To the Editors:

Lymphoma, thyrotoxicosis and thymic hyperplasia

We read with interest the article titled “A man with thyrotoxicosis, lymphoma and thymic hyperplasia” published in the September 2001 issue of your journal (1). The article does not indicate how thymic hyperplasia was diagnosed. The mediastinal mass which resolved after radiotherapy and chemotherapy could have been due to lymphadenopathy resulting from lymphoma and not due to thymic hyperplasia.

Thymic enlargement could be due to true thymic hyperplasia (defined as thymic enlargement beyond the upper limits of normal for the age) or due to thymic follicular hyperplasia (2). In the former the thymus is microscopically normal whereas in the latter there are secondary lymphoid follicles in the thymus (2). The weight of the thymus is within normal limits in most cases with thymic follicular hyperplasia (2). The thymic hyperplasias associated with thyrotoxicosis, Addison disease, myasthenia gravis, lupus erythematosus and other immune mediated diseases are due to a follicular hyperplasia. True thymic hyperplasia has been most often described in infants and children (3). It has also been described in adults sometimes after successful chemotherapy for Hodgkin’s disease and endodermal sinus tumour of the ovary (4,5).

With regard to the patient described in this case report (1), the most likely type of thymic hyperplasia would be thymic follicular hyperplasia due to the thyrotoxicosis. This requires a histological diagnosis. There is a report of histologically unconfirmed thymic hyperplasia occurring in a woman with thyrotoxicosis who developed mediastinal enlargement following chemotherapy for Hodgkin’s disease (5). In this case the mediastinal enlargement was presumed to have been due to thymic hyperplasia because it disappeared with the treatment of thyrotoxicosis without additional chemotherapy. In the present case (1), the patient had mediastinal enlargement before commencement of chemotherapy. This enlargement showed a significant regression with chemotherapy for non-Hodgkin’s lymphoma and treatment for thyrotoxicosis. As there was no histological confirmation of thymic hyperplasia we feel that it is wrong to presume that the mediastinal enlargement was due to thymic hyperplasia which regressed with therapy for thyrotoxicosis. It is possible that the mediastinal enlargement was due to non-Hodgkin’s lymphoma. The regression could have been a response to chemotherapy. In the absence of histological confirmation it is inaccurate to use the term “thymic hyperplasia” in this patient.

References
2. Rosai J. Ackerman’s Surgical Pathology. 8th ed. St.Louis: Mosby, 1996; 441.

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