Prognostic factors and natural history of non-ige mediated gastrointestinal food allergy:results from the first year follow-up of the NIGEFA project

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Objectives and Study: Non-IgE mediated gastrointestinal food allergies (non-IgE-GIFA) are an increasing problem in pediatric gastroenterology clinical practice. These conditions include food protein-induced: enterocolitis syndrome (FPIES), enteropathy (FPE), allergic proctocolitis (FPIAP), andmotility disorders (FPIMD). The NIGEFA project is focused on the investigation of main clinical features, prognostic factors (presence atopic dermatitis (AD), multiple food allergies, diagnostic delay, and familial history of allergy), and natural history (atopic march (AM) prevalence and timing of immune tolerance acquisition).

Methods: Prospective observational study evaluating children with non-IgE-GIFA diagnosed according to standard criteria observed at a tertiary center for pediatric gastroenterology and allergy (both sexes, aged <36 m, follow up 12 m after diagnosis). Main anamnestic, demographic, and clinicaldata were collected from all enrolled patients. Immune tolerance acquisition was evaluated by the result of oral food challenge.

Results: A total of 100 patients were enrolled: 58% male, mean age at diagnosis (SD) 8.5(8.8) m.Non-IgE-GIFA conditions were: FPE (44%), FPIES (11%), FPIAP (18%), FPIMD (27%). Mean
diagnostic delay was 5.3 (7.4) m. Multiple non-IgE-GIFA were observed in 47% at baseline. Familial history of allergy was observed in 64% of subjects. Presence of AD before the onset of non-IgE-GIFA was observed in 40% of subjects. The overall rate of immune tolerance acquisition at 12 m was 27%, with a higher rate in FPIAP (44%) compared with FPIMD (29.6%), FPE (22.7%) and FPIES (9.1%) subjects ($p<0.05$). The rate of immune tolerance acquisition at 12 m was significantly lower in children with familial history of allergy (-48%, estimated risk ratio (RR)0.52 (95% CI 0.28 to 0.99, $p<0.05$)) and in those with multiple non-IgE-GIFA (-61%, RR at 12 m 0.39 (95% CI 0.18 to 0.85, $p<0.05$)). At 12 m follow up, the rate of subjects presenting AM was 24% with no difference among the 4 disease groups.The occurrence of AM was significantly higher in subjects with multiple (38%) *vs.* mono non-IgE-GIFA (11%) ($p<.001$) at baseline, with an estimated RR of 3.38 (95% CI 1.47 to 7.81, $p<0.01$) at 12 m.
Moreover, for every 1-month of diagnostic delay there was an increase of 1.04 RR(95% CI 1.01 to 1.07) of AM occurrence at 12 m. No associations with other potential predictors (sex, familial allergyrisk, AD before the onset of GIFA, type of non-IgE-GIFA) were found.

Conclusions: These data shed lights on prognostic factors and natural history of non-IgE-GIFA suggesting the importance of early diagnosis in preventing the occurrence of AM occurrence in thesepatients.

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