

REVIEW

Tuberculous Myelopathy Associated with Longitudinally Extensive Lesion: A Clinicoradiological Review of Reported Cases

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Acute transverse myelitis is an inflammatory disorder of the spinal cord in which there is no evidence of spinal cord compression. Longitudinally extensive transverse myelitis (LETM) is a specific subtype of acute transverse myelitis that usually affects three or more vertebral levels and produces marked neurological deficits. While the most-common cause of LETM is neuromyelitis optica or neuromyelitis optica spectrum disorder, there are rare cases of other causes mimicking this condition, including tuberculosis (TB). We sought to review the clinicoradiological features of TB myelopathy associated with longitudinally extensive lesion, which may mimic LETM, in the English literature. We searched the PubMed, Google Scholar, Web of Science, and Scopus databases for relevant articles using search terms including "longitudinally extensive transverse myelitis," "tuberculosis," "TB spinal cord," and various combinations of these expressions. Full-text papers were selected without limiting the publication year. We also examined the reference lists of key papers to identify further articles that are potentially relevant. We found 10 cases in 7 papers describing TB myelopathy associated with longitudinally extensive lesion. The demographics, clinical features, relevant cerebrospinal fluid findings, and radiological findings were compiled and summarized. TB myelopathy associated with longitudinally extensive lesion is very rare, with no documented prevalence. Early and accurate diagnosis is important since the condition is potentially treatable.

Key Words tuberculosis, longitudinally extensive transverse myelitis, TB myelopathy associated with longitudinally extensive lesion, myelitis, multiple sclerosis, neuromyelitis optica, neuromyelitis optica spectrum disorder.

INTRODUCTION

Tuberculosis (TB) is the leading cause of death due to a single infectious disease among adults worldwide.¹ The World Health Organization estimated that 10 million people would have TB in 2017, with 8.7 million of them living in 30 high-burden countries.² The clinical manifestation depends on the organ system affected. TB of the central nervous system (CNS) comprises 1% of all TB infections, with 95% of these in the form of TB meningitis^{3,4} and half of them involving the spine. TB is an airborne infectious disease caused by the *My*-*cobacterium tuberculosis* complex that was discovered in 1882 by Robert Koch.⁵ Being airborne, it primarily affects the pulmonary system; however, it also affects other systemic organs throughout the human body. The clinical manifestation is dynamic, being asymptomatic in some cases but life-threatening in others.^{6,7} The incidence of TB is much higher in patients with human immunodeficiency virus (HIV), being in the range of 10–20%.^{8,9} A TB infection can affect the CNS in various ways. The manifestations of CNS TB include meningitis (in approximately 95% of cases), tuberculoma, abscesses, pachymeningitis, calvarial TB, and

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ReceivedOctober 2, 2019RevisedDecember 18, 2019AcceptedDecember 20, 2019

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spinal arachnoiditis.¹⁰⁻¹² TB involvement of the spinal cord is usually due to hematogenous spread;¹³ however, spinal cord involvement may also be secondary to compression via vertebral TB.¹⁴

TB transverse myelitis is very rare, which makes TB myelopathy associated with a longitudinally extensive lesion a much rarer clinical entity.15 Transverse myelitis describes inflammatory spinal cord lesions that usually (but not always) span up to two vertebral levels, with various causes.¹⁶ Longitudinally extensive transverse myelitis (LETM) is a subtype of acute transverse myelitis in which the spinal cord lesion spans three or more vertebral levels, usually with much more severe neurological symptoms,¹⁷ most commonly secondary to neuromyelitis optica (NMO) or neuromyelitis optica spectrum disorder (NMOSD).18 Other causes that need to be considered include multiple sclerosis (MS), acute disseminated encephalomyelitis, systemic lupus erythematosus, sarcoidosis, Sjogren's syndrome, vascular diseases (which may be due to spinal cord infarction, spinal cord arteriovenous shunts, or fibrocartilaginous embolism), neoplasms, trauma, nutritional deficiencies, and infections. TB is an important etiology in countries in which TB is endemic.19-22

In the present study we sought to determine the clinical presentation, laboratory, and radiological findings of TB myelopathy associated with longitudinally extensive lesion reported in the English literature.

METHODS

In order to identify relevant studies, we searched the PubMed, Google Scholar, Web of Science, and Scopus databases using search terms including "longitudinally extensive myelitis," "longitudinally extensive transverse myelitis," "longitudinal extensive transverse myelitis," "tuberculous," "tuberculosis," "TB," "myelitis," "TB spine," "TB spinal cord," "spinal TB," "TB myelitis," "TB LETM," and combinations of these terms. Full-text papers in the English language were selected without limiting the publication year. Articles not published in English, and those labeled "CNS tuberculosis" were excluded from the scope of this review. We also examined the reference lists of key papers to identify further articles that are potentially relevant for inclusion in this review.

RESULTS

After performing an extensive literature search and excluding irrelevant articles, we found 10 cases of TB myelopathy associated with longitudinally extensive lesion that were reported in 7 papers. All of the present authors read the articles and extracted the demographics, clinical presentation,

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relevant cerebrospinal fluid (CSF)/biochemical findings, and radiological findings. Six of the 10 cases had positive CSF findings confirming TB, while the CSF findings were negative in the remaining 4 cases, and so TB was diagnosed based on the involvement of other organs (brain in 1 case and lung in the other 3), with accompanying negative CSF findings for NMO/ NMOSD and/or MS. These cases are discussed briefly below and summarized in the Tables.

REVIEW OF REPORTED CASES

Demographics and clinical presentation

The 10 reported cases involved 5 males, 4 females, and 1 transgender patient. The age of the patients ranged from 17 to 53 years (mean age 33.8 years). The most-common clinical presentations were weakness of upper limbs, lower limbs, or both upper and lower limbs, as well as urinary retention. Other clinical manifestations included sensory changes (numbness and paresthesia), altered sensorium, gait ataxia, head-ache, fever, recurrent vomiting, blurring of vision, and anorexia. The reported duration of symptoms prior to the presentation ranged from 1 day to 2 months. While a proper neurological assessment could be performed on presentation in most of the patients, two of them had altered sensorium that prevented full assessments.

CSF findings

The polymerase chain reaction (PCR) for TB in the CSF produced positive results in six patients and negative ones in the other four. Three patients tested negative for HIV, one tested positive, while the HIV status was not documented in the remaining six patients. All of the patients had elevated CSF protein levels (40-440 mg/dL, normal range 20-40 mg/ dL), while their glucose levels were either normal or low (27-59 mg/dL, normal range 45-80 mg/dL). All of the six patients who were tested for aquaporin-4 antibodies (AQP4-IgG) were negative, as were the four patients tested for oligoclonal bands (OCBs). The statuses of the remaining patients for both AQP4 Ab and OCBs-to suggest the possibilities of NMO/ NMOSD and MS, respectively-were not documented. Pleocytosis was present in seven patients (predominantly lymphocytic), two patients had no CSF pleocytosis, and the status of the remaining patient was not documented.

Radiological findings

Whole-spine magnetic resonance imaging (MRI) had been applied to all of the reported cases. These had revealed longitudinally extensive intramedullary lesions that appeared iso- or hypointense on T1-weighted images and hyperintense on T2-weighted/fluid-attenuated inversion recovery

Reference/age, sex	Clinical findings	CSF/biochemical investigations	CSF/biochemical findings	MRI findings
Trebst et al. ¹⁸ / 53 years, female	Severe gait ataxia, nuchal neuropathic pain, fever, altered consciousness	Pleocytosis Protein Lactate Glucose PCR for TB in CSF ELISA for HIV	Present Elevated Elevated Reduced Positive Positive	T2-weighted hyperintensity from C2 to T2 vertebrae
Jain et al. ⁴⁴ / 18 years, male	Fever, headache, recurrent vomiting, confusion for 2 months, weakness of lower limbs, urinary incontinence	ELISA for HIV Pleocytosis Protein Glucose CSF AFB stain Skin tuberculin sensitivity test (pre-existing brain tuberculomas) AQP4-IgG OCBs	Negative Present Elevated Normal Negative Positive Negative Negative	T2 to T10 vertebrae with intramedullary cord lesion, T1-weighted isointense and T2-weighted hyperintense, no cord expansion or enhancement
Alkan et al.⁴/ 17 years, female	Bilateral blurring of vision, neck pain, headache	CSF pressure Pleocytosis Protein Glucose AQP4-IgG OCBs ELISA for HIV Tuberculin skin test Biopsy of the calcified right paratracheal lymph node found on chest CT	Normal pressure Present Elevated Normal Negative Negative Negative Positive (23 mm) Positive for necrotizing granulomatous inflammation and AFB, consistent with TB	T1-weighted isointense, T2- weighted hyperintense intramedullary lesion spanning from brainstem to full lumbar spine
Zhang et al. ⁴³ / 21 years, female	Bilateral lower limb numbness, loss of pain and touch below T4 level, hyperreflexia, urinary retention, up-going Babinski's sign bilaterally	Protein Pleocytosis AQP4-IgG MTB in CSF However, this patient had history of untreated PTB for 3 years, with chest CT showing a left upper lobe cavitating lesion	Elevated Absent Negative Negative	C3 to T11 T1-weighted hypointense, T2-weighted/ FLAIR hyperintense intramedullary lesion
Kasundra et al. ⁴² / 17 years, male	Low-grade fever, anorexia, acute sensorimotor paraparesis, absent tendon reflexes, anesthesia below T9 level	CSF pressure Protein Glucose PCR for TB in CSF ELISA for HIV AQP4-IgG	Normal Elevated Reduced Positive Negative Negative	T1-weighted isointense, T2- weighted hyperintense intramedullary lesion from T7 to conus, with no cord expansion. Cord surface enhancement
Coclitu et al. ⁴¹ / 52 years, male	Ascending bilateral lower limb paresthesia, urinary retention, muscle weakness, fever, brisk deep tendon reflexes, impaired sensation below T8 level, urinary retention	Pleocytosis CSF pressure Protein AQP4-IgG OCBs On chest CT, a lung nodule was found in the right lower lobe and biopsied	Absent Normal Elevated Negative Epithelioid granuloma and central caseous necrosis, confirming TB	T1-weighted isointense, T2- weighted hyperintense intramedullary lesion from C2 to T2. No gadolinium enhancement

 Table 1. Summary of the demographics, clinical, relevant CSF/biochemical, and MRI findings for the 10 reported cases

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Reference/age, sex	Clinical findings	CSF/biochemical investigations	CSF/biochemical findings	MRI findings
Sahu et al.³/	Sudden-onset lower	Pleocytosis	Present	Diffuse, long-segment
patient 1:35 years,	limb weakness,	Protein	Elevated	intramedullary lesion from
transgender	urinary retention,	Glucose	Normal	C7 to conus; T2-weighted
	abdominal reflexes absent below T10, loss of pain and touch below T10, bilateral plantar reflexes nonresponsive	PCR for TB in CSF	Positive	hyperintense. No cord expansion
Sahu et al. ³ /	Acute-onset lower	Pleocytosis	Present	T1-weighted hypointense,
patient 2: 40 years,	limb weakness,	Protein	Elevated	T2-weighted hyperintense
female	urinary retention,	Glucose	Normal	intramedullary lesion from
	loss of pain, touch, and temperature below L1 level	PCR for TB in CSF	Positive	T1 to conus
Sahu et al.³/	Low-grade fever,	Pleocytosis	Present	T1-weighted hypointense,
patient 3: 40 years,	acute-onset	Protein	Elevated	T2-weighted hyperintense
male	paraparesis with	Glucose	Normal	intramedullary lesion from
	urinary retention loss of power of both lower limbs (0/5 bilaterally)	PCR for TB in CSF	Positive	T6 to T10
Sahu et al.³/	Fever, urinary	CSF pressure	High	T2- to T9-level T2-weighted
patient 4: 45 years,	retention, unable	Pleocytosis	Present	hyperintense, ill-defined
male	to walk for 3 days,	Protein	Elevated	intramedullary lesion
	confusion	Glucose	Normal	
		PCR for TB in CSF	Positive	
		AQP4-IgG	Negative	
		OCBs	Negative	

 Table 1. Summary of the demographics, clinical, relevant CSF/biochemical, and MRI findings for the 10 reported cases (continued)

AFB: acid-fast bacilli, AQP4-IgG: aquaporin-4 antibodies, CSF: cerebrospinal fluid, FLAIR: fluid-attenuated inversion recovery, HIV: human immunodeficiency virus, MRI: magnetic resonance imaging, MTB: mycobacterium TB, OCBs: oligoclonal bands, PCR: polymerase chain reaction, TB: tuberculosis.

images. These mostly involved the cervicothoracic vertebrae spinal cord levels, with no cases of cord compression. Two patients had lesions which extended from the C7/T1 level to the conus, while one patient had full involvement from the level of the brainstem to the lumbar spine. No cord expansion was documented, but postgadolinium enhancement was evident in two patients, with no enhancement in the others.

The specific levels in each case are summarized in Table 1, along with the demographics, clinical findings, and relevant laboratory findings (Tables 1 and 2).

DISCUSSION

The clinical manifestations of patients with myelitis can be dramatic, including paraparesis/tetraparesis, altered gait, and bladder, bowel, and/or sexual dysfunction, as well as sensory

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impairment.²³ These dramatic losses of bodily function make early and accurate diagnosis vital. In 2002 the Transverse Myelitis Consortium Working Group introduced spinal MRI and CSF protein analysis as methods for identifying spinal cord inflammation into their diagnostic criteria (in addition to the clinical manifestation) to aid early and accurate diagnostic confirmation.²⁴ The present review has revealed that the clinical manifestations of TB myelopathy associated with longitudinally extensive lesion are mostly weaknesses affecting the upper limbs, lower limbs, or both upper and lower limbs, as well as urinary retention. Other clinical symptoms include altered sensorium, gait ataxia, headache, fever, recurrent vomiting, blurring of vision, and anorexia. The duration of symptoms in the reported cases included in our review ranged from 1 day to 2 months.

MRI of the spine is an important diagnostic tool in diag-

CSE findings	Results			
CSF findings	Present/positive/elevated	Absent/negative/reduced	Not documented	
Pleocytosis	70 (7)	20 (2)	10 (1)	
Protein	100 (10)	-	-	
Glucose	Normal in 60 (6)	Reduced in 20 (2)	20 (2)	
PCR for TB / AFB stain	60 (6)	40 (4)	-	
AQP4-IgG	-	60 (6)	40 (4)	
OCBs	-	40 (4)	60 (6)	
ELISA for HIV	10 (1)	30 (3)	60 (6)	
Relevant biochemical/laboratory and other				
imaging findings (for the 4 patients with	Present/positive/elevated	Absent/negative/reduced	Not documented	
negative CSF TB findings)				
Tuberculin skin test	20 (2)	-	-	
Biopsy	Biopsy Positive in 2 patients (1 paratracheal lymph node, 1 right lung nodule)		-	
Chest CT	2 patients with positive chest CT findings (1 right-lung nodule, 1 left upper lobe cavitating lesion)	-	-	

Table 2. Detailed CSF and relevant biochemical/laboratory and other imaging findings pertinent to the 10 reported cases

Variables are presented as % (n).

AFB: acid-fast bacilli, AQP4-IgG: aquaporin-4 antibodies, CSF: cerebrospinal fluid, HIV: human immunodeficiency virus, MRI: magnetic resonance imaging, OCBs: oligoclonal bands, PCR: polymerase chain reaction, TB: tuberculosis.

nosing TB myelopathy associated with longitudinally extensive lesion.²⁵⁻²⁷ Gupta et al.²⁸ found intramedullary involvement in 16 of 20 patients with intraspinal TB. This involvement appeared hypo- or isointense on T1-weighted images and hyperintense on T2-weighted images. None of the patients in their cohort showed cord swelling, or enhancement after administering gadolinium. It was not specified whether the patients had myelopathy associated with longitudinally extensive lesion; however, radiological evidence of myelitis was present. Wasay et al.29 found that involvement at the cervicothoracic level was the most common (affecting approximately 90% of patients), with hyperintensity on T2-weighted images being the most-consistent finding. They also identified two radiological features that were associated with a poor outcome: cord atrophy or cavitation, and the presence of a syrinx pattern on MRI. All patients had abnormal intramedullary signal intensities over long segments (covering at least three vertebral levels), which appeared iso- or hypointense on T1-weighted images and hyperintense on T2-weighted images. Cord enhancement after administering gadolinium was present in only 2 of the 10 patients, while there was no evidence of cord expansion or compression in any of the patients.

In addition to imaging, CSF studies play an important role in the diagnosis. In cases of TB myelopathy associated with longitudinally extensive lesion, a CSF analysis shows pleocytosis and increased protein levels, with normal glucose and opening pressures.³⁰⁻³² Another study found a low CSF glucose level in 60% of patients.²⁹ The reported percentage of normal CSF findings has ranged from 6% to 45%.31,32 All of the patients in the present cohort had an elevated CSF protein level (40-440 mg/dL, normal range 20-40 mg/dL), while their glucose levels were either normal or low (27-59 mg/ dL, normal range 45-80 mg/dL). Lymphocytic pleocytosis was evident in seven patients, while the PCR for TB in the CSF produced positive results in six patients. Tests for AQP4-IgG and OCBs (via cell-based assays) were all negative. While some studies have suggested an association between pulmonary TB and NMO/NMOSD, which suggests demyelination by NMO/NMOSD as the cause of the spinal cord findings,³³⁻⁴⁰ the present review found that only six patients had been tested for AQP4-IgG, all of whom turned out to be negative. However, it is difficult to confirm the absence of an association, since their clinical and radiological features were not fully described. More studies are therefore needed to confirm the absence or presence of an association between TB and NMO/NMOSD.

There is no specific guideline at present for the treatment regime to apply to patients with TB myelopathy associated with longitudinally extensive lesion. Previous reports have considered a combination of steroid and antitubercular therapy to be appropriate.^{3,4,18,41-44} Antiretroviral therapy may be added to the treatment regime for patients with HIV. All of the reported patients analyzed in this review received a combination of steroid and antitubercular therapy, with follow-

up clinical and radiological improvements evident in most of the patients. The average duration from the initiation of therapy to clinical improvement was 5-12 weeks in nine patients, while the tenth patient showed rapid deterioration despite receiving therapy.

CONCLUSION

TB myelopathy associated with longitudinally extensive lesion is a very rare clinical entity, with no documented prevalence. The extensive involvement of the spinal cord can results in a dramatic clinical manifestation, causing a significant burden on the patient and in some cases even a rapid deterioration in clinical symptoms. Appropriate knowledge about the clinical presentation and radiological and CSF findings is vital to ensuring an early and accurate diagnosis and hence the instigation of appropriate therapy.

Author Contributions

ICN

Conceptualization: Mohamad Syafeeq Faeez Md Noh. Data curation: Mohamad Syafeeq Faeez Md Noh, Anna Misyail Abdul Rashid. Formal analysis: Mohamad Syafeeq Faeez Md Noh, Norafida Bahari. Funding acquisition: Mohamad Syafeeq Faeez Md Noh. Investigation: all authors. Methodology: all authors. Project administration: Mohamad Syafeeq Faeez Md Noh, Norafida Bahari. Resources: Mohamad Syafeeq Faeez Md Noh, Anna Misyail Abdul Rashid. Software: Mohamad Syafeeq Faeez Md Noh. Supervision: Norafida Bahari. Validation: all authors. Visualization: all authors. Writing—original draft: Mohamad Syafeeq Faeez Md Noh. Writing—review & editing: all authors.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Acknowledgements .

None.

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