Occupational Hazards in India

From a pulomonologist’s point of view

R Lakshmi Narasimhan
History

- Mining and metallurgy goes back to ancient times
- Rock cutting and stone carving to build temples also present since a long time in India
- Occupational lung diseases mentioned in Ancient texts ➔ 4<sup>th</sup> century AD
- Emerged with increasing industrialisation
- C. Krishnaswami Rao was first to confirm cases of Silicosis in India in 1934*

*Current Science, Page No. 283-284 “Incidence of Silicosis in Kolar Gold Fields, Mysore”
Silicosis

- Most prevalent chronic occupational lung disease*
- Irreversible and chronic fibrotic disease caused by inhalation, retention and pulmonary reaction to large amounts of silica dust (SiO₂)
- Mining, stone cutting, ceramic, pottery, agate, brick making, slate pencil, etc. are a few of the many industries which are particularly at risk

*Silicosis - An Uncommonly Diagnosed Common Occupational Disease, ICMR Bulletin Sep 1999
Prevalence of Silicosis

- First Silicosis Survey in Kolar Gold Fields (1940-1946) by Caplan et al
- Of the 7653 workers examined in Kolar Gold Fields, 3402 (43.7%) cases of silicosis were detected
- Prevalence varies widely among various industries
- Lowest in Iron & Steel, Ordinance factories (2.5-3.5%)
- Highest in Agate, Slate Pencil, Lead, Zinc & Mica mining and Stone cutting/Quartz Grinding (>30%)
Silicosis in Indian Mines

<table>
<thead>
<tr>
<th>Mines</th>
<th>Year</th>
<th>Silicosis cases</th>
<th>No. of cases investigated</th>
<th>Prevalence of Silicosis</th>
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<tbody>
<tr>
<td>Kolar Gold fields</td>
<td>1940-1946</td>
<td>3402</td>
<td>7643</td>
<td>43.7</td>
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<tr>
<td>Mica</td>
<td>1953</td>
<td>112</td>
<td>329</td>
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<tr>
<td>Manganese</td>
<td>1960</td>
<td>10</td>
<td>243</td>
<td>4.1</td>
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<tr>
<td>Lead &amp; Zinc</td>
<td>1961</td>
<td>52</td>
<td>171</td>
<td>30.4</td>
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<tr>
<td>Iron Ore</td>
<td>1968</td>
<td>42</td>
<td>177</td>
<td>23.7</td>
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<tr>
<td>Mica</td>
<td>1976</td>
<td>19</td>
<td>110</td>
<td>17.2</td>
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<tr>
<td>Stone</td>
<td>1995</td>
<td>41</td>
<td>416</td>
<td>9.9</td>
</tr>
<tr>
<td>Gold</td>
<td>2001</td>
<td>313</td>
<td>4883</td>
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<tr>
<td>Zinc</td>
<td>2002</td>
<td>131</td>
<td>442</td>
<td>29.6</td>
</tr>
<tr>
<td>Uranium</td>
<td>2002</td>
<td>9</td>
<td>91</td>
<td>9.9</td>
</tr>
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</table>

*Chief Advisor of Factories  # Directorate General of Mines Safety  @ National Institute of Occupational Health
<table>
<thead>
<tr>
<th>Industry</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emery polishers</td>
<td>0.7</td>
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<tr>
<td>Iron and Steel</td>
<td>2.5</td>
</tr>
<tr>
<td>Ordnance factory</td>
<td>3.5</td>
</tr>
<tr>
<td>Mica processing</td>
<td>5.2</td>
</tr>
<tr>
<td>Glass bangle workers</td>
<td>7.3</td>
</tr>
<tr>
<td>Quartz crushing</td>
<td>12.0</td>
</tr>
<tr>
<td>Quartz mill-stone grinding</td>
<td>14.0</td>
</tr>
<tr>
<td>Ceramics and pottery</td>
<td>15.1</td>
</tr>
<tr>
<td>Brick makers</td>
<td>16.7</td>
</tr>
<tr>
<td>Stone cutters</td>
<td>19.1 – 35.2</td>
</tr>
<tr>
<td>Stone grinding</td>
<td>27.8</td>
</tr>
<tr>
<td>Agate workers</td>
<td>29.1 - 38.0</td>
</tr>
<tr>
<td>Slate pencil workers</td>
<td>54.6</td>
</tr>
</tbody>
</table>

Silicosis In Indian Factories
References

6. NIOH Annual report 1985-86
12. NIOH Annual report 1988-89
Clinical Course – 3 forms

- Chronic/Classic Silicosis
- Accelerated Silicosis
- Acute Silicosis
Chronic Silicosis

- Develops following low-to-moderate level exposure to silica dust for >20 yrs
- 1st ➔ Silica laden macrophages accumulate
- Later ➔ Silicotic nodules form as a result of host response to the foreign body
- Nodules mainly seen in upper lobes
- Calcified LN maybe seen
- Nodules enlarge and coalesce (>2cm) ➔ PMF or complicated silicosis
- Increased susceptibility to TB and cavitation
Silicotic nodule

- Central area organised with concentric whorl-like collagen fibres with inflammation in periphery

- Also called histological tornadoes
Accelerated Silicosis:
- Heavy silica exposure in <5-10 yrs
- Progresses faster than chronic silicosis
- Sometimes associated with CTD

Acute silicosis
- V.High concentration of silica exposure over weeks to months – eg. Sandblasters, rock drilling, etc
- B/l alveolar opacities without silicotic nodules
- Intense inflammatory reaction due to freshly fractured silica particles
- Hypertrophic Type II pneumocytes $\rightarrow$ produce excess surfactant $\rightarrow$ Resembles PAP
Complications

- *Tuberculosis*
- Cor pulmonale
- Spontaneous pneumothorax
- Broncholithiasis
- Tracheobronchial obstruction
- Lung cancer
- Hypoxemic ventilatory failure
Silico-tuberculosis

• The association of Silicosis and TB has been suspected several hundred years
• In 1902 JS Holdene committee reported that “Stone dust predisposes enormously to TB in the lung”
• Exposure to silica causes a renewed multiplication of bacilli in the healing TB lesions
Incedence

- In autopsy material – over 25 %
  \((\text{Gooding CG et al. Lancet, 2:891,1946})\)

- In India silicotuberculosis incidence -
  28.6% (Sikand BK, Pamra SP Proceedings of Seventh TB workers conference, 1949)

  10.7% in stone cutters, 22.5 % in Slate Pencil Workers
  (Tiwari RR et al, NIOH 2007)

  23% in stone quarries of Rajasthan (P K Sishodiya et al, NIMH 2012)

  12% with silicosis had Sputum Positive PTB (Keerthivasan et al, 2013)

- TB is 3 to 7 times higher in Indians with silicosis
  \((\text{Gupta SP et al. India J Med Res 1972})\)
Pathogenesis – Macrophage dysfunction

- **SILICA DUST**
  - Phagocytosis by AM
    - Damage cell membrane of AM
      - Inactivate AM/Death
        - IL-1/ TNF-α
          - Fibroblast activation
            - Fibrosis
          - Stimulate Neutrophils
            - Release oxidants
              - Local damage
Iron Hypothesis

- Mycobacteria are dependent on iron for growth and produce the iron chelator - mucobactin
- Silica particles absorbed body iron and act as a reservoir of iron
- Silicato-iron complexes may activate dormant tubercle bacilli
Interaction of silicosis with TB

- Increased risk of PTB in silicosis
- Exposure of silica has an unfavourable influence on the course of induced TB
- There is more fibrosis produced by combination
- Synergistic effect of silicosis and TB – proliferative fibrous reaction \( \rightarrow \) Rapid fibrosis
- TB may complicate simple silicosis as well as advanced disease
- It may develop PMF with cavitation
- Poor response to ATT \( \rightarrow \) Longer duration needed
Diagnostic dilemma

- Symptoms of silicosis and silicoTB are misleading
- Interpretation of the Chest X ray film of the silicotic is difficult
- The recovery of AFB in the sputum of patients suffering from silicotuberculosis is difficult.
- Because of walling in of the tubercle foci by silicotic fibrosis which prevents the discharge of tubercle bacilli in the sputum
Pointers to TB

- Clinical - Fever, Expectoration, Hemoptysis, LoA, LoW
- In miliary TB patient is toxaemic – compared to simple chronic silicosis
- Poorly demarcated “soft” infiltrates of variable size that do not cross the lung fissures s/o TB
- Opacities may surround pre-exiting silicotic nodules
- Presence of a cavity in a nodule
Pointers to TB

- The nodules in miliary tuberculosis are smaller than those in silicosis.
- The radiographs of patients with silicosis usually show increased translucency as against general loss of translucency in tuberculosis.
- The distinction between adult type (post-primary) tuberculosis and PMF radiological shadows difficult. However, the conglomerate shadows of silicosis do not show cavitation.
- Associated pleural/pericardial effusion.
- Rapid worsening in radiology.
ATT

- Prolongation of the continuation phase from 4 to 6 months decreased the rate of relapse from 22 to 7% (Blumberg et al. Am J Resp & crit care Med Feb 15-2003)
Treatment of Silicosis

- No specific therapy for silicosis
- Prevent further exposure to silica dust
- Strongly advise patients to quit smoking
- Immunize against influenza, pneumococci
- Experimental approaches tried without success are - whole-lung lavage, aluminum inhalation, and corticosteroids
- Screen for TB with sputum AFB x 2
- Complications should be treated appropriately
Prevention

- Dust suppression,
- Process isolation,
- Ventilation,
- Use of non–silica–containing abrasives.
- Respiratory masks
- Surveillance of exposed workers with respiratory questionnaires, spirometry, and chest x-rays is recommended
Chest X-ray Schedule

<table>
<thead>
<tr>
<th>Duration</th>
<th>Age</th>
<th>X-ray schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;10 years</td>
<td>All age</td>
<td>Every 5 years</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>&lt;30 years</td>
<td>Every 5 years</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>35-44 years</td>
<td>Every 2 years</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>&gt;45 years</td>
<td>Every year</td>
</tr>
</tbody>
</table>

*(Donaldson k et al. Ann Occ Hyg 1998;42)*
Diseases associated with exposure to Silica dust

- Occupational asthma
- Chronic obstructive pulmonary disease
  - Emphysema
  - Chronic bronchitis
- Mineral dust induced small airway disease
- Lung cancer
- Mycobacterial infection
  - MTB
  - NTM
- Immune –Related Disease
  - PSS, RA, CRD, SLE
Why silicosis is a problem in India?
### Population at risk for silicosis in India

<table>
<thead>
<tr>
<th>Industry</th>
<th>No. of workers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manufacturing of basic metals &amp; alloys (Steel, Copper, Ferro-alloys, etc.)</td>
<td>6,29,000</td>
</tr>
<tr>
<td>Mines and Quarries</td>
<td>17,00,000</td>
</tr>
<tr>
<td>Manufacturing of products (Refractory, Glass, Mica, etc)</td>
<td>6,71,000</td>
</tr>
<tr>
<td>Construction sector</td>
<td>70,00,000</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>1 Crore</strong></td>
</tr>
</tbody>
</table>

**DOES NOT INCLUDE WORKERS WHO ARE SELF-EMPLOYED OR IN UNORGANISED SECTOR**
Coal Workers Pneumoconiosis

- Coal dust consists of carbon (60-80%), apart from 50 different elements and oxides – including Silica
- Higher the quality of coal – higher the silica content in the dust
- 2 forms: simple CWP and PMF
- Three Criteria needed for diagnosis of CWP:
  - CXR consistent with CWP
  - A work history sufficient in exposure and latency to cause CWP
  - Absence of other illnesses which mimic CWP
Pathogenesis

- Direct toxicity of coal dust
- Release of oxidants, enzymes and cell membrane constituents from activated macrophages
- Cytokine release from macrophages which recruits other effector cells → fibroblast proliferation → Collagen synthesis
- But overall, coal dust less fibrogenic than silica
Simple CWP

- Small rounded opacities – from pinhead sized to 1 cm
- 1st upper zones → then all over lung fields
- Slowly progressive illness over decades
- Chest radiograph correlates with amount of coal dust inhaled
- Pathology: Coal macule is characteristic lesion
- Consists of coal dust, reticulin fibres and coal laden macrophages
- Later enlarges and forms coal nodule
- Surrounded by focal area of emphysema
- Silicotic nodules may coexist
Coal macule
Coal nodule
PMF

- When one or more nodules attain a size of >2cm
- MC in posterior segments of upper lobes or superior segments of lower lobes
- Asymmetrical
- Development influenced by:
  - Combined inhalation of silica
  - NTM infection
  - Immunologic response
CWP in India – ICMR study

- ICMR study (1986-1993) of 5777 underground coal miners and 1236 surface coal miners.
- Prevalence of pneumoconiosis in underground coal miners was 2.84% and in the surface coal workers it was 2.10%
- Majority of the cases of pneumoconiosis (84.1% of total cases) in underground coal miners belonged to category 1
- There were no cases of pneumoconiosis higher than category 2
- Only 3 cases of PMF were found in underground coal miners and none in surface coal workers.
CWP in India – ICMR study

- Prevalence of chronic respiratory symptoms amongst underground miners was 31.3%, significantly higher than surface coal workers (17.3%)

- Overall prevalence of functional abnormalities of lung in underground coal miners and surface coal workers was 45.4% and 42.2% respectively. Prevalence of obstructive type of abnormalities amongst underground coal miners and surface coal workers was 28.9% and 24.1% respectively.

- The environmental study indicated that the air borne dust concentrations were much higher than the suggested threshold limit values (TLV) in underground and surface coal mines.

- This study established a low prevalence of pneumoconiosis and absence of more severe cases of pneumoconiosis in Indian coal miners

- Also reported very high prevalence of non pneumoconiotic respiratory morbidity in coal miners
CWP in India – Parihar et al 1997

- 75351 coal workers in 72 collieries
- Overall prevalence found to be 3.03%, ranging from 1.52% to 4.76% between 10 areas
- Most cases were category-I (81.09%), followed by category-II (17.84%).
- Only 3 cases of PMF were detected.
- Round shaped opacities are predominant (89.59%) in Coal Worker's Pneumoconiosis.
- Among the opacities, 'p' type was more prevalent (48.29%) followed by 'q' type (40.62%).
### CWP in India- Decreasing trend

<table>
<thead>
<tr>
<th>Study</th>
<th>No. of Participants</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roy et al 1957</td>
<td>550</td>
<td>15</td>
</tr>
<tr>
<td>Ministry of Labour and Employment. Govt. of India. 1961 (Pilot study)</td>
<td>621</td>
<td>18.5</td>
</tr>
<tr>
<td>CMRS 1952</td>
<td>952</td>
<td>7</td>
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<tr>
<td>Vishwanathan R.et al 1972</td>
<td>8822</td>
<td>10.8</td>
</tr>
<tr>
<td>Vishwanathan R.et al 1977</td>
<td>455</td>
<td>3.5</td>
</tr>
<tr>
<td>ICMR study 1993</td>
<td>5777</td>
<td>2.84</td>
</tr>
<tr>
<td>Parihar et al 1997</td>
<td>75351</td>
<td>3.03</td>
</tr>
</tbody>
</table>
Reasons for declining incidence

- In 1978, Mines Act was amended vide which Periodical Medical Examination (P.M.E.) of all persons working in mines were made compulsory as well as the Initial Medical Examination (I.M.E.) as provided in Sec. 29 B of the Mines Rules.
- It stated that all workers have to undergo a P.M.E once in every 5 years → Clinical and CXR, AFB/Mtx as needed
- C.W.P has been made a notifiable disease under Mines Act and is a compensable disease as per the Workmen's Compensation Act.
- Increased preventive safety measures put in by Mining industry
Other diseases caused by coal

- COPD
- Industrial bronchitis
- Caplan’s syndrome

Complications:
- Cor pulmonale
- Pneumothorax
- Hypoxemic Respiratory failure
- Silicotuberculosis more common when exposed to high levels of silica in coal dust
Asbestosis

- Exposure to asbestos causes asbestosis, lung cancer and mesothelioma of pleura and peritoneum.
- In India, the total use of asbestos is 1.25 lakh tonnes, out of which more than 1.0 lakh tonnes is being imported. Significant occupational exposure to asbestos occurs mainly in asbestos cement factories, asbestos textile industry and asbestos mining and milling.
- Approximately 1,00,000 persons exposed at risk
Asbestos in Cement Industry

- There are 18 asbestos cement factories located in different parts of the country.
- The prevalence of asbestosis in these factories varied from 3% to 5%. The levels of asbestos fibres were found to be higher than the permissible levels of 2fibres/ml in two of the factories.*

*NIOH, 1996
Asbestos in Textile Industry

- Making of asbestos yarn and ropes is done mostly in the unorganised sector of industries with very poor safety measures.
- The average levels of air borne asbestos fibres varied from 216 to 418 fibres/ ml. The permissible level is 2 fibres/ml.
- The prevalence of asbestosis was 9%. This relatively low prevalence of asbestosis despite high environmental levels was attributed to high labour turn over.*
- Cases of asbestosis were observed in workers having less than 10 years exposure in contrast to the reported average duration of over 20 years in previous studies

*NIOH study
Asbestos Mining and Milling

- In asbestos mines the air borne fibre levels were within permissible limits.
- The average fibre levels in milling units varied from 45 fibres/ml to 244 fibres/ml of air. (Permissible level = 2 fibres/ml)
- The overall prevalence of asbestosis in mining and milling units was 3% and 21% respectively.*
- Another study in milling units revealed a prevalence of 22%.

*NIOH study
Mesothelioma

- No Indian studies available
- But it is predicted that incidence similar to Western countries
Byssinosis

- Byssinosis is an occupational lung disease caused by exposure to cotton, flax and hemp dust.
- Presents with asthma-like symptoms
- Maximum number of workers with byssinosis are reported in the cotton textile industry as it is one of the largest industries in the world.
- In India, there are about 1.07 million workers engaged in the manufacture of cotton textiles. The workers engaged in the initial processes of textile manufacturing (blow, card, frame and ring frame) are exposed to cotton dust and develop the disease after some years of exposure.
Prevalence of Byssinosis in India

- Several studies have reported byssinosis in India but they failed to demonstrate the severity and magnitude of the disease.
- The low prevalence reported in those studies created an impression that the disease is not an important problem.
- Studies conducted by NIOH have shown a very high prevalence of the disease especially in blow (30%) and card (38%) sections of textile industries.
Issues to be addressed in India

- Absence of National Policy on Prevention and Elimination of Silicosis
- Absence of central authority to coordinate activities of various agencies
- Official statistics on morbidity and mortality not available
- No large scale recent epidemiological studies
- Inadequate enforcement of legislation
- No central registry for cases of silicosis
- Lack of accountability on part of enforcement agencies and industry
Issues to be addressed in India

- Lack of awareness among workers, employers and doctors
- Inadequate infrastructure for diagnosis and management
- Small scale and unorganized sector not covered by legislation
- Poor quality or absence of health surveillance programme in industry
- Cases notified reflect only tip of Iceberg – most cases not recognised/reported
- Misdiagnosis and treatment of silicosis as tuberculosis
- Lack of coordination among stake holders for elimination of silicosis/asbestosis
- Asbestos not yet banned