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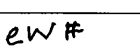
Insert word	Your proof	
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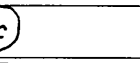
Make capital	your proof	
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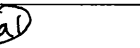
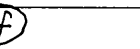
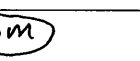
Make bold	Your proof	
Make roman (not italic or bold)	Your proof	
Let it stand; OK as is	Your proof	
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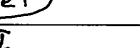
No paragraph	Marked. Your	
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When allergic rhinitis is not only allergic

Matteo Gelardi, M.D.,* Cosimo Russo, M.D.,* Maria Luisa Fiorella, M.D.,* Raffaele Fiorella, M.D.,* Giorgio Walter Canonica, M.D.,# and Giovanni Passalacqua, M.D.#

ABSTRACT

Background: *In clinical practice it can be observed that some patients with seasonal allergic rhinitis (AR) continue to have symptoms even when the exposure to allergens is expected to be low or absent. We studied the clinical and cytological characteristics of these atypical forms of (AR) in a large population of patients.*

Methods: *Consecutive patients with symptoms of rhinitis and with positive skin test to pollens only were interviewed for the duration of symptoms, correlation with sensitization pattern, and presence of reactivity to nonspecific stimuli. All underwent rhinoscopy and nasal scraping for cytology.*

Results: *Five hundred nineteen patients with AR were studied. Of these 519 patients 60 (11.5%) had an atypical or mixed form of rhinitis, with symptoms independent of the exposure and also elicited by nonspecific stimuli. These patients clearly differed from typical forms, especially for the nasal inflammation. They had a greater number of eosinophils and mast cells out of season ($p < 0.05$). Moreover, these atypical forms had, more frequently, asthma and eosinophilic polyps.*

Conclusion: *In ~12% of patients with AR, other mechanisms of inflammation seem to intervene. Nasal cytology can be helpful in discriminating these atypical forms.*

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Key words: Allergic rhinitis, atypical rhinitis, eosinophil, inflammation, mast cell, mixed rhinitis, nasal cytology, seasonal rhinitis

Allergic rhinitis (AR) is the most common IgE-mediated disease. Its prevalence is estimated to range between 5 and 30% and it is increasing worldwide.[1,2] In the majority of cases, the diagnosis of AR does not represent a problem, based on the presence of the typical symptoms (*i.e.*, sneezing, itching, obstruction, and rhinorrhea) and the demonstration of a specific IgE response either by skin testing or blood IgE assay.[3] This is particularly true in the case of sensitization to pollens, where there is a clear chronological pattern of symptoms that is related to the presence of pollens in the environment. For this reason, the term seasonal AR (SAR) is still largely used for those patients suffering from rhinitis occurring only during the pollen seasons of the sensitizing pollens.[4]

In everyday clinical practice it is sometimes observed that a proportion of patients with SAR continue to have symptoms also when the exposure to the allergens is totally absent, *i.e.*, out of the pollen season. These patients seem to have an increased sensitivity to nonspecific stimuli, such as dry or cold air, strong odors, or tobacco smoke. This resembles, from a clinical point of view, the so-called nonallergic rhinitis with eosinophilia syndrome (NARES), where mechanisms other than the IgE-mediated reaction could be hypothesized to intervene.[5,6] The aim of this study was to assess the prevalence and clinical characteristics of the "nonallergic component" in patients with ascertained AR, as well as to assess if they possess a distinctive nasal cytology.

*From the *Department of Otolaryngology II, University of Bari, Bari, Italy, and #Allergy and Respiratory Diseases, Department of Internal Medicine, University of Genoa, Genoa, Italy*

Address correspondence and reprint requests to Giovanni Passalacqua, M.D., Allergy and Respiratory Diseases, Department of Internal Medicine, Padiglione Maragliano, L.go R. Benzi 10, 16132 Genoa, Italy

E-mail address: passalacqua@unige.it

METHODS

Consecutive patients referred to our clinics, because of rhinitis symptoms, and diagnosed with allergic sensitization to pollens, were evaluated for the presence of symptoms also out of the pollen season. They had to have a skin positivity for at least one of the following allergens: grass, *Parietaria*, olive, birch, hazelnut, cypress, or *Compositae.* Patients with skin positivity to mite, *Alternaria*, *Cladosporium*, *Aspergillum,* cats, or dogs were excluded. Skin tests were performed with a commercial panel of the mentioned allergens and read according to the current recommendations.[7] A detailed clinical history was obtained to assess the presence of rhinitis (apart from common cold) out of the pollen season. In addition, patients were asked if rhinitis symptoms were elicited by one of the following: sudden temperature changes, dry air, strong odors, and/or cigarette smoke. The presence of hyposmia and/or hypogeusia, which are not common in AR, was also assessed. Patients with persistent symptoms according to Allergic Rhinitis and Its Impact on Asthma[3] were also evaluated for the presence of asthma, by detailed clinical history and pulmonary function test with bronchodilation or methacholine challenge as appropriate. The presence of rhinosinusitis symptoms, according to guidelines, was assessed as well, and diagnostic confirmation was then obtained by fiberoptic rhinoscopy and/or CT scan.[8] AQ: 1

All patients underwent a nasal scraping both in and out of the pollen season. Treatments, if any, were discontinued before nasal scrapings. The withdrawal period was at least 4 days for oral/nasal antihistamines and 10 days for intranasal corticosteroids. Samples were obtained with a Rhino-Probe and collected from the middle third of the inferior turbinate. After fixing with absolute alcohol for 3 minutes and drying, the samples were stained using May-Grünwald-Giemsa (Carlo Erba, Milan, Italy), and then examined at light microscopy (Nikon E600; Nikon, Italy). Cell count was performed on 10 fields at 1000× magnification under oil immersion.[9] Sam- AQ: 2 AQ: 3

Table 1 **Demography and clinical characteristics of patients with typical and atypical allergic rhinitis (AR)**

	Typical AR (n = 459)	Atypical AR (n = 60)	χ^2
Age range (yr)	4–68	9–76	
Mean age (yr)	32	26	
Sex	220 M (48%) 239 F (52%)	24 M (40%) 36 F (60%)	NS
Intermittent AR	170 (37%)	21 (35%)	NS
Persistent AR	289 (63%)	39 (65%)	NS
Family history for			
Asthma	23 (5%)	5 (8%)	NS
Polyposis	12 (3%)	3 (5%)	NS
Asthma	29 (6.3%)	8 (13%)	0.02
Polyposis	8 (1.7%) 7 Anthrochoanal (unilateral) 2 cystic fibrosis (bilateral) 1 Papilloma (unilateral)	7 (12%) 3 Eosinophilic polyps (bilateral) 4 Eosinophilic mast cell polyps (bilateral)	0.02

Table 2 **Pattern of sensitization (skin-prick test)**

Allergen	Typical AR (n = 459)	Percent	Atypical AR (n = 60)	Percent
Parietaria	29	6.3	6	10
Grass	21	4.7	4	6.7
Olive	11	2.4	2	3.3
Cypress	24	5.2	3	5
Compositae	11	2.4	1	1.7
Ambrosia	8	1.7	—	—
Multiple	355	77.3	44	73.3

AR = allergic rhinitis.

ples were examined blindly by two different investigators. According to the cellular prevalence, we distinguished an eosinophilic form (eosinophils >20% of total cells), a mast-cell form (mast cells >10% of total cells), a neutrophilic form (neutrophils >50%), and a mixed eosinophilic mast cell form (eosinophils >20% and mast cells >10%).[10] Cell counts were compared by Student's *t*-test.

RESULTS

Between January 2005 and December 2007, 519 patients (243 men; mean age, 36 years; age range, 6–76 years) with rhinitis and positivity to one or more pollen allergens were examined. of these patients, 60 (11.5%) could be defined as having an "atypical" AR because symptoms were present also out of the pollen season(s) of the sensitizing allergens. In all of these patients, symptoms were invariably elicited also by temperature changes/strong odors/dry air/tobacco smoke. Such signs of hyperreactivity were present in only 21% of the patients with typical AR. Of the 60 atypical patients, 21 (35%) suffered from intermittent rhinitis and 39 (65%) suffered from persistent rhinitis. According to Allergic Rhinitis and Its Impact on Asthma criteria, there was no difference between the two groups of patients in the demography (Table 1) and pattern of sensitization (Table 2). On the other hand, of the atypical AR patients, 8 (13%) also had asthma and 7 (12%) had nasal polyposis, as confirmed by endoscopy and CT scan. The percentages of asthma and polyposis (6.3% and 1.7%, respectively) were significantly lower in typical AR. In the typical AR, polyps were noneosinophilic (five anthrochoanal, two cystic fibrosis, and one papilloma), whereas in the atypical form polyps were always eosinophilic.

The nasal scraping of all of the patients displayed an intense cellular infiltration, mainly eosinophils or mast cells, during the pollen season, but this infiltration persisted out of the pollen season only in those patients with the atypical AR. The nasal cytology was clearly different between the two subpopulation of patients (typical AR and atypical AR), especially for the number of eosinophils and mast cells. In addition, the patients with atypical AR had a significantly greater number of mast cells during the pollen season (Fig. 1). The neutrophil count was not significantly different between groups. Concerning the type of infiltration, 4 (6.6%) of the 60 atypical patients had mainly mast cells, 35 (58%) had a predominant eosinophilic infiltration, and in 21 (35%) patients there was a mixed infiltration of eosinophils

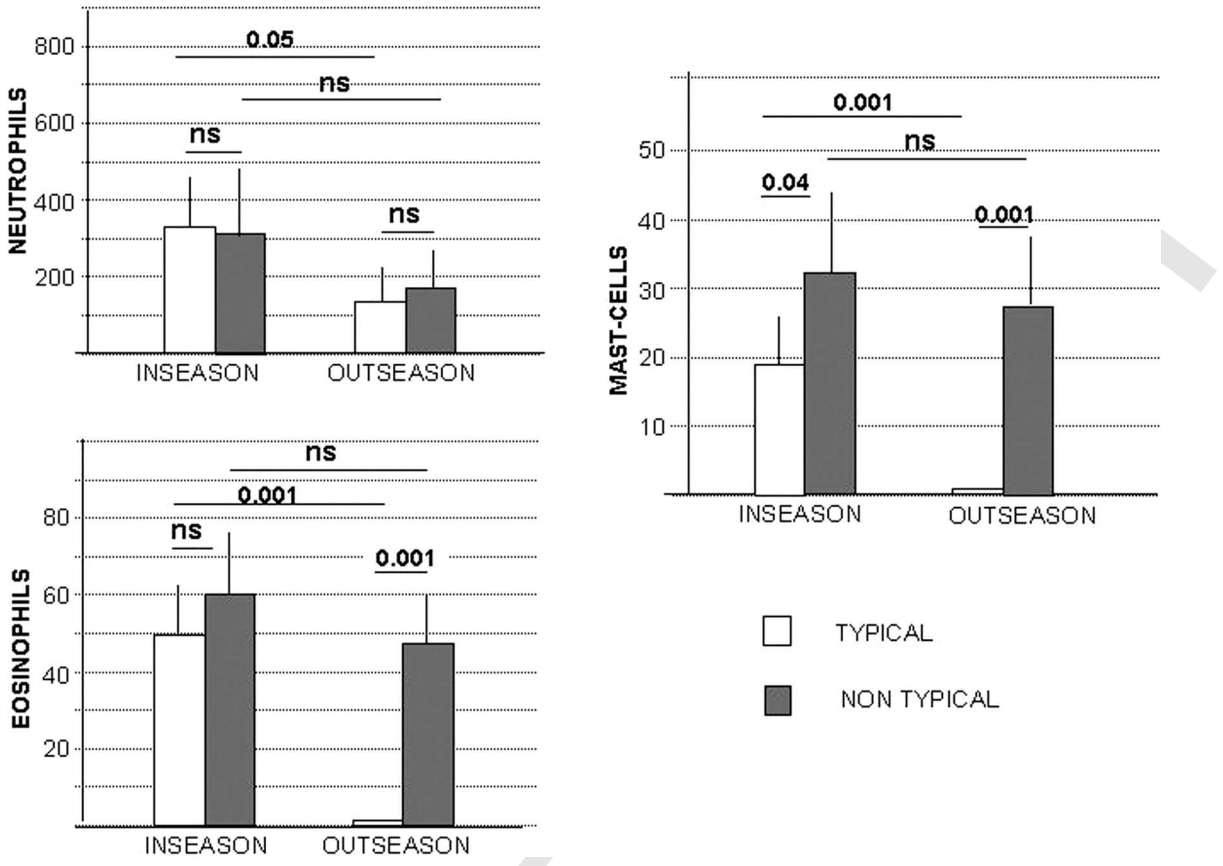

Figure 1. *Neutrophil, eosinophil, and mast cell counts (mean and SD) in nasal scraping in the typical and atypical allergic rhinitis. The p values are reported above the bars.*

and mast cells. Examples of the different cellularity patterns are shown in Fig. 2. F2

DISCUSSION

The IgE-mediated mechanism is of central relevance in AR, but it may happen that other pathogenic mechanisms are superimposed, so that symptoms do not follow the exposure and may be elicited by nonallergenic stimuli.[11] Based on this observation, derived from everyday clinical practice, we identified among subjects with SAR a subset of patients with an atypical form (strictly resembling the so-called NARES) and assessed if clinical and cytological differences exist among those subjects. The clinical subdivision was based on the persistence of symptoms out of season and on the sensitivity to nonspecific stimuli. We found that 11.5% of patients with SAR satisfied those clinical criteria. In those patients there were some clinical differences because they more frequently had asthma and polyposis. Nasal cytology by scraping is a cheap, simple, and safe technique, which is quite useful for the evaluation of nasal inflammation in allergic and nonallergic rhinitis.[12,13] At the nasal scraping we observed that in atypical forms a relevant eosinophilic infiltration with increased mast cells[14] was constantly present, independent of the exposure to the sensitizing allergen, whereas in typical AR, eosinophils were absent out of the pollen seasons, and the atypical ones had a persisting infiltrate when they were not exposed. In this regard, it is well known that in typical AR, AQ: 4 inflammation is triggered by the IgE–mast cell–allergen interaction and is then maintained if the exposure to allergen persists, whereas when the triggering event disappears, the inflammation subsides,[15] leaving sometimes a nasal hyperreactivity (that was in fact present in 21% of the typical forms).

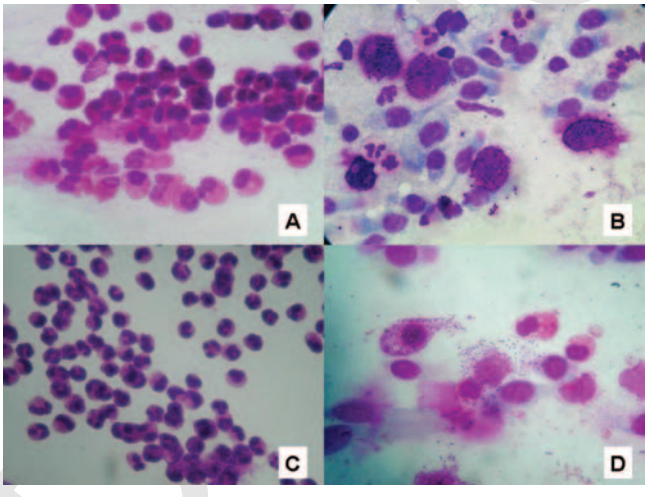

Figure 2. *Examples of nasal scraping with different cellular predominance. (A) eosinophilic, (B) mast cell, (C) neutrophilic, and (D) mixed.*

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In addition, a relevant fraction of patients had also significantly increased mast cells.[15]

In the previously described patients, it seems that two different inflammatory mechanisms coexist, one of which is not triggered by IgE. These atypical forms, despite their clinical characteristics, can not be grouped simply as NARES, because by definition in the NARES there is no IgE sensitization.[5] Otherwise, the atypical or mixed form may be identified at a certain extent as having a nonallergic noninfectious rhinitis overlapping AR.[16–18] A possible speculation is that these atypical or NARES-like forms of AR could explain the therapeutic failure of immunotherapy in some patients despite the fact that they are IgE sensitized, because immunotherapy is specific only for the IgE-mediated component. In conclusion, these observations suggest that AR is not always only allergic and that in the presence of clinical symptoms that are not in agreement with the pattern of sensitization, nasal cytology can provide additional information.

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