

Comparative Study Of Sputum Cytology In Construction Workers With A Control Population.

¹Madasamy Balamurugan, ²Jamunarani Srirangaramasamy,

³Duraipandian Jeyakumari, ⁴Aarthy Kumaraguru,

¹Professor and HOD, Department of Pathology, ²Assistant Professor, Department of Pathology,

³Professor, Department of Microbiology, Tagore Medical College and Hospital, Tamilnadu Dr MGR University, Chennai, Tamilnadu, India.

⁴Student, MBBS, Sri Manakula Vinayagar Medical College and Hospital, Pondicherry University, Pondicherry, Pondicherry Union Territory, India.

ABSTRACT:

Background and Objectives: Construction workers are exposed to a wide range of substances that are potentially hazardous to the respiratory system. In addition, factors, such as cigarette smoking also add to their morbidity. The prevalence of most respiratory disorders can be identified by the presence of inflammatory cells in the sputum. Sputum cytology is a simple, economic and a non- invasive method. The objective of this study is to estimate the susceptibility of lung diseases among the construction workers using sputum cytology with office workers as a control. **Methods:** 50 construction workers near our hospital were selected using inclusion and exclusion criteria formed test group. 50 clerical staff were taken as a control group. Sputum samples were collected from both groups. Smears were made and stained with Papanicolou, Giemsa and Toluidine blue stains. The smears were screened and cell populations were counted as cells per High Power Field (HPF). **Results:** Different cells were counted as cells/HPF, tabulated from both groups and analyzed statistically (Student T-test). **Interpretation and Conclusions:** Statistically significant increase in certain cells in the study group were observed. This study points to the exposure and risk of construction workers to diseases such as cancer.

Key-words: Construction workers, occupational exposure, sputum cytology.

Corresponding Author: M.Balamurugan, Professor and HOD, Department of Pathology, Tagore Medical College and Hospital, Tamilnadu Dr.MGR Medical University, Chennai-127. E-mail – balamuruganpath@tagoremch.com Mobile: 09551757371

INTRODUCTION

Occupational hazards are one of the leading causes of respiratory morbidity and mortality. According to National Mortality Statistics in 2008, pulmonary diseases rank 3rd among the causes of death in India.¹ The damaging effects of occupational exposure may be difficult to evaluate since workers may be exposed to several toxic substances and infectious materials in their place of work.² Many of the construction workers are exposed to a wide range of dust such as cement, wood dust from sawing, fumes from welding, dust from the soil, diesel exhaust from machines, dust from the air, which includes noxious gases like sulphur dioxide,

ozone [O₃], anthracotic dust and asbestos fibers.³ In addition, personal lifestyle factors, such as cigarette smoking have also been identified as etiological factors of respiratory diseases.

Several studies have demonstrated that long term exposure and inhalation of high concentration of toxic substances can provoke bronchoconstriction, increased nonspecific airway responsiveness and muco-ciliary dysfunction. The most common clinical features are cough, phlegm production, and impairment of lung function, bronchial asthma, emphysema, chest tightness and restrictive lung diseases.⁴ The inhalation of toxic substances also

induces atrophic changes in the nasal and pharyngeal mucosa, cancer of lung, stomach and colon.^{5,6} The toxic substances may enter into the systemic circulation and thereby reach all the organs of the body, including heart, liver, spleen bone and muscles.⁷ Also, it affects the iron homeostasis of the lung by causing oxidative stress injury of the alveolar macrophages.^{8,9,10}

The construction workers are also more susceptible to infective diseases which may be dust borne, soil borne, water borne or vector borne. Most common infectious agents affecting the construction workers are *Aspergillus*, *Mycobacterium tuberculosis*, *Blastomyces*, *Cryptosporidium*, *Coccidioidomyces* and *Histoplasma*. These organisms can be identified in sputum smears.^{11,12} A comprehensive review about the morbidities and mortality in construction workers and the usefulness of sputum cytology is discussed by Gary M.Liss.¹³

The efficacy of sputum cytology is proven and it is a simple, economic, and non-invasive method. The prevalence of most respiratory disorders can be identified by the presence of inflammatory cell changes in the sputum.^{14,15} Mast cells, which are metachromatic¹⁶ are known to be present in allergic and atopic conditions and their presence in sputum could be a marker for identifying individuals with these disorders. This present study is designed to observe and compare the cellular changes of sputum in construction workers with a control population.

MATERIALS AND METHODS

This study was conducted among the construction workers who fulfilled the inclusion criteria, working at the construction sites near the hospital.

Inclusion criteria:

- Construction workers and office workers of 18 to 50 years are included.
- The duration of exposure to construction work by the construction worker should be at least 2 years.
- Apparently asymptomatic, healthy subjects.

Exclusion criteria:

- Smokers were not considered for this study since it will affect the sputum cellularity. Acute respiratory illness, oral lesions and infections in ear, nose, throat
- Individuals having past history of any chronic respiratory disorders like tuberculosis etc.
- History of cardiopulmonary disease, and malignancy

Institute ethical committee approval was obtained prior to the study. After the briefing, exclusion criteria were used to shortlist the workers. To the selected workers, after taking the consent, the method of collection of sputum was explained. Clean, sterile, labeled plastic containers were given to 50 construction workers. They were advised to try and collect early morning sputum by forcible cough and were asked to deliver the samples to the Clinical Pathology laboratory within 2 hours as described by Pizzichini and coworkers¹⁴. If the sputum was not satisfactory deep cough expectoration was advised. The fact that many construction workers were always having some low grade, the subclinical reaction of the airways to the dusty atmosphere, sample collection was adequate most of the time.

The collected material was poured into a petri dish, and the more opaque and gelatinous portions, which looked different from saliva, are all selected with blunt forceps and put into another petri dish. The selected portion is then moved around to remove a bit more of the saliva and smeared and numbered¹⁶. Six smears were prepared from the most representative part of the sample and numbered for each subject. Two of them were fixed with Carnoy's fluid and four were air dried. Fixed slide was stained with Papanicolaou stain and air dried slides were stained with Giemsa stain and with Toluidine blue stain. Remaining slides were kept as reserve. These slides were examined for cell types and a differential was done on the Pap and Giemsa smears. Toluidine blue stained slides were used to identify the mast cells.

Sputum samples of 50 volunteers who are primarily working in our college office acted as control and similar procedures were carried out. Sputa without macrophages were considered not representative of pulmonary origin (inadequate). The presence of normal, metaplastic and abnormal epithelial cells, inflammatory leukocyte infiltrates which include macrophages, neutrophils, eosinophils, lymphocytes and mast cells were analyzed.¹⁶ The count of the cells in the smears was carried out using a light microscope with 10X ocular and 40X objective. A total of 20

fields per slide was analyzed, chosen randomly at 3mm intervals.¹⁷ The assessment of cellularity was done as cells/HPF by the Pathologist. The findings were recorded; the cells were classified and tabulated. Student's 'T'test was done using SPSS software.

RESULTS

The various cell populations in the sputum of both groups (Table-1) and their significance in variations (Table 2) are given in Table-1 & 2.

Table:1 Distribution of the cells in both groups

Group Statistics					
Cells	Groups	N	Mean	SD	SEM
Squamous cells	Control group	50	1.6800	.58693	.08300
	Const. group	50	2.2800	.49652	.07022
Metaplastic cells	Control group	50	.2400	.55549	.07856
	Const. group	50	1.8400	.68094	.09630
Columnar cells	Control group	50	.0600	.23990	.03393
	Cons. group	50	.1600	.37033	.05237
Macrophages	Control group	50	1.6000	.63888	.09035
	Cons. group	50	2.1200	.59385	.08398
Polymorphs	Control group	50	1.2600	.59966	.08480
	Cons. group	50	2.0400	.69869	.09881
Eosinophils	Control group	50	.1400	.35051	.04957
	Cons. group	50	.4200	.53795	.07608
Mast cells	Control group	50	.0000	.00000	.00000
	Const. group	50	.0600	.23990	.03393
Lymphocytes	Control group	50	1.2200	.41845	.05918
	Const. group	50	1.3200	.51270	.07251

Table:2 The significance and confidence interval of differences between both groups

Cell population	T-test for Equality of Means						
	T	df	p(2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
Squamous cells	-5.51	95.380	.000	-.60	.10872	-.815	-.384
Metaplastic cells	-12.87	94.200	.000	-1.60	.12428	-1.846	-1.353
Columnar cells	-1.60	83.967	.113	-.10	.06240	-.224	.024
Macrophages	-4.21	97.481	.000	-.52	.12335	-.764	-.275
Polymorphs	-5.99	95.797	.000	-.78	.13021	-1.038	-.521
Eosinophils	-3.08	84.252	.003	-.28	.09080	-.460	-.099
Mast cells	-1.76	49.000	.083	-.06	.03393	-.128	.008
Lymphocytes	-1.06	94.217	.288	-.10	.09359	-.285	.085

Squamous cells (p-value = <005), metaplastic squamous cells (p-value = <005), macrophages (p-value = <005), polymorphs (p-value = <005) and eosinophils (p-value = .003) show significant p-values and confidence intervals. Columnar cells, mast cells and lymphocytes do not show significant differences between the groups.

DISCUSSION

The cell populations in our control group were comparable to two similar studies, Twisha Lahiri et al., 2008, Delhi, India¹ and Jose Belda et al., 2000, Ontario, Canada.¹⁶ Our study showed an increased overall cellularity in the study group. This is comparable to a study done by Fell AK et al., in cement production workers in 2009, Norway.¹⁸ Polymorphs, macrophages and eosinophils showed statistically significant increase. This clearly shows that the construction workers have an increased baseline inflammation of the airways and can lead to complications like Chronic Obstructive Pulmonary Diseases (COPD).^{19,20} This result is comparable to studies conducted by Mauro Alderisio in

Italy, 2006 and Matti S. Huuskonen, Finland in 1978.^{17,21}

The squamous cells, particularly metaplastic squamous cells showed statistically significant increase in the study group. This result is also comparable to studies conducted by Mauro alderisio in Italy, 2006 and Matti S. Huuskonen, Finland in 1978.^{19,21} With metaplasia there is always the risk of dysplasia and increased incidence of cancer as indicated by Rafnsson et al., in 1997, who suggested it may be due to the presence of hexavalent chromium in cement.⁵

Indian studies in sputum cytology of construction workers were hard to find for comparison. Most of the information about sputum cytology in construction workers is from the book 'Occupational and Environmental Lung Diseases' by Susan M Tarlo, Paul Cullinan, Benoit Nemery¹³ which is European and Canadian data. More details of morbidity have been described in this book, like fungal infections in lungs. Special stains can be used to identify the parameters like fungal infections. Our study is confined to a small area in Pondicherry, southern India, with a moderate sample size. More

studies like lung function, atmospheric particle density, blood parameters and soluble sputum substances are required to assess the construction workers respiratory health status.

CONCLUSION

Construction workers are a specific group, which is more exposed to hazardous substances compared to the normal population. They have high cellularity in sputum particularly inflammatory cells and metaplastic cells, which makes them prone to chronic respiratory disorders and

malignancies. Construction is one of the largest ongoing industries in India and given the state of our construction worker's safety precautions, accommodation and other facilities, more positive findings can be obtained by studying them in large numbers.

Source of Funding: This study was funded by ICMR as STS project, done by Ms.Aarthy Kumaraguru guided by Dr.M.Balamurugan in 2011.

Conflict of Interest: None.

References

1. Lahiri T, Ray MR, Sengupta B, Trivedi RC. Epidemiological Study On Effect Of Air Pollution On Human Health (adults) In Delhi, Central Pollution Control Board, Ministry of Environment & Forests, Govt. of India Environmental Health Series, EHS/1/2008.
2. Yang CY, Huang CC, Chiu HF, Chiu JF, Lan SJ, Ko YC. Effects of Occupational dust exposure on the respiratory health of Portland cement workers. *J Toxicol Environ Health*, 1996;49:581-588.
3. Short S, Petsonk EL. Non-fibrous inorganic dusts. In: Philip Harber, Marc B Schenker and John R Balmes. Occupational and environmental respiratory disease, London. Mosby, 1999;356.
4. AbouTaleb ANM, Musaniger AO, Abdel Moneim RB. Health status of cement workers in the United Arab Emirates. *J Roy Soc Health* 1995;2:378-83.
5. Rafnsson V, Gunnarsdottir H, Kiilunen M. Risk of lung cancer among masons in Iceland. *Occup Environ Med*, 1997;54:184-188.
6. McDowall ME. A mortality study of cement workers. *Br J Ind Med*, 1984;41:179-182.
7. Meo S. Health hazards of cement dust. *J Occup Hyg*, 2004;25(9):1153-59.
8. Mateos F, Brock JH, Perez-Arellano JL. Iron metabolism in the lower respiratory tract, *Thorax*, 1998;53:594-600.
9. Andrew J, Jennifer LT. Iron homeostasis in the lung. *Biol Res*, 2006;39:67-77.
10. Roy S, Ray MR, Basu C, Lahiri P, Lahiri T: Abundance of siderophages in sputum: indicator of an adverse lung reaction to air pollution. *Acta Cytol*, 2001;45:958-64.
11. Baumgardner DJ, Burdick JS. An outbreak of human and canine blastomycosis. *Rev Inf Dis*, 1991;13:898-905.
12. SN Shrikhande, CA Chande, VR Shegokar, RM Powar. Pulmonary cryptosporidiosis in HIV negative, immunocompromised host. *Ind J Pathol Microbiol*, 2009;52:267-268.
13. Gary ML, Edward L, Petsonk, Kenneth DL. The construction industry. In: Susan M. Tarlo, Paul Cullinan, Benoit Nemery, editors. Occupational and Environmental Lung Diseases. Wiley Online Library, 2010:273-89.
14. Pizzichini E, Pizzichini M, Efthimiadis A, Evans S, Morris MM, Squillace D, et al. Indices of airway inflammation in induced sputum: reproducibility and validity of cell and fluid-phase measurements. *Am J Respir Crit Care Med*, 1996;154:308-17.
15. Frost JK, Gupta PK, Erozan YS, Carter D, Hollander DH, Levin ML, et al. Pulmonary cytologic alterations in toxic environmental inhalation. *Human Pathol*, 1973;4:521-536 & *Acta Cytol*, 2001; 45:958-64.
16. Belda J, Leigh R, Parameswaran K, O'Byrne PM, Sears MR, Hargreave FE. Induced sputum cell counts in healthy adults. *Am J Respir Crit Care Med*, 2000;161:475-78.
17. Alderisio M, Cenci M, Mudu P. Cytological Value of sputum workers Daily Exposed to Air Pollution. *Anticancer Res*, 2006;26:395-404.
18. Fell AK, Sikkeland LI, Svendsen MV, Kongerud J. Airway inflammation in cement production workers, 2010;67(6):395-400. Epub 2009 Oct 22.
19. Moermans C, Heinen V, Nguyen M, Henket M, Sele J, Manise M, et al. Local and systemic cellular inflammation and cytokine release in chronic obstructive pulmonary disease. *Cytokine*. 2011;29.
20. Karkhanis V, Joshi JM. All that caseate is not T.B. *Lung India* [serial online] 2007 [cited 2015 Nov 30]; 24:100-1. Available from: <http://www.lungindia.com/text.asp?2007/24/3/100/44226>
21. Matti SH, Eero T, Vesa V. Sputum cytology of asbestosis patients. *Scand J Work Environ & Health*, 1978;4:284-94.