Prevalence of disease among vinyl chloride and polyvinyl chloride workers

by

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The emergence in January 1974, of vinyl chloride as a new occupational carcinogen, producing hemangiosarcoma of the liver focused new and much enhanced attention on the toxicologic effects of this chlorinated hydrocarbon.

A considerable number of studies had been published on the rather puzzling and unique syndrome of "acroosteolysis" (Table 1) mainly describing the bone lesions in the distal phalanges of the fingers, but also the associated scleroderma-like skin lesions and the striking vascular (arterial and arteriolar) changes, pathologically the main lesion, leading to secondary bone and skin changes (Tables 2 and 3).

Much less attention had been given to the hepatotoxic effects of vinyl chloride, although convincing experimental evidence had been produced (Torkelson et al., 1961) and scattered reports of ill-defined liver changes were included in some of the studies of vinyl chloride exposed workers (Table 4).

There was evidence that liver damage occurred in workers exposed to higher levels of vinyl chloride. In some studies the liver changes (hepatomegaly, tenderness on palpation, abnormal liver tests) were found to be reversible after cessation of exposure. On the other hand, some observations mentioned the progressive nature of the liver changes, when toxic exposure continued, to what was stated to be "chronic hepatitis".

Recent work from Germany (Barateller et al., 1973) reporting a detailed study of 20 PVC workers, brought a very important contribution to our present knowledge on the liver changes induced by this toxic exposure. The authors were alerted to the problem by the occurrence of upper gastrointestinal bleeding in several workers of a PVC producing plant. Esophageal varices, splenomegaly, hepatomegaly, fibrosis of the liver capsule (by laparoscopy), delay in the secretion of bromsulphalein thrombocytopenia were the most prominent findings. The pathologic changes encompassed collagenous transformation of the liver sinusoids, focal activation of Kupffer cells, increased collagen deposition in the portal spaces, portal fibrosis, septal and intralobular fibrosis.

There are a few mentions in the literature on lung changes in PVC workers. One case of pneumoconiosis in a thirty year old worker who had inhaled PVC dust was reported. The lung biopsy showed granulomatous lesions (foreign body type) to be present. Fibrotic changes and altered pulmonary function tests were reported in 96 workers exposed to polyvinyl chloride dust; the changes were more pronounced in persons with long exposure (Vertkin et al. 1970).

In the available literature on vinyl chloride and polyvinyl chloride adverse health effects there were no accurate data on the prevalence of disease in exposed workers, the relative prelavence of the various pathologic changes, their possible association and time sequence.

We have undertaken clinical studies of a number of groups of vinyl chloride exposed workers in order to assess the prevalence of vinyl chloride induced changes and to define the natural history of this occupational disease. We are reporting the results of a survey (March 1974) of 267 workers currently employed in a VC polymerization plant, including virtually the entire current production workforce. 87 former workers were examined as well. In both groups, exposure for some had started in 1948, when the plant opened. Altogether 354 men were examined.

Table 5 shows the duration of exposure for the group and Table 6 the age distribution of the men. More than half were under 40 years, 20% were 51 or older.

METHODS

The medical examination included detailed occupational history, past medical history, complete physical examination, with special attention to the skin, hands and feet, arteries accessible to palpation, liver and spleen.

An Allen test for the assessment of the peripheral circulation in the areas supplied by the radial and ulnar arteries was performed. Each examined worker also had the following:

X-rays of the chest, hands and feet; pulmonary function tests; complete blood cell counts including platelets; blood chemistries including bilirubin, SGOT, SGPT, LDH, alkaline phosphatase, total protein, electrophoresis of serum proteins, fibrinogen split products; alpha fetoprotein, antinuclear antibodies, antimitrchondrial antibodies, antismooth muscle antibodies Australia antigen B by radioimmunoassay and liver antibodies. Urinalysis, including microscopic examination of the sediment was done. All examined workers were also tested for carcinogenic embryonic antigen. In a limited number, studies for chromosome breaks in circulating lymphocytes were undertaken.

RESULTS

Unfortunately, appropriate VC measurements had not been made in the past. We have findings which suggest, however (Table 7), that VC levels may have been considerable at times, sufficient to produce symptoms of acute overexposure; for example, 14 of the 354 workers had had loss of consciousness at one time or another.

In any case, our data indicate that measured levels would be of limited utility, unless integrated over time, since both total exposure and duration from onset of exposure seem to be of importance in relation to likelihood of abnormality. The influence of these variables may be demonstrated in a number of areas.