

Boolean Algebra in Diagnostics, Risk Stratification, and Treatment Decisions in Acute Lymphoblastic Leukemia

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Received date: April 20, 2026; **Accepted date:** May 12, 2026; **Published date:** May 20, 2026

Citation: Gerhard Zugmaier, (2026), Boolean Algebra in Diagnostics, Risk Stratification, and Treatment Decisions in Acute Lymphoblastic Leukemia, *J Clinical Research and Reports*, 24(1); DOI: [10.31579/2690-1919/631](https://doi.org/10.31579/2690-1919/631)

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Abstract

Background: Acute lymphoblastic leukemia (ALL) is a biologically heterogeneous malignancy of precursor lymphoid cells whose diagnosis and management require integrated interpretation of morphology, flow cytometry, cytogenetics, molecular genetic alterations, clinical context, and treatment response. Contemporary classification and management frameworks emphasize that ALL cannot be defined adequately by morphology alone and that diagnosis, risk assignment, and therapy selection must be based on a multiparameter approach.

Methods: Boolean algebra, which operates on binary variables using the operators AND ($\wedge$), OR ($\vee$), and NOT ($\neg$), provides a transparent framework for formalizing such decision processes. In this manuscript, Boolean algebra was applied across the three domains named in the title: diagnostics, risk stratification, and treatment decisions in ALL.

Results: Diagnosis was modeled as an integrated Boolean state that requires concordance among morphology, flow cytometry, cytogenetics, and genetic alterations, while also distinguishing ALL from lymphoblastic lymphoma (LBL). Risk stratification was represented as a staged clinical process rather than a single flattened equation. Baseline clinical and biologic risk were assigned first, after which treatment response—especially measurable residual disease (MRD)—can reclassify disease into a very high risk (VHR) state. Persistent or clinically decisive MRD was therefore incorporated within VHR rather than treated as an entirely separate parallel variable. Treatment decisions were modeled as downstream consequences of this hierarchy, with targeted therapy, treatment escalation, and consideration of hematopoietic stem cell transplantation (HSCT) linked to specific Boolean states.

Conclusion: Boolean formalization does not replace clinical judgment, but it clarifies the logical architecture of modern ALL care. Used carefully, it can improve conceptual precision, make decision pathways more explicit, and support interpretable computational approaches in hematologic oncology.

Keywords: boolean algebra; acute lymphoblastic leukemia; treatment decisions

Introduction

Acute lymphoblastic leukemia (ALL) is a malignant neoplasm of immature lymphoid progenitor cells characterized by clonal proliferation in the bone marrow (BM), peripheral blood, and extramedullary tissues such as the central nervous system (CNS). Contemporary diagnostic frameworks recognize ALL as a biologically diverse family of precursor lymphoid neoplasms defined by lineage, immunophenotype, cytogenetic abnormalities, molecular genetic alterations, age at presentation, and response to therapy. [1-4] The 2024 European Leukemia Net (ELN) recommendations and the 2024 National Comprehensive Cancer Network (NCCN) guideline update both emphasize integrated biologic

characterization, risk-adapted therapy, and treatment reassessment based on measurable residual disease (MRD). [1-4].

This modern view of ALL has profound implications for clinical reasoning. The clinician does not diagnose or treat ALL on the basis of a single finding. Instead, diagnosis emerges from concordance among morphology, flow cytometry, cytogenetics, and molecular genetics. Risk assignment begins with presenting features such as age, white blood cell count (WBC), and genotype, but it is subsequently modified by treatment response, particularly MRD. Therapeutic branching then depends on both biologic subtype and response-defined states. In practice, these steps are governed by combinations of conditions that are either present or absent,

even if clinicians do not usually describe them in formal symbolic language [1-4,6]

Boolean algebra provides a mathematical framework for expressing this type of reasoning. In Boolean systems, variables are binary and can be combined through conjunction (AND, $\wedge$), disjunction (OR, $\vee$), and negation (NOT, $\neg$). A growing systems-medicine literature has highlighted Boolean modeling as a mathematics - based dynamic approach that is especially useful when one wants transparent, interpretable rules rather than opaque prediction engines [5]. This makes Boolean algebra particularly attractive in diseases such as ALL, where classification and treatment are increasingly complex but still rest on rule-based clinical structure.

However, Boolean modeling of ALL is only useful if it is clinically accurate. Several recurrent oversimplifications must be avoided. First, diagnosis cannot be reduced to one test modality. Second, lineage assignment cannot be reduced to one marker such as CD19. Third, risk cannot collapse into a single static equation because modern ALL care is staged and response-adapted. Fourth, persistent MRD should not be modeled outside the highest-risk construct; clinically, it is one of the features that define a very high risk (VHR) state. [1-4,6].

The aim of this manuscript is to develop a clinically faithful Boolean framework for ALL that follows the structure of the title itself: diagnostics, risk stratification, and treatment decisions. The manuscript is intentionally hybrid. It is a clinical review of how ALL is diagnosed and managed, but it also layers Boolean formalization on top of that review to make the underlying mathematics explicit. The methodological background includes prior publications by Zugmaier and colleagues applying mathematical logic (Boolean algebra) to immunophenotyping, acute leukemia definition, cytogenetic risk stratification, myeloid neoplasms, laboratory diagnostics, and hematologic differential diagnosis [7-13]. The present manuscript extends that mathematics - based program specifically to the clinical pathway of ALL.

Methods

This manuscript is a conceptual and analytical review rather than a prospective or retrospective clinical study. Established diagnostic and therapeutic pathways in ALL were translated into Boolean expressions using binary encoding of clinically relevant variables. The objective was not to force hematologic biology into an artificial mathematical scheme, but to formalize the structure of real decision-making in a way that remains interpretable and clinically grounded [1-6].

Boolean representation of clinical variables

Clinical variables were encoded as present 1 or absent 0. This applied to immunophenotypic marker expression, lineage assignment, cytogenetic abnormalities, molecular genetic lesions, CNS involvement, relapse, and poor early response. Continuous variables were dichotomized only where clinical care already uses thresholds to activate specific decisions. Thus, high WBC count was treated as a threshold-defined variable in the context of baseline risk assignment, and MRD was treated as positive or negative relative to clinically meaningful assay cutoffs. [1-6].

Boolean operators

Three Boolean operators were used throughout the manuscript:

- **AND ($\wedge$):** all linked conditions must have the value 1

- **OR ($\vee$):** at least one linked condition must have the value 1
- **NOT ($\neg$):** the linked condition have the value 0

When treatment branching was described, an implication arrow ($\rightarrow$) was used in a clinical sense to indicate that a specific state opens a management pathway.

Methodological background from prior Boolean-algebra publications

The methodological framing of this manuscript was informed by prior applications of mathematical logic (Boolean algebra) in hematologic diagnostics by Zugmaier and colleagues. These publications addressed immunophenotyping of B-cell precursor acute lymphoblastic leukemia (BCP-ALL), cytogenetic definition and risk stratification of BCP-ALL, Boolean definition of acute leukemias, Boolean definition of myeloid neoplasms, laboratory diagnostics in medicine, diagnosis of hematologic disorders, and computational differential diagnosis in medicine. [7-13] In the present manuscript, those prior mathematics - based approaches are extended from diagnostic formalization into the broader clinical sequence of diagnosis, staged risk assignment, and treatment branching in ALL.

Results

I. Boolean Algebra in Diagnostics of Acute Lymphoblastic Leukemia

Diagnosis as an integrated four-pillar state

The first major result of the model is that diagnosis of ALL is not a one-modality event. In current clinical practice, ALL is defined through concordant interpretation of morphology, flow cytometry, cytogenetics, and molecular genetic alterations [1-4]. In other words, diagnosis is not merely “suggested” by these domains; rather, it is constituted by them when taken together. A clinically faithful Boolean representation is therefore:

$$ALL_{diagnosis} = Morphology_{ALL} \wedge Flow_{ALL} \wedge Cytogenetics_{ALL} \wedge Genetics_{ALL}$$

This expression does not imply that every patient must harbor one specific defining lesion in every domain. Instead, it indicates that a complete diagnosis of ALL requires all four domains to be assessed and interpreted as consistent with the ALL spectrum. Morphology provides the acute blast-rich marrow context. Flow cytometry establishes lineage and precursor status. Cytogenetics identifies recurrent chromosomal patterns associated with ALL subtypes. Molecular genetics defines or refines subtype and identifies biologically important lesions. Diagnosis is therefore a concordant Boolean state rather than a single-test conclusion. [1-4]

Morphology as the first diagnostic pillar

Morphology is the first pillar because it establishes the acute leukemia context. In Boolean terms, morphology in ALL can be expressed as:

$$Morphology_{ALL} = AcuteBlastPattern \wedge MarrowPredominance$$

Morphology shows that the disease is a blast-rich precursor neoplasm rather than a mature lymphoid process or a reactive marrow condition. It also contributes to the distinction between ALL and lymphoblastic lymphoma (**LBL**), which remains clinically important even though both entities lie on a biologic spectrum [14]. A simplified operational distinction can be expressed as:

$$ALL = PrecursorLymphoidNeoplasm \wedge MarrowDominant \\ \wedge (BM\ blasts \geq 20\% - 25\%)$$

$LBL = PrecursorLymphoidNeoplasm \wedge MassDominant \wedge$
($BM\ blasts < 20\% - 25\%$) This is an operational rather than ontologic division, but it remains a real diagnostic gate because it determines which pathway of disease characterization and treatment planning is entered. [1, 14].

Flow cytometry as the lineage and immaturity pillar

The second diagnostic pillar is flow cytometry. Here the value of Boolean representation is particularly clear because immunophenotypic diagnosis already behaves like a binary system. Lineage is determined by co-expression and non-expression patterns, not by one marker in isolation. For precursor B-lineage disease, a useful Boolean approximation is:

$$B-ALL = (CD19 \vee CD79a \vee CD22 \vee PAX5) \wedge \neg CD3 \wedge \neg MPO$$

For T-lineage disease:

$$T-ALL = (CD3 \wedge CD7) \wedge \neg CD19$$

These expressions formalize several important findings. First, lineage assignment is pattern-based, not one-marker based. Second, exclusion of incompatible lineage-defining features is as important as inclusion of the correct pattern. Third, precursor status often depends on the broader immunophenotypic context, including markers of immaturity such as TdT. Flow cytometry therefore functions not merely as a confirmatory test but as a co-defining diagnostic pillar. [1-4].

Cytogenetics as a co-defining diagnostic pillar

The third diagnostic pillar is cytogenetics. In contemporary ALL, cytogenetic analysis is not only prognostic; it is also diagnostic because it identifies recurrent chromosomal abnormalities associated with specific ALL entities and subtype families. In Boolean form:

$$Cytogenetics_{ALL} = DefiningCytogeneticAbnormality$$

Some cases contain clearly defining lesions, whereas others have a cytogenetic profile that is diagnostic by compatibility rather than by a single signature lesion. In either scenario, cytogenetic information contributes to disease definition and should be present in a complete Boolean model of diagnosis. [1-4].

Genetic alterations as the molecular diagnostic pillar

The fourth diagnostic pillar is molecular genetics. Modern classification increasingly defines precursor lymphoid neoplasms by recurrent gene fusions and other molecular alterations. A Boolean representation is:

$$Genetics_{ALL} = DefiningGeneticAlteration$$

This includes lesions that directly define subtype, as well as molecular contexts that strongly support an ALL diagnosis or help exclude alternatives. The increasing importance of genetic subclassification means that molecular findings are not merely prognostic refinements; they are part of what makes the modern diagnosis of ALL complete [1-4].

CD19 as a therapeutic branch rather than the whole diagnosis

A specific diagnostic and therapeutic result concerns CD19. CD19 is often treated informally as though it were synonymous with B-ALL, but

this is clinically unsafe. A patient can have precursor B-lineage disease without preserving CD19 targetability, particularly in the setting of antigen loss after immunotherapy. The correct Boolean separation is:

$$B-ALL = B\text{-lineage pattern} \\ CD19Targetability = B-ALL \wedge CD19$$

This distinction matters because it separates diagnostic identity from therapeutic eligibility. It is one of the clearest examples of how Boolean formalization improves clarity by forcing clinically distinct questions to remain distinct [1,15].

II. Boolean Algebra in Risk Stratification

Baseline risk assignment as the first stage

The second major domain is risk stratification. The model shows that risk is best understood as a staged process. The first stage is baseline risk assignment, based on variables known at presentation:

$$BaselineRisk = AgeRisk \vee WBC_{high} \vee CNS_{pos} \vee GeneticRisk \\ GeneticRisk = BCR::ABL1 \vee KMT2A \vee Hypodiploid \\ \vee OtherAdverseSubtype$$

Baseline risk remains clinically meaningful because it determines initial protocol allocation, the expected likelihood of treatment response, and the intensity of early monitoring. However, the Results make clear that baseline risk is only the first layer and is not sufficient as a final description of treatment-relevant risk [1-4].

Age as a multistate rather than binary variable

A second result is that age must be modeled as a multistate variable rather than a single threshold. In ALL, infancy, favorable-risk childhood, adolescent and young adult (AYA) status, and older adulthood each have distinct biologic and therapeutic implications. A Boolean representation that better reflects clinical reality is:

$$Age_{infant} = (Age < 1) \\ Age_{child\ favorable} = (Age \geq 1 \wedge Age < 10) \\ Age_{AYA} = (Age \geq 15 \wedge Age \leq 39) \\ Age_{older} = (Age \geq 60)$$

This layered structure is preferable to crude one-threshold models because it allows age to function as a context-dependent clinical category rather than as a simplistic split. [1-4].

Response-based reassessment as the second stage

The key departure from oversimplified prior models is the introduction of a second stage: response-based reassessment. Modern ALL management does not stop at baseline assignment. After induction and early treatment, response variables can redirect the patient into a substantially different treatment pathway. This can be represented as:

$$ResponseReclassification \\ = MRD_{persistent} \vee PoorEarlyResponse \\ \vee Relapse$$

This stage captures the clinical reality that persistent MRD, inadequate early cytorreduction, or frank relapse can supersede the implications of presenting features. In that sense, risk in ALL is not static; it evolves with treatment response [1-6].

Very high risk as an override state

The third stage is very high risk (VHR) designation. In the revised system VHR is not simply “more risk” added to a baseline score. It is an override state that changes treatment-relevant risk:

$$VHR = BCR::ABL1 \vee KMT2A \vee Hypodiploid \\ \vee ResponseReclassification$$

Expanded:

$$VHR = BCR::ABL1 \vee KMT2A \vee Hypodiploid \vee MRD_{persistent} \\ \vee PoorEarlyResponse \vee Relapse$$

This is the crucial correction. Persistent MRD belongs within VHR. It is not best modeled as a separate final-risk term outside the highest-risk construct. The treatment-relevant risk architecture is therefore staged:

$$InitialRiskAssignment = BaselineRisk \\ TreatmentRelevantRiskState \\ = \begin{cases} VHR, & \text{if VHR present after response assessment} \\ BaselineRisk, & \text{if VHR absent} \end{cases}$$

This staged representation is more clinically faithful than any flat “final risk equals X or Y” expression because it preserves chronology, reassessment, and override [1-6]. This risk classification is better expressed by formal logic in prose than by Boolean algebra

III. Boolean Algebra in Treatment Decisions

Escalation as a consequence of VHR

The third major result is that treatment decisions follow the risk architecture rather than being free-standing equations. Treatment escalation is best represented as a consequence of VHR:

$$Escalation = VHR$$

Expanded:

$$Escalation = BCR::ABL1 \vee KMT2A \vee Hypodiploid \\ \vee MRD_{persistent} \vee PoorEarlyResponse \\ \vee Relapse$$

Targeted therapy as a lesion-defined branch

Certain treatment branches map directly from disease biology. The clearest example is tyrosine kinase inhibitor (TKI) therapy in BCR::ABL1-positive disease:

$$TKI\ Branch = BCR::ABL1$$

This relation correctly identifies the lesion-defined nature of TKI selection without implying that the remainder of treatment is otherwise simple [1-4].

Hematopoietic stem cell transplantation (HSCT) consideration as a VHR-linked management branch

Unlike the earlier nonsensical formula that made HSCT a direct Boolean output identical to the whole treatment system, transplant should be represented more carefully as a management branch that opens under VHR conditions in an eligible patient:

$$HSCT\ Consideration = VHR \wedge TransplantEligible$$

This is still simplified, but it is clinically meaningful. It acknowledges that VHR opens the transplant branch while leaving room for real-world

determinants such as age, comorbidity, donor access, remission status, protocol, and patient preference. (1-4,69).

System-level treatment architecture

Because treatment in ALL is branching rather than monolithic, it is better described as a structured architecture than as one overloaded equation. This can also be done in prose by formal logic:

1. If BCR::ABL1 is present, a TKI branch is activated.
2. If VHR is present, intensified or redirected therapy is considered.
3. If VHR is present and the patient is an appropriate transplant candidate, the HSCT consideration branch opens.
4. If VHR is absent, management continues according to the baseline-risk pathway and protocol-specific response adaptation.

This formulation is clearer than forcing all treatment into a single conjunctive symbolic equation.

Discussion

This manuscript shows that Boolean algebra can represent acute lymphoblastic leukemia (ALL) most effectively when it is used to model diagnostic concordance, staged risk assignment, and branching treatment decisions rather than when it is forced into a single simplified formula. That point is especially important because earlier versions of the manuscript suffered from exactly that problem: over compression produced equations that looked elegant but were clinically misleading.

The first major implication concerns diagnosis. The diagnostic section now reflects what current ALL classification actually requires: concordance among morphology, flow cytometry, cytogenetics, and molecular genetics [1-4]. A manuscript on Boolean algebra in ALL that omitted one of these domains would fail to model the disease as it is currently diagnosed. Morphology provides the acute leukemia context and the marrow-based distinction from lymphoblastic lymphoma (LBL). Flow cytometry defines lineage and precursor state. Cytogenetics and molecular genetics place the case within a modern biologic subtype framework. Boolean algebra is especially useful here because it forces these domains to be written as co-required pillars rather than as loosely connected supporting tests.

The second implication concerns diagnostic hierarchy. The distinction between ALL and LBL remains operationally important even if the diseases lie on a biologic spectrum. Boolean formalization makes that first gate explicit. That matters because every later decision depends on the disease state that is chosen at the outset. The same is true for lineage assignment. By separating B-lineage diagnosis from CD19 targetability, the model avoids conflating diagnostic identity with therapeutic eligibility. This is not merely a technical correction; it has direct clinical meaning in the era of antigen-directed immunotherapy [15]

The third implication concerns risk. The present manuscript no longer treats risk as a one-line final equation. That was the core conceptual problem in the earlier flawed formulation. Real ALL risk assignment proceeds in stages. Baseline risk is assigned from presenting variables, but treatment response—especially persistent MRD—can subsequently reclassify the disease. The results support a model in which persistent MRD is part of VHR, not separate from it. This is more aligned with contemporary care, where MRD-driven risk adaptation is one of the

dominant determinants of therapeutic strategy [1-6]. Formal logic in prose is the better and more flexible matter for that.

The fourth implication concerns treatment. Treatment architecture in ALL is not one single Boolean sentence. It is a branching system. A targeted-therapy branch may open because of BCR::ABL1. An escalation branch may open because of VHR. A transplant-consideration branch may open because VHR is present in a patient who is actually transplant-eligible. Expressed this way, Boolean algebra clarifies the branching architecture of care rather than distorting it through artificial compression.

The methodological contribution of the prior Zugmaier Boolean-algebra publications also deserves emphasis. Those publications established the feasibility of applying mathematics to hematologic disease definition, immunophenotyping, cytogenetic risk classification, laboratory diagnostics, and differential diagnosis. [7-13]. Their relevance here is methodological rather than evidentiary: they show that this manuscript is part of a broader mathematics - based approach to diagnostic medicine rather than an isolated conceptual exercise.

The present model still has limitations. Biological systems are continuous rather than binary, antigen expression may be partial or heterogeneous, MRD thresholds differ by assay and protocol, and real treatment decisions depend on variables that cannot be neatly reduced to 0 or 1, including frailty, organ function, donor availability, and patient preference. Boolean algebra therefore cannot replace clinical judgment. What it can do is expose the algebraic skeleton of decision-making and make explicit where certain variables are required, where they are alternatives, and where they function as overrides.

In that sense, Boolean algebra is most useful in ALL when it is used not to oversimplify the disease, but to make its hidden structure visible. The revised manuscript supports that conclusion by treating diagnosis as four-pillar concordance, risk as a staged process, VHR as an override state, and treatment as a branching architecture rather than a single symbolic artifact.

Conclusion

Boolean algebra provides a structured and interpretable framework for modeling diagnostics, risk stratification, and treatment decisions in acute lymphoblastic leukemia (ALL) when applied with clinical rigor. Diagnosis is best represented as concordance among morphology, flow cytometry, cytogenetics, and molecular genetics. Risk is best represented as a staged process that begins with baseline assignment and may be overridden by very high risk (VHR) reclassification. Persistent measurable residual disease (MRD) belongs within VHR when clinically decisive. Treatment decisions are best represented as branching consequences of biologic subtype and treatment-relevant risk state rather than as one overloaded equation. Used in this way, Boolean algebra clarifies rather than distorts the structure of modern ALL care.

Abbreviations

ALL = acute lymphoblastic leukemia

LBL = lymphoblastic lymphoma

B-ALL = B-cell acute lymphoblastic leukemia

T-ALL = T-cell acute lymphoblastic leukemia

BM = bone marrow

WBC = white blood cell count

MRD = measurable residual disease

VHR = very high risk

CNS = central nervous system

HSCT = hematopoietic stem cell transplantation

TKI = tyrosine kinase inhibitor

CD = cluster of differentiation

TdT = terminal deoxynucleotidyl transferase

MPO = myeloperoxidase

PAX5 = paired box 5

AYA = adolescent and young adult

ELN = European LeukemiaNet

NCCN = National Comprehensive Cancer Network

BCR: ABL1 = fusion gene associated with the Philadelphia chromosome

KMT2A = lysine methyltransferase 2A

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DOI: [10.31579/2690-1919/631](https://doi.org/10.31579/2690-1919/631)

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