Long-term renal functions of the patients were investigated. **Results:** 80.3% of the patients with a median age of 9.8 years were male. The most common histopathological subgroup was BL (77.5%), while the majority of patients (76.7%) had advanced disease. Clinical TLS (CTLS) was observed in 12.1% of the cases, and isolated laboratory TLS (LTLS) was observed in 18.7%. Hyperhydration±alkalinization and allopurinol were used in first-line treatment and prophylaxis. A significant correlation was found between young age, advanced stage, high lactate dehydrogenase level at presentation and LTLS. Bone marrow involvement was found to be significantly higher in the group with CTLS. AKI was observed in 12.1% of the patients. Out of a total of 103 patients whose treatment was completed, 93 (90.3%) patients survived and 10 deaths were observed. No death due to TLS was observed. The mean survival time was 215.55±7.502 months. After an average of 6.9 years, when the glomerular filtration rate values of the patients at the first admission and at the last admission were compared, a mean decrease of 10 mL/min/1.73 m2 was detected. However, it was not found to be statistically significant. **Conclusion:** In our study, lower age, advanced stage, high LDH level at presentation were found to be risk factors for TLS. Long-term renal function loss was not detected in the survivors, for whom early and careful prophylaxis/treatment approaches were applied for TLS. The survivors are still being followed up.

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SUPPORTIVE CARE AND PALLIATIVE CARE

OP 22

IMPACT OF COVID-19 PANDEMIC ON DELAY OF CHILDHOOD CANCER DIAGNOSIS AND THE OUTCOMES IN A PEDIATRIC HEMATOLOGY/ ONCOLOGY DEPARTMENT

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Objective: Restriction of access to healthcare during COVID-19 pandemic is undoubtedly a major problem for patients with cancer. Although childhood cancers are highly curable, it is obvious that diagnostic and treatment disruptions will lead to poor Results. In this study we investigated the effects of pandemic on diagnosis and treatment delays of children with cancer along with their consequences. **Methodology:** We searched all pediatric patients treated for cancer between March 2020 and January 2022 for COVID-19 infection. Data were collected collected from medical files of patients diagnosed with COVID-19, confirmed by polymerase chain reaction (PCR), who received active antineoplastic treatment. **Results:** Fifty-eight patients developed COVID-19 infection at different stages of their anticancer treatment. Twenty-five had an asymptomatic COVID-19 infection, twenty-six had mild symptoms, three had moderate symptoms and four had severe disease. All of them recovered from COVID-19 infection. Chemotherapy courses were continued during active infection in four patients and interrupted in other patients. **Conclusion:** While strict measures are required to control the pandemic, patients with severe critical illness such as cancer should be carefully evaluated and treatment delays that may have vital consequences should be avoided. In pediatric patients with cancer whom infected by COVID-19, continuation of anticancer treatment may be considered by evaluating the clinical status of the patient.

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TUMOR BIOLOGY, IMMUNOLOGY AND IMMUNOTHERAPY

OP 23

INTRAPLEURAL THERAPY TO DISRUPT IL-6/IL-8 JUXTACTINE SIGNALLING TO BLOCK TUMOR EMT AND TO DRIVE SYSTEMIC ANTI-TUMOR IMMUNITY

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Objective: The goal of this study was to determine whether antiIL-6Rα block (tocilizumab) will alter the pleural secretome and will diminish tumor-specific immune responses. **Methodology:** Pleural T cells were isolated from freshly drained pleural effusions (n=6). Autologous pleural tumor was expanded in vitro using the Mammary Epithelial Growth Medium (Lonza). Pleural T cells were stimulated using anti-CD3/CD28 Dynal beads and low dose IL-2 (60 Cetus U/ml) for 2,4,7, 14 or 21 days in the presence of tocilizumab for the last 48h (0, 0.35, 0.72, 1.43, 2.86 and 5.72ug/ml). Pleural T cell effectors were counted and plated on tumor targets at 12.5:1 E:T in the presence of tocilizumab. **Results:** Ex vivo expanded pleural T cells were effector-memory phenotype (CD45RA-CD27-) and were highly cytotoxic against autologous tumor (89-100%). The majority of CD8+ T cells were central memory (CD45RA-/CD27-) or effector memory (CD45RA+/CD27-); the majority co-expressed granzyme B, perforin, 20-60% expressed PD-1. Most CD4+ co-expressed granzyme B and perforin and were PD-1+, suggesting cytotoxic CD4+ T cells. The presence of tocilizumab reversed tumor EMT but did not alter cytotoxicity. **Conclusion:** We show that the IL-6/IL-6Rα axis is prominent in MPE, drives tumor growth and inhibits anti-tumor immunity. Pleural T cells are neither exhausted nor dysfunctional but are suppressed by the pleural environment. Ex vivo expanded MPE CD8 and CD4 T cells are highly cytotoxic against