

LETTERS TO THE EDITOR

IgA Glomerulonephritis in a Plumber Working with Solvent-Based Pipe Cement

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While the etiology of auto-immune kidney disease remains obscure, IgA nephropathy accounts for over 20 percent of the primary glomerulonephritides in adults.¹⁾ Although there is a strong association with prior upper respiratory infections, the pathogenesis of IgA nephropathy is as yet unclear. A 1972 report by Beirne and Brennan²⁾ describes cases of Good pastures syndrome associated with massive exposure to hydrocarbon solvents. The authors proposed that the disease was secondary to chemical injury to the lung or kidney basement membranes. Churchill *et al.*³⁾ reviewed the literature since then for presumptive evidence associating solvent exposure with glomerulonephritis.

A request for health hazard evaluation was submitted to the National Institute for Occupational Safety and Health (NIOSH) from a 28-year-old male self-employed plumber who has been working with plastic pipe and pipe cement for 9½ years. Recently, he had been seen by a physician for gross hematuria. Needle biopsy of the kidney, with immunofluorescence, light, and electron microscopic examination showed segmental proliferative glomerulonephritis with IgA deposits. He had no family history of kidney disease. These findings, as well as the presence of capillary adhesions to Bowman's capsule and fibrin in the glomerular mesangial deposits, were indicative of progressive disease.

Industrial hygiene monitoring of his work identified massive, short-term (15-minute) exposures (range: 389–757 ppm) to tetrahydrofuran (THF), a component of polyvinyl chloride (PVC) pipe cement. These exposures were associated with the gluing of short sections of pipe, while working in the confines of a bathroom vanity. Other potential solvent exposures from the cement included: cyclohexanone, which was not measurable, and low levels of methylethyl ketone (MEK) (range: 3.9–5.0 ppm). The NIOSH-recommended short-term exposure limit for THF is 250 ppm.

While we are not suggesting cause and effect we feel that the situation which we evaluated merited discussion. It may be possible that a predisposition toward IgA nephropathy could be exacerbated by massive short-term exposure to solvent. Subsequent pulmonary, hepatic, or renal insult may initiate the disease process.

REFERENCES

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*National Institute for Occupational Safety and Health (NIOSH),
4676 Columbia Parkway, Cincinnati, Ohio 45226, U.S.A.*

**William N. ALBRECHT
James M. BOIANO and
Roger D. SMITH***

* *Department of Pathology,
University of Cincinnati,
Cincinnati, Ohio, U.S.A.*

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