Re: Atopy and Risk of Non-Hodgkin lymphoma

Melbye et al. (1) reported from their case–control study that participants with a history of hay fever or detectable serum antigen-specific immunoglobulin E (IgE) had a reduced risk of non–Hodgkin lymphoma (NHL). Two additional findings, however, led them to conclude that the inverse relationship between atopy and NHL, which was also observed in some other case–control studies, might be caused by NHL-induced suppression of the immunologic response to allergens. First, they reported that specific IgE levels were lower in case patients whose NHL was disseminated. Second, in a separate cohort of pregnant women, there was no association between low levels of specific IgE and subsequent risk of NHL, except among women who developed NHL less than 5 years after the blood was drawn. We would like to offer data in support of the first finding and an alternative interpretation of the second.

First, we agree that NHL might have led to a reduced immunologic response. In a population-based case–control study of NHL, we too reported (2) an inverse relationship between atopic conditions, including hay fever and food allergies, and risk of NHL. We measured serum IgG, IgM, and IgA by nephelometry in 597 (85%) of the 704 case patients and 522 (75%) of the 694 control subjects after diagnosis (in case patients) or interview date (in control subjects). Written informed consent was obtained from each participant, and the study was approved by the human research ethics committee of the University of New South Wales. Levels of all immunoglobulins that we measured were markedly lower in case patients than in control subjects (Table 1). This observation is consistent with an impaired immunologic response to antigens caused by NHL, as hypothesized by Melbye et al. (1), and a lessening of atopic symptoms, as previously reported by others (3).

Second, we believe that there is doubt about whether the cohort finding, that specific IgE levels in pregnant women did not predict later NHL risk, can be generalized to the wider population. There are some data to indicate that levels of serum immunoglobulin as well as the overall balance between Th1 and Th2 function may be different between pregnant and non-pregnant women (4,5). A previous cohort study that reported no association between specific IgE levels and lymphoma risk did not separate patients with Hodgkin lymphoma from those with NHL (6).

The possible inverse association between the immune state that is associated with atopic disease and NHL requires further investigation. Analysis of case–control studies that have collected data on the life course of atopic disease would be helpful. In our study, the inverse risk of NHL was similar in magnitude for history of hay fever in childhood and in adulthood (2). Studies that have collected data on indicators, such as birth order, of early exposure to infection and other exposures that condition immunity toward an atopic state are also needed (2). Our data support the finding of Melbye et al. (1) that the immunologic response to antigens is impaired by NHL. However, it remains possible that the pattern of immunity established early in life that is later manifest as atopy is truly protective against NHL.

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References


Notes

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