Chronic toxic liver damage in workers of PVC producing plants

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Polyvinylchloride (PVC) has been produced for more than 30 years; it is used in many industries and therefore is manufactured on a large scale throughout the world.

The fact that up to 1966 there were no published reports in the Western Hemisphere on chronic toxic effects is largely due to the widespread belief that only relatively inert substances are involved in the production of PVC.

Since 1967, there were reports from Romania, France, England, U.S. and Germany describing a disease characterized by Raynaud's syndrome, skin changes, pseudo-scleroderma; on the basis of a total number of 55 cases this disease was named "occupational acroosteolysis." In some of the papers there had been mention of hepatomegaly and hypothyroidism in the exposed workers. Out of the 55 mentioned cases, the symptomatology had brought up a suspicion of liver disfunction in eight. The main focus of attention being the occupational acroosteolysis, the problem of liver damage, first reported in 1949 in Russia in workers of PVC producing plants (in 15 out of 48 exposed workers), was obscured. The reported toxic hepatic damage in 50 out of 168 exposed workers, in a paper from Romania, and the "Subclinic chronic-epithelial hepatitis" in 15% of examined workers in another Russian paper did not receive the necessary attention.

In several workers of a PVC producing plant, the authors observed esophageal varicosities, splenomegaly and nonspecific liver damage. These observations, together with the above-mentioned reports on "subclinic liver damage" determined an investigation of the pathology of these changes.

Radiologic, biochemic, enzymatic methods, liver scanning, laparoscopy and liver needle biopsy were used in 20 workers of a PVC producing plant (age 30 to 56 years), length of exposure 1 1/2 to 21 years).

There were a total of 120 workers involved in the production of PVC; 45 of them had been examined at the Department of Dermatology, some because of manifest skin changes, but many were given a preventive examination.

In most of these 45 workers, the examination resulted in a clinical and/or biochemical suspicion of liver damage.

Until May, 1973, it was possible to perform laporascopy in 20 of these 45 workers mainly in those with a higher degree of suspicion. The prevalence of liver dammage in workers exposed in the PVC manufacturing plants could not be evaluated on this basis only.

The results are given in two tables. Table 1 presents clinical and biochemical changes.

In 13 cases out of 20 the liver was found to be enlarged (with 1.5 to 3cm). Two patients had pain on palpation in the RUQ; hyperlipidemia had been diagnosed in 1967 (after six years of exposure) in another patient, together with "liver damage."

In only one patient was there a history of jaundice in 1955, before the onset of exposure; in 1968 a diagnosis of chronic liver dysfunction had been established. There was no history of alcoholism. Splenomegaly was found in 7 out of 20 cases (range 1.5 to 5cm). Bilirubin (total) exceeded the upper normal limit of 1mg/100ml in three cases (range 1.1-1.7mg/100ml). The bromsulphalein test was abnormal (>5% after 45 minutes) in 19 cases (range 6.1-21.5). SGOT exceeded the upper normal limit of 12mU/ml in 17 cases; in 14 cases SGPT was elevated (range 15-30). An increased level of alkaline phosphatase was found in two patients (>18mU/ml). Hypotrombocytemia (less than 150 x $10^3/mm^3$) was found in 19 out of 20 patients, at least initially; in 12 the initial level was lower than 100 x $10^3/mm^3$. Acroosteolysis was present in four patients. BSR, CBC, glycemia, urinalysis, LDH, cholinesterase, acid phosphatase, iron and copper serum levels, proteinuria and electrophoretic separation of serum proteins, cholesterol, B-lipoproteins, tryglycerides and plasmatic coagulation factors were not found to be abnormal.

Rheumatoid factor, ANA, LEcells, immunoglobulins G, A and M, complement fractions B.A and cold antibodies were checked initially, for the purpose of differential diagnosis with scleroderma, but were not repeated.

Australia (SH) antigen was always negative. In only one case was there a cholelithiasis; a solitary gallbladder stone, not interfering with normal bile flow.

Table 2 presents the results of radiology, laparoscopy and histologic examination.

In three cases varicose esophageal veins (and in two cases also of the gastric fundus) were visualized.

Laparoscopy showed the following changes: capsular fibrosis (in 15 cases), reorganization of the liver architecture (in 8 cases), splenomegaly (in 7 cases), portal hypertension (in 2 cases) and hepatomegaly (in 4 cases).