Introduction

Although outcome for children with pediatric acute lymphoblastic leukemia (ALL) has improved considerably in recent years, a significant proportion of cases relapse ***[Hong et al., 2008].***

The complexity of the leukemogenic process, together with our limited understanding of the biology of this disease, presents a challenge to developing novel therapeutic approaches. ***[Cobaleda et al., 2000]***

Developments in flow cytometric techniques and the availability of lineage-associated monoclonal antibodies have permitted characterization of normal and leukemia cells and affirmed the immunophenotypic heterogeneity in ALL *[**Plasschaert et al., 2004**]*.

Evidence suggests that ALL have a primitive cell origin and share many immunophenotypic characteristics with normal progenitor cells. These leukemic stem cells may be resistant to current therapeutic strategies and subsequent relapses may arise from this population ***[Cox et al., 2005**]*.

These cancer stem cells have been shown to express CD133 (AC133), a primitive cell antigen ***[Horn et al., 1999**]*. That has been shown to be more specific marker of hematopoietic stem cells than CD34 ***[Toren et al., 2005**]*.

There have been conflicting reports on the expression of CD133 in ALL. Whereas some found high levels of CD133 expression on particular cases *[**Wuchter et al., 2001**]*. Others detected only few levels ***[Miraglia et al., 1997]*** or none at all *[**Horn et al., 1999**]*.

Despite several investigations, the biological role of the CD133 antigen on either normal or leukemic hematopoietic progenitors remains poorly defined ***[Lee et a.l, 2001].***