

Reminder of important clinical lesson

Paroxysmal symptoms in multiple sclerosis masquerading as transient ischaemic attacks

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Paroxysmal demyelinating events produced sudden onset, transient, recurrent symptoms that were troublesome to our patient and puzzled the referring clinician who mistook them for transient ischaemic attacks or epilepsy. It was important to recognise the true nature of the underlying problem because the symptoms could then be readily treated; this is especially critical because the symptoms, in this case, represent a relapse of multiple sclerosis and, therefore, are significant for examination during the diagnosis.

BACKGROUND

Clinicians recognise the multitude of symptoms that can occur during the course of multiple sclerosis. Of the many features of the condition, however, its paroxysmal manifestations are most frequently misinterpreted or simply missed. Affecting 8.6% of patients with multiple sclerosis at some point,¹ these are recurrent, stereotyped, collections of symptoms that persist for only a few seconds at a time. Particularly where these are the presenting feature of the disease, they are challenging for non-specialists while differentiating these symptoms from transient ischaemic attacks, epilepsy or functional disorders. A cluster of paroxysmal symptoms lasting days to months represent the clinical consequences of a new episode of inflammatory demyelination. It places the onus on the clinician to look for other demyelinating events in the past or future, in case a diagnosis of multiple sclerosis is justified.

This case illustrates the importance of recognising the readily treatable paroxysmal symptoms of demyelination and how a comprehensive clinical history remains critical in establishing the correct diagnosis of multiple sclerosis.

CASE PRESENTATION

A 45-year-old right-handed man was referred to the transient ischaemic attack clinic. He presented with 4 weeks of episodic, transient, sudden-onset blurred vision and a sensation of 'heaviness' in his left arm. During these attacks, he would often drag his left leg if walking and feel his fingers stiffen if typing, which made him pause until the symptoms passed and normal feeling returned. He experienced 10–20 stereotyped episodes daily, each lasting 5–10 s, passing spontaneously or when the patient made purposeful neck movements. The patient was completely well between attacks. He suspected the attacks were related to movement. With careful further questioning he recalled an episode of right-leg numbness a year previously that lasted 2 weeks. This had not come to medical attention at the time, as he had attributed the symptoms to an old sporting injury.

The patient was generally fit and well, with no history of neurological symptoms. Though he had recently seen an osteopath for problems with his right shoulder, he otherwise had no past medical history and was taking no regular medication. A non-smoker, he consumed little in the way of alcohol and had an unremarkable family history.

The cardiovascular examination was normal. Neurological examination was normal, apart from slightly brisk left-arm reflexes.

One attack was witnessed. Although the patient was able to speak normally and remained able to stand, finger–nose testing was slow and very hesitant, but accurate. Power remained normal. This resolved within 10 s, before the pulse was taken or further examination could be attempted.

INVESTIGATIONS

Routine blood tests and a 24-h electrocardiogram were normal. An MRI of the patient's brain, however, showed lesions of the corpus callosum, consistent with demyelination and an incidental cavernous haemangioma in the posterior fossa which was entirely confined to the cerebellum (see figures 1 and 2).

DIFFERENTIAL DIAGNOSIS

Paroxysmal demyelinating episodes. In context, probably multiple sclerosis.

TREATMENT

Membrane-stabilising drugs are known to be effective in dealing with the symptoms, and this patient was started on a single daily dose of 100 mg of carbamazepine.

OUTCOME AND FOLLOW-UP

The paroxysmal episodes resolved promptly with carbamazepine, and the drug was discontinued after 3 months. Given the earlier leg numbness, the paroxysmal symptoms qualified as a relapse of multiple sclerosis. The patient was subsequently started on -interferon, a disease-modifying drug for multiple sclerosis.



Figure 1 T2 FLAIR parasagittal slice from the patient's MRI showing lesions in the corpus callosum.

DISCUSSION

The numerous clinical manifestations of paroxysmal demyelination reflect the disruption of axonal conduction in different areas of the brain. This may arise idiopathically, in demyelinating syndromes other than multiple sclerosis (such as acute disseminated encephalomyelitis²), or as part of multiple sclerosis.

All variants share certain common features.² As in the case presented here, the symptoms are often brief, persisting for a few seconds but recurring many times each day, leaving the patient normal during the period between attacks. Although the individual episodes are brief, the time scale of a cluster of paroxysmal symptoms, typically days to months followed by remission, is similar to that of a relapse of multiple sclerosis. Symptoms are often stereotyped, may be individual to the patient and are frequently triggered by sensory stimuli, over breathing or, as in this case, by movement. With their sudden onset, they are easily attributed to epilepsy, transient ischaemic attacks, anxiety or labelled as *functional* and may even bewilder competent neurologists. Though probably underrecognised, they are most common early in the course of multiple sclerosis,³ with 8.6% of patients experiencing paroxysmal symptoms at some stage¹ and some experiencing more than one form over time, suggesting demyelination in different areas. They are particularly common in patients from Japan and Taiwan.⁴

The most well-recognised types of paroxysmal symptoms are given here in the order of likely prevalence on the basis of case series (eg, reference⁵). There is general agreement that 2% of people with trigeminal neuralgia have multiple sclerosis and 1% of people with multiple sclerosis will have trigeminal neuralgia.⁶ The pain is similar to that experienced with the idiopathic form, only more often bilateral.⁷ Frequently overlooked as dizziness, cerebellar ataxia frequently occurs with dysarthria. Tonic spasms can affect the face or limbs and may manifest as a transient disturbance in handwriting. Sensory symptoms, including numbness and pain typically occurring in the limbs or face, can be difficult to diagnose owing to their transient nature. Itching is recognised and is often intense. Akinesia, with paroxysmal loss of function in one or more limbs, may also be encountered. Paroxysmal involuntary movements, however, are usually the result of comorbidity rather than demyelination.⁸

The postulated mechanism provides a rationale for the treatment of these frequently troublesome symptoms. After a period of reduced excitability following demyelination, some axons become hyperexcitable. These then spontaneously discharge and may activate groups of neighbouring neurons via ephaptic transmission.⁹ Demyelinated sensory neurons are probably more prone to hyperexcitability owing to the greater expression of sodium channels.¹⁰ On this basis, membrane-stabilising drugs are

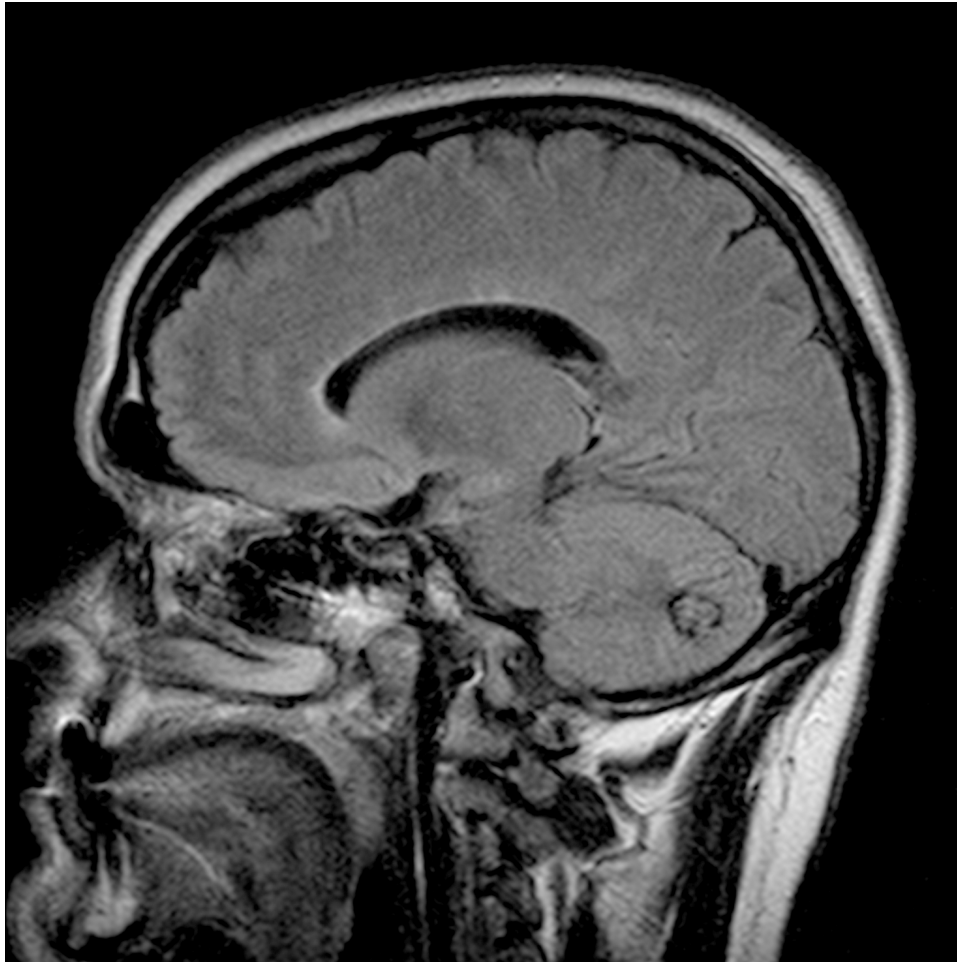


Figure 2 T2 FLAIR image showing the incidental posterior fossa cavernous haemangioma was contained entirely within the cerebellum.

prescribed in low dosages, and almost all patients respond rapidly.²

It is important that paroxysmal demyelinating events are not missed to facilitate a timely diagnosis, initiate appropriate treatment and identify relapses. In this case, with the history of leg numbness occurring a year prior to the paroxysmal symptoms and the positive MRI findings a diagnosis of probable multiple sclerosis was made.

Learning points

- ▶ It is important to consider paroxysmal demyelination in the differential diagnosis of transient neurological symptoms.
- ▶ The most valuable tool in identifying paroxysmal symptoms and placing them in an overall diagnostic context is the clinical history.
- ▶ The onset of paroxysmal demyelination should be considered a relapse relevant to a diagnosis of multiple sclerosis.
- ▶ Unlike many other manifestations of multiple sclerosis, paroxysmal symptoms are frequently abolished with medication.

Competing interests None.

Patient consent Obtained.

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