A rare case of papillary thyroid carcinoma with neurological presentation - a case report

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ABSTRACT

Background: Longitudinal extensive transverse myelitis, which designates inflammation of the spinal cord in more than 3 vertical vertebral segments. It is a rare presentation of paraneoplastic syndrome, usually occurring in lung and lymphoproliferative neoplasms, and even rarer in other neoplasms. Before diagnosing myelitis of paraneoplastic etiology, the aim is to exclude inflammatory, metabolic, infectious, and ischemic/traumatic causes.

Case Presentation: We present a 62-year-old man observed in the emergency department for low back pain that had lasted 2 weeks, paresthesia of the lower limbs, and severe inability to walk. He also referred sphincter anesthesia in the previous 3 days. Several examinations were carried out and the dorso-lumbar magnetic resonance imaging highlighted findings of longitudinal extensive transverse myelitis from D5-D6 to the conus medullaris. The etiological study showed papillary thyroid carcinoma as a condition for this paraneoplastic syndrome. After thyroidectomy, the patient progressively recovered from the deficits presented.

Conclusion: The clinical and imaging diagnosis of myelitis is not difficult; however, finding the underlying etiology can be challenging. A whole set of laboratory and imaging tests are necessary to confirm the cause and direct treatment with the intention of potentially reversing the neurological condition.

Keywords: Longitudinal transverse myelitis, paraneoplastic syndrome, papillary thyroid carcinoma, nuclear medicine.

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Background

Longitudinally extensive transverse myelitis (LETM) refers to inflammation of the spinal cord (SC) involving 3 or more vertebral segments vertically. The main etiology underlying LETM is neuromyelitis optica [1], followed by other inflammatory autoimmune etiologies such as multiple sclerosis, systemic lupus erythematosus, sarcoidosis, among others. There are also other etiologies such as vascular causes of SC infarction, trauma, infection, nutritional deficits, and those associated with neoplasms [2]. Paraplegia in neoplastic patients is often associated with SC metastasis and can also be associated with paraneoplastic syndromes [3]. Myelitis is a rare paraneoplastic manifestation of a neoplasm and is most common in lung and lymphoproliferative neoplasms, although it has been reported to occur in sarcomas, breast, esophageal, stomach, thyroid, ovarian, and liver neoplasms [4]. Neurological manifestations of papillary thyroid carcinoma are rare and usually occur due to cerebral metastasis, which is uncommon, or compression of nerves in the cervical region in cases of locally advanced tumors. Even rarer are neurological manifestations in a paraneoplastic syndrome associated with papillary thyroid carcinoma, as it is not, by excellence, a tumor that produces chemical signaling molecules [5].

Case Presentation

We present a 62-year-old caucasian man with hypertension, type 2 diabetes mellitus, dyslipidemia, and benign prostatic hyperplasia. He was medicated with Enalapril/Hydrochlorothiazide 20 + 12.5 mg id, Metformin 850 mg 2id, Simvastatin 20 mg id, and Dutasteride/Tamsulosin 0.5 + 0.4 mg id. The patient was admitted to the emergency room for low back pain lasting for 2 weeks and with a progressive sensation of numbness in the lower limbs and severe inability to walk. He also reported difficulty urinating and constipation over the previous 3 days. Symptoms of dysphagia, neck swelling, weight loss, cold/heat intolerance, or others compatible with thyroid dysfunction were denied.

On admission, he was hemodynamically stable, non-febrile, and had pain on hypogastric palpation. On neurological examination, he had no alterations to the upper nervous functions, cranial nerves, and upper limbs;

in the lower limbs, he had flaccid paraparesis grade 4 out of 5 (Medical Research Council scale), abolished osteotendinous reflexes, indifferent cutaneous-plantar reflex; sensory level by D12 and errors in kinesthetic sensitivity. Gait was possible with support.

Due to suspicion of bladder globus, urinary catheterization was required with an immediate output of 1,300 ml of urine. Blood tests showed increased inflammatory parameters (C-reactive protein 108 mg/l) and acute kidney injury (AKI) (serum creatinine 7.1 mg/dl). Type 2 urine with leukocyturia and nitrituria.

Cranioencephalic computed tomography showed no acute lesions or signs of intracranial hypertension. Cerebrospinal fluid (CSF) analysis by lumbar puncture showed 141 cells with a lymphocytic predominance, slight proteinuria, and no glucose consumption. Multiplex panel for viral/bacterial agents was negative; negative cultural examination. No oligoclonal bands were identified.

Non-contrast dorsal-lumbosacral magnetic resonance imaging (MRI) (Figure 1) showed an extensive area of hypersignal on T2-weighted sequences, extending from D5-D6 to the medullary cone (MC), with a pattern of centro-medullary involvement, with no association with anomalous vascular structures within the spinal canal.

Therefore, given the findings of LETM of undetermined etiology, it was decided to start Ceftriaxone+Ampicillin in meningeal doses and Methylprednisolone 1 g in order to treat the most frequent causes of myelitis: infectious and inflammatory. He was admitted for an etiological study.

During hospitalization, he underwent 7 days of antibiotic therapy and methylprednisolone 1 g for 5 days, followed by oral prednisolone in a weaning regimen from a dose of 60 mg. The lumbar puncture was repeated and the CSF showed 129 cells with a lymphocytic predominance and slight proteinuria.

An etiological study revealed negative onconeural, anti-MOG, and anti-Aquaporin 4 antibodies, a negative autoimmune study, and negative viral, parasitic, and bacterial serologies. No analytical or imaging evidence (chest CT) of granulomatous inflammatory disease. No vitamin deficits and normal serum copper and iron levels.

After managing post-renal AKI, he underwent neuroaxis MRI (Figure 2) with contrast without evidence of alterations at the cranioencephalic level and showing a hypersignal in T2 in the posterior aspect of the SC in the D12 plane and superior L1 plane. The extent of the signal in T2 was smaller when compared to the previous MRI, suggesting partial involution of the findings.

He was also screened for an occult neoplasm with a body CT scan which showed several right axillary adenomegalies, the largest measuring 19 and 23 mm in the minor axis. The study was complemented by a body positron emission tomography (PET) scan with 18F- fluorodeoxyglucose (FDG) (Figure 3) which showed a focus on intense anomalous FDG uptake in a hypodense nodular formation in the isthmus of the thyroid gland, Q.SUV max 29.9, and slight uptake in the two axillary adenopathies described on the CT scan, Q.SUV max 2.3 and 2.5. A thyroid ultrasound (Figure 4) showed a solid, hypoechogenic, and heterogeneous nodule, well delimited, measuring around 12 × 8 mm in the left isthmic region - TI-RADS 4. The two axillary adenopathy were biopsied with results compatible with reactive adenopathy. Fine needle aspiration biopsy was performed on the thyroid nodule and anatomopathological results were consistent with papillary thyroid carcinoma. Paraneoplastic LETM was assumed.

During 25 days of hospitalization, the patient remained apyretic and his neurological deficits progressively improved. At discharge, he had no motor deficits, normal tone and deep tendon reflexes were present. He preserved hypostasis by D12, with some sensation in the lower limbs

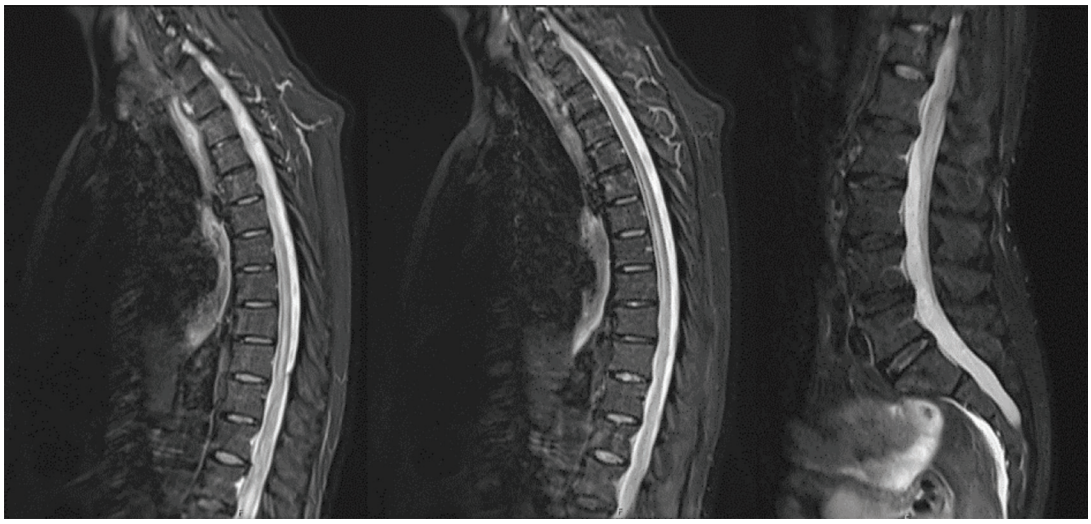

Figure 1. *Dorsolumbosacral MRI without contrast showing an extensive area of hypersignal in T2-weighted sequences, from planes posterior to the D5-D6 intersomatic disc to the medullary cone, with a predominantly centro-medullary pattern of involvement, with no obvious association with anomalous vascular structures within the spinal canal.*

up to the knees, but still no sensation in the perineal region, requiring urinary catheterization. Normal gait was possible at discharge and the patient was referred to General Surgery for thyroid neoplasm resection.

Discussion

Paraneoplastic lesions of the MC are rare and can occur before the neoplasm is detected [6]. Paraneoplastic myelopathy manifests itself in two ways: extensive necrosis of the MC with an ascending transverse lesion or irregular multifocal necrosis predominantly of the white matter of the MC, similar to multiple sclerosis [7].

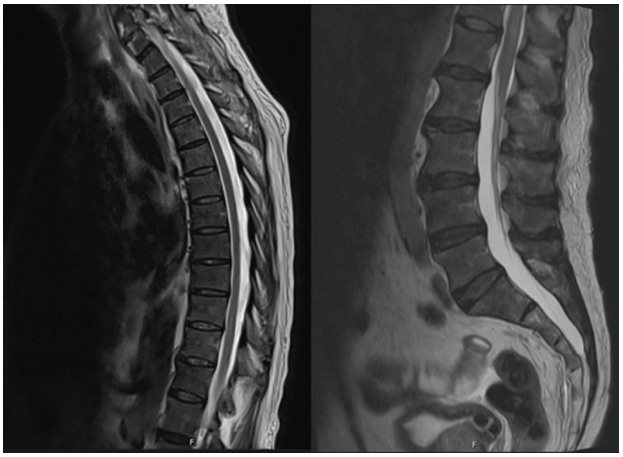

Figure 2. *Neuroaxis MRI showing hypersignal in T2 in the posterior aspect of the spinal cord in the plane of D12 and superior plane of L1, suggesting partial involution of the findings compared to the MRI at admission.*

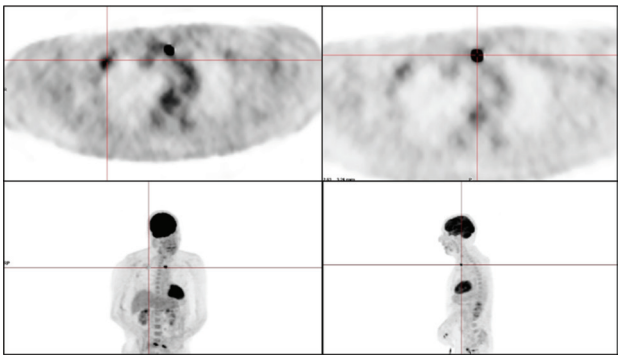

Figure 3. *Body PET scan with 18F-FDG showing a focus of intense anomalous FDG uptake in a hypodense nodular formation in the isthmus of the thyroid gland, Q.SUVmax 29.9, and slight uptake in the two axillary adenopathy described on CT, Q.SUVmax 2.3 and 2.5.*

Given the patient's age, a diagnosis of myelitis as a presentation of neuromyelitis optica or multiple sclerosis would be unusual, as these are diseases with rare presentations after 50 years of age, yet they were still ruled out. On the other hand, an infectious etiology was suspected due to increased inflammatory parameters upon admission, but these were explained by a urinary tract infection within the context of functional obstructive uropathy. A vascular etiology, such as spinal cord infarction, could also be considered given the patient's age and multiple cardiovascular risk factors, but the MRI did not show typical imaging findings. Myelitis due to trauma was excluded through clinical interview.

MRI, lumbar puncture, and serological analysis should guide diagnostic suspicion and treatment. CSF analysis usually allows us to infer an infectious, inflammatory, or autoimmune etiology, but it can also leave a diagnostic void when it is nonspecific, as in the present case.

Although onconeural antibodies were negative, this did not exclude paraneoplastic syndrome, as they can frequently be negative in patients with non-classical symptoms of paraneoplastic syndrome, as was this case [8]. Negative anti-MOG and anti-Aquaporin-4 antibodies helped to exclude demyelinating disease, particularly Multiple Sclerosis, as an etiology of myelitis.

Due to pleocytosis in the CSF, empirical antibiotic therapy in meningeal doses was decided on until culture results were obtained. Given the inflammatory findings on the initial MRI scan, Methylprednisolone mega doses were initiated and imaging and clinical improvement were obtained. Once we found a thyroid neoplasm and considering that all the other studies were negative, including the search for anti-aquaporin 4 antibodies and anti-MOG antibodies, a paraneoplastic etiology was presumed.

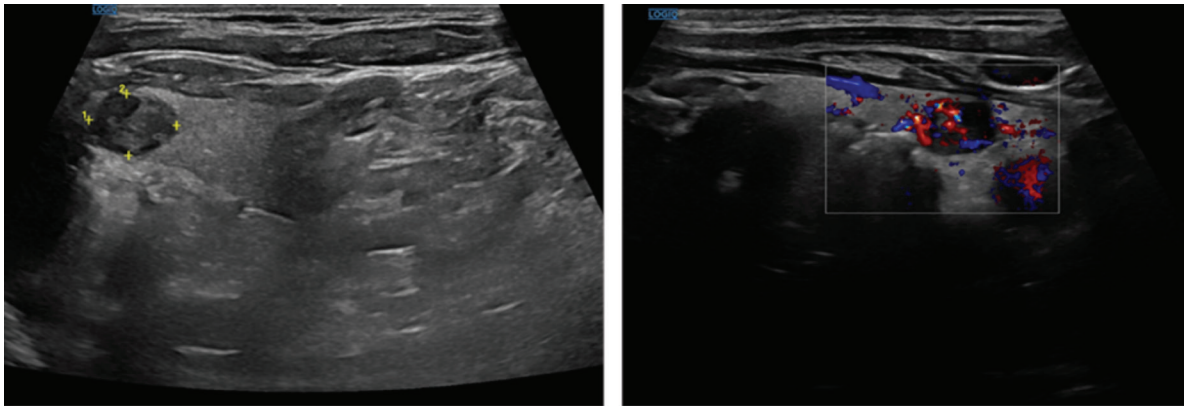

Figure 4. *Thyroid ultrasound showing a solid, hypoechogenic, and heterogeneous nodule, well delimited, measuring around 12 × 8 mm in the left isthmic region, classified as TI-RADS 4.*

Conclusion

Given the initial finding of LETM with no defined cause, leading to severe neurological deficits, it was not unreasonable to start empirical antibiotic therapy in meningeal doses and corticosteroid pulses in mega doses in order to reduce the inflammatory load and save time in treatment if bacterial infectious cause is found. With the course of corticosteroid therapy, the patient showed slight improvements, and although only a minority improved after treatment for the underlying neoplasm, a total thyroidectomy was performed and the patient progressively recovered all the deficits he had at admission. The patient had semiannual surveillance for recurrence of neurological deficits and was started on levothyroxine supplementation.

This case underscores the importance of including occult neoplasms in the differential diagnosis of extensive transverse myelitis and early identification and management of rare paraneoplastic syndromes related to thyroid cancer.

What's new?

Myelitis can be a life-threatening condition or cause serious neurological changes. Differential diagnosis can be challenging and is essential for targeted treatment. Other associated organic dysfunctions must be resolved simultaneously.

Conflict of interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

Funding

None.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Ethical approval

Ethical approval is not required at our institution to publish an anonymous case report.

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Summary of case

1	Patient (gender, age)	62 years, male
2	Final diagnosis	Paraneoplastic Longitudinal extensive transverse myelitis
3	Symptoms	Flaccid paraparesis and errors in kinesthetic sensitivities
4	Medications	Antibiotics, corticosteroids
5	Clinical procedure	Total thyroidectomy
6	Specialty	Neurology