

# Correlation between dyslipidemia and severity of allergic rhinitis

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Received 30 November 2017

Accepted 14 January 2018

The Egyptian Journal of Otolaryngology

2018, 34:111–115

## Background

Allergic rhinitis is a common problem affecting between 20 and 25% of the population lowering the quality of life (QOL) more than any other disease. Dyslipidemia is known to impact potently the development of atopy as it promotes proatopic Th2 immunity and allergic inflammation.

## Objective

The aim was to test the correlation between severity of allergic rhinitis and dyslipidemia.

## Materials and methods

A comparative study carried out on 350 allergic rhinitis patients were subjected to full serum lipid assays, visual analog scale assessing their nasal symptoms, and QOL assessment using a seven-point scale.

## Results

Patients were divided into two groups (according to their lipid profile): abnormal dyslipidemia group (33%) and normal lipid profile group (67%).

The abnormal dyslipidemia group showed a more intense allergic rhinitis symptoms compared with the normal lipid profile with poor QOL score (1.97).

## Conclusion

Dyslipidemia might play an important role in increasing the severity of allergic rhinitis symptoms with impaired patients' QOL; therefore, its control could achieve better treatment outcomes.

## Keywords:

allergy, lipid, quality of life, rhinitis

Egypt J Otolaryngol 34:111–115

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1012-5574

## Introduction

Allergic rhinitis is an inflammation of the nasal mucous membrane caused by an immunoglobulin E (IgE)-mediated reaction to one or more allergens, clinically manifested as an adverse immune response after repeated contact with usually harmless substances such as pollens, mold spores, animal dander, dust mites, foods, and stinging insects affecting between 20 and 25% of the population [1].

Although allergic rhinitis is not a life-threatening disease, its burden and economic costs had significantly affected quality of life (QOL) more than any other disease [2].

Allergic rhinitis is type I hypersensitivity characterized by sneezing, nasal congestion, nasal itching, and rhinorrhea due to immediately released mediators including histamine, tryptase, chymase, kinins, heparin, leukotrienes, and prostaglandin D2 [3,4].

Dyslipidemia, defined as increased serum lipids including triglycerides (TGs), cholesterol, and/or fat phospholipids, is usually noticed with high prevalence

in the developed countries due to bad dietary habits and lifestyle [5].

Dyslipidemia is known to impact potently the development of atopy by promoting proatopic Th2 immunity and allergic inflammation [6,7].

In addition, cholesterol enhances latex-specific IgE and Th2 cytokine production by mononuclear cells of patients with atopy [6].

Some authors have investigated the possible link between metabolic syndrome such as dyslipidemia either high levels of apoB lipoproteins and TGs, and/or low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) with the development of allergic conditions especially bronchial asthma. They mentioned that the blood lipid levels are associated with the development of asthma and sensitization to aeroallergens. The team

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suggests that asthma and allergies share certain features such as dyslipidemia with other chronic inflammatory disorders [8].

The aim of our study was to test the correlation between severity of allergic rhinitis and dyslipidemia.

### Materials and methods

A comparative study was conducted in Suez Canal University, Ismailia, Egypt from June 2015 to April 2017. The local ethics committee of the Faculty of Medicine approved the study protocol and a written consent was obtained from all patients.

The patients were in the range of 40–70 years age group known to have allergic rhinitis with the inclusion criterion of allergic rhinitis (nasal congestion and obstruction, watery rhinorrhea, postnasal drip, sneezing, itchy nose, and watery eyes). Patients with acute illness like high-grade fever and first, 2 weeks following surgery were excluded from the study to obtain a pure picture of relationship between allergic rhinitis and serum lipids. Patients with diabetes mellitus, hypothyroidism, and those receiving lipid-altering drugs were excluded.

All patients were subjected to a structured and validated, designed questionnaire assessing their nasal symptoms. A visual analog scale was used to assess subjective symptoms, with 0 indicating no symptoms and 10 indicating severe and/or constant symptoms for data collection [9]. This questionnaire was in the Arabic language and was filled in by the patients themselves with the help from the questionnaire administrators. The tool was validated by including the inputs from three experts in the subject area.

Also, sociodemographic history and detailed present, past, family clinical history, and drug history were taken. General physical examinations were performed. The anterior rhinoscopic and rigid nasal endoscopic examinations (the latter using a 4 mm diameter, 0°, Hopkins II endoscope; Karl Storz, Tuttlingen, Germany) were performed with the sinus computed tomography scan if needed. Skin prick test was done to all patients to exclude any negative data from our study as a negative skin prick test was considered as one of the exclusion criteria.

The blood samples were drawn from all the patients after 10–12 h of fasting. The patients were asked to have a light, fat-free diet on the day before the sampling. The venipuncture was done in the cubital fossa and about

10 ml of blood was drawn using a perfectly dry and sterile syringe and blood was transferred to a vacutainer. Care was taken to avoid hemolysis and within 2 h of collection, serum was separated by centrifugation at 5000 rpm for 10 min. The serum samples were analyzed on the same day. For full lipid profile such as fasting lipid profile, total cholesterol (TC), HDL, LDL, very low-density lipoprotein, and TGs using the fasting serum, samples were collected for estimation of TC by cholesterol oxidase method with a reference range of 150–200 mg/dl, TGs by lipase/lipase and glycerol dehydrogenase method (GOL) dehydrogenase method with a reference range of 120–150 mg/dl, and HDL-C by direct non-immunological absolute HDL method using Siemens Dade Dimensions (Order No. A91DX-CAI-150351-GC2-4A00-06-2015, Siemens Healthcare Diagnostics Inc., 511 Benedict Avenue, Tarrytown, NY 10591-5005, USA) with a reference range of 30–80 mg/dl. LDL-C was calculated using the Friedwald formula [10].

We defined dyslipidemia, according to the National Cholesterol Education Program Adult Treatment Panel III, as high TC ( $\geq 240$  mg/dl), high TG ( $\geq 200$  mg/dl), and low HDL-C ( $< 40$  mg/dl). Additionally, we defined high non-HDL-C ( $\geq 160$  mg/dl), high TC to HDL-C ratio greater than or equal to 4, high TG to HDL-C ratio greater than or equal to 3.8, and high LDL-C to HDL-C ratio greater than or equal to 2.5 as abnormal dyslipidemia parameters [11,12].

QOL assessment was performed for all patients using a seven-point scale for severity of allergic rhinitis on sleep pattern at night, work performance, and social and/or recreational activities [13].

The objective was to verify clinically the relationship between allergic rhinitis and dyslipidemia aiming to improve patients' quality of outcomes.

### Statistical analysis

Descriptive analysis of demographic and relevant clinical parameters was done. Quantitative variables were presented as mean and SD. The odds ratio and 95% CI of the association between dyslipidemia and allergic rhinitis were calculated. The 'Z' test is also used. *P* value of less than 0.05 was considered to be statistically significant. IBM SPSS version 21 was used for statistical analysis (SPSS Inc., Chicago, Illinois, USA).

### Ethical considerations

Written consents were obtained from all patients. The institutional ethics committee approved the study.

## Results

Our study included 350 patients with allergic rhinitis as a study group with a mean age of  $47\pm 3.8$  years old, 212 (61%) men and 138 (39%) women.

The presenting symptoms are summarized in Table 1. Sneezing was the most predominant symptom in 310 (89%) patients followed by watery nasal discharge in 295 (84%) patients, while irritative cough is the least presented symptom in 89 (25%) patients.

The main finding during nasal examination was edematous nasal mucosa in 304 (87%) patients.

All patients were positive to skin prick test, mostly positive reaction to *Dermatophagoides pteronyssinus*, house dust and domestic pets.

According to the full lipid profile investigation results, the patients were divided according to the criteria of the National Cholesterol Education Program Adult Treatment Panel III [14] under dyslipidemia and normal lipid profile.

A total of 114 (33%) patients had dyslipidemia and 236 (67%) patients had normal lipid profile.

The mean intensity of nasal symptoms according to visual analog scale among the dyslipidemia group and normal lipid profile group had a statistically significant difference between the two groups as shown in Table 2.

QOL scale was calculated and assessed in both groups. There was a relatively better QOL in the normal lipid profile group (4.61) compared with the dyslipidemia group (1.97), and the difference was statistically significant as shown in Table 3.

## Discussion

Dyslipidemia is a recognized, independent key risk factor for the development of metabolic conditions and is known to impact potently the development of atopy when cholesterol enhances latex-specific IgE and Th2 cytokine production by mononuclear cells of patients with atopy [6].

In addition, some authors have found that dyslipidemia promotes proatopic Th2 immunity and allergic inflammation [7].

Cholesterol may increase allergen-specific IgE production, which may in turn aggravate allergic symptoms with correlation between an altered

lipoprotein profile and atopy as association has been hypothesized to be due to alterations in the dietary fat intake, a factor possibly contributing to the increase of allergic diseases in industrialized countries [15].

Allergic sensitization is also related directly to LDL-C, and inversely to HDL-C in Chinese men [16].

Allergic asthma and IgE reactivity have increased over the past decades with symptoms that can also contribute to reduced sleep quality, which is associated with an increased risk of metabolic diseases as Schäfer *et al.* [17] investigated serum cholesterol on atopy and found that a high level of serum cholesterol is associated with the occurrence of atopic diseases.

**Table 1 Allergic nasal and extranasal symptoms among the study group**

Symptoms	N=350 [n (%)]
Sneezing	310 (89)
Runny nose	295 (84)
Stuffiness	277 (79)
Itchy nose	241 (69)
Nasal obstruction	234 (67)
Postnasal drip	207 (59)
Eye symptoms	186 (53)
Throat irritation	141 (40)
Cough	89 (25)

**Table 2 Mean degree of different allergic nasal and extranasal symptoms in both groups**

Symptoms	Dyslipidemia (N=114) (mean±SD)	Normal lipid profile (N=236) (mean±SD)	P value
Sneezing	9.66±0.9	6.79±0.8	0.0001**
Runny nose	9.14±0.33	6.15±0.12	0.0001**
Stuffiness	9.08±0.72	5.98±0.19	0.0001**
Itchy nose	9.01±0.81	5.41±0.23	0.0001**
Nasal obstruction	8.79±1.08	5.11±0.48	0.0001**
Postnasal drip	8.77±1.15	4.97±1.08	0.0001**
Eye symptoms	8.51±1.37	4.81±1.71	0.0001**
Throat irritation	8.19±1.6	4.28±0.15	0.0001**
Cough	7.99±1.98	4.11±0.84	0.0001**

\*\* $P < 0.01$ , highly significant.

**Table 3 Mean scores of quality of life scale in both groups**

	Dyslipidemia (N=114) (mean±SD)	Normal lipid profile (N=236) (mean±SD)	P value
Mean QOL	1.97±0.95	4.61±1.21	0.0001**

QOL, quality of life; \*\* $P < 0.01$ , highly significant.

Sarria *et al.* [18] found that children with high LDL-C have higher blood T lymphocyte CD3, CD4, and CD8 subset counts than those with normal LDL-C.

We found a strong statistically significant correlation between allergic rhinitis symptoms with abnormal dyslipidemia level as the symptom intensity was more observed among them compared with the normal lipid profile

Pesonen *et al.* [19] mentioned that the inverse association between the cholesterol level in infancy and subsequent manifestations of atopy seems not to be due to atopy-related dietary alterations, because it was already present in early infancy, when virtually all the infants were on a similar diet, that is, on exclusive breastfeeding.

Ramaraju *et al.* [20] also investigated serum cholesterol as a risk factor for developing bronchial asthma and they found significant association between higher levels of serum cholesterol and asthma.

The chronic airway inflammation is associated with airway hyperresponsiveness to allergy related to many factors. Such proinflammatory host factor that has gained interest among researchers in recent years is the serum cholesterol level. It is well established that hypercholesterolemia is associated with an enhanced expression of proinflammatory mechanisms leading to increased levels of proinflammatory cytokines, cellular adhesion molecules and inflammation-sensitive plasma proteins [21–23].

Serum cholesterol may also potentiate eosinophilic inflammation in those with genetic susceptibility for atopy with significant correlation between serum cholesterol and elevated inflammatory markers in bronchoalveolar lavage fluid such as eosinophil counts, interleukin-5, prostaglandin E<sub>2</sub>, and monocyte chemoattractant protein-1. The authors have also found that the administration of pravastatin decreased pulmonary allergic inflammation. Similar anti-inflammatory effect of statins at varying doses has been demonstrated in other animal studies indicating its therapeutic potential in asthma [20].

We found a strongly statistically significant better QOL scale in the normal lipid profile group compared with the dyslipidemic group. We can explain the poor QOL in the dyslipidemic group due to burden from allergic rhinitis symptoms with more intense severity, socioeconomic and financial burden from treatment costs plus the metabolic

systemic burden of dyslipidemia. The inflammatory nature of chronic, persistent allergic rhinitis leads to nasal obstruction, difficulties in sleep-wake cycle, snoring, hypersomnolence, memory loss, diminished work performance, and finally insomnia which leads to a negative impact on patient's QOL [24].

The interaction of the immune, nervous, and endocrine systems may drive an individual to a well-recognized biological hypersensitivity and the development of allergic symptoms followed by distinct behavioral patterns characterized as affective hypersensitivity. The nervous and immune systems may interact through the action of neurotransmitters on mast cells. Both the immune and nervous systems are interacting reciprocally to affect each other [25].

Our raw data demonstrated a statistically significant association between allergic rhinitis symptoms severity and dyslipidemia.

Finally, we have to mention that our results indicate that some patients with persistent allergic rhinitis should be investigated for dyslipidemia as a risk factor for developing more symptoms in allergic rhinitis patients and its control could improve the patients QOL aiming to achieve better outcome.

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## Conclusion

Dyslipidemia might play an important role in increasing the severity of allergic rhinitis symptoms with impaired patients' QOL; therefore, its control could achieve better treatment outcomes.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

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