making, as MRD negativity is the strongest predictor of favorable outcomes. 2. Reducing treatment-related toxicity, particularly in elderly or frail patients, by minimizing intensive chemotherapy and incorporating TKIs with immunotherapy. 3. Individualizing the role of allo-HSCT, reserving it primarily for patients with persistent MRD, high-risk features, or early relapse. 4. Integrating novel agents such as blinatumomab, inotuzumab, and CAR-T therapies earlier in the treatment course to improve long-term survival and potentially reduce the need for transplantation. In summary, modern management of Ph+ ALL emphasizes TKI-based regimens, MRD-guided therapeutic decisions, and the incorporation of targeted immunotherapies. While allo-HSCT remains relevant for selected patients, emerging evidence suggests that long-term remission may increasingly be achievable without transplantation, especially when potent TKIs and immunotherapies are combined. This evolving paradigm reflects a shift toward personalized, less toxic, and more effective treatment strategies for Ph+ ALL.

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Abstract 030

CRS AND ICANS MANAGEMENT

Mehmet Gündüz

Biruni University Hospital, Türkiye

CRS (Cytokine Release Syndrome) CRS is an exaggerated systemic inflammatory response triggered by treatments such as Bispecific Antibodies (BsAb), which activate T cells and cause the release of inflammatory cytokines. CRS symptoms range from mild flu-like symptoms to severe multiorgan failure. Symptoms: Fever, hypotension, hypoxia, tachycardia, organ dysfunction. Physical Examination - Temperature, blood pressure, pulse oximetry or arterial blood gas (or mixed venous blood gas/O2 saturation), skin, heart, and lung examination Laboratory Tests - Complete blood count with differential diagnosis; Coagulation (PT/PTT, fibrinogen, fibrin D-dimer); Chemistry (serum electrolytes, kidney and liver function, uric acid, lactate, LDH; C-reactive protein and ferritin (inflammation); Microbiological tests, especially in neutropenic patients (blood and urine cultures); cardiac markers are clinically indicated. Do not await laboratory results. Laboratory findings: Cytopenias, elevated creatinine, elevated liver enzymes, irregular coagulation parameters, elevated C-Reactive Protein • Management of CRS (see Management Section below) does not require laboratory testing and should not be delayed pending laboratory results. **Management by grade:** • Grade 1: Support only (antipyretic, fluid support, close monitoring). • Grade 2: Low-dose oxygen, IV fluids, low-dose vasopressors if necessary. Tocilizumab may be initiated. • Grade ≥3: High-dose oxygen, intensive care support, vasopressor requirement. **Medical Treatment:** • First choice: Tocilizumab (anti-IL-6 monoclonal antibody) • If no response: Corticosteroids (e.g., dexamethasone, methylprednisolone) are added. • Other support: Antibiotic prophylaxis/treatment, electrolyte balance, close monitoring of organ functions. **Immune Effector Cell-Associated Neurotoxicity Syndrome (ICANS):** Neurological toxicity caused by the inflammatory effects of cytokines released after BsAb treatment results in disruption of the blood-brain barrier and accumulation of inflammatory cytokines in the central nervous system. ICANS is a diagnosis of exclusion after other possibilities have been excluded. Neurological toxicity develops after immune activation. Flu-like symptoms: Fever (≥38.0°C/<100.4°F) (unattributable to another cause); nausea; fatigue; headache; rash; diarrhea, arthralgia, myalgia Hypotension Systemic inflammatory response syndrome (circulatory collapse; vascular leakage; peripheral and/or pulmonary edema; renal failure; cardiac dysfunction; multiorgan failure) Respiratory symptoms: cough; tachypnea; hypoxia, ARDS Rash and Urticaria (allergic reaction) Low-grade CRS is common and high-grade is rare **Diagnosis:** • ICANS should be suspected if there are new or worsening neurological symptoms following recent immune effector cell (IEC) therapy, such as CAR-T cell therapy or BsAb therapy. • Initial symptoms may be mild, such as loss of attention and/or slurred speech or tremors. • Further evaluation to investigate other possible causes should include review of concomitant medications or recent use of CNS-active drugs (e.g., opiates, benzodiazepines). Investigation may include a head CT or brain MRI, and a lumbar puncture to investigate infectious causes. Management: It may occur with or without CRS. **Treatment:** • Grade 1 (mild): Close neurological monitoring, supportive care. • Grade ≥2: Corticosteroids (Dexamethasone or Methylprednisolone) are initiated. • Tocilizumab is generally not effective for ICANS (because the IL-6 antibody does not cross the blood-brain barrier well). • Seizure prophylaxis/treatment: Levetiracetam is preferred. • Intensive care support in severe cases.

Keywords: CRS, ICANS, management, treatment.

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Abstract 031

GRAFT VERSUS HOST DISEASE PROPHYLAXIS

Şebnem İzmir Güner

İstanbul Hisar Hospital İntercontinental, Türkiye

Graft−versus−host disease (GvHD) is an important complication that can be observed after allogeneic hematopoietic stem cell transplantation (allo-HSCT). The incidence of Acute GvHD (aGvHD) is around 30%-50% in HLA fully matched allo-HSCT. aGvHD is also common in haploidentical and matched unrelated donor transplantation. The mechanism underlying tissue damage in aGvHD is massive inflammatory cytokine secretion. Proinflammatory cytokines [tumor necrosis factor (TNF)-α, interleukin (IL)-1β, and IL-6] are seen, as well as the increased expression of the receptor repertoire (pattern recognition receptors) on antigen-presenting cells. The most important risk factor for GvHD is HLA mismatch. Other risk factors include sex disparity between donor and recipient, the intensity of the conditioning regimen, increased age, multiparous female donors, ineffective GvHD prophylaxis, and the source of the graft. A study showed that aGvHD was