venetoclax-azacitidine has become a first-line treatment for elderly AML patients worldwide who are unfit for intensive therapy. Similarly, the VIALE-C trial, which randomized patients to LDAC/venetoclax versus LDAC/placebo, demonstrated improved CR/Cri (48% vs 13%) and OS (8.4 vs 4.1 months) in the venetoclax arm.(8) The combination of HMAs with other agents, together with the establishment of genetic risk profiles and identification existing mutations, underscores the importance of individualized therapy. Among promising agents, Ivosidenib monotherapy or its combination with HMA has shown superiority in OS, CR/Cri, and EFS for IDH- 1mutated de novo AML (AGILE trail) (9). Patients with TP53 alterations, however, continue to experience significantly worse survival outcomes (10). The CD47 monoclonal antibody magrolimab has demonstrated clinical efficacy when combined with azacitidine or with azacitidine/venetoclax (11).Several multiple novel agents and combinations are under investigation, including fromtline FLT3i, oral HMAs, and triplets combining HMA, venetoclax and targeted agents (12). Considering that none of these regimens are curative, it remains a matter of debate whether dynamically assessing patient frailty and using non-intensive therapies can provide a bridge to allogenic stem cell transplantation.

https://doi.org/10.1016/j.htct.2025.106197

Abstract 021

HEPATIC VENO-OCCLUSIVE DISEASE

Barbaros Şahin Karagün

Adana City Training and Research Hospital, Türkiye

Hepatic veno-occlusive disease, also called sinusoidal obstruction syndrome (VOD/SOS), is a severe complication which usually occurs due to conditioning regimens used for hematopoietic stem cell transplantation (HSCT). It is characterized by hepatomegaly, hyperbilirubinemia, ascites and right upper quadrant pain and usually develops within the first 20-30 days after transplant. It is accepted to be a result of endothelium and hepatocyte damage caused by chemotherapy and radiotherapy of the conditioning regimen. Current studies suggest that the primary site of toxic injury is the hepatocyte, subsequently followed by damage to the central veins in zone 3 of the hepatic acinus and sinusoidal endothelial cells. Early changes include fibrin deposition, venous occlusion, progressive venous micro-thrombosis and sinusoidal occlusion. These changes lead to severe clinical problems including portal hypertension, hepatorenal syndrome and hepatocellular necrosis, which may ultimately result in multiorgan dysfunction (MOD) and death. Previously, the Baltimore and Seattle criteria were used for VOD/SOS diagnosis; however, the limitations of these criteria for VOD/SOS diagnosis (especially in anicteric children and those who have symptom onset after 21 days), led to establishment of the EBMT (European Society for Blood and Marrow Transplantation) 2017 VOD/SOS criteria which evaluates pediatric and adult patients separately. The EBMT 2017 criteria is comprised of laboratory and clinical findings such as transfusion-resistant thrombocytopenia, unexplained weight gain, hepatomegaly, ascites and elevation in bilirubin levels. Despite the advantages brought by this criteria, it is still difficult to diagnose VOD/SOS. Several approaches to prevent its development of VOD/SOS were put forth, including individualized dosing of chemotherapy, reduction of the intensity of the conditioning regimens, close monitoring of the levels of busulfan and cyclophosphamide and also reducing their use. Prostaglandin E1 and tissue-plasminogen activator with or without concurrent heparin have been explored in VOD/SOS treatment; however, these approaches have shown little success, as is the case with supportive treatments. Defibrotide (DF) emerged as the most promising medication for both prophylaxis and treatment in patients with VOD/SOS. DF is a single-stranded polydeoxyribonucleotide with anti-inflammatory, anti-ischemic, anti-thrombotic, and thrombolytic properties in addition to its protective effects on endothelial cells. DF is approved for adult and pediatric patients with VOD/SOS with renal or pulmonary dysfunction after HSCT in the United States, and for severe VOD/SOS post-HSCT in patients aged >1 month in the European Union. In addition, several studies have examined DF prophylaxis can reduce the incidence of VOD/SOS in high-risk patients. Although the literature is unanimous for the use of DF in patients diagnosed with VOD/SOS, its use as a prophylactic agent has not been approved; even though many studies have reported reduced VOD/SOS incidence and severity with DF prophylaxis.

https://doi.org/10.1016/j.htct.2025.106198

Abstract 022

TREATMENT OF RELAPSED/REFRACTORY DLBCL

Hakan Kalyon

Koç University Faculty Of Medicine, Türkiye

Fifteen percent of DLBCL patients are refractory to the first line of therapy, while 25% experience relapse after response. The management of these patients is planned according to the patient's suitability for high-dose chemotherapy and whether the disease is refractory/early relapse (BSH guideline, 2025). While HSCT provides long-term survival in patients who are suitable for treatment and are chemosensitive (CORAL study), long-term survival compared to HSCT has been achieved in non-chemosensitive patients with CAR-T therapies ZUMA-7 and TRANSFORM studies. CAR-T therapies are approved as first-line treatment for patients with refractory/early relapse. However, some r/r DLBCL patients are not suitable for HSCT and CAR-T treatments due to age and comorbidities, and some are resistant to these treatments or relapse after these treatments. Tafasitamab – Lenalidomide combination is approved for patients with relapsed DLBCL, NOS who are not eligible for HSCT or CAR-T therapies (L-MIND study). The efficacy of Glofitamab – GemOx has also been proven in patients with relapsed DLBCL, NOS who are not suitable for HSCT or CAR-T therapy in the STARGLO study. Loncastuximab is a single-agent ADC used in r/r DLBCL. Due to its cumulative toxicity, long-term use is not suitable, and a one year treatment was planned in the LOTIS-