guided by a combination of mutational status and comorbidities. Specific mutations confer resistance to certain TKIs, making mutation-directed sequencing essential. At the same time, patient comorbidities such as cardiovascular, pulmonary, or metabolic disease influence drug tolerability and safety, thereby shaping the optimal therapeutic choice [1,7]. **Beyond TKIs:** For patients failing multiple TKIs, allogeneic hematopoietic stem cell transplantation (allo-HSCT) remains the only potentially curative approach, particularly in younger and high-risk patients [1,2]. Novel strategies under investigation include rational TKI combinations (e.g., asciminib plus ponatinib), immunotherapeutic approaches, and targeted inhibition of epigenetic regulators [8]. **Conclusion:** Refractory CML reflects the biological and clinical complexity of disease progression beyond BCR::ABL1 dependence. While ponatinib and asciminib have redefined therapeutic opportunities, additional high-risk mutations highlight the need for precision medicine strategies. Tailored TKI sequencing, integration of comorbidity profiles, and timely transplantation remain central pillars, while ongoing translational research promises to expand future options [7,8].

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Abstract 009

HYPERCOAGULABILITY: ETIOLOGY, DIAGNOSIS AND TREATMENT PRINCIPLES

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Thrombosis occurs when the delicate balance between prothrombotic and anticoagulant forces is impaired. It usually develops due to multiple factors. When multiple risk factors come together, the anticoagulant systems cannot resist procoagulant forces and thrombosis may develop as a result. Thrombosis due to hypercoagulability is usually seen clinically as venous thromboembolism (VTE) and rarely as arterial thrombosis. VTE can be seen as deep vein thrombosis (DVT) or pulmonary embolism. DVT most often manifests itself in the legs and rarely in the abdominal or intra-pelvic veins. The hereditary or acquired factors are involved in the etiology of venous thromboembolism. Clinically, VTE is observed in those who are due to hereditary factors, while venous or arterial thromboses may be observed in those who are due to acquired causes. Hypercoagulability due to acquired causes is observed more often (70%) and they have a greater risk of thrombosis. Venous thromboembolism is reported to occur in 1/10,000 people per year under the age of 40 and 1/1000 people per year over the age of 75. Hereditary thrombophilia causes are rare in the population. Although different rates are reported according to the world geography, The R506Q mutation in coagulation factor V, also known as the Factor V Leiden (FVL) mutation is the most common among them (3-8%). It is rare in far east countries. FVL mutation is the most common cause among hereditary hypercoagulabilities (50%). Clinically, young age, idiopathic thrombosis, thrombosis in an unusual place (upper extremity, mesenteric vein, portal vein, renal vein, cerebral vein) are noteworthy. Recurrence of thrombosis and a family history of venous thromboembolism are common. Since the findings are not specific in the diagnosis of venous thromboembolism, the patient's medical history, family history and examination findings should be evaluated together. Determination of thrombosis risk scores, D-Dimer test, blood chemistry, lung X-ray and ECG are included as the first examinations in the patient. In patients with a negative D-Dimer test, a further examination is usually not needed. The subject of which tests to perform and when to perform in VTE cases requires expertise. In cases of idiopathic thrombosis, occurring at a young age, or recurrent, genetic or coagulation tests may be planned. Since test results may be misleading during the acute thrombosis period, it is more appropriate to schedule the tests a few weeks later or after the end of treatment. In patients with a high thrombosis risk score and elevated D-dimer levels, extremity vein Doppler ultrasonography and computed pulmonary angiography are used as imaging studies. Oral or parenteral anticoagulants are used in the treatment of venous thromboembolism. These include low molecular weight heparin, FXa inhibitors (apixaban, rivaroxaban), and vitamin K antagonist (warfarin). The most commonly used are low-molecular-weight heparin, FXa inhibitors (apixaban, rivaroxaban), and vitamin K antagonists (warfarin). Anticoagulant therapy should last at least 3 months, after which patients should be evaluated based on their risk status. Anticoagulant therapy should be longer-term in patients with ongoing diseases or conditions that trigger thrombosis (such as antiphospholipid syndrome, active autoimmune disease, cancer). Patients should be carefully monitored for bleeding during anticoagulant therapy. Thrombolytic or interventional treatments may be administered to patients presenting with acute heart failure and hypotension. Patients should continue to be monitored after anticoagulant therapy, and physical therapy should be provided for patients with postthrombotic syndrome.

Key words: Hypecoagulability, venous tromboembolism, anticoagulant therapy.

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Abstract 010

THE PLACE OF IMMUNOTHERAPY IN ALL

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In patients with acute lymphoblastic leukemia (ALL), although 80-90% of adult patients achieve a complete response (CR), cure rates are only 40% with initial treatment and 10%-20% with subsequent salvage treatments. Ten percent of patients are refractory to initial treatment, and 40%-70% relapse. Allo-HCT is the standard of care for a fit and eligible group. Immunotherapies are an important choice in improving treatment success and reducing side effects. The primary immunotherapies include bispecific antibodies (BsAbs), antibody-drug conjugates, CAR T-cell, and CAR NK-cell therapies. Blinatumomab activates T cells by binding to