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Original Article

Movement Disorders in Central Nervous System Tuberculosis: A Systematic Review

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ABSTRACT: ***Background:*** Tuberculosis (TB) affecting the central nervous system (CNS) can lead to a broad range of movement disorders, which are frequently overlooked in clinical settings. This review explores the various presentations, underlying mechanisms and patient outcomes related to these disorders. ***Methods:*** We systematically reviewed published case reports, series and cohort studies that described patients with CNS TB who developed movement disorders. Extracted data included patient characteristics, type of CNS TB, imaging and CSF findings, types of movement disorders, treatments used and outcomes. ***Results:*** A total of 61 patients with CNS TB and associated movement disorders were analyzed. The most common manifestations were ataxia, dystonia, chorea or hemiballismus and parkinsonism. Less frequent symptoms included opsoclonus-myoclonus, segmental myoclonus, tremors, cervical dystonia and stereotypy. TB meningitis was the predominant form, often accompanied by infarcts, hydrocephalus or tuberculomas. Proposed causes included vascular injury, inflammatory lesions, immune mechanisms and drug-related effects. All patients received anti-tuberculosis treatment, and nearly half required corticosteroids or surgical procedures. About 25 patients (41%) fully recovered, 17 (27.9%) had significant improvement, 13 (21.3%) showed partial improvement, 11 (18.0%) had ongoing problems and 1 (1.6%) died. Cohort studies also reveal that movement disorders – particularly tremors, dystonia, parkinsonism and ataxia – are frequent but underrecognized in CNS TB. These typically emerge early, often within three weeks, and are linked to lesions in the basal ganglia or thalamus. ***Conclusions:*** Movement disorders in CNS TB are more common than generally recognized. Prompt diagnosis through imaging and timely therapy can greatly improve neurological outcomes.

RÉSUMÉ: **Troubles du mouvement dans la tuberculose du système nerveux central : résultats d'une revue systématique.** ***Contexte :*** La tuberculose (TB) du système nerveux central (SNC) peut se manifester par un large éventail de troubles du mouvement qui, bien souvent, passent inaperçus en clinique. La revue visait à relever les différentes manifestations de la maladie, les mécanismes sous-jacents et l'état de santé des patients en lien avec ces troubles. ***Méthode :*** L'équipe a procédé à un examen systématique d'exposés de cas, de séries et d'études de cohortes publiés, portant sur des patients atteints de la TB du SNC qui présentaient des troubles du mouvement. Les données extraites comprenaient les caractéristiques des patients, le type de TB du SNC, les résultats des examens par imagerie et des analyses du liquide céphalorachidien, les types de troubles du mouvement, les traitements et les résultats obtenus. ***Résultats :*** Au total, les dossiers de 61 patients atteints de TB du SNC qui présentaient des troubles du mouvement associés à la maladie ont fait l'objet d'analyse. Les manifestations les plus fréquentes étaient l'ataxie, la dystonie, la chorée ou l'hémiballisme et le syndrome parkinsonien. Des symptômes moins fréquents comprenaient l'opsoclonie-myoclonie, la myoclonie segmentaire, les tremblements, la dystonie cervicale et la stéréotypie. La forme la plus courante était la méningite tuberculeuse, qui s'accompagnait souvent d'infarctus, d'hydrocéphalie ou de tuberculomes. Parmi les causes envisagées, il y avait les lésions vasculaires, les lésions inflammatoires, les mécanismes immunitaires et les effets de certains médicaments. Les patients ont tous été soumis à un traitement antituberculeux, et presque la moitié d'entre eux ont dû recevoir des corticostéroïdes ou être opérés. Environ 25 patients (41 %) ont connu un rétablissement complet; 17 (27, 9 %), une amélioration importante de leur état et 13 (21,3 %), une amélioration partielle; 11 (18,0 %) ont fait face à problèmes persistants, et 1 (1,6 %) est mort. En outre, d'après les études de cohorte, les troubles du mouvement, notamment les tremblements, la dystonie, le syndrome parkinsonien et l'ataxie, sont fréquents, mais ils passent inaperçus dans la TB du SNC. Ces troubles apparaissent généralement tôt, souvent au cours des trois premières semaines de la maladie, et ils sont liés à des lésions des noyaux gris centraux ou du thalamus. ***Conclusion :*** Les troubles du mouvement dans la TB du SNC sont plus fréquents que ne le laisse croire, en général, leur reconnaissance. Ainsi, la pose rapide d'un diagnostic par l'imagerie et la mise en route d'un traitement en temps opportun peuvent améliorer grandement les résultats neurologiques.

Keywords: Dystonia; chorea; myoclonus; tuberculous meningitis; tuberculoma

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Highlights

- Movement disorders are frequent but underrecognized in central nervous system tuberculosis.
- Common types include ataxia, dystonia, chorea and parkinsonism.
- Basal ganglia or thalamic lesions are commonly linked to these disorders.

Introduction

Central nervous system (CNS) tuberculosis (TB) is the most devastating form of extrapulmonary tuberculosis, accounting for approximately 1% of all TB cases and up to 10% of extrapulmonary cases. It remains a significant cause of neurological morbidity and mortality, particularly in endemic regions and among immunocompromised individuals.[1,2] CNS TB presents primarily as tuberculous meningitis, intracranial tuberculomas, spinal arachnoiditis or a combination thereof. The pathological processes involved – granulomatous inflammation, vasculitis, infarction, hydrocephalus and immune-mediated injury – can result in a variety of neurological syndromes.[1,2]

Several case reports and case series have documented a diverse array of movement disorders in patients with CNS TB, particularly tuberculous meningitis, highlighting its complex and often underrecognized neurological manifestations.[3–5]

Although individual case reports and small series have described diverse movement disorders in CNS TB, the evidence remains fragmented and inconsistent. Cohort studies are limited, and outcome predictors such as lesion location, pathophysiological mechanisms and treatment response have not been systematically synthesized. No prior comprehensive review has pooled available cases and compared them with cohort findings. A systematic review at this time is needed to consolidate dispersed evidence, clarify clinical patterns and identify prognostic factors, thereby addressing a critical gap in the literature and guiding both diagnosis and management.

Methods

This systematic review followed the PRISMA 2020 guidelines for reporting. The review protocol was prospectively registered in the PROSPERO database with the registration number CRD420251075389.[6]

Search strategy

A thorough and systematic literature search was conducted across four major databases: PubMed, Embase, Scopus and Google Scholar, to identify all relevant studies reporting movement disorders in patients with CNS TB. We limited Google Scholar screening to the first 50 pages, as results on earlier pages are more relevant, while later ones are largely low-yield or duplicate. The final search was completed on May 31, 2025. Detailed search strategy has been provided in Table 1.

Non-English studies were translated using Google Translate. Critical medical terminology, including diagnoses and outcome measures, was cross-verified against bilingual medical dictionaries, if necessary.

Definitions

CNS tuberculosis was defined as microbiologically, radiologically or histologically confirmed tuberculous infection of the brain or spinal cord, including manifestations such as tuberculous meningitis, intracranial or spinal tuberculomas, abscesses and spinal TB with CNS involvement[7,8] (Figure 1).

Movement disorders were defined clinically and classified into hyperkinetic (e.g., chorea, dystonia, myoclonus, tremor) and hypokinetic (e.g., parkinsonism, rigidity) types. Cases with insufficient neurological detail to confirm movement disorders were excluded.[9]

Eligibility criteria

We included case reports, case series and cohort studies that provided a confirmed diagnosis of CNS TB, documented the presence of one or more clinically observed movement disorders and offered sufficient clinical, radiological or treatment-related details for meaningful analysis. Studies were excluded if they involved non-human subjects, lacked adequate clinical descriptions or were purely radiological or experimental without clinical correlation.

Study selection

Two reviewers (RKG and SS) independently assessed all titles and abstracts for eligibility. Full texts of potentially relevant articles were retrieved and reviewed. Any disagreements were resolved through discussion or, when necessary, consultation with a third reviewer (SP).

EndNote 21 (Clarivate Analytics, Philadelphia, USA) was used for reference management and to remove duplicates. Duplicate verification was independently performed by HSM, with final adjudication by SS. The selection process was documented in a PRISMA flow diagram.

Data extraction

Data were extracted using a predefined structured format by three reviewers (SP, SS and RAM). The following data were extracted from each included study: patient demographics (including age and sex), subtype of CNS TB (such as tuberculous meningitis or tuberculoma), type of movement disorder observed, time to onset of movement-related symptoms, findings from neuroimaging and CSF analysis and treatment approaches including antitubercular therapy, corticosteroids, symptomatic medications and surgical interventions, as well as clinical outcomes encompassing recovery status, persistence of symptoms, complications or death. All discrepancies were resolved through consensus in consultation with a senior reviewer (RKG). Data were compiled in Microsoft Word tables and verified for accuracy before analysis.

Quality assessment

Case reports and case series were evaluated using the criteria proposed by Murad et al., which assess four key domains: selection, ascertainment, causality and reporting. Records meeting all four domains were rated as "good," those fulfilling three as "fair" and those satisfying only one or two as "poor." Cohort studies were appraised using the Newcastle–Ottawa Scale, focusing on three domains: selection, comparability and outcome assessment. Disagreements in scoring were resolved by consensus among the reviewers.

Data synthesis

A descriptive synthesis of data was performed. Categorical variables were reported as frequencies and percentages. Results were grouped by type of movement disorder, type of TB, underlying mechanism (vascular, immune-mediated, compressive), diagnostic modalities,

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Table 1. Search strategy

PubMed
("Tuberculosis, Central Nervous System"[Mesh] OR "Tuberculosis, Meningeal"[Mesh] OR "Tuberculoma"[Mesh] OR "Spinal Tuberculosis"[Mesh]
OR "CNS tuberculosis"[tiab] OR neurotuberculosis[tiab] OR "tuberculous meningitis"[tiab] OR tuberculoma[tiab] OR "spinal tuberculosis"[tiab])
AND
("Movement Disorders"[Mesh] OR "Parkinsonian Disorders"[Mesh] OR "Chorea"[Mesh] OR "Dystonia"[Mesh] OR "Myoclonus"[Mesh] OR "Tremor"[Mesh] OR "Ataxia"[Mesh]
OR "movement disorder*"[tiab] OR parkinsonism[tiab] OR chorea[tiab] OR dystonia[tiab] OR myoclonus[tiab] OR tremor[tiab] OR rigidity[tiab] OR ataxia[tiab])

Embase
('central nervous system tuberculosis'/exp OR 'tuberculous meningitis'/exp OR 'tuberculoma'/exp OR 'spinal tuberculosis'/exp
OR 'CNS tuberculosis':ab,ti OR neurotuberculosis:ab,ti OR 'tuberculous meningitis':ab,ti OR tuberculoma:ab,ti OR 'spinal tuberculosis':ab,ti)
AND
('movement disorder'/exp OR 'parkinsonism'/exp OR 'chorea'/exp OR 'dystonia'/exp OR 'myoclonus'/exp OR 'tremor'/exp OR 'ataxia'/exp
OR 'movement disorder*':ab,ti OR parkinsonism:ab,ti OR chorea:ab,ti OR dystonia:ab,ti OR myoclonus:ab,ti OR tremor:ab,ti OR rigidity:ab,ti OR ataxia:ab,ti)

Scopus
(TITLE-ABS-KEY("CNS tuberculosis" OR neurotuberculosis OR "tuberculous meningitis" OR tuberculoma OR "spinal tuberculosis"))
AND
(TITLE-ABS-KEY("movement disorder*" OR parkinsonism OR chorea OR dystonia OR myoclonus OR tremor OR rigidity OR ataxia))

Google Scholar
("CNS tuberculosis" OR neurotuberculosis OR "tuberculous meningitis" OR tuberculoma OR "spinal tuberculosis")
AND
("movement disorder" OR parkinsonism OR chorea OR dystonia OR myoclonus OR tremor OR rigidity OR ataxia)

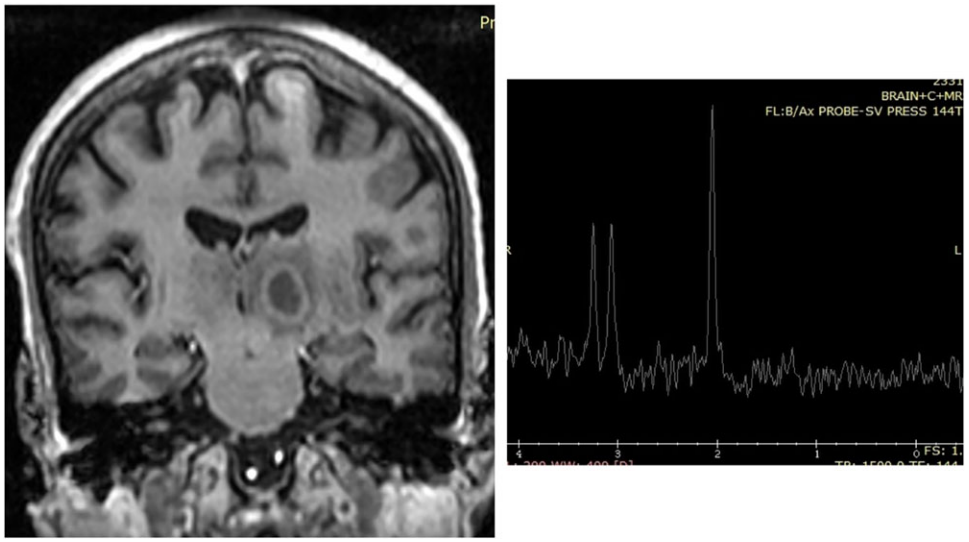


Figure 1. MRI of the brain showing a ring-enhancing lesion in the left thalamus with surrounding edema on post-contrast T1-weighted coronal image (left). Magnetic resonance spectroscopy from the lesion (right) demonstrates a lipid-lactate peak, reduced N-acetylaspartate and elevated choline, suggestive of a tuberculoma. The patient had right hemichorea.

treatments given and clinical outcomes. Structured tables and illustrative figures were developed to present a comprehensive summary of findings and clinical patterns in CNS TB-associated movement disorders. Missing data were extracted as "not reported," and no imputation was performed. Study authors were not contacted for additional clarification. Only descriptive synthesis was undertaken, as the available evidence consisted solely of case reports, case series and a few heterogeneous cohorts, precluding a meaningful meta-analysis.

Results

This review includes 61 patients with CNS TB who developed movement disorders, with data derived from 56 published reports.[12–67] All included cases were assessed for quality, with each rated as either good or fair (Supplementary Item 1). The PRISMA checklist is available in Supplementary Item 2. Detailed patient-level data are summarized in the tables. Figure 2 presents the PRISMA flow diagram outlining the case selection process. Of 4310 records identified, 3514 were screened after removing 796 duplicates, 158 full texts were retrieved, 131 assessed for eligibility and finally, 60 reports (including 4 cohort studies) met inclusion criteria.

The mean age was 26.9 years, with a median of 24 years and a nearly equal male-to-female distribution. The majority of patients were immunocompetent, and most cases were reported from Asia, predominantly India (Table S1 and Table 2). Tuberculous meningitis was the most frequent type of CNS TB, accounting for nearly two-thirds of the cases, often associated with infarcts, hydrocephalus or tuberculomas. Intracranial tuberculomas without meningitis accounted for about one-fifth, and spinal TB, miliary TB and other forms such as cerebritis and demyelination were less frequent. Neuroimaging showed tuberculomas in about half of the patients, hydrocephalus in 44% and infarcts in 34%, particularly involving the thalamus, basal ganglia and brainstem. Leptomeningeal enhancement, basal exudates and arachnoiditis were also commonly noted. CSF findings typically included

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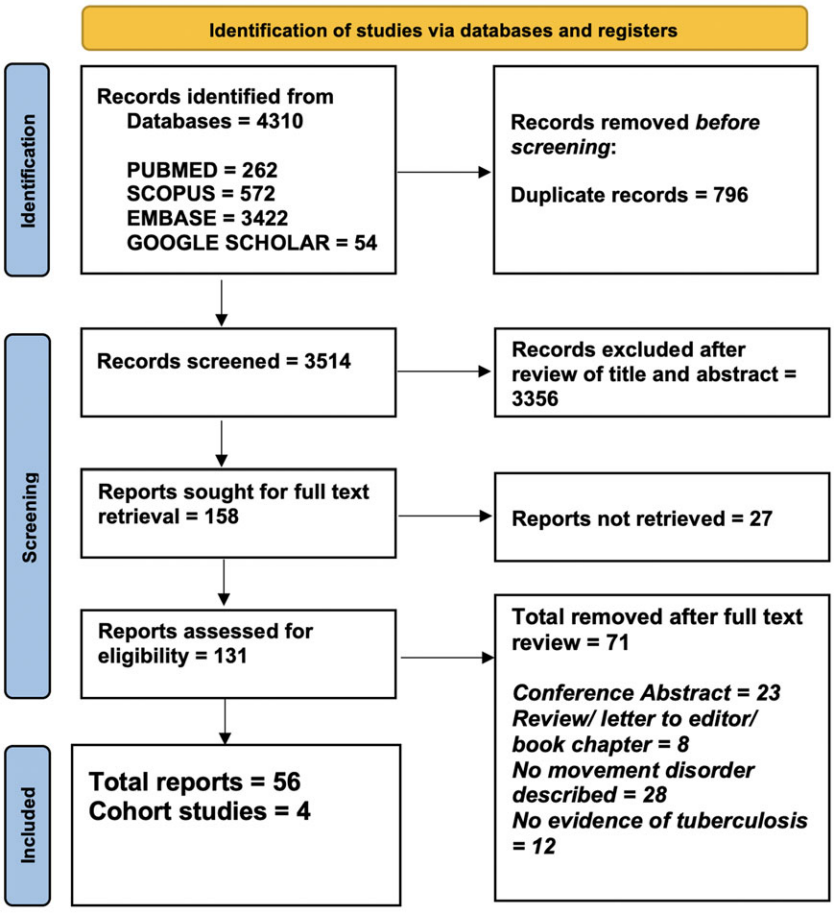


Figure 2. PRISMA 2020 flow diagram illustrating the study selection process for inclusion in the systematic review. The diagram shows the number of records identified, screened, excluded and included at each stage of the review, along with reasons for exclusions.

lymphocytic pleocytosis with elevated protein and low glucose. Microbiological or histopathological confirmation of tuberculosis was obtained in about two-thirds of the cases, most commonly via CSF GeneXpert, culture or biopsy (Table S1 and Table 3).

The movement disorders observed were diverse and reflected the underlying pathology and lesion location. Ataxia was the most common manifestation, followed closely by dystonia, chorea or hemiballismus and parkinsonism. Less commonly reported were opsoclonus-myoclonus, segmental myoclonus, postural and resting tremors, torticollis, cervical dystonia, blepharospasm, orofacial dyskinesia, stereotypy, trismus and bobble-head doll syndrome. The onset of movement disorders was subacute in most cases, though some occurred acutely or following initiation of anti-tuberculosis therapy, and a few developed after clinical recovery or months to years later. Associated neurological features included motor weakness, cranial nerve palsies, altered consciousness, raised intracranial pressure, autonomic instability, seizures and behavioral or cognitive abnormalities. Anti-tuberculosis therapy was administered in nearly all patients, often with adjunctive corticosteroids. Symptomatic treatment for movement disorders, including agents such as levodopa, trihexyphenidyl, clonazepam, baclofen and midazolam, was provided in nearly half the cases. Neurosurgical interventions, including ventriculoperitoneal shunting, surgical decompression or lesion excision, were performed when indicated.

Among the patients, 25 (41%) achieved full recovery, while 17 (27.9%) showed marked or significant improvement. Partial improvement was observed in 13 (21.3%) cases, and 11 (18.0%) continued to have persistent or serious problems. One patient (1.6%) died (Table S1 and Table 3).

Table 2. Summary of demographic characteristics of CNS tuberculosis cases presenting with movement disorders ($n = 61$)

Variables	Values
Age (in years)	NA = 1 Mean = 26.9 Median = 24 Mode = 4 Range = 1–78 IQR = 27.5
Sex	Female: 30 (49.2%) Male: 31 (50.8%)
Country-wise distribution of published reports ($n = 56$)	Asia: 39 (69.6%) *India = 24 (42.9%)* Africa: 4 (7.1%) Europe: 7 (12.5%) North America: 5 (8.9%) South America: 1 (1.8%)
Immune status	Immunocompetent: 53 (86.9%) HIV-positive: 3 (4.9%) Not explicitly stated: 5 (8.2%)

CNS = central nervous system; HIV = human immunodeficiency virus.

The proposed etiologies of the movement disorders included infarcts and vasculitis affecting the basal ganglia, thalamus or

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Table 3. Summary of clinical, neuroimaging and treatment features of CNS TB cases presenting with movement disorders ($n = 61$)

Variables	Values
Type of CNS TB	Tuberculous meningitis (including infarcts, hydrocephalus, tuberculomas or complications): 39 (63.9%) Intracranial tuberculomas (isolated/multiple): 13 (21.3%) Spinal tuberculosis: 3 (4.9%) Miliary tuberculosis: 2 (3.3%) Other forms (cerebritis, demyelination, probable CNS TB): 4 (6.6%)
Neuroimaging	Hydrocephalus: 27 (44.3%) Infarcts: 21 (34.4%) Tuberculomas (ring-enhancing or nodular lesions): 31 (50.8%) Leptomeningeal enhancement/basal exudates/arachnoiditis: 17 (27.9%) Spinal involvement: 6 (9.8%) Normal brain imaging: 3 (4.9%) Atrophy, encephalomalacia or demyelination patterns: 5 (8.2%) NA: 2 (3.3%)
CSF findings	Lymphocytic pleocytosis with elevated protein and low glucose: 30 (49.2%) Lymphocytic pleocytosis (protein/glucose not fully specified): 9 (14.8%) Polymorphonuclear (PMN) or mixed pleocytosis: 5 (8.2%) Normal or near-normal CSF: 4 (6.6%) CSF not performed/not reported/not specified: 13 (21.3%)
Diagnostic confirmation of tuberculosis	Confirmed (microbiological/histopathological): 41 (67.2%) - CSF GeneXpert/CBNAAT/PCR: 13 - CSF AFB positive: 4 - CSF culture positive: 6 - Histopathology (biopsy-proven): 11 - Other microbiological (e.g., sputum, BAL, gastric aspirate): 7 Presumptive diagnosis (clinical + imaging ± CSF findings): 20 (32.8%)
Extra-CNS Tuberculosis	Pulmonary TB (active or past): 14 (23.0%) Disseminated/miliary TB: 4 (6.6%) Spinal TB/Pott's spine: 3 (4.9%) Lymph node TB (e.g., cervical, mediastinal): 2 (3.3%) Other extrapulmonary sites (e.g., GI tract, spleen/liver): 3 (4.9%) No extra-CNS TB/not reported/none evident: 35 (57.4%)
Spectrum of movement disorders	Ataxia: 17 (27.9%) Dystonia: 15 (24.6%) Chorea/hemichorea/choreoathetosis: 14 (23.0%) Parkinsonism: 9 (14.8%) Myoclonus/opsoclonus-myoclonus: 6 (9.8%) Tremor: 5 (8.2%) Torticollis/cervical dystonia: 2 (3.3%) Others (blepharospasm, stereotypy, etc.): 6 (9.8%)
Onset of movement disorders	Subacute onset (weeks to months): 26 (42.6%) Acute onset (sudden or within a few days): 13 (21.3%) After starting ATT: 10 (16.4%) During active TBM: 6 (9.8%) Paradoxical: 3 (4.9%) Delayed (months to years after TBM): 3 (4.9%)
Clinical features other than movement disorders	Motor weakness: 21 (34.4%) Cranial nerve palsy: 15 (24.6%) Altered consciousness: 13 (21.3%) Raised intracranial pressure: 7 (11.5%) Cognitive/behavioral changes: 6 (9.8%) Seizures: 5 (8.2%) Autonomic symptoms: 3 (4.9%) NA: 14 (23.0%)
Treatment received	**Primary Treatment:** ATT given: 59 (96.7%) Not treated/supportive care only/died before treatment: 2 (3.3%) **Adjunctive Therapies:** Corticosteroids (IV or oral): 38 (62.3%) Surgical intervention (e.g., VP shunt, decompression, biopsy): 13 (21.3%) ART: 1 (1.6%) **Other Medical Support** (e.g., physiotherapy, nutritional support, ICU care): 20 (32.8%)
Treatment for movement disorders	Symptomatic drugs for movement disorders used: 27 (44.3%) No specific drug used/not required: 25 (41%) Not specified/not mentioned: 9 (14.7%)

(*Continued*)

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Table 3. Summary of clinical, neuroimaging and treatment features of CNS TB cases presenting with movement disorders ($n = 61$) (*Continued*)

Variables	Values
Surgical intervention done	VP shunt or ventricular drainage: 13 (21.3%) Biopsy (stereotactic or surgical): 7 (11.5%) Surgical excision/craniotomy/myelotomy: 6 (9.8%) Pallidotomy (for movement disorder): 1 (1.6%) No neurosurgical intervention: 35 (57.4%
Clinical and functional outcomes	Fully recovered: 25 (41%) Marked or significant improvement: 17 (27.9%) Partial improvement: 13 (21.3%) Persistent or serious problems: 11 (18.0%) Death: 1 (1.6%) NA: 2 (3.3%)

AFB = acid-fast bacillus; ART = antiretroviral therapy; ATT = anti-tuberculous therapy; BAL = bronchoalveolar lavage; CBNAAT = cartridge-based nucleic acid amplification test; CNS = central nervous system; EVD = external ventricular drainage; GI = gastrointestinal; *H* = isoniazid; HIV = human immunodeficiency virus; HRZE = isoniazid, rifampicin, pyrazinamide, ethambutol; ICP = intracranial pressure; INH = isoniazid; IQR = interquartile range; IVIG = intravenous immunoglobulin; NA = not available; OMS/OMAS = opsoclonus-myoclonus syndrome/opsoclonus-myoclonus-ataxia syndrome; PAID = paroxysmal autonomic instability with dystonia; PCR = polymerase chain reaction; PMN = polymorphonuclear; *R* = rifampicin; RIPE = rifampicin, isoniazid, pyrazinamide, ethambutol; TB = tuberculosis; TBM = tuberculous meningitis; VP = ventriculoperitoneal.

Table 4. Proposed enteropathogenic mechanisms ($n = 61$)**

Proposed etiopathogenesis	1. Infarcts and Vasculitis: 18 (29.5%) *Due to TB-related inflammation of brain vessels, mainly affecting the thalamus, basal ganglia and brainstem.* 2. Tuberculomas and Mass Effect: 17 (27.9%) *Granulomas compressing brain areas like the basal ganglia, cerebellum or brainstem, disrupting motor pathways.* 3. Basal Ganglia Involvement: 11 (18.0%) *Direct TB infection or inflammation of the basal ganglia causing movement problems.* 4. Immune-Mediated Causes: 7 (11.5%) *Autoimmune responses or parainfectious processes affecting motor control areas.* 5. Drug or Toxin Effects: 4 (6.6%) *Side effects of TB drugs (like isoniazid, phenytoin) or electrolyte imbalance causing brain damage.* 6. Structural Damage: 2 (3.3%) *Bone or nerve compression (e.g., CV junction TB, Wallerian degeneration).* 7. Hydrocephalus and CSF Effects: 2 (3.3%) *Increased pressure or CSF blockage affecting motor regions.*

TB = tuberculosis; CV = craniovertebral. ***Etiopathogenesis was derived from author reports in original case descriptions where explicitly stated; otherwise, categorization was inferred by the reviewers based on clinical, radiological and treatment details.*

brainstem, compressive effects of tuberculomas, direct basal ganglia involvement, immune-mediated mechanisms, drug-induced neurotoxicity (such as isoniazid toxicity) and complications of hydrocephalus or structural lesions. Outcomes appear less favorable in cases with deep basal ganglia or thalamic infarcts, where persistent parkinsonism, dystonia or hemichorea were commonly reported. Conversely, patients with isolated tuberculomas or cerebellar involvement more often achieved complete or near-complete recovery after anti-TB treatment and adjunctive therapy (Table S1 and Table 4).

We also identified four cohort studies that met the PICO criteria.[3,4,68,69] Cohort studies further substantiated these findings. In a pediatric cohort from North India, tuberculous meningitis accounted for 4.3% of all movement disorder cases and typically presented with ataxia or tremors. In a large prospective adult cohort, secondary movement disorders due to CNS TB commonly manifested as dystonia, parkinsonism and tremors, with imaging frequently showing lesions in the thalamus or basal ganglia. Another study of patients with intracranial tuberculomas highlighted the role of focal or unilateral movement disorders caused by mass lesions affecting motor circuits. A separate prospective study of tuberculous meningitis patients found that 87% developed movement disorders, predominantly tremors, chorea, dystonia and parkinsonism, typically within the first three weeks of illness. These cohort findings emphasized that movement disorders are common, often underrecognized consequences of CNS TB, with early imaging and targeted therapy being crucial for improving neurological outcomes (Table 5).

Discussion

CNS TB, especially tuberculous meningitis, gives rise to a wide variety of movement disorders driven by inflammation, infarction, hydrocephalus or immune-mediated phenomena. Hyperkinetic forms such as chorea, dystonia, tremor, myoclonus, opsoclonus and stereotypies are commonly associated with basal ganglia or brainstem involvement, whereas hypokinetic syndromes like parkinsonism and akinetic mutism are usually linked to thalamic or frontal lobe injury. From a clinical perspective, early recognition of these disorders is crucial since they may precede or persist despite anti-TB therapy and significantly influence long-term outcomes. Active screening, timely neuroimaging and appropriate use of corticosteroids, surgery or symptomatic drugs can substantially improve prognosis (Table 6).

Immune-mediated mechanisms

In some patients, movement disorders arise without visible lesions, suggesting an immune basis. Molecular mimicry and cytokine-driven inflammation may lead to parainfectious syndromes such as opsoclonus-myoclonus or dystonia. These cases often respond to corticosteroids or immunotherapy, reinforcing the immune-mediated pathogenesis.[69]

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Table 5. Summary of four cohort studies on movement disorders in central nervous system (CNS) tuberculosis

Reference	Country	Summary	Quality assessment
Pandey et al., 2019[68]	India	In a hospital-based observational study of 93 children with movement disorders (excluding cerebral palsy) from North India, tuberculous meningitis (TBM) was identified in 4.3% of cases. **Clinical Presentations of TBM-Associated Movement Disorders** Children with TBM most often presented with ataxia or tremor. These manifestations reflect the common involvement of cerebellar and basal ganglia pathways in TBM. TBM was part of a broader infectious category that accounted for nearly half of all movement disorder cases in the cohort. **Comparative Burden** Although dystonia (24.7%), tremor (22.6%) and ataxia (19.4%) were the most frequent overall movement disorders, TBM contributed specifically to the ataxia–tremor subgroup, underscoring its neurological impact. Other infection-related etiologies included acute encephalitis syndrome and subacute sclerosing panencephalitis, but TBM remains unique because it is a treatable bacterial cause with potential for prevention and early intervention. **Outcomes** Severity was assessed using the Battini Childhood Movement Disorder Rating Scale. Most children, including those with infection-related etiologies such as TBM, showed gradual improvement over 3 months, with many recovering fully or partially. **Key Implications for TBM in Low-Resource Settings** TBM is an important but under-recognized cause of childhood movement disorders. Ataxia and tremor should raise suspicion of TBM in endemic regions, especially when associated with systemic or neurological signs of CNS tuberculosis. Infections remain the largest etiological category of pediatric movement disorders in such settings, emphasizing the need for early recognition, neuroimaging and prompt antitubercular therapy.	Newcastle–Ottawa Scale score = 6
Netravathi et al., 2012[69]	India	In a prospective study of 103 patients with secondary movement disorders (age range 2–70 years), tuberculosis contributed under two categories: vascular causes and space-occupying lesions. **Clinical Presentations** Tuberculous arteritis was identified among vascular causes and commonly presented with post-hemiplegic dystonia. Intracranial tuberculomas (three patients) were classified as space-occupying lesions and were most often associated with tremor and dystonia. **Comparative Burden** Vascular causes overall accounted for 22.3% of secondary movement disorders, with dystonia being the most frequent (56.6%). Tuberculous arteritis was one of these vascular insults. Space-occupying lesions contributed to 17.5% of cases, and tuberculomas were a notable subgroup, typically linked to tremor or combined tremor with dystonia. **Outcomes** The time interval between the insult and the onset of movement disorder varied. Tuberculous arteritis often caused subacute presentations within weeks to months, while tuberculomas were associated with delayed onset, with the average latency for space-occupying lesion-related disorders being about 129 weeks **Key Implications** Central nervous system tuberculosis, in the form of arteritis or tuberculomas, is a recognized but often underappreciated cause of movement disorders. Presentations include post-stroke dystonia from tuberculous arteritis and tremor or dystonia from tuberculomas.	Newcastle–Ottawa Scale score = 7
Alarcon et al., 2011[4]	Ecuador	A hospital-based cohort study from Ecuador evaluated 49 patients with intracranial tuberculomas (mean age 31.7 years; 53% male). **Clinical Presentations** Sixteen patients (32.6%) developed abnormal involuntary movements. The types were chorea (43.8%), tremor (31.3%), dystonia (18.8%) and myoclonus (6.3%). Most cases (87.6%) developed these manifestations within the first two weeks of hospitalization. **Comparative Burden** Patients with movement disorders had more severe illness on admission, greater motor (75%) and sensory deficits (43.8%) compared with controls. They were more likely to have multiple tuberculomas (68.8 vs. 36.4%) and deep supratentorial lesions involving the basal ganglia (31.3% vs. 21.2%). **Outcomes** At hospital discharge, most patients with movement disorders showed complete recovery (68.8%), some had partial recovery (12.5%), while a minority had persistent symptoms or died. **Key Implications** Intracranial tuberculomas are a major cause of secondary movement disorders, especially chorea, tremor and dystonia. The risk is highest with multiple and deep lesions, particularly when the basal ganglia are involved. Early diagnosis and antituberculous treatment lead to good outcomes in most cases, though some patients develop sequelae or fatal progression.	Newcastle–Ottawa Scale score = 6

(*Continued*)

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Table 5. Summary of four cohort studies on movement disorders in central nervous system (CNS) tuberculosis (*Continued*)

Reference	Country	Summary	Quality assessment
Alarcon et al., 2001[3]	Ecuador	A hospital-based study in Quito, Ecuador, reviewed 180 patients with TBM. Thirty patients (16.6%) developed movement disorders. **Clinical Presentations** Tremor: 20 patients (11.1%) Chorea: 7 patients (3.8%) Dystonia: 3 patients (1.6%) Some patients had overlapping features: tremor with myoclonus (1 case), dystonia with tremor (1 case). Chorea was more frequent in young children (mean age 5.9 years). Tremor predominated in adolescents and adults (mean age 30.5 years). **Comparative Burden** Patients with chorea and dystonia had more severe disease compared with those with tremor. Neuroimaging showed a higher frequency of deep vascular lesions (basal ganglia, internal capsule, diencephalon, mesencephalon) among patients with movement disorders compared with matched controls without movement disorders. Hydrocephalus was present in 60% and infarcts in 46.6% of those with movement disorders. **Outcomes** Follow-up extended to 24 months after completion of antitubercular therapy. Tremor cases often had the poorest functional prognosis, with residual disability more common. Mortality was recorded in six patients: three with tremor, two with chorea and one with dystonia. In most cases, the movement disorders were self-limiting, resolving over weeks to months, although some left persistent deficits. **Key Implications** Tremor is the most common movement disorder in TBM, while chorea is more frequent in children. Deep infarcts and basal ganglia involvement strongly predispose to movement disorders in this setting. Although often self-limited, these disorders may indicate more severe disease and are sometimes associated with poor outcomes or death.	Newcastle–Ottawa Scale score = 6

Table 6. Key findings of our review ($n = 61$)

- Most common movement disorder: Ataxia: 17 cases, 27.9
- Next most frequent: Dystonia: 15 cases, 24.6%; chorea/hemichorea/choreoathetosis: 14 cases, 23.0%
- Most common neuroimaging abnormality: Tuberculomas (isolated or multiple): 31 cases, 50.8%
- Other frequent findings: Hydrocephalus: 27 cases, 44.3%; infarcts (thalamic, basal ganglia, etc.): 21 cases, 34.4%
- Most common etiopathogenesis: Infarcts and vasculitis: 18 cases, 29.5%
- Overall outcome: 41.0% fully recovered, 18.0% had persistent/severe problems, 1.6% died

Vascular mechanisms

Vasculitis is one of the most common causes of movement disorders in CNS TB. In tuberculous meningitis, infectious vasculitis frequently produces infarcts involving deep brain structures such as the basal ganglia, thalamus, internal capsule and brainstem. These infarcts disrupt motor circuits, leading to manifestations such as chorea, dystonia, tremor or parkinsonism.[70]

Structural and compressive lesions

Tuberculomas can exert a mass effect or cause perilesional edema, particularly when located in motor regions like the basal ganglia, thalamus or brainstem.[4] Hydrocephalus, often due to obstruction of CSF pathways by basal exudates, can compress the thalamus or midbrain and disturb motor relays.[71] Involvement of the craniovertebral junction or brainstem in spinal or meningeal tuberculosis may also produce abnormal movements through direct compression or inflammation.[17,72]

Drug-related toxicity

Anti-TB drug neurotoxicity, particularly from isoniazid, may contribute to reversible encephalopathy involving motor pathways, especially in the absence of pyridoxine supplementation.[24] Clinical presentations can mimic vascular or structural complications.

Delayed sequelae

Rarely, movement disorders appear months to years after the initial CNS TB due to sequelae such as gliosis, cortical atrophy or Wallerian degeneration.[73] These delayed complications reflect long-term injury and remodeling of motor pathways after the acute infection has subsided.

Taken together, these mechanisms highlight the complex interplay of vascular, inflammatory, immune and drug-related factors underlying movement disorders in CNS TB (Table 3).

This review has several limitations. Most data come from case reports or small series, risking publication bias toward severe or atypical presentations. Variability in clinical descriptions, imaging and diagnostic criteria hindered meta-analysis. Many studies lacked long-term follow-up, consistent treatment details or microbiological confirmation. Immune, drug-induced and infectious causes were often not clearly differentiated. Additionally, geographic bias – mostly from high TB-burden regions like India – limits global generalizability. Future research should focus on prospective cohorts to define the incidence and course of movement disorders in CNS TB. Advanced MRI can clarify mechanisms, while long-term follow-up may reveal prognostic factors. Studies on immune and drug-induced causes are also needed to improve understanding and management.

Conclusions

This review shows that movement disorders are common yet underrecognized in CNS TB. Ataxia, dystonia, chorea and

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parkinsonism predominate. Causes include infarcts, tuberculomas, hydrocephalus and immune or drug effects. Early diagnosis, neuroimaging, anti-TB treatment and symptomatic therapy improve outcomes. Clinicians should actively screen for these treatable complications to reduce long-term disability.

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Data availability. All data generated or analyzed during this study are contained within the published article and its supplementary materials.

Author contributions. RKG: Conceptualization; Data curation; Formal analysis; Methodology; Project Software; Supervision; Validation; Visualization; Writing – original draft; Writing – review and editing. SP: Methodology; Writing – original draft; Writing – review and editing. RAM: Data curation; Formal analysis; Methodology; Writing – original draft; Writing – review and editing. SS: Project Software; Supervision; Validation; Writing – original draft; Writing – review and editing.

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Ethical statement. This study did not involve human or animal subjects; therefore, ethical approval was not required.

Ethics approval and consent to participate. As the study did not involve human participants, ethics approval and informed consent were not applicable.

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