

Introduction

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The current classification of acute myeloid leukemia (AML) is based predominantly on the cytogenetic abnormalities and morphology of malignant blasts but it is not always helpful for optimization of the treatment strategy (*Yamashita et al., 2006*).

CD34 is the most commonly used antigen to define immature hematopoietic progenitor cells. In acute leukemia immunophenotyping, CD34 is not lineage restricted and thus not useful for distinguishing acute myeloid leukemia (AML) from acute lymphoblastic leukemia (ALL) (*Russell NH, 1999*).

Human CD133 (AC133) is a novel five-transmembrane molecule, which is expressed on primitive normal hematopoietic progenitor. AC133 reacts with a population of non committed or granulomonocytic GM-committed CD34⁺ cells in normal hematopoiesis. AC133 reactivity was observed in cases of AML especially myelomonocytic different AML FAB M4/M5 cases (*Buhring et al., 2002*).

In the hematopoietic system, CD133 is expressed on a subset 30-70% of the CD34⁺ cells in the human bone marrow, fetal liver, umbilical cord blood and growth factor primed peripheral blood (*Corbeil et al., 2003*).

The expression of CD133 on primitive AML cells is unknown, while it has been reported that CD133 is expressed on the majority of bulk CD34⁺ AML cells whereas CD133 expression on CD34⁻ AML cells is low to absent in most but not all cases (*Kratz-Albers et al., 2001*).

Thus, studies suggest that CD133 can be useful as an additional marker for CD34 to purify the primitive hematopoietic progenitors. Recent evidence suggests that stem cell rarely CD34⁻ also express CD133 allowing their purification (*Gallacher et al., 2003*).

There have been conflicting results regarding a correlation between CD133 expression and FAB type, with increased CD133^{bright} cells in the M4 & M5 FAB subtypes and in FAB M0 reported. CD133 expression on FAB M3 cases has been low to

absent in all reports and this is consistent with an absence of CD34 expression on these samples. No correlation between CD133 expression and cytogenetics or prognosis has been reported (*Snell et al., 2001*).