Multiple Sclerosis and Related Disorders 4 (2015) 555–566

Contents lists available at ScienceDirect

Multiple Sclerosis and Related Disorders

journal homepage: www.elsevier.com/locate/msard

Review

Neuro-oncology dilemma: Tumour or tumefactive demyelinating lesion

Mohammad Abdoli [a,b], Mark S. Freedman [a,b,*]

[a] University of Ottawa, Canada
[b] The Ottawa Hospital Research Institute, Canada

ARTICLE INFO

Article history:
Received 22 April 2015
Received in revised form
15 June 2015
Accepted 27 July 2015

Keywords:
Multiple sclerosis
Tumefactive demyelinative lesions-TDLs
Brain tumour mimics
Comorbidity in MS

ABSTRACT

Tumefactive demyelinating lesions (TDLs) are not an uncommon manifestation of demyelinating disease but can pose diagnostic challenges in patients without a pre-existing diagnosis of multiple sclerosis (MS) as well as in known MS patients. Brain tumours can also arise in MS patients and can be seen in chronic MS patients as co-morbidities. Delayed diagnosis or unnecessary intervention or treatment will affect the ultimate prognosis of these patients. In this article, we will review some typical cases illustrating the dilemma and review the information that helps to differentiate the two conditions.

The intention is not to present an extensive differential diagnosis of both entities, but to examine some typical examples when the decision arises to decide between the two. We take a somewhat different approach, by presenting the cases in "real time", allowing the readers to consider in their own minds which diagnosis they favour, discussing in detail some of the pertinent literature, then revealing later the actual diagnosis. We would urge readers to consider re-visiting their first thoughts about each case after reading the discussion, before reading the follow-up of each case.

The overall objective is to highlight the real possibility of being forced to decide between these two entities in clinical practise, present a reasonable approach to help differentiate them and especially to focus on the possibility of TDLs in order to avoid unnecessary biopsy.

Contents

* Corresponding author at: University of Ottawa, Canada.
E-mail addresses: mabdoli@toh.on.ca (M. Abdoli), mfreedman@toh.on.ca (M.S. Freedman).

http://dx.doi.org/10.1016/j.msard.2015.07.013
2211-0348/

1. Introduction

Typical demyelinating lesions in MS appear as small, often ovoid, T2 and fluid attenuated inversion recovery (FLAIR) hypersignal lesions which may involve corpus callosum, periventricular, deep white matter, juxtacortical regions, the infratentorial compartment and spinal cord. These lesions may enhance with gadolinium or show restricted diffusion in diffusion weighted imaging (DWI) in the acute phase. Chronic lesions are non-enhancing and demonstrate hypointensity or black holes on T1 sequences (Barkhof et al., 1997).

One of the rarer variants of demyelinating lesions is the tumefactive demyelinating lesion. Early reports described MS variants with tumour-like presentation as Shilder's disease or Marburg's variants (Poser et al., 1992). There is no consensus however, regarding the definition of TDLs, but typically represent lesions larger than 2 cm in patients with or without an established diagnosis of MS. TDLs by definition are large and can present as a space occupying lesions (SOL), posing a particular diagnostic dilemma in patients with or without MS. There are some typical imaging and clinical manifestations of TDLs, but none are pathognomonic. Without a clear clinical or imaging differential such patients are often referred for brain biopsy, which itself carries certain morbidity and eventual results may be uninformative or misleading and could lead to unnecessary surgery, or even radiotherapy which could further aggravate TDLs.

There are growing numbers of middle aged and older MS patients who experience other co-morbidities, including brain tumour, throughout the course of their long-lasting disease. Early presentation of brain tumour may not be easily differentiated from MS, or can be diagnosed incorrectly as a relapse, or even progressive multifocal leukoencephalopathy (PML) in a patient taking certain MS therapies and this further delays the actual diagnosis of brain tumour, thus postponing potentially life-saving treatment impacting overall prognosis.

2. Case presentation

2.1. Case A

A 55-year-old female was diagnosed with MS in 2001 and took beta-interferon for more than 10 years with a stable course (Fig. A1). She then presented with burning dysesthesia in her right lower abdominal area and clumsiness of her right hand, but no new objective findings were seen. A “mild relapse” was considered but no acute treatment was given.

2 months later, she returned with increased numbness with associated clumsiness in her right hand and a 3-day-course of high-dose oral prednisone was given as a relapse treatment. She only experienced a temporary improvement and her next month follow up visit showed increased weakness and numbness right upper extremity with spreading of numbness into her face. A 3-day-course of IVMP was given and a new MRI was performed (Fig. A2).

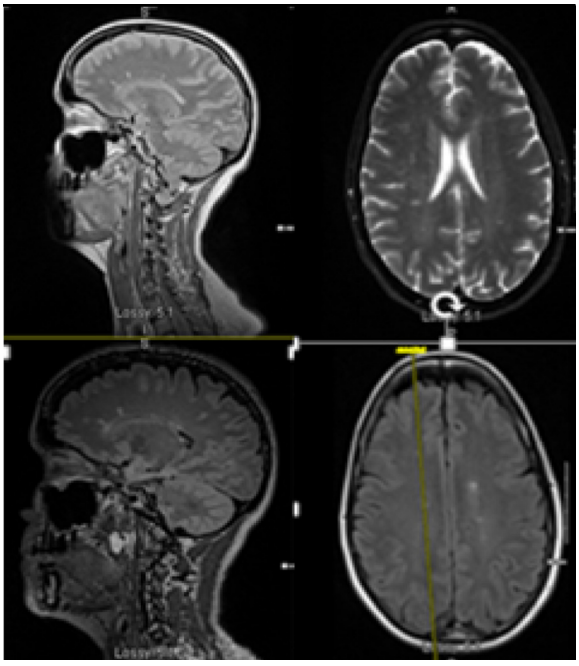

Fig. A1. 2010-2011 imaging.

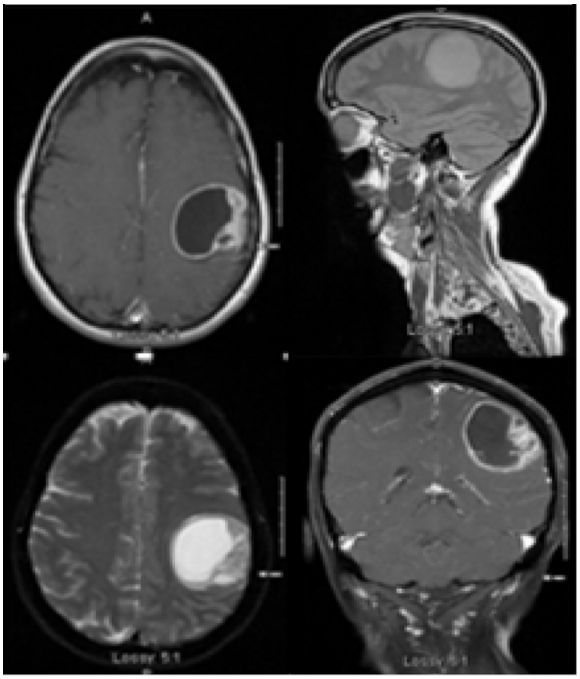

Fig. A2. New 2013.