and Kidd blood group systems were discovered. Today, there are over 360 different blood group antigens within 48 blood group system. Landsteiner won the Nobel Prize in 1930 for his discovery of blood groups. In 1907, it was recognized that blood group compatibility between donor and patient was necessary, and the first cross-matching tests were performed by Ruben Ottenberg. With these studies, Ottenberg demonstrated that the O blood group is a universal donor. A milestone in blood banking was the use of sodium citrate, an anticoagulant, in blood transfusions (1914-1915) (Hustin, Agote, Levishon). Prior to this discovery, transfusions were performed by transferring blood from the donor to the patient using syringes or vascular anastomoses. However, with the ability to store blood without clotting, transfusions began to be performed by transferring blood from the donor into a glass bottle containing citrate and then to the patient. The world's first blood bank was established in England in 1921 by Oliver Percy. Later, with the addition of dextrose, phosphate, adenine, and mannitol mixtures, blood could be stored for up to 42 days in four-degree blood refrigerators. In 1930, Russian Shamov performed the first transfusion of cadaver blood to a living person. In the following years, transfusions were performed on 2,500 people using this method. In 1935, the International Society of Blood Transfusion (ISBT) was founded. At its 1937 congress, the ISBT adopted the ABO terminology for blood grouping. In 1950, plastic blood bags were developed. In 1953, blood components were obtained using a refrigerated centrifuge method. In 1968, the first apheresis devices were developed. In Turkey, the first human-to-human transfusion was performed at Haydarpaşa Numune Hospital in 1932. Starting in 1945, small blood units were established in some hospitals. In 1957, Red Crescent blood banks were established first in Ankara and then in Istanbul. In 1983, Law No. 2857 on Blood and Blood Products was enacted in Turkey. In ....., a new blood law and related regulations were enacted in light of scientific developments. Accordingly, Red Crescent Regional Blood Centers and Hospital Transfusion Centers were established. Guidelines were developed. Mandatory screening tests were initiated for diseases transmitted through transfusion, including HBV, syphilis, malaria, HIV, and most recently HCV. In 1996, the Blood Centers and Transfusion Association (KMTD) was established. In 1997, a donor screening form was created and its use was made mandatory throughout Turkey. When KMTD was established, whole blood usage in Turkey was over 95%. KMTD, in collaboration with the Ministry of Health, held 118 educational meetings in 74 provinces, explaining blood components, transfusion indications and complications, and blood bank-clinic relationships. As a result, component usage was adopted throughout the country. Annual courses and conferences were held to keep pace with developments worldwide and in Turkey. Recently, training has focused particularly on Hemovigilance (blood monitoring system) and Patient Blood Management. Currently, components are used not only for component requirements but also for various treatment options. For this purpose, platelets, mesenchymal stem cells, and plasma are used in regenerative medicine and wound healing. In light of scientific and technological developments, the following developments are expected in the field of transfusion in the future: Artificial blood (oxygen-carrying hemoglobin derivatives and engineered products), Universal blood production and conversion of erythrocytes from various blood groups to O-type erythrocytes (cell tissue engineering), digital and automation systems, and artificial intelligence will enable fast and accurate data analysis, reduction of human error, reduction of infection risk, and the use of advanced bioprinters.

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Abstract 014

PLATELET FUNCTION DISORDERS: CONTEMPORARY INSIGHTS AND FUTURE DIRECTIONS

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Platelet function disorders (PFDs) represent a diverse group of qualitative platelet defects that often remain underdiagnosed despite normal platelet counts. Their clinical relevance extends beyond hematology, as undetected PFDs contribute to perioperative bleeding, complications in oncology, and challenges in balancing hemostasis with cardiovascular protection during antiplatelet therapy. For hematologists, timely recognition of these disorders is critical for optimal patient care. Inherited PFDs (IPFDs) include Glanzmann thrombasthenia, Bernard–Soulier syndrome, and RUNX1-associated familial platelet disorder, each characterized by distinct receptor or signaling abnormalities. These range from impaired fibrinogen binding (αIIbβ3 defects) to defective adhesion (GPIb–IX–V complex deficiencies). Syndromic forms such as Wiskott–Aldrich syndrome illustrate the intersection of platelet dysfunction, immune dysregulation, and malignancy predisposition. The spectrum of bleeding can vary considerably. Acquired PFDs are more frequent and clinically impactful. Drugs such as aspirin and P2Y12 inhibitors, uremia, advanced liver disease, myeloproliferative neoplasms, and xtracorporeal circulation all compromise platelet activation or secretion. Given their prevalence, distinguishing pharmacologic platelet inhibition from true dysfunction is a practical challenge in routine hematology. Diagnosis requires a structured, tiered approach. Clinical history and bleeding scores remain the foundation, but must be complemented by laboratory assays. Initial testing should exclude von Willebrand disease, while light transmission aggregometry, flow cytometry, and secretion assays provide functional insights. Next-generation sequencing now allows precise molecular classification of many IPFDs, though accessibility remains uneven. Novel technologies, including microfluidics and whole-blood shear assays, ... Therapeutic strategies depend on etiology and severity. Antifibrinolytics and desmopressin are often sufficient for mild bleeding; platelet transfusions and recombinant factor VIIa are mainstays for severe inherited forms, particularly Glanzmann thrombasthenia complicated by alloimmunization. Hematopoietic stem cell transplantation offers curative potential in selected syndromic disorders. In acquired dysfunction, correcting underlying disease or adjusting medications is essential. Personalized perioperative

pla... Future challenges include diagnostic delays, variability in laboratory availability, and unequal global access to advanced therapies. However, rapid integration of genomics, standardized testing protocols, and emerging hemostatic agents promise to redefine clinical management. Collaborative registries and international networks will be essential to accelerate discovery and translate innovation into equitable care. In conclusion, PFDs embody a nuanced and evolving frontier in hematology. By integrating advanced diagnostics with personalized management strategies, hematologists can reduce morbidity, anticipate complications, and contribute to reshaping the future of bleeding disorder care., Türkiye

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Abstract 015

CNS INVOLVEMENT IN PRIMARY AND SECONDARY ALL AND TREATMENT STRATEGIES

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Abstract Central nervous system (CNS) involvement is an important prognostic factor in acute lymphoblastic leukemia (ALL). Both primary and secondary CNS disease are associated with increased relapse risk and inferior survival. In adults, CNS involvement at diagnosis occurs in 5–10% of cases, with relapse rates of 4–15%. Before the introduction of prophylaxis in the 1980s, CNS relapse rates were as high as 30–40%. The pathophysiology of CNS involvement in ALL is complex, involving early migration of leukemic blasts across the blood –brain barrier, facilitated by adhesion molecules, integrins, and vascular endothelial growth factor (VEGF). VEGF-mediated endothelial disruption increases vascular permeability and plays a pivotal role in the development of posterior reversible encephalopathy syndrome (PRES). Targeting VEGF with monoclonal antibodies has been shown to reduce CNS leukemic burden, suggesting a promising future strategy in both pediatric and adult ALL. The immune-privileged microenvironment of the CNS provides a sanctuary for leukemic cells, supporting their persistence and relapse risk. Traditionally, cerebrospinal fluid (CSF) cytomorphology has been considered the gold standard for assessing CNS involvement. However, this method has low sensitivity and specificity, particularly in samples with low cell counts or technical artifacts. In recent years, flow cytometric immunophenotyping of CSF has demonstrated superior sensitivity, identifying CNS disease more frequently and serving as a strong biomarker for relapse prediction. Minimal CNS involvement not only increases the risk of relapse but is also associated with treatment-related neurotoxicities. Data from the NOPHO group indicate that minimal CNS involvement in pediatric ALL is linked to higher rates of seizures and PRES. Standard treatment approaches continue to rely on intrathecal chemotherapy (methotrexate, cytarabine, corticosteroids) and high-dose systemic agents. However, repeated intrathecal administration and cranial irradiation carry substantial risks of long-term neurotoxicity, highlighting the need for more selective and less toxic strategies. Radiation therapy may still be considered in selected cases, particularly in the context of hematopoietic stem cell transplantation (HSCT). HSCT remains a potentially curative option, especially when preceded by effective cytoreduction with immunotherapy. In conclusion, CNS involvement in ALL represents a biologically and clinically distinct entity requiring tailored management. Primary involvement demands sensitive diagnostics and a careful balance between efficacy and neurotoxicity, while secondary CNS relapse necessitates aggressive multimodal therapy, often incorporating novel immunotherapies and HSCT. Advances in CNS-directed diagnostics and therapeutics are expected to further individualize treatment, aiming to reduce relapse risk while minimizing late toxicities.

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Abstract 016

GAUCHER DISEASE

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Gaucher disease is an autosomal recessive lysosomal storage disorder caused by pathogenic variants in the GBA1 gene on chromosome 1q21, resulting in reduced or absent activity of the enzyme glucocerebrosidase. Consequently, glucosylceramide accumulates primarily in macrophages, leading to the formation of Gaucher cells. The disease most commonly presents with anemia, thrombocytopenia, bleeding tendency, hepatosplenomegaly, fatigue, and skeletal involvement. Bone pathology includes decreased mineral density, bone marrow infiltration, infarction, and fibrosis, all of which contribute to impaired hematopoiesis and cytopenias. From a hematological standpoint, bone marrow aspiration may reveal Gaucher cells with the typical “wrinkled tissue paper” cytoplasm; however, this finding is not pathognomonic and may be seen in other lysosomal storage disorders. Definitive diagnosis therefore requires demonstration of deficient glucocerebrosidase activity or identification of pathogenic GBA1 variants through molecular analysis. In clinical practice, hematological parameters remain essential both for diagnosis and longitudinal monitoring. Complete blood counts provide information on cytopenias and treatment response, while coagulation studies and platelet function tests assist in evaluating bleeding risk. Biomarkers such as chitotriosidase and glucosylsphingosine, together with organomegaly assessment, are increasingly employed in follow-up. Historically, hematopoietic stem cell transplantation was considered a potential curative approach but was limited by high morbidity, mortality, and donor-related challenges. With the advent and efficacy of enzyme replacement therapy and substrate reduction therapy, hematopoietic stem cell transplantation is now reserved only for rare, severe cases without access to standard treatment. In summary, Gaucher disease is a multisystemic disorder with prominent hematological manifestations. Early recognition, accurate diagnosis, and systematic monitoring