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Mohie-eldin Tharwat Mohamed
Department of Neurology, Faculty of Medicine for boys, Al-Azhar University, Cairo, Egypt.

Abdel-Ghaffar Ismail Fayed
Department of Neurology, Faculty of Medicine for boys, Al-Azhar University, Cairo, Egypt.

Islam Shaaban al-Emam
Department of Neurology, Faculty of Medicine for boys, Al-Azhar University, Cairo, Egypt.,
eslamsalman211@gmail.com

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ORIGINAL ARTICLE

Evaluation of Non-compressive Myelopathy in a Sample of Egyptian Patients

Mohie-eldin Tharwat Mohamed, Abdel-Ghaffar Ismail Fayed, Islam Shaaban Al-Emam*

Department of Neurology, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt

Abstract

Background: **'Spinal cord malfunction in the lack of clinico-radiological evidence of spinal cord compression' is the definition of non-compressive myelopathy. In the absence of imaging modalities that show compression, it may be caused by demyelinating, viral, autoimmune, vascular, degenerative, and metabolic illnesses.**

Aim and objectives: **The goal of this research was to assess the etiological factors of non-compressive myelopathy in a sample of Egyptian patients.**

Subjects and methods: **Between January 1 and June 30, 2022, researchers at Cairo, Egypt's Al-Azhar University Hospitals (Al-Hussein and Sayed Galal) performed a prospective observational study on patients with non-compressive myelopathy. At three and six months, patients were followed up on to assess any lingering impairments and disabilities.**

Results: **The study had 60 patients with median age of 30.70 ± 12.4 years where female: male ratio was 1.3 : 1. Diagnostic classification/NCM was MS spectrum in 32 (53.3%) patients, Idiopathic ATM in 10 (16.7%), NMO spectrum in 6 (10.0%), HSP in 8 (13.3%), Infections/Para infection (Viral myelitis) in 2 (3.3%), and ADEM in 2 (3.3%).**

Conclusion: **According to the finding in the present study, a wide range of diseases present with non-compressive myelopathies. In the majority of cases, the definitive diagnosis can be reached with a thorough history and clinical examination along with the proper workup. Our diagnostic precision has improved because of neuroradiology and serological indicators. Immediate diagnosis of these diseases makes them reversible, therefore, a proper treatment can improve the outcomes of this disease.**

Keywords: Myelopathy, Neuroradiology, Non-compressive, Spinal cord

1. Introduction

A pathologic disease or neurological deficiency involving the spinal cord is collectively referred to as myelopathy. A common and possibly incapacitating neurologic emergency is myelopathy.[1]

Imaging investigations are crucial for the detection of spinal cord diseases since they can have fatal effects. The clinical context, the length of time from the start of symptoms and indications, and the imaging results must all be taken into consideration when developing a diagnosis.[2]

Myelopathies may be compressive or non-compressive, regarding to subarachnoid space obstruction.[3]

Non-compressive myelopathy (NCM) is described as 'spinal cord involvement without detectable clinical and radiological evidence of spinal cord compression causing neurological deficit.[4]

NCM is a challenging condition to evaluate because of the wide differential diagnosis. The value of observational studies resides in the recognition of significant illnesses and in offering inspiration for future study.[5]

Immediate diagnosis of these diseases makes them reversible, so the physicians and neurologists need to realize the relation between clinical presentation and radiological findings in magnetic resonance imaging, where prediction relays on early and accurate diagnosis.[6]

In light of updated diagnostic criteria and serological testing, detailed data on Egyptian individuals with non-compressive myelopathy (NCM) are scarce. The clinic-radiological characteristics of non-

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* Corresponding author at: Department of Neurology, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt.
E-mail address: eslamsalman211@gmail.com (I.S. Al-Emam).

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compressive myelopathy (NCM) has not yet been investigated in any studies. With particular attention to its radiological aspects, we are attempting to share our knowledge of the etiological pattern of non-compressive myelopathy (NCM) in this work.

The goal of this study was to study the etiological factors of non-compressive myelopathy in a sample of Egyptian patients.

2. Patients and methods

Between January 1 and June 30, 2022, researchers at Cairo, Egypt's Al-Azhar University Hospitals (Al-Hussein and Sayed Galal) performed a prospective observational study on patients with non-compressive myelopathy.

2.1. Inclusion criteria

Patients with subacute, or chronic neurologic malfunction (motor and sensory deficit, sphincteric involvement, and a well-defined upper sensory segmental level) consistent with myelopathy (whether or if encephalopathy, neuropathy, or radiculopathy are also present).

2.2. Exclusion criteria

Patients with clear cerebral symptoms inconsistent with spinal pathology (e.g., aphasia, hemianopia, neglect, facial involvement), motor neuron disease (MND), and degenerative ataxias, as well as patients with myelopathy who have not had magnetic resonance imaging (MRI) of the spinal cord. Although there was considerable spinal cord pathology in MNDs and several degenerative ataxias, these conditions were omitted from the research because they were diffuse processes with extensive cerebral cortex, brainstem, and cerebellar involvement.[4]

2.3. Operational design

The following procedures were applied to all patients.

2.4. History taking

Personal information (Name, age, and sex), past medical history, drug history, family medical history, neurological symptoms, skin rash, photosensitivity, joint pain, bone pain, edema, jaundice, anemia, gastrointestinal hemorrhage, cough, chest pain, exposure to radiation and other toxic substances, travel history, and high-risk sexual history.

General checkup in the form of Vital signs (Temperature, Blood pressure, pulse rate, Respiration rate), **signs of** (Pallor, jaundice, cyanosis, and enlarged lymph nodes). Evidence of a systemic illness or cancer, uveitis, scleritis, and conjunctival xerosis symptoms, lesions from the zoster virus, erythema migrans, erythema nodosum, etc.

2.5. Neurological examination

2.5.1. Mental status

Detailed mental examination with particular attention to memory impairment, inattention and mood changes.

2.5.2. Cranial nerves

Preserved cranial nerves confirm spinal cord lesions. Assessment of all cranial nerves with focusing on optic nerve as optic neuritis was found in many inflammatory disorders related to myelitis. **Motor Exam:** Full Testing was done to confirm distribution of weakness, as well as compare the left and right sides, looking for lower motor neuron signs at the level. Determination of grade of the weakness, Examination for **Ataxia** and **Gait. Deep Tendon Reflexes**: The assessment of reflexes had A precise localizing value (confirm level). **Sensory Examination**: detect and confirm sensory level. Examination of all sensory modalities.

2.5.3. Investigations

Spinal MRI: In the sagittal (cervical, dorsal, and lumbar) sections, we recorded the number of lesions, their size, and their location, while in the axial sections, we noted centromedullary versus a peripheral site, cord edema, and the alterations seen after gadolinium-contrast administration. **Brain MRI:** Siemens Magnetom 1.5 T MRI machine with a 4 mm slice thickness was used for the MRI. **Laboratory investigations**: **Routine lab:** Complete blood picture (CBC): Red blood cells (RBCs), white blood cells (WBCs), and platelet count, hemoglobin level (Hb%). Renal function test: urine, blood urea, and serum creatinine analysis. Liver Test Profile: Serum erythrocyte sedimentation rate, C-reactive protein, thyroid profile, urinalysis, serum aspartate and alanine aminotransferases (AST and ALT), serum albumin, serum bilirubin, prothrombin time, and international normalized ratio (INR). **Special tests were done when needed according to clinical evaluation:** Tests for vitamin B-12 status, antinuclear antibodies, anti-aquaporin-4 antibodies, and analyses of the total count, differential counts, protein, and sugar in the cerebrospinal fluid (CSF) are all available. Polymerase chain reaction (PCR) assays

for varicella-zoster virus (VZV), herpes simplex virus (HSV), cytomegalovirus (CMV), Epstein-Barr virus, Mycobacterium tuberculosis, and toxoplasma gondii were performed on the cerebrospinal fluid (CSF). Human immunodeficiency virus, hepatitis B, and hepatitis C serological testing. **Electrophysiological studies:** in patients having co-existent neuropathy or radiculopathy was done using EMG/NCV/EP system and visual evoked potentials (VEPs).

2.5.4. Treatment and follow up

All included patients received treatment according to protocol of therapy in our hospital. At three and six months, patients were followed up on to assess any lingering impairments and disabilities. A successful result was deemed to be a partial or full recovery, the capacity to walk unassisted, isolated urine problems, or a modified Rankin Scale (mRS) < 3. mRS ≥3 or death, being unable to walk without assistance, were considered poor outcomes.[4]

2.5.5. Ethical considerations

The Neurology Department, Faculty of Medicine, Al-Azhar University, Cairo, Egypt, code 0000044, granted approval for the study's execution. All participants were asked for their written permission. The research was entered into the ClinicalTrials.gov database.

2.6. Data statistical analysis

Utilizing SPSS version 20, data input, processing, and statistical analysis were completed (Statistical Package for the Social Sciences). The Kruskal-Wallis, Wilcoxon, Chi-Square, logistic regression analysis, and Spearman's correlation tests of significance were applied. According to the kind of data (parametric and non-parametric) collected for each variable, data were presented, and an appropriate analysis was carried out. *P* values of 0.05 or below (5%) were regarded as statistically substantial.

3. Results

From January 1 to June 30, 2022, 60 patients with non-compressive myelopathy were included in this prospective observational research at the Department of Neurology at Al-Hussein and Sayed Galal Hospitals of Al-Azhar University in Cairo, Egypt.

A total of 124 adult patients with non-compressive myelopathy were initially screened for inclusion in the present study. Of them, 24 patients were excluded due to not meeting inclusion criteria ($n = 15$), or refused to sign the written informed consent ($n = 9$). Then, patients who lost follow up ($n = 22$), or did not undergo MRI of the spine ($n = 18$). Thus, a total of 60 patients were included in the present analysis (Fig. 1).

The patients median age was 30.70 ± 12.4 years, 34 (56.7%) female and 26 (43.3%) male patients. The onset of the disease was acute in 21 (35.0%) patients, chronic in 12(20.0%) patients, subacute in 12 (20.0%) patients, and relapsing in 15 (25.0%) patients as shown in Table 1.

There were previous attacks of demyelination/myelopathy in 23 (38.3%) patients, preceding illness in 8 (13.3%) patients, consanguinity in 12 (20.0%) patients, and similar familial illness in 6 (10.0%) patients as shown in Table 2.

There were pyramidal signs in 50 (83.3%) patients, peripheral neuropathy in 15(25.0%) patients, brain stem involvement in 5 (8.3%) patients, flexor spasm in 5(8.3%) patients, encephalopathy in 3 (5.0%) patients, dystonia in 1 (1.7%) patient and radiculopathy in 1 (1.7%) patient. There were cerebellar symptoms in 12(20.0%) patients. the clinical findings of the patients were summarized in Table 3, CSF was done in 27 patients, and it was normal in 2 (3.3%) patients, there was +ve OCB/high IG g index in 13 (21.7%) patients, pleocytosis in 7 (11.7%) patients, and protein 5 (8.3%) patients. MRI spine showed short segment in 43 (71.7%) patients, LETM in 10 (16.7%) patients and Gadolinium enhancement in 10 (16.7%) patients.

Diagnostic classification/NCM was Idiopathic ATM in 10 (16.7%) patients, MS spectrum in 32 (53.3%) patients, NMO spectrum in 6 (10.0%) patients, Hereditary spastic paraplegia (HSP) in 8 (13.3%) patients, Infections/Para infection (Viral myelitis) in 2 (3.3%) patients, and ADEM in 2 (3.3%) patients as shown in Fig. 2.

Treatment strategy was Steroids in 50 (83.3%) patients, Plasmapheresis in 16 (26.7%) patients, and supportive in 10 (16.7%) patients. **At admission**, mRS mean was 3.38 ± 1.61, **After 3 months**, it was 2.98 ± 1.7, **After 6 months** it became 1.67 ± 1.22 with values ranging between 0 and 5 in the patients. There was highly statistically significant improvement in mRS after 3 months and more improvement after 6 months than at admission (Fig. 3).

Fig. 4.

4. Discussion

Spinal cord disorders can result in catastrophic and debilitating effects. Usually, after thorough inquiry, the cause is still unknown. In the preceding

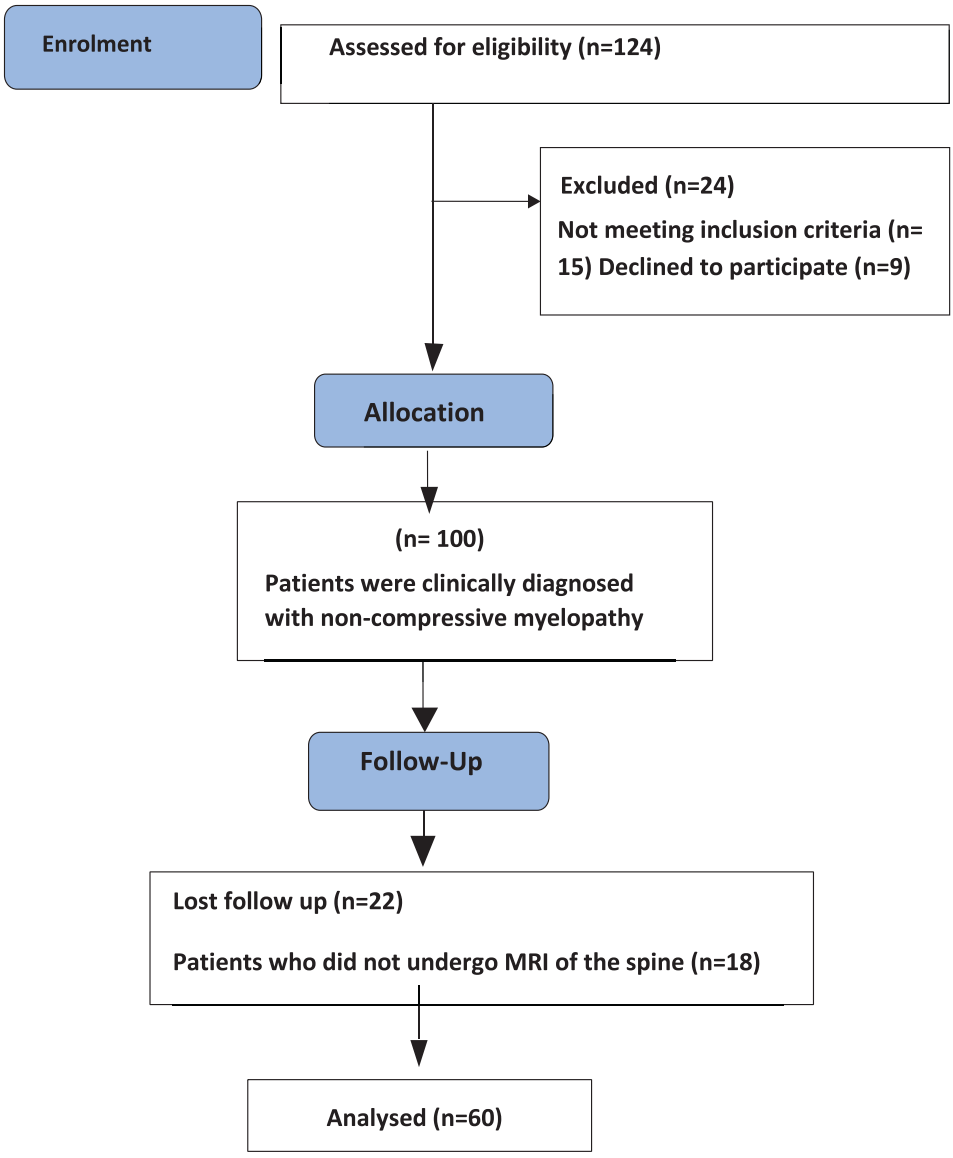

Fig. 1. Allocation and flow of cases.

studies, patients with myelopathy, around fifth of them were idiopathic or of unknown origin. Myelitis may manifest for the first time in some conditions including Multiple Sclerosis (MS), Neuromyelitis Optica Spectrum Disorders (NMOSD), and ANTI-MOG Spectrum Disorders. and the exact illness may not be identified during the initial episode, leading to the designation of Idiopathic. As a result, there is a chance of recurrence after a first episode,

Table 1. Demographic data and onset of disease of patients.

Age (mean ± SD)	(30.70 ± 12.4)
Sex	*N* (%)
Female	34 (56.7%)
Male	26 (43.3%)
Onset	*N* (%)
Acute	21 (35.0%)
Chronic	12 (20.0%)
Subacute	12 (20.0%)
Relapsing	15 (25.0%)

Table 2. Previous attacks, preceding illness, consanguinity and family history.

Previous attacks of demyelination/myelopathy	*N* (%)
None	37 (61.7%)
Yes	23 (38.3%)
Preceding illness	
None	52 (86.7%)
Yes	8 (13.3%)
Consanguinity	
None	48 (80.0%)
Yes	12 (20.0%)
Positive family history	
None	54 (90.0%)
Yes	6 (10.0%)

Table 3. Clinical findings of the patients.

Items	*N* (%)
Muscle state	
Normal	24 (40.0%)
Spastic	32 (53.3%)
Wasting	4 (6.7%)
Superficial Reflexes	
Normal	8 (13.3%)
Lost	52 (86.7%)
Deep Reflexes	
hyper-reflexia	30 (50.0%)
Normal	22 (36.7%)
hypo-reflexia	8 (13.3%)
Pattern of sensory loss	
Posterior column sensory loss	30 (50.0%)
Spinothalamisensory loss	24 (40.0%)
None	6 (10.0%)
Weakness pattern	
Monoparesis	4 (6.7%)
Paraparesis	17 (28.3%)
Hemiparesis	3 (5.0%)
Quadriparesis	36 (60.0%)
Sensory loss pattern	
None	20 (33.3%)
Hemisensory	8 (13.3%)
Definite Sensory level	32 (53.4%)
Bladder involvement	
None	22 (36.7%)
Yes	38 (63.3%)
Bowel involvement	
None	48 (80%)
Yes	12 (20%)
Speech involvement	
None	45 (75.0%)
Yes	15 (25.0%)

hence thorough clinical and radiological follow-up is advised. During follow-up, patients who have been given the idiopathic diagnosis may modify it.

In light of updated diagnostic criteria and serological testing, detailed data on Egyptian individuals with non-compressive myelopathy (NCM) are scarce.

The median **age** of patients was 30.70 ± 12.4 years in the current study. This due to most cases were MS, and the symptoms of MS usually begin between the ages of 20 and 40 with a peak incidence of 24 years. This result was lower than reported in **Kayal *et al.*,**[7] study which showed that median age of patients with non-compressive myelopathies was 35 years. Similarly, the median age of clinically diagnosed patients with NCM was 34.5 years in **Thangaraj & v. R.,**[4] study and 34.45 years in **Prabhakar *et al.*,**[8] study.

In the current study, there was **females' preponderance** compared with males. Similar results reported in **Barhate *et al.*,**[9] study where female: male ratio was 7.8:1. This may be explained by females' preponderance in neuromyelitis optica spectrum disease[10] and multiple sclerosis.[11]

With regards to the disease's **onset** in the present research, acute onset showed a larger prevalence (35.0%) followed by relapsing (25%) then chronic and subacute (20% for both), this is because most of our cases were diagnosed with inflammatory diseases of CNS which often present in acute onset, and was relapsing in MS cases in particular. Our result agreed with **Kayal *et al.*,**[7] also, likewise, **Kamble *et al.***[12]

In contrary to our result, **Thangaraj & v. R.,**[4] study results revealed that most of non-compressive myelopathy patients were chronic in 54 (72%) patients followed by acute in 10 (13.3%) patients, relapsing in 6 (8%) patients, subacute in 5 (6.67%)

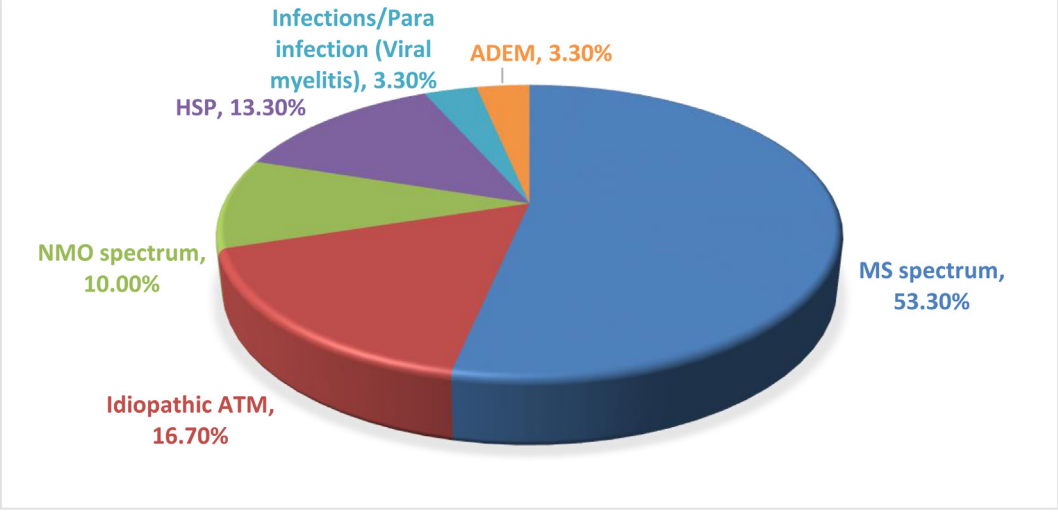

Fig. 2. Diagnostic classification.

patients, this is because most of their diagnoses were degenerative myelopathy as hereditary spastic paraplegia and ALS.

Regarding **weakness pattern** in the present study, most of cases were quadriparesis 36 (60.0%) patients; as most lesions in the spinal MRI were in the cervical region These results were matched to, **Kayal *et al.*,**[7] study results showed that a higher percentage of the included patients presented with quadriparesis in 81 (56%) patients. Similarly, among patients with non-compressive myelopathies in **Kamble *et al.*,**[12] study, there was quadriparesis in 54 (67.5%) patients. By contrast, **Thangaraj & v. R.,**[4] study results which showed that patients with non-compressive myelopathies presented with paraparesis in 38 (52.05%) patients, quadriparesis in 21 (28.77%) patients.

In the present study, as a well-defined truncal sensory level, below which the sense of pain and temperature is changed or eliminated, separates myelopathy from brain lesions and peripheral neuropathies, the **sensory loss pattern** was largely clear in 32 (53.3%) instances, our study agreed with **Thangaraj & v. R.,**[4] Posterior column sensory loss was predominant in 30 (50.0%) patients in the present study. As most of cases were MS where lesions of MS affect the posterior-lateral aspect of spinal cord, our result was comparable to **Kamble *et al.*,**[12] study, **Kayal *et al.*,**[7] study, and **Thangaraj & v. R.,**[4] study.

The current study found that 53.3% of patients had spasticity in their muscles, 86.7% lost their superficial reflexes, 50.0% had hyperreflexia in their deep reflexes, and acutely, limb tone and muscle

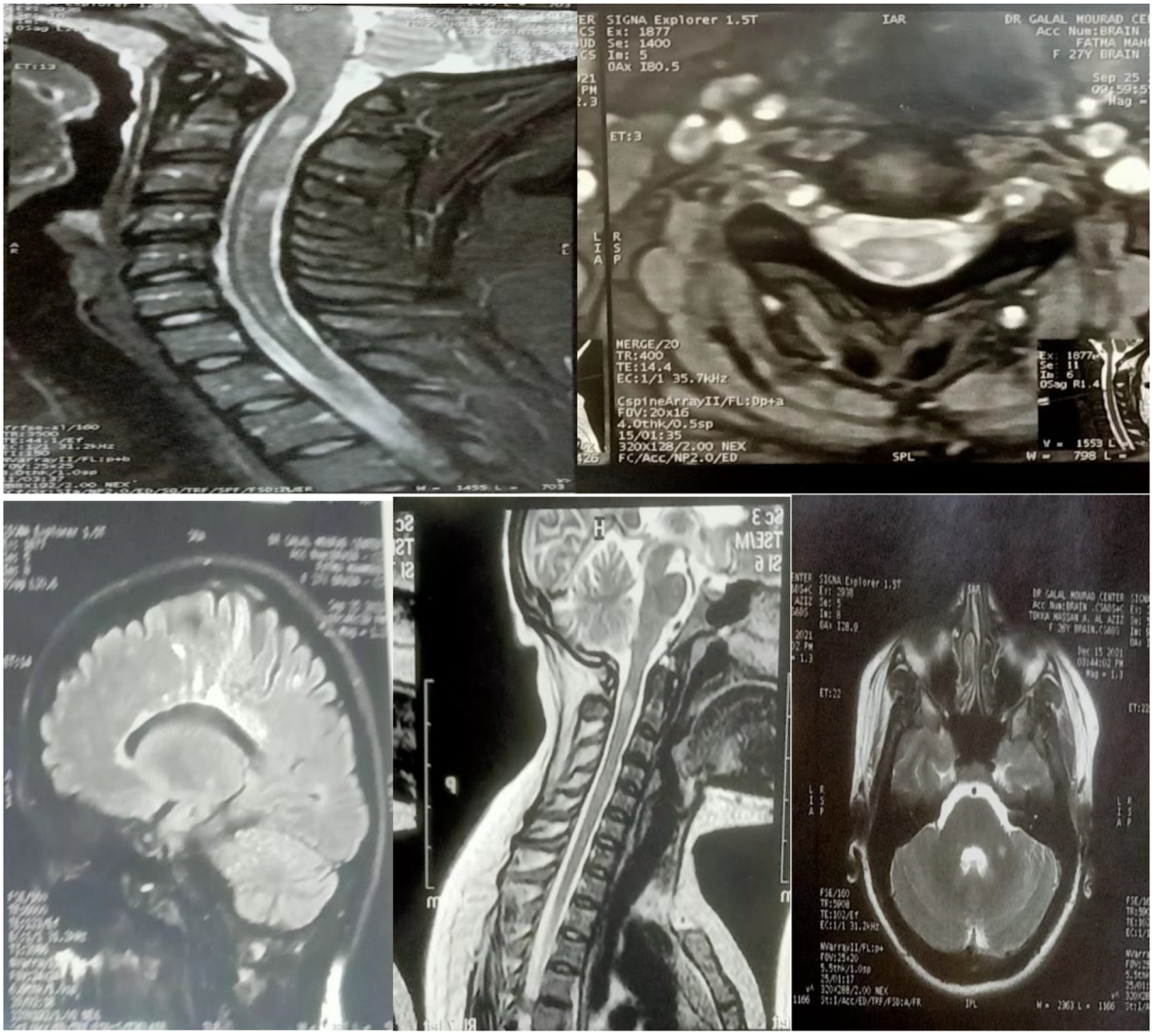

Fig. 3. Multiple sclerosis: spinal short segment lesions and brain lesions (supratentorial and infratentorial).

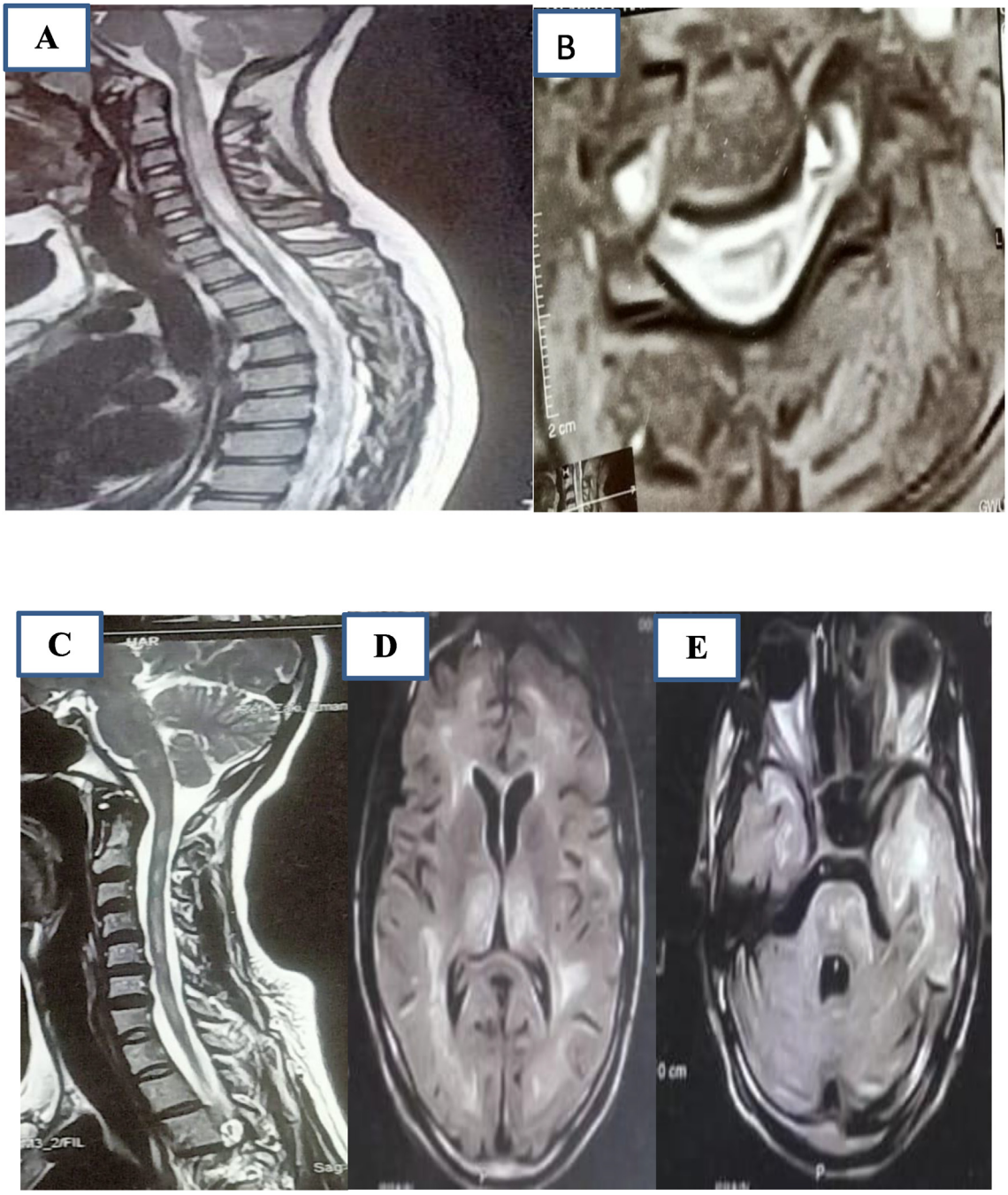

Fig. 4. A, B: Neuromyelitis Optica: Longitudinally extensive transverse myelitis. C, D, E: Acute disseminated encephalomyelitis: Cervical cord lesions and subcortical hyperintensities.

stretch reflexes may be diminished or even absent ('spinal shock syndrome'), raising the possibility of diagnostic confusion with Guillain-Barre syndrome (GBS). Clinically, spinal shock often lasts 4–6 weeks after an injury, ranging from days to weeks. Spasticity, hyperreflexia, and extensor plantar responses (i.e., the traditional symptoms of the upper motor neuron [UMN] syndrome) become more noticeable with time. These results were comparable to **Kayal *et al.*,**[7] study which showed that there was spasticity in 87 (57.6%) patients among patients with non-compressive myelopathies.

However, our results disagreed with **Thangaraj & v. R.,**[4] study which stated that there was more spasticity in 58 (77.33%) patients than the current study results, as most of their cases were chronic.

The present study showed that there were **cerebellar symptoms** in 12 (20.0%) patients, these

symptoms were due to previous attacks of demyelination in MS patients. Cerebellar symptoms occur in 50% of MS patients at some point throughout their disease. In **Thangaraj & v. R.,**[4] study, there were cerebellar signs in 11 (14.66%) patients.

In our study there were **urinary bladder dysfunction** in 38(63.3%) patients most of them were acute urine retention. **Bowel dysfunction** less common but still present in 12(20%) patients. presence of this symptoms early in the course of the disease confirm diagnosis of NCM.

Regarding **clinical features** in the present study, there were peripheral neuropathy 7(11.7%) patients, brain stem involvement in 5 (8.3%) patients, flexor spasm in 5 (8.3%) patients, encephalopathy in 3 (5.0%) patients, dystonia in 1 (1.7%) patient, radiculopathy in 1 (1.7%) patient. the small percentage of these symptoms are consistent with myelopathy. Our study agreed with **Thangaraj & v. R.,**[4] and **Kayal *et al.***[7]

In the current study, **MRI spine** showed short segment in 43 (71.7%) patients, LETM in 10 (16.7%) patients and Gadolinium enhancement in 10(16.7%) patients. The cervical or the cervicodorsal cord was affected in 44 (73.3%) patients. short segments which is typical of our MS patients, while long segments in NMO patients. Regarding MRI issue in the study by **Kamble *et a*l.,**[12] In 14 and 18 individuals, respectively, the spine revealed long segment lesions and short segment lesions. Similar outcomes have been seen in earlier Indian studies of NMOSD **Barhate *et a*l.**[9] Unexpectedly, Kamble and colleagues found long segment lesions in both of their multiple sclerosis patients.

CSF analysis is performed to confirm an inflammatory process and to look for possible infectious etiologies by serologic assessments, polymerase chain reaction studies for specific organism and to differential diagnosis of MS and NMO. In our study, CSF was done in 27 patients, and there was +ve OCB and high IGg index in 13 (21.7%) patients. The most reliable factor leading to the first diagnosis of idiopathic ATM seems to be the lack of OCB. The existence of OCB is very indicative of this etiology, even while their absence does not rule out MS. According to the 2017 update of the McDonald's criteria, positive OCB are now a time dissemination condition for MS. Patients initially diagnosed with an idiopathic ATM may eventually develop MS despite a negative OCB.

In **Prabhakar *et al.***,[8] study, CSF was studied in 23 patients out of 31 cases of ATM. OCB was present in 4 out of 14 patients (28%) in the ATM group.

In our study infectious cause of Acute Transvers Myelitis was VZV, CMV and EPV. The neurological abnormalities in these individuals first manifested 7–12 days following the commencement of the rash, although the latent phase may last up to several months.

In our current study, **the diagnosis** of non-compressive myelopathy was classified as MS spectrum, which was the most often reported diagnosis, then idiopathic ATM, HSP, NMO spectrum, infections/para infections (viral myelitis), and ADEM. The increase in demyelinating cases diagnosis in our study is due to the presence of MS unit in our hospitals, increase of awareness of demyelinating diseases, and the availability of the disease modifying therapy of MS. An observational study that investigated the clinical, radiographic, and cerebrospinal fluid profile of non-compressive myelopathy in Kota revealed that 52% of the patients had an underlying etiology such as demyelinating, infectious/post-infectious, autoimmune, or nutritional **Kamble *et al.***,[12] study.

In **Shukla *et al.***,[13] study, 28 cases were categorized under the causes of non-compressive myelopathy in India. Myelitis was the most common cause, which was seen most in isolated cases (50% patients) followed by association with MS, NMO, subacute combined degeneration of the spinal cord and ADEM.

Matching with our results, the study of **Zakaria *et al.***,[14] in Egypt about the evaluation of the clinical criteria and enhanced MR images in diagnosis of acute transverse myelitis. In this study they focused only on MRI findings to differentiate between extramedullary and intramedullary causes.

Also, **Kimbrough *et al.***,[15] investigated the indicators of transverse myelitis recurrence after a first episode in various racial groups. The goal of the research was to determine whether variables raised the probability of a subsequent transverse myelitis (TM) presentation.

In the current trial, patients with ATM received oral prednisolone 60 mg (or 1–1.5 mg/kg) per day tapered over a one-month period after receiving intravenous methylprednisolone 1 g (or 25 mg/kg) for 5 days. For patients that did not respond to steroids, plasmapheresis was the next step. Particular antibiotics or antivirals were used to treat patients with infectious myelopathies. Symptomatic treatment and disease modifying therapy were used after discharge.

There was highly statistically **significant improvement** in mRS after 3 months and more improvement after 6 months than after admission, this improvement because of immediate diagnosis and starting protocol of treatment and follow up. Our result agreed with **Bhowmick *et al.***[16], also, likewise, In **Thangaraj & v. R.**[4].

4.1. Conclusion

According to the finding in the present study, a wide range of diseases present with non-compressive myelopathies. In the majority of cases, the definitive diagnosis can be reached with a thorough history and clinical examination along with the proper workup. Our diagnostic precision has improved because of neuroradiology and serological indicators. Therefore, a proper treatment can improve the outcomes of this disease.

Authorship

All authors have a substantial contribution to the article.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

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Conflicts of interest

The authors declared that there were NO conflicts of Interest.

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