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Extrinsic allergic alveolitis in a sawmill worker

A case report

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TERHO EO, HUSMAN K, KOTIMAA M, SJÖBLOM T. Extrinsic allergic alveolitis in a sawmill worker: A case report. *Scand j work environ health* 6 (1980) 153-157. Extrinsic allergic alveolitis, with acute attacks of the disease, developed in a sawmill worker. The diagnosis was based on the history of exposure to moldy wood, on the symptoms and clinical signs, and on physiological and radiographic findings. A microbiological analysis of the ambient air of the patient's work environment showed that the air contained a considerable number of spores, both of fungi and actinomycetes. The most common fungal genus was *Aspergillus*. *Thermoactinomyces vulgaris* predominated the actinomycetal flora. The patient's serum contained precipitins to *Thermoactinomyces vulgaris* and *Aspergillus fumigatus*. Based on the microbiological and serological data, it was concluded that the causative agent(s) of the disease was either *T vulgaris* or species of the genus *Aspergillus*.

Key terms: aerobiology, allergic alveolitis, *Aspergillus* species, moldy wood, occupational disease, precipitins, *Thermoactinomyces vulgaris*.

Extrinsic allergic alveolitis (8) is a form of hypersensitivity lung disease in which the allergic reactions take place in the peripheral parts of the bronchopulmonary tree and give rise to a defect in gas exchange. Various biological dusts with sufficiently small particle size for penetration into the alveoli may serve as allergens. Exposure stimulates the formation of circulating precipitating antibodies to the offending allergens. Thus, the main immunologic mechanism of the disease is considered to be the immune-complex mediated (type III) reaction, although other immune reactions may also be involved (9).

During less than 20 a several differently named alveolitis have been reported (8, 12), most of them being occupational diseases in nature. Rather few reports have been published concerning alveolitis associated with the wood-processing industry (3, 4, 13). In the present communication we report a case of extrinsic allergic alveolitis that developed in a Finnish sawmill worker.

Case report

Clinical history and findings

The patient was a 31-year-old previously healthy, male nonsmoker who had worked in a sawmill for more than 10 a. No allergic diseases were reported in relatives.

In the beginning of March 1978, he started to experience mild fatigue with a dry cough. Within a short period of time he lost about 10 kg of body weight and

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developed breathlessness while working. In the beginning of April 1978, in the evening after a workday, he had a fever of 40° C, a dry cough, and shortness of breath. The fever continued at home for several days, after which he sought the help of a doctor. Dyspnea at rest was noticed and basal crepitant rales were heard over both lungs. The condition was considered an infection and doxycycline treatment was prescribed. This treatment had no effect on the fever, and after a few days the antibiotic was changed to amoxycillin, whereafter the fever and other symptoms disappeared during one week's sick leave.

On the evening of the second day after his return to work he again experienced the same symptoms and the condition cleared up during a new sick leave. The same situation occurred two more times;

each time the symptoms reappeared shortly after his return to work. During the third attack of the disease, a chest radiograph was taken which showed minimal diffuse densities in both lungs. The erythrocyte sedimentation rate was 22 mm/h. After the fourth attack the patient was admitted to the hospital.

On admission, about one month later, at the end of May 1978, the patient was symptomless and physical findings, including breath sounds, were normal. A chest radiograph was normal. Mantoux tuberculin tests with 1 and 10 TU were negative, and so were skin prick tests with common inhalant allergens. The erythrocyte sedimentation rate was 13 mm/h, blood hemoglobin 152 g/l, hematocrit 0.49, blood erythrocyte count $5.44 \times 10^{12}/l$, and leukocyte count $6.1 \times 10^9/l$. There was neither blood, sputum, nor nasal discharge

Fig 1. The trimming and sorting level of planks (sampling site A). From this level the trimmed and sorted planks fall down to storage ("pockets").

Fig 2. The storage ("pockets") of the trimmed and sorted planks (sampling site B).

eosinophilia. No antinuclear antibodies, LE-cells, or rheumatoid factor was found, and serum immunoelectrophoresis was normal. Serum precipitin tests showed positive reactions to *Thermoactinomyces vulgaris* and *Aspergillus fumigatus*, but not to *Micropolyspora faeni*. Spirometric findings were normal, as well as the pulmonary diffusing capacity measured by the single-breath method. Arterial blood oxygen tension at rest (10.5 kPa) was slightly decreased in comparison to the age-adjusted normal value (14) of 12.0 kPa. After two month's sick leave the patient returned to his normal work and had no further respiratory trouble. The arterial blood oxygen tension, however, remained impaired at later checkups (9.1 kPa in September 1978, 8.9 kPa in November 1978), and therefore an exercise test was performed on a bicycle ergometer in September 1978. During the exercise the arterial blood oxygen tension fell to 8.4 kPa. To exclude right to left shunt, a catheterization of the right side of the heart with blood pressure measurements was performed. The investigation showed no shunt. A transbronchial lung biopsy in November 1978 did not yield an adequate specimen. The arterial blood oxygen tension did not return to normal until the beginning of 1979. It was considered that the patient had suffered from bouts of extrinsic allergic alveolitis.

Aerobiological investigations

On closer inquiry, the patient reported that he had handled moldy wood at work, especially during the wintertime. The worst symptoms had appeared shortly after he had trimmed and sorted moldy fir planks. To explore the nature of the mold exposure, aerobiological measurements in the work environment were considered necessary.

The investigation took place at the end of June 1978. Two methods were used, the Petri dish method (11) and spore sampling with an Andersen sampler (6). Sampling was performed in the following two sites: on the trimming/sorting level of the planks (sampling site A) (fig 1) and close to the storage ("pockets") of trimmed and sorted planks (sampling site B) (fig 2). After exposure the culture media were incubated

at room temperature and at +40° C. When a good growth was noticed, the colonies were analyzed microbiologically, and both the relative and absolute amounts of spores in the air of the work environment were estimated. The results are shown in tables 1 and 2.

Discussion

The diagnosis of extrinsic allergic alveolitis (EAA) in our case was based on the history of exposure to moldy wood, on the symptoms and clinical signs, and on physiological and radiographic findings. Although the physiological investigations were not performed during any of the acute phases of the patient's disease and the patient was symptomless on admission to the hospital, the measurements of the arterial blood oxygen tension at rest and during exercise imply that in the hospital he still had an impairment of alveolar oxygen diffusion, which is one of the hallmarks of EAA.

The positive precipitin reactions (5) and the results of the aerobiological investigation of the work environment gave final support to the diagnosis. It is very probable that either *T. vulgaris* (7) or species of the genus *Aspergillus* (10) were the causative agents of this alveolitis. A summary of the other reported cases of EAA associated with the woodprocessing industry is given in table 3. In Sweden, a case report (1) has been published of sawmill workers suffering from symptoms compatible with EAA, even though their chest radiographs were normal and adequate physiological investigations were not performed, so that the diagnosis of EAA in these cases could not be considered definite.

Conifer wood, especially, tends to get moldy after felling. In Finnish sawmills in the summertime the wood is treated after sawing, usually with chlorinated phenols, to prevent the growth of molds causing the wood to turn blue. This treatment has been considered unnecessary during the wintertime. This work procedure agrees well with the findings of our patient, who became sick during the winter, and with the fact that rather low spore counts were found in the work

Table 1. Results of the mold analysis of the ambient air in the work environment — Petri dish technique, exposure time 10 min. Number of colonies/dish and portions in percentages calculated from the total number of colonies. Sampling site A: the trimming/sorting level of the planks, sampling site B: storage ("pockets") of the planks.

Fungal genus or group	Sampling site A		Sampling site B	
	N	%	N	%
Incubation at + 20°C				
<i>Aspergillus</i>	33	20	105	80
<i>Aureobasidium</i>	33	20	—	—
<i>Paecilomyces</i>	25	15	13	10
<i>Penicillium</i>	17	10	—	—
Other fungi	8	5	13	10
Yeasts	49	30	—	—
Total	165	100	131	100
Incubation at + 40°				
<i>Aspergillus</i>	94	80	83	90
<i>Mucor</i>	6	5	5	5
<i>Paecilomyces</i>	18	15	5	5
Total	118	100	93	100

Table 2. Spore content of the ambient air in the work environment — Andersen sampler counts, exposure time 3 min. The number of spores/m³ of air and portions in percentages calculated from the total number of spores. The same sampling sites as in table 1.

Genus or group	Sampling site A		Sampling site B	
	N	%	N	%
Fungi				
<i>Aspergillus</i>	1,150	50	1,800	60
<i>Aureobasidium</i>	230	10	150	5
<i>Mucor</i>	115	5	150	5
<i>Paecilomyces</i>	460	20	600	20
<i>Penicillium</i>	115	5	—	—
Other fungi	115	5	150	5
Yeasts	115	5	150	5
Total	2,300	100	3,000	100
Actinomycetes				
<i>Thermoactinomyces</i>				
<i>vulgaris</i>	560	70	1,200	80
Others	240	30	300	20
Total	800	100	1,500	100

environment in June. This minimal exposure, however, might explain the slight deterioration of the patient's arterial blood oxygen tension values found in checkups in September and November. Later, in another sawmill, we measured spore con-

centrations of several thousands per cubic meter of air (unpublished finding).

It seems that there are new health hazards in the modern sawmill industry. Special attention should be drawn to reducing the mold exposure of the work

Table 3. Extrinsic allergic alveolitis found among workers in the wood processing industry.

Alveolitis	Causative agents (confirmed or suspected)	Reference
Maple-bark disease	<i>Cryptostroma corticale</i>	Emanuel et al (4)
Sequoiosis	<i>Graphium</i> <i>Aureobasidium</i>	Cohen et al (3)
Wood-pulp workers' disease	<i>Alternaria</i>	Schlueter et al (13)
"Sawmill worker's lung"	<i>Thermoactinomyces</i> <i>vulgaris</i> <i>Aspergillus species</i>	Present report

environments to a minimum. Sawmill workers should always use effective dust respirators when handling moldy materials. Irreversible pulmonary damage may be the consequence from attacks of EAA (2). Therefore, the officers in occupational health services should be especially alert to the development of recurrent respiratory trouble among sawmill workers.

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