Prolonged exposure to an epoxy resin leading to interstitial nephritis

A 51 year old administrator was transferred with a four week history of malaise and intractable vomiting. His creatinine had deteriorated over 10 days from 343 μmol/l to 524 μmol/l. There was no notable personal or family history. He was not taking any medications or herbal remedies. Further questioning showed that over the previous 18 months he had been building his own aeroplane in a large but enclosed aircraft hangar. This involved the use of a blended epoxy resin (SP Ampreg 20) and the associated SP Ampreg 20 standard hardener (3-amino-methyl-3, 5, 5-trimethylcyclohexylamine and 4, 4’- isopropylidenephenol) (Standard Polymer Systems). Six months previously he had developed severe contact dermatitis which resolved on use of occlusive hand protection.

Physical examination was unremarkable. Urinary analysis showed 850 mg protein/24 hours and negative microscopy. Routine immunological tests and ultrasound of the renal tract were normal. Histological evaluation of a percutaneous renal biopsy showed an interstitial nephritis with lymphocytes, plasma cells, and a few eosinophils. There was mild interstitial fibrosis.

The patient was started on oral prednisolone at 0.5 mg/kg/day. This was slowly reduced over eight weeks to 10 mg daily and the creatinine improved to 155 μmol/l six months later.

We think that this patient developed interstitial nephritis secondary to inhalation of volatile substances associated with the use of an epoxy resin. He had no contact with other chemicals that could cause this disease. He experienced general malaise which cannot be accounted for by the degree of renal impairment, but is consistent with the systemic effects of resin exposure. Previous severe dermatitis and the symptoms had improved with the end of direct contact.

Epoxy resins are formed by the condensation of epichlorohydrin and a diphenol in the presence of a tertiary amine hardener. Allergic illnesses, particularly dermatitis, can be caused either by the resin or the hardening agents. Contact can be direct or by inhalation of volatile hydrocarbons.

Workers chronically exposed to volatile organic solvents have been shown to have significantly more protein in their urine than controls. Cases of acute interstitial nephritis have been reported associated with volatile hydrocarbons. Two were related to chronic exposure in gas laboratories, one after a single episode of exposure to polymide epoxy high gloss paint fumes.

Interstitial nephritis is a pathological entity characterised by a mononuclear cell infiltrate of the renal tubular interstitium. Although lymphocytes predominate eosinophilia can occur particularly when drugs are identified as the cause. Clinically the picture is an acute decline in renal function which can be associated with heavy proteinuria and peripheral oedema. Improvement often occurs after removal of the offending agent, but uncontrolled observations suggest that moderate doses of corticosteroids hasten recovery.

Recovery should be complete although evidence of interstitial fibrosis at biopsy is associated with a poorer outcome.

IAN D DITTMER
ALISON J ARMITAGE
The Richard Bright Renal Unit, Southmead Hospital, Westbury-on-Trym, Bristol BS10 5NB, United Kingdom

Correspondence to: Dr Ian D Dimmer, The Richard Bright Renal Unit, Southmead Hospital, Westbury-on-Trym, Bristol BS10 5NB, UK.


This is a short booklet on an important topic. It is based on contributions made by some two dozen experts participating in a World Health Organisation (WHO) meeting focusing on asbestos, crystalline silica, and coal mine dusts. It aims to be a step by step approach to the development of programmes particularly for developing countries, where "... effective measure are not taken because of a lack of awareness of the problem." The reader may well wonder whether there still exist countries in which even when multinationals giants are not involved either in financial or advisory roles, management is sophisticated enough to have mastered mining and fibre processing technology and yet has succeeded in maintaining ignorant of dust hazards. For that matter, it is too nihilistic to think that where the infrastructure for dust control is absent, that it will be unlikely that the facilities recommended by WHO would be available, and if they were, whom would use them? The report states that "...medical screening is presented as an integral part of a disease prevention programme, intended to detect disease in its preclinical phase at a stage when its progression can be arrested. Surveys to determine the study of trends and distribution of disease incurrence through the systematic collection, analysis, and evaluation of morbidity and mortality reports and other data. To it the authors add the requirements for reporting findings, and for intervention to prevent disease. As a test of the recommendations of the committee, consider the case of asbestos workers, where the hygiene standard set to protect workers remains to be evaluated. Further, as the author notes, there is uncertainty about the natural history of asbestosis so that we do not know whether it can be detected at a stage where further progression will not occur if exposure ceases."

This was recognised in the discussions leading up to the United Kingdom 1969 Asbestos Regulations. The inadequacy of statutory medical examinations as then practiced and understood was recognised. In this place, Lloyd Davies was able to obtain agreement for the need for a national longitudinal survey of asbestos workers, by a few dedicated physicians, who standardised the medical, radiological, and physiological investigations. As the examinations were primarily for research purposes and for the indirect measures of the effectiveness of the regulations or their implementation, and could not be shown to benefit individual workers at risk, it was agreed that participation should be voluntary for workers, but mandatory for employers to permit examination. In parallel, measures of environmental exposure began to be collected, and cancer morbidity and mortality registers were established.

Other times, other ways. Somewhat belatedly, the European Community woke up, and despite the fact that clinical benefit to individual workers had not been established, and in the absence of advance in treatment of any of the asbestos diseases since 1969, directed that asbestos workers shall be compulsorily examined. According to this WHO report (annexe 2), United Kingdom asbestos workers currently undergo primarily a screening programme with some surveillance. The target of the programme it declares is to detect asbestosis and pleural changes. The two years examination cycle should include: medical and occupational histories; chest x ray films; and pulmonary function tests (forced expiratory volume in one second and forced vital capacity (FEV1, and FVC). In fact, Health and Safety Executive regulations implementing the European Directive do not prescribe the details of examination, they are left to the discretion and clinical judgement of the appointed doctor. The clock is stopped if the worker is reported to have an abnormality, but sometimes continues to tick. The worker is seen periodically and followed up, usually by the same doctor. The programme was supposed to be cancelled in 1973 (2). The WHO states that the effectiveness of the United Kingdom production of dust has been assessed by published statistics. Annual audit of the mesothelioma register is certainly published, and there has been a mortality analysis of subjects on the asbestos register, but


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I D Dittmer and A J Armitage

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