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VERTIGO AS AN INITIAL MANIFESTATION OF CHRONIC CEREBELLAR ABSCESS - CASE REPORT

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Abstract

Chronic cerebellar abscess is a rare clinical condition, considering the protective function of the blood-brain barrier. The prevalence of brain abscess is increasing with the increase in the number of people living with HIV, especially abscesses caused by a fungal infection.

We present the case of a 67 year old male patient with acute onset of dizziness, associated with nausea, vomiting and unstable gait. Initial neurological examination revealed dysarthric speech, with discrete central paresis of the facial nerve, latent left-sided ataxic hemiparesis. In order to clarify the etiology of the expansive change in the left cerebellum, a series of examinations were performed. MRI of the brain was performed with IV contrast, which showed an oval intraaxial lesion with a maximum diameter of 20 x 16 mm, on the left cerebellar side. Routine biochemical analyzes was normal, Immunoassay analysis of serum detected a multiple increase in the serum concentration of Immunglobulin E. In consultation with an infectologist, several samples of cerebrospinal fluid were sent for microbiological, parasitological (antibodies to toxoplasmosis, echinococcus and cysticercosis) and serological analysis. The patient was referred to a hematologist again, when a PET scan was performed which showed the presence of metabolically inactive lymph nodes, the same with benign characteristics. A craniotomy and surgical extirpation of the lesion were performed in consultation with a neurosurgeon.

The treatment of brain abscesses is multidisciplinary and includes: a neurologist, infectologist, radiologist, neurosurgeon and an internal medicine specialist

Key words: brain abscess, magnetic resonance imaging

Introduction

Chronic cerebellar abscess is a rare clinical condition, considering the protective function of the blood-brain barrier. The prevalence of brain abscess is increasing with the increase in the number of people living with HIV, especially abscesses caused by a fungal infection. Almost half of the cases of brain abscess (45-50%) are due to direct spread of the infection from the surrounding organs — subacute or chronic otitis media, mastoiditis, sinusitis, or infection of dental structures. In 25% of cases it is caused by a hematogenous spreading of the infection in the vascular area of arteria cerebri media. These types of abscesses are often associated with congenital heart defects, pulmonary arteriovenous malformations, chronic lung infections (eg. abscess, epyema, bronchiectasis), skin infections, and immunodeficiency conditions, transplantation, and HIV infection [1].

Facial injuries and open skull fractures account for about 10% of abscess cases. The availability of rapid neuroimaging diagnostic methods (CT/MR), as well as the discovery of new antimicrobial drugs have reduced the mortality rate to 5-15%. However, there is an increased risk in individuals over the age of 60 with a history of stroke, pneumonia, hepatitis or meningitis.

The most common clinical manifestations of brain abscess are headaches, changes in mental status, focal neurological deficits, nausea and vomiting, dizziness, fever, seizures, nuchal rigidity, and papilledema.

Case report

We are going to present the case of a 67 year old male patient with acute onset of dizziness, associated with nausea, vomiting and unstable gait. There is no information about previous febrile state,

upper respiratory infection, or constitutional symptoms such as weakness and malaise, other than the loss of about 15 kg of body weight over a period of one year. Two weeks before the onset of symptoms, he had had surgical extirpation of an enlarged and inflamed lymph node in the right inguinal region (with an increase in inflammatory markers CRP). A CT scan of the brain showed an oval change in the cerebellum, which was interpreted as a secondary deposit in the brain (with no known primary malignancy) and the patient was referred to an oncologist. The oncologist ruled out the possibility of malignancy. Additionally, MRI of the brain was performed with IV contrast, which showed an oval intra-axial lesion with a maximum diameter of 20 x16 mm on the left side, in the area of the fossa crania posterior, and in the projection of the middle cerebellar peduncle. It showed irregularly marked surrounding vasogenic edema, which had a compressive effect on the cerebellum on the left side as well as on the brainstem, predominantly on the left pons, but edema was also found at the level of the left segment of the mesencephalon and medulla oblongata. The described change is radiologically most similar to toxoplasmosis, but does not rule out granulomatosis, parasitosis, or neoplasm.

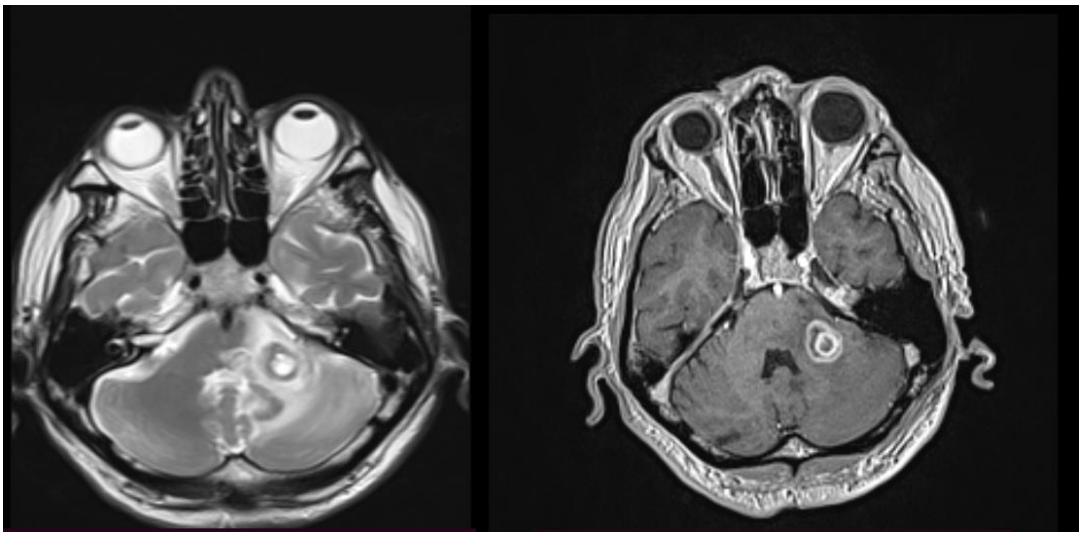

Fig. 1. MRI scans of the brain with i.v native and postcontrast series

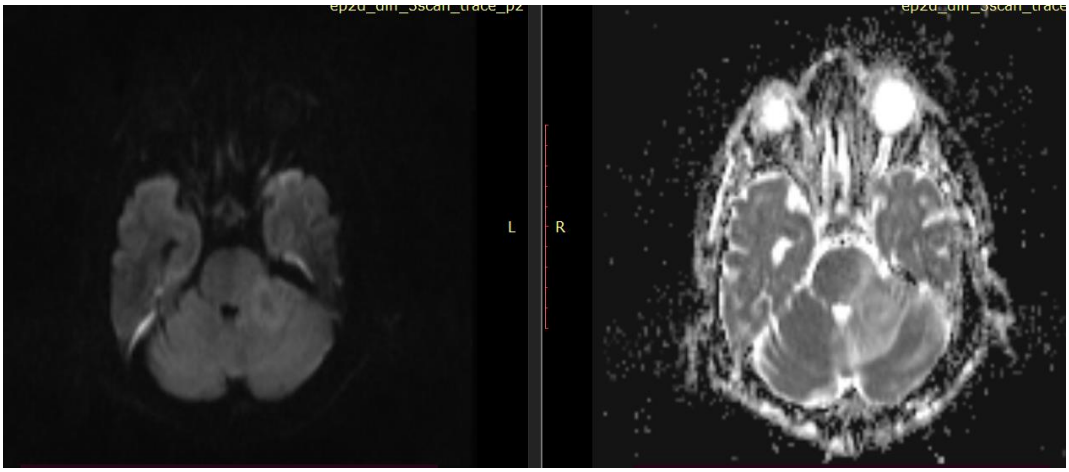

Fig. 2. MRI scans of the brain

The patient has not provided information neither about allergic diathesis, frequent infections or immunodeficiency conditions, nor about traveling in the tropic/ subtropic regions.

Consequent to the finding of palpable enlarged lymph nodes in the left inguinal region, the patient was examined by a hematologist, a biopsy was performed with the finding of a chronically inflamed lymph node. Additionally, a CT scan of the abdomen and small pelvis with IV contrast was done - with findings of focal hypodense change with a diameter of 10 mm (segment 6), spleen without focal changes and dimensions 158x51 mm, retroperitoneally, paraaortically, in addition to the right perigastric cruciate ligament, there were enlarged lymph nodes in the hilus of the spleen with a maximum diameter of 20 mm. In the right inguinal region there was residual collection with a transverse diameter of 16 mm, with fibro-adhesive changes, enlarged lymph nodes up to 12 mm, and also, enlarged lymph nodes were also observed in the left inguinal region with a diameter of about 16.5 mm. Tests for infectious diseases such as borrelia, brucellosis and toxoplasmosis were negative. The patient was thought to have Castelman disease.

Initial neurological examination revealed dysarthric speech, with discrete central paresis of the facial nerve, latent left-sided ataxic hemiparesis. In order to clarify the etiology of the expansive change in the left cerebellum, a series of examinations were conducted.

Routine biochemical analyzes showed SE 26 / -, a haemogram with mild leukocytosis (Le 15.9; Granulocytes 8.4), the rest of the findings were in normal range. Glycaemia 4.44 mmol/l, Urea 8.4 mmol/l Creatinine 81.2 μmol/l, Sodium 144 mmol/l, Potassium 4.2 mmol/l, Cholesterol 6.4 mmol/l, LDL 3.8 mmol/l, Triglycerides, 27 mmol/l, the enzyme status was within normal range. Serum iron 21.9 mmol/l. Analysis of the urine- turbid, yellow, acidic pH, protein 30 mg/dl.

Immunoassay analysis of serum (serum protein electrophoresis) detected a multiple increase in the serum concentration of Immunglobulin E (IgE = 951 IU/ml (<10) (because of that finding a series of diagnostic tests have been conducted to detect conditions with high production of immunoglobulins – parasitosis/granulomatosis - sarcoidosis / allergic diathesis). For that purpose, serological tests were performed as there was suspicion of an existing autoimmune diseases-collagenosis (ANA, ANCA, anti Ds DNA, RF, CRP, a SS-A, a SS B, CCP) - all with a negative finding. Serum angiotensin converting enzyme concentrations were in a normal range, so laboratory parameters ruled out sarcoidosis. Additionally, due to generalized lymphadenopathy, CT scans of the lungs and mediastinum were performed, where no enlarged lymph nodes or any changes in the lung parenchyma were detected. In consultation with an infectologist, several samples of cerebrospinal fluid were sent for microbiological, parasitological and serological analysis.

Parasitological analysis aimed at detecting the presence of antibodies to toxoplasmosis, echinococcus, and cysticercosis - as possible causes of IgE overproduction, lymphadenopathy, and CNS changes. They were negative (confirmed by two independent laboratories). Protein electrophoresis of the cerebrospinal fluid has shown a transudative type of electrophoregram - finding of blood-brain barrier dysfunction, but excluding CNS immune disease (total protein 1.07 g/l (0.15–0.45), albumin 824 mg/l (50-250), IgG 108 mg/l (3-30), albumin coefficient 20.1 (1.8-7.4). IgG synthesis in the CNS 0). Feces for parasites was tested with ELISA (Taenia solium) - no specific IgG antibodies were detected. Serological blood test for Toxoplasma gondii (IgM, IgG in serum) was negative. Control MR of the brain with IV contrast showed an oval intraaxial lesion measuring 22x17 mm on the left at the height of the posterior cranial fossa, and in projection of the middle cerebellar peduncle with surrounding irregular vasogenic edema, which has a compressive effect on the cerebellum on the left side, as well as the brainstem, predominantly the left pons, but edema was also found to exist at the level of the left segment of the mesencephalon and medulla oblongata. No significant shift or compromise of the lumen of the 4th ventricle was observed. After IV given contrast, inhomogeneous eccentric bifocal postcontrast accentuation was obtained. The rest of the brain parenchyma was without pathological staining.

Additionally, MR was performed on a cervical segment of the spine with a post-contrast series, where spondyloarthritic changes were detected, dorsocentric protrusions at the level of C5/C6, C6/C7,

without myelopathic changes in the medulla spinalis. Lung and mediastinal CT, native and post-contrast series - no CT signs of focal changes or pleural effusion were visible. Increased lymph nodes were not observed in the mediastinum. On the distal sections mesenterically at the level of the splenic artery on the left, 9 mm lymph nodes were observed. Fundus oculi- OU PNO at retinal level with clear boundaries. Right macula lutea with clear reflex, left with regrouping of pigment - atrophic changes. Blood vessels on both sides with normal lumen and blood flow.

The overall clinical symptomatology of generalized lymphadenopathy, in the absence of fever, weight loss, verified brain lesion, and hypersecretion of IgE did not rule out the presence of lymphoproliferative disease. The patient was referred to a hematologist again, where a PET scan was performed showing the presence of metabolically inactive lymph nodes, which displayed benign characteristics.

Nowever, asymmetry of FDG distribution at the cerebellum level was detected. Meanwhile, the patient had a progression of neurological symptomatology, developing peripheral paresis of the left facial nerve, severe dysarthria, and severe ataxic hemiparesis.

Additionally, MR spectroscopy was performed showing an increase in the size of the lesion at the level of the left cerebellar peduncle (29x27 mm), with surrounding perilection edema. A craniotomy and surgical extirpation of the lesion were performed in consultation with a neurosurgeon. Pathohistological analysis showed that it was a chronic abscess of the cerebellum at the stage of organization. Postoperatively, the patient still had dizziness and severe left-sided ataxic hemiparesis.

Discussion

A brain abscess is a localized zone of necrosis of the brain parenchyma, with a surrounding membrane, which usually occurs as a result of an infectious process or trauma. In terms of etiology, brain abscesses are usually a consequence of direct local spread of the infectious process in infections of the structures in the head and neck: otitis media, mastoiditis, pansinusitis, tooth infections, facial trauma, the presence of metal foreign bodies in the brain parenchyma. However, they are often due to hematogenous dissemination, most often in lung infections (pneumonia, empyema), pulmonary arteriovenous malformations, bacterial endocarditis, congenital heart anomalies with cyanosis in children. Brain abscesses due to bacteraemia are usually multiple, localized in the vascular area of the middle cerebral artery, on the border of gray and white matter. The incidence of brain abscesses is about 8% on average of all cases of intracranial processes; especially in western countries with an increased number of people living with HIV/AIDS. They occur more often in men than women and that ratio is 3:1 [2].

Otogenic brain abscesses are a rare complication of chronic inflammation of the middle ear and surrounding structures, given the possibility of early diagnosis and the availability of antibiotic therapy. They are most commonly localized in the cerebellum and temporal lobe of the cerebrum [3].

In terms of the clinical presentation, the most common symptom is headache, which occurs in almost 70% of cases. This is followed by changes in the mental status such as lethargy and quantitative disturbances of consciousness; focal neurological deficits, epileptic seizures (in 25% of cases), nausea, vomiting, and signs of elevated intracranial pressure, fever, and cranial nerve involvement (n. III, n. VI). The diagnosis is made by history and examination, neuroimaging examinations (computed tomography of the brain-CTM, magnetic resonance imaging MRI (native, postcontrast, diffusion, spectroscopy), cytochemical and microbiological analysis of cerebrospinal fluid [4].

The results are mostly non-specific, consisting of an elevated protein level, pleocytosis with the variable neutrophil count, typically a normal glucose level, and sterile cultures. A brain abscess can lead to an elevated intracranial pressure and it has significant morbidity and mortality. Management can vary between medical or surgical approach.

Medical management can be considered for deep-seated, small abscesses (less than 2 cm), cases of coexisting meningitis, and few other selected cases. Usually, a combination of both medical and surgical approaches is considered [5].

The complications that might occur secondary to a brain abscess are: meningitis, ventriculitis, increased intracranial pressure, brain herniation, seizures, thrombosis of intracranial blood vessels, death.

The treatment of brain abscesses is multidisciplinary and includes: a neurologist, infectologist, radiologist, neurosurgeon and an internal medicine specialist. There is still controversy as to what the most appropriate approach for the choice of antibiotic therapy is, on the deciding on the indication and the timing of surgery, and the long-term outcome.

References

1. Lange N, Berndt M, Jörger AK, Wagner A, Wantia N, Lummel N, Ryang YM, Meyer B, Gempt J. Clinical characteristics and course of primary brain abscess. Acta Neurochir (Wien). 2018;160(10):2055-2062.
2. Maher G, Beniwal M, Bahubali V, Biswas S, Bevinahalli N, Srinivas D, Siddaiah N. Streptococcus pluranimalium: Emerging Animal Streptococcal Species as Causative Agent of Human Brain Abscess. World Neurosurg. 2018;115:208-212.
3. Sennaroglu L, Sozeri B. Otogenic brain abscess: review of 41 cases. Otolaryngol Head Neck Surg. 2000;123(6):751-755.
4. Longo D, Narese D, Fariello G. Diagnosis of brain abscess: a challenge that Magnetic Resonance can help us win! Epidemiol Infect. 2018;146(12):1608-1610.
5. Widdrington JD, Bond H, Schwab U, Price DA, Schmid ML, McCarron B, Chadwick DR, Narayanan M, Williams J, Ong E. Pyogenic brain abscess and subdural empyema: presentation, management, and factors predicting outcome. Infection. 2018;46(6):785-792.