Interesting image

FDG PET/CT findings of thyrotoxicosis induced thymic hyperplasia associated to interferon

Hallazgos de la FDG PET/TC en la tirotoxicosis inducida por hiperplasia tímica asociada a interferon

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Interferon-alpha (IFNα) is a therapeutic agent which is used for infectious and malignant diseases. Side effects such as flu-like symptoms, hematologic abnormalities and thyroid disease have been reported associated with this therapy. Thyroid disease manifests as development of thyrotoxicosis and it may be secondary to destructive thyroiditis or Graves’ disease.

Here we report a 61-year-old female patient with malignant melanoma. After an IFNα treatment duration of 18 months, she started to complain of nervousness, sweating and palpitation. Serum free T3, free T4, thyroid stimulating hormone (TSH) and antithyroglobulin antibody (TGAb) levels were 22.27 pg/ml (normal: 2.5–3.9 pg/ml), 5.14 ng/ml (normal: 0.61–1.12 ng/ml), 0.05 μIU/ml (normal: 0.34–5.6 μIU/ml) and 312.5 IU/mL (normal: 0–115 IU/mL), respectively. Thyroid scintigraphy with 99mTc pertechnetate was consistent with diffusely increased uptake throughout the thyroid. FDG PET/CT imaging, which was performed for restaging of the melanoma, revealed anterior mediastinal mass in thymus location with elevated FDG uptake (SUVmax 2.91). Thyrotoxicosis induced thymic hyperplasia must be kept in mind in the differential diagnosis of an anterior mediastinal mass which obviates additional diagnostic work-up. To our knowledge, this is the first report in which FDG PET/CT findings of IFNα associated thyrotoxicosis induced thymic hyperplasia have been shown (Figs. 1 and 2).

Although disappearance of metabolic activity in subsequent FDG PET/CT evaluations could not be demonstrated, thymic rebound, which was attributed to thyrotoxicosis induced thymic hyperplasia, was thought.

Thymic enlargement is a rare phenomenon in adults and is mainly observed after chemotherapy.1 Thyrotoxicosis may also cause thymic enlargement which is partly due to the stimulatory effect of raised circulating thyroid hormones2 and partly due to the binding of immunoglobulins to the thyocytes.3 Formation of thymic medullary lymphoid follicles and thymic hyperplasia is found in 38% of patients with Graves’ disease which supports the immunological aspects of thymocyte proliferation.3

Since the description of the association between IFNα and thyroid disease in 1985,4 high incidence of thyroid abnormalities including destructive thyroiditis and Graves’ disease has been reported in patients treated with IFNα.5,6 In our case, presence of TSH receptor antibody or thyroid stimulating immunoglobulin could not be shown but the patient was thyrotoxic and antithyroglobulin antibodies were present. With the findings of laboratory tests and thyroid scintigraphy, Graves’ disease, instead of destructive thyroiditis, was thought.

Thymic rebound and uptake of FDG in thymus have been reported following chemotherapy and also after radioiodine treatment.7,8 Unusual point of our case was observing FDG uptake

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Fig. 1. Thyroid scintigraphy revealed enlargement and diffusely increased 99mTc pertechnetate uptake in both thyroid lobes.
in thymus after IFNα therapy secondary to thyrotoxicosis induced thymic hyperplasia.

FDG uptake in thymus must be interpreted with caution. In order to prevent further invasive procedures, apart from chemotherapy and radioiodine therapy, IFNα therapy should be evaluated in patient history as well.

References