A CLUSTER OF TOXIC HEPATITIS FROM EXPOSURE TO AN INDUSTRIAL CHEMICAL: PYRROLE

Tan H.H[1] DABT, FRCSEd(A&E); Teo S.[2]

[1]*Clinical Toxicology Consultation Service, Changi General Hospital, Singapore*

[2]*Principal Specialist (Occupational Medicine), OSH Specialist Department. Occupational Health Division. Ministry of Manpower, Singapore*

Objectives: We report 3 healthy young patients who presented to our hospital over a course of 5 years and were diagnosed with toxic hepatitis. They all worked in the same electronics factory. As the cases presented about 2 years a part, the confirmation of the possible chemical involved was delayed.

Methods and results: All 3 patients had hepatocellular hepatitis and were extensively worked up for other causes of hepatitis. The average latency period between work exposure and hepatic symptoms was between 1 to 8 months. Liver biopsy for the last 2 patients confirmed the presence of toxic hepatitis. The first two patients had returned back to the same job scope in the same area after discharge from hospital resulting in a flare-up of their symptoms again, within 8 to 20 days, both requiring re-hospitalization, suggesting evidence of work-relatedness. The second worker was treated for suspected autoimmune hepatitis and continued to work in the same environment. He had frequent mild relapses of hepatitis requiring treatment with steroids. After being taken out of the environment, he did not have any relapse. The last patient was not allowed back to work in the same environment. The RUCAM score for drug induced liver injury of 6 - 7 for all 3 patients suggested a probable toxic cause.

Two of these workers worked at the polymerization section and the third at the masking and punching section at the same level. However, there was no partitioning between the 2 sections and they shared the same air conditioning system. In addition there were inadequate engineering control measures, resulting in contamination of air in the entire production area. The workers also did not wear appropriate personal protective equipment.

The work process was evaluated and out of the dozen chemicals involved, the most likely agent is pyrrole. In the polymerization section, this heterocyclic pyrrole compound was used as a monomer. After polymerization, it formed a polypyrrole, which enhanced the conductivity of the capacitor. Pyrrole level in the air was detected but there is no PEL for this chemical.

Conclusions: Pyrrole has been reported to be hepatotoxic in animal studies but there are no human reports available in the literature. To our knowledge, these are the first reported cases of acute toxic hepatitis possibly due to pyrrole or a pyrrole related compound. Further studies are needed to evaluate the mechanism of toxicity.