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EDİTÖRE MEKTUP / LETTER TO THE EDITOR

Rebound thymic hyperplasia

Ribaund timus hiperplazisi

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Dear Editor,

Rebound hyperplasia of the thymus, after cessation of chemotherapy, raises major concern both in family and among clinicians in contex of tumor recurrence. It is a well documented entity following chemotherapy and occurs generally within the first year after stopping chemotherapy. Later presentation makes differatiation difficult benign disorders from a second malignancy in survivors of childhood cancer. Rebound hyperplasia of the thymus generally occurs in children but it may also be observed in young adults.

It is frequently reported after cessation of treatment in survivors of lymphoma[1]. The thymus is a lymphoid organ essential to the maturation of T lymphocytes. It is large in pre-adolescent children before it gradually involutes. Although rebound hyperplasia of the thymus has been documented in various malignancies, it has only been reported once in Wilms' tumor[2].

In this report, an unusual case of rebound thymic hyperplasia is described, presenting 3 months after completion of chemotherapy for Wilms' tumor. A 6-year-old girl was admitted with a complaint of abdominal pain and distention. Physical examination revealed a solid mass in left flank region which was confirmed on computed tomography as a tumor originating from the left kidney. The patient did not demonstrate hemihypertrophy or any other physical stigmata of Beckwith-Wiedemann syndrome. Patient and family history were unremarkable. Her thoracal computed tomography revealed no pulmonary metastasis. After resection of the renal tumor, pathologic examination revealed Wilms' tumor with positive surgical margins without anaplasia. Nine Gy radiotherapy was delivered to the left flank area and the patient was treated with chemotherapy consisting of vincristine, carboplatin and doxorubicin for 12 months. Three months after cessation of treatment, control computed tomography showed no relapse or residual mass in abdomen but an anterior mediastinal mass most probably consistent with thymic hyperplasia. Because of the location of the mass and negative results of the other examinations a rebound thymic hyperplasia was the favored diagnosis. The mass was followed with monthly thoracal ultrasound and with computed tomography every 3 months. Six months later the mass was no longer detectable on tomography scans. The patient is in remission without an event for 12 months.

The thymus is primarily responsible for fetal T-cell development[3]. T cells undergo positive and negative selection in the thymus before entering the circulating immune system. Outside the thymus, the CD45 isoform CD45RA becomes expressed. CD45RO is expressed on memory T cells in circulation after activation. Larger ratios of CD45RA:CD45RO positive T cells have been found in patients with thymic rebound, indicating that thymic enlargement is associated with increased thymopoiesis[4]. With thymic rebound, thymus contributes significantly to the reconstitution of the

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peripheral T-cell population following chemotherapy-induced lymphopenia to re-establish immune function[5]. Moreover, some researchers proposed that hyperplasia of the thymus following chemotherapy would appear to be a good prognostic factor[6].

It is important for clinicians to be informed about rebound hyperplasia of thymus in the differential diagnosis of anterior mediastinal masses in children with solid tumors. Thoracal ultrasound can be used to detect if the mass is thymic tissue in order to avoid unnecessary procedures and treatment. It may occur following intensive chemotherapy for childhood cancers and it should not be misdiagnosed as malignant tumors and overtreated.

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