

Differentiation of Tumefactive Demyelinating Lesions from Metastatic Brain Disease with FDG PET-CT: A Case Report

Kresimir Dolic^{1*}, Ivica Bilic², Ante Buca¹, Darijo Radovic³ and Marina Titlic²

¹Clinical Department of Interventional and Diagnostic Radiology, University Hospital Split, Croatia

²Department of Neurology, University Hospital Split, Croatia

³Medikol polyclinic, PET/CT Center, Split, Croatia

*Corresponding author: Kresimir Dolic, MD, PhD, Clinical Department of Interventional and Diagnostic Radiology, University Hospital Split, Spinciceva 1, 21000 Split, Croatia, Tel: +385 21 556-243; E-mail: kresimir.dolic@st.t-com.hr

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Abstract

Tumefactive demyelinating brain disease can pose a diagnostic challenge in patients without a pre-existing diagnosis of multiple sclerosis by mimicking intra-cranial neoplasms, infarction, as well as infections. Choosing when to biopsy a tumefactive lesion to exclude alternative pathology can be difficult. Here we report a case of a 44 year old female patient without a previous history of multiple sclerosis who presented with two brain lesions mimicking metastatic brain disease which pose a serious diagnostic dilemma. FDG PET CT and follow up magnetic resonance imaging (MRI) helped to rule out tumor etiology of those lesions and spared patient from invasive procedures such as biopsy or radiotherapy.

Keywords: Tumefactive demyelination; PETCT; Multiple sclerosis; Magnetic resonance imaging

Introduction

Tumefactive demyelinating lesions (TDL) are generally thought of as solitary lesions, greater than 2 cm with mass effect and edema which represent major diagnostic challenge since their clinical, radiologic and even pathohistological features are not uniform, often mimicking brain neoplasms [1]. Symptoms are atypical for multiple sclerosis (MS) and include focal neurologic deficit, seizure or aphasia. They can be found in 2.8% of MS patients or in patients without preexisting MS history, although they usually develop typical MS features in near future [1,2]. Here we report a unique case of tumefactive demyelinating disease that was diagnosed with the help of PET/CT. The report has been approved by our Institutional Review Board.

Case Report

A 44-year-old Caucasian woman, without past medical history, presented to the Emergency Neurology Department with seizures, postictal confusion and speech difficulties. Brain computed tomography (CT) on admission showed 2.5 cm large demarcated hypodense lesion in the left parietal lobe with perifocal edema and ring enhancement. Further diagnostic work-up with brain MRI showed one more small lesion in the right frontoparietal subcortical region as well as the bigger one in the left parietal lobe with surrounding edema, mass effect and a partial ring-like pattern of contrast enhancement (Figure 1).

Based on clinical data of first seizure attack differential diagnosis included metastatic tumor disease. Oncologist and neurosurgeon were consulted and they suggested specific oncologic treatment for suspected metastatic tumors. She received a 5 day course of high dose

corticosteroids (methylprednisolone) and had been discharged with no neurologic deficit. Patient decided to take second opinion in another neurology clinic where PET-CT has been done and revealed no pathologic metabolic activity in the lesions (Figure 2).

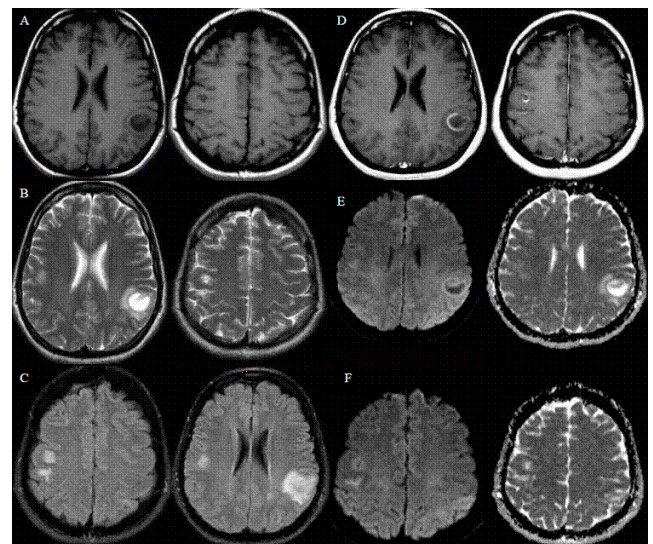


Figure 1: Brain MRI. Axial T1 (A), T2 (B) and FLAIR (C) images show two oval brain lesions with perifocal edema. Axial T1 postcontrast images (D) show open ring enhancement of the lesions with peripheral restriction on diffusion-weighted imaging (E,F).

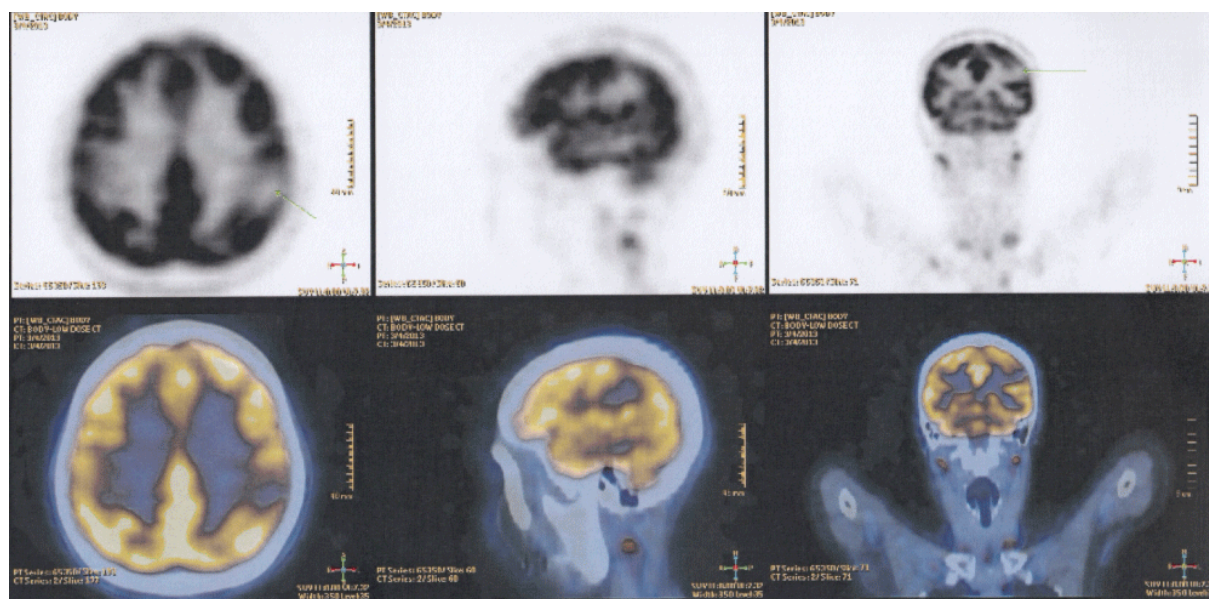


Figure 2: PET/CT images. PET (black/white) and fused (colored) PET/CT images show decrease accumulation of 18F-fluoro-2- deoxy-glucose (FDG) in the left parietal brain lesion (arrow).

Follow up MRI of the brain showed reduced size of the lesions, still with incomplete ring of enhancement, which together with positive oligoclonal bands in the cerebrospinal fluid and patient's considerable clinical improvement after steroid therapy helped to establish the diagnosis of tumefactive demyelinating disease.

Discussion

Typical brain lesions in multiple sclerosis (MS) include multiple or focal small ovoid periventricular (often oriented perpendicular to the long axis of the ventricular system) or subcortical white matter lesions, bright on T2-weighted and FLAIR images, with or without contrast enhancement. There is usually good clinical-radiological correlation, emphasized with oligoclonal-band positivity in cerebrospinal fluid [3]. On the other hand, tumefactive demyelinating lesions (TDL), which can be isolated entity or presenting feature of MS (tumefactive MS lesions - TMS) pose a serious diagnostic challenge, to both clinicians and radiologists, that frequently prompts biopsy in initial evaluation. TMS often presents large, greater than 2 cm, enhancing lesions with mass effect and edema mimicking neoplasm, abscess, or stroke [4]. The frontal lobe and parietal lobe are the most common locations of tumefactive MS and it presents as a single neurologic episode like in our case. Although, some MRI characteristics, such as open-ring enhancement, peripheral restriction on diffusion-weighted imaging, or venular enhancement [3,4], location in the subcortical hemispheric white matter, relative lack of mass effect and perifocal edema [5] may help to distinguish TMS from neoplastic lesions it still represents big diagnostic challenge as it was also in our case (Figure 1). Because the clinical presentation of TMS is more like in neoplastic disease, distinguishing active demyelination from neoplasm is critical, since a misdiagnosis can result in unwarranted procedures and treatments that can even exacerbate underlying inflammatory demyelinating disease [1-3,5]. It is critical for the neurologist to be aware of this diagnostic pitfall because sometimes histopathologic findings from the biopsy can also be misleading depending which part of the lesion was

biopsied: for TDL the target of biopsy should always be the wall of the lesion, unlike in tumours where the central core of the lesion is more important [6]. However, a pre-existing diagnosis of multiple sclerosis does not exclude the possibility of a coexisting tumour, or additional pathology (e.g. infection) [1].

In contrast to TMS neoplastic lesions more often show complete ring enhancement and greater mass effect while abscesses show more extensive edema and central restriction on diffusion-weighted imaging as well as complete ring enhancement [1-3]. Owing to its unifocal features and its good response to steroid therapy brain lymphoma is easily confused with tumefactive MS but it shows marked homogeneous postcontrast enhancement on T1 and iso- or mild hyperintensity on T2-weighted images [7].

Although CT hypoattenuation of MR enhancing lesions was found to be highly specific for distinguishing TDL from primary gliomas or central nervous system lymphomas conventional MR imaging is still considered to be the most sensitive method for detecting white matter lesions of multiple sclerosis [6]. Some additional MR techniques [8,9] as well as other imaging modalities [10] can help to increase sensitivity and specificity for correct diagnosis especially prior to possible brain biopsy. MR perfusion shows low cerebral blood volume (rCBV) in demyelinating lesions in contrast to high rCBV in neoplastic lesions [3,6]. Although the role of MR spectroscopy (MRS) in helping to clarify the diagnosis of TMS is not yet definitely established an elevated choline/creatine ratio, increased lactate, and normal N-acetylaspartate/creatine ratio may be regarded as suggestive of inflammation or demyelination [8,9]. In our case positron emission tomography-computed tomography (PET/CT) was used to evaluate metabolic activity of the suspected lesions which together with follow up MR helped to rule out focal brain as well as systematic neoplastic disease. Some authors have found that follow up MR imaging may be used as a diagnostic aid to confirm the diagnosis, with interval improvement of these lesions following steroid therapy [5]. Further,

because many of the patients with TMS develop definite MS (70%) most will have new lesions characteristic for time-space phenomenon of MS lesions on follow up imaging which may help to avoid biopsy [1,2,5].

Conclusion

This case emphasise the fact that in case of young patients with tumor-like lesions, tumefactive demyelination should always be considered in the differential diagnosis. Since TDL represent major diagnostic challenge it calls for team work of many specialists involved in diagnostic process: neurologists, neuroradiologists, neuropathologists, oncologists and neurosurgeons. Timely and exact diagnosis is prerequisite for optimal treatment and can spare patients from unnecessary risks of surgical, avoiding resection of viable brain tissue, or oncology procedures.

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