In autologous HSCT, stem cells are collected from the patient following prior exposure to chemotherapy. The standard mobilization approach is granulocyte colony-stimulating factor (G-CSF) alone or in combination with chemotherapy, such as cyclophosphamide. While chemotherapy-based mobilization may increase CD34+ yields and contribute to disease cytoreduction, it is associated with increased infectious and hematologic complications. Plerixafor, a CXCR4 antagonist, has emerged as a highly effective adjunct in patients with poor mobilization, particularly those heavily pretreated or with impaired marrow reserve. Predictors of mobilization failure include advanced age, extensive prior therapy, and low baseline blood counts. In allogeneic HSCT, stem cells are obtained from healthy donors. G-CSF administration for 4 –5 days remains the standard strategy, providing sufficient peripheral blood stem cell (PBSC) yields and enabling rapid hematopoietic recovery. Compared with bone marrow harvest, PBSC collection is less invasive and results in higher CD34+ cell counts, but is associated with an increased incidence of chronic graft-versus-host disease. Plerixafor has been investigated as an alternative or adjunct in specific donor populations with inadequate mobilization, though its use remains limited. Donor safety, tolerability of mobilization agents, and long-term health implications are major considerations in the allogeneic context. Despite distinct indications, both autologous and allogeneic mobilization share key challenges: ensuring adequate stem cell yield, minimizing toxicity, and reducing the need for multiple apheresis procedures. Recent advances have improved mobilization outcomes, yet the problem of poor mobilizers persists. Novel mobilizing agents, optimization of dosing schedules, and risk-adapted strategies are under evaluation to enhance efficiency and safety. Stem cell mobilization remains a critical determinant of HSCT success. Autologous mobilization is challenged by prior therapy and patient-related factors, whereas allogeneic mobilization prioritizes donor safety and graft quality. The incorporation of agents such as plerixafor has significantly expanded the mobilization armamentarium. Future directions include individualized mobilization protocols, novel pharmacologic combinations, and strategies aimed at improving long-term transplant outcomes.

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Abstract 025

LABORATORY EVALUATION IN MYELOMA: WHICH TESTS SHOULD BE PREFERRED DURING DIAGNOSIS AND FOLLOW-UP?

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Introduction: Multiple myeloma (MM) is a plasma cell malignancy characterized by clonal proliferation of abnormal plasma cells, production of monoclonal immunoglobulins, and organ dysfunction, often defined by the CRAB criteria (hypercalcemia, renal impairment, anemia, and bone disease). Laboratory testing is central to diagnosis, risk assessment, and monitoring during therapy and remission. **Baseline Evaluation at Diagnosis: Hematology and Biochemistry** - CBC with differential → detection of anemia, leukopenia, or thrombocytopenia. - Biochemistry panel → creatinine, urea, calcium, albumin, LDH. - β2-microglobulin and albumin → incorporated into the Revised International Staging System (R-ISS). - CRP may reflect disease activity (IL-6 driven). **Monoclonal Protein Studies:** - Serum protein electrophoresis (SPEP): quantifies the M-spike. - Urine protein electrophoresis (UPEP, 24 h): detects Bence Jones proteinuria. - Immunofixation (serum and urine): confirms the type of heavy and light chain. - Serum free light chain (sFLC) assay: critical for light-chain, non-secretory, and oligo-secretory myeloma. **Bone Marrow Examination** - Morphology: percentage of plasma cells. - Multiparameter flow cytometry: demonstrates clonality and immunophenotype. - Cytogenetics/FISH: identifies high-risk abnormalities (del[17p], t[4;14], t[14;16]) that influence prognosis. **Laboratory Evaluation During Follow-Up Routine Monitoring** - M-protein quantification (SPEP/UPEP): mainstay of monitoring. - Immunofixation: required to confirm complete response. - sFLC assay: sensitive tool for relapse, especially in light-chain disease. - CBC, renal function, calcium, LDH, β2-microglobulin: routine for treatment toxicity and disease burden. **Advanced Monitoring** - Minimal Residual Disease (MRD): assessed via next-generation flow cytometry or next-generation sequencing. MRD negativity correlates with superior survival and is increasingly used as a response endpoint. - Mass spectrometry and liquid biopsy are promising future tools for detecting residual disease with high sensitivity. **Preferred Tests in Clinical Practice** - At diagnosis: a comprehensive panel including SPEP, UPEP, serum/urine immunofixation, sFLC, bone marrow studies (with cytogenetics/FISH), and advanced imaging is essential. - During follow-up: routine monitoring can be streamlined to SPEP and sFLC, supplemented by basic hematology and chemistry. UPEP is reserved for patients with baseline significant proteinuria. - In specialized centers: MRD testing should be incorporated, especially in clinical trials, to refine response evaluation. **Conclusion** Laboratory evaluation remains the cornerstone of myeloma diagnosis and long-term management. While a full diagnostic panel is indispensable at baseline, streamlined monitoring with SPEP and sFLC is sufficient in most patients during follow-up. Advanced tools such as MRD assessment and mass spectrometry are reshaping the landscape, providing unprecedented sensitivity in disease monitoring. The optimal combination of tests ensures accurate diagnosis, appropriate risk stratification, and effective treatment monitoring in multiple myeloma.

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Abstract 026

ACUTE AND CHRONIC GRAFT-VERSUS-HOST DISEASE: INSIGHTS INTO ETIOPATHOGENESIS

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Graft-versus-host disease (GvHD) remains one of the most significant complications following allogeneic hematopoietic stem cell transplantation (HSCT), contributing substantially to morbidity and mortality despite advances in conditioning regimens, donor selection, and prophylactic strategies. Understanding the etiopathogenesis of acute and chronic GvHD is essential for improving risk stratification, tailoring prophylaxis, and designing novel targeted therapies. Acute GvHD (aGvHD) typically develops within the first 100 days post-transplant and arises from a multi-step immunopathological cascade. Conditioning regimens induce extensive tissue damage, releasing danger-associated molecular patterns (DAMPs) and pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6, which activate host antigen-presenting cells (APCs). Activated APCs prime donor T cells, leading to the expansion of alloreactive effector T cells. These T cells infiltrate target organs—most prominently the skin, gastrointestinal tract, and liver—mediating tissue destruction via cytotoxic molecules (perforin, granzyme) and further amplification of the inflammatory milieu. Regulatory T cell (Treg) dysfunction, microbial translocation from intestinal damage, and loss of epithelial integrity amplify these effects. Emerging evidence highlights the contribution of innate immune cells, the microbiome, and cytokine networks in shaping the severity and trajectory of aGvHD. Chronic GvHD (cGvHD), in contrast, is a complex, multifactorial syndrome that shares features with autoimmune and fibrotic disorders. It generally manifests beyond day 100, although temporal overlap with aGvHD is increasingly recognized. The pathogenesis of cGvHD involves sustained immune dysregulation, including aberrant thymic recovery, impaired central and peripheral tolerance, and persistence of autoreactive and alloreactive T and B cells. B cell hyperactivity, autoantibody production, and activation of germinal center–like reactions contribute to chronic inflammation. Crosstalk between T follicular helper cells, pathogenic B cells, and fibroblasts drives tissue remodeling and fibrosis. Key target organs include the skin, lungs, liver, eyes, and mucous membranes, with progressive organ dysfunction severely impacting quality of life. Recent studies underscore the importance of profibrotic cytokines (e.g., TGF-β, PDGF) and aberrant tissue repair pathways in perpetuating cGvHD. Advances in molecular and cellular profiling have provided novel insights into both acute and chronic disease mechanisms. High-throughput sequencing, proteomic analyses, and microbiome studies have identified candidate biomarkers for early diagnosis, disease monitoring, and therapeutic stratification. These findings are paving the way toward precision medicine approaches, including selective inhibition of JAK/STAT pathways, B cell depletion strategies, adoptive Treg therapy, and microbiota modulation. Despite these promising developments, challenges remain in balancing graft-versus-host effects with graft-versus-leukemia (GvL) activity, underscoring the need for therapeutic interventions that preserve antitumor immunity while mitigating alloreactivity. In summary, both acute and chronic GvHD arise from complex, overlapping yet distinct immunopathological processes that reflect dysregulated interactions between donor-derived immune cells, host tissues, and the microenvironment. Ongoing research continues to refine our understanding of GvHD biology, which is critical for developing innovative therapies and improving long-term outcomes in allogeneic HSCT recipients.

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Abstract 027

CHELATION THERAPY IN THALASSEMIA

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Thalassemia major is a severe hereditary hemoglobinopathy characterized by ineffective erythropoiesis and transfusion-dependent anemia. Regular red blood cell transfusions remain the cornerstone of supportive treatment; however, they inevitably result in progressive iron overload due to the absence of physiological mechanisms for iron excretion. Iron accumulation predominantly affects the liver, heart, and endocrine organs, leading to cirrhosis, cardiomyopathy, arrhythmias, and multiple endocrinopathies. Consequently, iron chelation therapy constitutes a fundamental component of long-term management in patients with thalassemia major. The first clinically available chelating agent was deferoxamine (DFO) promotes urinary and fecal iron excretion. Long-term use of DFO has significantly improved survival by reducing iron-related cardiac mortality. Nevertheless, its administration—via subcutaneous or intravenous infusion for 8–12 hours on most days of the week—poses substantial challenges to adherence, particularly in pediatric and adolescent populations. To address these limitations, oral chelators were developed. Deferiprone (DFP) is effective in reducing myocardial iron burden and preventing cardiac dysfunction, although it carries the risk of agranulocytosis, requiring strict hematological monitoring. Deferasirox (DFX) has demonstrated efficacy in maintaining negative iron balance and reducing hepatic iron concentration, thereby improving adherence and overall patient satisfaction. In cases of severe or refractory iron overload, combination therapy has been employed. The concurrent use of DFO and DFP exhibits synergistic effects, particularly in the clearance of cardiac iron. Emerging data also support the potential benefits of combining DFO with DFX in select clinical scenarios. These strategies allow for individualized treatment based on iron burden, organ involvement, and patient tolerance. Monitoring of chelation efficacy is essential. Serum ferritin is widely utilized as a surrogate marker of body iron, though it may be confounded by inflammation or hepatic injury. T2-star magnetic resonance imaging provides a more reliable and non-invasive quantification of cardiac and hepatic iron, enabling timely therapeutic adjustments and prevention of irreversible organ damage. Chelation therapy has transformed the prognosis of thalassemia major, shifting the natural history from early mortality to survival into adulthood with improved quality of life. Nevertheless, challenges persist, including variability in drug availability, treatment adherence, and adverse event profiles. Future perspectives include optimization of chelation regimens, development of safer agents, and curative