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B. Mark Keegan
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Chapter 1

Pitfalls in identifying the classical clinical features of MS

Introduction

Multiple sclerosis (MS) is the prototypical inflammatory demyelinating disease of the central nervous system (CNS). While it is the most common presentation of this spectrum of diseases, other inflammatory, infectious, structural, genetic disease processes and even normal presentations are competing diagnoses. While in the past it was difficult in some cases to make a clear diagnosis of MS based mostly on clinical evaluation, current investigational technologies, particularly brain and spinal cord magnetic resonance imaging (MRI), have made this far easier today. It is critical to make a confident and definitive diagnosis of MS prior to administering effective MS medications in order to avoid potentially toxic therapies aimed at different disease processes. In this introductory chapter we approach a simplified three-step assessment when diagnosing MS:

Three-step diagnosis of MS

Step 1: Identify the classical clinical features of MS
Step 2: Conduct a neurological examination for signs of MS
Step 3: Assess investigational evidence of MS

Step 1: Identify the classical clinical features of MS

See Table 1.1.

One way to diagnose multiple sclerosis with confidence, as opposed to competing diagnoses, that will be encouraged in this book is to undertake a three-step diagnostic evaluation. The first step involves identifying the presence or absence of the classical, clinical features of multiple sclerosis. This includes, but is not limited to, optic neuritis, diplopia, trigeminal neuralgia, dysarthria, ataxia, hemiparesis, hemisensory loss, Lhermitte symptom and symptoms of acute or progressive myelopathy (sensory level, paraparesis or quadriparesis, sphincteric dysfunction). Apart from paroxysmal symptoms of MS, such as trigeminal neuralgia and painful tonic spasms, which are discussed further in Chapter 3, most symptoms that are classified as new clinical attacks of multiple sclerosis should last at least twenty-four hours but usually last days to weeks in duration. For instance, a common presenting symptom of multiple sclerosis is optic neuritis. The typical history in a patient with optic neuritis is of painful, unilateral, central visual loss that worsens over hours to days. The visual loss may plateau over a number of days to weeks and then improve spontaneously or with the use of corticosteroids, again over many weeks. Visual improvement often may be excellent but recovery may be incomplete. Painless loss of vision is less common a manifestation of optic neuritis due to MS, as is binocular loss of vision. These latter findings should prompt a careful search for an alternative cause for the visual impairment.

In contradistinction to the painful nature of optic neuritis, diplopia due to MS should be painless. This condition should also be "binocular" in nature, in that if the patient closes one eye, the diplopia is abolished as the visual input is coming solely from the uncovered eye. It may be vertical or horizontal or oblique in nature. Diplopia of most etiologies comes on acutely, but the severity of the diplopia due to a demyelinating cause may worsen over hours to days.

MS patients may develop Lhermitte symptoms. This is an unusual paresthesia of a "shock-like" or "buzzing" sensation that occurs classically on extreme neck flexion. This is indicative of a demyelination within the cervical spine and is produced by the neck flexion stretching the spinal cord, which leads to demyelinated axons being hyperirritable and discharging unnaturally with this stimulus. This symptom, while characteristic of MS, may also occur in patients with other causes of cervical myelopathy

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Table 1.1

Symptom	Classical clinical features	Pitfall symptoms
Optic neuritis	Painful, monocular visual loss	Painless, binocular loss, refractive error, migrainous visual auras
Diplopia	Painless, binocular, long duration	Monocular, brief duration (seconds to minutes)
Trigeminal neuralgia	Lancinating shock-like pain with typical triggers (e.g. touching, speaking, cold air)	Constant facial pain
Sensory symptoms	Sensory level (spinal cord level); hemisensory impairment (cortical/subcortical level); alternating sensory loss ipsilateral face and contralateral limb (brainstem level)	Random, fleeting sensory disturbance in non-neurological distributions
Lhermitte symptom	Shock or buzz on neck flexion	Cracking, popping on neck rotation
Bladder/bowel impairment	Urge-related incontinence	Urinary frequency, anatomical explanations (e.g. pregnancy, surgeries)
Motor and gait impairment	Weakness, or ataxia, often asymmetrical; assess functional limitation; no. of blocks walked, stairs climbed	Restricted activity due to fatigue, pain or deconditioning

such as compressive cervical spondylotic myelopathy and subacute combined degeneration of the spinal cord due to B12 deficiency.

Hemiparesis or hemisensory deficit involving the face, arm, and leg could reflect CNS demyelination within the cerebral white matter. Again, the clues to CNS demyelination as an etiology are the onset and worsening over hours to days, lasting for days to weeks and then improving spontaneously or with corticosteroid treatment. Importantly, symptoms of a sensory myelopathy are common in multiple sclerosis, and this is generally heralded by an ascending sensory level and may be accompanied by quadriparesis or paraparesis (depending on cervical or thoracic location of the lesion) and bowel and bladder dysfunction.

Gait impairment is common in multiple sclerosis, and it is important on a clinical evaluation to identify the actual functional limitations experienced and the exact reasons behind it. For example, many patients have limitations that are due entirely to fatigue or deconditioning. While this limitation is restricting and very important, more compelling evidence of a progressive myelopathy is a reliable onset of impairing symptoms with less exertion. To use a common clinical example indicative of a progressive myelopathic gait disorder, a patient may describe walking for two miles three years previously, followed by leg dragging at one mile two years ago, then at half a mile one year ago and is now dragging after only walking one city block. If the gait limitation is due solely to ataxia, the number and frequency of falls and requirement of gait aids (walking stick, furniture, other people) constitutes a key informative history. Multiple sclerosis–related gait disorders are generally due to corticospinal track impairment with progressive dragging of one limb or significant gait unsteadiness with falls. Bowel and bladder dysfunction may occur with multiple sclerosis, particularly with lesions of the spinal cord, and may herald as urinary or bowel urgency with associated urge-related incontinence. This should be distinguished from stress urinary incontinence symptoms (e.g. incontinence with coughing, Valsalva strain or laughing) that are often due to anatomical impairment rather than CNS disease.

MS is commonly associated with sensory loss. By far the most sensitive clinical finding of sensory loss due to MS is loss of distal vibratory sense in the

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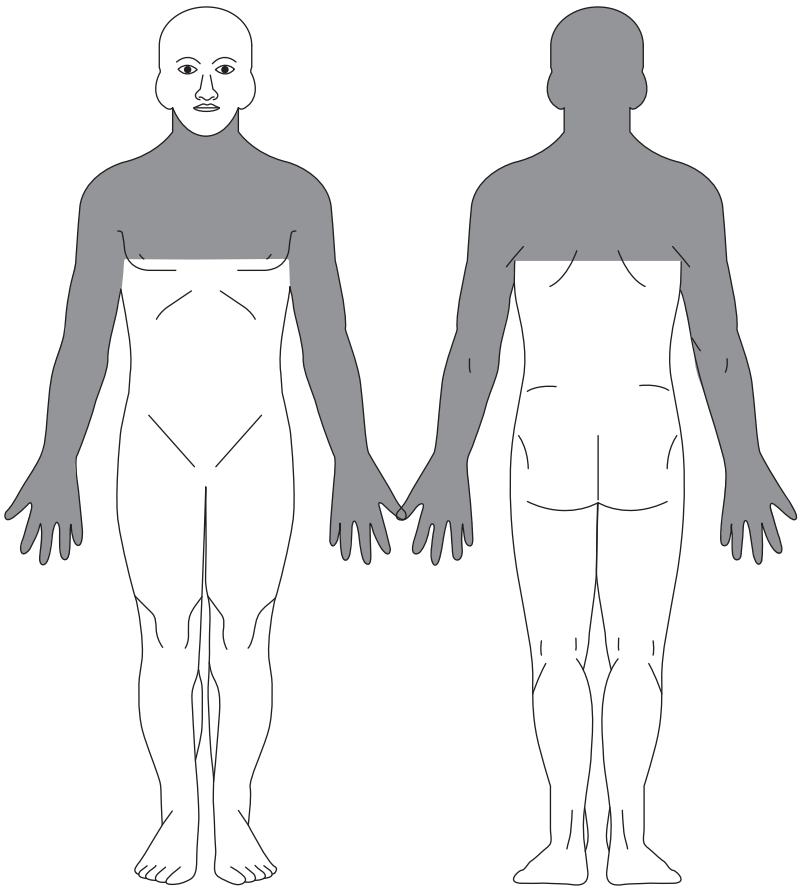

Figure 1.1 Sensory level described by patients with myelopathies.

feet. It should be noted that a "sensory level" indication of a spinal cord impairment, as demonstrated in Figure 1.1, is best evaluated based on clinical history rather than findings on neurological examination. For example, the patient may inform the clinician of a sensory-level pattern. On examination the clinician may or may not be able to identify or confirm the abnormal signs reflecting this impairment, but the clinical history of a definite sensory level should be taken as a highly compelling indication of a spinal cord lesion. An important distinction should be made between a spinal cord sensory level and "large fiber" peripheral neuropathies which may cause similar distal loss of vibratory sense. The clinician should be concerned about a spinal cord lesion when the hands are involved prior to or simultaneously with the feet, and of course if saddle (genital and buttock) region numbness is prominent or if there are signs of upper motor neuron motor impairment consistent with CNS (not peripheral nervous system [PNS]) disease.

Step 2: Conduct a neurological examination for signs of MS

See Table 1.2.

The second step in diagnosing multiple sclerosis, following a detailed evaluation for the classical clinical symptoms of MS, is a comprehensive neurological examination. One way to simplify the results of neurological evaluation is to classify it as normal neurological examination, neurological examination consistent with multiple sclerosis or neurological examination consistent with an alternative neurological disease as the etiology. It should be strongly emphasized that many MS patients will have an entirely normal neurological examination. A neurological examination consistent with MS means that they exhibit one or more characteristic, but not defining or pathognomonic, abnormalities. For instance, patients may have signs of optic neuropathy such as visual acuity impairment, central scotoma, color vision impairment and a pale optic disc,

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Table 1.2

Examination	Examination findings	Comment
Dementia	Impaired cognition on mental status exam	Pitfall: non-specific memory concerns are common in MS but not reliably distinguishing; severe dementia is uncommon but occurs in MS
Optic neuropathy	Scotoma, acuity deficit, color vison impairment, relative afferent pupillary deficit	Pitfall: acuity deficit due to refractive errors
Internuclear ophthalmoplegia (INO)	May be bilateral, often incomplete with slowed adduction and dysconjugate nystagmus	Rapid saccades back and forth from extreme horizontal gaze identifies INO
Limb ataxia	Often asymmetrical	Pitfall: palatal myoclonus is a feature of stroke and Alexander disease
Upper motor neuron weakness	Pyramidal distribution; extensor plantar responses (Babinski signs)	Bilateral extensor plantar responses strongly suggest a myelopathy
Sensory loss	In pattern of cortical, brainstem or spinal cord impairment	Distal lower extremity vibratory loss most sensitive; pitfall: pin, light touch sensation is highly subjective and better appreciated on clinical history rather than findings on examination
Gait	Spastic, ataxic quality is most common, often asymmetrical	Pitfall: some patients have functional non-neurological gait disorders, or are impaired by pain

all of which are characteristic of MS, particularly if the patient has a clinical history of typical optic neuritis. Extraocular movement abnormalities such as unilateral or bilateral internuclear ophthalmoplegia (INO) indicative of interruption of the brainstem medial longitudinal fasciculus connecting the oculomotor and contralateral abducens nerve are classically caused by MS. It should be noted, however, that INO due to MS is often incomplete and the identification of slowed adduction with dysconjugate nystagmus is best seen by having the patient make repeated, rapid saccades back and forth from one extreme horizontal gaze to the other. Findings of corticospinal tract impairment with upper motor neuron–type weakness (hyperreflexia, "pyramidal" distribution weakness, extensor plantar responses [Babinski signs]) are commonly found in MS. MS patients may also have signs of cerebellar dysfunction with gait and limb ataxia as well as a cerebellar dysarthria. Distal upper or lower extremity sensory loss particularly to vibration is characteristic of dorsal spinal cord dysfunction in MS.

Step 3: Assess investigational evidence of MS

The third step in diagnosing MS is evaluating the results of appropriately selected investigations (Table 1.3). The most sensitive way to investigate a possible diagnosis of MS is the use of magnetic resonance imaging (MRI) of the brain, cervical, and thoracic spinal cord. Typical ovoid T2-weighted hyperintense lesions located periventricularly within the brain, within the posterior fossa, juxtacortically, chronic T1-weighted hypointense lesions ("black holes") and acute T1 lesions that enhance following gadolinium administration are particularly characteristic of multiple sclerosis (see Figure 1.2). Similar areas of ovoid abnormal T2 signals that are short in length and generally laterally placed are found within the cervical and thoracic spinal cord (Figure 1.3). While it is extremely common to find non-specific MRI lesions within the brain, such as those related to aging, migraine headaches, hypertension and other vascular risk

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factors, these types of typical T2 lesions within the cervical and thoracic spine are characteristic of multiple sclerosis and do not arise simply due to these conditions.

Table 1.3 Investigational evidence of MS.

- Brain MRI
- Cervical and thoracic spinal cord MRI
- CSF examination: IgG index, unique CSF oligoclonal bands
- Evoked potentials: visual, somatosensory, rarely brainstem auditory evoked potentials
- Serological evaluations for MS mimickers
- Additional evaluations for MS mimickers

The cerebrospinal fluid (CSF) examination remains an important diagnostic test in the evaluation of MS. In particular, CSF elevations in the immunoglobulin G (IgG) index and unique CSF oligoclonal bands are characteristic of an immune-mediated CNS disease process generally and often of multiple sclerosis specifically. Examination of the white blood cell count, protein levels and other values are of benefit, but generally only to rule out mimickers of multiple sclerosis, including CNS infectious diseases, CNS neoplasms or other non-MS CNS diseases.

Electrophysiological evaluation of CNS pathways with evoked potentials also assists in MS diagnosis. Visual evoked potentials (VEP) assess for conduction deficit in the optic nerves. Somatosensory evoked potentials (SSEP) may identify impaired conduction

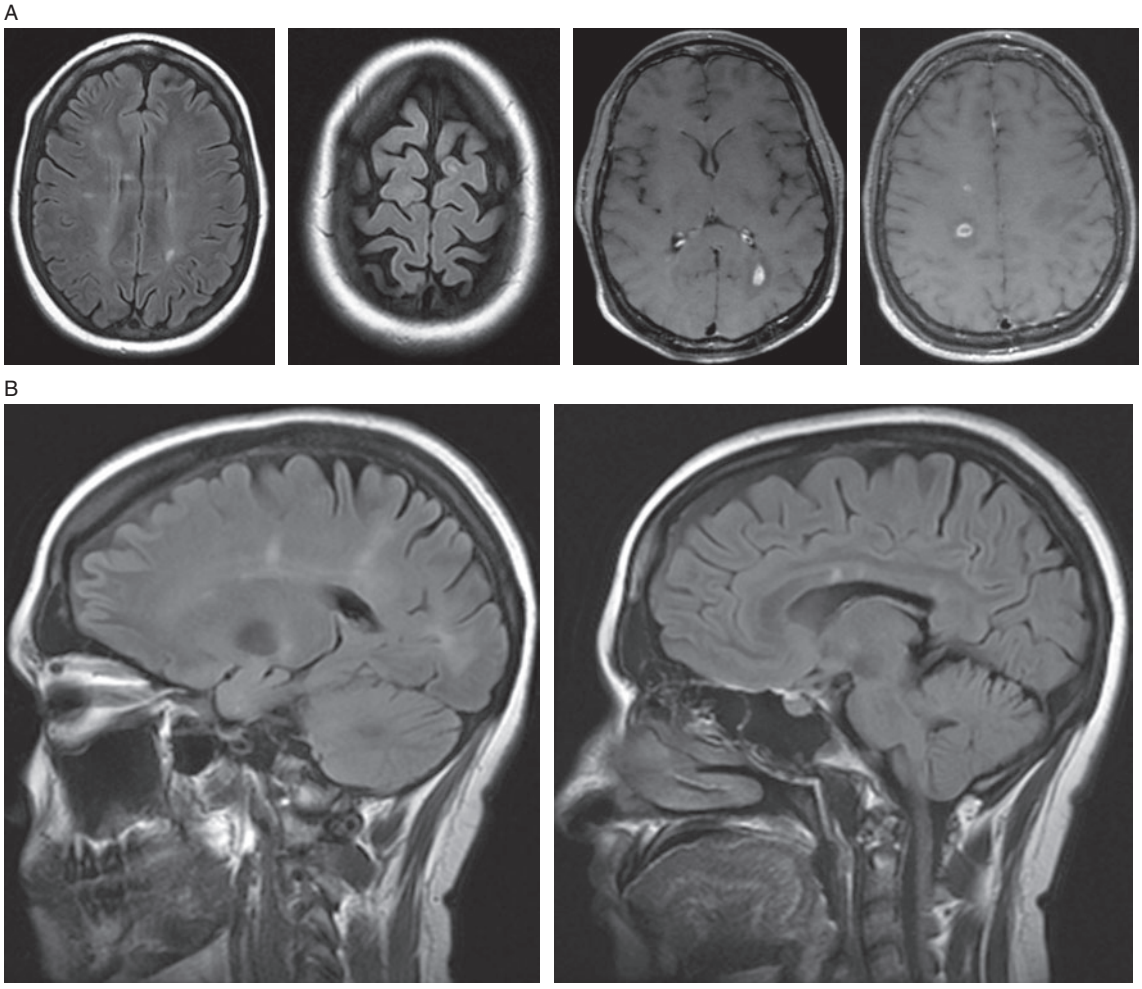

Figure 1.2 A) Axial MRI brain showing typical FLAIR periventricular, juxtacortical and T1 gadolinium–enhancing lesions. B) Sagittal MRI brain FLAIR sequence showing typical "Dawson's fingers" and corpus callosum MS lesions.

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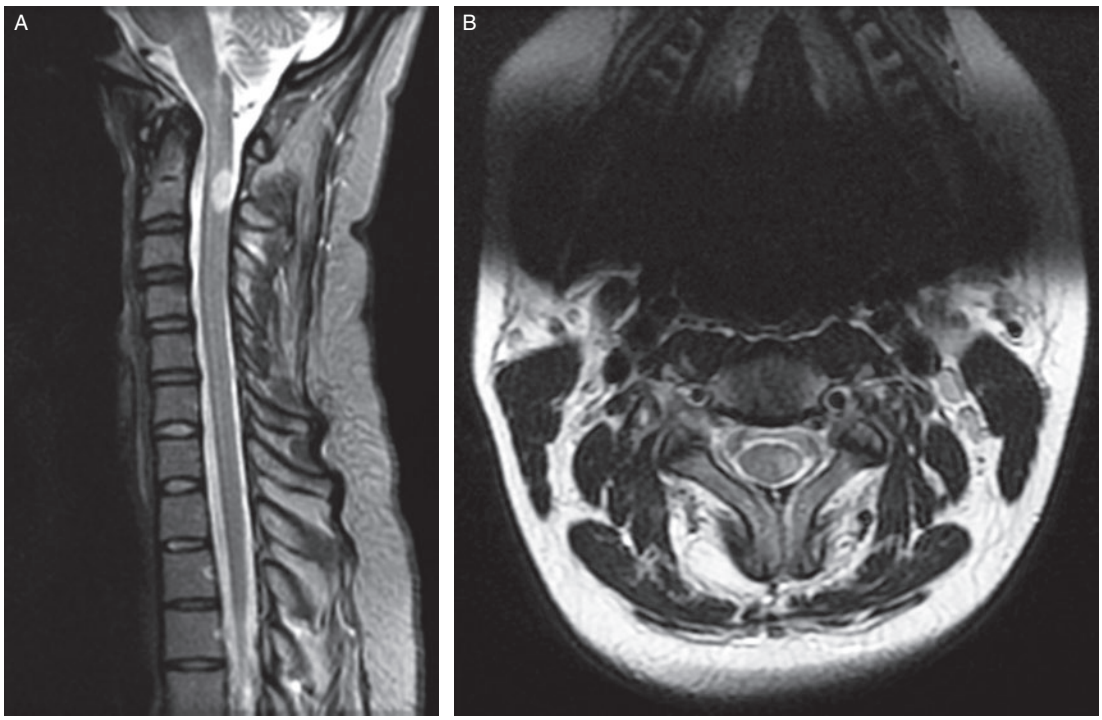

Figure 1.3 Sagittal T2 MRI cervical spine dorsally placed, short-segment, ovoid lesion characteristic of multiple sclerosis. Axial T2 MRI cervical spine in the same patient shows the lesion is in the left lateral aspect of the spinal cord.

in the central proprioceptive pathways. Brainstem auditory evoked potentials (BAEP) may, in rare cases, be useful in identifying central versus peripheral hearing impairment in the evaluation of multiple sclerosis.

Serological and neuroradiological testing, as well as other selected investigations searching for infectious, vascular, traumatic, neoplastic, inherited and other causes that may mimic multiple sclerosis, may be required in cases where a diagnosis of MS remains uncertain (see Table 1.4). Many MS cases are straightforward and few if any investigations outside of MRI, CSF and evoked potentials are required.

Case 1: A woman with numbness and prior visual loss: possible MS

A 41-year-old woman noted one day while showering and shaving her legs that her right leg and thigh sensation was gone. This involved the whole limb, both anterior and posterior. The sensory loss then progressed the next day to involve the foot of that lower extremity, her buttocks, her genitalia, and the next day up to the lower back, all on the right side. She then had moderate, but incomplete, spontaneous improvement in the sensation over roughly the next three weeks. She had no left-side impairment, nor any motor weakness or bowel or bladder dysfunction. She had noticed higher levels of fatigue over the previous few months.

She recalled that five years previously, during a stressful time, she had left eye blurriness with mild ocular pain that resolved spontaneously over about three or four weeks. She had had no diplopia, dysarthria, dysphagia or hearing loss and no other episodes of visual or sensory impairment. Her ambulation was normal and she could easily walk a mile and recently was able to cycle thirteen miles.

On examination, her mental status was normal, while visual acuity and color vision were normal bilaterally. Her motor exam was normal. A sensory exam revealed decreased temperature sensation up to T5 on the right side, with preserved pin vibratory and joint position sense. Muscle stretch reflexes were normal and her plantar responses were downgoing bilaterally. Her gait was normal.

A brain MRI demonstrated areas of radially oriented nonenhancing increased T2 signals within the cerebral hemispheres, and T2 lesions in the cerebellum and pons suggestive of MS. Gadolinium-

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Table 1.4 Clinical and laboratory red flags

Clinical red flags	Implication
Headache/meningismus	Sarcoidosis, SLE, Lymphomatosis
Stroke-like events	SLE, Antiphospholipid Syndrome, CNS angiitis, embolic strokes
Myopathy	Mitochondrial disease, Sarcoidosis
Neuropathy	B12 deficiency, Dysmyelinating D/o
Diabetes insipidus	Sarcoidosis, Histiocytosis
Bone lesions	Histiocytosis/Erdheim Chester disease
Pulmonary symptoms	Sarcoidosis, SLE
Cardiac symptoms	Embolic cerebral infarcts
Mucosal ulcers	Behçet's disease
Arthritis/arthralgia	SLE, Sjögren's disease
Rash	SLE, Fabry's disease, Lyme
Oculomasticatory myorhythmia	CNS Whipple
Uveitis	Behçet's disease, SLE
Prominent family history	CADASIL, Hereditary Spastic Paraparesis, Dysmyelinating disorder
Endocrinopathy	Sarcoidosis, Histiocytosis
Retinopathy	Mitochondrial disease, Susac syndrome
Thrombotic events	Antiphospholipid Syndrome, SLE
Laboratory red flags	
Elevated ESR	Vasculitis, SLE, Sjögren's
High titer ANA	Connective tissue disease
Elevated serum lactate	Mitochondrial disease
Anemia/cytopenia	SLE, B12 deficiency
Persistent/marked CSF pleocytosis	Lymphoma
Neutrophilic CSF pleocytosis	Behçet's disease, CNS Whipple

SLE – systemic lupus erythematosus, CADASIL – cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy

enhancing lesions were found throughout, raising suspicions of active demyelinating disease (Figure 1.4). A cervical spine MRI was normal. A thoracic spine MRI showed subtle, short segment T2–signal abnormality at T4 (not shown). A CSF examination showed elevations in oligoclonal bands and IgG index and normal other values. Visual evoked potential showed mild slowing on the left visual pathway.

Three-step assessment

1 Classical clinical features of MS: sensory myelopathy with resolution, monocular visual loss with pain typical of optic neuritis
2 Neurological examination: myelopathic sensory level
3 Investigations: MRI brain consistent with MS, MRI cervical spine normal, thoracic spine consistent

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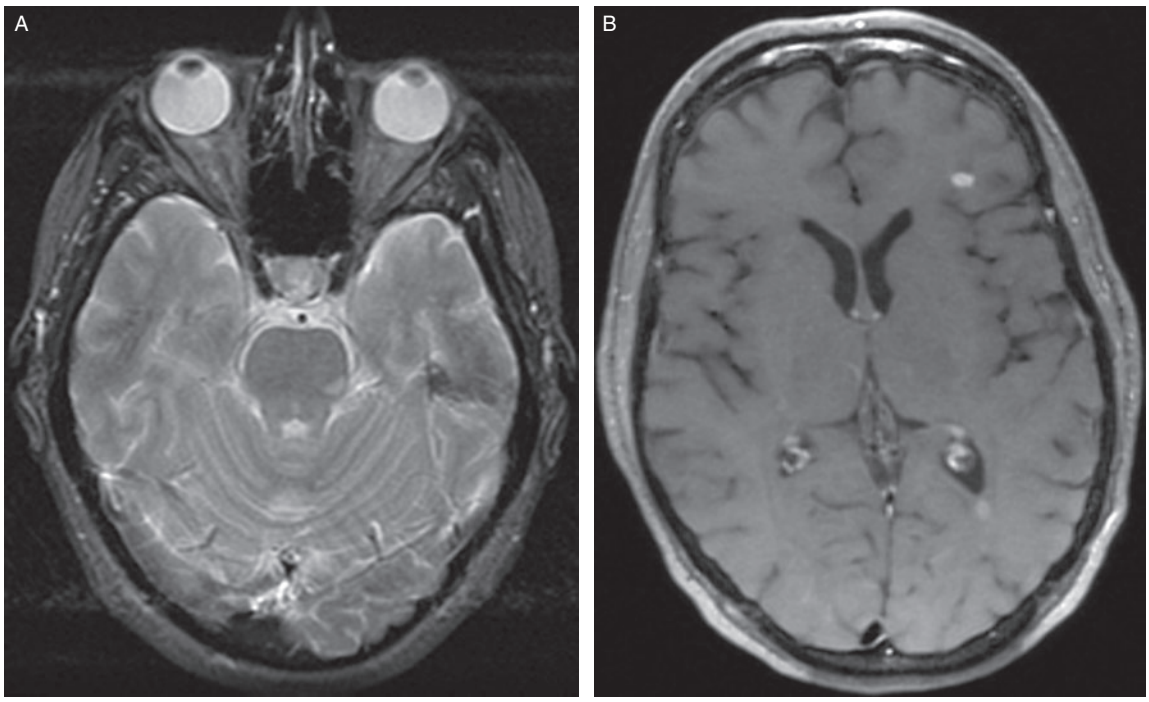

Figure 1.4 A) Axial T2 MRI brain showing left pontine MS lesion. B) Axial T1 MRI brain with gadolinium showing enhancing white matter lesion in left frontal lobe.

with MS; CSF consistent with MS; visual evoked potentials abnormal on left

Diagnosis: Relapsing remitting multiple sclerosis.

Tip: Using the three-step assessment, this patient satisfies all of the steps. She has had classical symptoms of MS attacks with spontaneous resolution including a sensory myelopathy and optic neuritis; her neurological examination is consistent with MS with a thoracic spinal cord sensory level. Important investigations including the brain and spinal cord MRI, CSF examination and evoked potentials are all consistent with MS as the correct diagnosis.

The patient initiated treatment with interferon beta -1a subcutaneous injections three times weekly for relapsing remitting MS.

Case 2: A woman with diffuse pain and abnormal brain MRI scan. It is MS?

A 44-year-old woman presented for evaluation of possible MS. She had a long history of typical migraine headaches without aura, and brain neuroimaging was performed to evaluate the headaches. She described typical migraines with severe headaches that worsened with activity and were associated with photophobia, phonophobia and nausea and vomiting. She had a history of multiple foot surgeries with significant allodynia and complex regional pain syndrome. More recently, she developed more diffuse pain symptoms and it was suspected that she had fibromyalgia. She had symptoms of non-specific memory concerns. Despite this, she was able to prepare all of her meals, go shopping independently and drive without difficulty.

While she had no classical clinical attacks of multiple sclerosis, she reported rare, random, brief, slurring dysarthria as well as word-finding difficulties, but no clear history of aphasia, paraphasic errors, or prolonged dysarthria. She did not have significant dysphagia but occasionally reported rare choking episodes. She had tinnitus but no significant hearing loss. She had a "cracking" sensation on neck movement but no typical Lhermitte symptom (also known as Lhermitte sign). She was limited to walking only one block, but this was entirely due to pain; and she would hold on to her husband because of this.

On neurological examination, she was anxious and appeared depressed but formal mental status

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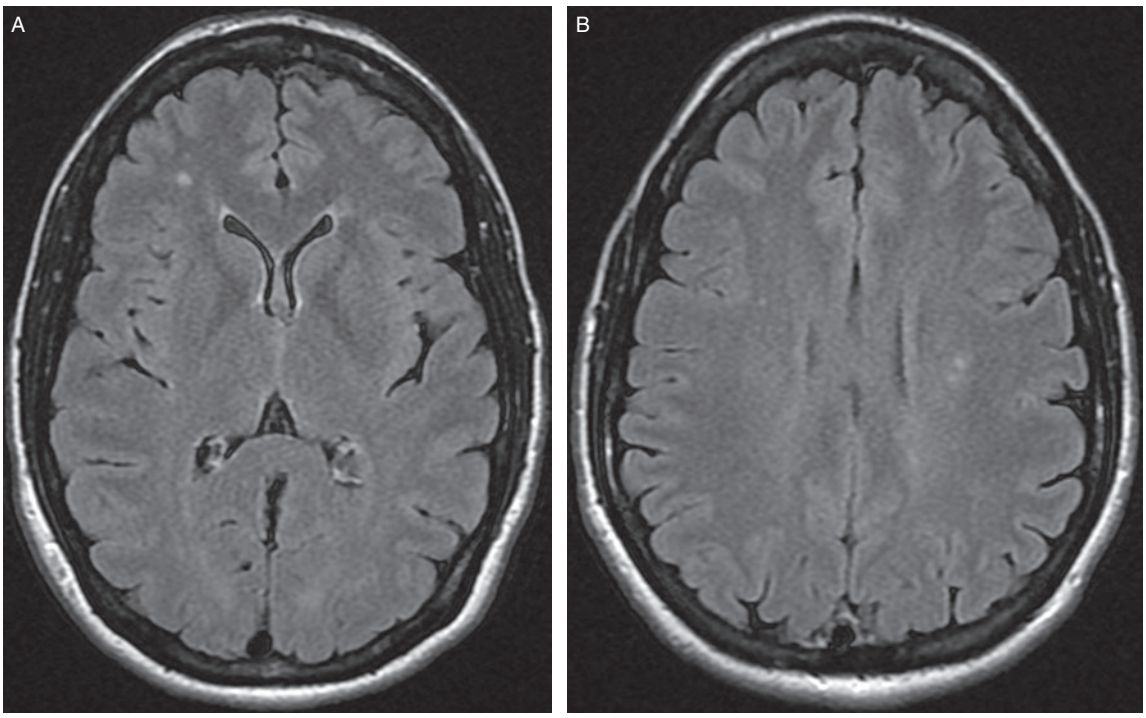

Figure 1.5 Axial FLAIR MRI brain showing commonly found, non-specific, randomly placed signal change, not highly suggestive of MS lesions.

testing was normal. She had an antalgic gait but the rest of her neurological examination was normal.

A brain MRI showed small, non-specific areas of abnormal T2 signal (Figure 1.5). A cervical and thoracic spine MRI was normal and negative for radiological features of demyelinating disease. Visual and somatosensory evoked potentials were both normal. A CSF examination was normal with no elevation in CSF oligoclonal bands or IgG index.

Three-step assessment

1 Classical clinical features of MS: none
2 Neurological examination: normal
3 Investigations: MRI brain: non-specific findings; MRI cervical and thoracic spine negative; CSF normal; evoked potentials negative

Diagnosis: Migraine without aura, fibromyalgia and complex regional pain syndrome, abnormal brain MRI scan with non-specific findings likely related to migraine.

Tip: Diffuse pain, while perhaps common in MS, is not a highly specific, clinically discriminating feature of multiple sclerosis and needs to be investigated thoroughly for alternative causes, such as fibromyalgia. Complex regional pain syndrome can be the cause of severe focal pain disorders. Undertaking further investigational studies to try to confirm further MS-related abnormalities on spinal cord MRI, evoked potentials and CSF examination is often important to evaluate an abnormal brain MRI with non-specific findings such as in this case.

Case 3: A patient with numbness of the feet rapidly involving the hands. Where is the lesion in the nervous system?

A 43-year-old woman was wrapping holiday gifts when she developed bilateral foot numbness. The next day she noticed similar numbness, now involving the hands bilaterally, and following that had ascending loss of sensation in both lower extremities. The numbness then progressed up the lower extremities to the upper thighs and lower abdomen including the genital and buttocks region, and finally it involved the costal margin. She had no motor weakness or bowel or bladder incontinence despite the lack of perineal sensation. She had no accompanying

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symptoms of cranial nerve dysfunction and no prior significant symptoms. She had no family history of multiple sclerosis.

On neurological examination, her mental status was normal. Her motor exam was normal throughout, muscle-stretch reflexes were brisk, but plantar responses were flexor bilaterally. Vibratory and joint position senses were normal. Her ambulation was normal.

Brain MRI showed one non-specific area of abnormal T2 signal but was otherwise normal. A cervical spine MRI showed a short segment, oval area of abnormal T2 signal with subtle gadolinium enhancement within the cervical cord at C2 consistent with an inflammatory demyelinating lesion (Figure 1.6). A thoracic spine MRI was normal. The lumbar spine MRI scan was normal with no abnormality of the cauda equina nerve roots.

Three-step assessment

1 Classical clinical features of MS: inflammatory cervical myelopathy
2 Neurological examination: normal
3 Investigations: MRI brain: non-specific findings; MRI cervical spine: typical MS lesion; MRI thoracic and lumbar spine negative

Diagnosis: Clinically isolated syndrome (CIS) of demyelination with inflammatory cervical myelopathy. This is within a relatively low-risk group for future development of multiple sclerosis given the lack of other inflammatory demyelinating lesions compared to CIS patients with >2 asymptomatic non-enhancing T2 MS lesions.

Tip: A sensory "glove" distribution coming immediately following or coinciding with "stocking" distribution should suggest the cervical spinal cord as the level of the nervous system involved. It should not as strongly suggest a peripheral nerve distribution which, if length-dependent and symmetrical, would affect the feet and progressively much travel further up the lower extremities prior to hand involvement ("stocking", then "glove" distribution). A sensory level as was described in the patient above is a symptom that is typically obtained on a clinical history rather than as a clinical finding on neurological examination.

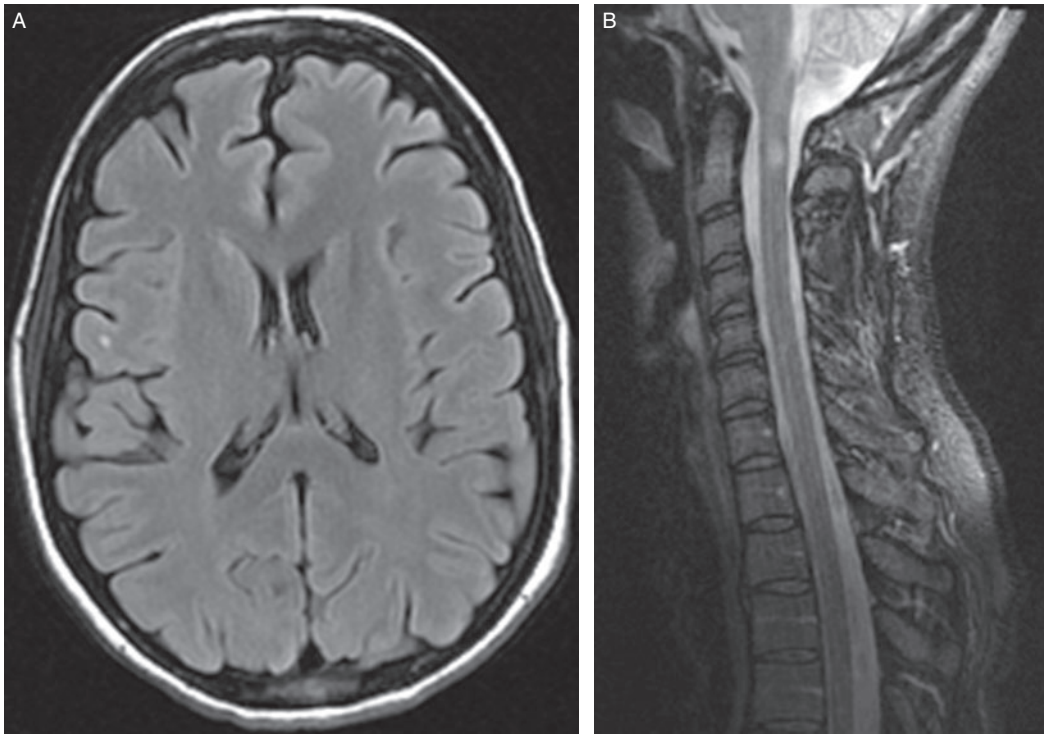

Figure 1.6 A) Axial FLARI MRI brain showing small, non-specific lesion in right hemispheric white matter. B) Sagittal T2 MRI cervical spine short-segment, ovoid lesion characteristic of multiple sclerosis.

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