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ASBESTOSIS IN A WORKER ENGAGED IN AUTOMOBILE UNDERCOATING

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THE term pneumoconiosis applies to diseases of the lungs caused by inhalation of foreign material. A more specific diagnosis will depend upon correct identification of noxious substances in air samples, sputum and, whenever possible, lung tissues. Among the occupational hazards causing penumoconiosis, asbestos has been identified in exposed mine workers, workers handling insulating materials composed largely of asbestos, workers in the brake-lining industry and workers engaged in asbestos spinning, carding and weaving.[1] Although asbestos bodies have been found in the lungs or sputum of patients with no evidence of pulmonary disease or heavy exposure,[2] the presence of asbestos bodies in the lungs of a worker in an occupation hitherto not connected with asbestos makes the following case worth reporting.

CASE REPORT

F.T., a 41-year-old automobile mechanic, was admitted to the hospital in the vicinity of Boston on December 15, 1958, with an acute febrile disease. He complained of cough, shortness of breath and yellowish expectoration. He had been well until several weeks previously, when he had noted the gradual onset of productive cough, weight loss and excessive fatigue.

On admission he was febrile and coughing. The temperature was moderately elevated. On physical examination there was a congenital anomaly of the right arm and cyanosis of the ears." Rales and rhonchi were present over both lungs. There was no clubbing of the fingers. Laboratory studies showed "yeastlike bodies" in the sputum. Gastric washings were negative for acid-fast bacilli, and a Papanicolaou smear was negative for malignant cells. Blood studies showed a hematocrit of 47 per cent, a white-cell count of 10,800, with 72 per cent neutrophils, and a sedimentation rate of 7 mm. per hour. A tentative admission diagnosis of pneumonitis was made.

X-ray films taken on the following day showed diffuse bilateral infiltration of the lungs by minute, discrete nodular densities and considerable accentuation of the peribronchial fibrovascular markings, particularly of the lower lobes. The cardiac contours were not clearly defined, being partially obscured by the infiltration. There was considerable bilateral adenopathy.

A bronchoscopy and bronchography performed on December 17 showed "tracheobronchitis." Thoracotomy in the region of the left lingula on January 5, 1959, revealed that the lung had a fine, granular appearance, suggesting pulmonary fibrosis. Two wedge biopsies from the lingula and the lower lobe showed, microscopically, many areas of foreign-body reaction as well as a few typical asbestos fibers. The foreign-body reaction appeared most marked around areas that stained blue and contained unidentifiable homogenous material. Marked connective-tissue reaction was present. There was no evidence of cancer, tuberculosis or Boeck's sarcoidosis.

The patient made an uneventful recovery, his fever subsided and he was discharged on January 9 greatly improved. He continued to have some greenish tenacious sputum and slight shortness of breath, with wheezing. He returned to work to the same company and was assigned as a parts distributor in the Service Department. On re-examination he still had a moderate cough, with slight dyspnea on exertion, 12 months later.

The occupational history revealed that this man was engaged in his present occupation at the same company as an automobile mechanic for ten years. Previously, he had worked for two years at a saw-manufacturing company, where he operated a cold press, and before this he had worked at a gasoline station. He had a complete physical examination, including a chest film, which was interpreted as normal, when he was first hired ten years before admission to the hospital.

His work consisted of the servicing of new automobiles, essentially from one American car manufacturer.

With the exception of fiberglass in small quantities, no material of significance in occupational diseases was used by this worker in his job.

In addition to his routine duties for the last six years he had been engaged on a contract basis in applying an asbestos-base undercoating on new cars.

This operation was performed in a wing of the garage measuring 15 by 26 feet and closed at both ends by wide sliding doors, which permitted the entrance and exit of automobiles. There were large windows at one side of this wing. The undercoating operation was carried out by means of a hand-type spray gun operated at a pressure of 110 pounds per square inch and directed against the underside of the raised car. No mechanical ventilation and no respiratory protective devices were in use during this operation. A considerable amount of the tarry asbestos-base material was dispersed through the work area and onto the worker's clothes and face. The undercoating material was pumped from a 55-gallon drum. Each car required approximately 4 gallons of the material and thirty to sixty minutes of spraying time. About 1 to 3 cars were sprayed daily by the patient during the first five years, and less during the last year.

Since the illness suggested the possibility that the undercoating of automobiles was responsible for or contributory to the acute pulmonary disease, it was deemed advisable to investigate the undercoating operation presently performed by many automobile sales and service agencies in Massachusetts. To obtain background information, contact was made with the industrial-hygiene units of the large automobile as-

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anatomic findings. The material that the patient used contained asbestos of various fiber sizes in an asphalt base and was held in a mastic form by a solvent of the petroleum-distillate type. Lung biopsy revealed the presence of asbestos bodies of characteristic appearance. The unusual finding in this case was the presence of numerous amorphous bluish-staining deposits fairly widely dispersed throughout the specimen and surrounded by inflammatory and connective-tissue reaction. The x-ray appearance of finely granular infiltration throughout both lower lobes and the possibility that the sputum also contained asbestos bodies ("yeastlike bodies") indicate that the lung process in this man was of a chronic nature consistent with pneumoconiosis and the direct local action of foreign material such as asphalt and asbestos. This is also suggested by some of the air tests obtained in the survey.

From the point of view of occupational health it is significant that the patient, in contrast to the workers observed in the survey, had been daily and continuously engaged in the undercoating operation for more than five years, that no history of previous lung disease or exposures to noxious material could be demonstrated and that the survey had shown that the spraying procedure was accompanied by considerable back splash into the breathing zone of the worker.

The cross-sectional survey here reported indicates that under prevailing methods of operation, the use of an asbestos-base undercoating, when limited to short exposures, has not resulted in an occupational hazard. However, if this operation is performed continuously without adequate ventilation and protective respiratory equipment it may expose the worker to the potential danger of pneumoconiosis.

As a protective measure, it is therefore recommended that workers using asbestos-base undercoating material over a period of ten hours weekly or more for periods longer than one year should observe the following rules:

Pre-employment and periodic x-ray studies of the chest, with the use of large-sized films (14 by 17 inches) should be made.

When possible the workers should be rotated.

Approved respiratory devices, with additional protection for the eyes, should be worn.

Mechanical ventilation should be provided in the immediate vicinity of the work area.

Summary

On the basis of a clinical and pathological study of a case of pneumoconiosis in a worker engaged in the undercoating of automobiles with an asbestos-base compound, a cross-sectional survey of this operation in the Commonwealth of Massachusetts was undertaken. Appropriate recommendations to protect workers with prolonged and continuous exposure to this operation are made

We are indebted to R. E. Farrand, M.D., of Fitchburg, for permission to report this case and to H. J. Sparling, M.D., pathologist, Burbank Hospital, Fitchburg, for review of the pathological specimens

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NEPHROTIC SYNDROME CAUSED BY PROBENECID*

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THE nephrotic syndrome has been reported to occur in the course of therapy with a number of drugs including trimethadione,[1,2] paramethadione,[3] mercurial diuretics,[4] tolbutamide[5] and perchlorate,[6] as well as in association with sensitivity to poison oak[7] and bee sting.[8]

In the case reported below, the nephrotic syndrome appeared to be the result of a reaction to probenecid.¶ Although proteinuria and renal failure are known to occur in the course of chronic gout, the nephrotic syndrome has not heretofore been reported as a result of gouty nephritis or in association with probenecid therapy.

Case Report

A 52-year-old man was first seen at the Grace-New Haven Hospital in January, 1954, because of acute arthritis of the right knee. Physical examination was negative except for the painful swelling of the knee. The blood pressure was 118/85. Urinalysis was negative. The nonprotein nitrogen was 38 mg., the fasting blood sugar 100 mg., and the serum uric acid on 2 occasions 9.0 mg. and 9.9 mg. per 100 ml. The acute attack of gout responded in 24 hours without specific therapy and he was discharged on probenecid, 1 gm. twice a day, and a low-purine diet. Eighteen days later he experienced urticaria, which subsided when probenecid was discontinued. He was then advised to stop probenecid and to take colchicine, 1.2 mg. daily, as prophylaxis against further acute attacks of gout.

In March, 1954, probenecid, 1 gm. daily, was restarted. When next seen in May, 1954, the patient had gained 10 kg. (22 pounds), but no evidence of edema was present. Two weeks later the weight had increased further, and gross pitting edema was noted. No urinalysis was done at that

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¶In the form of Benemid, Merck Sharp and Dohme, West Point, Pennsylvania.

semblers and other state organizations responsible for the investigation of occupational health hazards. The case history was summarized and forwarded to these organizations with a request for information regarding whether similar cases had been observed in the past. None of these agencies had knowledge of any similar experiences. The information revealed that in plants outside of Massachusetts, an undercoating compound that contained a rubber or cork base was in use. In Massachusetts most of the automobile agencies are using an undercoating material that contains asbestos in an asphalt medium.

The undercoating compounds used by the dealers included in this survey were manufactured by 5 different companies. The material consisted of an asphalt base containing approximately 50 per cent of asbestos by weight. About 10 per cent of the constituents were fillers such as clay. The material was of a mastic consistence, with a petroleum distillate such as a high-flash naphtha or kerosene as the solvent.

Fifteen different types of American automobiles sold and serviced in Metropolitan Boston were selected for a cross-sectional survey to study the undercoating operation and its potential hazards. Fifty-five automobile sales and service agencies were visited to obtain information on the number of cars undercoated annually, the number of employees engaged in this operation, the methods of operation, the protective equipment worn by the operators and the composition of the undercoating compound being utilized.

The survey revealed that the number of cars undercoated by these establishments ranged from as low as 2 to as high as 600 per year. There was a recent tendency to reduction in the amount of undercoating as compared with previous years, since most of the new cars are delivered undercoated by the assemblers.

The undercoating operation was carried on as a rule by 1 or 2 employees at the close of the workday, when the operator performed the undercoating operation on his own time, with extra compensation for each car undercoated. The location of the operations varied at the different agencies. In most cases a section of the garage area was set aside for the operation, which might be performed in an open area or segregated from the main repair operations by means of canvas curtains used to prevent the material, during spraying, from lodging on other automobiles in the area. About 25 per cent of the agencies had separate workrooms used exclusively for this operation. The undercoating material was usually received in 55-gallon drums and dumped into 275-gallon storage tanks. Under a pressure of approximately 100 pounds per square inch the mastic material was forced through a spray gun of the type used in spray painting. Parts of the automobile that were not undercoated were protected during spraying by masking tape. The wheels were removed, and the automobile raised by means of hydraulic lift. The operator sprayed the underside of the automobile. The actual spraying operation required approximately fifteen to thirty minutes. There was considerable back splash of the material, striking all parts of the operator, including the face. This contamination resulted in a reluctance of workers to undertake this work as a full-time occupation and led to frequent rotation of workers in this particular job. General means of ventilation such as a wall fan were provided at a few locations. It was noted that no special precautions were taken to prevent the inhalation of the dispersed undercoating compound. Some of the operators were provided with plastic face shields or coated their faces with petroleum jelly.

Industrial-hygiene units that had surveyed the undercoating operations in the past were interested primarily in the solvents used in the undercoating material. Their analysis did not reveal any substantial hazard attributable to these solvents. Our survey was directed at determining whether asbestos constituted a hazard after the prolonged use of certain undercoating compounds. Atmospheric samples were obtained during the actual spraying of the undercoating material. The average concentrations varied from 500,000 to 1,500,000 particles of asbestos per cubic foot of air. Microscopical analysis showed that the asbestos was present in the form of dust particles, less than 10 microns in length. The present maximum allowable concentration for asbestos dust particles less than 10 microns long dispersed in the working atmosphere, as set by the American Conference of Governmental Industrial Hygienists, is 5,000,000 particles of asbestos per cubic foot of air. The maximum allowable concentration for long fibers greater than 10 microns in length has been tentatively set in Massachusetts as 1,000,000 fibers per cubic foot of air. The atmospheric contamination due to asbestos in the operations surveyed, based on available standards, was not excessive. It was noted that in some cases the air samples picked up asbestos particles surrounded by an asphalt coating.

Additional information about the health of certain individual operators was obtained. At establishments where at least 150 cars were undercoated annually it was recommended that an x-ray film of the chest (14 by 17 inches) be taken of each employee engaged in this operation. In some establishments arrangements were made with the local health unit for free x-ray studies; in others, the expenses were underwritten by the employer. Twenty-five of the employees involved had such films taken. All x-ray studies were interpreted as negative.

Discussion

The observations here recorded raise the question whether the use of an undercoating material containing asbestos had a direct relation to the clinical and