MULTIPLE CEREBRAL MASS LESIONS IN A YOUNG PATIENT – ABSCESSES OF UNKNOWN ORIGIN

MIRCEA VICENŢIU SĂCELEANU[1], VLAD-IOAN SUCIU[2], CORINA SCOROBEŢ[3], ADRIANA SĂCELEANU[4]

[1,4] "Lucian Blaga" University Sibiu, [1,2,3,4]Clinical County Emergency Hospital Sibiu

Keywords: *multiple cerebral abscesses, no immunosupression*

Abstract: *We present the case of an 18-year old patient, formerly with unknown illness, who is brought to the emergency department for a state of altered consciousness, right hemiparesis and frontal headache. The symptoms started a week before the day of admission. On arrival, the patient had fever, an altered state of consciousness, positive meningeal irritation signs, right hemiparesis and bilateral Babinski's sign. The emergency cerebral tomography showed two lesions: left frontal and left temporal hypodense masses with peripheral contrast enhancement and surrounding edema and the biological markers showed an inflammatory response. HIV testing was negative. The histological samples showed inflammatory reaction, while the Gram stain of the collection showed Gram positive diplococcus bacteria. Conclusion: The primary cause of the multiple abscesses remained uncertain.*

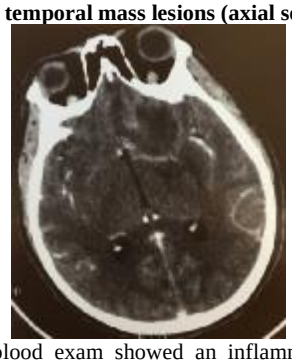
INTRODUCTION

Brain abscesses are seen frequently in immunocompromised patients. The etiology can be bacterial, fungal or parasitic. The clinical picture resembles a bacterial meningitis.(1,2)

CASE REPORT

We present the case of an 18-year old patient, formerly with unknown illness, who is brought to the emergency department for a state of altered consciousness, right hemiparesis and frontal headache. The symptoms started approximately a week before the day of admission, according to the family. On arrival, the patient had fever with abundant sweating, normal respiratory parameters, coughing, low blood pressure, altered consciousness, positive meningeal irritation signs, no cranial nerves deficits, 3 out of 5 MRC scale right hemiparesis, brisk reflexes, bilateral Babinski's sign and no other signs. The emergency cerebral tomography (CT) showed two lesions: left frontal and left temporal hypodense masses with peripheral contrast enhancement and surrounding edema. Also the pharyngeal lymph nodes were enlarged (figure no. 1).

Figure no. 1. Contrast enhanced cerebral CT scan showing the frontal and temporal mass lesions (axial section)



The blood exam showed an inflammatory response with 18.850 leucocytes/mm^3 with 16.680 granulocytes/mm^3, a erythrocyte sedimentation rate of 41 mm/h, a C-reactive protein of 48 units (normal < 6 units) and slightly modified coagulation parameters (INR 1,3). The plain thoracic radiography was normal (figure no. 2).

Figure no. 2. Plain thoracic radiography (AP incidence)

An infectious diseases consultation suspected a possible cerebral abscess in the context of an immunosupression and recommended high doses of Ceftriaxone, Clyndamycin and Fluconazole.

Ophthalmology consultation showed bilateral papillary edema with a slight asymmetry favouring the left side.

Magnetic resonance imaging could not be done because of the patient's critical state.

HIV testing was negative. Blood cultures were negative for aerobic, anaerobic bacteria and fungi.

The patient's state aggravated: left midriasis, abolished light reflexes on the left side and a coma of 4 points on the Glasgow scale.

The emergency surgery revealed a dura mater under tension after the craniotomy. The incision of the mass in the frontal lobe revealed a massive collection of pus. The collection

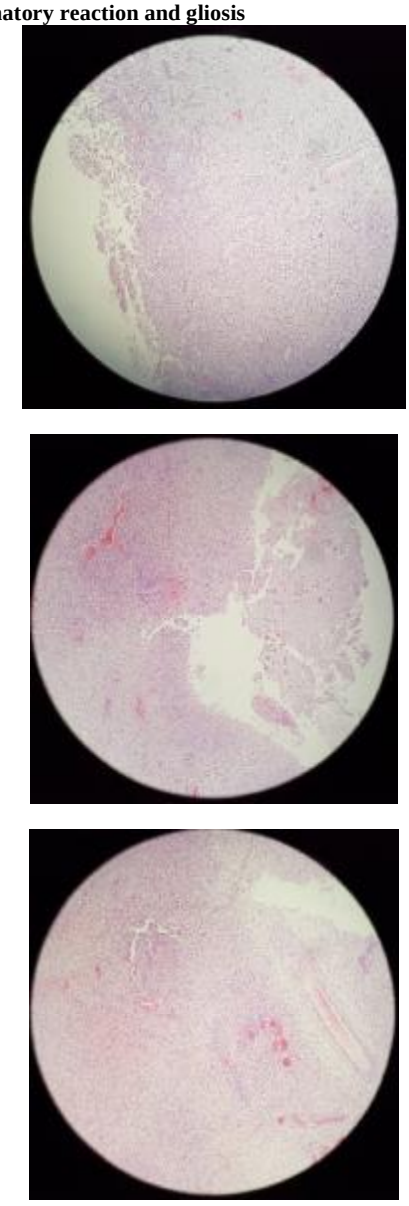
[4]Corresponding author: Adriana Săceleanu, Str. Corneliu Coposu, Nr. 2-4, Sibiu, România, E-mail: vicentiu.saceleanu@gmail.com, Phone: +4070 022931

Article received on 18.07.2017 and accepted for publication on 29.11.2017
ACTA MEDICA TRANSILVANICA December 2017;22(4):39-41

is drained and a small satellite lesion of fish-meat-like consistency is visualised and removed. The fluid collection did not have the specific smell of pus.

The histopathological samples of the satellite lesion and pus were sent to the pathology department for diagnosis. The Gram stain of the intraoperatory drained collection revealed frequent leucocytes and Gram positive diplococcus bacteria. The histopathological fragments showed acute inflammatory reaction, gliosis, necrosis and no tumour cells (figure no. 3).

Figure no. 3. Set of images from the histopathological samples taken from the resected cerebral tissue showing inflammatory reaction and gliosis



After the surgery, the patient presented left areactive midriasis and right trochlear paresis with 4 points on the Glasgow scale. The patient's health rapidly declined.

DISCUSSIONS

Cerebral abscess is a purulent infection localized in the brain parenchyma and has lifethreathening potential. It has 4 stages of evolution:

1. early cerebritis (nonenhancing hypodense lesion in the first 3 days),
2. late cerebritis (ring-enhancing lesion with central necrosis and surrounding edema within 4-9 days),
3. early capsule (first 10-14 days),
4. final maturation (2 weeks).(1,2,3)

This is more common in males, in the fourth decade of life, in immunocompromised patients and in children with right-left cardiac shunt. The most common symptoms are headache, fever, nuchal rigidity, nausea, lethargy, focal deficits, seizures, papilledema and oculomotor palsies. Cerebral sinus thrombosis can accompany an abscess.(1,2)

The pathological mechanisms of brain abscess include hematogenous spreading from a distal infection site, direct extension from sinusitis or oto-mastoiditis, or a direct inoculation. The most common reported pathological mechanism is hematological spread.(1,2,4)

The most common risk factors for developing cerebral abscesses are right-left cardiac shunts, endocarditis, chronic lung infections, abdominal or pelvic infections, sepsis, sinusitis, oto-mastoiditis, dental infections and immunosupression of different causes.(2,4)

The most common microorganisms are Staphylococcus aureus, Streptococci (35-50 %), Enterobacteriaceae and anaerobes. Fungal or parasitic infections are found in immunocompromised patients due to HIV infection, malignant conditions or immunosupressive drugs. New born children can develop abscesses with group B Streptococci or E. Coli. Children have gram negative bacteria, while adults have gram positive and anaerobes isolated from the necrotic pus. Listeria monocytogenes is found in pregnant women or elderly patients. No microorganisms are found in 25 % of patients.(1,2,4)

Lumbar puncture is contraindicated because of the risk of herniation. Highly suggestive, but still not pathognomonic for a cerebral abscess are the following: thin wall, extensive perilesional edema and restricted diffusion in DWI sequences. MRI is the most sensitive imaging tehnique. MRI features are: hypointense T1/ hyperintense T2, FLAIR and DWI core; surrounding vasogenic edema; elevation of succinate and lactate peak in magnetic resonance spectroscopy. For positive diagnosis it is recommended corroborating clinical, imaging and intraoperatory data.(1,3,5,6,7)

The ring-enhancing focal mass lesions are considered as differential diagnosis to cerebral abscesses:

- glioblastoma (no thick ring enhancement; cystic areas do not show restricted diffusion in DWI sequences)
- primary central nervous system lymphoma
- infarcts (not a specific context)
- metastasis (no prior history of cancer, no round and nodular lesions)
- acute demyelinating disease (excluded by the infectious profile of the presentation, no incomplete ring enhancement)
- infected primary tumour
- tuberculoma (no pulmonary lesions, no immunosupression)
- neurocysticercosis (no multiple < 1 cm calcified lesions).(1,3,5,8,9,10)

Treatment implies antimicrobial therapy for 4-8 weeks (at least 2 broad spectrum antibiotics) started before surgery for aspiration. Dexametasone is indicated in increased intracranial pressure, multiple abscesses or perilesional edema. Surgical options are stereotactic aspiration or open craniotomy. Open craniotomy was chosen because of the emergency procedure and the uncertain etiology of the lesions.(1,11,12,13)

Risk factors for poor outcome are: delayed diagnosis or treatment, multiple abscesses, intraventricular pus, altered consciousness on admission and immunosupression.(1)

CONCLUSIONS

This case presented numerous atypical characteristics. The inflammatory profile clearly suggested an infection, but it was not clear where the infection started. The patient had no prior history of a documented illness. Multiple metastases were improbable but not impossible, moreover multiple infected primary tumours were also highly improbable. We excluded other causes as well, and suspected the lesions to be abscesses.

The primary cause of the multiple abscesses remained uncertain. Because the pharyngeal tonsils and lymph nodes were enlarged, we had arguments to believe that the primary infection situs was pharyngeal. Still, the answer why multiple brain abscesses appeared in an immunocompetent patient remains unknown.

REFERENCES

1. Louis ED, Mayer SA, Rowland LP. Merritt's Neurology. Thirteenth Edition. Philadelphia: Wolters Kluwer; 2016.
2. Ropper AH, Samuels MA, Klein JP. Adams and Victor's Principles of Neurology. Tenth Edition. China: McGraw-Hill; 2014.
3. Osborn A, Blaser S, Salzman K. Encyclopedia of Diagnostic Imaging. AMIRSYS; 2008 ISBN:0721629059.
4. Greenberg MS. Handbook of neurosurgery. George Thieme Verlag; 2006 ISBN:313110886X.
5. Lin E, Garg K, Escott E, et al. Practical differential diagnosis for CT and MRI. Thieme Medical Pub.; 2008 ISBN:1588906558.
6. Holmes TM, Petrella JR, Provenzale JM. Distinction between cerebral abscesses and high-grade neoplasms by dynamic susceptibility contrast perfusion MRI. AJR Am J Roentgenol. 2004;183 (5):1247-52.
7. Schaefer PW, Grant PE, Gonzalez RG. Diffusion-weighted MR imaging of the brain. Radiology. 2000;217 (2):331-45.
8. Viaccoz A, Ducray F, Tholance Y, et al. CSF neopterin level as a diagnostic marker in primary central nervous system lymphoma. Neuro Oncol. 2015;17(11):1497-503. doi: 10.1093/neuonc/nov092. Epub 2015 May 25.
9. Garg RK, Sinha MK. Multiple ring-enhancing lesions of the brain. J Postgrad Med. 2010;56 (4): 307-16. doi:10.4103/0022-3859.70939.
10. Mutu CC, Suciu VI. Atypical primary central nervous system lymphoma or cryptogenic leukoencephalopathy. Case report. AMT. 2016;21(4):19-21.
11. Popp A, Popp AJ, Deshaies EM. A Guide to the Primary Care of Neurological Disorders. Thieme Medical Pub.; 2007, ISBN:1588905160.
12. Johnson RT, Griffin JW, McArthur JC. Current Therapy in Neurologic Disease. Mosby. (2006) ISBN:0323034322
13. Hufschmidt A, Lücking CH. Neurologie integrală. De la simptom la tratament. Iași: Polirom; 2002.