



REVIEW

Global characteristics of childhood acute promyelocytic leukemia



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ARTICLE INFO

Keywords:

Acute promyelocytic leukemia

AML-M3

Pediatric leukemia

Therapy-related leukemia

Environmental exposure

Risk factors

ABSTRACT

Acute promyelocytic leukemia (APL) comprises approximately 5–10% of childhood acute myeloid leukemia (AML) cases in the US. While variation in this percentage among other populations was noted previously, global patterns of childhood APL have not been thoroughly characterized. In this comprehensive review of childhood APL, we examined its geographic pattern and the potential contribution of environmental factors to observed variation. In 142 studies (spanning >60 countries) identified, variation was apparent—*de novo* APL represented from 2% (Switzerland) to >50% (Nicaragua) of childhood AML in different geographic regions. Because a limited number of previous studies addressed specific environmental exposures that potentially underlie childhood APL development, we gathered 28 childhood cases of therapy-related APL, which exemplified associations between prior exposures to chemotherapeutic drugs/radiation and APL diagnosis. Future population-based studies examining childhood APL patterns and the potential association with specific environmental exposures and other risk factors are needed.

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1. Introduction

1.1. Leukemia is the most common type of cancer in children

Leukemia, the most common type of cancer in children [1], accounts for 25–35% of cases of childhood cancer in most populations [1,2]. Acute myeloid leukemia (AML) and acute lymphocytic leukemia (ALL) comprise the two major subtypes of childhood leukemia, with ALL

accounting for 76% of childhood leukemia cases [3,4]. AML, the second largest subgroup in children but the most common leukemia type among adults [3], represents 15–20% of leukemia cases in children, and is responsible for up to 30% of pediatric leukemia related deaths [4,5].

De novo acute promyelocytic leukemia (APL), a subtype of AML, represents about 5–10% of childhood AML cases in the United States [6]. Previous studies, in which the majority of cases were reported from clinical trials or treatment protocols, rather than population-based

Abbreviations: ALL, Acute Lymphocytic Leukemia; AML, Acute Myeloid Leukemia; APL, Acute Promyelocytic Leukemia; ATRA, All-trans Retinoic Acid; BMI, Body Mass Index; CI, Confidence Interval; CLIC, The Childhood Leukemia International Consortium; FAB, French–American–British; HL, Hodgkin Lymphoma; ICD-10, 10th revision of the International Statistical Classification of Diseases and Related Health Problems; LCH, Langerhans Cell Histiocytosis; MS, Multiple Sclerosis; NHL, Non-Hodgkin Lymphoma; OR, Odds Ratio; t-AML, Therapy-related AML; t-APL, Therapy-related APL; WHO, World Health Organization.

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analyses, have suggested that in certain Latin American, European and African populations, APL comprises relatively higher percentages of childhood AML [7]. However, variation in incidence among geographic regions has not been formally explored at a global level.

This review not only provides an overview of childhood APL, but also aims to: 1) examine childhood APL as a proportion of AML in countries around the world in order to gain insight into potential global geographic patterns; 2) analyze whether a previously hypothesized gender predominance in childhood APL cases exists; and, 3) discuss the potential contribution of environmental risk factors to the development of APL, using the example of exposure to previous therapy for primary diseases.

1.2. APL is a relatively well-characterized subtype of AML

AML encompasses a heterogeneous group of leukemias characterized by increased proliferation of myeloid cells in the bone marrow [8]. Among the subtypes of AML, APL is of particular interest due to its well-characterized etiology. With targeted treatment involving chemotherapy and all-trans retinoic acid (ATRA), the survival rate of APL in children is relatively high (75–80%) [9]. Additionally, variation in the incidence of APL as a percentage of total childhood AML across certain racial/ethnic groups and geographic regions has been previously observed, and is potentially attributable to certain environmental exposures.

The etiology, molecular mechanisms, and treatment of APL have been comprehensively studied. In 1990, based on the observation that retinoic acid, a vitamin A derivative, is able to induce in vivo

differentiation of APL cells into mature granulocytes, a French team of researchers examined the retinoic acid receptor gene (*RARα*) and discovered that the t(15;17) translocation, characteristic of the majority of APL cases, involved the *RARα* gene (located on chromosome 17) and the *PML* locus on chromosome 15, resulting in *PML/RARα* fusion products [10,11].

1.3. APL classification is based on morphological and cytogenetic information

Under the French–American–British (FAB) classification system, AML is categorized into eight subtypes (AML-M0 to M7) based on morphological features, as well as percentage and maturation of myeloblasts [12]. Under the FAB system, APL is characterized as subtype AML-M3, in which the predominant cells are promyelocytes with heavy granules and Auer rods. Diagnosing the microgranular variant of APL (AML-M3v) can be difficult because its morphological and cytochemical features are often non-specific, leading to misdiagnosis as AML-M4 or AML-M5 [13]. For these reasons, APL diagnosis by morphology alone has its limitations.

Random somatic chromosomal abnormalities resulting in fusion gene rearrangements are common in the malignant cells of patients with AML. About 95% of APL cases are characterized by recurrent chromosomal rearrangements of the *RARα* gene located on chromosome 17 [14], with the majority involved in a t(15;17)(q24;q21) translocation where the *RARα* gene fuses to the *PML* gene on chromosome 15. Eight

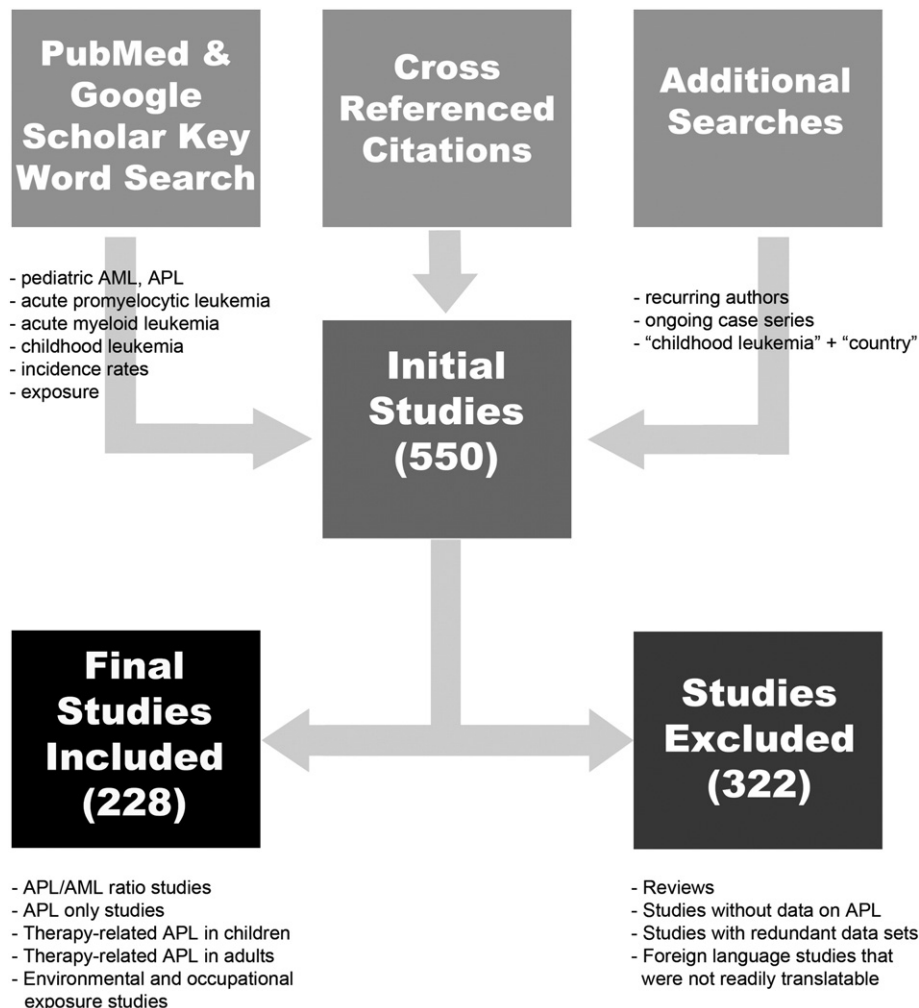


Fig. 1. Study selection process. This flow diagram depicts the logic of the study selection process, the results of which are included in this review. In total, 228 studies were included.

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