

INTISARI

Latar belakang: Demam neutropenia merupakan salah satu tantangan yang dihadapi dalam penatalaksanaan berbagai jenis penyakit keganasan, termasuk leukemia limfoblastik akut (LLA) anak. Beberapa penelitian menunjukkan adanya hubungan antara profil imunofenotipik limfoblas dan derajat pendesakan sumsum tulang, yang merupakan salah satu dasar patofisiologi neutropenia pada LLA anak.

Tujuan: Mengetahui pengaruh koekspresi antigen prekursor limfosit T, mieloid, dan *non-lineage* (CD34 dan *cytoplasmic terminal deoxynucleotidyl transferase* [cTdT]) pada limfoblas anak dengan LLA prekursor sel B (LLA-B) terhadap kejadian demam neutropenia pada pengobatan fase induksi.

Metode: Penelitian kasus-kontrol melibatkan penderita LLA-B anak yang mengalami demam neutropenia (kasus) dan tidak mengalami demam neutropenia (kontrol) pada pengobatan fase induksi. Penderita-penderita tersebut menjalani pengobatan dengan protokol Indonesia-ALL-2006 di Rumah Sakit Umum Pusat (RSUP) Dr. Sardjito, Yogyakarta, dalam kurun waktu tahun 2006 - 2011. Terhadap masing-masing kelompok kasus dan kontrol, dilakukan penelusuran hasil analisis imunofenotipik pada saat penegakan diagnosis.

Hasil: Sebanyak 28 orang kasus dan 29 orang kontrol diikutsertakan dalam penelitian ini. Koekspresi antigen prekursor limfosit T, mieloid, dan *non-lineage* masing-masing ditemukan pada 22 (39%), 12 (21%), dan 48 (84%) dari 57 orang anak dengan LLA-B. Analisis bivariat menunjukkan adanya hubungan antara kejadian demam neutropenia dan tidak terdapatnya koekspresi antigen prekursor limfosit T (*odds ratio* [OR] 0,31; interval kepercayaan [IK] 95% 0,10 - 0,96).

Simpulan: Koekspresi antigen prekursor limfosit T pada limfoblas anak dengan LLA-B mengurangi risiko terjadinya demam neutropenia pada pengobatan fase induksi. Meskipun demikian, pengaruh faktor-faktor lain yang berkaitan dengan patofisiologi demam neutropenia masih perlu dipertimbangkan.

Kata kunci: LLA-B anak, demam neutropenia, antigen prekursor limfosit T, antigen mieloid, antigen *non-lineage*

ABSTRACT

Background: Febrile neutropenia is one of the major challenges in the management of various neoplastic diseases, which include childhood acute lymphoblastic leukemia (ALL). Previous studies revealed the presence of association between immunophenotypic profiles of the lymphoblasts and the degree of bone marrow suppression, which constitutes the basic mechanism in the pathophysiology of neutropenia in childhood ALL.

Objectives: To determine the influence of T-lineage, myeloid, and non-lineage (CD34 and cytoplasmic terminal deoxynucleotidyl transferase [cTdT]) antigen coexpression in the lymphoblasts of children with B-lineage ALL (B-ALL) on the incidence of febrile neutropenia in the remission induction phase of treatment.

Methods: A case-control study involved children with B-ALL, who did (the cases) and did not (the controls) experience febrile neutropenia in the remission induction phase of treatment, according to the Indonesia-ALL-2006 protocol. Each of the cases and controls was admitted to Dr. Sardjito General Hospital, Yogyakarta, during the period of 2006 - 2011. The diagnostic immunophenotypic profiles of each case and control were reviewed.

Results: Twenty-eight cases and 29 controls were eligible for this study. The presence of T-lineage, myeloid, and non-lineage antigens was observed in 22 (39%), 12 (21%), and 48 (84%) of 57 children with B-ALL, respectively. Bivariate analysis revealed the presence of association between T-lineage antigen coexpression and the absence of febrile neutropenia (odds ratio [OR] 0.31; 95% confidence interval [CI] 0.10 - 0.96).

Conclusion: The presence of T-lineage antigens in the lymphoblasts of children with B-ALL may reduce the risk of febrile neutropenia in the remission induction phase of treatment. Nevertheless, the influence of other factors, which are involved in the pathophysiology of febrile neutropenia, toward these findings remains under consideration.

Keywords: childhood B-ALL, febrile neutropenia, T-lineage antigens, myeloid antigens, non-lineage antigens