total protein 64 g/l, albumin 36 g/l, autoimmune screen negative, haemoglobin 11.6 g/dl, white blood cells 8.9 × 10⁹/l, platelets 473 × 10⁹/l. Urine: normal microscopy, 24 hour protein excretion 2.2 g, calculated creatinine clearance 110 ml/min. Renal ultrasound: two normal sized kidneys.

A percutaneous renal biopsy was performed. Light microscopy showed diffuse mild thickening of the basement membrane of the glomeruli. Electron microscopy demonstrated electron dense subepithelial deposits distributed in a patchy manner around the capillary loops. Immunofluorescence also showed rather patchy staining predominantly for IgG, in relation to the capillary loops. All of these changes were compatible with a diagnosis of membranous nephropathy.

Shortly after discharge the results of a mercury analysis became available. The cream contained 1%
mercury by weight, and the urinary content was 33 nmol Hg/mmol creatinine (normal < 5). The patient stopped using the cream at this point.

Discussion

Mercury may damage the kidney in a number of ways depending on the form in which it is absorbed and the rate at which this happens. In general, organic and metallic mercury are more lipophilic than inorganic mercury and tend to be more neurotoxic than nephrotoxic (and vice versa for inorganic mercury). However, this is not an absolute distinction and all forms of mercury may cause either tubular or glomerular damage. The distinction between these patterns of injury is partly determined by the rate of administration: acute exposure tends to cause tubular necrosis and chronic exposure, as in our patient, to cause glomerular disease. However it is also clear that immunological and other idiosyncratic factors play a major role in causing glomerular damage. The histological and immunofluorescent findings in our patient are typical of mercury nephropathy: light microscopy is often interpreted as minimal change or early membranous nephropathy, but the findings on electron microscopy and immunofluorescence provide evidence for the latter diagnosis.

The diagnosis rests mainly on a history of exposure.

Supporting evidence may be provided by a raised urinary concentration of mercury, but this tends to be rather variable. Furthermore, the concentration usually falls to normal within weeks of removal from exposure, although the renal abnormalities may persist for much longer. Under these circumstances raised concentrations of the metal may be found in the hair for up to six months.

The treatment of mercury nephropathy due to chronic intoxication consists simply of removal from exposure. With this the prognosis is good: in a previous series of nephrotic syndrome, largely secondary to skin lightening creams, 50% of the patients entered remission, 77% of these doing so spontaneously when use of the cream was stopped.

Despite the fact that topical mercury preparations were condemned as both useless and dangerous almost twenty years ago, these creams are apparently still available. It is important that clinicians are aware of this cause of what is usually an eminently treatable renal problem, and inquire specifically about cosmetic use in appropriate patients, particularly those without access to preparations from abroad.

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References