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Original Research Article

Transverse Myelitis: Physiotherapy Assessment and Rehabilitative Interventions – A Review of Literature

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Abstract:

Objective: Acute transverse myelitis (ATM) is a rare inflammatory demyelinating disorder characterized by relatively acute onset of motor, sensory and autonomic dysfunction. Children comprise 20% of total cases of ATM. In this review, we described the current literature on ATM, focusing on the epidemiology, pathogenesis, clinical presentation, approach to diagnosis, differential diagnosis, treatment and outcome in the affected population.

Materials & Methods: We searched the related articles in electronic databases such as Scopus, EMBASE, Google Scholar, Springer and PubMed. All study designs were included.

Results: The related data focusing on the epidemiology, pathogenesis, clinical presentation, diagnostic approach and differential diagnosis, treatment and outcome of ATM were gathered and described.

Conclusion: The study concluded that no standard protocol for physiotherapy assessment and management is identified. The exercise program needs to incorporate task based functional training and recent advances such as Robotic therapy and Virtual Reality.

Keywords: Acute transverse myelitis, Transverse Myelitis, Transverse myelopathy, Acquired demyelinating syndromes, Myelitis, Physiotherapy in ATM.

Introduction

Transverse Myelitis (TM) is a rare neurological condition that causes bilateral lesions of the spinal cord.[1] According to National Institute of Neurological Disorders and Strokes (NINDS), it is an inflammation of the spinal cord that destroys the myelin sheath in Central Nervous System (CNS)[2] and is characterized by a marked sensory disturbance at a defined sensory level.[3] It is often referred to as myelitis, acute transverse myelitis (ATM) or partial myelitis in literature.[1,4] It usually effects three to four segments[1], involving one or more levels of the spinal cord.[5] TM may be completely characterized by long segment (more than three) and central cord affection whereas incomplete TM has a short segment (less than three) and peripheral cord affection.[5] The onset of symptoms are rapid & progressive.[6] It leads to impairments between spinal cord and nerve cells communication.[1]

The English neurologist Henry Bastian described the earliest signs and symptoms of transverse myelitis and were published in 1882 and 1910.[7,8] In 1928, Frank Ford

noted that in mumps patients who developed acute myelitis, symptoms only emerged after the mumps infection and associated symptoms began to recede. In an article in The Lancet, Ford suggested that acute myelitis could be a post-infection syndrome in most cases rather than an infectious disease where a virus or some other infectious agent caused paralysis. His suggestion was consistent with reports in 1922 and 1923 of rare instances in which patients developed "post-vaccinal encephalomyelitis" subsequent to receiving the rabies vaccine which then was made from brain tissue carrying the virus. The pathological examination of those who had died from the disease revealed inflammatory cells and demyelination as opposed to the vascular lesions predicted by Bastian.[9]

Ford's theory of an allergic response being at the root of the disease was later shown to be only partially correct, as some infectious agents such as mycoplasma, measles and rubella[10] were isolated from the spinal fluid of some infected patients, suggesting that direct infection could contribute to the manifestation of acute myelitis in certain cases.

In 1948, Dr. Suchett-Kaye described a patient with rapidly progressing impairment of lower extremity motor function that developed as a complication of pneumonia. In his description, he coined the term transverse myelitis to reflect the band-like thoracic area of altered sensation that patients reported.[7] The term 'acute transverse myelopathy' has since emerged as an acceptable synonym for 'transverse myelitis', and the two terms are currently used interchangeably in the literature.[11]

The definition of transverse myelitis has also evolved over time. Bastian's initial description included few conclusive diagnostic criteria; by the 1980s, basic diagnostic criteria were established, including acutely developing paraparesis combined with bilateral spinal cord dysfunction for <4 weeks and a well-defined upper sensory level, no evidence of spinal cord compression, and a stable, non-progressive course.[12,13] Later definitions, were written to exclude patients with underlying systemic or neurological illnesses and to include only those who progressed to maximum deficit in fewer than 4 weeks.[14]

As cited in an article by the Transverse Myelitis Consortium Working Group,[2,3] the incidence is 1-4 per million people with peak rates from 10-19 years and 20-39 years.[2,3,6] Another study in 2018 reveals that incidence rates are reported to be around 1.34-4.6 per million per year.[1] The incidence in children is 0.2 per 100,000 per year.[3] Incidence varies between developed and developing countries with higher incidence in the latter.[4] Approximately 20% of patients with TM are younger than 18 years of age.[15] Adults are more affected than children but males and females are equally impacted.[1]

In most cases, researchers doesn't know what initiates the inflammatory process.[2] According to Bhat et al. (2010) the condition can be idiopathic, but upto 50% of the cases are caused post-infection. Shin et al. (2013) in his study found that causes include demyelinating diseases, viral, bacterial or fungal infections, atopic myelitis, parasite infections, drugs or toxins, autoimmune disease, or can be considered idiopathic [1] It can exist as a part of a multifocal CNS disease, a CNS infection, a multisystem disease or as an isolated idiopathic entity.[15] 15-34% of the cases are idiopathic, 20.5 % are due to connective tissue disorder, 18 % are due to infarction of spinal cord, 1-17% are due to neuromyelitis optica and uncommonly post vaccination[5] including one for chicken pox[2] and other for COVID-19 which came to the limelight during the ongoing massive vaccine drive against coronavirus disease .[5] A study in 2021 found TM to be an unexpectedly frequent neurological complication of COVID-19. The pathogenesis of TM remains unknown, but the suggested mechanism is that SARS-CoV-2 antigens present in COVID- 19 vaccine may induce immune

mechanisms leading to TM. Most cases of SARS-CoV-2 associated TM have longer latency periods suggesting a post infectious origin.[16]

Another study conducted in April 2022 revealed TM as an emerging and serious neurological manifestation of COVID-19.[17] A case report held in 2021 has highlighted the association of TM with Acinetobacter Baumannii associated UTI.[5]

Histologic findings are different in idiopathic and disease-associated ATM, but inflammation and neural loss are seen in both Monocyte and lymphocyte infiltrations in the lesion and axonal degeneration are reported and both gray and white matters are involved. In fact, ATM is a combined inflammatory disease that involves multiple components of CNS including neurons, axons, oligodendrocytes, and myelin rather than a pure demyelinating disease. Histopathologic studies in adults show intralesional infiltration of monocytes and CD4+ and CD8+ T lymphocytes associated with astrocyte and microglia activation. Necrosis and cavitation may occur especially in NMO, resulting in severe disability. There have been described two potential mechanisms of autoimmunity in ATM including molecular mimicry and superantigen effect. A pediatric study showed significant increase in the Interleukin-6 (IL-6) levels in the CSF of the children with ATM. IL-6 plays role in cellular injury of the spinal cord. An association between increased IL-6 levels and disability has been shown. Studies showed a decreased IL-6 response to monoclonal antibody trial in early phase of ATM with appropriate outcome in NMO and ATM patients. Various autoantibodies are implicated in ADS by crossing the blood-brain barrier such as aquaporin-4 and myelin oligodendrocyte glycoprotein (MOG) antibodies in NMO and childhood ADS, respectively. The latter may be predicted a non-MS course. Some autoantibodies also may directly damage neurons that expose antigens which cross-react with antibodies against infectious organisms.[18].

It can affect motor, sensory and autonomic functions. The extent to what effects and their magnitude depends on the severity of the condition. Typical symptoms include bowel/bladder dysfunction, dermatomal pattern sensory loss, and/or weakness of most commonly affecting lower extremity flexors and upper extremity extensors depending on the level of lesion. In addition, back pain and fatigue can occur.[1] There can also be signs of spinal shock, areflexia, flaccidity, abnormal imaging, abnormal somatosensory evoked potentials, and a high deficit score at the onset. Fatigue is one of the most common symptoms in people with TM.[6]

Over a span of hours or days from the initiation of the immune response, the patient start losing motor control of the extremities, may also loose bowel and bladder control and even diaphragm control depending on the level of inflammation.[2]

Urinary retention may be the first sign of myelitis an should warrant further investigation into myelopathy.[19] Motor symptoms may vary according to the level of spinal cord involved. Upper cervical lesion (C1-C5) affects all four extremities, diaphragmatic dysfunction (C3, C4, C5) and respiratory failure. Lower cervical lesion (C5-T10) may develop UMN & LMN signs in UE and UMN signs exclusively in LE. Cervical lesion accounts for about 20% of cases. Lesion in thoracic region (T1-T12) may cause both UMN & LMN signs in the LE. It is the most commonly affected region (70%) in TM cases. Lesion in lumbosacral regions (L1-S5) may cause both UMN & LMN in the LE. Lumbar lesion accounts for 10% of cases approximately.[19]

Need of Study

First of all, there is very limited study available on TM due to rarity of the disease. Most of the research available are directed towards the diagnostic & medical treatment of the disease. Evidence on optimal physical therapy for TM is even more limited.[1]

Secondly, there is limited clinical research on physical therapy for patients diagnosed with TM.[6]

There is ample opportunity for more research to be done with this patient population.[1] Third , the effect of physical therapy on the speed of TM progression has not been shown in research yet.[2]

Fourth, none of the previous study has differentiated between the direct effect of physiotherapy interventions and other pharmacological and medical interventions such as administration of antibiotics, corticosteroids and muscle relaxants.[3]

Fifth, there is a surge seen in TM cases post covid vaccination as reported by studies held in 2021-2022.[5,16,17].

Aim of Study

The aim of this literature review is to find the already known and emerging assessment & management strategies for Transverse Myelitis

Assessment and Differential Diagnosis

The diagnosis of TM is proposed when the patient present with signs and symptoms of bilateral sensory, motor and autonomic dysfunction localized to one or more spinal segments without evidence of a cord compression. Therefore, the following criteria are necessary for the diagnosis of ATM:

1-Exclusion of compressive lesions, and
2-Confirmation of spinal cord inflammation as detected by the following:

i) the gadolinium enhancing lesion in MRI, or
ii) CSF evidence of either pleocytosis or elevated immunoglobulin type G (IgG) index

However, the lack of the inflammation markers does not exclude ATM. Developing symptoms of myelopathy considered as an emergency condition since severe squealed can occur if the disorder is not diagnosed and treated promptly . Emergent spinal MRI is required to exclude a compressive lesion. After that, determination of inflammatory or non-inflammatory myelopathy is necessary according to abovementioned markers. If inflammation is present and TM is suspected, then some investigations are recommended.

Brain MRI with and without contrast for detecting MS or ADEM; CSF analysis for cell count, protein, glucose, OCBs, IgG index, and cytology; serum NMO IgG antibodies (anti-aquaporin-4 IgG); serum B12; methylmalonic acid; human immunodeficiency antibodies; thyroid function test; antiphospholipid antibodies, antinuclear antibodies, rheumatoid factor and anti-dsDNA. In more than 40% of children, asymptomatic brain MRI lesions are seen and may be a risk factor for developing MS or NMO . Ophthalmologic consultation for detecting comorbid optic neuritis is recommended for all patients . Some children with encephalopathy and young children may not have complaints of vision impairments. Additionally, neurophysiological findings of subclinical optic neuritis may exist.[18]

Differential Diagnosis

Three categories in the differential diagnosis of idiopathic ATM are as follows:

1. Other forms of myelopathy such as compressive or non-inflammatory including epidural hematoma, intervertebral disk herniation, vertebral body fracture, ischemic myelopathy due to arterial compromise or venous hypertension. Spinal cord tumors occasionally present with subacute pain and myelopathic symptoms. Extramedullary tumors consist of nerve sheet tumors, meningioma, and metastasis of medulloblastoma. Intramedullary tumors include astrocytoma and ependymoma [(1)]. Spine tuberculosis is another cause of compressive myelopathy. Other noninflammatory causes are vitamins deficiency including B12, D, and E along with copper deficiency.
2. Secondary ATM including cases due to identified causes such as infectious

myelitis, a rheumatological disease (e.g. SLE and SS), paraneoplastic syndromes, demyelinating CNS disease (e.g. ADEM, MS, and NMO). Enteroviruses have recently been reported to be mediated in acute flaccid myelitis due to direct invasion to motor neurons of spinal cord.· Clear diagnosis of disease-associated TM by clinical and paraclinical features is important since the prognosis, treatment and recurrence risks are different. Patients with NMO typically have LETM.

3. Non myelopathic disorders can mimic ATM essentially Guillain-Barre syndrome (GBS). Clinical features of GBS including acute weakness and progressive motor and sensory dysfunctions resemble to ATM. However, some clinical and paraclinical factors help to discriminate them. Autonomic involvement in ATM present with intestinal or urinary urgency or retention, rather patients with GBS have cardiovascular instability. Sensory level is characteristic in ATM, while is never detected in GBS. IgG index in CSF and distinct spinal cord lesion in MRI are two findings in ATM but not detected in GBS patients. Finally, in GBS, conduction block or delayed conduction of peripheral nerves may be seen in electrodiagnostic studies, but these studies are usually normal in ATM patients.

Regarding the onset time of the symptoms of spinal cord syndrome, differential diagnosis is classified as follows: symptoms with acute or hyperacute onset should prompt consideration of spinal infarct, hemorrhage or disk herniation. Slowly progressive onset of the symptoms is in the favor of compressive myelopathy such as tumor, nutritional deficits, toxin exposure and hereditary disorders such as hereditary spastic paraplegia. Subacute presentation is in the favor of demyelination (TM or NMO), infections, and vasculitis (SLE).[18]

Management And Prognosis

Medical Management

The first line therapy for TM is Intravenous glucocorticoids (methylprednisolone or dexamethasone) for 3-5 days.[19] Medical management is not standardized and includes plasmapheresis, steroid therapy, intravenous immunoglobulin and chemotherapy.[2]

Physiotherapy Management

Physiotherapy focuses on reducing pain, muscle spasm, normalizing muscle tone, improve joint range of motion[3], prevention of bed sores & airway maintenance, maximize functional mobility. Traditional rehabilitation includes maximizing physical independence, capabilities and potentials. An interdisciplinary approach is used and the rehabilitation team consists of physicians, physiotherapists, nurses, family members, psychologists, speech pathologist, occupational therapists and orthotics etc. Because of its broad differential diagnosis, etiology, and prognosis, rehabilitation must be tailored on the specific setting in which TM occurred. Age at onset should also be considered.[15]

Prognosis and Outcome

Outcome of ATM in children is better than adults, as almost 50% of children obtain recovery after 2 year. In general, outcomes are very different from complete recovery without sequelae to entire paralysis and even death. 33%-50% of the patients show complete recovery and 10%-20% of cases have poor outcome. Infants have the worst outcome. More frequency of LETM in infants and inability of immature brain to recover from injury in them are two reasons. Recovery phase can continue up to several years. Death is usually due to a high cervical cord injury and respiratory insufficiency. Sensory deficits and urinary dysfunction are the most common sequelae (15%-50%). Nearly 10%-20% of patients never obtain mobility or urinary function. Poorer outcomes have been associated with: younger age at the onset of the

disease, rapid onset of symptoms, shorter time (less than 24 h) to maximal deficit, complete paraplegia, need to assisted ventilation, longer time to treatment, absence of CSF pleocytosis, higher border of sensory level, T1 hypo intensities in spinal cord MRI and longitudinal extent of the cord lesions. Prognosis is better in TM associated with ADEM. TM in most patients is monophasic, but 25%-33% of the patients with idiopathic TM and as high as 70% of patients with disease-associated TM develop recurrences. Factors that predict recurrence include: longitudinally extensive lesions in spinal cord, brain lesions on MRI, existence of one or more autoantibodies (ANA, ds DNA, phospholipid antibody, C-ANCA), underlying collagen vascular disease, OCBs in CSF, presence of NMO-IgG (anti-Aquaporin-4)antibody and female sex. Patients with LETM have an increased risk for NMO, LETM, recurrent ATM, ATM with concurrent or rapidly sequential optic neuritis suggest NMO. In addition, OCBs can be detected in 30% of patients with NMO. Besides, patients with OCBs in CSF have an increased risk for evolving MS.

According to a Canadian study, MS develops in 13% of children with ATM. In addition, patients with patchy lesions involving 1-3 segments in cord, partial myelitis, and brain lesions in MRI have a rate of 60%-90% progression to MS during 3-5 years. A study has reported cognitive impairment in 10% of pediatric ATM patients.

We suggested monitoring of this population for cognitive problems. Apart from initial outcome, ATM patients should be monitored longitudinally, either to clearing diagnosis or to provide rehabilitative interventions for motor deficits, urinary dysfunction, psychological and cognitive impairment.

ATM is a heterogeneous disorder in children with a broad spectrum of clinical presentation, etiology, and outcome. It may be the first presentation of relapsing acquired demyelinating syndromes or occur as an isolated post-infectious condition. It also must be distinguished from compressive and non-inflammatory myelopathies. Diagnostic evaluation, prognosis, recurrence risk, and treatment plans are different among these entities. Therefore, the correct diagnosis is crucial for treatment and prognosis.[18]

Methodology

Inclusion Criteria

- Control Trials
- Randomized Control Trials
- Review Articles
- Systematic Review
- Case Study
- Only English Language Articles

Exclusion Criteria

- News Blog
- Conference Proceeding
- Article in other Language

Search Strategies

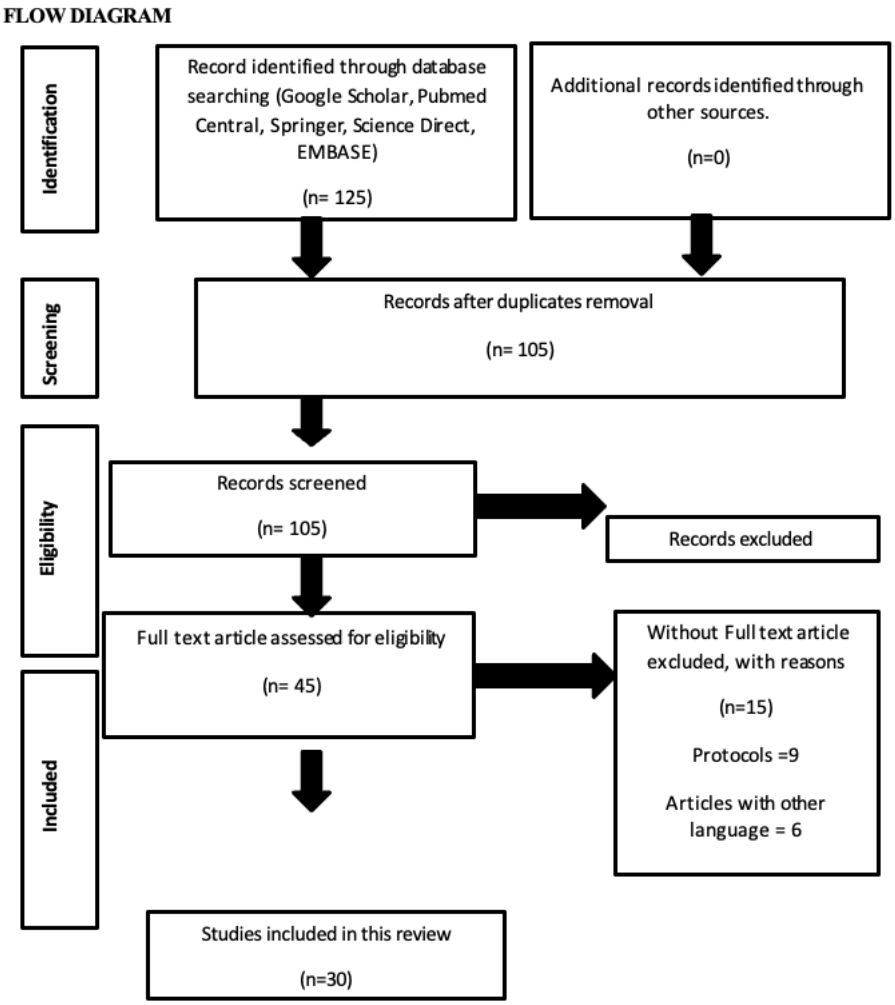
Comprehensive literatures in English language were identified by searching through various bibliographic database and search engine like PubMed Central, Google Scholar, Science Direct, Springer and EMBASE.

Search Duration

The study limited from 2010-2023.

Keywords

Searching for studies in English language using the Medical Subject Headings (MeSH) terms like Transverse Myelitis, Myelopathy, Physiotherapy in TM, Neurophysiological Imaging.



Result

A total of 30 articles were included in the study out of which 10 are case studies, 9 are review articles, 5 are cohort studies, 3 are review of literatures, 2 are RCT and 1 is a cross sectional study.

Out of 30 articles 1 article suggested nomenclature, 1 article suggested the prevalence, 1 article reported about the classification system, 4 articles explained etiology , 2 articles explained about pathophysiology, 4 articles explained clinical presentation, 5 articles reported about differential diagnosis, 3 articles suggested medical management, 2 articles compared traditional versus novel method of treatment, 8 articles highlights the effects of physiotherapy in TM rehabilitation, 3 Articles explained the close relation of TM and MS, 5 articles explained COVID-19 is not an uncommon cause of TM explaining its association with it, 2 articles explained about GBS overlap ATM syndrome.

Discussion

ATM is a heterogenous disorder in children with a broad spectrum of etiology, clinical presentation, pathophysiology and outcome. It may be the first presentation of relapsing acquired demyelinating syndrome therefore correct diagnosis is crucial for treatment and prognosis.[18]

The current study illustrates the review for assessment and management which can lead to accurate diagnosis and better rehabilitation program for the patients suffering from TM. Although the prevalence of TM is very low 1- 4 in 1 million per year[2,3,6], but it leads to deterioration in the quality of life of the patients suffering from the condition. The

common age group affected by TM is 10-19 years and 20-39 years,[2,3,6] but approximately 20% of affected individuals are younger than 18 years of age.[15] In a study conducted by **Onyekere CP et al. (2018)**, the incidence rate was reported to be around 1.34-4.6 per million per year.[3] **Gupta A et al. (2016)** in his study found that incidence of TM can vary between developed and developing countries with higher rate of incidence in countries with poor socioeconomic status.[4]

Although the exact etiology of TM is unclear, most of the cases are idiopathic and post infectious.[18] According to various studies different causes are bacterial, fungal, viral and parasitic.[1,5] According to a case series by **Simone CG et al. (2022)** 64% of cases were idiopathic and 36% were associated with disease,[9] while in another report by **Beh SC et al. (2013)** idiopathic TM accounts for 15-30% of cases.[36] **Sharma DJ et al. (2021)** in a case report has highlighted that *Acinetobacter Baumannii* that infect the catheter can cause urinary tract infection which further leads to TM.[5] In the recent years after the pandemic attack of COVID-19, there has been various researches that highlight the after effects and neurological complications of COVID-19 vaccines that include GBS, Stroke, Dizziness and Headache. One of such study by **Lingas EC et al. (2022)** discussed that the ATM is a common neurological complication after COVID-19.[17] In a study based on single hospital conducted by **Roman GC et al. (2021)** in 1760 COVID-19 patients form Italy, TM alone accounted for 1.2% of all neurological complications.[16]

As mentioned above the two most common etiologies of TM are idiopathic and inflammatory, studies have found that both have different histopathology, but inflammation and neuronal loss are seen in both causes. There is intralesional infiltration of monocytes, CD4+ and CD8+ T lymphocytes which leads to activation of astrocytes which leads to necrosis and finally disability. The other two proposed theory for the pathogenesis of TM in idiopathic cause are molecular and superantigen effect.[18] ATM is a combined inflammatory disease that involves many components of CNS. A study conducted by **Tavasoli A et al. (2018)** found that neurons, axons, oligodendrocytes and myelin sheath are the various CNS components that are affected.[18]

Magnitude of clinical presentation in TM depends on the severity of the disease.[1] Its clinical presentation commonly matches with other spinal cord injuries. In my opinion it carries a vast clinical picture that includes motor, sensory, autonomic and immune system dysfunction. In a study conducted by **Schrader C et al. (2018)**, various components that are affected by TM is explained. Motor weakness in flexors of lower extremity and extensors of upper extremity depending upon the level of lesion, dermatomal pattern sensory loss, loss of bowel and bladder functions, loss of reflexes along with back pain and fatigue.[1]

Diagnosis of TM is a crucial component in treatment and prognosis. Developing symptoms of TM is considered as an emergency condition since severe sequel can occur if not diagnosed correctly. **Tavasoli A et al. (2018)** proposed a set criteria for the diagnosis of ATM that is exclusion of compressive spinal cord lesion and conformation of spinal cord lesion by MRI and CSF.[18] So it can be concluded that the diagnosis is purely based on radiological and laboratory findings. In this review it has been found that there are no studies that highlight the assessment and evaluation in physiotherapy.

Early intervention in TM can lead to better prognosis and outcome,[19] so there need to be a standard protocol in medical and physiotherapy management. There is no standard protocol available in medical management that includes plasmapheresis, corticosteroids, IVIG and

chemotherapy.[2] Also there is no set protocol in PT for TM. The primary goal is to treat patients according to their clinical presentation.[6] Physiotherapy rehab includes active and passive range of motion exercise, strengthening exercise, joint mobilization, neuromuscular re-education, passive stretching, resistance exercises, functional exercises along with various electrotherapeutic modalities, splints and orthosis.[3,6,15,19] According to a study conducted by **Hartono et al. (2018),** a patient diagnosed with TM having weakness and loss of sensation in lower limb was given high dose corticosteroid as first line medical management along with PT management that includes AROM exercises with walking 3 session/week of 30 minutes, TENS 2 session/week for 20 minutes, sensory stimulation 2 session/week for 20 minutes and bladder training for 3 weeks.[39,40]

Conclusion

As per the findings of the literature review, it is found that the diagnosis of Transverse Myelitis is purely based on radiological and laboratory investigations and no standard protocol for physiotherapy assessment or evaluation has been revealed. Focusing on the physiotherapy management it is concluded that the physiotherapy treatment program is specifically tailored based & include conventional physiotherapy such as range of motion exercises, strengthening exercises, joint mobilisations, neuromuscular re-education, gait training etc. and no standard protocol has been identified. The literature also suggests that there is a need to incorporate activity-based functional tasks and movements along with recent advances such as Robotic Therapy and Virtual Reality emphasizing the impairments into the exercise program for better recovery and outcome in patients with Transverse Myelitis.

Limitation and Future Scope

Limitations:

1. Due to very less prevalence, many studies have not been conducted on Transverse Myelitis.
2. There is lack of literatures on Indian population.
3. There is lack of experimental studies done on this disease.

Future Scope Of Study:

1. Direct effect of physical therapy apart from medical management needs to be studied.
2. RCTs are required to replicate the findings of physiotherapy management in ATM.
3. Experimental research with more TM patients is required in the future to determine whether a measurable benefit of PT exists for this population.
4. Studies on proper physiotherapy assessment and evaluation on TM are required.
5. Ample opportunity for more research to be done with this patient population.
6. Research to identify the responsible antigen and immune-pathogenesis of COVID-19 associated ATM must be encouraged.

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