

Persistent Liver Dysfunction among Workers at a Vinyl Chloride Monomer Polymerization Plant

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Summary

Thirteen workers with persistent abnormalities in one or more liver function tests (LFT) at a vinyl chloride monomer (VCM) polymerization plant were investigated.

Twelve workers were found to have VCM-induced liver dysfunction based on circumstantial evidence. They were employed between 1971 and 1982 when the VCM levels ranged from 1 to 21 p.p.m. After 1982 when the environmental VCM levels were controlled to below 1 p.p.m., no cases of VCM-induced liver dysfunction were detected. In most cases, glutamic pyruvic transaminase was the earliest parameter to be raised. The second most common parameter is serum gamma glutamyl transpeptidase. The latent period ranged from 1 to 13 years. An improvement in their LFT results was shown by 83.3 per cent of workers within 6 months to 2 years after removal from further VCM exposure. For workers who returned to VCM work, their LFT became abnormal again. Liver scans showed hepato and/or splenomegaly in most cases. Liver biopsies on 9 workers were reported as 'non-specific fatty changes' of varying degrees.

These observations highlight the need for continual vigilance with environmental monitoring and medical surveillance of VCM-exposed workers.

Introduction

The detection of hepatic angiosarcoma among workers in a vinyl chloride polymerization plant in the United States¹ led rapidly to surveys of workers in such factories^{2,3}. Since then, industrialized countries throughout the world have become increasingly concerned over the potential health effects of vinyl chloride monomer (VCM). This paper presents the findings on 13 workers with persistent liver dysfunction detected during a medical surveillance programme at a VCM polymerization plant and proposes a systematic plan of investigation.

Materials and Method

A VCM plant was set up in Singapore in the early 1970s to produce polyvinyl chloride (PVC) powder by the polymerization of VCM.

Medical examination for workers at this plant started in 1975 on a voluntary basis. Tests were done to look for evidence of liver disease. These tests comprised the biochemical liver function tests (LFT): serum bilirubin, serum alkaline phosphatase (AP), serum gamma glutamyl transpeptidase (GGT) and the transaminases, serum glutamic oxaloacetic transaminase (GOT) and serum glutamic pyruvic transaminase (GPT). If the initial LFT was normal, similar tests would be repeated annually. If there was one or more abnormalities, the abnormal test(s) would be repeated in 3 months after advising the worker to abstain from alcohol. If the abnormality persisted, the worker would be required to undergo a comprehensive physical examination including biochemical tests such as carcino-embryonic antigen (CEA), alpha foeto-protein (AFP), hepatitis B antigen (HBsAg) and a technetium-99m liver colloid scan. However, this surveillance

programme was not carried out systematically nor was there proper supervision. Liver function tests were analysed at 3 different laboratories. However, the type of LFT abnormality reported was fairly consistent among the 3 laboratories. All these were later rectified by 1983.

All workers who had persistent abnormalities (on at least 1 repeat test) in one or more LFT and with 1 or more years of exposure formed the basis of this report. A detailed occupational, medical and alcohol history was obtained from each worker. A complete physical examination with special attention to the liver, spleen, skin and extremities was done. The laboratory tests included technetium-99m liver colloid scan, HBsAg, CEA and AFP. The latter 3 were done by radioimmunoassay method. Liver biopsy was only done on 9 workers who were still with the plant at the time of this review. The specimens were read by 2 pathologists independently.

Environmental assessments comprising area and personal sampling were carried out on a periodic basis at different sections of the plant. Area sampling had been carried out at monthly intervals since 1976 by the company's safety officer using Kitagawa detector tubes and a photo-ioniser (HNU model P101).

Personal sampling was started in 1983 by our department, using 3M organic vapour monitors. Sampling duration was about 5-7 hours. The samples were analysed by carbon disulphide desorption and gas chromatography in accordance with the NIOSH Method No: P & CAM 178.

The results of the medical and environmental monitoring are discussed in this report.

Results

The Plant

The VCM was purified before it was charged into reactors containing de-ionized water and peroxidase initiators. The reaction took place in batches in the 6 autoclaves (reactors) to produce PVC slurry. At the end of the polymerization, the unreacted VCM was recovered by evacuation. The PVC slurry was further stripped of VCM before it was centrifuged to remove the water to produce PVC powder. The PVC powder was then packed and supplied to other factories where it would be used for the manufacture of PVC pipes, resins, etc. Our 13 workers came from various sections of the plant.

Exposure to vinyl chloride monomer

The highest exposure occurred during reactor cleaning. Here, workers were required to enter the reactors to clean

and scrape the scales formed during the reaction. Although the reactors were purged with air before they were opened, residual VCM still remained in the PVC scales. Prior to 1976, cleaning was done manually and workers were required to enter the reactors after every batch. In 1976, this method of cleaning was mechanized by the use of a high pressure water hose. This reduced the frequency of entry of workers into the reactors from once per batch to once in every 10 batches. The general environmental VCM levels measured in 1976 ranged between 1 and 20 p.p.m. with a geometric mean of 6 p.p.m. No measurements were done before 1976, but we can expect levels to have been higher.

The installation of a VCM recovery system in 1980 further reduced the residual VCM in the reactor after polymerization. All control rooms were air-conditioned by 1982 and fresh air intake for this air conditioned system was taken from the environment away from the process area. In 1983, the residual VCM in the PVC slurry after leaving the reactor was greatly reduced from 10000 p.p.m. to less than 10 p.p.m. after incorporating a stripper to further remove residual VCM. This reduced the VCM exposure at the drying and packing sections. To further reduce exposure, a quality control valve was installed in 1986 to obviate the need to open the reactors during the taking of samples for quality checks.

As a result of the various engineering measures instituted, the general environmental VCM levels were significantly reduced from a geometric mean of 5.9 p.p.m. in 1976 to 1.5 p.p.m. after 1983. However, workers could still be exposed to high, albeit short term levels of 10 p.p.m. especially when entering the reactors and when taking samples.

Personal samples of VCM were much lower (range: 0.1 to 0.5 p.p.m.) than the results of area sampling. They were more representative of workers' exposure during a workshift.

Personal and Occupational Profile

Since 1975, a total of 271 workers had undergone medical examinations. Thirteen of them (4.8 per cent) had persistent abnormalities in their liver function tests. They were males aged between 19 and 55 at the time of the first abnormal LFT result (mean = 28.6 years). All had worked at least one year in the VCM plant. They began employment in the period 1971 to 1982. Their mean exposure duration (at the time of first abnormal LFT result) was 5.1 years (range = 1 to 13). Table 1 gives the summary of the nature of their work and their degree of VCM exposure.

Alcohol consumption was very low except for 1 worker, J. He drank about 3 bottles of toddy (an alcoholic drink obtained from the juice of the cut flower of the coconut palm) every week and brandy/whisky occasionally. Three were ex-drinkers (had stopped drinking for at least 1 year prior to the time of the first abnormal LFT results). Four were teetotalers. The rest were social drinkers.

Medical History

None had a history of jaundice, blood transfusion or Raynaud's phenomenon. Two of the workers were diabetics (J, M). Six workers had symptoms during work. The symptoms were usually non-specific and vague. All had complaints of nausea with four experiencing dizziness as well. Two of the workers (M, G) mentioned that they

Table 1. Nature of work and degree of exposure to vinyl chloride monomer (VCM) in the 13 workers

Section	No of workers	Nature of work	Range of environmental VCM levels (p.p.m.)	
			Before 1983	1983 and after†
Purification	1	Checking for leaks; Monitoring of VCM levels; control the pump valves and condenser	2-21	0.6-2.8
Polymerization	3*	Charging of VCM, catalyst, etc into reactors; Taking of samples for quality checks; cleaning of reactors; opening of valves to release unreacted VCM into storage tanks	2-16	0.7-2.9
Drying and packing	4	Checking and taking of PVC samples for moisture test; washing of slurry tank; packing PVC into bags; general housekeeping	1-11	0.7-2.8
Maintenance including electrical service	5	Maintenance and servicing of pumps, compressors; welding of faulty VCM pipes (including work in a sister company with no VCM exposure)	Exposure varied depending on the type of maintenance work and where it was carried out.	

* One worker had worked for 1 year at the drying section and about 6 years at the polymerization section.

† VCM levels after the implementation of various engineering control measures.

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Table II. Summary of the clinical findings and investigation results of the 13 workers

Worker	Age at first abnormal LFT result	Section	Year joined factory	Symptoms				Drinking history	Duration of exposure (years) at first abnormal LFT result	Type of LFT abnormalities	Liver scan results	Diagnosis
				Dizziness	Nausea	LOW	LOA					
A	25	Purification	1978	-	-	-	-	Drinker (2-3 bottles of beer/month x 5-6 years)	6	Bil	Mild hepatomegaly	† Gilberts with probable VCM-induced liver dysfunction
B	23	Drying & packing	1972	-	+	-	-	Drinker (1-2 glasses of beer/month x 1-2 years. Stopped in 1979)	6	Bil	Hepatomegaly. Moderate liver parenchyma damage	† Gilberts with probable VCM-induced liver dysfunction
C	19	Drying & packing	1982	-	-	-	-	Non-drinker	1	GPT	Mild hepatomegaly	VCM-induced liver dysfunction
D*	24	Polymerization	1971	+	+	-	-	Drinker (1-2 bottles of beer/month. Stopped in 1979)	13	GPT, GGT	Mild hepatomegaly	† VCM-induced liver dysfunction
E	24	Polymerization	1979	-	-	-	-	Non-drinker	4	GPT, GOT	Normal	† VCM-induced liver dysfunction
F	34	Maintenance	1973	+	+	+	+	Ex-drinker (1 glass of beer/week. Stopped in 1974)	7	GPT, GGT, GOT	Mild liver parenchymal damage. Mild splenomegaly	† Probable VCM-induced liver dysfunction
G	22	Drying & packing	1981	+	+	-	+	Non-drinker	2	GPT, GGT, GOT	Mild hepatomegaly	† Probable VCM-induced liver dysfunction
H	30	Maintenance	1973	-	-	-	-	Drinker (1-2 bottles of beer/month. Stopped in 1981)	7	GPT, GGT, GOT	Mild hepatomegaly with early liver parenchymal damage	† Probable VCM-induced liver dysfunction
I	32	Maintenance	1976	+	+	-	+	Ex-drinker (1/2 bottle of beer/month x 14 yrs. Stopped in 1979)	4	GPT, GGT, GOT	Mild splenomegaly	† Probable VCM-induced liver dysfunction
J	55	Maintenance	1971	-	-	-	-	Drinker (3 bottles of toddy/week + brandy/whisky occasionally x 20 years)	7	GPT, GGT, AP	Normal	Alcoholic liver cirrhosis with diabetes mellitus
K	29	Maintenance	1980	-	-	-	-	Non-drinker	3	GPT, GGT, GOT, AP	Normal	VCM-induced liver dysfunction
L	28	Polymerization	1975	-	-	-	-	Ex-drinker (1 litre beer every 2-3 days x 7 years. Stopped in 1977)	5	GPT, GGT, GOT, AP	Mild hepatomegaly	† Probable VCM-induced liver dysfunction
M	27	Drying & packing	1981	-	+	+	-	Drinker (2-3 bottles of beer per month x 4 years. Stopped in 1982)	1	GPT, GGT, GPT, AP, Bil	Hepatomegaly	VCM-induced liver dysfunction with diabetes mellitus

* Worker had worked for 1 year at the drying section and about 6 years at the polymerization section

† Liver biopsy done

Ex-drinker (one who stopped drinking for at least a year prior to date of first abnormal result)

LOW, loss of weight; LOA, loss of appetite; LFT, liver function test; Bil, bilirubin; GPT, serum glutamic pyruvic transaminase; GGT, serum gamma glutamyl transpeptidase; GOT, serum glutamic oxaloacetic transaminase; VCM, vinyl chloride monomer; AP, alkaline phosphatase

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experienced nausea especially during the washing of the slurry tank. Three had loss of appetite while 2 had complaints of weight loss. Worker F had all of the above symptoms. *Table II* summarizes the clinical findings and investigation results of the individual workers.

On examination, all were well built and clinically well. None had any jaundice, hepatosplenomegaly or signs of liver disease, sclerodermatous skin changes or acro-osteolysis.

Hepatitis B antigen, carcino-embryonic antigen and alpha foetoprotein were not present in all 13 workers.

Serial Liver Function Test Results

Table III gives the serial LFT results. Of the 13 workers investigated, 3 workers had only one persistent LFT abnormality, 2 had 2 abnormalities and 8 had 3 or more abnormalities in the battery of tests.

WORKERS WITH ONE LFT ABNORMALITY

Workers A and B had raised bilirubin and worker C had raised GPT. Workers A and B were confirmed to have benign unconjugated hyperbilirubinaemia (Gilbert's syndrome). Their bilirubin levels fluctuated from 1.4 to 2.6 mg/100 ml and remained so even after removal from VCM exposure.

Worker C's serum GPT level became normal after removal from VCM exposure and was raised again on return to VCM work. These findings support the diagnosis of VCM-induced liver dysfunction.

WORKERS WITH TWO LFT ABNORMALITIES

Workers D and E had raised GPT and GGT levels, and GPT and GOT levels respectively. They worked in the polymerization section and their LFT improved when they were removed from VCM exposure. However, their LFT became abnormal within a year of returning to VCM work. As alcohol and viral hepatitis were already excluded as possible causes, these findings were consistent with the diagnosis of VCM-induced liver dysfunction.

WORKERS WITH THREE OR MORE LFT ABNORMALITIES

Eight workers had 3 or more abnormal LFT results. All except M (there were no previous LFT results for comparison) had normal LFT results prior to the development of the persistent abnormalities. These abnormalities developed within 1 to 7 years of starting work (mean 4.5 years).

Three workers showed an improvement in their LFT results within 6 months of removal from VCM exposure and in workers G and K, the results returned to normal. These 2 workers were removed from further VCM exposure within one and a half years of developing the abnormal LFT result. Workers G and M who returned to VCM work re-developed abnormal LFT results. These findings supported the diagnosis of VCM-induced liver dysfunction. In addition, M had diabetes mellitus which was controlled with insulin injections.

The remaining 5 workers had fluctuations in their LFT results. Workers F, H and I were in the maintenance section where the exposure to VCM varied depending on the type and place of work. This may account for the fluctuation in their LFT results. However, where there was documented removal from VCM exposure, their LFT results improved suggesting that they probably had VCM-induced liver dysfunction. In the case of L, we could

not explain the fluctuation in the LFT results, although there was definite improvement when he was removed from further VCM exposure. He probably had VCM-induced liver dysfunction. Worker J had a history of drinking about a bottle of brandy/whisky and 3-4 bottles of toddy at least once a week for the last 20 years. His LFT results fluctuated probably with variation in his drinking habits. There was also no improvement in the results after removal from further VCM exposure, thus suggesting that alcoholic liver cirrhosis was the most probable cause of his persistent liver dysfunction. He also had diabetes mellitus which was well controlled by tolbutamide.

Liver Scan Results

Four workers had hepatomegaly and four had hepatosplenomegaly. Two workers had only splenomegaly. The rest had normal liver scans. Enlargement of the liver and spleen was based on an enlargement of 2 cm or more when compared to the normal spans for liver (15 cm) and spleen (8 cm) in our local population.

Biopsy Findings

Liver biopsies were carried out on 9 workers. The liver tissues showed normal lobular architecture. There were mild to moderate fatty changes. Portal tracts were normal except slightly widened by aggregates of chronic inflammatory cells in the case of worker C. In 3 cases, there were aggregates of mononuclear cells among the fat vacuoles. Kupffer cells appeared active in 3 cases. Sinusoidal dilatation was present in worker I and in workers B and D, the hepatocytes displayed megalocytosis with enlarged darkly stained nuclei. No malignant change was observed. The histological diagnosis was non-specific fatty changes of varying degrees although none was obese clinically, 3 were ex-drinkers, 2 were teetotalers and the rest were social drinkers.

Diagnosis

For the 9 workers who had liver biopsies, three had VCM-induced liver dysfunction and 4 were probable cases as explained earlier. The remaining 2 were confirmed as having Gilbert's syndrome. In addition, they probably had VCM-induced liver dysfunction in view of the abnormal liver scan and biopsy reports which could not be explained by Gilbert's syndrome alone.

Hence, of the 13 workers with persistent liver dysfunction, 6 were caused by exposure to VCM (1 had in addition diabetes mellitus), 6 were probably VCM-induced (2 had in addition Gilbert's syndrome) and 1 had alcoholic liver cirrhosis with diabetes mellitus.

Discussion

Our study showed 13 (4.8 per cent) workers with persistent liver dysfunction. Of these, 12 workers had VCM-induced liver dysfunction (6 possible and 6 probable cases) based on circumstantial evidence and after excluding alcohol and viral hepatitis as possible causes. The discussion centres on these 12 cases.

They started employment in the plant between the early 1970s and 1982. While there were reports of high VCM exposures of about 100 p.p.m. in the 1970s^{1,4,5}, VCM levels in our plant would be considered low, ranging from 1 to 21 p.p.m. before 1983. After the institution of the various engineering control measures, the VCM exposure levels in the production area ranged from 0.6 to 2.9 p.p.m. and were less than 1 p.p.m. most of the time since 1983. These levels were well below the recommended ACGIH threshold limit values of 5 p.p.m.⁶. However, the VCM exposure could be high during the washing of the slurry tank as 2 workers had complaints of nausea whilst carrying out such work (the odour threshold being 2000 to 5000 p.p.m.⁷). Workers employed after 1982 showed no evidence of liver dysfunction suggesting that VCM exposure with regard to inducing liver dysfunction has a biological threshold. It appears that VCM-induced liver dysfunction is unlikely to occur at levels below 1 p.p.m. The strict supervision on the usage of air-line respirators during reactor cleaning in 1983 may be an added factor. Since 1974, most of the countries having PVC manufacturing plants have lowered their threshold limit values for VCM. The United States and Sweden had lowered their threshold limits (8 hours time weighted average for VCM to 1 p.p.m.)⁸.

Raynaud's phenomena, acro-osteolysis and sclerodermatous skin changes had been associated with past heavy exposures to VCM^{1,7,9}. It is not surprising that these were not detected in our workers as the general VCM levels in our plant are low.

Aryanpur⁸ and Lili³ found the liver changes (hepatomegaly, tenderness on palpation, abnormal LFT results) to be reversible after cessation of exposure. The liver progressed to what was stated to be 'chronic hepatitis' when toxic exposure continued. Clinically, the liver was not palpable in all our cases although the liver scan showed hepato/splenomegaly of varying degrees in 10 of our workers.

It is reported that the ability of the traditional biochemical tests to predict the presence of underlying histological hepatocellular damage is at best 80-85 per cent accurate¹⁰. The most effective test was GPT followed by GGT. Other studies showed that GGT was the most useful test in detecting abnormality and reflecting the extent of liver damage^{11,12}.

On reviewing the serial LFT results of our workers, we noted an elevated GPT to be the most common initial finding with GGT being the second most frequently raised parameter.

In 9 of our cases, the liver dysfunction progressed rapidly upon developing the first abnormality. (The rise in LFT results were 1-2 times normal as compared to many more times in cases of infective hepatitis.) It took from 6 months to 4 years for the second parameter to become abnormal if the affected workers were not removed from further VCM exposure.

Of our workers 83.3 per cent had improvements (10 out of 12) in their LFT results within 6 months to 2 years after removal from further VCM exposure. For the 3 workers who had only one LFT abnormality initially, their LFT returned to normal on cessation of exposure. Of the 8 workers who returned to VCM work, their LFT results became abnormal again within 3 months to a year. These observations suggest that if progression of liver dysfunction is to be avoided, a worker should be removed from further VCM exposure upon developing an

abnormality. The elevated LFT results would then improve, indicating reversibility of dysfunction, at least in the early stages.

In our 12 workers, the latent period for the development of liver dysfunction (from first exposure to date of first abnormal LFT result) ranged from 1 to 13 years (possible cases: range = 1 to 13 years; median = 2.5 years; probable cases: range = 4 to 7 years; median = 6.0 years). This latent period is much shorter than that reported for the development of angiosarcoma, which takes at least 10 years^{7,13,14}. Thus, for the present, it is not known whether our workers will suffer from angiosarcoma of the liver at a later time. The full magnitude of this site-specific cancer risk among our workers will only be determined following full lifetime observation. One worker (H) has died of a cerebrovascular accident after a fall at home.

The chronic manifestations of VCM injury to the liver were well illustrated by the sinusoidal dilatation with sinusoidal cell activation, subcapsular fibrosis, peliosis hepatitis, cirrhosis and finally angiosarcoma¹². The liver biopsies of our 9 workers were reported as 'non-specific fatty changes' of varying degrees although 2 were teetotallers, 3 were ex-drinkers, and the rest were social drinkers. Whether these were a misdiagnosis or the precursor stage of angiosarcoma is yet to be determined, as these features may be found in a variety of clinical conditions such as obesity, diabetes mellitus, non-A, non-B hepatitis and vinyl chloride toxicity^{15,16}.

Conclusion

Exposure to VCM in industrial settings can produce hepatic fibrosis with angiosarcoma of the liver as a late manifestation^{3,8}. The early detection of such liver disease may be of great industrial and public health importance because it remains a possibility that very early liver abnormalities may be reversible or non-progressive after workers are removed from further VCM exposure.

Our cases of VCM-induced liver dysfunction are a relatively young group of workers in which little disease and few deaths would be expected to occur. All were below 35 years of age at the time of diagnosis. These data indicate the need for continual vigilance with environmental monitoring and medical surveillance.

Until specific tests for hepatic angiosarcoma or VCM-induced liver damage are available, we believe that our medical surveillance programme will aid detection of liver abnormalities in a possibly reversible or curable state (Table IV). Our programme involves early removal of workers with trial re-exposure. In addition, we also started a system whereby all workers exposed to carcinogens are followed-up on a regular basis for life and are registered with the local Cancer and Death Registries who will notify us upon their deaths or if they develop cancer. This may later provide useful information on the cancer risk among our exposed workers.

Acknowledgements

We are grateful to the Permanent Secretary (Labour) for permission to quote from departmental records and Mrs Marsita Zain for typing the manuscript.

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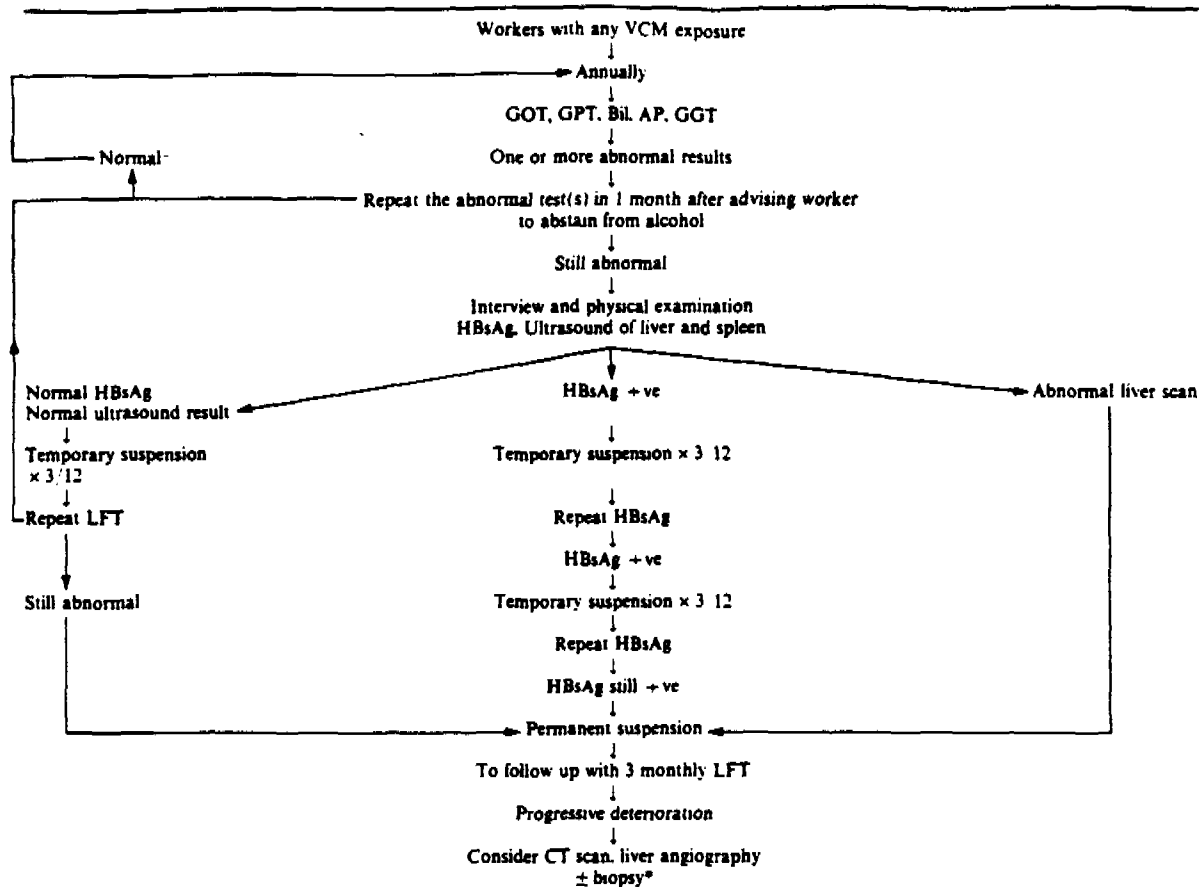
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Table III Follow-up liver function test results in 13 workers exposed to vinyl chloride monomer polymerization

Worker	Year of first abnormal result	First abnormal result	Serial abnormal liver function test results															
			Before 81	Jul 81	Jul 82	Aug 83	Jul 84	Oct 84	Jan 86	May 86	Jun 86	Oct 86	Feb 87					
A	1984	Bil	Before 81 NA	Normal	Normal	Normal	Bil	Normal	Normal	Bil*	Bil	Bil	Bil					
B	1978	Bil	Before 78 NA	Aug 78	Normal	Mar 79	Oct 79	Aug 80	Jul 81	Oct 81	Jun 82	Aug 83	May 85	Jan 86	Jun 86	Oct 86	Feb 87	
C	1983	GPT	Before 83 NA	Aug 83	Apr 84	Jul 84	Oct 84	Jun 85	Normal	Normal	Normal	Bil	Normal	Bil	Bil*	Bil	Bil	Bil
D	1984	GGT	Before 77 NA	Aug 77	Sep 78	Oct 79	Aug 80	Aug 81	Jul 82	Jul 84	Oct 84	May 85	Jun 86	Oct 86	Jul 87			
E	1983	GGT	Before 81 NA	Jul 81	Jul 82	Aug 83	Apr 84	Jul 84	May 85	Jan 86	Mar 86	Jun 86	Jul 87	Died of cerebrovascular accident after a fall at home				
F	1980	GPT	Before 80 NA		Oct 80	Mar 81	Jul 82	Mar 83	Jul 84	Oct 84	Apr 85	May 85	Sep 85	Oct 85	Mar 86	Aug 86	Jan 87	Apr 87
G	1983	GPT	Before 82 NA	Aug 82	Aug 83	Mar 84	Aug 84	Nov 84	May 85	Oct 86	Oct 87							
H	1980	GPT	Before 77 NA	Aug 77	Sep 78	Oct 79	Aug 80	Oct 80	Feb 81	Jul 81	Oct 81	Jul 82	Sep 82	Jul 84	Oct 84	Jan 85	May 85	Oct 85
I	1980	GPT	Before 79 NA	Oct 79	Aug 80	Oct 80	Feb 81	Jul 81	Jul 82	Sep 82	Aug 83	Jul 84	Nov 84	Jan 85	May 85	Oct 85	Jan 86	Oct 86
J	1978	AP	Before 77 NA	Aug 77	Aug 78	Sep 78	Mar 79	Sep 80	Feb 81	Jul 81	Oct 81	Nov 81	Dec 81	Jul 82	Aug 83	Jul 84	Oct 84	May 85
K	1983	GGT	Jul 81 Normal	Jul 82 Normal	Aug 83 GGT	Jul 84 GGT	Oct 84 GGT*	May 85 GGT	Oct 85 GGT	Jan 86 Normal	Resigned							
L	1980	GPT	Before 78 NA	Aug 78	Oct 79	Aug 80	Oct 80	Feb 81	Jul 81	Jul 82	Sep 82	Oct 82	Jan 83	Mar 83	Apr 83	Aug 83	Jul 84	May 85
M	1982	GPT	Before 82 NA	Jul 82	Sep 82	Jan 83	Mar 83	Apr 83	Jul 83	Aug 83	Oct 83	Jul 84	Oct 84	Dec 84	May 85	Dec 85		

NA, Not available. The LFT normal range: bilirubin (Bil) < 1 mg/100 ml, alkaline phosphatase (AP) 25-100 IU/l, serum glutamic pyruvic transaminase (GPT) < 30 IU/l, serum glutamic oxaloacetic transaminase (GOT) < 30 IU/l, serum gamma glutamyl transpeptidase (GGT) < 50 IU/l. * Removal from VCM exposure. ** Returned to VCM work.

Table IV. Medical surveillance of vinyl chloride monomer (VCM) exposed workers



* Consent for a biopsy should be obtained from the worker.

Suspension, removal from further VCM exposure.

LFT, liver function test: GOT, serum glutamic oxaloacetic transaminase; GPT, serum glutamic pyruvic transaminase; Bil, bilirubin; AP, alkaline phosphatase; GGT, serum gamma glutamyl transpeptidase; HBsAg, hepatitis B antigen.

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