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Case Report

Subacute Sensitive Neuropathy Revealing Lung Cancer About of a Case

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Abstract

Lung cancer is the second most common malignant disease worldwide. It is estimated that around 2.2 million new cases of lung cancer are diagnosed each year. The clinic is very polymorphous, the neurological manifestation can be part of a paraneoplastic syndrome or secondary damage by tumor infiltration. The damage can be central or be central or peripheral. Here we report a case of a known smoking patient who presented with peripheral neurogenic syndrome which revealed lung cancer.

Keywords: Sensitive Neuropathy; Lung Cancer

Introduction

Supported by the Global Burden of Disease (GBD) Study, several recently published articles report cancer statistics, including incidence, mortality and disability-adjusted-life-years (DALYs), for 195 countries and territories from 1990 to 2017. Overall, lung cancer was the second most common cancer, after non-melanoma skin cancer, with 2.2 million incident cases in 2017 [1]. Lung cancer was also the most common cause of cancer death and DALYs for males, and the second leading common cause of cancer death and DALYs for females—with a total of 1.9 million deaths and 40.9 million DALYs in both sexes [1].

Neurological damage falls within the framework of a paraneoplastic syndrome. Paraneoplastic neurological syndromes (PNS) are rare (approximately one in 300 cancer patients). They depend on a remote action of a malignant tumor on the nervous system involving autoimmune mechanisms usually evidenced by the presence of specific autoantibodies [3]. These antibodies therefore play a crucial role in the diagnosis of SNPs. In 2004, an international group of experts proposed a series of criteria for the diagnosis of these syndromes [2]. However, over time, the number of identified antibodies has increased considerably and things have become more complex with the observation that the most recent antibodies, which for the most part recognize membrane antigens, are frequently observed outside of any cancer. The development of easily accessible commercial kits has also made the diagnosis of SNPs easier in daily practice but due to problems of sensitivity and specificity has opened the way to new difficulties. It is for these reasons that the same group of experts decided in 2021 to update the 2004 criteria.

Peripheral neuropathy is a rare form, hence the interest of this case.

Case

We report the case of a 65-year-old adolescent with smoking history, admitted to the neurology department of Dalal Jamm hospital for lower limb paresthesia. The symptoms began several years ago with the occurrence of repeated falls associated with walking difficulties for which they consulted a hospital center where medication was prescribed with amendment for a week.

Follow-up of recurrences of falls associated with paresthesias. After going through various primary and secondary medical structures as well as traditional healers for about 15 months without amendment, hence the reason for consultation in neurology for support. On examination, the patient was in good general condition, mucosa of normal color, anicteric, supple calf, no objectified edema of the limbs. Vital parameters were normal, with no disturbance of consciousness, A motor deficit of the 4 limbs with a segmental muscle strength of 4/5 in the 4 limbs according to the scale [Medical Research consul] MRC at the distal levels - Abolition osteotendinous reflexes in the four limbs.

Faced with the onset of progressive paresthesias associated with chronic osteotendinous areflexia, a diagnosis of a Neurogenic syndrome was made.

The electroneuromyography of the four limbs had objectified a sensory neuropathy of the four limbs from which the etiological research with an infectious, inflammatory blood test without particularity and the thoracoabdomino-pelvic scanner had highlighted: A suspicious lung lesion with right alveolar and diffuse reticular opacity. The biopsy was in favor of small cell lung cancer; chemotherapy had been instituted, the evolution was characterized by the occurrence of death two years later in a picture of motor deficit associated with convulsive seizures then a coma the brain CT showed a secondary cerebral localization.

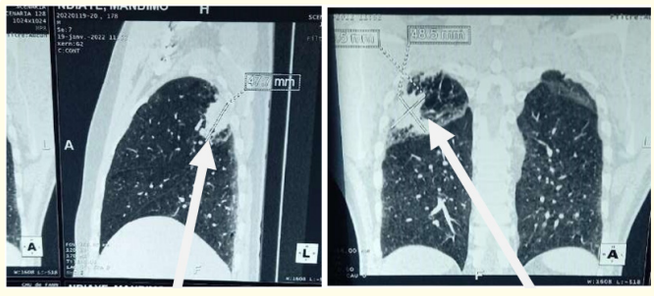

Figure 1: CT THORACIC: Right alveolar opacity and diffuse reticular.

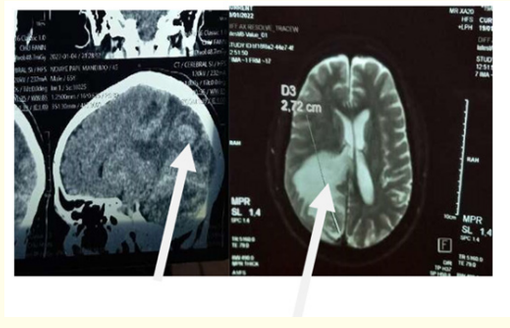

Figure 2: CT ENCEPHALIC: secondary localization.

Discussion

The etiology of neuropathies in malignant patients is considered to be multifactorial, including metabolic and nutritional deficits, but also including unknown factors, which typically appear at the advanced stage of the underlying disease [4,5]. The causation of neuropathies in elderly patients is particularly controversial, as many possible causes of peripheral nerve damage are already present [6]. The mechanism of damage is associated with onconeural antibodies and onconeural antigen-specific T cells. However, the absence of known onconeural antibodies in patients with typical clinical manifestations of neurological damage does not exclude the possibility of paraneoplastic neuropathy. These forms of neuropathies often prefigure the onset of the underlying disease and sometimes, independent of the cancer, create a high level of functional disability (e.g., subacute sensory neuropathy) [8].

A diagnosis of subacute sensory neuropathy (SSN) can be made when the following criteria are all met: subacute progression in less than 12 weeks, severity of at least 3 (moderate disability) assessed by a Rankine score, onset of numbness with often pain and sensory disturbances, impairment of the arms and legs (so-called glove and stocking area), and often asymmetry at the beginning. In addition, electrophysiological findings showed the significant disruption of sensory fibers and, in at least one sensory nerve, the absence of sensory nerve action potentials from the sensory nerve [9].

Motor nerves may also be minimally disturbed. Tendon reflexes are depressed or absent [10] and patients often present with autonomic, cerebellar, or cerebral abnormalities [8]. SSN occurs in approximately 75% of patients with paraneoplastic encephalomyelitis, is predominant in 50%, and is clinically pure in 25% [11]. In a review of 26 patients with paraneoplastic SNS, 19 (73%) had SCLC [12]. There was a striking female predominance [10].

In general, patients with PNS metastases have a poor prognosis. However, it is important to get an accurate diagnosis as early as possible because therapeutic strategies can significantly improve pain and stabilize or even ameliorate neurological deficits. Malignant nerve sheath tumors malignant nerve sheath tumors arise from plexiform neurofibromas or normal peripheral nerves [15]. Nearly 50% of these tumors occur as part of neurofibromatosis type 1. Radiation therapy may be a causative factor with or without neurofibromatosis [16]. Malignant transformation of plexiform neurofibromas into malignant nerve sheath tumors have been associated with p53 and INK4a gene mutations and aberrant Notch pathway signaling [17].

The clinical presentation depends on the peripheral nerve involved, but severe pain and rapidly growing tumors are common and suggestive of malignant transformation. Overall, the prognosis is poor. Leptomeningeal metastases Leptomeningeal infiltration occurs in 5-15% of patients with solid tumors, primarily in those with breast, lung, head, or melanoma cancer, or in those with cancer gastric [18]. About 5-10% of lymphomas spread to the leptomeninges, especially acute lymphoblastic leukemia, and lymphoblastic lymphoma and Burkitt 's lymphoma for which prophylactic treatment is warranted [19]. With other lymphomas, advanced age,

high initial tumor grade, or location of the tumor in the testicle, sinuses, bone marrow, blood, or digestive tract are associated with an increased risk of infiltration meningeal [18]. Regardless of tumor type, involvement of the cranial nerve and inferior spinal roots is typical [20]. The most commonly affected cranial nerves are the oculomotor nerves followed by the facial, optic, and auditory nerves. Spinal root infiltration induces motor deficit, sensory loss, areflexia and sometimes root pain. The progressive extension of symptoms with a multiradicular distribution is highly suggestive of leptomeningeal metastases MRI with gadolinium infusion is the best radiological examination [21].

Linear and nodular contrast of meninges and sulci, ventricular ependyma, basilar cisterns, tenta, cranial nerves, cauda equina, and hydrocephalus are indicative of meningeal metastases, but none of these radiological effects signs is specific because they can occur with other chronic meningitis. The key point in diagnosis occurs when the cerebrospinal fluid examination is positive for malignant cells. However, to detect 80-90% of malignant cells, three lumbar punctures may be needed [22]. Adequate cerebrospinal fluid volume and a short delay between lumbar puncture and analysis greatly minimize false negative results [23]. To detect malignant cells in carcinomas, in situ hybridization20 may be useful. When lymphoma is suspected, immune phenotypic analysis of cerebrospinal fluid lymphocytes or immunoglobulin light chain gene rearrangement study [24] can be used to show monoclonal. When a neurological disorder occurs in patients with no known malignancy and repeated cerebrospinal fluid examinations remain negative, meningeal biopsy should be considered. Plexus metastases About 1% of cancer patients develop metastases in their plexus.3 Head and neck tumors can extend to the cervical plexus and down to the base of the skull and invade the cranial nerves lower. Breast tumor and apex lung cancer infiltrate the brachial plexus, while gynecological, prostate and colorectal cancer and pelvic sarcoma invade the lumbar and sacral plexus.

Conclusion

The paraneoplastic syndrome represented a frequent entity characterized by various clinical manifestations, the symptomatology of which may be present a few years even before the symptomatology, hence the importance of doing a good semiological analysis in order to properly carry out a reliable diagnostic approach.

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