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Poster abstracts

Maternal pregestational diabetes and risk of childhood leukaemia in the offspring: a population-based study in Northern Italy

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Introduction

The aetiology of childhood cancer is not well understood, but the typically early onset suggests that genetic, prenatal and perinatal factors, such as maternal diabetes, may be involved (Wiemels, 2015). We aimed to examine the influence of maternal pregestational diabetes on Acute Lymphoblastic Leukaemia (ALL) risk in the offspring.

Methods

All children born in Bologna and Reggio Emilia, two provinces in Northern Italy, between 1998–2010 (n = 161,804) were followed from birth until first cancer diagnosis, age 15 years, or December 31, 2017. Data on newborns, maternal diabetes and covariates were obtained from hospital discharge records, data on ALL cases from the National Childhood Cancer Register of the Italian Association of paediatric haematology and oncology (AIEOP). We calculated annual incidence rate of ALL per 100,000 person-years, and Hazard Ratio (HR) with 95% confidence interval (CI) by using Cox regression adjusted for maternal age and nationality.

Results

We observed 97 cases of ALL, with annual incidence rate of 16.3 (95% CI: 4.1–65.2) per 100,000 person-years among children born to women with pregestational diabetes and 4.6 (95% CI: 3.8–5.6) among those born to women without pregestational diabetes. In multivariable analysis, maternal pregestational diabetes was associated with higher risk of ALL in the offspring (adjusted HR [95% CI]: 3.5 [0.8–14.1]).

Conclusions

In this population-based cohort study, we estimated higher risk of ALL among offspring of women with pregestational diabetes. These results are consistent with previous findings (Contreras *et al*, 2016; Deleskog *et al*, 2017; Søgaard *et al*, 2017) and they are compatible with a role of prenatal and perinatal glycaemic environment in childhood cancer aetiology. Given the overall rarity of both maternal diabetes and childhood leukemia, studying their association is challenging. Further studies, including larger cohorts of newborns, are needed to refine our understanding of this association and to identify biological mechanisms underlying childhood ALL.

References

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- Wiemels (2015) *Eur J Epidemiol* 30: 1225 – 1227