

# NASAL LAVAGE FLUID EXAMINATION IN DIAGNOSTICS OF OCCUPATIONAL ALLERGY TO CHLORAMINE

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## Abstract

**Objectives:** Chloramine T is a known sensitising agent in the occupational environment of health care workers. In cases of occupational hazards induced by this agent, a clinical history may be far from conclusive, hence appropriate provocation tests are absolutely essential. The aim of the study was to evaluate the usefulness of the nasal challenge test in diagnostics of respiratory allergy to chloramine T. **Materials and Methods:** A single-blind, placebo-controlled study was conducted in 6 subjects with chloramine T asthma and rhinitis. Two control groups comprised 7 atopic subjects with asthma and rhinitis and 6 healthy persons. All the controls had negative results of skin prick tests with chloramine T and none displayed any respiratory symptoms under exposure to the agent. A “nasal pool” technique was used to evaluate morphological and biochemical parameters (mast cell tryptase, eosinophil cationic protein, permeability index) in nasal washings before and 30 min, 4 h and 24 h after the provocation with chloramine T and placebo. **Results:** A significant increase was found in the total count and percentage of eosinophils and basophils, albumin, tryptase and eosinophil cationic protein levels in the nasal lavage fluid from patients with chloramine T respiratory allergy when compared to both control groups. Also a dual asthmatic reaction in 4 patients and an isolated late reaction in 2 cases were observed in chloramine-sensitive subjects. **Conclusions:** The results indicate the applicability of the “nasal pool” technique as a diagnostic procedure in chloramine T-induced airway allergy.

## Key words:

Occupational asthma, Chloramine T, Nasal lavage

## INTRODUCTION

Chloramine, the sodium salt of *N*-chloro-*p*-toluene sulphonamide (chloramine T) or *N*-chloro-*p*-benzene sulphonamide (chloramine B) is a water-soluble, oxida-

tive agent with antiseptic and disinfectant properties that has been used in cleaning of floors and other surfaces in dairies, breweries, kitchens and hospitals (in operating theatres and in treating infected wounds) [1].

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Hypersensitivity reactions to chloramine T (CLT) have been known for a long time. Such reactions were reported as early as 1935 in a Swedish medical journal [2]. In 1945 Feinberg and Watrous [3] reported 14 cases of occupational asthma and rhinitis among workers exposed to the substance. They also observed wheal-and-flare reactions to CLT. In 1968, Hard and Bengtson [4] reported six cases of allergy to CLT among 40 workers at the factory hall of a paper pulp mill. Of this number, five workers experienced both asthma and rhinitis, while one worker showed rhinitis as the only symptom. All six workers were found to have positive skin "scratch" tests to CLT. In 1979, Bourne et al. [5] described seven brewery workers with CLT-induced asthma and positive wheal-and-flare reactions to the substance. Two years later, Dijkman et al. [6] reported five cases with respiratory symptoms due to CLT hypersensitivity, confirmed by positive results of skin prick tests (SPT) in four patients and positive results of inhalation test in three others.

One decade later, Blomqvist et al. [7] described the presence of specific anti-CLT IgE antibodies demonstrated by radio-allergosorbent test (RAST) in subjects occupationally exposed to this agent.

Studies aimed at evaluating changes in nasal lavage fluid after provocation with chloramine have not as yet been reported. Some authors performed provocation tests with CLT, obtaining only data on clinical symptoms and spirometric changes. In many cases, these methods are inadequate to differentiate between the non-specific (irritant) and specific (allergic) character of the reaction.

The aim of this study was to assess the applicability of the "nasal pool" technique to the diagnosis of CLT respiratory allergy and to evaluate cellular response, changes in the protein level and the biomarkers of allergic inflammation like eosinophil cationic protein (ECP) and mast cell tryptase (MCT) concentrations in nasal lavage fluid.

## MATERIALS AND METHODS

### Subjects

Three groups of non-smoking subjects were enrolled for the study.

Group A comprised 6 health care workers (5 women and 1 men) – 4 nurses and 2 ward attendants (mean age,  $43.7 \pm 7.9$  years) with positive history of respiratory symptoms (asthma and rhinitis) related to CLT exposure and positive skin prick test to CLT. In four of them, sensitisation to CLT was confirmed by the presence of specific anti-chloramine IgE. In addition, specific anti-latex IgE were found in two nurses.

Group B consisted of 7 atopic patients (5 women, 2 men; mean age,  $37.5 \pm 6.1$  years) with perennial respiratory symptoms, asthma and rhinitis, and a positive SPT result with at least one common aeroallergen.

Group C was composed of 6 healthy women (mean age,  $38.5 \pm 7.5$  years).

All the controls (groups B and C) had negative results of SPT and CAP RAST to CLT.

The study participants did not receive any systemic or local medication, except for inhaled short acting  $\beta_2$ -agonists, 14 days prior to the study. The nurses were not exposed to chloramine and natural rubber latex (NRL) for at least 14 days preceding the study. All the patients were hospitalized in the NRL- and chloramine-free environment during the course of the study.

### Study protocol

The study was designed as a biphasic, single blind trial. At phase I, the subjects were challenged with 0.9% saline as placebo. At least 7 days later the allergen challenge with 2% CLT was performed. The testing was done by painting the solution onto a 2 m<sup>2</sup> piece of cardboard in a challenge chamber for 15 min.

The Regional Medical Ethics Committee approved the study protocol and all the participants gave written informed consent prior to the trial.

### Skin prick tests

SPT were performed on the volar part of the forearm with a standard battery of common allergens, including tree and grass pollen, *Dermatophagoides pteronyssinus*, moulds, feathers (Allergopharma, Germany) and with chloramine

T (0.1, 1, 1.0 mg/ml). Allergen diluent and histamine dihydrochloride solution at 1 mg/ml were negative and positive controls, respectively. All the tested sites were examined after 15 min; the wheal of 3 mm above negative control was considered positive.

#### **Total and specific IgE determination**

The evaluation of total serum IgE, NRL RAST (Phadezym k82) and CLT RAST (Phadezym k85) was performed using Uni-CAP system (Pharmacia Diagnostics, Sweden). The results were expressed quantitatively in kilo units per liter and considered positive at values higher than 0.35 kU/l.

#### **Nasal lavage and challenge procedure**

All the procedures were performed like in the "nasal pool" method [8]. Before the provocation, each nostril was washed 10 times with 6 ml saline solution using the "nasal pool" device, a 10 ml syringe closely fitting the nostril. Saline in the volume of 6 ml was inserted into the nasal cavity for 5 min and then recovered. Nasal washings were collected immediately before the provocation and 30 min, 4 h and 24 h afterwards. All washings were always performed on the same side of the nasal cavity.

#### **Symptom score**

The number of sneezes and the degree of mucosal oedema, rhinorrhea and itching were evaluated for the following time periods: 30 min before the challenge, 0–30 min after the challenge, and one hour before all next control points. Total symptom score (SS) ranged from 0 to 8 and represented the sum of scores for sneezing (0 sneezes – 0 points, 1–4 sneezes – 1 point, >4 sneezes – 2 points), rhinorrhea (none – 0 points, mild – 1 point, abundant – 2 points), mucosal oedema (none – 0 points, mild – 1 point, nasal block – 2 points) and itching (none – 0 points, itching of the nose or throat – 1 point, itching of the nose and throat – 2 points).

#### **Nasal washings processing**

Nasal washings were centrifuged (for 10 min at 1000 rpm) to isolate the cell pellet and the supernatant. The obtained

sediment was washed with sterile phosphate-buffered saline (Dulbecco, Sigma, USA) and 0.1% human serum albumin (HSA, Behringwerke A.G., Germany) and then suspended in 1 ml buffer with HSA. The cells were stained using: (a) Turk method for leukocytes, (b) Dunger method for eosinophils and (c) 0.06% toluidine blue in 30% ethanol for basophils (metachromatic cells). The cells were counted in a Fuchs-Rosenthal chamber. The number of cells in 1 ml of the recovered fluid was determined.

The samples were further centrifuged at 2000 rpm for 5 min, transferred onto a slide, and air-dried. The slides were stained following Giemsa method. On each slide, the first 200 cells were classified into epithelial cells, eosinophils, neutrophils, basophils and mononuclear cells – a category including lymphocytes and monocytes.

The total protein content in the supernatant was evaluated following the method of Lowry [9]. Albumin concentration was measured using the "rocket" method of Laurell [10] (the assay ranged between 20 and 200 µg/ml). The permeability index, i.e., albumin to total protein ratio was also calculated.

#### **Mediator levels**

Nasal ECP and MCT concentrations were measured by radioimmunoassay (RIA kits, Pharmacia Diagnostics, Sweden) according to the manufacturer protocol. The samples for these assays were collected before and 30 min, 4 h and 24 h after the provocation test.

#### **Pulmonary function**

Bronchial response was measured by monitoring Forced Expiratory Volume in one second (FEV<sub>1</sub>) using a spirometer (Vicatest 2A, Mijnhardt, Holland), before and 5 min, 30 min, 1 h, 2 h, 4 h, 5 h and 24 h after the provocation. Histamine challenge was performed according to Cockcroft [11].

#### **Statistical analysis**

We analysed the results of CLT and placebo provocations separately. The data were analyzed with a repeated measures ANOVA. For all the tests the significance level  $\alpha = 0.05$  was accepted.

## RESULTS

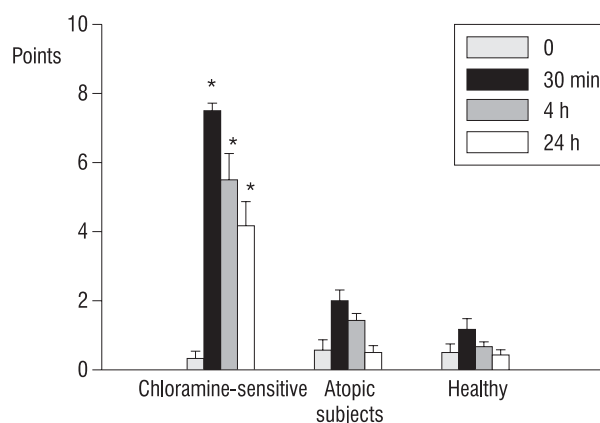
The results of SPT and evaluation of total IgE level are presented in Table 1. Only in one subject of group A, positive SPT to common allergens were found.

The inhalation challenge with CLT induced isolated late asthmatic reaction in 2 patients sensitized to CLT and dual reaction in 4 others, with a significant decrease ( $\geq 20\%$ ) in FEV<sub>1</sub> and increase in bronchial hyperreactivity (Table 2).

Placebo provocation in the subjects of all groups, as well as CLT challenge in controls, did not induce significant changes in FEV<sub>1</sub>. Provocation with placebo induced neither significant changes in symptom score nor in the biochemical and cellular composition of nasal washings (Figs. 1–6). The changes observed after CLT challenge are described below.

### Symptom score

Allergen challenge induced very severe symptoms of rhinitis in all subjects of group A (score  $7.50 \pm 0.55$ ). The reaction to CLT in subjects of groups B and C was slight



**Fig. 1.** Chloramine – induced changes in symptom score in subjects participating in the study.

(scores  $1.17 \pm 0.75$  and  $1.0 \pm 0.82$ , respectively). Statistical analysis revealed significant differences between groups 30 min ( $F_{2,16} = 138.68$ ,  $p < 0.001$ ), 4 h ( $F_{2,16} = 32.00$ ,  $p < 0.001$ ) and 24 h after the provocation ( $F_{2,16} = 24.99$ ,  $p < 0.001$ ). Furthermore, within-groups comparisons revealed significant differences in group A ( $F_{1,16} = 75.27$ ,  $p < 0.001$ ) and C ( $F_{1,16} = 5.27$ ,  $p = 0.035$  (Fig.1).

**Table 1.** Results of skin prick test to common and occupational allergens, evaluation of total and specific IgE in subjects participating in the study

Group	Number of subjects	Positive SPT to common allergens (N)	Positive SPT with CLT (N)	Total IgE (ku/l) Mean $\pm$ SD	Positive NRL-RAST (N)	Positive CLT-RAST (N)
A	6	1	6	$141 \pm 58$	2	4
B	7	7	0	$105 \pm 154$	0	0
C	6	0	0	$52 \pm 21$	0	0

SPT – skin prick test.

CLT – chloramine T.

NRL – natural rubber latex.

RAST – radio-allergosorbent test.

SD – standard deviation.

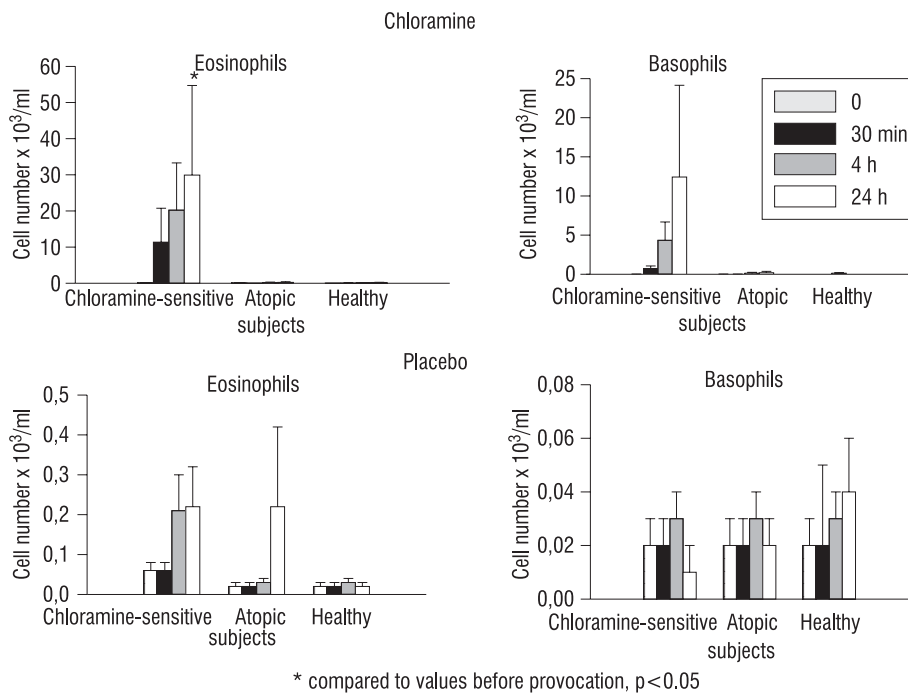
**Table 2.** Type of asthmatic response and changes in non-specific bronchial hyperreactivity in patients sensitised to CLT after specific inhalation challenge test with CLT (N = 6)

No	Asthmatic response during SIPT with CLT	PC20H before SIPT with CLT	PC20H after SIPT with CLT
1	Dual	6,4	1,0
2	Dual	4,8	2,4
3	Dual	6,0	0,44
4	Dual	3,2	0,12
5	Late	1,68	0,12
6	Late	4,8	1,4

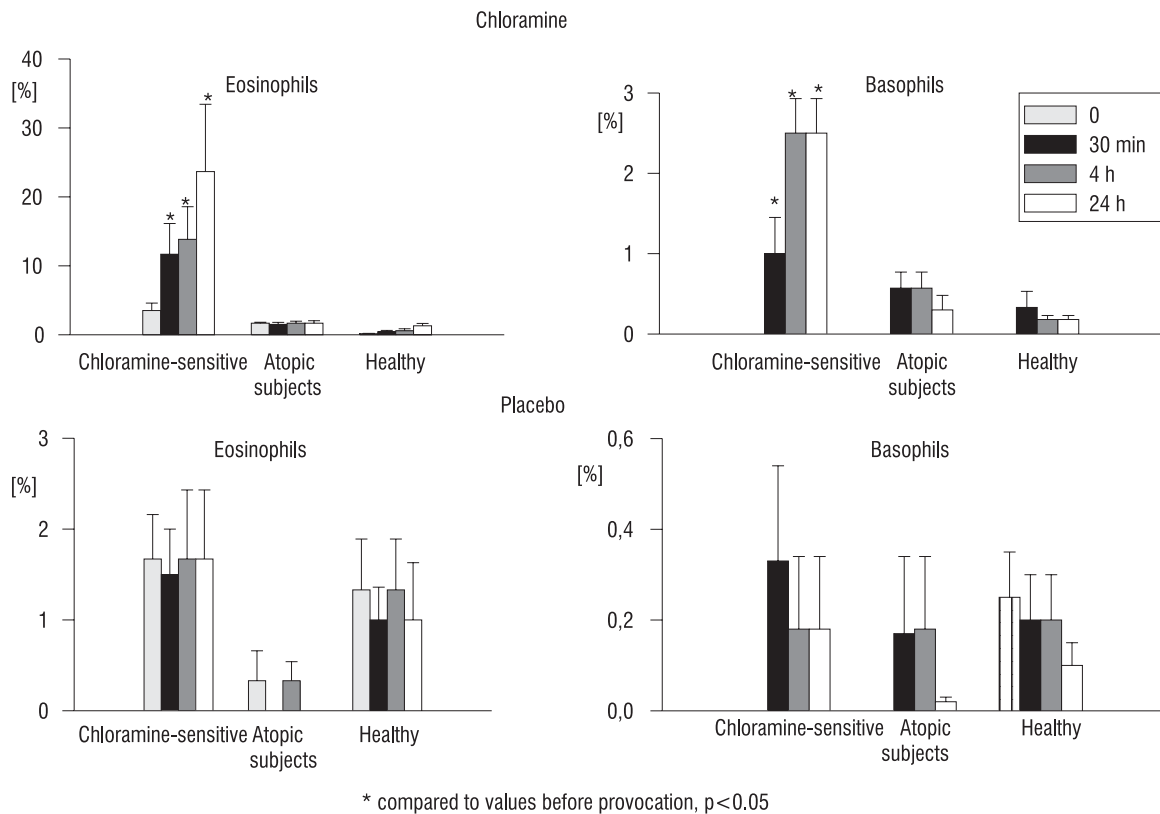
SIPT – specific inhalatory provocation test.

CLT – chloramine T.

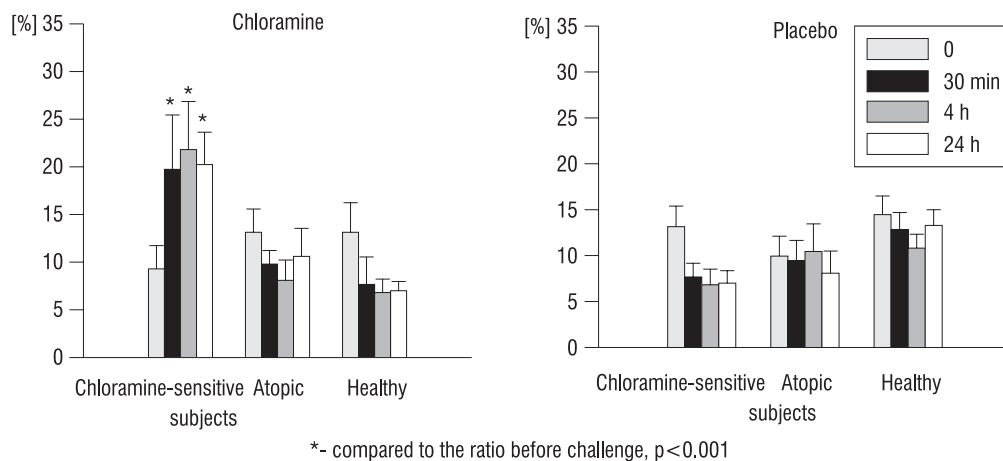
PC20H – provocation concentration of histamine producing a 20% fall in FEV<sub>1</sub>.



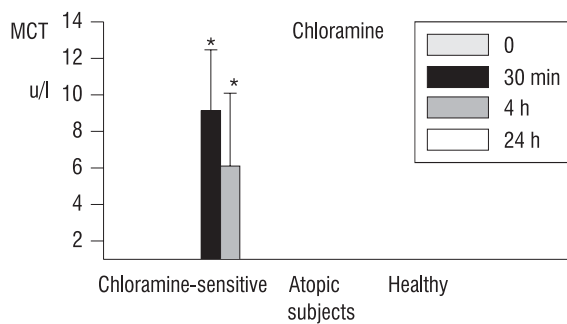
**Fig. 2.** Changes in total count of eosinophils and basophils in nasal lavage fluid induced by challenge with chloramine T or placebo.



**Fig. 3.** Changes in the proportion of eosinophils and basophils in nasal lavage fluid induced by challenge with chloramine T or placebo.



**Fig. 4.** Changes in permeability index (albumin to total protein ratio) in nasal lavage fluid induced by challenge with chloramine T or placebo.



**Fig. 5.** Changes in the level of mast cell tryptase (MCT) in nasal lavage fluid induced by challenge with chloramine T or placebo.

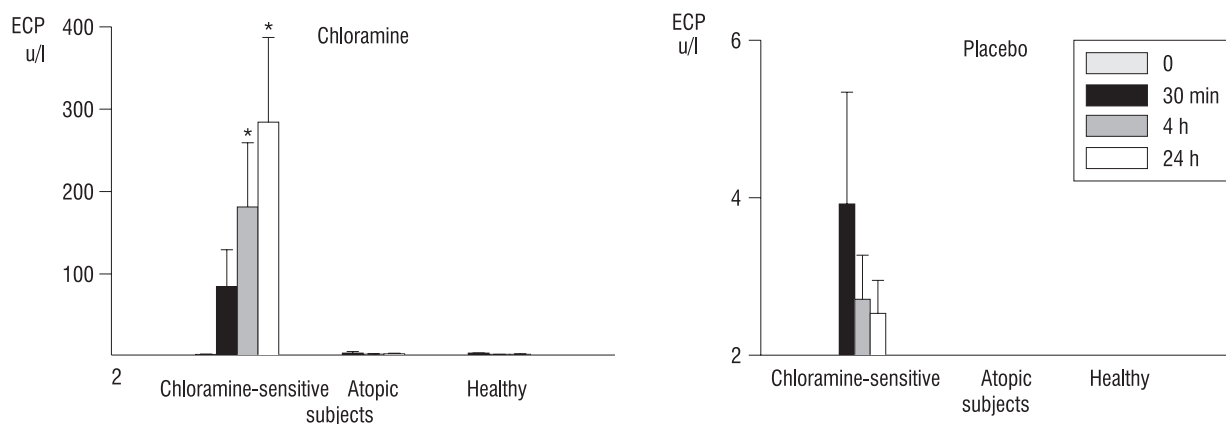
**Cellular findings**

The provocation with CLT resulted in an increase in the total number of leukocytes in nasal washings from all the groups at all time points. Within-groups comparisons revealed significant differences in total leukocyte count only in group A ( $F_{1,16} = 4.59, p = 0.048$ ).

An analysis of the total eosinophil count revealed that the groups differed significantly 24 h after the provocation ( $F_{2,16} = 6.13, p = 0.011$ ) (Fig. 2).

An analysis of the proportion of eosinophils revealed significant differences between the groups 30 min ( $F_{2,16} = 5.11, p = 0.019$ ), 4 h ( $F_{2,16} = 7.72, p = 0.004$ ) and 24 h after CLT challenge ( $F_{2,16} = 4.38, p = 0.030$ ). Within-groups comparisons revealed significant differences only in group A after CLT provocation ( $F_{1,16} = 8.86, p = 0.009$ ) (Fig. 3). CLT provocation increased total count of basophils. It was higher in group A than in groups B and C. In the former group, it was higher 24 h after than before the challenge but the differences did not reach the level of significance (Fig. 2).

An analysis of the proportion of basophils revealed significant differences between the groups 4 h ( $F_{2,16} = 18.74,$



**Fig. 6.** Changes in the level of eosinophil cationic protein (ECP) in nasal lavage fluid induced by challenge with chloramine T or placebo.

$p < 0.001$ ) and 24 h ( $F_{2,16} = 10.69$ ,  $p = 0.001$ ) after challenge with CLT, but within-groups comparisons revealed significant differences only in group A ( $F_{1,16} = 24.02$ ,  $p < 0.001$ ) (Fig. 3).

### Permeability index

Only CLT provocation caused significant changes in permeability index. Significant differences between the groups were observed 30 min ( $F_{2,16} = 3.57$ ,  $p = 0.05$ ), 4 h ( $F_{2,16} = 6.48$ ,  $p = 0.009$ ) and 24 h ( $F_{2,16} = 5.89$ ,  $p = 0.012$ ) after the challenge. Moreover, significant differences between permeability indices at successive time points after CLT challenge could be observed only in group A ( $F_{1,16} = 6.17$ ,  $p = 0.024$ ) (Fig. 4).

### Mediator levels

The analysis of MCT levels revealed significant differences between groups 30 min after the provocation with CLT ( $F_{2,16} = 6.63$ ,  $p = 0.008$ ). Within-groups comparisons revealed statistically significant differences only in group A ( $F_{1,16} = 10.73$ ,  $p = 0.001$ ) (Fig. 5).

The analysis of ECP concentration revealed significant differences between groups 4 h ( $F_{2,16} = 5.76$ ,  $p = 0.013$ ) and 24 h ( $F_{2,16} = 8.19$ ,  $p = 0.004$ ) after CLT challenge. Within-groups comparisons showed significant differences only in group A ( $F_{1,16} = 22.64$ ,  $p < 0.001$ ) (Fig. 6).

The results of the score evaluation and of the cellular and biochemical count of nasal washings, expressed as means  $\pm$  standard deviations, are presented in Table 3.

**Table 3.** The results of the evaluation of symptom score and the cellular and biochemical analysis of nasal washings

Evaluated parameter	Study group	Time			
		0	30 min	4 h	24 h
Symptom score	A	0.33 $\pm$ 0.52	7.50 $\pm$ 0.55	5.50 $\pm$ 1.87	4.17 $\pm$ 1.72
	B	0.57 $\pm$ 0.79	2.00 $\pm$ 0.82	1.43 $\pm$ 0.53	0.50 $\pm$ 0.55
	C	0.50 $\pm$ 0.55	1.17 $\pm$ 0.75	0.67 $\pm$ 0.52	0.43 $\pm$ 0.53
Leukocytes	A	2.93 $\pm$ 1.88	20.48 $\pm$ 19.49	30.67 $\pm$ 32.51	60.82 $\pm$ 97.59
	B	4.36 $\pm$ 6.49	12.57 $\pm$ 15.02	22.47 $\pm$ 23.46	19.01 $\pm$ 12.98
	C	1.68 $\pm$ 1.02	3.03 $\pm$ 3.66	5.58 $\pm$ 3.97	8.60 $\pm$ 7.61
Total eosinophil count	A	$\pm$ 0.01	11.35 $\pm$ 22.99	20.22 $\pm$ 32.03	29.93 $\pm$ 60.71
	B	0.06 $\pm$ 0.05	0.06 $\pm$ 0.05	0.21 $\pm$ 0.21	0.22 $\pm$ 0.25
	C	0.01 $\pm$ 0.01	0.02 $\pm$ 0.01	0.12 $\pm$ 0.30	0.19 $\pm$ 0.45
Proportion of eosinophils	A	3.50 $\pm$ 4.51	11.68 $\pm$ 12.16	13.83 $\pm$ 11.62	23.67 $\pm$ 27.12
	B	1.67 $\pm$ 1.21	1.50 $\pm$ 1.22	1.67 $\pm$ 1.86	1.67 $\pm$ 1.86
	C	0.14 $\pm$ 0.38	0.43 $\pm$ 0.79	0.57 $\pm$ 0.79	1.29 $\pm$ 0.95
Total basophil count	A	$\pm$ 0.01	0.68 $\pm$ 0.92	4.33 $\pm$ 5.75	12.42 $\pm$ 28.72
	B	0.02 $\pm$ 0.01	0.02 $\pm$ 0.01	0.03 $\pm$ 0.01	0.01 $\pm$ 0.01
	C	0.02 $\pm$ 0.01	0.02 $\pm$ 0.01	0.13 $\pm$ 0.29	0.19 $\pm$ 0.45
Proportion of basophils	A	$\pm$ 0.01	$\pm$ 1.09	2.50 $\pm$ 1.05	2.50 $\pm$ 1.64
	B	0.15 $\pm$ 0.37	0.57 $\pm$ 0.79	0.57 $\pm$ 0.53	0.30 $\pm$ 0.48
	C	0.01 $\pm$ 0.00	0.33 $\pm$ 0.52	0.18 $\pm$ 0.40	0.18 $\pm$ 0.40
Permeability index	A	9.30 $\pm$ 5.99	19.73 $\pm$ 13.98	21.80 $\pm$ 12.37	20.23 $\pm$ 8.34
	B	13.15 $\pm$ 5.52	9.80 $\pm$ 3.77	8.09 $\pm$ 5.63	10.60 $\pm$ 7.80
	C	13.13 $\pm$ 6.45	7.67 $\pm$ 3.64	6.82 $\pm$ 4.19	7.00 $\pm$ 3.33
MCT	A	0.92 $\pm$ 0.01	9.13 $\pm$ 8.19	6.10 $\pm$ 9.76	0.90 $\pm$ 0.01
	B	0.90 $\pm$ 0.01	0.90 $\pm$ 0.01	0.90 $\pm$ 0.00	0.90 $\pm$ 0.00
	C	0.91 $\pm$ 0.01	0.91 $\pm$ 0.02	0.90 $\pm$ 0.00	0.90 $\pm$ 0.00
ECP	A	2.38 $\pm$ 1.15	84.75 $\pm$ 109.32	181.32 $\pm$ 190.94	284.23 $\pm$ 251.96
	B	1.94 $\pm$ 0.07	3.92 $\pm$ 3.48	2.71 $\pm$ 1.36	3.07 $\pm$ 1.96
	C	1.91 $\pm$ 0.00	3.93 $\pm$ 5.32	2.47 $\pm$ 1.33	2.53 $\pm$ 1.02

Data expressed as means  $\pm$  SD.  
ECP – eosinophil cationic protein.  
MCT – mast cell tryptase.

## DISCUSSION

In cases of exposure to occupational allergens, a clinical history may be far from conclusive, hence appropriate provocation testing is absolutely essential. A specific challenge test is used to confirm the relationship between occupational agent and these symptoms and to reproduce the temporal relationship between exposure and the onset of symptoms. We adopted the "nasal pool" method as more useful and safer than bronchial lavage fluid examination during the specific challenge [12].

Although it is not a standardized method and the commercial reagents for it are not available, the skin testing performed with chloramine seems to be accurate for screening and diagnosing CLT allergy and thus we consider this technique a suitable reference method. Moreover, specific anti-chloramine IgE antibodies were found in the majority of CLT-sensitized subjects. The relevance of the laboratory findings and clinical symptoms was confirmed by a biphasic or isolated late reaction.

When diagnosing the airway sensitisation induced by disinfectants, it may be difficult to differentiate between allergic and irritant reactions. Therefore, the bronchial provocation test combined with the lavage technique and subsequent morphological, biochemical and mediators analyses of nasal washings appears to be a highly objective method. Nevertheless, it should be stressed that the combination of work-related symptoms, positive histamine inhalation challenge and positive skin prick responses to CLT allows to identify patients with occupational asthma and rhinitis induced by CLT. It is then suggested to use these three elements as a substitute for specific inhalation challenge. However, in cases of patients who claim for compensation, but their history of work-related symptoms is not reliable, more objective methods can be needed.

Atopy is a well-documented risk factor for the development of allergy to high molecular weight allergens, whereas its role as a risk factor for chloramine allergy is still controversial [7]. In our study, of the 6 patients with occupational airway allergy to chloramine only 1 presented positive SPT to common inhalant allergens. It seems that sensitization to chloramine occurs mainly in non-atopic individuals.

We also found positive NRL RAST results in two CLT-sensitized subjects. Both exposures to natural latex and chloramine T lead to nasal, pulmonary and skin symptoms, hence the similarity of clinical symptoms induced by these agents [13]. One should consider the possibility that the health care workers may be hypersensitive to more than one agent in their work environment. Even if sensitivity to chloramine is known, appropriate laboratory tests for latex allergy should always be performed, as it is extremely important for a latex-sensitive subject to be aware of his/her reactivity and to avoid that allergen [14].

Our study confirms previous data on cell influx and an increase in albumin/protein ratio after specific challenge [15–17]. We also observed a prolonged, significant influx of eosinophils and basophils in nasal lavage in patients with occupational allergy after allergen provocation. The persistence of the influx of these cells up to 24 h after the challenge suggests their involvement in an active inflammatory process. Eosinophils are obvious participants and play the role of effectors in promoting the pathogenesis of allergic diseases [17–20].

Basophils also seem to be of importance during the late reaction phase. Some studies indicate that the basophil function may correlate with that of asthma markers [18,19]. Our data support the concept that basophils are recruited to the airway after allergen challenge and release mediators during the late phase of allergic reaction. Previous studies have revealed that a prolonged increase in the albumin/protein ratio as an index of mucosal permeability, is also specific for the allergic response [16,17,21]. In the present study we also demonstrated an increased vascular permeability in nasal washings of patients with occupational airway allergy after specific challenge.

An increase in the number of granulocytes and the total protein level has been observed in the irritant type reaction. However, this was rather brief and did not affect the relative number of eosinophils, basophils and permeability index [22].

For high molecular weight allergens, it has been proposed to consider the nasal challenge test positive when the increase in eosinophils proportion and the permeability index persisted for up to 24 h after the provocation and



when a two-fold increase in these parameters was observed; the cut off point for the test was estimated at least at 5% of eosinophils [15]. All our CLT-sensitised patients fulfill the aforesaid criteria.

Allergen challenge triggers an activation of mast cells and eosinophils with a pronounced increase in the concentrations of their biomarkers – MCT and ECP [23,24]. MCT, a tetrameric neural protease is preferentially found in mast cell secretory granules; in basophil, its levels are about 0.2–0.4% of those found in mast cells [25]. Studies of mediator release during nasal allergen challenge indicate that the increase in tryptase concentration is the most specific marker of mast cell activation during the immediate allergic response [26]. The increased ECP levels were found in the late phase of allergic reactions, both in broncho-alveolar lavage and nasal fluid [23]. The observed increase in the MCT and ECP levels was found in all CLT-sensitised subjects, but not in controls.

Although chloramine is a chemical of low molecular weight, it is rather similar to high molecular weight allergens. First, there is a very good correlation between clinical symptoms and specific IgE antibodies found in the serum and skin. Second, the character of CLT-induced, cellular and biochemical changes in nasal lavage fluid is similar to that caused by high molecular weight allergens. Although allergic rhinitis is a typical, always present symptom of CLT respiratory allergy, it seems to be disregarded when diagnosing some subjects with isolated rhinitis. Third, it should always be considered that clinical picture of chloramine sensitisation is very similar to latex allergy, therefore health care workers with a history of work-related asthma, rhinitis and urticaria, should be thoroughly diagnosed with both allergens.

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