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Chronic rhinitis in glassblowers

Hronični rinitis kod stakloduvača

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Abstract

Background/Aim. Glassworkers, especially glassblowers are in close contact with a variety of chemical and physical harmful agents at their workplace. Upper aerodigestive pathway is predominantly vulnerable to these agents. Breathing of warm volatile substances and dust, and mouth touch with glassblower's pipe are the main ways for chronic respiratory mucosa inflammation. The aim of this study was to estimate effect of workplace environment in a glass manufacturer plant, as a causative factor, on the prevalence of chronic rhinitis in glassblowers. **Methods.** Studied groups, one hundred glassblowers and 100 nonglassblowers in a same factory, were examined for diagnosis of chronic rhinitis. **Results.** This investigation confirmed that chronic rhinitis prevalence among glassblowers was significantly higher than that in non-glassblowers. The duration of exposure to harmful factors was not a significant factor for chronic rhinitis development. **Conclusion.** On their workplace, glassblowers are exposed to greater influence of noxious factors, and they have statistically greater risk for getting chronic rhinitis than nonglassblowers who work in the same work environment. Glass production by glassblowing is highly significant risk factor for getting chronic rhinitis, but the exposure period is not.

Key words:
glass; occupational exposure; prevalence; rhinitis; workplace; risk assessment.

Apstrakt

Uvod/Cilj. Stakloduvači su na radnom mestu izloženi različitim fizičkim i hemijskim štetnim agensima. Sluznica gornjeg aerodigestivnog trakta (nosa, usne šupljine, ždrela i larinksa) je naročito izložena ovim faktorima. Udisanje toplog vazduha, gasova, čestica prašine i oralni kontakt sa stakloduvačkom lulom su najvažniji faktori koji mogu uzrokovati hronično zapaljenje sluznice gornjeg respiratornog trakta. Cilj ove studije je bio da ispita da li je i u kojoj meri radna okolina u fabrici stakla uzročni faktor za visoku prevalenciju hroničnog rinitisa kod stakloduvača. **Metode.** Eksperimentalna grupa se sastojala od 100 slučajno odabranih stakloduvača muškog pola, dok je kontrolnu grupu činilo 100 muškaraca, zaposlenih u istom pogonu za proizvodnju stakla, koji nisu bili stakloduvači. **Rezultati.** Ovo istraživanje je potvrdilo da je prevalencija hroničnog rinitisa kod stakloduvača bila značajno veća nego kod radnika kontrolne grupe. Dužina ekspozicije štetnim faktorima nije bila značajan faktor u nastanku hroničnog rinitisa. **Zaključak.** Stakloduvači su na radnom mestu izloženi većem uticaju štetnih faktora i imaju značajno viši rizik od dobijanja hroničnog rinitisa od radnika drugih zanimanja u istom radnom okruženju. Proizvodnja stakla je visokorizičan faktor za dobijanje hroničnog rinitisa, ali period ekspozicije štetnim agensima nije.

Ključne reči:
staklo; profesionalna izloženost; prevalenca; rinitis; radno mesto; rizik, procena.

Introduction

Glass production is an essential aspect of the economy, especially due to wide use of different glass types in human everyday life. Glassblowing is one of the main ways of glass manufacturing. Glassblower's employment is very difficult and associated with diverse serious health threats. Severe infrared emission from glass furnaces, warm gases, evaporations and dust and glassblower's pipe are the main forms of exposure to harmful agents in glassblowers.

Chronic rhinitis is nonspecific inflammation of the nasal mucosa in duration of more than 12 weeks. According to the histopathological changes of the mucosal layer, chronic rhinitis can be divided into hypertrophic and atrophic and based on main causative factors, chronic rhinitis can be divided into allergic, infective and nonallergic noninfective rhinitis[1]. Occupational rhinitis ("work-related rhinitis") could be defined as chronic inflammation of the nasal mucosa, characterized by intermittent or persistent nasal congestion, sneezing, rhinorrhea, itching, and/or hypersecretion, which

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are consequences attributable to a workplace setting, but not to factors outside the workplace [1, 2]. This form of rhinitis may be allergic, consequent to exposure to a sensitizing factors through an immunological mechanism, and nonallergic, mediated by nonimmunological mechanism [1]. The most severe form of occupational rhinitis is corrosive rhinitis, which is characterized by permanent inflammation of the nasal mucosa sometimes associated with ulceration and perforation of the nasal septum [1].

Yoruk et al. [3] have found that denim sandblasters exposed to crystalline silica had considerable upper airway complaints in addition to pulmonary ones. The findings on the upper airway of the patients were: higher rate of rhinitis and adenoid vegetation, increased pH value in the nasal secretions and increased time of mucociliary clearance.

Irritation and inflammatory responses, epithelial changes, nasal host defense effects, systemic immune response, and nasal airflow resistance changes are sinonasal responses to various inhaled chemicals. Earliest physiologic response mediated by trigeminal nerve are irritative effects, which include a nasal and eyes burning sensation, nasal congestion, sneezing, headaches, cough, and reflex apnea. The initial nonspecific nasal inflammatory responses on inhaled pollutants are dependent on irritation response via the mechanism of neurogenic inflammation [4], and later through cytotoxic damage of mucosa, which cause recruitment of inflammatory cells. Impaired mucociliary clearance due to exposure to harmful chemicals in air could result in retention of secretions and consequent infection. Immunotoxic effect to nasal mucosa exerted by many airborne chemicals and compromised phagocytic and killing ability could lead to impaired host resistance and clinical infection [5]. Epithelial changes are result of increased epithelial permeability and consequent hyperresponsiveness to inhaled stimuli. Chronic decrease in nasal mucus flow caused by constant or repeated exposures to various air pollutants has been concerned as an etiologic factor in chronic rhinitis [6].

Moreover, intensive infrared radiation and high air temperature from glass furnaces and low humidity cause irritation of nasal mucosa. These factors lead to significant increase of nasal glands secretion and vasodilatation via trigeminal reflex. Nasal mucosa becomes wet, edematous and hyperemic, that is initial stage of chronic rhinitis. Longer exposure leads to hypertrophy and finally to atrophy of nasal glands, decreasing of their secretion and blood perfusion, and dryness of nasal mucosa. Final point is generalized atrophy of whole nasal mucosa. Nasal mucosa becomes pale, dry, atrophic, while mucociliary defense considerably decreases [4–6].

A diversity of chemicals like metal oxides (aluminum, antimony, arsenic, cadmium, chromium copper, manganese, and nickel), silica, sulfur dioxide, acrolein and asbestos have important role for melting and coloring of glass. Fumes and dust that include these substances have irritant and noxious influence to upper respiratory tract, particularly to the nasal mucosa [7].

Inhalation of fumes, gases and dust and primarily blowing glassworker's pipe are essential forms of contact to harmful influences in glassblowers. Red-hot glass in furnaces and on the end of glassblower's pipe is on temperature of 1,100°C. Therefore, high temperature and different volatile substances and fumes arise from molten glass to the glassblower's mouth and other parts of upper aerodigestive pathways via blow-pipe.

In four German glass factories, Raithel et al.[8] have found significant higher air concentration of nickel. Concentration of this metal was significantly higher in glassblower's urine than in an unexposed control group, too. Correlation of nickel compounds with upper respiratory malignancies is well known (IARC, 2018)[9].

Occupational exposition to hexavalent chromium compounds is confirmed to be causative factor for paranasal sinuses, laryngeal and lung cancer, which prevalence is 15–20 times higher than in unexposed population [10].

Szmeja et al. [11] reported high incidence of the chronic inflammation of upper respiratory pathways in workers employed in glass industry. They claimed that this was probably related to silica dust exposition.

The aims of this study were to determine the prevalence of chronic rhinitis in glassblowers and nonglassblowers, to check whether or not glassblowers have significantly higher prevalence of chronic rhinitis than the control group, as well as to establish which etiologic factors have most significant influence on prevalence of chronic rhinitis in glassblowers.

Methods

The investigation was conducted in the Serbian Glass Factory, Paraćin, Serbia. One hunderd randomly selected male glassblowers made the experimental (exposed) group, while the control group was made of 100 male nonglassblowers workers from the same factory, which worked near glassblowers. All procedures were conducted in accordance with the Helsinki Declaration. All participants provided written informed consent for participating in this analytical cross sectional study.

For this study specific questionnaire was prepared, with participant's general data (age, workplace, years of employment), hazardous life-style behavior and anamnesis of earlier illness, injuries, surgery of upper aerodigestive tract and nasal related symptoms.

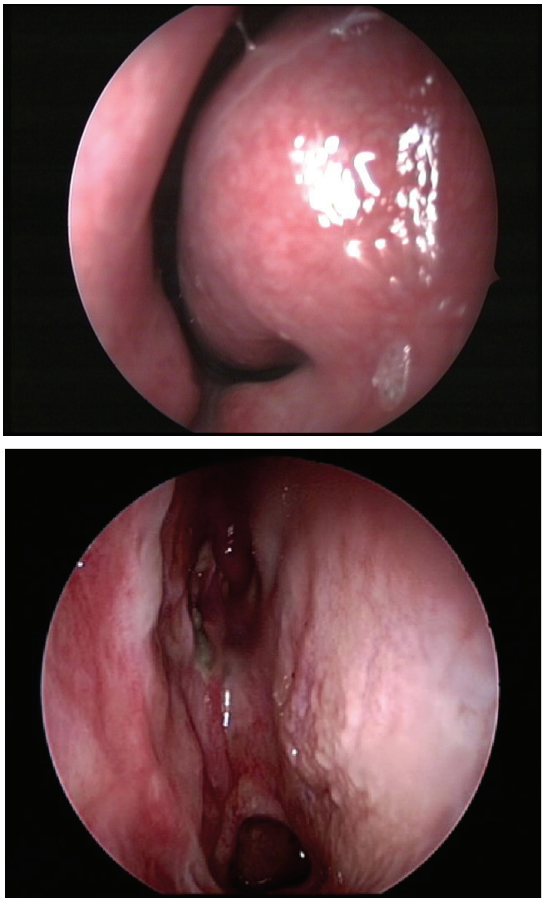
In view of smoking practice, participants were divided in the groups of current smokers and non-smokers (never smoked). In the smokers group, number of cigarettes per day was noted.

Regarding alcohol abuse, three groups according to the daily intake of alcohol were created: up to one beverage per day, drinking one to two beverages a day and serious drinkers – more than two drinks a day, based on guidelines of the National Institute on Alcohol Abuse and Alcoholism [12].

Diagnostic criteria

Only workers with clinically confirmed nonallergic, nonpolypoid and noninfectious inflammation of the nasal mucosa in duration for more than 12 weeks were considered

for this study. Main symptoms of chronic rhinitis were nasal congestion, rhinorrhea, sneezing and itching in the nose. A routine ear, nose, throat examination including anterior and posterior rhinoscopy and nasal endoscopy was performed in all participants. Endoscopical signs of nasal chronic inflammation were long lasting edema, mucosal hyperrhemia and hypertrophy, viscous nasal secretions (Figure 1A), or, rarely, atrophy and dryness of the nasal mucosa (Figure 1B), particularly in the region of the inferior turbinates. Negative X-rays of paranasal sinuses and absence of nasal polyps by endoscopy were made for differentiation from chronic rhinosinusitis with nasal polyps.



A)

B)

Fig. 1 – Endoscopic view on nasal cavity of a patient with: A) hypertrophic, and B) atrophic form of occupational rhinitis.

The diagnosis of nonallergic noninfectious rhinitis was based on exclusion criteria, i.e. the absence of clinical signs of infection and sensitization to inhalant allergens, demonstrated by skin-prick test (SPT) results or serological analysis for immunoglobulin E (IgE) [13–15].

Subjects with perennial allergic rhinitis, infectious rhinitis, non-allergic rhinitis with eosinophilia syndrome (NARES), medicamentous rhinitis, hormonal rhinitis, etc. were excluded using appropriate diagnostic methods, according to the Diagnostic Tools in Rhinology EAACI Position Paper [14]. The subjects with systemic illness, with positive anamnesis of abuse any of drugs (like cocaine etc.), long-term use of nasal decongestants, previous injuries and surgical procedures on the nasal cavity and paranasal sinuses were excluded too.

Differentiation from perennial allergic rhinitis

SPT was done in all participants with the standard set of respiratory allergens: birch, timothy, mugwort (*Artemisia vulgaris*), dog, cat, horse, mite (*Dermatophagoides farinae, Dermatophagoides pteronyssinus*), moulds (*Alternaria alternata, Aspergillus fumigatus, Cladosporium herbarum*), *Olea europaea*, *Parietaria judaica*, *Plantago lanceolata*, *Platanus acerifolia*) [14]. Saline solution (0.9% NaCl) and 1 mg/mL histamine solution were also used in SPT as negative and positive controls, respectively. SPT result was noted as positive if the width of wheal was larger than 3 mm in comparison to the negative control.

ELISA kit (Elitech Diagnostics, France) was used for measurement of total serum IgE level. The level of IgE of more than 100–150 IU/mL considered to be higher than normal [14].

All subjects with positive SPT and/or IgE level above normal were excluded from this study.

Differentiation from infectious rhinitis

Swabs for microbiological evaluation of nasal secretion were provided in all workers with clinical confirmation of chronic rhinitis. Any recognized microbial pathogen existed in more than 1,000 colony per mL was considered as the cause of infectious chronic rhinitis, and these workers were excluded too.

Differentiation from NARES

Profound nasal eosinophilia was revealed by cytology evaluation of scraped nasal mucosa in all participants. Nasal leukocyte counts were determined after fixing of the specimen on plain slide with 95% ethanol and staining with May-Grünwald-Giemsa, by light microscopy (x400) under oil immersion. Twenty percent or more eosinophils in total leukocyte count was considered to be characteristic of NARES [16], and these subjects were excluded from the study.

Statistical analysis

For presentation of numeric variables, descriptive statistics was used as mean values ± standard deviation (SD), while for categorical variables percentages were used. Student *t*-test was used for evaluation of differences in average of age and length of service between evaluated groups. Differences in smoking habits, alcohol abuse, and the prevalence of confirmed chronic rhinitis were evaluated by χ^2 test.

Binary logistic regression model was used to calculate the relative risk for the occasion of chronic rhinitis based on independent predictor variables (age, years of service, smoking, alcohol consumption and group membership). A p value of 0.05 and less was considered to be statistically significant.

For statistical analysis, we used the PASW Statistics 2018 programme.

Results

General characteristics of the investigated cohorts at the moment of the investigation demonstrated no statistically significant differences between the groups in view of the average age, duration of employment, alcohol abuse and smoking practice (Table 1).

Table 1
Main characteristics of the studied population

Parameter	Group		*p*
	exposed	control	
Age (years), mean ± SD	37.5 ± 7.9	39.6 ± 8.9	0.077†
Employment (years), mean ± SD	19.3 ± 8.2	17.5 ± 8.1	0.131†
Smoking habits, n (%)			
nonsmokers	34 (34.0)	26 (26.0)	
up to 10 cigarettes/day	10 (10.0)	12 (12.0)	0.426*
11–20 cigarettes/day	56 (56.0)	62 (62.0)	
Alcohol consumption, n (%)			
rarely or never	23 (23.0)	26 (26.0)	
moderate (1–2 drink/day)	68 (68.0)	61 (61.0)	0.731*
heavy (> 2 drink/day)	9 (9.0)	13 (13.0)	

SD – standard deviation; †Student *t*-test; *Pearson χ^2 test.

Using χ^2 test for assessment of overall chronic rhinitis prevalence in studied groups, we got result: $\chi^2 = 7.498$, DF = 1, $p = 0.006$ (Table 2). We concluded that exposed group had considerably higher prevalence of chronic rhinitis than nonexposed population.

Table 2
Chronic rhinitis prevalence in the exposed group and the control group

Group	Chronic rhinitis		Pearson χ^2	Df	*p*
	yes	no			
Exposed	78	22	1	7.498	0.006
Control	21	97			

Figure 2 presents the prevalence of chronic rhinitis for both studied groups regarding the exposure duration.

By means of binary logistic regression model, we found that only membership to exposed group – glassblowers had statistically significant contribution to the model, with relative risk of 8.387 (Table 3). That means that glassblowers have almost 8.4 times greater risk for occurrence of chronic rhinitis than the control group. Other examined predictor variables (age, years of employment, smoking and alcohol abuse) had not contribution to getting chronic rhinitis.

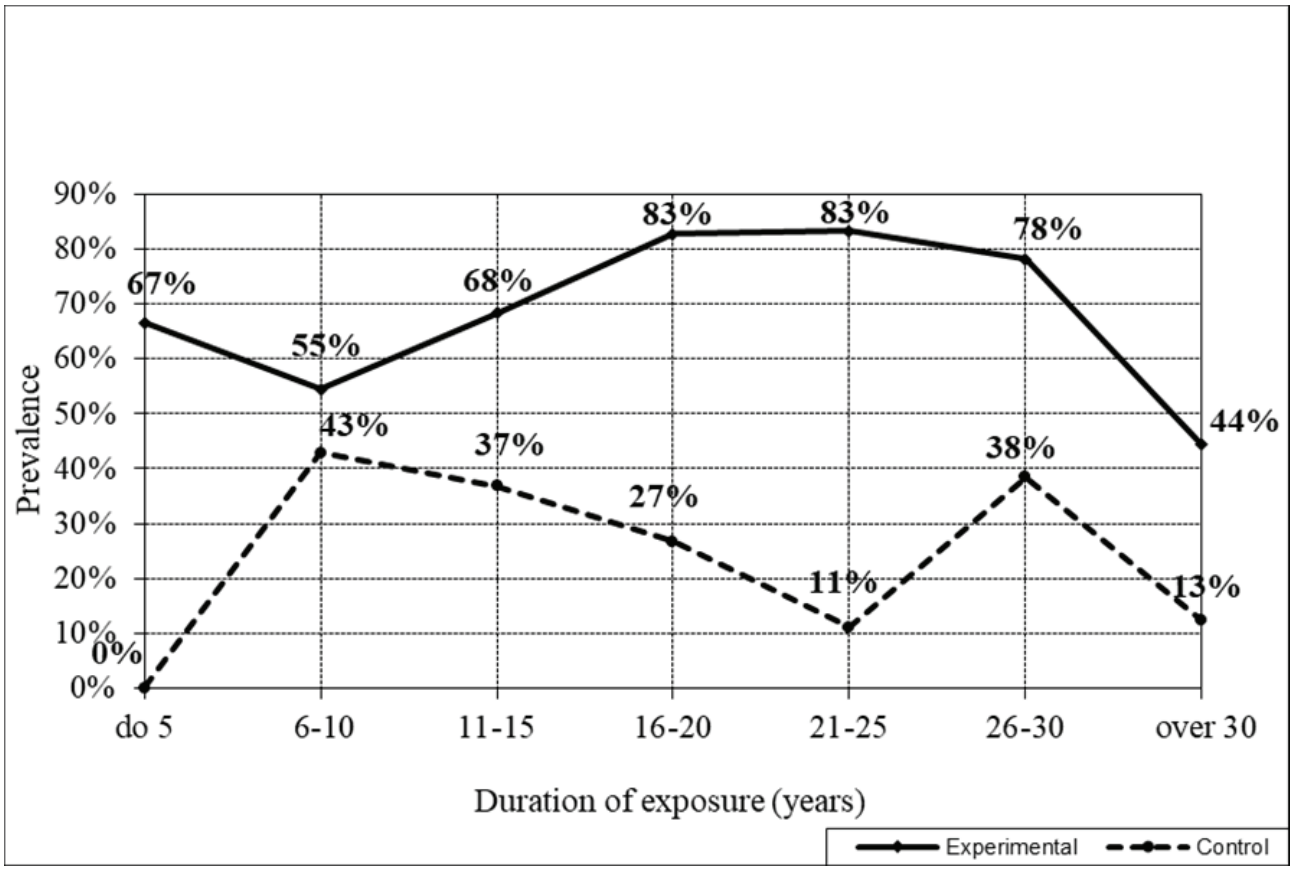


Fig. 2 – Prevalence of chronic rhinitis in the exposed (experimental) group and the control group during exposure period.

Table 3

Relative risk for occurrence of chronic rhinitis in glassblowers and control group

Predictor variables	B	S.E.	Sig.	RR*	95% CI	
					lower	upper
Group						
control				1.000		
exposed	2.127	0.357	0.000	8.387	4.163	16.897
Age	0.056	0.034	0.099	1.057	0.990	1.129
Years of employment	-0.053	0.035	0.124	0.948	0.886	1.015
Smoking	0.039	0.352	0.912	1.040	0.521	2.073
Abuse of alcohol	-0.197	0.376	0.601	0.821	0.393	1.718
Constant	-2.195	0.967	0.023	0.111		

***Relative risk (binary logistic regression); CI – confidence interval.**

Discussion

Among other roles, the nose has the protective function of the lower parts of respiratory system from the ambient harmful influences. More intensive contact of glassblowers with noxious influences could be explained by previous noted closer and more intensive contact with harmful factors in contrast with the control group. This fact could be explanation of more than 8 times higher prevalence of chronic rhinitis in the exposed than in the control group. Additionally, we found that the years of service was not a statistically significant factor for occurrence of chronic rhinitis.

Although glassblowers are exposed to several carcinogenic factors, malignant tumors of the nose and upper aerodigestive tract were not found in our investigation. Some other surveys [8, 9] have noted increased occurrence of malignancies of the nose and paranasal sinuses in glassworkers.

The curves of prevalence distribution of chronic rhinitis in studied groups of workers through years of service were interesting in shape (Figure 2).

Unexpectedly high prevalence of chronic rhinitis (67%) in the exposed group was found at the beginning of their work (0–5 years). At that time, in the control group, no one case of chronic rhinitis was diagnosed. This fact could be explained by rapid and intensive exposition of the glassblower's nasal mucosa to harmful occupational environmental factors. Nasal mucosa, at this time, was not adapted to rapidly and intensively changed microclimatic factors. These facts reveal how harmful microclimatic conditions have more significant influence on the glassblower's nasal mucosa than on that of the control group of workers.

In the second exposure period (6–10 years) prevalence of chronic rhinitis among the glassblowers decreased (55%), whereas increased within the control group (43%), but still less than in the exposed group. During years of service many glassblowers probably acquire some adaptation mechanisms to harmful influences of work ambient, and this could be the explanation for decreased prevalence of chronic rhinitis among glassblowers in later period. Mechanisms of this adaptation were not considered in this study. The control group of workers were employed near glassblowers, but they did not blow glass, so they were less exposed to harmful workplace factors. This fact could explain slower increasing of prevalence of chronic rhinitis in the control group.

After this period, we observed smooth rise of chronic rhinitis prevalence in the exposed group, while this prevalence decreased among control nonglassblowers. The difference in frequency of chronic rhinitis between examined groups increased during time too, and raised maximum in exposition interval 21–25 years of service, when prevalence of chronic rhinitis in the exposed group maximized (83%), and in the control group minimized (11%).

The interval of 26–30 years of service in both studied groups was characterized by the decrease of chronic rhinitis prevalence. The retirement of workers who had the most prominent symptoms and signs of chronic rhinitis or other diseases could explain this fact. Therefore, only workers with relatively good health status remained in manufacturing plant.

Conclusion

On their workplace, glassblowers are exposed to greater influence of noxious factors, and they have statistically greater risk for getting chronic rhinitis than nonglassblowers who work in the same glass factory.

The prevalence of chronic rhinitis increased in both groups of workers during exposure time (years of service), but difference between them was not statistically significant. Therefore, we can conclude that the glass production by glassblowing is highly significant risk factor for getting chronic rhinitis, but the exposure period is not.

We noted the decrease of chronic rhinitis prevalence among glassblowers after 5 to 10 years of service that can be explained by the possible adaptation of the laryngeal mucosa to harmful influences.

On the basis of our results, it is imperative to insist on using adequate standard protective devices on the working place, as well as an adequate ventilation of the workspace. We consider that it is necessary to include at least periodic otorhinolaryngology examination in the regular systematic examinations of glassblowers.

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