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Nasal hypersensitivity in purulent middle ear effusion

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SUMMARY

The existence of a physiopathologic connection between nose and middle ear is widely accepted so that chronic purulent middle ear effusion (CPMEE) could be expected to be usually associated with nasal chronic disease or impaired function. Nevertheless such association is less frequently observed in clinical practice than one could expect, possibly because of inadequate nasal function evaluation. Thirty-five patients affected by CPMEE were included in this study in order to assess the incidence of nasal disorders. E.N.T. clinical history was obtained and E.N.T. physical examination, nasal endoscopy by fiberoptics, anterior rhinorheomanometry, non-specific nasal provocation test with histamine, mucoliary transport test, and allergic skin tests were performed. In the clinical history assessment 26 patients were affected by chronic rhinopathies, 16 by chronic pharyngitis, and 20 by frequent headache. At rhinoscopy we registered nasal septum deviation in 24 cases and mean and inferior turbinates hypertrophy in 31 cases. CPMEE and nasal septum deviation or turbinates hypertrophy were more frequently omolateral ($p < .001$ and $p < .05$, respectively). Total nasal resistance was 0.99 ± 0.49; it was abnormally high in 11 subjects bilaterally and in 4 subjects monolaterally and increased significantly in 32 patients following nasal provocation test. Mucociliary transport time was longer in CPMEE subjects than in 10 healthy subjects (18 ± 5 *vs* 13 ± 4 min; $p < .05$). Finally 10 patients presented positive skin tests. On the whole, 96% of non allergic patients included in this study showed signs of non-specific nasal hypersensitivity which could theoretically cause purulent middle ear effusion to chronicize. Indeed recurrent histamine release in response to specific and/or aspecific stimuli could cause the obstruction of the Eustachian tube and consequently inadequate middle ear ventilation.

Key words: Nasal hypersensitivity. Purulent middle ear effusion. Nasal provocation test.

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INTRODUCTION

In a few patients affected by acute purulent middle ear effusion antbiotic therapy fails to eradicate microbial infection, tympanic drum does not repair, and chronic purulent middle ear effusion (CPMEE) develops (1, 2). Recurrent bacterial infections from upper airways making the development of bacterial resistance to antibiotics easier are often facilitated by the presence of anatomical or immunological factors. The former include facial abnormalities causing inadequate middle ear ventilation through the Eustachian tube; the latter include the deficit of secretory IgA in upper airways or the increased levels of IgE, causing allergic reactions (3-5).

It is widely accepted that the nose plays a particularly important role in the protection of the ear and airways against microbial infections and that anatomical or functional obstruction or chronic phlogosis of the nose are major factors predisposing purulent middle ear effusion to chronicize (6). Nevertheless about 28.5% of subjects affected by CPMEE do not present any apparent nasal disease in clinical practice. This observation could be explained by admitting that the presence of nasal alterations is not necessary to PMEE chronicization or by admitting that the nasal examination routinely carried out could be inadequate to point out minimal alterations of nasal function, particularly concerning immunologic factors. The purpose of this study was to test nasal reactivity in patients affected by CPMEE.

MATERIAL AND METHODS

Thirty-five subjects, 17 males and 18 females, aged 7-73, and affected by CPMEE, were included in this study. Data were collected by:

Clinica history — Life habits, job, diseases, traumas, or previous surgical procedures concerning E.N.T. districts.

E.N.T. physical examination — Including nasal endoscopy by fiberoptics.

Skin tests — Prick tests for pollens, inhalants, and mycophites were utilized.

Anterior rhinorheomanometry (RRM) — Partial and total nasal resistance ($Pa/cm^3\ sec^{-1}$) was calculated by dividing pressure by flow.

Non-specific nasal provocation test (NPT) — The test was carried out according to a previously described method (7, 8). One puff (100 µl) of 2% histamine solution was administred by spray into both nostrils; nasal resistance was measured by RRM before and following the exposure to histamine; the number of sneezes caused by the exposure to histamine was registered (10-13).

Mucociliary transport test — The effectiveness of mucociliary clearance was tested by Tremble's coloured indicator test (9).

Means and standard deviations are reported. Statistical analysis was performed by t-test and Chi square corrected according to Yates.

RESULTS

Thirty-two patients were affected by monolateral CPMEE, 3 by bilateral CPMEE. At audiometry the 38 ears affected by CPMEE presented neurosensorial hypoacusia in 20 cases and mixed hypoacusia in 18 cases; pure transmissive hypoacusia was observed in any case. The 32 ears which were not affected by CPMEE presented normoacusia in 17 cases, transmissive hypoacusia in 2 cases, mixed hypoacusia in 6 cases, and neurosensorial hypoacusia in 7 cases.

Anamnestically 26 patients reported chronic symptoms regarding nose (obstruction 23; rhinorrhea 15) and 16 ones regarding pharyn (chronic pharyngitis); 20 patients complained of recurrent headaches.

Anterior rhinoscopy pointed out significant nasal septum deviation in 24 subjects, which occluded both nostrils in one case, and the hypertrophy of mean and inferior turbinates in 19 cases bilaterally and in 12 cases monolaterally. Table I reports the distribution of the nasal obstruction caused by nasal septum deviation or turbinate hypertrophy whether homolateral to CPMEE or not. Both nasal septum deviation and turbinate hypertrophy were significantly associated to homolateral CPMEE (septum deviation: $p < .05$; hypertrophy: $p < .05$). Skin tests were positive in 10 patients (28.5%); the involved allergens are reported in table II.

Table I

Distribution of turbinates hypertrophy and nasal septum deviation in relation to the presence of homolateral CPMEE

	Homolateral CPMEE Yes	Homolateral CPMEE No	
Turbinates hypertrophy Yes	30	20	50
Turbinates hypertrophy No	8	12	20
Total	38	32	Total: 70 Chi square $p < .05$
Nasal septum deviation Yes	20	4	24
Nasal septum deviation No	18	28	46
Total	38	32	Total: 70. Chi square $p < .001$

Table II

Results of the allergy skin tests

Positive skin tests	10
Graminacee	3
Parietaria Officinalis	2
D. Pt.	2
Multple positive answers	3
Negative skin tests	25

RRM pointed out a significant obstruction in 11 subjects bilaterally and in 1 monolaterally; nasal resistance was 0.47 ± 0.23. NPT caused total nasal resistance to increase both in Skin test positive patients (R changed from 0.54 ± 0.20 to 2.52 ± 1.35; $p < .001$) and in Skin test negative patients (R changed from 0.45 ± 0.24 to 1.87 ± 1.19; $p < .01$) (Fig. 1). The number of sneezes observed following NPT was significantly higher in skin test positive patients than in skin test negative ones (6.5 ± 3.8 *vs* 3.2 ± 3.7). Mucociliary transport time was 18 ± 5 min and was significantly longer than the time registered in 10 healthy subjects (13 ± 4; $p < .05$).

DISCUSSION

This study pointed out that nasal alterations are present in 71.5% of patients affected by CPMEE. Anatomical alterations like nasal septum deviation, chronic IgE-mediated or complement-mediated phlogistic alterations, and nasal non-specific reactivity can progressively involve both paranasal sinuses and

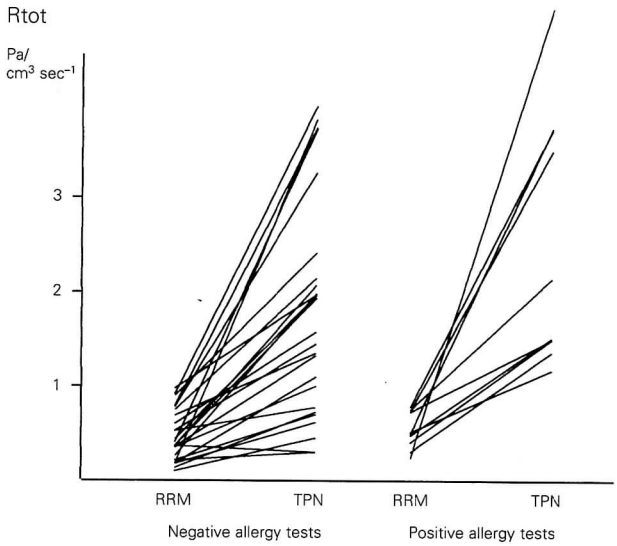

Fig. 1—Total resistance before and after NPT in negative and positive allergy skin test subjects

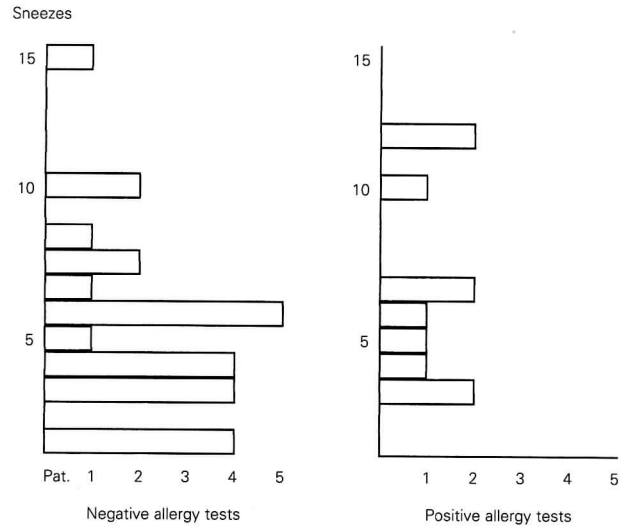

Fig. 2—Sneeze number following NPT in negative and positive allergy skin test subjects

middle ear. Particularly all the phlogistic alterations share non-specific nasal hypersensitivity (96%) that is likely to arise from the allergen challenge in positive test subject and mainly from bacteric superimposition in negative test subjects (10, 11). The significant association between CPMEE and homolateral nasal affections gave further evidence of the link between CPMEE and nasal alterations.

In conclusion our results showed that patients affected by CPMEE are usually affected by nasal hypersensitivity (mostly by non-specific hypersensitivity); as a consequence, environmental stimuli can possibly cause local phlogosis which facilitates bacterial superimposition both in the nose and in the ear. To search for nasal affections and non-specific nasal hypersensitivity and to treat them early could theoretically prevent CPMEE.

RESUMEN

La relación patofisiológica entre la nariz y el oído medio es ampliamente aceptada, de modo que pudiera esperarse que la otitis media purulenta crónica (OMPC) se asociara con rinopatía crónica o con disminución de la función nasal. No obstante, esta asociación se observa menos frecuentemente que lo que sería de esperar en la práctica clínica, posiblemente debido a la evaluación inadecuada de la función nasal. Treinta y cinco pacientes afectados por OMPC fueron incluidos en este estudio con el fin de valorar la incidencia de alteraciones nasales. Se realizaron la historia clínica y exploración física ORL, endoscopia fibraóptica nasal, rinorreomanometría anterior, prueba de pro-

vocación nasal no específica con histamina, prueba de transporte mucociliar y pruebas cutáneas de alergia. Por la historia clínica, 26 pacientes tenían rinopatía crónica, 16 faringitis crónica y 20 cefalea frecuente. En la rinoscopia encontramos desviación del tabique nasal en 24 casos e hipertrofia de los cornetes medio e inferior en 31 casos. La OMPC y desviación del tabique nasal o hipertrofia de cornetes fue más frecuentemente homolateral ($p < 0,001$ y $p < 0,05$, respectivamente). La resistencia nasal total fue 0,99 ± 0,49; con elevación anormal bilateral en 11 sujetos y monolateral en 4 sujetos, y fue significativamente aumentada en 32 pacientes después de la prueba de provocación nasal. El tiempo de transporte mucociliar fue más prolongado en los sujetos con OMPC que en 10 sujetos sanos (18 ± 5 *vs* 13 ± 4 min; $p < 0,05$). Finalmente, 10 pacientes presentaron pruebas cutáneas positivas. Globalmente, el 96% de los pacientes no atópicos incluidos en este estudio mostraron signos de hipersensibilidad nasal no específica, que en teoría podría dar lugar a la cronificación de la otitis media purulenta. De hecho, la liberación recurrente de histamina en respuesta a estímulos específicos y/o no específicos puede producir obstrucción de la trompa de Eustaquio y, consecuentemente, la ventilación inadecuada del oído medio.

Palabras clave: Hipersensibilidad nasal. Efusión purulenta del oído medio. Prueba de provocación nasal.

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