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Review

# Evaluation and management of longitudinally extensive transverse myelitis: a guide for radiologists

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Longitudinal extensive transverse myelitis (LETM) is defined as an intramedullary spinal cord T2 signal abnormality extending craniocaudally over at least three vertebral bodies on an MRI study. Timely and appropriate diagnosis greatly facilitates patient management. The radiologist should review the relevant clinical information and determine the patient demographics and acuity of symptoms. Herein, we review the spectrum of diseases causing LETM and propose interpretation to guide the radiologist when presented with the MRI finding of LETM.

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## Introduction

Longitudinally extensive transverse myelitis (LETM) is defined as intramedullary hyperintense T2-weighted signal abnormality that spans three or more vertebral segments.[1] LETM encompasses a broad list of differential diagnoses,[2] many of which have widely variable treatments and outcomes. The presence of LETM should prompt a comprehensive clinical assessment and paraclinical testing that includes not only imaging findings, but also timing of symptom onset, serum and cerebrospinal fluid (CSF) laboratory studies, and presence or absence of previous neurological symptoms (Fig 1). When a patient first presents with myelopathy, the incidence of longitudinally extensive signal abnormality on imaging is not clear and depends on the underlying aetiology and timing of the scan; however, it has been reported to be as high as 65% in non-multiple sclerosis (MS) or collagen vascular related transverse myelitis (TM).[3] In considering the onset of TM symptoms, with an acute presentation, compressive myelopathy must be excluded rapidly, as emergent surgical decompression is required. Demyelinating and inflammatory diseases can also present acutely. For example, TM typically presents with progressive acute symptoms such as neurogenic bladder and bowel

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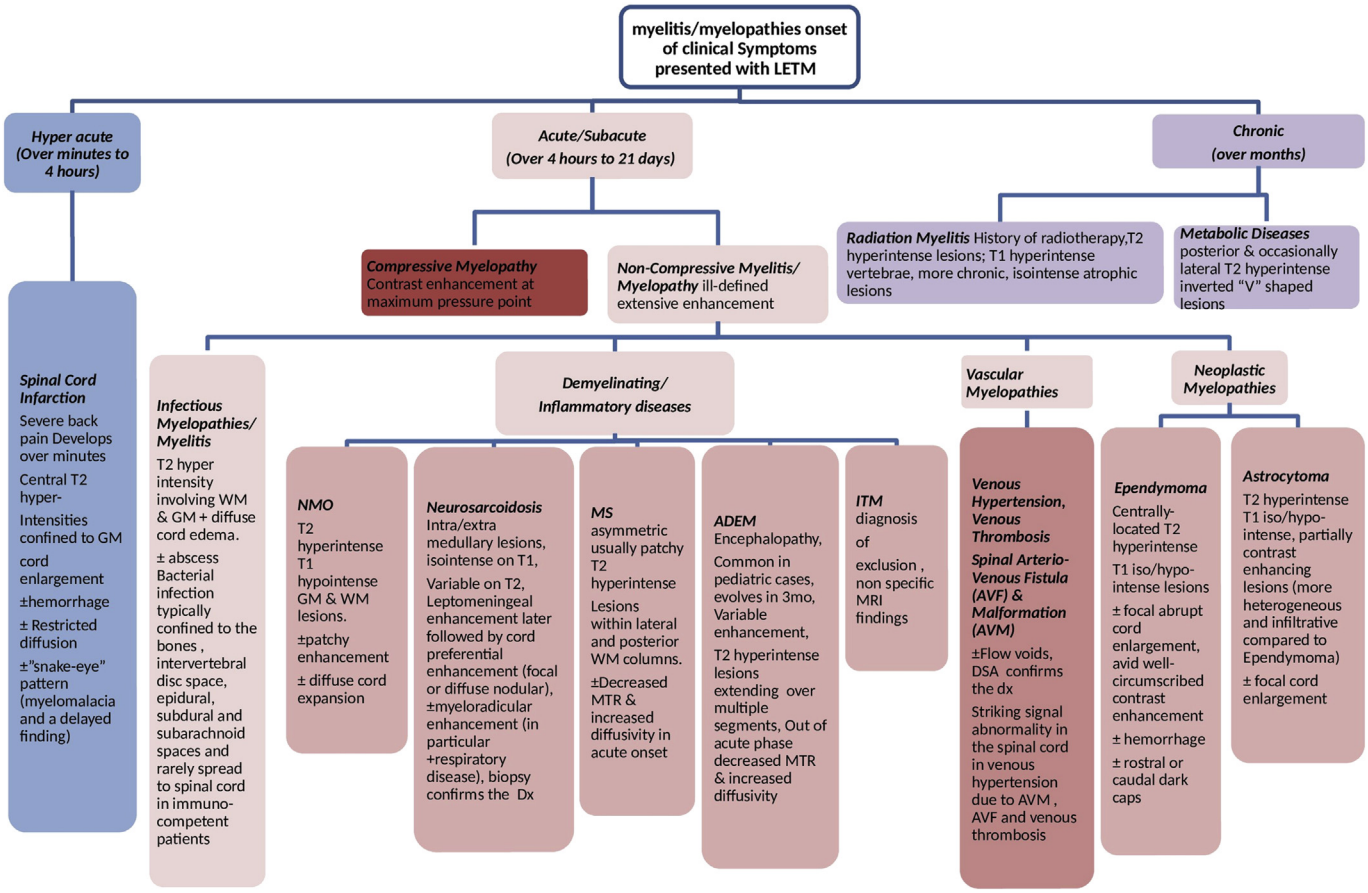


**Figure 1** ADEM, NMO, LETM, MS, MTR, idiopathic TM. In paediatric cases spinal cord infarction may have late presentation (12–24 hours to days). Although onset of clinical symptoms are considered as the main initial clue to narrow down the differential diagnosis of LETM, the time of imaging in the course of disease should also be taken into account as many of the above discussed diseases have different imaging manifestations at various time points. Neoplasm, especially low-grade tumours, of the cord may grow slow and present sometimes in months or years, therefore, this group could be included in the chronic myelopathy group as well.

dysfunction, sensory or motor deficits, or gait instability developing over hours to days. Less commonly, however, it may present subacutely, with symptoms developing over 1–2 weeks.[1,4] Although much less common than inflammatory TM, infectious myelitis is also an important diagnosis that must be excluded in the acute or subacute setting. Vascular myelopathies including spinal cord infarction or dural arteriovenous fistulas (AVFs) causing venous hypertension within the spinal cord can mimic TM with an acute onset of symptoms.

The radiologist should evaluate the appearance of the brain magnetic resonance imaging (MRI) in all cases of LETM after compressive myelopathy has been excluded, as it may be valuable in narrowing the differential diagnosis. LETM in patients with chronic symptoms is more challenging, where the presence of extensive signal abnormality may reflect an acute deterioration superimposed on chronic symptoms. Inflammatory and demyelinating diseases are the primary considerations in this setting. Idiopathic LETM accounts for 15–30.5% of all acute TM cases.[5,6]

Herein, we review the spectrum of diseases causing LETM and propose interpretation aids to guide the radiologist when presented with the spinal MRI finding of LETM. The review is organised with respect to clinical onset of myelopathy/myelitis (Fig 1).

## Hyperacute presentation of LETM

### *Arterial spinal cord infarction*

Arterial occlusion often presents with severe back pain and develops over minutes, leading to spinal cord infarction. The anterior spinal artery (ASA) is most commonly affected and classically produces the MRI appearance of T2 hyperintense "snake eyes" due to ischaemia of the ventral grey matter, with sparing of the dorsal horn. The posterior spinal arteries are paired; therefore, ischaemia/infarction is rarely seen in the posterolateral hemicord. Watershed ischaemia can also occur. Predisposing factors of spinal cord infarction are spinal and aortic surgery,[7] vertebral artery dissection,[8] hypotension,[9] vasculitis,[10] fibrocartilaginous embolism,[11] atherosclerosis, and others. The characteristic MRI presentation (T2 hyperintensities in anterior grey matter distribution referred to as a "snake eye" or "owl eye" pattern) is usually seen in the chronic stage; at the acute/

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