

Modern Treatment Methods for Acute Leukemias: Chemotherapy, Targeted Therapy, and Cellular Immunotherapy

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ABSTRACT

Acute leukemias are among the most severe malignant diseases of the hematopoietic system. Despite significant progress in diagnosis and treatment, they remain an important medical and social problem. Conventional combination chemotherapy remains the foundation of treatment; however, its use is limited by toxicity, the development of drug resistance, and the risk of relapse. In recent years, the treatment of acute leukemias has changed considerably owing to the introduction of molecular genetic diagnostics, targeted drugs, monoclonal and bispecific antibodies, CAR T-cell therapy, and hematopoietic stem cell transplantation.

The aim of this study is to analyze modern treatment methods for acute lymphoblastic leukemia and acute myeloid leukemia, as well as to evaluate their advantages, limitations, and prospects. The paper examines conventional chemotherapy, tyrosine kinase inhibitors, FLT3 and IDH inhibitors, drugs targeting the BCL-2 protein, monoclonal and bispecific antibodies, antibody–drug conjugates, CAR T-cell therapy, and hematopoietic stem cell transplantation. Particular attention is paid to the importance of measurable residual disease and treatment personalization.

The modern approach to the treatment of acute leukemias is based on the combination of clinical, immunological, and molecular genetic characteristics of the disease. A promising direction is the transition from universal chemotherapy to risk-adapted treatment combinations, which may improve treatment effectiveness and reduce toxicity.

Keywords: acute leukemia, acute lymphoblastic leukemia, acute myeloid leukemia, chemotherapy, targeted therapy, immunotherapy, CAR T cells, measurable residual disease, transplantation.

Introduction

Acute leukemias are a group of clonal malignant diseases of hematopoietic tissue that develop as a result of the genetic transformation of hematopoietic precursor cells. This transformation disrupts normal cell differentiation and maturation, while the abnormal clone acquires the ability to proliferate and survive uncontrollably. Tumor cells accumulate in the bone marrow, replace normal hematopoietic lineages, and may infiltrate the peripheral blood, lymph nodes, liver, spleen, central nervous system, and other organs.

The main types of acute leukemia are acute lymphoblastic leukemia and acute myeloid leukemia. Acute lymphoblastic leukemia occurs more frequently in children and is the most common hematological malignancy in childhood. Acute myeloid leukemia is diagnosed more often in adults, although it can also occur in children

and adolescents. These diseases differ in the origin of the malignant cells, molecular abnormalities, clinical course, and response to treatment.

For many decades, intensive combination chemotherapy was the main treatment method for acute leukemias. It has led to a significant improvement in survival, particularly among children with acute lymphoblastic leukemia. However, chemotherapy is not completely specific: it damages not only malignant cells but also normal cells that divide rapidly. This may result in myelosuppression, infectious complications, organ damage, impaired fertility, and other early and late effects.

Another major problem is relapse. Even after morphological remission has been achieved, small numbers of leukemic cells may remain in the body. Methods for detecting measurable residual disease are used to identify these cells. A positive result may indicate an increased risk of relapse and the need to modify the treatment strategy.

Modern treatment of acute leukemias is developing toward a personalized approach. The choice of drugs is increasingly determined not only by the type of disease and the patient's age, but also by the presence of specific genetic mutations, the expression of tumor antigens, the response to initial treatment, and the level of measurable residual disease. FLT3 and IDH inhibitors are used for certain molecular subtypes of acute myeloid leukemia, while tyrosine kinase inhibitors are used for BCR::ABL1-positive acute lymphoblastic leukemia. In relapsed and refractory B-cell acute lymphoblastic leukemia, bispecific antibodies, antibody–drug conjugates, and CAR T-cell therapy are becoming increasingly important.

Relevance of the Study

- Acute leukemias progress rapidly without timely treatment.
- Standard chemotherapy is associated with significant toxicity.
- Some patients develop primary resistance or relapse.
- Molecular subtypes of the disease require different treatment approaches.
- Modern treatment methods require an assessment of the genetic and immunological characteristics of the tumor.
- Some innovative drugs are currently available only in specialized centers and within clinical trials.

Aim of the Study

To analyze modern treatment methods for acute leukemias, determine the role of chemotherapy, targeted therapy, immunotherapy, and transplantation in current clinical practice, and examine their advantages and limitations.

Objectives of the Study

1. To characterize the biological features of acute leukemias.
2. To examine the role of conventional chemotherapy.
3. To analyze the main types of targeted therapy.
4. To describe modern methods of immunotherapy.
5. To examine the possibilities and limitations of CAR T-cell therapy.
6. To determine the significance of hematopoietic stem cell transplantation.
7. To analyze the role of measurable residual disease in treatment selection.
8. To evaluate the prospects for the development of modern acute leukemia therapy.

Materials and Methods

This study was conducted as an analytical review of contemporary scientific literature. Official clinical materials from the National Cancer Institute, publications on the diagnosis and treatment of acute leukemias, reviews of molecular classification, and studies of new immunotherapy methods were used in the preparation of the article.

The literature search was conducted using the following keywords: acute leukemia, acute lymphoblastic leukemia, acute myeloid leukemia, targeted therapy, immunotherapy, CAR T-cell therapy, bispecific antibodies, venetoclax, FLT3 inhibitors, IDH inhibitors, measurable residual

disease, and hematopoietic stem cell transplantation.

The analysis included publications concerning the treatment of adults and children with acute leukemias; molecular genetic classification; targeted drugs; monoclonal and cellular immunotherapy; the clinical use of transplantation; and the evaluation of treatment effectiveness and toxicity.

Biological Features of Acute Leukemias

Acute leukemias develop as a result of the clonal transformation of a hematopoietic precursor cell. A genetic abnormality blocks normal cell maturation and increases the ability of the abnormal cells to proliferate. As a result, blast cells accumulate in the bone marrow and displace normal erythroid, granulocytic, and megakaryocytic lineages.

Suppression of normal hematopoiesis causes the main clinical syndromes of the disease. Reduced erythropoiesis leads to anemia, a low platelet count causes a hemorrhagic syndrome, and a decreased number of functionally mature neutrophils increases the risk of infections.

Leukemic cells can infiltrate various tissues. Involvement of the central nervous system may cause headache, vomiting, seizures, visual disturbances, and cranial nerve impairment. Infiltration of the liver and spleen leads to their enlargement. Involvement of the bones and periosteum may cause severe pain, particularly in children.

The modern understanding of acute leukemias is based on recognition of their biological heterogeneity. Even patients with the same morphological diagnosis may have different genetic abnormalities and different responses to treatment. Therefore, the optimal treatment strategy requires the combination of morphological, immunological, and molecular genetic data.

Acute Lymphoblastic Leukemia

Acute lymphoblastic leukemia develops from lymphoid precursor cells. Depending on the immunophenotype, B-cell and T-cell subtypes are distinguished.

B-cell acute lymphoblastic leukemia is the most common form, particularly in childhood. It may be associated with various genetic rearrangements, including ETV6::RUNX1, TCF3::PBX1, BCR::ABL1, and other abnormalities. T-cell acute lymphoblastic leukemia is more frequently associated with mediastinal lymphadenopathy and may cause symptoms resulting from compression of thoracic organs. Treatment depends on the patient's age, white blood cell count, genetic characteristics, and response to induction therapy.

Acute Myeloid Leukemia

Acute myeloid leukemia develops from cells of the myeloid lineage. It includes various molecular subtypes that may differ in prognosis and sensitivity to treatment.

Mutations and rearrangements involving the FLT3, NPM1, CEBPA, IDH1, IDH2, and other genes are particularly important. Identifying these abnormalities makes it possible to classify patients into prognostic groups and use targeted drugs.

Acute promyelocytic leukemia is considered a distinct form. It is associated with the PML::RARA rearrangement and carries a high risk of severe coagulopathy. At the same time, it is one of the most successful examples of molecularly targeted treatment, because the use of all-trans retinoic acid and arsenic trioxide can achieve high remission rates.

Conventional Chemotherapy

Despite the development of innovative methods, chemotherapy remains central to the treatment of acute leukemias. It may be used as an independent treatment or in combination with targeted drugs, immunotherapy, and transplantation.

Chemotherapy for Acute Lymphoblastic Leukemia

Treatment of acute lymphoblastic leukemia usually consists of several stages:

9. Remission induction.
10. Consolidation.
11. Intensification.
12. Maintenance therapy.

13. Central nervous system prophylaxis.

The aim of induction therapy is to achieve complete remission. During this phase, a combination of several drugs is used to target different stages of the leukemic cell life cycle. After remission has been achieved, consolidation therapy is administered to eliminate residual tumor cells that cannot be detected by routine microscopic examination. Intensification is then used to further reduce the risk of relapse.

Maintenance therapy is usually longer and less intensive. It is necessary to prevent the reaccumulation of leukemic cells. Central nervous system involvement may be clinically silent; therefore, prophylaxis against central nervous system leukemia is an important part of treatment. This may include intrathecal administration of drugs and, in selected situations, systemic therapy capable of crossing the blood–brain barrier.

Chemotherapy for Acute Myeloid Leukemia

Induction and consolidation chemotherapy are traditionally used in acute myeloid leukemia. In patients who are able to tolerate intensive treatment, regimens containing cytarabine and anthracyclines may be used. The aim of induction therapy is to achieve complete remission. Consolidation is then administered to eliminate the residual leukemic clone. Allogeneic hematopoietic stem cell transplantation may be considered for patients at high risk of relapse.

In older patients and individuals with severe comorbidities, intensive chemotherapy may be contraindicated. In such cases, less intensive regimens are used, including hypomethylating agents in combination with venetoclax.

Limitations of Chemotherapy

The main limitation of chemotherapy is its lack of absolute specificity. Chemotherapeutic drugs damage malignant cells but also affect normal tissues with a high rate of cell division.

- Neutropenia.
- Thrombocytopenia.
- Anemia.
- Febrile neutropenia.
- Mucositis.
- Nausea and vomiting.
- Alopecia.
- Liver injury.
- Kidney injury.
- Cardiotoxicity.
- Impaired reproductive function.
- Secondary malignancies.

Late effects are particularly important in children. Intensive treatment may affect growth, the endocrine system, cognitive development, and fertility. Therefore, modern protocols aim to adjust treatment intensity according to the individual patient's risk.

Targeted Therapy

Targeted therapy is treatment directed at a specific molecular target in a tumor cell. The target may be a mutated enzyme, receptor, signaling protein, or cell-surface antigen. Its advantage is greater selectivity compared with conventional chemotherapy. However, it is effective only when the corresponding target is present in the tumor cells.

Tyrosine Kinase Inhibitors

In some leukemias, pathologically active tyrosine kinases are produced. The best-known example is BCR::ABL1, which is associated with the Philadelphia chromosome. BCR::ABL1 activates signaling pathways that stimulate tumor-cell growth and survival. Tyrosine kinase inhibitors block this enzyme and suppress proliferation of the abnormal clone.

- Imatinib.
- Dasatinib.
- Nilotinib.
- Ponatinib.
- Other agents.

Tyrosine kinase inhibitors are primarily important in chronic myeloid leukemia, but they are also used in BCR::ABL1-positive acute lymphoblastic leukemia. In acute lymphoblastic leukemia, they are usually administered in combination with chemotherapy or immunotherapy. Mutations that cause drug resistance may develop; in such cases, molecular monitoring and modification of treatment are required.

FLT3 Inhibitors

FLT3 mutations occur in a proportion of patients with acute myeloid leukemia. They cause persistent activation of a signaling pathway that stimulates leukemic-cell proliferation and survival. FLT3 inhibitors, including midostaurin and quizartinib, are used for patients with FLT3-altered acute myeloid leukemia. They may be administered during induction, consolidation, after transplantation, or in relapsed or refractory disease. Treatment effectiveness depends on the type of mutation, cellular sensitivity, depth of remission, and the possibility of combining treatment with other methods.

IDH Inhibitors

IDH1 and IDH2 mutations lead to the production of abnormal metabolites that impair cell maturation. IDH inhibitors partially restore the ability of tumor cells to differentiate. Ivosidenib and enasidenib may be used in selected patients. Their use depends on the mutation type, patient age, general condition, and previous treatment. A major limitation is the need for prior genetic testing.

BCL-2 Inhibition

The BCL-2 protein participates in the suppression of apoptosis. Tumor cells may use this mechanism to survive and resist treatment. Venetoclax blocks BCL-2 and increases the likelihood of leukemic-cell death. In acute myeloid leukemia, it is mainly used in combination with hypomethylating agents or low-dose cytarabine in patients who cannot tolerate intensive induction.

- Treatment of older patients.
- Use when intensive chemotherapy is not possible.
- Activity in some relapsed forms.
- Combination with other drugs.

Important complications include severe neutropenia, thrombocytopenia, infections, tumor lysis syndrome, and the need for dose adjustments because of drug interactions.

Immunotherapy

Immunotherapy uses the patient's immune system to recognize and destroy tumor cells. Unlike conventional chemotherapy, it acts not only directly on the tumor cell but also by restoring or enhancing the antitumor immune response.

Monoclonal Antibodies

Monoclonal antibodies bind to a specific antigen on the surface of a tumor cell. After binding, the cell may be destroyed through activation of the immune system, complement-dependent cytotoxicity, or other mechanisms. Rituximab targets CD20 and is used in B-cell malignancies when tumor cells express this antigen.

- Relatively high specificity.
- The possibility of combination with chemotherapy.
- Action through immune mechanisms.
- Use when the relevant antigen is present.

Limitations include limited effectiveness in the absence of the antigen, infusion reactions, immunodeficiency, infections, and possible development of resistance.

Bispecific Antibodies

Bispecific antibodies bind simultaneously to a tumor cell and a T lymphocyte. In this way, they bring the two cells together and activate the T cell to destroy the tumor. Blinatumomab binds CD19 on the B lymphoblast and CD3 on the T lymphocyte. It is used in certain forms of B-cell acute lymphoblastic leukemia, particularly in relapse, refractory disease, or measurable residual disease.

- Direct activation of T lymphocytes.
- No need to remove and genetically modify the patient's cells.
- Potential use in measurable residual disease.
- Activity in some relapsed forms of acute lymphoblastic leukemia.

Major complications include cytokine release syndrome, fever, neurological toxicity, infections, and the need for continuous infusion in some treatment regimens.

Antibody–Drug Conjugates

Antibody–drug conjugates combine a specific antibody with a cytotoxic substance. The antibody recognizes an antigen on the tumor cell and delivers the toxic component into the cell. Inotuzumab ozogamicin targets CD22 and may be

used in some cases of relapsed or refractory B-cell acute lymphoblastic leukemia. Its use may be complicated by myelosuppression and liver injury, particularly in patients who are candidates for hematopoietic stem cell transplantation.

CAR T-Cell Therapy

CAR T-cell therapy is one of the most important achievements in modern hemato-oncology. It is a form of cellular immunotherapy based on the genetic modification of the patient's own T lymphocytes.

Main Stages of Therapy

14. Leukapheresis and collection of the patient's T lymphocytes.
15. Isolation and activation of the cells.
16. Insertion of the chimeric antigen receptor gene.
17. Expansion of the modified cells.
18. Quality control of the cellular product.
19. Lymphodepleting chemotherapy.
20. Intravenous administration of CAR T cells.
21. Patient monitoring and management of complications.

The chimeric antigen receptor consists of an antigen-recognition region and intracellular signaling domains that activate the T lymphocyte. In B-cell acute lymphoblastic leukemia, CD19 is the most common target. After infusion, CAR T cells recognize CD19-positive cells and destroy them.

Indications

- Relapsed B-cell acute lymphoblastic leukemia.
- Refractory disease.
- Insufficient response to chemotherapy.
- Multiple relapses.
- Selected cases in which transplantation has failed.

Advantages

- Treatment after failure of standard chemotherapy.
- A strong antitumor response.
- Activity against cells resistant to conventional drugs.
- Potentially long-term immune control.
- The possibility of achieving remission in patients with severe relapse.

Limitations and Complications

- Cytokine release syndrome.
- Neurotoxicity.
- Prolonged B-cell aplasia.
- Hypogammaglobulinemia.
- Infections.
- Cytopenias.
- Relapse with loss or alteration of the tumor antigen.

Cytokine release syndrome results from massive activation of immune cells and the release of inflammatory mediators. It may present with high fever, hypotension, tachycardia, hypoxia, and organ dysfunction. Neurotoxicity may include headache, confusion, speech impairment, seizures, and decreased consciousness. Therefore, CAR T-cell therapy is performed in specialized centers experienced in managing immune-related complications. Additional limitations include high cost, the complexity of manufacturing an individualized cellular product, and the need for specialized laboratory infrastructure.

Hematopoietic Stem Cell Transplantation

Allogeneic hematopoietic stem cell transplantation remains an important treatment method for patients with a high risk of relapse or refractory disease. Its therapeutic effect is associated with two mechanisms: replacement of the abnormal hematopoietic system with healthy donor cells and the graft-versus-leukemia effect, in which donor immune cells recognize and destroy residual tumor cells.

Main Stages

- Selection of a compatible donor.
- Assessment of the patient's condition.
- Conditioning therapy.
- Infusion of stem cells.
- Prevention of graft-versus-host disease.
- Anti-infective prophylaxis.
- Long-term follow-up.

Indications

- High risk of relapse.
- Adverse genetic abnormalities.
- Failure to achieve deep remission.
- Relapsed acute myeloid leukemia.

- Refractory disease.
- Selected forms of acute lymphoblastic leukemia after achieving a second remission.

The decision is individualized. Physicians must compare the expected benefit of transplantation with the risk of severe complications.

Complications

- Graft-versus-host disease.
- Severe viral, bacterial, and fungal infections.
- Liver injury.
- Lung injury.
- Mucositis.
- Infertility.
- Secondary malignancies.
- Chronic immune deficiency.

Measurable Residual Disease and Treatment Personalization

Measurable residual disease is a small population of leukemic cells that remains after treatment. It may not be detectable by standard morphological methods but can be identified using highly sensitive flow cytometry, PCR, and sequencing. Morphological remission means that obvious blast cells are absent on bone marrow examination; however, it does not always mean complete elimination of the tumor clone.

Detection Methods

- Multiparameter flow cytometry.
- PCR for specific genetic rearrangements.
- Digital PCR.
- Highly sensitive sequencing.

Clinical Significance

If measurable residual disease persists after induction or consolidation, the risk of relapse may be higher. In this situation, physicians may consider intensification of chemotherapy, addition of immunotherapy, use of a targeted drug, transplantation, or participation in a clinical trial. When measurable residual disease is absent, treatment intensity may sometimes be reduced in patients with favorable characteristics, thereby reducing toxicity without compromising antitumor efficacy.

Thus, measurable residual disease is an important tool for treatment personalization. It helps move from identical treatment for all patients toward individually adapted protocols.

Comparative Analysis of Modern Methods

Method	Main target or principle	Main advantages	Most important limitations
Combination chemotherapy	Rapidly dividing cells	Broad effectiveness; established protocols	Myelosuppression and systemic toxicity
Tyrosine kinase inhibitors	BCR::ABL1 and related kinases	High effectiveness with specific genetic abnormalities	Appropriate molecular target required
FLT3 inhibitors	Mutated or activated FLT3	Treatment of a distinct AML subtype	Resistance and adverse effects
IDH inhibitors	IDH1 or IDH2	Targets a metabolic mechanism	Only applicable with specific mutations

Venetoclax	BCL-2 protein	Suitable for patients unable to tolerate intensive therapy	Neutropenia, infections, tumor lysis syndrome
Monoclonal antibodies	Tumor antigens	Immunological specificity	Dependent on antigen expression
Bispecific antibodies	T cell and tumor cell	Rapid activation of immune response	Cytokine release syndrome and neurotoxicity
CAR T-cell therapy	Genetically modified T cells	Effective in relapse and resistance	Cost, complexity, and severe complications
Transplantation	Replacement of hematopoiesis and graft-versus-leukemia effect	Potential for long-term remission	Infections, graft-versus-host disease, toxicity

Treatment of Children

Childhood leukemias require a special approach. Because the child's body continues to grow and develop, toxic treatment effects may have consequences years after therapy has ended.

Treatment of childhood acute lymphoblastic leukemia is usually based on protocols that take into account age, white blood cell count, immunophenotype, genetic abnormalities, central nervous system involvement, early treatment response, and measurable residual disease.

Modern protocols aim not only to improve survival but also to reduce late toxicity. In patients with a favorable prognosis, treatment intensity may be reduced, whereas targeted or immunological therapy may be added in high-risk cases. Long-term follow-up should assess growth and physical development, thyroid function, sexual development, cardiovascular health, cognitive function, psychological well-being, reproductive function, and the risk of secondary malignancies.

Future Research Perspectives

Despite considerable progress, modern acute leukemia therapy still has several unresolved problems. Leukemic cells may acquire drug resistance through new mutations, changes in antigen expression, activation of alternative signaling pathways, and interactions with the bone marrow microenvironment.

Relapse remains one of the main causes of treatment failure. Current approaches include repeat chemotherapy, immunotherapy, targeted drugs, CAR T-cell therapy, and transplantation. Research aims to identify patients at high risk of relapse before clinical relapse develops by using molecular monitoring and measurable residual disease assessment.

Reducing adverse effects without reducing efficacy is a major goal, particularly in pediatrics. Researchers are studying less toxic combinations, reduced treatment intensity for low-risk patients, prevention of cardiotoxicity, fertility preservation, neuroprotective strategies, individualized dosing, and drugs with narrower mechanisms of action.

Innovative therapies require expensive drugs, genetic testing, and specialized centers.

Important healthcare priorities include development of laboratory diagnostics, access to basic blood testing, creation of specialized centers, medical staff training, participation in international clinical trials, and adequate anti-infective support.

Promising areas include off-the-shelf cellular products, multi-antigen CAR T cells, natural killer cells, bispecific CAR constructs, gene editing, and combinations of cellular and targeted therapies. Many of these approaches remain investigational and should not yet be considered standard treatment for all patients.

Discussion

The analysis shows that the development of acute leukemia treatment is occurring in three main directions: molecular personalization, enhancement of the immune response, and reduction of toxicity.

The first direction involves genetic profiling. Identifying FLT3, IDH1, IDH2, BCR::ABL1, and other abnormalities allows clinicians to select drugs that target specific pathological mechanisms. The second direction involves the immune system. Bispecific antibodies and CAR T cells direct T lymphocytes against tumor cells and are particularly important in relapse and resistance. The third direction is reduction of toxicity. Modern treatment should be evaluated not only by remission and survival rates but also by quality of life, preservation of cognitive function and fertility, and prevention of late complications.

Innovative methods do not completely replace conventional treatment. Chemotherapy remains the basis of many treatment protocols, and the best results are often achieved through rational combinations of treatment methods. For example, a patient with acute myeloid leukemia may receive chemotherapy combined with an FLT3 inhibitor; a patient with BCR::ABL1-positive acute lymphoblastic leukemia may receive a tyrosine kinase inhibitor with chemotherapy or immunotherapy; and a patient with relapsed B-cell acute lymphoblastic leukemia may be considered for blinatumomab, inotuzumab, or CAR T-cell therapy. Thus, treatment is determined by the biology of the disease, not only by its name.

Conclusion

Acute leukemias are biologically heterogeneous diseases that require an individualized approach. Conventional chemotherapy remains the foundation of treatment, but its effectiveness is limited by toxicity and the possibility of resistance.

Targeted therapy has made it possible to act on specific molecular abnormalities. Tyrosine kinase inhibitors are used in BCR::ABL1-positive disease, FLT3 inhibitors in FLT3-altered acute myeloid leukemia, IDH inhibitors in patients with corresponding mutations, and venetoclax mainly in combination with less intensive therapy for patients unable to tolerate standard induction.

Immunotherapy has significantly expanded treatment options for relapsed and refractory disease. Monoclonal antibodies, bispecific antibodies, and antibody–drug conjugates allow targeted action against tumor cells. CAR T-cell therapy has become an important treatment for selected forms of B-cell acute lymphoblastic leukemia, although its use is limited by cost, manufacturing complexity, and the risk of severe complications.

Hematopoietic stem cell transplantation remains important in patients at high risk of relapse and in refractory disease. The decision should be individualized according to disease biology, the patient's condition, and the risk of complications.

Measurable residual disease is one of the main tools for treatment personalization. It helps assess the depth of response, identify patients at high risk of relapse, and modify treatment in a timely manner.

Modern treatment of acute leukemias is moving toward combinations of chemotherapy, targeted drugs, and immunotherapy. Future research should focus on improving efficacy, overcoming drug resistance, reducing early and late toxicity, improving quality of life, and increasing access to innovative treatments.

Therefore, modern leukemia treatment cannot be reduced to a single drug or method. The most effective strategy is a comprehensive approach based on accurate diagnosis, molecular characterization of the tumor, assessment of measurable residual disease, and individualized risk stratification.

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