Inorganic particulates in pneumoconiotic lungs of hard metal grinders

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ABSTRACT Data from the analysis of lung dust in 16 metal grinders who had been exposed to hard metals between five and 44 years is reported. The mean latent time between the first exposure and analysis in biopsy or necropsy specimens was 33.6 years. Mineralogical and elementary analysis by a variety of techniques showed small or trace amounts of hard metal in all lungs. Many specimens, however, did not contain all hard metal components, cobalt, for example, being detected in four cases only. All the lungs contained quartz and silicates and in most of the necropsy cases carborundum and corundum could also be shown. Histologically no specific pattern was found. The appearances included mixed dust nodular pneumoconiosis, diffuse interstitial lung fibrosis, and foreign body and sarcoid like granulomatous changes. In view of the mixed dust exposure of the hard metal grinders and the variable histological appearance we think that the term "mixed dust pneumoconiosis in hard metal grinders" is more appropriate than "hard metal lung" to describe this condition.

The mineralogical and chemical analysis of lung tissue has proved successful in detecting aetiological particulates in the pneumoconioses. This is particularly true for the uncommon pneumoconioses, to which group belong the rare cases of Cerpneumoconiosis^{1,2} and the so called hard metal disease that has attracted considerable attention since it was first described by Jobs and Ballhausen in Germany in 1940.³

Hard metals are metallurgical materials characterised by their extreme physical hardness and resistance to corrosion and heat, and they have found wide application in industries such as toolmaking, instrument production, and the manufacture of drills. The process of manufacturing hard metals includes the sintering of powders of tungsten carbide (WC), titanium carbide (TiC), niobium carbide (NbC), tantalum carbide (TaC), molybdenum carbide (MoC), vanadium carbide (VC), and chromium carbide (CrC), with cobalt (Co) and nickel (Ni) as binding materials. The composition varies according to the technical requirements. Tungsten carbide (WC) is the main component and is regularly present in high amounts, around 80-90%.⁴ More than 75% of hard metals contain between 6% and 9% cobalt.⁵ Dust is produced both in the manufacture of hard metals and

during the grinding and polishing of hard metal products. In Switzerland, with a highly developed toolmaking industry, instrument grinders are especially exposed. Grinders usually use corundum (Al₂O₃), carborundum (SiC), and diamond buffing disks. Dust measurement studies in the patients' working environment report concentrations of particles below 5 microns of between 0.3 mg/m³⁵ and 15 mg/m³ and more.⁶ The particle size in the working area varies between 1 and 20 microns.⁷

Clinically, two types of hard metal induced lung lesions are described: (1) diffuse interstitial lung fibrosis with a restrictive impairment of lung function and (2) hard metal asthma with an obstructive disturbance.^{4,8,9} Pathologically, an interstitial lung fibrosis has been reported in most of the published cases.¹⁰⁻²⁰

In the present paper we report the results of a mineralogical analysis of lung dust in 16 metal grinders and polishers exposed to hard metal dust.

Occupational history and dust exposure

Of the 16 patients, 15 had a clear history of exposure to hard metal dust. In one case (No 547), a 74 year old man, no occupational data were available but lung dust analysis was performed because of clinical idiopathic diffuse lung fibrosis and the presence of hard

Table 1 *Hard metal grinders: exposure time and age distribution*

Mean exposure time (t_{exp}):
21.2 $\pm$ 13.3 years (n = 13) 5–55 years
Mean latent period (t_{lat}):
33.6 $\pm$ 15.9 years (n = 12) 7–54 years
Mean age (years):
All cases: 58.7 $\pm$ 14.8 (n = 16) 31–76
Biopsies: 46.1 $\pm$ 12.5 (n = 7) 31–65
Necropsies: 68.4 $\pm$ 7 (n = 9) 55–76

metals was shown. The 15 subjects had been metal grinders, polishers, and winders in plants where hard metal tools and instruments were produced. Two of the patients had worked as grinders in foundries (Nos 253, 792), another (No 758) had worked in a quartz mill before working as a grinder. None had been engaged in the manufacture of hard metals. Unfortunately the data about dust concentration at their places of work are incomplete. In many cases the exposure was in the distant past and apparently low. Single measurements resulted in values around 1.3–2.8 mg/m³ but in one extreme case it was up to 15.6 mg/m³ (Swiss National Accident Insurance Fund (SNAIF), personal communication). The patients had frequently been exposed to mixed dust containing particles other than hard metals. These values, however, may not be representative. Reliable values for the concentration of cobalt in the working atmosphere are also not available. In one case, however, it was estimated to be 0.1 mg/m³ (SNAIF).

Table 1 documents the average exposure time (t_{exp}) and the latent period (t_{lat}) between the first contact with hard metal dust and the time of analysis—that is, the time of biopsy or necropsy. The shortest exposure time was five years and the longest 44 years. The mean

latent time (12 cases) was 33.6 years.

The ages at diagnosis ranged between 31 and 62 in the biopsy cases and between 55 and 76 in the necropsy cases (table 1).

Histological findings

The histological findings are given in table 2. In three cases (Nos 253, 296, 758) there was a mixed dust pneumoconiosis of the type seen in foundry workers with starlike pinhead sized nodules that showed fibrosis accompanied by centrilobular emphysema.²¹

In 10 cases there was a diffuse interstitial lung fibrosis (DILF) with honeycombing. In two of these (Nos 511 and 880) the DILF was associated with granulomatous lesions having an appearance like sarcoidosis. Three biopsy cases showed granulomatous lesions only. In two (Nos 451, 454) there was a foreign body reaction with giant cells containing dust particles. The third case showed sarcoid like granulomas.

Analytical methods and results

We used seven lung biopsy specimens and nine whole lungs from necropsies for lung dust analysis. For isolation of the minerals the lungs and the biopsy specimens were degraded either by plasma incineration or by formamide. The procedures used are described in full elsewhere.^{22 23}

To identify mineralogical particulates in biopsy specimens we used transmission electron microscopy and energy dispersive x ray analysis (EDXA) on thin sections from histological preparations.

In necropsy cases representative probes of isolated lung dust were used for x ray diffraction and infrared

Table 2 *Exposure time and pathological diagnoses*

Case no	Age (years)	t_{exp} Years	t_{lat} Years	Diagnosis
Biopsies				
451	40	18	21	GRAN (F)
454	31	15	—	GRAN (F)
584	43	16	22	GRAN (S)
627	39	13	16	DILF
669	43	7	7	DILF
694	62	—	—	DILF
792	62	—	—	DILF
Necropsies				
253	72	5	51	MIPN + + +
296	65	22	23	MIPN +
511	55	36	41	DILF + + GRAN (S)
547	74	—	—	DILF + + +
578	71	32	38	DILF + + +
672	66	43	47	DILF + + +
724	75	44	54	DILF + + +
758	76	9	53	MIPN + +
880	62	16	30	DILF + GRAN (S)

MIPN = Mixed dust pneumoconiosis; DILF = diffuse interstitial lung fibrosis; GRAN = granulomatous changes; severity: + = mild, + + = moderate, + + + = severe; t_{exp} = time of dust exposure; t_{lat} = time between first exposure to dust and biopsy or necropsy; (F) = foreign body reaction; (S) = sarcoid like.

Table 3 Elemental analysis (AAS, EDXA)

Case No	Hard metal components	Binding metals	Other elements
<i>Biopsy specimens:</i>			
451	W	Fe, Ni	Cu, Al, P, Si, S, Mg, Ca, K
454	Ti, W	Fe	Al, Mn, Cu, Zn, Si, P
584	Ti, Cr	Co, Fe, Ni	Al, Ce, P, Si
627	Nb, Ti, W, Cr	Fe, Ni	Mo, Al, Si, Ca, K, P, Sn, Cl
669	Ti, W, Cr	Fe, Ni	Al, Ca, Si
694	Ti, W, Zr, Cr	Fe, Ni	Si, Al, Ba, Ca
792	Ti, W, Cr	Fe, Ni	Al, P, Si, Zn, Sn, S

spectroscopy and also for electron microscopy and EDXA. In some instances atomic absorption spectroscopy (AAS) was applied. The results are summarised in tables 3 (biopsy specimens, elemental analysis) and 4 (necropsies, elemental and mineralogical analysis).

It has to be emphasised that it is not always possible to correlate the results of the EDXA analysis with the results of the mineralogical analysis. The EDXA method allows for the detection of elements with an atomic number greater than 9 (F) only. The sensitivity of EDXA is much higher than the x ray diffraction and infrared-spectroscopy methods. For example, small quantities of Cr and Co were detected with EDXA in case No 547 whereas no corresponding compounds of these elements were observed with x ray diffraction and infrared-spectroscopy. The EDXA analysis of biopsy specimens allows only the detection of elements but no compounds. In combination with other methods and the occupational history it is a valuable indicator for a given dust exposure.

Discussion

In all the lungs analysed hard metal components were found. Not all the isolated dust samples, however, contained all the most frequently used hard metal components, thus W, Mo, Ti, Cr, and Co were not detected in each and every lung. Apart from the hard metals, considerable amounts of quartz and silicates such as mica and feldspar, and other minerals were also present in all the lungs. In the lungs showing mixed dust pneumoconiosis the mineral content of the lung was high (with the exception of case 296) compared with the cases with diffuse interstitial lesions. Carborundum and corundum, the most frequently used grinding materials, were found in seven of the nine necropsy lungs of metal grinders that have been analysed in total. In the biopsy tissue, however, as mentioned before, carborundum and corundum could not be shown by EDXA analysis.

Histologically three types of lung lesions were found:

- (1) diffuse interstitial lung fibrosis (DILF),
- (2) sarcoid like granulomatous lesions and foreign body reactions (GRAN), and
- (3) typical mixed dust pneumoconiosis with starlike elementary nodules (mixed dust silicosis) (MIPN).

The histological pattern was not always uniform and different lesions often appeared in the same lung. No specific changes induced by hard metal were detected. This is perhaps what would be expected as it is not the hard metals alone but dust mixtures containing other fibrogenic mineral particles such as

Table 4 Elemental analysis (AAS, EDXA)

<i>Elemental analysis (AAS, EDXA)</i>			<i>Mineralogical analysis (x ray diffraction, infrared-spectroscopy)</i>	
Case No	Hard metal components	Binding metal	Min cont % of G dry weight lung	
<i>Necropsies:</i>				
253	Ti, V, Cr	Co, Fe, Ni	5.5	Haematite 8%, magnetite 20%, quartz 70%, mica 2%
296	Ti, Mo, Ta, W	—	1.3	Magnetite, carborundum, quartz
511	Ti, Mo, Cr	—	2.42	Haematite 5%, corundum 5%, carborundum 2%, ca-phosphate 80%, quartz 5%, feldspar 3%
547	Ti, Cr	Co, Fe	1.07	Haematite 1%, magnetite 1%, amorphous iron compounds 50%, rutile + anatase 2%, quartz 7%, kaolin 1%, mica 1%, hydroxylapatite 30%
578	Ti, W	Fe	1.42	Haematite 1%, magnetite 1%, amorphous iron compounds 70%, corundum 5%, carborundum 5%, rutile 1%, quartz 7%, mica 1%, kaolin 2%, feldspar 1%, ca-phosphate 1%
672	Ti, V, Nb, W	Fe	0.52	Haematite 2%, magnetite traces, amorphous iron compounds 75%, corundum 8%, carborundum 2%, WC 4%, TiC traces, Nb ₂ O ₅ traces, quartz 4%, mica 1%, kaolin 1%, feldspar 1%
724	Zr, Mo, Cr	Fe, Ni	0.52	Haematite 15%, magnetite 7%, amorphous iron compounds 10%, corundum 25%, carborundum traces, rutile traces, quartz 25%, mica 3%, talc 1%, kaolin 9%, feldspar 6%
758	Ti, W	Fe	3.01	Haematite traces, corundum 20%, carborundum 12%, WC 5%, TiC traces, rutile 20%, anatase 10%, quartz 30%, mica 2%, feldspar 2%, ca-fe-phosphate 4%
880	Cr, W	Co, Ni	0.43	Amorphous iron compounds 30%, corundum 20%, carborundum 5%, WC traces, quartz 21%, mica 2%, talc 10%, kaolin 5%, feldspar 2%

quartz and silicates which are inhaled. These other minerals probably play a contributory part in inducing the lung changes. From our experience we know that plate minerals and fibrous minerals (mica, for example) produce a diffuse interstitial fibrosis.²⁴ The aetiological and pathogenic role of the metal binding cobalt is unclear in this process. From clinical evidence it seems that cobalt may be responsible for the asthmatic form of hard metal disease. Cobalt increases the risk of asthma five times when an exposure at a concentration of 0.1 mg/m³ occurs.²⁵ Other authors suspect that cobalt is an important causal agent in hard metal disease.^{17 18 26-28} The search for cobalt by chemical and mineralogical analysis of lung tissue was not, however, rewarding and it was found in four cases only. This may be a consequence of the high solubility of this metal in water by which it evades detection.

Nevertheless, based on clinical observation cobalt may have a toxic allergic influence.^{4 17 25 29} The origin of the sarcoid like lung lesions may be a cobalt mediated immunological process. Analysis of giant cells with the electron microscope and EDXA in our material, however, showed no cobalt. Instead the particulates sometimes seen in the foreign body giant cells resembled quartz crystals and micacrystals. Measurements of particles containing tungsten in the electron microscope and in some of the deposits in the fibrosed interstitial lung tissue showed that most are less than 1 micron in diameter. These small particles, together with the fibrogenic plate minerals (silicates), are believed to be an essential factor in the production of diffuse interstitial lung fibrosis.

Taking the results of the analysis and the histopathological features together there appears to be no specific lung lesion in workmen exposed to hard metal. The appearances are rather those of a non-specific mixed dust pneumoconiosis. For this reason we prefer the term "pneumoconiosis in hard metal grinders" to "hard metal lung" which we do not believe is a distinct pathological entity.

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