Polyvinyl Chloride Pneumoconiosis: Epidemiological Study of Exposed Workers

Giuseppe Mastrangelo, M.D.; Maurizio Manno, M.D.; Guido Marcer, M.D.; Giovanni Battista Bartolucci, M.D.; Carla Gemignani, M.D.; Giulia Saladino, M.D.; Lorenzo Simonato, M.D.; and Bruno Saia, M.D.

Among 1216 workers who were employed in a polyvinyl chloride production factory and who had had no previous dust exposure elsewhere, 20 cases of pneumoconiosis were found. Chest x-ray abnormalities were characterized by limited profusion, irregular type and low gravity. All 20 workers had been exposed to high PVC dust levels. The chest x-ray changes were observed after a minimum exposure of five years and, in a small percentage of cases, were associated with slight restrictive respiratory function impairments. Moreover, in the whole group of workers 388 cases (31.9%) were found with non-specific x-ray abnormalities mainly related to age and smoking.

Polyvinyl chloride (PVC) dust is particularly relevant to a consideration of the anatomical, clinical and radiological signs of pneumoconiosis found in workers exposed to "inert" dusts because of the high annual production of PVC and the number of exposed workers.

Within the framework of a national survey on vinyl chloride hazards to humans, co-ordinated by the Chemical Workers Unions, the authors examined the working population of plants producing PVC in Porto Marghera, Italy. One group of workers was exposed to PVC dust; another group worked in departments polluted only by vinyl chloride monomer (VCM).

The characteristics and prevalence of pulmonary radiological abnormalities found are reported and their pathogenic mechanism is discussed in this article.

Methods and Population

Environmental dust samples were collected and both concentration in air and particle size were measured. In the drying, sacking and blending departments, PVC dust concentrations were higher than the TLV (10 mg/m³ of total dust) in about 60% of the samples. In the polymerization departments, no concentration higher than the TLV was found. In the samples taken, particles with diameters of 1 μm to 5 μm constituted 4.5% to 30.9% of total dust weight.

All the workers answered the European Coal and Steel Community (ECSC) questionnaire for chronic bronchitis and emphysema,[1] and were submitted to chest x-ray (according to ILO standards) and spirographic examination (Godart-Expirograph, Bilthoven, Holland). Chest x-ray films were read by two independent physicians utilizing the ILO/UC Pneumoconiosis Classification, 1971.[2] For statistical analysis, a consensus reading was used. For each worker examined, present and past working histories were carefully recorded and particular consideration was given to previous dust exposure. Of the total, 1,216 subjects had had no past exposure to organic or inorganic dusts; 731 had been exposed to PVC dust (employed in drying, sacking and blending of polymer), and 485 had been exposed to monomer alone.

Results

Table 1 shows the mean and standard deviation of age and of length of exposure, the number of subjects and the percentage of smokers in the two groups; those exposed to PVC dust and those not exposed. There are no significant differences in age and smoking habits, but the duration of exposure is higher in the workers not exposed to dust.

In 20 subjects (1.6%), we diagnosed a typical pneumoconiosis, i.e., chest x-ray changes (irregular opacities or frankly micronodular images) of at least class 1 profusion, according to the ILO/UC classification. The

From the Institute of Industrial Medicine, University of Padova, Padova, Italy

Table 1. — Age, Exposure, Number of Subjects and Percentage of Smokers in PVC Dust Exposed and Non-Exposed Groups.

	Age (Years)		Exposure (Years)		No. of	% of
	Mean	S.D.	Mean	S.D.	Subjects	Smokers
PVC dust exposed group	37.7	8.8	6.1	4.0	731	73.9
PVC dust non-exposed group	35.7	8.5	8.6	4.5	485	68.7

mean age of this group was 44.9 (± 5.2) years and the mean length of exposure was 11.6 (± 5.4) years; 16 subjects (80%) were either smokers or ex-smokers.

Table 2 summarizes the distribution of cases in relation to age and length of exposure. In all age groups there is an increase of disease prevalence associated with increasing length of exposure. In the case of x-ray changes, 16 subjects had class 1/0 profusion; two cases, class 1/1; one case, class 2/1; and one case, class 2/2. In spite of age and the considerable exposure, the majority of cases observed were in a low profusion category, thus indicating a slow evolution of the disease. The changes were characterized by irregular opacities: ten cases were type "s"; three cases, type "t"; six cases, type "p"; and one case, type "r." The opacities were diffused, mainly over median lobe areas. Two subjects showed slight restrictive spirographic impairment. All had had jobs which involved high airborne dust levels (mostly drying and sacking) for at least five years. None of the 20 subjects was in a group unexposed to dust. None of them had experienced previous occupational exposure to organic or inorganic dusts. We considered the above alterations to be PVC pneumoconiosis.

In 388 subjects (31.9%) we found slight chest x-ray alterations consisting of linear or irregular vanishing opacities, or both, classified as class 0/1 profusion; the remaining 808 subjects were class 0/0.

Table 3 reports the total population distribution, excluding 20 subjects with PVC pneumoconiosis. Results are presented in a two-way table — each entry reports the number of observations. Samples are classified according to age (three classes) and polyvinyl chloride dust exposure: "PVC +" represents presence and "PVC −," absence. In the table, "x-ray +" indicates the classes with class 0/1 profusion. To assess the influence of both risk indicators we performed a two-way analysis of variance for proportions (Snedecor and Cochran).[1] The column mean is adjusted for row effects and vice versa. We also tested whether row and column effects were additive or whether interaction was present. The results (Table 4) show that both age and exposure were significant factors influencing x-ray abnormalities — since the interaction is not significant, the effects are simply additive.

The square of a multiple-partial association coefficient for qualitative data was calculated to measure the degree of association between the dependent variable (x-ray changes) and each of the two predictor variables (age and exposure).* Age and exposure are alternately held constant. Age is a most important factor. When exposure is held constant, 33.2% of the x-ray changes are shown to depend upon age; when age is held constant, 6.5% are shown to depend upon exposure.

Table 5 summarizes the distribution of cases (same subjects as Table 3) according to smoking habits in workers exposed and not exposed to PVC dust. In Table 6, the two-way analysis of variance for proportions shows that both risk indicators have a significant influence on chest abnormalities.

There is no interaction of factors. Habitual smoking plays a major role and, when exposure is held constant, is shown to be responsible for 17.1% of the changes. When smoking habits are held constant, exposure to PVC dust is shown to be responsible for 8.9% of the x-ray abnormalities.

Discussion

X-ray changes of at least class 1 profusion, found in 20 subjects, are considered PVC-induced pneumoconiosis. This claim is based on the following: (1) Reading of the chest films was performed by two independent physicians, who were not informed of the age, job, length of exposure or clinical history of workers. A consensus reading was required for the pneumoconiosis diagnosis. (2) Special attention was given to the absence of any previous occupational exposure to organic or inorganic dusts. (3) The changes were found in workers who had experienced prolonged exposure in departments with demonstrated PVC dust pollution. The length of exposure was about 12 years on the average and never less than five. No radiographic signs of pneumoconiosis were found in subjects working in departments free of PVC dust, although the average period of exposure was longer

Table 2. — Distribution of PVC Pneumoconiosis in Age and Exposure Classes (The percentage on the group of same age and exposure is shown in parentheses).

PVC Dust Exposure (Years)	Age (Years) ≤ 30	31-40	41-50	> 50
< 5	—	—	—	—
5-10	—	2 (2.4)	5 (8.3)	1 (5.9)
> 10	—	2 (3.8)	8 (9.0)	2 (9.5)

Table 3. — Distribution of Cases with Class 0/1 and 0/0 Profusion According to Age and PVC Dust Exposure.

	Age (Years) ≤ 30	31-40	41-50	> 50
PVC +				
X-ray +	20	78	123	35
X-ray -	133	197	108	17
PVC -				
X-ray +	17	45	55	15
X-ray -	119	158	67	9

Table 4. — Two-Way Analysis of Variance for Proportions (Same cases as in Table 3).

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Squares	F
Age	3	30.9713	10.3238	53.81*
PVC dust exposure	1	0.7843	0.7843	4.09†
Interaction	3	0.9882	0.3294	1.72
Error	1188	227.9403	0.1919	

*p < 0.01
†p < 0.05

Table 5. — Distribution of Cases with Class 0/1 and 0/0 Profusion According to Smoking and PVC Dust Exposure.

	Nonsmokers	Smokers
PVC +		
X-ray +	40	216
X-ray -	147	308
PVC -		
X-ray +	25	107
X-ray -	127	226

in these workers (Table 1).

Previous experimental data on animals and reports of human lung biopsy indicated the possibility that the impairments described above can be attributed to PVC dust inhalation. In 1970, Szende et al[5] reported a case of pneumoconiosis in a worker exposed to PVC dust. Chest x-ray films showed small patches which became denser toward the hilum. Biopsy revealed multiple granulomatous centers consisting of foreign amorphous material surrounded by granulation tissue composed of histiocytes, lymphocytes, plasma cells, eosinophils and foreign body giant cells. Such centers were associated with moderate diffuse fibrosis.

A histiocytic-macrophage reaction in alveolar septa, observed by Frongia et al[6] in guinea pigs exposed to PVC dust in a sacking department, developed into lesions with multiple granulomatous centers with giant cells during the course of exposure. Rats exposed to a similar working environment developed marked thickening of interalveolar septa with an evolution similar to that in the guinea pigs.

Such experimental and pathologic data confirm the results of this epidemiological study. Lung changes are directly related to PVC dust while VCM exposure alone fails to cause these changes. Therefore, unlike Lilis et al,[7] we believe that pulmonary changes are not pathogenically similar to other vinyl chloride induced abnormalities (fibrosis of the liver, scleroderma-like skin changes, peripheral vascular damage).

In the present study there was only a 1.6% prevalence of pneumoconiosis in a total population of 1,216 subjects. Nevertheless, in workers exposed to effective risk of pneumoconiosis (731 subjects) the prevalence rose to 2.7%. Waxweiler et al[8] reported pulmonary fibrotic changes associated with respiratory function impairments in 96 subjects exposed to PVC dust. Lilis et al[7] studied the chest x-ray films and changes in respiratory function of groups of PVC production workers who had experienced exposure of different degree and length. They found a 4.3% to 22.7% prevalence of chest x-ray changes (small, linear, reticular and/or rounded opacities) and spirographic impairments related to the duration of exposure and to the presence of chest x-ray changes. As these authors did not employ either the ILO/UC International Classification or any other, no comparison concerning the reported prevalence can be made with the present data.

Apart from 20 cases of pneumoconiosis, mild nonspecific alterations (profusion of 0/1 class) were found both in the group exposed to PVC dust and in the group exposed to VCM alone. Such changes are related mainly to age and smoking habits, and the role of exposure is minor.

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Table 6. — Two-Way Analysis of Variance for Proportions (Same cases as in Table 5).

Source of Variation	Degrees of Freedom	Sum of Squares	Mean Squares	F
Smoking	1	7.8850	7.8850	37.31*
PVC dust exposure	1	1.7847	1.7847	8.44*
Interaction	1	0.1023	0.1023	< 1
Error	1192	251.9125	0.2113	

*p < 0.01