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Effects of vanadium on the upper respiratory tract of workers in a vanadium factory

A macroscopic and microscopic study

by MARKKU KIVILUOTO, M.D.,¹ OLAVI RÄSÄNEN, M.D.,² ARI RINNE, M.D.,³ and MARTTI RISSANEN, M.D.⁴

KIVILUOTO, M., RÄSÄNEN, O., RINNE, A. and RISSANEN, M. Effects of vanadium on the upper respiratory tract of workers in a vanadium factory: A macroscopic and microscopic study. *Scand. j. work environ. & health* 5 (1979) 50—58. An epidemiologic cross-sectional case-history study on the injurious effects of vanadium was carried out among the workers of a vanadium factory. The upper respiratory tract of 63 male workers exposed to vanadium dust was examined macroscopically and microscopically, and the findings were compared with those of a reference group of workers who were exposed to inert dust only. The groups compared were of similar ages and had similar smoking habits. Nasal smears and sputum cells were studied microscopically, and biopsies for histological study were taken from the nasal mucosa. The biopsies from the vanadium workers showed a significant increase in the number of plasma and round cells, and the histological picture was almost characteristic. There were no increased numbers of secretion eosinophils or other signs indicative of allergic inflammation.

Key words: cytology, histopathology, nasal mucosa, vanadium dust, vanadium exposure.

Vanadium is a silvery-white metal, which is usually separated from carnotite and titanomagnetite ores and is commonly used as an alloy to improve the properties of steel. Some is also used in the chemical industry.

The world production of vanadium was about 35,000 t in 1975. The largest pro-

ducers were South Africa, the United States, the Soviet Union, Finland, Namibia, Norway, and Chile.

Exposure to the dust of vanadium pentoxide can occur during the different stages of its manufacture, in the steel industry, and in certain branches of the chemical industry. It can also occur while boilers operated by oil are being cleaned or repaired, as many oils contain vanadium, which originates from ancient sea organisms.

Vanadium is mostly transmitted to the human organism through the lungs and is mainly excreted in the urine. On the whole, the gastrointestinal tract absorbs none at all.

It was Dutton who first described the symptoms of vanadium poisoning (6).

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Later Symanski (23), Wyers (28, 29), Sjöberg (19, 20, 21), Pielsticker (17), Gulko (8), Mantantseva (13), Vintinner et al. (25), Frost (7), and Williams (26) described the symptoms caused by vanadium and noted that vanadium dust causes irritation in the upper respiratory tract, e.g., nasal catarrh, even hemorrhage from nasal mucosa, stinging pain in the throat, irritation causing coughing as well as whooping breathing. Several scientists have described chronic rhinitis, bronchitis and even conjunctivitis, probably due to a prolonged exposure to vanadium dust. Exposure, even if prolonged, has not been proved to cause such permanent damage to the lungs as emphysema, bronchiectasis, or pneumoconiosis, nor has vanadium been shown to be carcinogenic (19). There is no indication that vanadium affects the central or peripheral nervous system detrimentally (19).

The action of the kidneys, liver, and bone marrow has been found to remain normal during exposure to vanadium. Vanadium causes local irritating symptoms in the gastrointestinal tract, but no chronic systemic intoxication has been observed (19). The aforementioned studies have concerned man in vivo.

Biochemically vanadium may have various effects on metabolism, obviously because of its oxidizing ability. These effects are primarily associated with enzyme systems (1, 2, 3, 4, 5, 9, 12, 14, 16).

It is not possible to detect vanadium in the blood of test animals 8 h after exposure. More than half of the vanadium is excreted in the urine in 24 h (19). Changes indicating chronic inflammation, emphysema, and broncho-pneumonic-atelectatic changes appeared in the mucous membranes of rabbits after prolonged exposure. Microscopic examination revealed a fairly large leucocyte and plasma cell infiltration in the nasal mucosa, which contained an abundance of blood vessels subepithelially. Slightly hyperemic and chronic inflammatory changes were found in the trachea. Bronchial emphysema and broncho-pneumonic-atelectatic changes were found in all of the 12 test animals (19). These studies were made on test animals in vivo and in vitro.

Symptoms of irritation in the respiratory

tract were fairly common as subjective symptoms among the workers of the vanadium factory of the present study in a periodic examination in 1973. So far no studies on simultaneous macroscopic, cytological, and histological changes have been made on humans exposed to vanadium. The purpose of the present study was to explain these changes.

METHODS AND SUBJECTS

Determinations of the concentration of vanadium dust in the factory air have been carried out since 1970. According to these measurements, 0.2–0.5 mg of vanadium per cubic meter can be regarded as the average level of previous exposure (determined from the total dust gathered on a paper filter with the atomic absorption spectrophotometric method, a total of 64 samples between 1970 and 1975).

At the beginning of 1976 the management intensified the flushing of the floors in order to stop the dust from rising. Thereafter the exposure level was about 0.01–0.04 mg/m³ (194 determinations of dust concentration in the spring of 1976, 114 samples of which were taken in the breathing zones of individual workers and the rest at stationary sites).⁵ The dust was collected by suction on a paper filter. The concentration of vanadium was determined by an atomic absorption spectrophotometer with the graphite furnace method. The total dust concentrations were very small in the factory. In addition to vanadium, the air contained inert magnetite dust. In ordinary workplaces near sintering furnaces, the concentration of sulfur dioxide was also determined with indicator tubes, the determining limit of

⁵ The American Conference of Governmental Industrial Hygienists, as of 1972, recommends a threshold limit value of 0.5 mg/m³ for vanadium dust and a maximal allowable concentration of 0.05 mg/m³ for vanadium fumes. The accuracy of the determinations made with the atomic absorption spectrophotometric method was about 0.002 mg and the standard deviation on the level of 0.05 mg was about 0.003 mg.

which, $0.5 \text{ cm}^3/\text{m}^3$, was not exceeded. The samples were taken by methods commonly used by the Institute of Occupational Health in Finland (22).

Since the subjects had worked an average of 11 years at the factory, attention had to be paid to the previous levels of dust before a reliable picture of the possible chronic effects of vanadium could be formed.

An epidemiologic cross-sectional study was chosen as the method of investigation. The subjects were comprised of process workers, day workers, repairmen, foremen, and a laboratory worker, i.e., altogether 63 men. A reference group was composed of male workers living in the same area, and working in the Otanmäki Mine with possible exposure to inert dusts of the concentrating plant, the repair shop, the mine, and the office. The referents were matched with regard to age and cigarette consumption within two years and five cigarettes a day, respectively.

The results were statistically analyzed by means of matched pairs according to the McNemar chi-square test. The changes noticed within the vanadium group after exposure were also examined according to McNemar's chi-square test. The pathologic-anatomic changes were also analyzed by these means after the 10 persons with the shortest periods of employment at the factory were eliminated from the group.

An otologist examined the upper respiratory tract of the workers in both groups before they returned to work after their 2- to 4-week summer vacation in June-August 1975; he also examined the exposed group again in March-June 1976 after several months of exposure. (The measurements of dust concentration were also made at this time.) At the time of the former examination the cytological smears of the nasal mucosa and mucosal biopsies were taken from the middle part of the nasal inferior turbinates for microscopic examination. There was no evidence of epidemic upper respiratory infection in the examined subjects at the time of the study. The subjects took the sputum cell specimens at home on the morning of the examination. Measurements of pulmonary ventilation and some laboratory tests were also made at the same time. Sputum

samples were coughed into a jar containing 50 % ethanol. After one to two days' fixing, smears were made on clean slides.

Nasal secretion smears were made and studied according to the methods of Holopainen (10). The smears were taken by a very light hand with a cotton swab from the surfaces of the nasal mucosa and spread onto a clean slide with great care. The samples were fixed immediately with cytofixative (Pro-Fixx®).

Two pathologists examined the samples independently of each other in a blind test and counted the cells per 10 fields of vision using the 480-fold magnification of the microscope as well as the symbols 0, +, ++, +++, and +++++. In the categories of 0 and + the number of cells was regarded as being within normal limits, and in those of ++, +++, and +++++ increased.

The histological biopsies were fixed in 10 % neutral formalin and embedded in paraffin blocks. They were cut into sections which were 5μ thick.

The following stainings were used: (a) Hematoxylin-eosin to demonstrate the overall morphology and the tissue plasma cells and eosinophils and (b) May-Grünwald-Giemsa to demonstrate tissue plasma cells and eosinophils.

The following stainings were made from the sputum samples: (a) Papanicolaou staining (15) according to Holopainen (10) to demonstrate ordinary findings and (b) May-Grünwald-Giemsa staining especially to demonstrate eosinophils.

The following stainings were made from the nasal secretion smears: (a) Papanicolaou staining as for the sputum samples, (b) May-Grünwald-Giemsa staining as for the sputum samples, and (c) Wright's staining (27) especially to demonstrate mast cells but also neutrophils and eosinophils.

Two pathologists examined the cells by the earlier mentioned means. The metaplastic cells and eosinophilis were examined from the sputum.

In smears taken from the nasal inferior turbinates, the columnar epithelial cells, goblet cells, metaplastic cells, basal cells, squamous epithelial cells, cilia cytophorias cells, neutrophils, eosinophils, mast cells, lymphocytes, plasma cells, and bacteria were examined.

Columnar epithelial cells with or without cilia, possible degeneration, goblet cells, metaplastic cells, inflammatory cells (the plasma cells included), eosinophils, and mast cells were examined in the biopsies taken from the nasal inferior turbinates. In addition, we examined the thickness of the connective tissue membrane, changes in the mucous glands and in the vessels, and the stromal fibrosis and also made a histopathological diagnosis considering a possible acute, subchronic or chronic inflammation.

RESULTS

Macroscopic findings in the respiratory tract

The findings in the anterior rhinoscopy did not reveal any significant differences between the groups according to McNemar's test.

No significant differences were revealed in the posterior rhinoscopy in the inspection of palatal mucosa, the rear wall of the pharynx, larynx and vocal cords, or in the frequency of common colds at the time of the examination.

Cytology

According to McNemar's chi-square test, the number of neutrophils had increased significantly ($p < 0.001$) in the nasal smears of the exposed group (table 1).

After the exposed group had been reduced by the 10 workers with the shortest periods of exposure, there were still significantly more of the neutrophils ($p < 0.001$) in the exposed group (table 2).

Histopathological findings

A statistically significant change ($p < 0.05$) was found in the amount of plasma cells, which appeared more frequently in the exposed group (table 3).

There were also more plasma cells in the biopsies of the nasal mucosa from the exposed group once the 10 workers with the shortest periods of employment were eliminated, but the difference was not significant ($p > 0.05$) (table 4).

Table 1. Number of persons according to the amount of neutrophils in the nasal smears of the exposed and reference groups (55 pairs).^a

Exposed group	Reference group	
	Normal	Increased
Normal	35	1
Increased	16	3

^a Significantly ($p < 0.001$) more neutrophils in the nasal smears from the exposed group.
 $\chi^2 = 11.53$ (DF = 1)

Table 2. Number of persons according to the amount of neutrophils in the nasal smears of the exposed group without the 10 workers with the shortest periods of employment at the vanadium factory and their matched referents (45 pairs).^a

Exposed group	Reference group	
	Normal	Increased
Normal	30	0
Increased	12	3

^a Significantly ($p < 0.001$) more neutrophils in the nasal smears from the exposed group.
 $\chi^2 = 10.08$ (DF = 1)

Table 3. Number of persons according to the amount of plasma cells in the biopsies of nasal mucosa from the workers in the exposed and reference groups (57 pairs).^a

Exposed group	Reference group	
	Normal	Increased
Normal	38	4
Increased	15	0

^a Significantly ($p < 0.05$) more plasma cells in the biopsies of nasal mucosa from the exposed group.
 $\chi^2 = 5.26$ (DF = 1)

There was a statistically significant increase ($p < 0.05$) in the round cells of the exposed group (table 5).

Table 4. Number of persons according to the amount of plasma cells in the biopsies of nasal mucosa of the exposed group without the 10 workers with the shortest periods of employment at the vanadium factory and their matched referents (47 pairs).^a

Exposed group	Reference group	
	Normal	Increased
Normal	32	4
Increased	11	0

^a More, but not significantly more ($p > 0.05$), plasma cells in the biopsies of nasal mucosa from the exposed group.
 $\chi^2 = 2.40$ (DF = 1)

Table 5. Number of persons according to the amount of round cells in the biopsies of nasal mucosa from the exposed and reference groups (56 pairs).^a

Exposed group	Reference group	
	Normal	Increased
Normal	19	10
Increased	21	6

^a Significantly ($p < 0.05$) more round cells in the biopsies of nasal mucosa from the exposed group.
 $\chi^2 = 3.23$ (DF = 1)

Table 6. Number of persons according to the amount of round cells in the biopsies of nasal mucosa of the exposed group without the 10 workers with the shortest periods of employment at the vanadium factory and their matched referents (46 pairs).^a

Exposed group	Reference group	
	Normal	Increased
Normal	16	7
Increased	19	4

^a Significantly ($p < 0.05$) more round cells in the biopsies of nasal mucosa from the exposed group.
 $\chi^2 = 4.65$ (DF = 1)

More numerous round cells were found statistically significantly ($p < 0.05$) in the exposed group without the 10 least exposed men (table 6).

Table 3 indicates an increase in the plasma cells. This increase was statistically significant ($p < 0.05$) for the mucous membrane biopsies from the exposed persons. Table 5 reveals that, in the biopsies, round cells were more numerous among the exposed workers than among the referents, and the difference was almost statistically significant. In addition the histological picture was almost characteristic in the biopsies of the exposed persons. It was typical that the round cell reaction could be seen in the lamina propria as a zonelike infiltration directly under the surface of the epithelium (fig. 1). Deeper in the lamina propria, only a small increase in the inflammatory cells was found, or there was hardly any sign of them at all. The round cell infiltration contained abundant plasma cells (fig. 2), which also showed a statistical difference when compared with the biopsies from the referents. Also, in several samples, fairly typical features of the biopsies of the exposed persons were papillarity on the surface of the mucous membranes (fig. 3) and dilated and hyperemic capillaries in papillae (fig. 4), although no evident statistical differences appeared between the number of blood vessels in the exposed workers and the referents. In the nasal smears the amount of neutrophils had increased significantly in the exposed group (table 1). The rest of the examined factors of the biopsies and cell findings did not significantly differ between the exposed workers and the referents. The changes were parallel when the 10 persons with the shortest periods of employment at the vanadium factory were eliminated from the material; among the rest of the subjects the exposed group had almost significantly more plasma cells (table 4) and significantly more round cells (table 6).

When the cell changes were examined, no significant difference could be found during the period of exposure according to McNemar's chi-square test. Thirty-one workers of the exposed group underwent a reexamination after 7 to 11 months of exposure at the factory. Cell findings in

Fig. 1. The nasal mucosal biopsy taken from a 44-year-old furnace man after 19 years of exposure to vanadium. The surface columnar epithelium is slightly irritated and hyperplastic. A fairly rich zonellike subepithelial round cell infiltration can be seen (bracket). There are only a few inflammatory cells deeper in the stroma of the mucosa. Hematoxylin-eosin staining. Original magnification $\times 120$.

Fig. 2. A higher magnification from the biopsy above indicating an abundance of plasma cells among the round cells. Original magnification $\times 480$.

Fig. 3. A histological finding common to the persons exposed to vanadium was the papillarity of the nasal mucosa associated with subepithelial zones of round cell infiltration in lamina propria as in fig. 1. This biopsy was taken from a 36-year-old filter man after 13 years of exposure. Hematoxylin-eosin staining. Original magnification $\times 100$.

Fig. 4. A solitary mucosal papilla from the above biopsy. A strong hyperemia can be seen in the capillaries. The surface columnar epithelium is degenerated to some extent on the head of the papilla. Original magnification $\times 480$.

different age groups (19—29, 30—39 and 40—56 years; 18, 14 and 31 persons, respectively) indicated no further significant changes between the studied groups. In the exposed group there was no correlation between the cell findings and the duration of exposure to vanadium (4—109, 110—169 and 170—241 months of exposure; 23, 20 and 20 persons, respectively).

DISCUSSION

No previous study of the detrimental effects of vanadium has used such a large reference material. In the literature, no case history study can be found with a simultaneous examination of macroscopic, cytological and histological changes of the respiratory tract of man after exposure to vanadium.

It is probable that the statistically significant increase in the neutrophils of the smears is due to an irritating effect of vanadium dust on the mucous membranes of the upper respiratory tract. The statistically significant increase in the number of plasma cells and the round cells of the mucous membrane biopsies taken from the inferior nasal turbinates would seem to support this view too.

The reliability of the results is confirmed by the fact that the smoking habits and the age distribution of the two groups studied are similar (matched pairs) and by the fact that the cytological and histological samples were studied in a blind test. When the significance of the inflammatory changes is estimated, the earlier level of exposure ($0.2\text{--}0.5\text{ mg/m}^3$ on an average, determined from the total dust content in a cubic meter of air at the factory) and the average work period (11 years) must be taken into account.

Because there were no statistically significant changes in the number of the eosinophils of the cytological and histological samples from the exposed group during 7—11 months of exposure, we are

inclined to believe that the changes in the number of cells caused by vanadium are not of an allergic origin. No changes suggesting a carcinogenic effect of vanadium could be found. In our opinion it would be reasonable to carry out a follow-up study later, since carcinogenicity is known to appear after 20—35 years of exposure.

In spite of the fact that the vanadium concentrations of air inhaled were minute, there was an almost typical finding in the nasal mucosal biopsies of the workers with the longest periods of exposure to vanadium, i.e., a zonelike subepithelial infiltration of mononuclear cells and frequent papillarity in the mucous membrane surface with its hyperemic capillaries. Similar subepithelial inflammatory cell localization and hyperemia were described by Sjöberg (19) in the nasal mucosa of rabbits exposed to high vanadium concentrations.

It would seem that the subepithelial lymphocyte and plasma cell infiltration functions as a protective mechanism in the irritated mucosa. We have planned additional studies to determine the main classes of immunoglobulins contained in these round cell infiltrations and whether there are immunoglobulins in the mucosa or not. We will use a peroxidase-antiperoxidase method (PAP) as modified in the Department of Pathology in the University of Oulu.

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