

0073 - ASSESSMENT OF TESTICULAR FUNCTION IN BOYS AND ADOLESCENTS WITH HEMATOLOGICAL MALIGNANCIES: PROSPECTIVE LONGITUDINAL STUDY FROM DIAGNOSIS UP TO 3 YEARS AFTER THE END OF CHEMOTHERAPY

Mode: Research paper (Oral Awards)

Thematic Unit: Puberty

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Background/Objectives: Survival after pediatric hematologic malignancies has improved substantially, increasing concern regarding endocrine and reproductive sequelae. While gonadotoxic effects on germ cells are well characterized in adult survivors, the early impact of cancer and chemotherapy on hypothalamic–pituitary–testicular axis function during childhood and adolescence remains insufficiently studied. The aim of this study was to characterize testicular function of boys and adolescents with hematologic malignancies, from diagnosis up to 3 years after the end of treatment.

Methods: We conducted a prospective longitudinal cohort study in boys and adolescents with acute lymphoblastic leukemia (ALL), acute myeloid leukemia (AML), or non-Hodgkin lymphoma (NHL) treated at a tertiary pediatric center in Argentina between 2013 and 2019. Hormonal markers of Sertoli cell function (AMH, inhibin B, FSH) and Leydig cell function (testosterone, LH) were prospectively assessed from diagnosis, throughout chemotherapy, and up to 3 years after treatment completion. Hormone concentrations were analyzed adjusted to age and pubertal stage.

Results: 63 patients were included. ALL was the most prevalent diagnosis (65.1%), followed by NHL including lymphoblastic lymphoma (LBL) and other types of NHL (27.0%), and AML (7.9%). Patients with ALL and LBL were pooled for analysis, given the homogeneity of their chemotherapy protocols. Prepubertal boys at diagnosis, who represented most of the cohort (77.8%), showed mildly impaired Sertoli cell function at baseline, characterized by reduced AMH and FSH levels. AMH increased during chemotherapy induction, fluctuated throughout the remaining treatment, and normalized thereafter, remaining stable up to 3 years post-treatment. Notably, transient FSH suppression occurred concomitantly with high-dose corticosteroid exposure. Boys entering puberty during follow-up showed persistently elevated AMH relative to pubertal stage, suggesting delayed Sertoli cell maturation. In pubertal patients, chemotherapy induced transient seminiferous tubular dysfunction, reflected by increased FSH and decreased inhibin B, with recovery by 3 years after treatment. Mild compensated Leydig cell dysfunction persisted, characterized by elevated LH despite normal testosterone concentrations.

Conclusion: Testicular dysfunction associated with pediatric hematologic malignancies is mostly reversible. However, delayed Sertoli cell maturation and persistent compensated Leydig cell insufficiency indicate residual testicular vulnerability, underscoring the importance of long-term endocrine surveillance during and after the end of chemotherapy.

