CDP was mainly post-operative and with parenteral nutrition. Post-operative complications were common and mainly affected those over 70 years of age, so this group would benefit the most from prehabilitation programmes.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Transient thymic hyperplasia associated with thyroiditis

Hiperplasia tímica transitoria asociada a tiroiditis

Thymic hyperplasia is characterised by a generally diffuse and symmetrical increase in the size of the thymus, which may be caused by epithelial cell proliferation or increased lymphoid follicles in this organ. It may be an incidental finding or alternatively manifest compressive or systemic symptoms. Its association with several autoimmune diseases, including myasthenia gravis, systemic lupus erythematosus, rheumatoid arthritis and, above all, Graves' disease, has been reported.[1] We present a case of transient thymic hyperplasia, incidentally found, associated with silent or painless thyroiditis.

This was a 51-year-old female patient, a one pack-a-day smoker, with no other relevant medical or surgical history, who was examined by the Pulmonology department due to a one-year history of asthma-like symptoms, with night-time coughing and wheezing fits, exertional dyspnoea and recurrent bronchitis. During the medical interview, she reported symptoms of nervousness, irritability and 5-kg weight loss in two months without a reduction in dietary intake. The patient's blood test results revealed hyperthyroidism with thyrotropin or thyroid-stimulating hormone (TSH) below 0.01 uIU/mL (normal range 0.38–5.33) and free thyroxine (T4) 2.49 ng/dl (0.38–1.5), while she was posi-

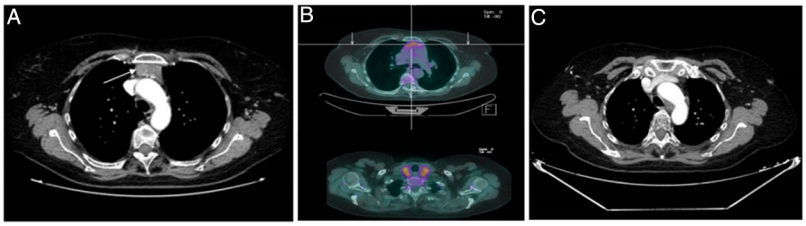

Figure 1 CT and PET-CT thymoma images.

tive for thyroid peroxidase antibodies (428.3 IU/mL, normal value below 10) and negative for TSH receptor antibodies (thyroid stimulating immunoglobulin [TSI]). A thyroid ultrasound was requested, which showed glandular parenchyma with decreased echogenicity, a heterogeneous structure and a pseudonodular appearance, and a chest X-ray, which revealed mediastinal widening. In light of these findings, a computed tomography (CT) of the chest was performed, which incidentally revealed a vascularised solid mass in the anterior and superior mediastinum measuring 44×24 mm, consistent with a thymoma (Fig. 1A). The study was completed with a positron emission tomography (PET/CT) scan that identified a high-uptake lesion in the anterior mediastinum, consistent with thymic hyperplasia, and bilateral and diffuse high thyroid uptake (Fig. 1B). A diagnosis of hyperthyroidism probably associated with Graves' disease was established, and treatment with thiamazole 10 mg/day was started. Once blood test results and incompatible clinical symptoms had ruled out myasthenia gravis, a watchful waiting approach was adopted to the mediastinal lesion following assessment for thoracic surgery. After two months of follow-up, the patient quickly developed hypothyroidism, leading us to change our diagnosis to probable subacute lymphocytic thyroiditis given the marked reduction in free T4, and antithyroid medication was suspended. After six months, thyroid hormone levels returned to normal and a follow-up CT scan found no trace of the mediastinal lesion (Fig. 1C).

Although rare, the onset of thymic hyperplasia in patients with Graves' disease is well documented and has been reported in over one hundred cases published in the scientific literature.[1–4] The prevalence of this association is probably underestimated given that routine imaging studies for its detection are not performed. Its pathogenesis is related both to hyperthyroidism itself as well as to an autoimmune mechanism. On the one hand, thyroid hormones could stimulate thymus growth, particularly the epithelial component and cortical lymphoid tissue through the action of local regulatory proteins such as thymulin, while on the other, TSH receptors have been found in the thymus, in addition to in the orbit or in the dermis of the pretibial region, whose direct stimulation by TSI could induce cell proliferation.[1,2,5] It has also been hypothesised that inflammatory cytokines may induce increased angiogenesis in this organ in Graves' disease patients.[2] Thymic hyperplasia has a benign clinical course and is asymptomatic in the majority of cases. A differential diagnosis must be performed with other lesions located in the anterior mediastinum such as thymomas, germ cell tumours or mesenchymal tumours, lymphomas, masses of thyroid or parathyroid origin or metastases. Recognising its radiological features is therefore essential (thymic hyperplasia is homogeneous, is not accompanied by cystic areas or calcifications and it does not invade neighbouring structures), as is close monitoring, as it almost always remits during Graves' disease treatment and once the thyroid profile has returned to normal.[1,6] As such, biopsy or surgical removal are not usually required, but it is recommended to perform a follow-up CT scan six months after normal thyroid function is restored.[1]

The unusual aspect of our case is that the finding of thymic hyperplasia is not associated with Graves' disease but rather with thyroiditis. The absence of goitre and ophthalmopathy, negativity for TSI, elevated thyroid peroxidase antibodies and the clinical course, with rapid resolution of hyperthyroidism, point to a diagnosis of silent or painless subacute lymphocytic thyroiditis or hashitoxicosis, which have rarely been associated with thymic hyperplasia in the literature.

The pathogenic mechanism of thyroid growth in this context is probably related to the transient increase in thyroid hormones. In this regard, cases of thymic hyperplasia in association with other thyroid disorders, such as follicular neoplasms, have also recently been published.[7,8] It is therefore essential to be aware of these associations and to conduct comprehensive thyroid function tests in all patients with lesions and masses in the anterior mediastinum.

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Diabetes insipidus as initial manifestation of extranodal NK/T cell lymphoma nasal type

Diabetes insípida como manifestación inicial de linfoma extraganglionar nasal de células T/NK

Central diabetes insipidus (CDI) is a disease caused by a decrease in or the absence of antidiuretic hormone (ADH) or arginine vasopressin (AVP), characterised by polydipsia and polyuria with hypotonic urine emission.[1] Possible causes include neoplasms, such as germinomas or craniopharyngiomas, accidental trauma or trauma secondary to intracranial surgery, midline malformations, diseases caused by accumulations such as sarcoidosis and autoimmune and/or infiltrative diseases such as Langerhans cell histiocytosis.[2] There are cases in which there are underlying genetic defects in AVP synthesis, while in others the cause is not fully understood and may be related to an autoimmune component.[3] The diagnosis of CDI represents a major challenge in clinical practice, and the water deprivation test (WDT) or thirst test is the standard for diagnosis. However, the test is complex and the results are sometimes inaccurate, so new tools have been developed in recent years, such as measuring arginine- or hypertonic saline-stimulated copeptin.[4,5]

Extranodal natural killer (NK)/T-cell lymphoma, nasal type (ENKL-NT) is a subtype of lymphoma whose aetiology is not fully understood. It is related to the Epstein–Barr virus (EBV), detection of which is a requirement for diagnosis. It is more common in Asia and America than in the West, it predominates in 40–80-year-old men and affects the nasal area in 80% of cases, presenting with symptoms of nasal obstruction. The definitive diagnosis is histopathological. The treatment of choice is radiotherapy (RT) combined with chemotherapy (CT) in different regimens. The disease has a poor short-to-medium-term prognosis.

We present the case of a 52-year-old male with no known allergies. He was a smoker, with a history of pneumothorax in 2001. One week before admission, he consulted for acute low-back pain, for which he was given an intramuscular injection of 40 mg of methylprednisolone. Three days later, he went to Accident and Emergency complaining of polydipsia and polyuria of seven litres a day, with intermittent paraesthesia on the right side of his face and bilateral nasal congestion. Chest X-ray showed no abnormalities. He was admitted to endocrinology, where a WDT was consistent with the diagnosis of partial CDI (urinary osmolarity of 97 mOsm/kg, which increased after administration of desmopressin >100% up to 535 mOsm/kg), for which treatment was started with desmopressin, 60 mcg/day, at night. Magnetic resonance imaging (MRI) of the pituitary showed an increase in the stalk and the pituitary gland itself, with absence of physiological enhancement of the posterior pituitary on T1, and no evidence of tumour-type lesions. Computed tomography (CT) of neck and chest revealed no significant lymphadenopathy suspicious for sarcoidosis.

The patient's nasal congestion worsened, requiring assessment by Ear, Nose and Throat, who only observed bilateral mucosal thickening with a deviation of the nasal septum. During the first few days of his admission, his low-back pain worsened. A lumbar spine X-ray and lumbar MRI were performed without significant findings. Also performed were a bone series, showing no osteolytic lesions and a blood test for c-ANCA, p-ANCA, anti-cardiolipin Ab, ENA, ANA, anti-DNA Ab, which was negative. Angiotensin-converting enzyme, IgG4 immunoglobulin, alpha-foetoprotein, and human chorionic gonadotropin levels were all normal. The QuantiFERON® test for *M. tuberculosis* was negative, as were serology for human immunodeficiency virus and hepatitis C and B viruses, PCR for SARS-CoV-2, and the serological diagnoses of syphilis and Lyme disease. Urinary sediment was normal. While the patient was in hospital, several skin lesions appeared in the form of non-pruritic erythematous macule-papules, and Dermatology took a biopsy. The paraesthesia rapidly worsened, extending to the lower limbs with no clear metameric distribution. Neurology carried out a lumbar puncture (LP), with flow cytometry revealing a 66% infiltration of NK cells, findings consistent with the preliminary results of the skin biopsy. At that point the patient was transferred to Haematology.

PET/CT scan with 18-fluorodeoxyglucose (FDG) showed marked metabolic activity, both nodal and extranodal, with intense pituitary uptake suggestive of tumour invasion (SUVmax 10.1) (Fig. 1), as well as a large sinonasal hypermetabolic mass and intense activity in the left L3, bilateral L5 and right S1 nerve roots (SUVmax 3.1), consistent with the patient's symptoms of low-back pain. These results, in conjunction with those of the skin biopsy and LP, confirmed the diagnosis of stage IV ENKL-NT with skin and central nervous system involvement.

The patient suddenly developed severe dysphagia, which required treatment with high-dose intravenous dexam-