



Pneumonoultramicroscopicsilicovolcanoconiosis: The largest English word of 45 letters causing largest health hazards

Dhrubo Jyoti Sen, Kinsuk Kalyan Sarker, Clive Dadida and Paul Dhliwayo

Department of Pharmaceutical Chemistry, Shri Sarvajanic Pharmacy College, Gujarat Technological University, Arvind Baug, Mehsana-384001, Gujarat, India

Received: 09-10-2014 / Revised: 17-11-2014 / Accepted: 21-11-2014

ABSTRACT

PNEUMONULTRAMICROSCOPICSILICOVOLCANOCONIOSIS is a largest English word of 45 letters cause silicosis which is a lung disease caused by the inhalation of very fine silica dust, causing inflammation in the lungs!! Silicosis is a chronic lung disease caused by breathing in tiny bits of silica dust. Silica is the second most common mineral in the earth's crust. It is a major component of sand, rock and mineral ores like quartz. People who work in jobs where they can be breathing in these tiny silica bits—like sandblasting, mining, construction and many others—are at risk for silicosis. When people breathe silica dust, they inhale tiny particles of silica that has crystallized. This silica dust can cause fluid buildup and scar tissue in the lungs that cuts down your ability to breathe. There are three types of silicosis: Chronic silicosis, the most common type of silicosis, usually occurs after 10 or more years of exposure to crystalline silica at low levels, Accelerated silicosis occurs 5-10 years after exposure and is caused by exposure to higher levels of crystalline silica. Acute silicosis can occur after only weeks or months of exposure to very high levels of crystalline silica. Acute silicosis progresses rapidly and can be fatal within months. It occurs due to: Highway and bridge construction and repair, Building construction, Demolition and repair, Abrasive blasting, Masonry work, Concrete finishing, Drywall finishing, Rock drilling, Mining, Sand and gravel screening and Rock crushing (for road base). Silicosis is chronic and cannot be cured. Treatments ease symptoms and address infections that people with silicosis are prone to getting. Depending on the type of silicosis, people may live for many years or only a few months. Generally, the silica dust affects the lungs ability to work correctly. Each type of silicosis affects the body somewhat differently: In simple chronic silicosis, the silica dust causes areas of swelling in the lungs and chest lymph nodes, which causes breathing difficulty. In accelerated silicosis, swelling in the lungs and symptoms occur faster than in simple silicosis. In acute silicosis, the lungs become very inflamed and can fill with fluid, which causes severe shortness of breath and low blood oxygen levels. Anyone with silicosis may suffer from several complications: Increased risk for lung infections and tuberculosis. Progressive massive fibrosis—severe scarring and stiffening of the lung, which makes it difficult to breathe. Progressive massive fibrosis can occur in either simple or accelerated silicosis, but is more common in the accelerated form with respiratory failure.

Keywords: Pneumonoultramicroscopicsilicovolcanoconiosis, volcano, silica, silicosis, chronic simple silicosis, accelerated silicosis, complicated silicosis, acute silicosis, coniosis, scleroderma, SLE, PMF, leukotriene B₄, cytokines.



The largest English word which makes a great wonder:

PNEUMONULTRAMICROSCOPICSILICOVOLCANOCONIOSIS

It's phonetic analysis is **NOO-muh-noh-UL-truh-MY-kruh-SKOP-ik-SIL-i-koh-vol-KAY-no-KOH-nee-O-sis!** It means *A lung disease caused by the inhalation of very fine silica dust, causing*

inflammation in the lungs!!

PNEUMONO+ULTRAMICROSCOPIC+SILICO+VOLCANO+CONIOSIS

is **PNEUMONO** refers to pneumatic system which is filled with air (lungs), **ULTRAMICROSCOPIC** refers to ultra fine matter, **SILICO** refers to silica, **VOLCANO** refers to volcano, **CONIOSIS** means the disease caused by inhalation of dust.¹ *It is an English word that refers to a lung disease that is otherwise known as silicosis. It is the longest word*

*Corresponding Author Address: Prof. Dr. Dhrubo Jyoti Sen, Department of Pharmaceutical Chemistry, Shri Sarvajanic Pharmacy College, Gujarat Technological University, Arvind Baug, Mehsana-384001, Gujarat, India; E-mail: dhrubosen69@yahoo.com

in the English language published in a dictionary, the Oxford English Dictionary. According to the Oxford English Dictionary, it is "an artificial long word said to mean a lung disease caused by inhaling very fine ash and sand dust."

This word was invented in 1935 by **Everett M. Smith**, president of the National Puzzlers' League (N.P.L.), at its annual meeting. The word figured in the headline for an article published by the New York Herald Tribune on February 23, 1935, titled

"Puzzlers Open 103rd Session Here by Recognizing 45-Letter Word".

Pneumonoultramicroscopicsilicovolcanoconiosis succeeded electrophotomicrographically as the longest word in the English language recognized by the National Puzzlers' League at the opening session of the organization's 103rd semi-annual meeting held yesterday at the Hotel New Yorker. The puzzlers explained that the forty-five-letter word is the name of a special form of pneumoconiosis caused by ultra-microscopic particles of silica volcanic dust.²



Volcano



Silica Dust



Lungs

Figure-1: Volcano with Silica Dust and Lungs^[1-3]

(1. Definition of pneumonoultramicroscopicsilicovolcanoconiosis in Oxford dictionary (British and World English). Oxford Dictionaries. Oxford University Press.

2. Cole Chris (1999). Wordplay, A Curious Dictionary of Language Oddities. Sterling Publishing Co., Inc. 106–107.

3. Pelucchi C, Pira E, Piolatto G, Coggiola M, Carta P, La Vecchia C (2006). Occupational silica exposure and lung cancer risk: a review of epidemiological studies 1996–2005. *Ann. Oncol.* 17(7): 1039–1050.)

This cause **Silicosis** which is a form of occupational lung disease caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in the form of nodular lesions in the upper lobes of the lungs. It is a type of pneumoconiosis. Silicosis (particularly the acute form) is characterized by shortness of breath, cough, fever, and cyanosis (bluish skin).³ It may often be misdiagnosed as pulmonary edema (fluid in the lungs), pneumonia, or tuberculosis. The name *silicosis* is classified as:
Chronic simple silicosis: Usually resulting from long-term exposure (10 years or more) to relatively low concentrations of silica dust and usually

appearing 10–30 years after first exposure. This is the most common type of silicosis. Patients with this type of silicosis, especially early on, may not have obvious signs or symptoms of disease, but abnormalities may be detected by x-ray. Chronic cough and exceptional dyspnea are common findings. Radiographically, chronic simple silicosis reveals a profusion of small (<10 mm in diameter) opacities, typically rounded and predominating in the upper lung zones.³

Accelerated silicosis: Silicosis that develops 5–10 years after first exposure to higher concentrations of silica dust. Symptoms and X-ray findings are

similar to chronic simple silicosis, but occur earlier and tend to progress more rapidly. Patients with accelerated silicosis are at greater risk for complicated disease, including progressive massive fibrosis (PMF).⁴

Complicated silicosis: Silicosis can become "complicated" by the development of severe scarring (progressive massive fibrosis, or also known as conglomerate silicosis), where the small

nodules gradually become confluent, reaching a size of 1 cm or greater. PMF is associated with more severe symptoms and respiratory impairment than simple disease. Silicosis can also be complicated by other lung disease, such as tuberculosis, non-tuberculous mycobacterial infection and fungal infection, certain autoimmune diseases and lung cancer. Complicated silicosis is more common with accelerated silicosis than with the chronic variety.⁵



Silica Crystals **Silica Powder** **Silica Fibers**
Figure-2: Silica^[4-6]

(4. Cassel SL, Eisenbarth SC, Iyer SS, et al. (2008). "The Nalp3 inflammasome is essential for the development of silicosis". Proc. Natl. Acad. Sci. U.S.A. 105(26): 9035.
5. Wagner GR (1997). Asbestosis and silicosis". Lancet 349(9061): 1311–1315.
6. Sahu AP, Saxena AK. (1994). Enhanced Translocation of Particles from Lungs by Jaggery. Environmental Health Perspectives (Environmental Health Perspectives, Vol. 102) 102(S5): 211–214.)

Acute silicosis: Silicosis that develops a few weeks to 5 years after exposure to high concentrations of respirable silica dust. This is also known as silicoproteinosis. Symptoms of acute silicosis include more rapid onset of severe disabling shortness of breath, cough, weakness and weight loss, often leading to death. The X-ray usually reveals a diffuse alveolar filling with air bronchograms, described as a ground-glass appearance and similar to pneumonia, pulmonary edema, alveolar hemorrhage and alveolar cell lung cancer.⁶

Signs and symptoms: Because chronic silicosis is slow to develop, signs and symptoms may not appear until years after exposure. Signs and symptoms include: Dyspnea (shortness of breath) exacerbated by exertion, Cough, often persistent and sometimes severe, Fatigue, Tachypnea (rapid breathing) which is often labored, Loss of appetite and weight loss, Chest pain & Fever. Gradual dark shallow rifts in nails eventually leading to cracks as protein fibers within nail beds are destroyed. In advanced cases, the following may also occur: Cyanosis (blue skin), *Cor pulmonale* (right ventricle heart disease). Respiratory insufficiency. Patients with silicosis are particularly susceptible to tuberculosis (TB) infection—known as silico-tuberculosis. The reason for the increased risk—3 fold increased incidence—is not well

understood. It is thought that silica damages pulmonary macrophages, inhibiting their ability to kill mycobacterium. Even workers with prolonged silica exposure, but without silicosis, are at a similarly increased risk for TB. Pulmonary complications of silicosis also include Chronic Bronchitis and airflow limitation (indistinguishable from that caused by smoking), non-tuberculous Mycobacterium infection, fungal lung infection, compensatory emphysema, and pneumo-thorax. There are some data revealing an association between silicosis and certain autoimmune diseases, including nephritis, Scleroderma and Systemic Lupus Erythematosus (SLE), especially in acute or accelerated silicosis.⁷

Pathophysiology: When small silica dust particles are inhaled, they can embed themselves deeply into the tiny alveolar sacs and ducts in the lungs, where oxygen and carbon dioxide gases are exchanged. There, the lungs cannot clear out the dust by mucous or coughing. When fine particles of silica dust are deposited in the lungs, macrophages that ingest the dust particles will set off an inflammation response by releasing tumor necrosis factors, interleukin-1, leukotriene B₄ and other cytokines. In turn, these stimulate fibroblasts to proliferate and produce collagen around the silica particle, thus resulting in fibrosis and the formation of the nodular lesions.

The inflammatory effects of crystalline silica are apparently mediated by the Nalp3 in inflammasome. Characteristic lung tissue pathology in nodular silicosis consists of fibrotic nodules with concentric "onion-skinned" arrangement of collagen fibers, central hyalinization and a cellular peripheral zone, with lightly bi-refringent particles seen

under polarized light. The silicotic nodule represents a specific tissue response to crystalline silica. In acute silicosis, microscopic pathology shows a periodic acid-Schiff positive alveolar exudates (alveolar lipoproteinosis) and a cellular infiltrate of the alveolar walls.⁸



Figure-3: Lungs affected by silica^[7,8]

(7. Steenland K, Goldsmith DF (1995). Silica exposure and autoimmune diseases. *Am. J. Ind. Med.* 28(5): 603–608.

8. Nouh MS (1989). Is the desert lung syndrome (nonoccupational dust pneumoconiosis) a variant of pulmonary alveolar microlithiasis? Report of 4 cases with review of the literature. *Respiration* 55(2): 122–126.)

Silica: Silicon (Si) is the second most common element in the Earth's crust (oxygen is the most common). The compound silica, also known as silicon dioxide (SiO_2), is formed from silicon and oxygen atoms. Since oxygen and silicon make up about 75% of the Earth, the compound silica is quite common. It is found in many rocks, such as marble, sandstone, flint and slate and in some metallic ores. Silica can be a main component of sand. It can also be in soil, mortar, plaster and shingles. The cutting, breaking, crushing, drilling, grinding or abrasive blasting of these materials may produce fine silica dust. Silica occurs in 3 forms: crystalline, microcrystalline (or cryptocrystalline) and amorphous (non-crystalline). "Free" silica is composed of pure silicon dioxide, not combined with other elements, whereas silicates (e.g. talc, asbestos and mica) are SiO_2 combined with an appreciable portion of cations.

Crystalline silica exists in 7 different forms (polymorphs), depending upon the temperature of formation. The main 3 polymorphs are quartz, cristobalite, and tridymite. Quartz is the second most common mineral in the world (next to feldspar).

Microcrystalline silica consists of minute quartz crystals bonded together with amorphous silica. Examples include flint and chert. **Amorphous silica** consists of kieselgur (diatomite), from the skeletons of diatoms, and vitreous silica, produced by heating and then rapid cooling of crystalline

silica. Amorphous silica is less toxic than crystalline, but not biologically inert, and diatomite, when heated, can convert to tridymite or cristobalite. Silica flour is nearly pure SiO_2 finely ground. Silica flour has been used as a polisher or buffer, as well as paint extender, abrasive, and filler for cosmetics. Silica flour has been associated with all types of silicosis, including acute silicosis. Silicosis is due to deposition of fine respirable dust (less than 10 micrometers in diameter) containing crystalline silicon dioxide in the form of alpha-quartz, cristobalite, or tridymite.⁹

Diagnosis: There are three key elements to the diagnosis of silicosis. First, the patient history should reveal exposure to sufficient silica dust to cause this illness. Second, chest imaging (usually chest x-ray) that reveals findings consistent with silicosis. Third, there are no underlying illnesses that are more likely to be causing the abnormalities. Physical examination is usually unremarkable unless there is complicated disease. Also, the examination findings are not specific for silicosis. Pulmonary function testing may reveal airflow limitation, restrictive defects, reduced diffusion capacity, mixed defects, or may be normal (especially without complicated disease). Most cases of silicosis do not require tissue biopsy for diagnosis, but this may be necessary in some cases, primarily to exclude other conditions. For uncomplicated silicosis, chest x-ray will confirm the presence of small (< 10 mm) nodules in the lungs, especially in the upper lung zones. Using the ILO classification system, these are of

profusion 1/0 or greater and shape/size "p", "q", or "r". Lung zone involvement and profusion increases with disease progression. In advanced cases of silicosis, large opacity (> 1 cm) occurs from coalescence of small opacities, particularly in the upper lung zones. With retraction of the lung tissue, there is compensatory emphysema. Enlargement of the hilum is common with chronic and accelerated silicosis. In about 5-10% of cases,

the nodes will calcify circumferentially, producing so-called "eggshell" calcification. This finding is not pathognomonic (diagnostic) of silicosis. In some cases, the pulmonary nodules may also become calcified. A computed tomography or CT scan can also provide a more detailed analysis of the lungs, and can reveal cavitations due to concomitant mycobacterial infection.



Figure-4: Silica Dust inhalation by workers^[9,10]

(9. Griffin DW (2007). Atmospheric movement of microorganisms in clouds of desert dust and implications for human health. Clin. Microbiol. Rev. 20(3): 459–747.

10. Norboo T, Angchuk PT, Yahya M, et al. (1991). Silicosis in a Himalayan village population: role of environmental dust. Thorax 46(5): 341–343.)

Prevention: The best way to prevent silicosis is to identify work-place activities that produce respirable crystalline silica dust and then to eliminate or control the dust ("primary prevention"). Water spray is often used where dust emanates. Dust can also be controlled through dry air filtering. Following observations on industry workers in Lucknow (India), experiments on rats found that jaggery (a traditional sugar) had a preventive action against silicosis.

Treatment: Silicosis is an irreversible condition with no cure. Treatment options currently focus on alleviating the symptoms and preventing complications. These include: Stopping further exposure to silica and other lung irritants, including tobacco smoking, Cough suppressants, Antibiotics for bacterial lung infection. TB prophylaxis for those with positive tuberculin skin test or IGRA (Interferon- γ release assays) blood test. Prolonged anti-tuberculosis (multi-drug regimen) for those with active TB. Chest physiotherapy to help the bronchial drainage of mucus. Oxygen administration to treat hypoxemia, if present. Bronchodilators to facilitate breathing.

Lung transplantation to replace the damaged lung tissue is the most effective treatment, but is associated with severe risks of its own. For acute silicosis, broncho-alveolar lavage may alleviate symptoms, but does not decrease overall mortality. Experimental treatments include:

Inhalation of powdered aluminium, d-penicillamine and polyvinyl pyridine-N-oxide. Corticosteroid therapy. The herbal extract tetrandine may slow progression of silicosis.¹⁰

CONCLUSION

Silicosis is a lung disease that is caused by inhaling tiny bits of silica. Silica is a common mineral that is part of sand, rock and mineral ores like quartz. People who work in jobs where they could breathe in these tiny silica bits—like sandblasting, mining, construction and many others—are at risk for silicosis. The silica dust can cause fluid buildup and scar tissue in the lungs that cuts down your ability to breathe. Silicosis cannot be cured, but you can prevent it if you take specific steps to protect yourself and your family.

ACKNOWLEDGEMENT

This article has been contributed by four international authors (**Dr. Dhrubo Jyoti Sen** from **India**, **Kinsuk Sarker** from **Bangladesh**, **Clive Dadida** & **Paul Dhliwayo** from **Zimbabwe**).



Prof. Dr. Dhrubo Jyoti Sen



Kinsuk Sarker



Clive Dadida



Paul Dhliwayo

Mr. Sarker, Mr. Dadida & Mr. Dhliwayo all are currently studying in B.Pharm. Sem-III at Shri Sarvajani Pharmacy College, Mehsana, Gujarat, India (under the ICCR scholarship scheme) and guided by Dr. Sen. All the authors are thankful to the internet facility as well as the reference books which were provided by the library of Shri Sarvajani Pharmacy College for collecting the matters regarding the topic.

REFERENCES

1. Definition of pneumonoultramicroscopicsilicovolcanoconiosis in Oxford dictionary (British and World English) (2012). Oxford Dictionaries. Oxford University Press.
2. Cole Chris (1999). Wordplay, A Curious Dictionary of Language Oddities. Sterling Publishing Co., Inc. 106–107.
3. Pelucchi C, Pira E, Piolatto G, Coggiola M, Carta P, La Vecchia C, et al. (2006). Occupational silica exposure and lung cancer risk: a review of epidemiological studies 1996-2005.. *Ann. Oncol.* 17(7): 1039–1050.
4. Cassel SL, Eisenbarth SC, Iyer SS, et al. (2008). The Nalp3 inflammasome is essential for the development of silicosis. *Proc. Natl. Acad. Sci. U.S.A.* 105(26): 9035.
5. Wagner GR (1997). Asbestosis and silicosis. *Lancet* 349(9061): 1311–1315.
6. Sahu AP, Saxena AK, et al. (1994). Enhanced Translocation of Particles from Lungs by Jaggery. *Environmental Health Perspectives.* 102(S5): 211–214.
7. Steenland K, Goldsmith DF, et al. (1995). Silica exposure and autoimmune diseases. *Am. J. Ind. Med.* 28(5): 603–608.
8. Nouh MS (1989). Is the desert lung syndrome (nonoccupational dust pneumoconiosis) a variant of pulmonary alveolar microlithiasis? Report of 4 cases with review of the literature. *Respiration* 55(2): 122–126.
9. Griffin DW (2007). Atmospheric movement of microorganisms in clouds of desert dust and implications for human health. *Clin. Microbiol. Rev.* 20(3): 459–747.
10. Norboo T, Angchuk PT, Yahya M, et al. (1991). Silicosis in a Himalayan village population: role of environmental dust. *Thorax* 46(5): 341–343.