Jurnal Medical Aradean (Arad Medical Journal)
Vol. XXI, issue 2, 2018, pp. 22-23

PARANEOPLAZIC NEUROLOGY SYNDROME

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ABSTRACT. The patient is a 64-year-old male, hypertensive, expressing severe bilateral paresis, more pronounced on the left side with a staggering character, associated with ataxia, leading to impossibility in conducting daily activities as a driver. The performed investigations reveal cervical medullary canal stenosis as well as NCV and EMG with an suggestive pattern of chronic demyelinating inflammatory polyneuropathy. The patient does not tolerate corticosteroids, thus undergoes cervical neurosurgery with a temporarily favorable response at first followed by a worsening neurological clinic condition, subsequent recurrence of symptoms in combination with weight loss, loss of appetite, as well as depressive syndrome. Tomographic investigations conducted during development reveal a thickened gastric wall with suspicion of gastric neoplasm confirmed in subsequent surgery. The conclusion of the complex case, ackwnoledges the hypothesis of a complex paraneoplastic neurological syndrome in a patient with staggering neurological symptomatology with differential diagnoses which raised various discussions regarding case management.
KEYWORDS: paraneoplazic, ataxia, paresthesia, polyneuropathy.

INTRODUCTION

Patient, without significant personal pathological history, known as hypertensive, is initially present in the polyclinic for a bothersome, parenteral bilateral paresis neurological symptomatology more pronounced on the left side. The patient is sent to various investigations, rising various diagnostic hypotheses. Clinical evolution is unfavorable under various drug and neurosurgical therapies, the patient's case becoming much more complex, gastric oncology pathologies discovered being possibly responsible for clinical manifestation of neurological symptoms.

CASE DESCRIPTION

A 64-year-old male, taxi driver, known hypertensive, with no other semminificative heredo-collateral or pathological history, accuses paresthesia in both inferior limbs, more pronounced in the left side, later with extension to the upper limbs and left hemithorax, onset about 1-2 months before the progressive ascending trend. The patient performs a series of investigations in ambulatory as well as a cranial CT: no pathological changes, heart ultrasound normal, abdominal ultrasound, normal, normal biological samples. As the symptoms become more pronounced causing the patient to be unfit to perform his activities as a driver, the patient presents himself for admission to the neurology department. Cervical spine MRI shows the enhancement of physiological cervical lordosis, cervical disseminated osteophytic changes, inferior cervical zig-apophysis arthrosis. C2, C3 and C4 rear disc protrusion causing an imprint on the thecal sac, without radicular conflict or identifiable bone marrow compression. C5 important intra and extraforaminal circumferential disc bulge causing an imprint on the thecal sac, moderate spinal cord reduction and anterior medullary cord compression, foraminal narrowing with bilateral radicular damage. C5-C6 Modic II. C6: intra and extraforaminal circumferential bilateral disc bulge, more pronounced postero-lateral left side, causing an imprint on the thecal sac, reduction of the spinal canal (AP diameter of 5-6 mm at this level) moderate / significant compression of the anterior left lateral medullary cord, foraminal narrowing and bilateral radicular damage, more pronounced on the left side, Modic II C6-C7. No detectable change in bone structure in the segment considered suspicious. Without hyper intramedullary signal showing in the cervical cord. Cervical spine MRI

conclusions: C5-C6 discopathy with moderate anterior medullary cord compression and bilateral radicular conflict, more expressed on the left side. Lumbar puncture reveals mild proteinorrhagia without significant alterations. Subsequently EMG and NCV are performed. EMG, NCV: study reveals conduction blocks on bilaterally common peroneal and right ulnar nerves. NCVs are asymmetrically lowered on all the examined nerves. No sensitive responses are obtained. EMG obtains a MUP with subacute chronic neurogen denervation pattern in all the examined muscles in inferior and superior limbs.

Conclusions of NCV and EMG: CIDP (Chronic inflammatory demyelinating polyneuropathy)

The patient was discharged with the recommendation of corticosteroid treatment with prednisone and with indication of neurosurgical consultation. Corticotherapy treatment was poorly tolerated by the patient with inappetence, nausea, vomiting, sincopal episode at home, and was subsequently progressively interrupted. Taking into account that the neurological symptoms become progressively worse, the decision of a neurosurgical intervention for cervical canal stenosis is made in Timisoara University Center. The postoperative progression is favorable with partial improvement of neurological symptoms, but for a temporary period of several weeks, with the help of recovery and kinetotherapy, followed by a subsequent and more pronounced return of paraesthesic symptomatology associated with ataxia. Over time, the patient experiences digestive symptoms like inappetence, nausea, weight loss, associated with a progressive depressive syndrome.

Taking into consideration the tremendous digestive symptoms, it is decided to perform an abdominal CT scan where the gastric body tumor is highlighted.

Abdominal CT with constrast highlights liver with right lobe longitudinal diameter of 14 cm without focal lesions without inner or extrahepatic bile duct dilation, spleen,10 cm long without focal lesions. Above the pancreatic corporeal region, evolving cranically and coming into contact with the small gastric curvature, a round oval shaped nodular formation measuring 70/56 mm diameter lesion with inhomogeneous contrast medium uptake.

The patient undergoes a surgical procedure where a gastric formation with macroscopic malignancy is revealed, which is then supported by the microscopic examination. The subsequent evolution of the patient was unfavorable, with multiple post surgical infectious complications, the patient dying shortly after surgery.

The particularity of the case consisted in the neurological symptomatology onset, difficult to assign to clear characteristics of medullary canal stenosis or chronic demyelinating inflammatory polyneuropathy, which posed problems of therapeutic conduct, later, in about 6 months time, the patient being diagnosed with advanced stage gastric cancer.

DISCUSSIONS

Paraneoplastic neurological syndromes are clinical manifestations in the neurological sphere, whether of a central or peripheral type, occurring in the neoplastic context, often being primary clinical manifestations prior to the first manifestations of primary neoplasia and without being determined by neurological secondary determinations or associated oncology therapy. It is very important to frame neurological symptomatology in the sphere of paraneoplastic diseases because by recognizing them, it is possible to perform specific analyzes of antineuronal antibodies that are suggestive of certain neoplasms, being thus closer to the etiological diagnosis of the symptomatology.

The production mechanism of neurological symptomatology is an immunological type with primary neoplasm being its starting point. In recent years, a large number of antineuronal antibodies have been detected which, by cross-reaction with neuronal protein-like tumor antigens, produce central neurological syndromes (cerebellar degeneration, encephalomyelitis, NMDA cerebellar encephalitis, necrotizing myelopathy, stiff-person syndrome) or syndromes which affect the visual system (optic neuritis, retinopathy, uveitis) or syndromes affecting the peripheral nervous system (sensory neuronopathy, subacute or chronic peripheral motor-sensory neuropathy, autonomic neuropathy, peripheral nerve hyperexcitability) or syndromes affecting neuro-muscular junction and muscles (myasthenic Eaton-Lambert syndrome, myasthenia gravis, dermatomyositis, acute necrotizing myopathy, cachectic myopathy)

CONCLUSIONS

In retrospect, the patient's symptomatology, which was purely neurological at first, posing difficult problems of differential diagnosis, can be labeled as being paraneoplastic neurological syndrome, primary neurological followed by clinical digestive due to the detection of advanced stage gastric cancer.

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