



HEPATIC TOXICITY ASSOCIATED WITH EXPOSURE TO DIMETHYLACETAMIDE IN HEALTHY WORKER

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Background: Dimethylacetamide (DMAC) is a dipolar solvent which is colorless to pale yellow liquid with an odor of ammonia at or around 20 ppm. It is most widely used in the acrylic fiber, polyester films and pharmaceutical industries. Occupational exposure to DMAC has been linked to adverse effect in hepatitis, rhabdomyolysis, and hallucinations. However, although DMAC have the similar characteristics as dimethylformamide(DMF), less human toxicities were reported. Here we report a case of dimethylacetamide -associated hepatitis in healthy worker working in acrylic fiber industry.

Case report:This 31 y/o male has no underlying disease except pneumothorax history caused by car accident 10 years ago. He started his work in an acrylic fiber industry for 2 months and general weakness with yellowish appearance was noted then. Within a few weeks, he experienced epigastralgia, poor appetite and admitted to a hospital where total-bilirubin level increased dramatically up to 18 mg/dl, other liver functions showed AST/ALT:181/469, GGT:241 U/L, ALK-P:231 U/L, LDH 230 U/L. No leukocytosis, electrolyte imbalance or renal dysfunction was found. Further survey including HBs antigen, anti HCV antibody, EBV, CMV, HSV and auto-antibodies were all showed non-specific findings. We went through the factory for investigation. No special finding was found but the worker usually soaked his hands into the DMAC solution without gloves protection, which may be a clue to us about the overexposure of DMAC. Besides, the patient took metoclopramide, dimethicone, rabeprazole up to seven days for his constipation at outpatient department before hepatitis occurred, which may result in the **interference** of DMAC's metabolism at liver and induced toxicity. The liver dysfunction improved during hospitalization with total bilirubin decreased to 6.47 mg/dl, AST/ALT:105/196, ALK-P:161 U/L 20 days later after admission. N-methylacetamide (**NMA**), the major metabolite of DMAC in urine showed 64.9 (mg/g of creatinine) after seven days away from exposure, which is still much higher than normal range. The final diagnosis was dimethylacetamide -associated hepatitis according to the clinical judgement.

Conclusion: The mechanism of DMAC-related liver toxicity remains unclear and some researchers have found a dose-response relation between DMAC exposure and the development of liver toxicity. The toxicity may also be partly attributable to acetamide given that DMAC and dimethylformamide both result in liver toxicity. Future studies are warranted to better understand the toxic mechanisms as well as the management of DMAC related toxicity. Medications may disturb the metabolism of chemical substance and induce toxicity. Workers working in such industry still need to pay caution to this hepatotoxicity chemical substance.