

Spontaneous resolution of a mediastinal mass in a woman with myasthenia gravis[☆]



Resolución espontánea de una masa mediastínica en una mujer con miastenia gravis

Dear Editor:

Myasthenia gravis is associated with thymomas in approximately 10% of cases; surgery is the recommended treatment, except in cases of advanced age or poor general health. Spontaneous regression of thymomas is rare: according to our literature review, only 9 cases have been reported^{1–5}; 7 of these were reported in Japan and most cases were women. Only one of these patients also had myasthenia gravis.²

We present the case of a 55-year-old woman who was diagnosed with generalised myasthenia gravis (Osseman type IIb) based on the presence of acetylcholine receptor antibodies. A chest CT scan performed in October 2010 revealed a homogeneous mass in the left side of the anterior mediastinum measuring 111 × 58 × 167 mm; there was no mass infiltration (Fig. 1). The CT scan also revealed small adenopathies in the inferior paratracheal area plus another in the aortopulmonary window; these findings were consistent with thymoma. The patient started treatment with pyridostigmine dosed at 60 mg/8 hour and prednisone dosed at 60 mg/day; the dose was progressively decreased the following month. Resection of the thymoma was suggested, but the patient refused surgery. Medical treatment achieved clinical stabilisation, but the patient experienced a relapse after withdrawal of corticosteroid treatment. Prednisone achieved stabilisation; we further administered azathioprine dosed at 150 mg/day, which enabled prednisone to be withdrawn. The patient progressed favourably; she experienced mild fatigability but could independently perform the activities of daily living. A second CT scan was performed one year later, revealing that the mass had decreased considerably. One year after onset of treatment with azathioprine, the patient had experienced no further relapses and recovered progressively until she was nearly asymptomatic. A subsequent follow-up CT scan performed recently showed that the mass had disappeared completely (Fig. 2).

The real rate of spontaneous thymoma regression is unknown since most thymomas are surgically removed. The mechanism underlying the process is still to be determined. In our patient, symptoms progressed favourably with tumour regression. Complete tumour regression has been reported in only one of the 9 cases published⁴; the other patients underwent thymectomy. Radiological follow-up may be an alternative to surgery in certain cases.

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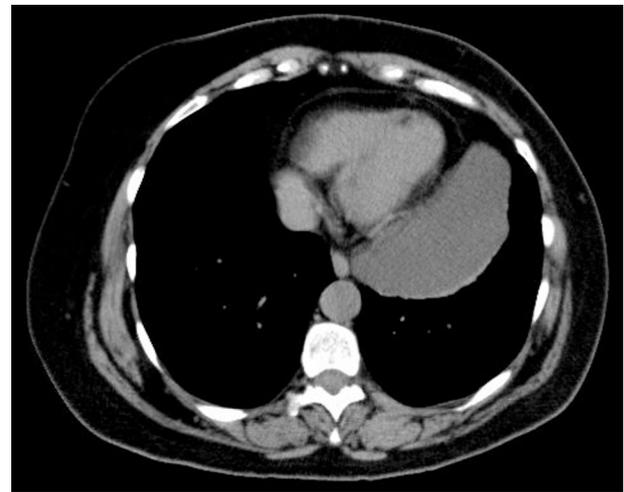


Figure 1 Chest CT scan: mass in the left side of the anterior mediastinum extending from the aortic arch to the diaphragm, with no signs of infiltration and a density similar to that of water. Measurements: 111 mm (anteroposterior) × 58 mm (transverse) × 167 mm (vertical).

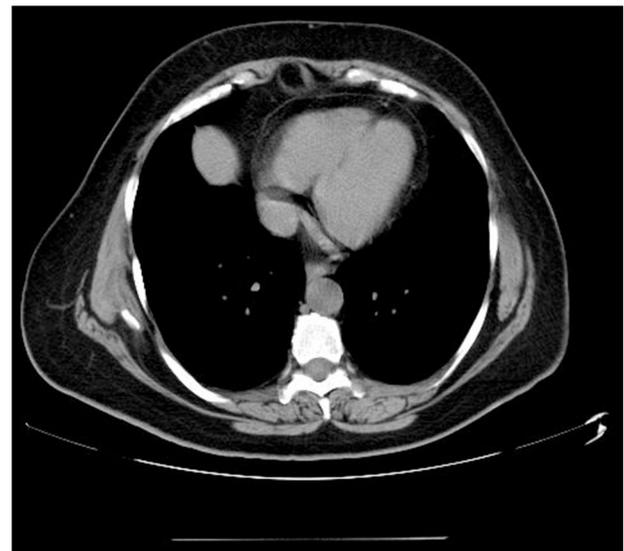


Figure 2 Chest CT scan at 6 years showing complete mass regression.

Although we have no histopathological data from our patient, we were surprised to find complete spontaneous regression of a mass of such dimensions.

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Recurrent meningoencephalitis due to allopurinol[☆]



Meningoencefalitis recurrente por alopurinol

Dear Editor:

Drug-induced meningitis is an infrequent entity but should be considered in recurrent or unexplained episodes of meningitis. It has mainly been described in association with the use of nonsteroidal anti-inflammatory drugs, antibiotics, intravenous immunoglobulins, immunosuppressants, vaccines, and intrathecal agents.¹ The literature includes 3 exceptional reports of cases related to allopurinol.^{2–4} We describe a new case of recurrent meningoencephalitis probably caused by allopurinol. Our patient was a female smoker aged 74 years, with a history of hypertension and hyperuricaemia, who was being treated with allopurinol, olmesartan/hydrochlorothiazide, and omeprazole. She was admitted to our hospital due to a third episode of language impairment and confusion. In the first episode, 2 months before, the patient was admitted to another hospital due to symptoms of disorientation and aphasia upon awakening. The patient experienced general discomfort with asthenia and headache in the days leading up to the episode. No fever was reported. A cranial CT scan yielded normal results, whereas lumbar puncture (LP) revealed pleocytosis of 104 cells (70% PMN), high protein levels (163 mg/dL), and a glucose level of 59 mg/dL. Microbiology tests (conventional culture and PCR study for HSV in the CSF; serology tests for cytomegalovirus, Epstein–Barr, varicella-zoster, *Borrelia burgdorferi*, HIV, and syphilis) yielded negative results. The patient received treatment for 14 days with acyclovir at 10 mg/kg/8 h, with symptoms resolving on the fifth day after admission. Brain MRI results were normal. Two months later, the patient was admitted a second time, with similar symptoms, but again with no fever. A new LP revealed 410 cells (95% PMN), with high protein levels (166 mg/dL) and a glucose level of 65 mg/dL. The same microbiology tests were performed as in the previous episode, in addition to a PCR for mycobacteria; all tests returned negative results. A new CT scan showed no abnormalities. The patient was treated with intravenous ceftriaxone dosed at 2 mg/24 h for

10 days, and symptoms fully resolved at 48–72 h of admission. The night of her discharge, she started experiencing headache and nausea, and again displayed confusion and language impairment the following morning. The patient was transferred to our hospital at this time. At arrival, she presented bradypsychia, disorientation, and severe mixed aphasia, with no signs of long pathway involvement, meningitis, or fever. A new LP revealed 900 cells (85% PMN), with high protein levels (207 mg/dL), and a glucose level of 48.9 mg/dL. Gram stain was negative and adenosine deaminase (ADA) level was <4 IU/L. Therefore, this was a third episode of headache, aphasia, and confusion, with increasing PMN pleocytosis in the CSF and high CSF protein levels, with full clinical resolution between episodes. We performed a new cranial MRI scan, as well as microbiology tests, antineuronal antibodies, a whole-body CT scan, and autoimmune profiling; all yielded normal results. We started treatment with cefotaxime (2 g/4 h), ampicillin (2 g/4 h), vancomycin (1 g/12 h), and tuberculostatic drugs (isoniazid, 250 mg/24 h, pyrazinamide, 1500 mg/24 h, rifampicin, 600 mg/24 h, and ethambutol, 1000 mg/24 h). Symptoms resolved 3 days after admission. Due to the low level of clinical suspicion, tuberculostatic drugs were suspended; the triple antimicrobial therapy was continued for 14 days. Once infectious aetiology of the symptoms could reasonably be ruled out, we considered the possibility of drug-induced aseptic meningoencephalitis. Considering the absence of CSF lymphocytosis, we ruled out a diagnosis of HaNDL syndrome. The patient was receiving several drugs, including allopurinol. We became aware that the patient had started treatment with allopurinol 2 months before her first admission. During her first stay in hospital, she did not receive allopurinol; this coincided with the improvement she experienced a few days later. Two months later, in her home, she resumed treatment and experienced symptom relapse just 2 days later. Again, during the second admission, treatment with allopurinol was unintentionally suspended, and her clinical symptoms again improved rapidly within a few days. We could not confirm whether the patient took allopurinol in her home the day of discharge; she was admitted the following day due to a third episode. Considering the chronology of the episodes of meningoencephalitis and their temporal association with the administration of allopurinol, we recommended replacing the drug with an alternative urate-lowering drug, febuxostat. After one year and 3 months of follow-up, the patient has remained asymptomatic and has suffered no further episodes.

The causes of recurrent aseptic meningitis include chronic inflammatory diseases, structural lesions (craniopharyngioma and epidermoid cyst), chronic

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