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Case Report

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Guillain-Barré syndrome as a clinical manifestation of Burkitt's lymphoma case report

Abstract

A 17-year-old male who began with a 3-month history of focal motor seizures with no apparent cause that warranted antiepileptic treatment. He presents a diagnosis of bacterial meningitis for which treatment is given with antibiotics and steroids with improvement. In April 2022, he began with symmetrical, ascending distal weakness of the lower limbs, with involvement of the upper limbs and asymmetric bilateral facial paralysis, with a diagnosis of Guillain syndrome. Barre variant AMSAN confirmed with electromyography, received treatment with immunoglobulin 5 doses without improvement and worsening of muscle weakness at the end of it. Magnetic resonance imaging shows neurolymphomatosis-type lesions of the cervical plexus and cauda equina with enhancement of vertebral bodies and splenomegaly, a T5 vertebral body biopsy was performed with histopathological result of probable Burkitt lymphoma vs. lymphoblastic lymphoma.

Male who presented with focal motor seizures with no apparent etiology that was controlled with anticonvulsants and was discharged, later readmitted with data compatible with bacterial meningitis, with a good response to treatment initially, however three months later, he presented distal weakness with a diagnosis of Guillain barre AMSAN variant, receives treatment with immunoglobulin and presents clinical worsening, in magnetic resonance imaging with neurolymphomatosis-type lesions of the cervical plexus and cauda equina, with reinforcement in vertebral bodies, for which a T5 vertebral body biopsy is performed, with histopathological result of lymphoma from Burkitt.

Keywords: Guillain Barre, Burkitt's lymphoma, neurolymphomatosis

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Introduction

Guillain-Barre syndrome is the most common cause of flaccid paralysis in the world. lit is a polyradiculoneuropathy of autoimmune origin and with a heterogeneous clinical presentation. The classic presentation is an ascending flaccid paralysis mainly associated with areflexia, although the pattern of weakness It is not exclusive to limbs, but can extend to muscles iinnervated by cranial nerves, respiratory muscles and have autonomic manifestations. The diagnosis of Guilllain-Barre syndrome requires the presence of flaccid paralysis and a decrease or absence of muscle stretch reflexes that cannot be explained by another cause.[1] There are entities that can be imitators of Guillain-Barre syndrome in their clinical presentation. In a 2018 publication that describes 12 cases of patients with pathologies that mimiicked an acute polyradiculoneuropathy syndrome, a 57-year-old patient is rellated.

With loss of sensiitivity in patches and neurophysiological result with axonal length dependent polyneuropathy, sensory and motor, secondary to meningeal metastases secondary to liymphoma, a diffuse large B celll lymphoma among other diagnoses of the rest of the cases such as Wernicke's encephalopathy, vasculitic neuropathy, porphyriia acute intermiittent, neuropathy induced by treatment for diiabetes mellitus HIIV-related chronic inflammatory demyelinating polyneuropathy.[2]

Neurolymphomatosis is known as that entity in which there is invasion and infiltration of the endoneurium by lymphoma cells. This can affect different segments of the periipheral nervous system such as cranial!nerves, nerve roots, cervical and lumbosacral plexuses and peripheral nerve.[3] Cases of invasiion into the endoneurium by diffuse large B-cell lymphoma are generally reported, although rare lymphoma have been reported.[4] In a work publiished in 2019, they make an association of 23 published cases of neurolymphomatosis and of these, 21 cases (92.3%) were secondary to B cell lymphoma, and two cases related to T3 cell lymphoma.

Aim

Case presentation

17-year-old male, resident of a rural community in central Mexico, student, and no significant family or personal history. First hospitalization in December 2021 in a rural hospital due to focal motor seizures that became bilateral on 3 occasions lasting 5 minutes, with 40-miin intervals between each one, was approached with simple and contrasted head tomography and general laboratories which were reported as normal, the reason why their diischarge was decided hospital with treatment based on magnesium valproate and an electroencephalogram is requested for follow-up by outpatient consultation.

Second hospitalization in the same month, December 2021, due to very intense headache, accompanied by diplopia, photophobia, fever of 38 degrees Celsius and meningeal siigns, with simple and contrast-enhanced tomography without alterations, and without iinfectious focus demonstrated by laboratories or office and with biometry hematic without alterations of the cell lines in all those carried out from His first hospitalization, it was decided to perform a lumbar puncture, and the result of cerebrospinal fluiid showed protein of 111.8 mg/di, with pleocytosis of 450 cells x mm3, of which 70% are polymorphonuclear, and hypoglycorrhachiia of 5 mg/dl, with negative gram stains, diagnosis of central nervous system infection is suspected and he is treated with empiriical antibiiotic therapy as welll as dexamethasone. An electroencephalogram was performed during this hospital stay, which showed right frontal focal epileptic activity

with secondary generaliization. Due to improvement, the patient was discharged home with antiepileptic treatment.

In February 2022, the patiient was referred to a speciialty hospital for evaluation by the neurology service due to distal weakness of the symmetrical, ascending lower limbs, with involvement of the upper limbs and asymmetric bilateral facial paralysiis, and on examinatiion we found him with VII asymmetric bilateral infranuclear muscle, force 2/5 distall and proximal lower limbs and 3/5 distal and proximal!upper limbs, as well as generalized areflexia and apalesthesia, so a neurophysiological study was decided which reported a sensory and motor polyradiculoneuropathy, an axonal variant compatiblle with Guillain Barre Syndrome type AMSAN. (Figure 2)

Based on this diagnostic suspiicion, a lumbar puncture was performed, which showed the foUowing results: 240 cells x mm3, 100% mononuclear predominance, protein 164.5 mg/dl, gllucose 2 mg/dl, with gram negative stain, acid fast bacilli negative and negative chinese Ink, a panel of PCRs for viral, bacterial, fungal and parasitic meningitis was performed, which was negative, and cernbrospinal fluid was sent for cytology for pathollogy. Whiile waiting for the result of the pathology, we performed magnetic resonance imaging of the simple neuroaxis (Figure 1) and contrast with which we demonstrated images suggestive of neurolymphomatosis of the cervical plexus and at the level of the cauda equina, the C4 and C5 vertebral bodies with heterogeneous siignal intensity of the bone marrow involving both pedicles with heterogeneous enhancement to the administration of contrast with gadolinium, T11 vertebral body to the administration of contrast with gadolinium presents sliight enhancement homogeneous, both kidneys with increased volume, with lobulated edges and heterogeneous parenchyma due to multiplle wedge-shaped lesions with an infiltrative appearance, which after the administration of contrast present heterogeneous reinforcement, iincireased spleen volume with a maximum longitudinal diameter of 13.5 cm.

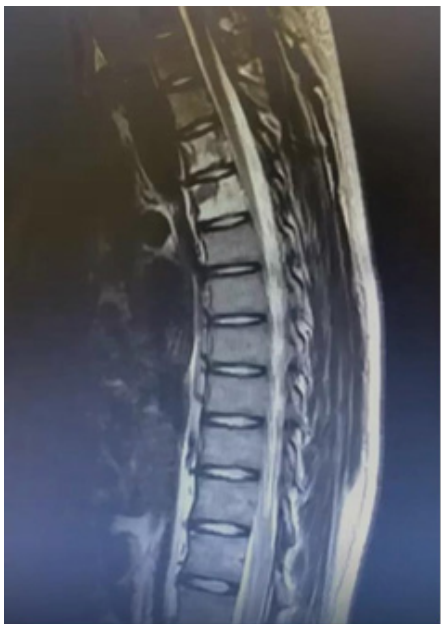

Figure 1 Simple MRI and neuroaxis, lateral view.

The preliminary report from the pathology service in relation to the cytology of the cerebrospinal fluiid was of infiltratiion of malignant cells (Figure 2), so it was decided to take a biopsy of the pedicle of the T5 vertebral body, and the histopathological result was probable Burkitt's lymphoma vs lymphoblastic lymphoma.

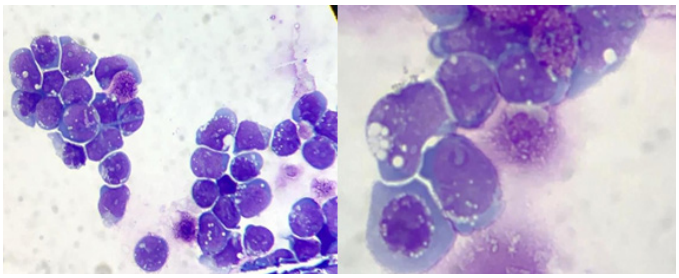

Figure 2 Cerebrospinal fluid showing monomorphic infiltrate of lymphocytes of moderate size with fine immature chromatin and basophilic cytoplasm, highlighting the presence of vacuoles in a perinuclear arrangement.

Upon evaluation by the hematology service, it was decided to perform a bone marrow aspirate, whiich was positive for infiiltration due to a lymphoproliferative process with a positiive immunophenotype for CD 10 +++, CD20 +++, CD 79 A+, BCL2-2, +++, Kl 67 40%, DTT - which guides the Diagnosis of Burkitt lymphoma, chemotherapy regimen with Cytarabine and intrathecal methotrexate was started.

Discussion

Burkitt lymphoma is an aggressive mature B-cell non-Hodgkin lymphoma characterized by translocation and deregulation of the MYC gene on chmmosome 8, and according to the world health organization (WHO) revision of the classification of hematological malignancies. It can present as a leukemoid form corresponding to Burkitt cell acute lymphoblastic leukemia or Burkitt leukemia.[5] The three main clinical presentations are endemic, sporadic and associated with immunodeficiency[6] It represents less than 5% in adults and up to 40% iin children, and the sporadic presentation involves extra nodal sites such as the gastrointestinal tract, bone marrow and system. Central nervous system,[7] in addition to unusual presentations of Burkitt lymphoma with acute apendicitis.[8] In the central nervous system, hematological malignancies can be accompanied by paraneoplastic neurological syndromes; however, this presentatiion is usually rare and affects less than 0.01% in patients with cancer. Burkitt lymphoma originates from the germinal center of B cells that express lgM, BCL6, CD1o, CD20, CD45, and CD79a, but not BCL2 or TdT; Ki67 expression reflects proliferation.

Morphollogical characteristics and immunophenotype often allow an earlly diagnosis, but genomic lesions are needed to differentiate Burkitt lymphoma from other high grade pathologiies such as: Burkitt-type lymphoma with 11q aberration, high-grade Bcell lymphoma (HGBL) with MYC, BCIL2 and/or BCIL6 rearrangements, other unspecified (NOS), and diiffuse large B-celll lymphomas with MYC rearrangements, iin many cases Burkitt-like lymphoma (BLL) shares phenotypes intermediate between diffuse large B-cell lymphoma and classic Burkitt lymphoma, which makes the histopathological diagnosiis difficult, so currently a high growth fractiion with Ki67 of more than 99% is required for the diagnosis of Burkitt-type lymphoma. In contrast to Burkitt leukemia, it does not express surface immunoglobulins and are usually TdT+,[9] which guiides the immunohistochemical diagnosis of Burkitt lymphoma in the patient.

The most important characteristic genetic expression iis the role of the MYC oncogene, the most frequently mutated (70%) in Burkitt's lymphoma, and the profile of mutations in Burkitt's lymphoma, which is a transcription factor that promotes growth and prolliferation through several effector genes (>10% of all genes), and the MYC6 translocation alone is not sufficient for the oncogenesis of Burkitt's

lymphoma, double hit lymphoma since it simultaneously sensitizes the cells to apoptosis, so mechanisms operate additional protectors for the uncontrolled malignant proliferation characteristic of high-grade lymphomas, for example in "double hit" lymphomas,[10] MYC rearrangements co-occur with the BCIL2 or BCL6 translocation, leading to a more aggressive clinical course.[11,12.]

Acknowledgments

None.

Conflicts of interest

No conflict of interest.

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