The revised McDonald criteria in the diagnosis of multiple sclerosis (MS) - what has changed?

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Learning objectives

To inform radiologists of changes to previous versions of the McDonald criteria in the diagnosis of MS. In this poster we will mainly focus on the 2010 revisions to the McDonald criteria with particular reference to the MRI requirements for demonstrating lesion dissemination in space (DIS) and time (DIT). We will also emphasise the fact that, although key aspects of the McDonald criteria have changed as a result of new research by the MAGNIMS (Magnetic Imaging in MS) group, it remains essential that the criteria are only applied to patients with symptoms compatible with a CNS inflammatory demyelinating disease, and that alternative diagnoses are excluded [1].
Background

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disorder of the CNS [2]. MS usually presents in the third to fourth decade, is more common in women, and has an incidence of 1-10/100 000 in Europe and North America [3]. As a result, MS is one of the most common causes of non-traumatic disability in young adults [2], and is a frequently encountered presentation, both within a general radiology and specialist setting.

A conclusive diagnosis of MS requires relevant clinical features as well as dissemination of lesions in space (DIS) and time (DIT) [4]. Whilst MS can be diagnosed on clinical grounds alone, MRI is regarded as an important diagnostic tool in this setting [5], and MRI findings may support or even replace some clinical criteria [1]. The use of MRI often facilitates an earlier diagnosis than would otherwise be possible with clinical assessment alone, thereby enabling earlier commencement of treatment. This is of key importance, as disease-modifying immunomodulatory therapies (such as interferon and glatiramer acetate) are most effective in the relapsing-remitting period of the disease [2].

In 2010, following the International Panel on Diagnosis of MS meeting in which the most recent published research regarding MS diagnosis was reviewed; the magnetic resonance imaging (MRI) requirements for demonstrating DIS and DIT were updated. The updated criteria are simpler to use and may enable earlier diagnosis, with equal or even improved specificity and sensitivity.

Whilst the new McDonald criteria are easier to use, have improved sensitivity and may enable earlier definitive diagnosis, there are several areas in which caution should be exercised. Firstly, the criteria have not been validated in Latin American and Asian populations, and further studies to confirm sensitivity and specificity are needed in these patient groups [1]. Secondly, application of the criteria to paediatric patients is challenging, as approximately 20% do not have an adult-type clinically isolated syndrome (CIS) presentation [8]. Where children present with multifocal neurological symptoms and encephalopathy then acute disseminating encephalomyelitis (ADEM) should be considered and serial clinical and MRI assessment should be undertaken.
Dissemination in Space (DIS)

The new 2010 criteria requirements for DIS state that a minimum of 1 T2 hyperintense lesion should be present in at least 2 of 4 characteristic locations: periventricular, juxtacortical, infratentorial and/or spinal. Previously, 1 gadolinium-enhancing (GdE) or 9 T2 hyperintense lesions were required, 3 located in a periventricular distribution, 1 juxtacortical and 1 infratentorial/spinal. The updated criteria, whilst being simpler to apply, have also been shown to be slightly more sensitive than the original McDonald Criteria (72% with 2010 criteria compared with 60% for 2005 criteria), with preservation of specificity and accuracy [6]. It should be noted that in subjects with a brainstem or spinal cord syndrome, symptomatic lesions do not contribute to lesion count.

Periventricular lesions appear as high signal foci on T2-weighted sequences and characteristically run through the corpus callosum, perpendicular to the lateral ventricles (in a perivenular distribution). This imaging appearance is termed Dawson's fingers [Figure 1]. A further example of a periventricular distribution of MS plaques is shown in Figure 2. Figures 1, 3 and 4 are MRI images from a 36 year-old male with known relapsing-remitting MS. In addition to the periventricular lesions shown in Figure 1, this patient also has high signal foci within the pons [Figure 3] and within the proximal spinal cord [Figure 4]. The MRI findings for this patient therefore satisfy the 2010 McDonald criteria for DIS, as 1 or more lesion is seen in greater than 2 characteristic locations; periventricular, infratentorial and spinal cord. The fourth characteristic location for MS plaques is in a juxtacortical distribution, as shown in Figure 5. Juxtacortical lesions are seen adjacent to the cortex and must touch the cortex. They are best appreciated on fluid-attenuated inversion recovery (FLAIR) sequences, and may contribute to cortical atrophy and memory impairment in MS [7].

Dissemination in Time (DIT)

Dissemination in time (DIT) can be demonstrated in two ways; either the presence of a new T2 hyperintense lesion in a characteristic area on MRI performed any time after onset of symptoms, or by the simultaneous presence of asymptomatic GdE and non-enhancing lesions.

Figure 6 shows an axial image from an unenhanced CT head in a 37 year old female presenting with left arm weakness and numbness. There is a subtle area of low attenuation in the right basal ganglia. MRI subsequently revealed a T2 hyperintense region in the right thalamus [Figure 7] and further multifocal areas of signal abnormality
in a periventricular distribution. Whilst these findings could be in keeping with acute infarction, the morphology of the lesions in combination with their periventricular distribution is more in keeping with demyelination. A further MRI brain performed in the same patient one month later revealed a new curvilinear focus of high T2 signal in the left parietal region [Figure 8]. This fulfils the criteria for DIT.

The new DIT criteria have two advantages. Firstly, clinicians are no longer required to delay performing an MRI until 30 days after the onset of new symptoms. Secondly, in some patients presenting with CIS, it may be possible to diagnose MS on the basis of a single MRI [8].

Gadolinium enhancement of MS plaques is indicative of active demyelination. Figure 9 shows several T2 hyperintense lesions in a periventricular distribution (predominantly left-sided on this image). There was faint nodular enhancement of these lesions following administration of gadolinium, suggestive of acute demyelination [Figure 10].

**Optic Neuritis and Neuromyelitis Optica (NMO)**

Patients with underlying MS present with optic neuritis in approximately 20% of cases [9]. Optic neuritis (ON) is defined as inflammation of the optic nerve, and is a common cause of acute painful visual loss [10]. Uncomplicated optic neuritis is a clinical diagnosis; however brain MRI is often performed in order to detect white matter plaques. MS cannot be diagnosed until a patient has a second demyelinating episode, at which point ON can be called MS-Associated Optic Neuritis (MSAON).

Figure 11 shows abnormal high T2 signal in the right optic nerve. There is abnormal enhancement of the right optic nerve following administration of gadolinium [Figure 12]. These imaging findings are characteristic of optic neuritis.

Devic's Neuromyelitis Optica (NMO) is an additional, rare cause of optic neuritis and is more common in Asian populations. NMO is an uncommon, severe demyelinating disease characterised by optic neuritis and myelitis [5]. It can be difficult to differentiate between NMO and MS, however doing so is important, as NMO carries a poorer prognosis and is managed differently. There are several clinical, biochemical (i.e. the presence of serum anti-aquaporin-4 antibodies) and imaging features which favour NMO. With regards imaging, spinal cord MRI is the investigation in which NMO can be most easily distinguished from MS [5]. NMO lesions usually extend over 3 or more vertebral segments, whereas MS cord lesions are typically short (extend <2 vertebral bodies). Additionally NMO lesions are located centrally within the spinal cord and are T1 hypointense, whereas MS cord lesions are T1 isointense and have a more posterolateral location.
Figure 13 gives a concise overview of the new 2010 McDonald criteria, and compares the current requirements for DIS and DIT with the previous criteria (see below).
Fig. 1: Sagittal T2-weighted brain MRI image. There are multiple ovoid high signal lesions in a periventricular distribution, characteristic of MS. Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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**Fig. 2:** Axial T2-weighted brain MRI image. Multiple hyperintense plaques are again noted in a periventricular distribution. There is also a background of marked involutional changes. Image courtesy of Dr R Guy (Radiology Consultant, Conquest DGH, Hastings).

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Fig. 3: Axial T2-weighted fluid-attenuated inversion recovery (FLAIR) brain MRI image. There are several high signal foci within the pons, in keeping with demyelination in an infratentorial distribution. Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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Fig. 4: Sagittal T2-weighted MRI image of the cervical spinal cord. There is an area of abnormally increased signal within the proximal spinal cord at the C2/C3 vertebral level. Note the length of the plaque, which is typically short (2 vertebral bodies or less) in MS. Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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**Fig. 5:** Axial T2-weighted FLAIR brain MRI image showing a focus of high signal in the left frontal lobe, in a juxtacortical location (arrow). Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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**Fig. 6:** Axial unenhanced CT head image showing an ill-defined focus of low attenuation at the lateral border of the right thalamus. The nature of this abnormality was further characterised using MRI (please see Figure 6). Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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Fig. 7: Axial T2-weighted brain MRI image of the same patient as in Fig. 6 showing a high signal focus within the lateral aspect of the right thalamus. Several additional high signal foci were also noted in a periventricular distribution (not shown on this image), suggestive of possible demyelination. Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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Fig. 8: Axial T2-weighted brain MRI image in the same patient as shown in Figure 6. This follow-up MRI performed one month later shows a new curvilinear focus of increased signal in the left parietal region (arrow). This suggests disease progression, and satisfies the new criteria for DIT. Image courtesy of Dr E Owens (Consultant Radiologist, Eastbourne DGH).

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Fig. 9: Axial T2-weighted FLAIR brain MRI image. Several periventricular hyperintense lesions are noted (predominantly left-sided in this image). Image courtesy of Dr R Guy (Radiology Consultant, Conquest DGH, Hastings).

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Fig. 10: Axial post-gadolinium T1-weighted brain MRI image. Some of the periventricular lesions noted in Figure 9 show faint nodular enhancement following gadolinium administration. This is in keeping with acute demyelination. Image courtesy of Dr R Guy (Radiology Consultant, Conquest DGH, Hastings).

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**Fig. 11:** Coronal T2-weighted short T1 inversion recovery (STIR) brain MRI image. There is abnormal T2 hyperintensity in the right optic nerve, in keeping with optic neuritis.

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Fig. 12: Coronal T1-weighted post-gadolinium brain MRI image. There is abnormal enhancement in the right optic nerve, in keeping with optic neuritis.

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**Fig. 13:** Table 1. A concise overview of the new 2010 McDonald criteria for MRI-demonstration of DIS and DIT, with comparison to the 2005 criteria.

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Conclusion

A diagnosis of MS may have potentially devastating consequences for a patient. Equally, uncertainty of the diagnosis may be stressful for the individual and unnecessarily delay treatment. Use of the new consensus McDonald criteria may facilitate the management pathway without compromising diagnostic accuracy. However, it is imperative that subjects to whom the criteria is applied have a presentation compatible with an inflammatory demyelinating condition of the CNS, as the McDonald criteria are only valid in such cases and isolated T2 hyperintense lesions could otherwise represent other inflammatory demyelinating conditions such as neuromyelitis optica (NMO), ADEM, metabolic, ischaemic or other aetiologies [4]. Caution should also be exercised if the McDonald criteria are applied to paediatric patients or patients of non-Caucasian origin.
References


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