

INFLUENCE OF INDUSTRIAL DUST EXPOSURE ON THE DEVELOPMENT OF ATROPHIC RHINITIS AMONG CEMENT FACTORY EMPLOYEES

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Abstract

Atrophic rhinitis is a chronic degenerative disease of the nasal mucosa characterized by progressive atrophy of mucosal epithelium, glandular structures, vascular components, and underlying osseous tissue accompanied by dryness, crust formation, unpleasant odor, impaired mucociliary clearance, and respiratory dysfunction. Occupational exposure to industrial dust represents one of the most important environmental risk factors contributing to chronic irritation and degenerative changes within upper respiratory pathways. Cement factory employees are continuously exposed to high concentrations of cement dust particles containing silica, calcium oxide, aluminum compounds, and various chemical irritants capable of inducing chronic inflammatory and atrophic processes within nasal tissues. Prolonged inhalation of industrial dust leads to epithelial injury, vascular compromise, oxidative stress, inflammatory infiltration, impaired local immunity, and progressive destruction of nasal mucosal structures. Clinical manifestations commonly include nasal dryness, obstruction, crusting, epistaxis, reduced olfactory sensitivity, mucosal ulceration, chronic irritation, and secondary bacterial colonization significantly affecting respiratory comfort and occupational quality of life. The present study investigates the relationship between occupational cement dust exposure and development of atrophic rhinitis among industrial workers with emphasis on pathophysiological mechanisms, occupational risk factors, clinical manifestations, diagnostic evaluation, respiratory complications, and preventive strategies. Modern diagnostic approaches including rhinoscopy, endoscopic examination, cytological analysis, microbiological evaluation, pulmonary assessment, and occupational exposure monitoring significantly improve early identification of degenerative nasal changes and facilitate individualized management. Contemporary prevention and treatment strategies increasingly incorporate environmental protection measures, respiratory safety programs, nasal hydration therapy, anti-inflammatory management, occupational monitoring, and multidisciplinary rehabilitation aimed at preserving respiratory health and preventing progression of chronic mucosal degeneration. Clinical findings demonstrate that prolonged occupational exposure to cement dust substantially increases the risk of chronic atrophic rhinitis and negatively influences respiratory function and quality of life among industrial employees.

Keywords: Atrophic rhinitis, industrial dust, cement factory workers, occupational respiratory disease, nasal mucosal atrophy, cement dust exposure, respiratory health, occupational pathology, chronic rhinitis, mucociliary dysfunction

1. Introduction

Atrophic rhinitis is a chronic progressive disorder of the nasal cavity characterized by degeneration and thinning of nasal mucosa, loss of glandular structures, vascular insufficiency, impaired mucociliary activity, and widening of nasal passages due to underlying tissue atrophy. The disease represents an important occupational and environmental health problem, particularly among individuals exposed to chronic inhalation of industrial pollutants and airborne particulate matter. Cement factory employees constitute a high-risk occupational group because of continuous exposure to large quantities of cement dust containing silica particles, alkaline compounds, heavy metals, and chemical irritants capable of causing persistent inflammatory and degenerative respiratory changes. Industrialization, inadequate environmental control, insufficient respiratory protection, prolonged occupational exposure, and poor workplace ventilation significantly increase risk of chronic respiratory diseases among industrial workers. Cement dust particles possess strong irritative and abrasive properties that directly damage respiratory epithelium and disrupt physiological defense mechanisms of upper airways. Repeated inhalation of particulate matter causes chronic inflammation, epithelial desquamation, vascular compromise, oxidative stress, mucus gland dysfunction, and progressive atrophy of nasal mucosa. Chronic inflammatory activation additionally impairs mucociliary clearance and facilitates bacterial colonization leading to recurrent infection, crust formation, foul odor, and secondary mucosal destruction. Clinical manifestations of atrophic rhinitis commonly include persistent nasal dryness, crusting, nasal obstruction, epistaxis, reduced olfactory sensation, burning sensation, headache, discomfort during respiration, and chronic irritation affecting daily occupational functioning and psychosocial well-being. Severe cases may progress to extensive mucosal ulceration, chronic sinus infection, and structural deformities of nasal tissues. Occupational respiratory diseases associated with industrial dust exposure continue to represent significant public health concerns worldwide due to increasing industrial activity and inadequate implementation of preventive occupational health measures. Modern understanding of occupational respiratory pathology emphasizes the role of chronic oxidative stress, inflammatory cytokine activation, epithelial barrier dysfunction, vascular ischemia, and impaired regenerative capacity in development of chronic degenerative airway disorders. Accurate diagnosis requires comprehensive occupational history, clinical examination, rhinoscopy, nasal endoscopy, cytological analysis, microbiological investigation, respiratory assessment, and evaluation of workplace environmental conditions. Advances in occupational medicine, pulmonology, otolaryngology, industrial hygiene, and respiratory rehabilitation have significantly improved understanding of dust-related respiratory pathology and facilitated development of preventive and therapeutic strategies. Contemporary management increasingly incorporates respiratory protection systems, workplace environmental monitoring, nasal irrigation therapy, anti-inflammatory treatment, humidification measures, occupational rehabilitation, and employee health surveillance programs aimed at reducing respiratory morbidity and improving quality of life among industrial workers. Effective prevention and management therefore require multidisciplinary collaboration between occupational physicians, otolaryngologists, pulmonologists, industrial hygienists, environmental specialists, and respiratory rehabilitation professionals. Occupational respiratory diseases remain an important global public health challenge due to rapid industrialization, increasing environmental pollution, prolonged workplace exposure to airborne contaminants, and insufficient implementation of occupational safety measures. Among industrial respiratory disorders, atrophic rhinitis represents a chronic progressive condition characterized by degeneration of nasal mucosa, reduction of glandular secretion, impaired vascularization, mucociliary dysfunction, widening of nasal passages, and gradual destruction of supporting respiratory structures. Cement factory employees constitute one of the highest-risk occupational groups because of continuous exposure to airborne cement dust particles containing silica, calcium oxide, aluminum compounds, magnesium salts, and other irritative chemical substances capable of inducing chronic inflammatory and degenerative changes within upper respiratory pathways. Repeated inhalation of fine industrial particulate matter directly damages respiratory epithelium and disrupts physiological protective mechanisms responsible for humidification, filtration, immune defense, and mucociliary transport within nasal cavities. Persistent contact between respiratory mucosa and abrasive dust particles contributes to epithelial desquamation, inflammatory mediator release, oxidative cellular injury, vascular compromise, impaired mucus production, and progressive mucosal atrophy. Chronic inflammatory activity further weakens local immune defense and promotes secondary bacterial colonization leading to recurrent infection, crust formation, unpleasant odor, and worsening

respiratory dysfunction. Clinical manifestations of occupational atrophic rhinitis commonly include nasal dryness, crust accumulation, recurrent epistaxis, impaired smell perception, mucosal irritation, headache, respiratory discomfort, and progressive decline in nasal physiological function. Severe disease progression may result in extensive mucosal ulceration, chronic sinus inflammation, structural nasal deformities, and persistent respiratory impairment significantly reducing occupational productivity and quality of life. Duration of occupational exposure, concentration of airborne industrial dust, smoking habits, inadequate workplace ventilation, and insufficient use of respiratory protective equipment substantially influence severity and progression of respiratory pathology among industrial workers. Modern scientific understanding emphasizes the important role of oxidative stress, epithelial barrier dysfunction, chronic inflammatory activation, vascular ischemia, impaired tissue regeneration, and environmental toxicity in development of chronic degenerative airway disorders. Accurate diagnosis requires comprehensive occupational history, detailed respiratory assessment, rhinoscopy, endoscopic examination, cytological investigation, pulmonary function evaluation, and environmental monitoring of workplace air quality. Advances in occupational medicine, environmental health sciences, otolaryngology, pulmonology, and respiratory rehabilitation have significantly improved understanding of industrial respiratory pathology and facilitated development of effective preventive and therapeutic strategies. Contemporary occupational healthcare increasingly emphasizes implementation of industrial ventilation systems, respiratory safety protocols, periodic medical examinations, environmental monitoring, anti-inflammatory respiratory management, humidification therapy, and worker education programs aimed at reducing occupational respiratory morbidity and preserving long-term airway function. Effective prevention and management consequently require multidisciplinary cooperation involving occupational physicians, pulmonologists, otolaryngologists, industrial hygienists, environmental specialists, and respiratory rehabilitation professionals.

2. Materials and Methods

This study was conducted using clinical, occupational, otolaryngological, and respiratory evaluation of cement factory employees exposed to industrial dust between 2020 and 2025. Comprehensive assessment included analysis of occupational history, duration of dust exposure, workplace environmental conditions, use of respiratory protective equipment, smoking habits, respiratory symptoms, and quality-of-life indicators. Physical examination focused on nasal mucosal condition, crust formation, mucosal dryness, epistaxis, olfactory disturbances, respiratory function, and associated upper airway abnormalities. Diagnostic procedures included anterior rhinoscopy, nasal endoscopy, cytological examination of nasal secretions, microbiological analysis, pulmonary function testing, radiological evaluation when indicated, and environmental monitoring of industrial dust concentration within workplace settings. Patients were categorized according to severity of atrophic rhinitis manifestations and duration of occupational exposure. Preventive and therapeutic interventions including workplace ventilation improvement, respiratory protective equipment, nasal hydration therapy, saline irrigation, anti-inflammatory medications, humidification measures, and occupational rehabilitation were comparatively analyzed to determine clinical effectiveness and respiratory outcomes.

3. Results

Clinical evaluation demonstrated that cement factory employees exposed to industrial dust most frequently presented with persistent nasal dryness, crust formation, nasal obstruction, mucosal irritation, epistaxis, burning sensation, unpleasant odor, and reduced olfactory sensitivity. Workers with prolonged occupational exposure exceeding ten years demonstrated significantly greater severity of mucosal atrophy, epithelial degeneration, crust accumulation, and respiratory discomfort compared with employees having shorter exposure duration. Rhinoscopic and endoscopic examination revealed widened nasal cavities, pale atrophic mucosa, reduced glandular secretion, crust deposition, mucosal ulceration, and impaired mucociliary activity. Cytological analysis demonstrated epithelial degeneration, inflammatory infiltration, reduced goblet cell density, and chronic inflammatory changes within nasal tissues. Environmental monitoring confirmed elevated airborne cement dust concentrations within poorly ventilated industrial areas. Pulmonary function testing demonstrated reduced respiratory efficiency and increased prevalence of chronic upper airway irritation among workers with advanced atrophic changes. Secondary bacterial colonization and

recurrent inflammatory exacerbations were more common among patients with severe mucosal degeneration. Employees reporting inconsistent use of respiratory protective equipment demonstrated significantly greater frequency of respiratory symptoms and mucosal damage. Preventive interventions involving workplace ventilation improvement, respiratory masks, nasal hydration therapy, saline irrigation, humidification measures, and anti-inflammatory treatment significantly reduced symptom severity and improved respiratory comfort. Workers participating in occupational rehabilitation and regular medical monitoring demonstrated improved nasal function, reduced inflammatory complications, and enhanced quality of life during long-term observation. Clinical evaluation demonstrated that cement factory employees exposed to industrial dust most frequently experienced persistent nasal dryness, excessive crust formation, mucosal irritation, burning sensation, recurrent epistaxis, impaired olfactory sensitivity, nasal obstruction, headache, and chronic respiratory discomfort. Workers exposed to industrial dust for prolonged periods demonstrated significantly greater severity of mucosal degeneration and respiratory dysfunction compared with employees having shorter occupational exposure. Endoscopic and rhinoscopic examinations revealed widened nasal cavities, pale atrophic mucosa, epithelial thinning, crust deposition, ulcerative lesions, impaired mucus production, and substantial reduction of mucociliary activity. Cytological analysis demonstrated epithelial cell degeneration, inflammatory infiltration, decreased goblet cell density, chronic inflammatory remodeling, and evidence of oxidative tissue injury. Environmental workplace monitoring identified elevated concentrations of airborne cement particulate matter in poorly ventilated industrial sections with insufficient dust control systems. Pulmonary function testing demonstrated reduction in respiratory efficiency and increased prevalence of chronic upper airway irritation among workers presenting advanced atrophic mucosal changes. Secondary microbial colonization and recurrent inflammatory exacerbations occurred more frequently in individuals with severe mucosal degeneration and prolonged occupational exposure. Employees demonstrating inconsistent use of respiratory protective equipment experienced significantly higher incidence of respiratory symptoms, inflammatory complications, and structural mucosal damage. Preventive interventions including improvement of industrial ventilation, respiratory mask utilization, nasal saline irrigation, humidification procedures, mucosal hydration therapy, and anti-inflammatory treatment significantly reduced symptom severity and improved respiratory comfort. Workers participating in regular occupational health monitoring and respiratory rehabilitation programs demonstrated better preservation of nasal physiological function, lower frequency of complications, and improved quality-of-life indicators during long-term observation.

4. Discussion

The findings confirm that chronic occupational exposure to industrial cement dust plays a major role in development and progression of atrophic rhinitis among cement factory employees. Persistent inhalation of alkaline particulate matter contributes to epithelial injury, chronic inflammation, oxidative stress, vascular impairment, glandular dysfunction, and progressive degeneration of nasal mucosal structures. Cement dust particles possess abrasive and cytotoxic properties capable of disrupting physiological mucosal defense mechanisms and impairing mucociliary clearance, thereby increasing susceptibility to chronic irritation and secondary infection. The study additionally demonstrates that prolonged exposure duration and inadequate respiratory protection substantially increase severity of mucosal atrophy and respiratory dysfunction. Chronic inflammatory activation involving cytokine release, oxidative stress pathways, epithelial barrier disruption, and vascular compromise appears to represent a central pathogenic mechanism in occupational atrophic rhinitis. Reduced mucosal hydration and glandular secretion further contribute to crust formation, unpleasant odor, and impaired nasal function. The findings emphasize the importance of early occupational health surveillance and implementation of preventive workplace strategies aimed at minimizing respiratory exposure to industrial pollutants. Modern occupational diagnostics significantly improve identification of early degenerative respiratory changes and facilitate timely therapeutic intervention. Nasal hydration therapy, humidification measures, saline irrigation, anti-inflammatory treatment, and environmental control remain important components of symptomatic management. However, prevention through effective industrial ventilation systems, respiratory protective equipment, reduction of airborne dust concentration, and regular medical examination remains the most effective strategy for limiting occupational respiratory morbidity. Despite advances in occupational medicine and industrial hygiene, several important challenges remain including insufficient workplace safety

compliance, prolonged environmental exposure, delayed diagnosis, recurrent inflammatory exacerbations, and progression of irreversible mucosal degeneration. Future scientific research increasingly focuses on molecular mechanisms of occupational respiratory injury, oxidative stress biomarkers, regenerative mucosal therapy, precision occupational medicine, and advanced industrial hygiene technologies aimed at improving prevention and treatment of dust-related respiratory disorders. Multidisciplinary integration of occupational medicine, otolaryngology, pulmonology, industrial hygiene, environmental health, and respiratory rehabilitation therefore remains essential for protecting respiratory health among industrial workers. The findings confirm that chronic occupational exposure to cement dust is a major contributing factor in development and progression of atrophic rhinitis among industrial workers. Continuous inhalation of fine abrasive particulate matter induces persistent respiratory irritation, epithelial destruction, inflammatory activation, oxidative stress, vascular impairment, and progressive degeneration of nasal mucosal structures. Cement dust particles possess cytotoxic and alkaline properties capable of disrupting physiological respiratory defense mechanisms and impairing mucociliary transport, thereby increasing vulnerability to chronic inflammation and secondary infection. The study additionally demonstrates that occupational exposure duration, inadequate respiratory protection, poor workplace ventilation, and elevated airborne particulate concentration substantially influence severity of respiratory pathology. Oxidative stress mechanisms involving free radical generation, inflammatory cytokine release, epithelial barrier dysfunction, and vascular compromise appear to play central roles in chronic degenerative remodeling of nasal tissues. Reduced glandular secretion and impaired mucosal hydration further contribute to crust formation, unpleasant odor, recurrent bleeding, and progressive respiratory discomfort. The findings emphasize the critical importance of early occupational surveillance and implementation of preventive industrial health strategies aimed at minimizing respiratory exposure to harmful airborne pollutants. Modern diagnostic technologies significantly improve identification of early degenerative respiratory changes and facilitate timely therapeutic intervention before development of irreversible mucosal destruction. Nasal hydration therapy, humidification procedures, saline irrigation, anti-inflammatory treatment, and environmental modification remain important supportive measures for symptomatic management. However, prevention through effective industrial ventilation systems, respiratory safety equipment, workplace environmental control, and regular medical examinations remains the most effective approach for reducing occupational respiratory morbidity. Despite advancements in occupational medicine and industrial hygiene, several important challenges persist including inadequate compliance with respiratory safety standards, delayed diagnosis, prolonged environmental exposure, recurrent inflammatory exacerbations, and progression of irreversible tissue degeneration. Future scientific research increasingly focuses on molecular mechanisms of occupational respiratory injury, oxidative stress biomarkers, regenerative respiratory therapy, precision occupational medicine, artificial intelligence-based environmental monitoring, and innovative industrial dust control technologies aimed at improving prevention and treatment of chronic occupational respiratory disorders. Integration of occupational medicine, environmental science, pulmonology, otolaryngology, respiratory rehabilitation, and industrial hygiene therefore remains essential for comprehensive protection of respiratory health among industrial workers exposed to cement dust.

5. Conclusion

Chronic occupational exposure to cement dust significantly contributes to development and progression of atrophic rhinitis among cement factory employees. Industrial particulate matter induces persistent inflammatory and degenerative changes within nasal mucosa leading to epithelial atrophy, impaired mucociliary clearance, glandular dysfunction, crust formation, and respiratory discomfort. Prolonged exposure duration and inadequate respiratory protection substantially increase severity of respiratory pathology. Early occupational monitoring, comprehensive respiratory assessment, and implementation of preventive workplace strategies significantly improve respiratory health outcomes and reduce progression of chronic mucosal degeneration. Contemporary management approaches including environmental control, respiratory protection, nasal hydration therapy, anti-inflammatory treatment, and occupational rehabilitation effectively improve nasal function and quality of life. Continued advancement in occupational medicine, industrial hygiene, respiratory pathology, and preventive healthcare will further enhance protection and management of workers exposed to industrial respiratory hazards. Chronic occupational exposure to cement dust

significantly contributes to development of atrophic rhinitis and progressive respiratory dysfunction among cement factory employees. Industrial particulate matter induces persistent inflammatory and degenerative changes within nasal mucosa leading to epithelial atrophy, impaired mucociliary clearance, glandular dysfunction, crust formation, recurrent irritation, and chronic respiratory discomfort. Prolonged occupational exposure and insufficient respiratory protection substantially increase severity of airway pathology and risk of long-term respiratory complications. Early occupational monitoring, comprehensive respiratory assessment, environmental workplace control, and implementation of preventive protective strategies significantly improve respiratory outcomes and reduce progression of degenerative mucosal disease. Contemporary management approaches including respiratory protection systems, industrial ventilation improvement, nasal hydration therapy, anti-inflammatory treatment, and occupational rehabilitation effectively preserve respiratory function and enhance quality of life among industrial workers. Continued advances in occupational medicine, respiratory pathology, industrial hygiene, and preventive healthcare will further improve protection and management of workers exposed to industrial respiratory hazards.

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