European Journal of Molecular & Clinical Medicine

ISSN 2515-8260 Volume 09, Issue 07, 2022

ORIGINAL RESEARCH

Study of spectrum of non-compressive myelopathy at a tertiary care hospital

Vilas Shingare[1], Karale M S[2], Yogesh Rasal[3]

[1]Assistant Professor, Department of General Medicine, Smt. Kashibai Navale Medical College, Pune, Maharashtra, India.
[2]Associate Professor, Department of Medicine, Vilasrao Deshmukh Government Medical College, Latur, Maharashtra, India.
[3]Assistant Professor, Department of General Medicine, Smt. Kashibai Navale Medical College, Pune, Maharashtra, India.

ABSTRACT

Background: Myelopathies result in motor, sensory and autonomic disability thereby restricting patients mobility and lead to a poor quality of life. Non-compressive myelopathy encompasses various diseases that are intrinsic to the cord and are considered to have primarily inflammatory, infectious and vascular etiologies. Present study was aimed to discuss patients of non-compressive myelopathy admitted at our tertiary care hospital.

Material and Methods: Present study was single-center, prospective, observational study, conducted in patients admitted in wards/ICU OR attending the outpatient department with signs and symptoms of myelopathy, labelled as a case of non-compressive myelopathy.

Results: 28 patients of non-compressive myelopathy were studied. Majority cases were from 31-40 years age group (39.29 %) & 41-50 years age group (28.57 %), were male (60.71 %) & had chronic course of illness (67.86 %).Common Motor manifestations observed were quadriparesis (71.43 %), paraparesis (39.29 %), spasticity (35.71 %) & flexor spasms (7.14 %). While common sensory manifestations noted were sphincter involvement (71.43 %), posterior column sensory loss (57.14 %), paresthesia (53.57 %), spinothalamic sensory loss (46.43 %) & peripheral neuropathy (14.29 %). Acute transverse myelitis (35.71 %) was most common etiology, followed by multiple sclerosis (14.29 %), vitamin B12 deficiency (14.29 %), tubercular (7.14 %), neuromyelitis optica (7.14 %) & vascular (3.57 %). In 5 cases exact etiology could not be made out (17.86 %).

Conclusion: In cases of non-compressive myelopathies, detailed history and clinical examination coupled with neuroradiological imaging and serological markers have increased our diagnostic accuracy.

Keywords: non-compressive myelopathies, transverse myelitis, quadriparesis, multiple sclerosis

Corresponding Author: Dr.Karale M S, Associate Professor, Department of Medicine, Vilasrao Deshmukh Government Medical College, Latur, Maharashtra, India.
Email:drmskarale13@gmail.com

INTRODUCTION

The term myelopathy describes pathologic conditions that cause spinal cord, meningeal or perimeningeal space damage or dysfunction. Myelopathies result in motor ,sensory and autonomic disability thereby restricting patients mobility and lead to a poor quality of life.[1] The role of MRI is important to distinguish compressive from non-compressive myelopathy. Once compressive lesions have been excluded, non-compressive causes of acute Myelopathy that are intrinsic to the cord are considered primarily vascular, inflammatory, and infectious etiologies.[2]

Non-compressive myelopathy encompasses various diseases such as acute transverse myelitis, primary demyelinating disorder such as multiple sclerosis and neuromyelitis optica, subacute combined degeneration of the cord, HIV myelopathy, radiation and toxin induced myelopathy and degenerative diseases of the cord.[3]

Dysfunction of ascending and descending axons and local neural circuits is reflected by various myelopathic signs and symptoms. Spinal cord diseases often have devastating consequences, ranging from quadriplegia and paraplegia to severe sensory deficits. Many of these diseases are potentially reversible if recognized and treated at an early stage.[4] Present case series was aimed to discuss patients of non-compressive myelopathy admitted at our tertiary care hospital.

MATERIAL AND METHODS

Present study was single-center, prospective, observational study, conducted in Department of General Medicine, at Smt. Kashibai Navale Medical College, Pune, India. Study duration was of 2 years (January 2020 to December 2021). The study was ethically approved by Institutional Ethical Committee.

Inclusion criteria

- Patients admitted in wards/ICU OR attending the outpatient department with signs and symptoms of myelopathy, labelled as a case of non-compressive myelopathy.

Exclusion criteria

- Patients who did not undergo magnetic resonance imaging (MRI) of the spinal cord
- Patients with spinal cord compression on MRI, anterior horn cell involvement in Japanese encephalitis
- Patients with motor neuron disease (MND), degenerative ataxias.

Study was explained to patients/close relatives in local language & written consent was taken for participation & study. All patients underwent relevant routine biochemical analysis including complete hemogram, liver function tests, renal function tests serum electrolytes, thyroid profile, urinalysis and appropriate neuroimaging studies (MRI of spine with contrast / MRI of brain). Whenever required, additional investigations such as HIV, VDRL, ESR, X-ray chest, collagen disease profile (ANA, RA factor, anti-dsDNA, serum vitamin B12 level, copper level, angiotensin converting enzyme (ACE) levels, cerebrospinal fluid (CSF), additional radiological investigations (USG or CT abdomen) & visual evoked potentials (VEPs) were done.

Data was collected and compiled using Microsoft Excel, analysed using SPSS 23.0 version. Statistical analysis was done using descriptive statistics.

RESULTS

28 patients of non-compressive myelopathy were studied. Majority cases were from 31-40 years age group (39.29 %) & 41-50 years age group (28.57 %), were male (60.71 %) & had chronic course of illness (67.86 %).

Table 1: General characteristics

Characteristics	No. of patients	Percentage
Age groups (in years)		
≤20	2	7.14%
21-30	7	25.00%
31-40	11	39.29%
41-50	8	28.57%
Mean age (mean±SD)	41.35 ± 8.46	
Gender		
Female	11	39.29%
Male	17	60.71%
Duration		
Acute	4	14.29%
Chronic	19	67.86%
Relapsing	3	10.71%
Subacute	2	7.14%

Common Motor manifestations observed were quadriparesis (71.43 %), paraparesis (39.29 %), spasticity (35.71 %) & flexor spasms (7.14 %). While common sensory manifestations noted were sphincter involvement (71.43 %), posterior column sensory loss (57.14 %), paresthesia (53.57 %), spinothalamic sensory loss (46.43 %) & peripheral neuropathy (14.29 %).

Table 2: Clinical features

Clinical features	No. of patients	Percentage
Motor manifestations		
Quadriparesis	20	71.43%
Paraparesis	11	39.29%
Spasticity	10	35.71%
Flexor spasms	2	7.14%
Bibrachial weakness	1	3.57%
Hemiparesis	1	3.57%
Sensory manifestations		
Sphincter involvement	20	71.43%
Posterior column sensory loss	16	57.14%
Paresthesia	15	53.57%
Spinothalamic sensory loss	13	46.43%
Peripheral neuropathy	4	14.29%
Optic neuritis/atrophy	1	3.57%

Acute transverse myelitis (35.71 %) was most common etiology, followed by multiple sclerosis (14.29 %), vitamin B12 deficiency (14.29 %), tubercular (7.14 %), neuromyelitis optica (7.14 %) & vascular (3.57 %). In 5 cases exact etiology could not be made out (17.86 %).

Table 3: Etiological profile of Non-compressive myelopathy

Etiology	No. of patients	Percentage
Acute transverse myelitis	10	35.71%
Unknown	5	17.86%
Multiple sclerosis	4	14.29%
Vitamin B12 deficiency	4	14.29%

Tubercular	2	7.14%
Neuromyelitis optica	2	7.14%
Vascular	1	3.57%

DISCUSSION

The history, neurological examination and the study of the cerebrospinal fluid guide the diagnosis of spinal cord injuries. However, imaging is of great importance in order to diagnose and classify the aetiology appropriately. In addition to the pattern of clinical presentation, analysis of certain features on MRI may help to narrow down the differential diagnosis. These features include distribution and location of the abnormal signal, the longitudinal extent of cord involvement (short segment or long segment), the cross-sectional area involved, and the enhancement pattern.[5] Transverse Myelitis (TM) is characterised by sudden onset of bilateral sensory, motor and autonomic dysfunction with a well-defined sensory level, in the absence of a pre-existing neurologic or systemic disease.[6]

Singh R et al.,[7] studied 73 patients (M:F:50:23), the mean age was 32 years. Etiology was established in 66 (90.4%) cases and those were acute transverse myelitis (ATM) (post infectious) (41.1%); multiple sclerosis (13.7%); Vitamin B12 deficiency (12.3%); tubercular myelitis (8.2%); neuromyelitis optica (NMO) (5.5%), sarcoidosis (4%), vascular (1.3%); hereditary spastic paraplegia (1.3%); post-radiation myelitis (1.3%), and post lightening myelitis (1.3%).

In study by Yavnika Jain et al.,[8] among 70 patients, 50 (71.4 %) were males, mean age of presentation was 31.95 years. Onset of illness was acute to sub-acute in nature in all the cases. Bladder and bowel involvement was seen in maximum cases (78.6 %). Patients found to have long segment myelitis (71.4 %), short segment myelitis (18.5 %) and spinal cord without changes (10 %). Demyelinating illness was most common in occurrence (28.6 %) comprising 10 NMO positive (14.3 %), 8 MOG positive (11.4 %) and 2 multiple sclerosis cases (2.8 %). Six cases (8.6 %) each of tubercular myelitis and post infectious (non-tubercular) ATM including 1 case (1.4 %) of post herpetic myelitis were found along with 5 cases (7.1 %) of subacute combined degeneration and 2 cases (2.9 %) of spinal AVM (Arterio-venous malformation) 31 (44.3 %) cases remained undiagnosed.

M. Thangaraj,[9] studied 75 patients with a median age of 34.5 years and male: female ratio of 1.35:1. Presentation was acute in 10 patients (13%), subacute in 5 (6.5%), chronic in 54 (72.5%) and history of relapse and remission in 6(8%) patients. Degenerative etiology was found for 42 (56%) others were non degenerative (demyelinating, autoimmune, vascular, nutritional, or physical agent). MRI study carried out in all cases showed signal changes in 51 cases (68%) which included myelomalacia, demyelination, atrophy of cord, infarction of cord.

Kayal AK et al.,[10] studied 151 patients [96 acute‐to‐subacute myelopathy (ASM) and 55 chronic myelopathy (CM)] with a median age of 35 years and male: female ratio 1.4:1. The causes of ASM were neuromyelitis optica spectrum disorder (23), multiple sclerosis (MS) (8), postinfectious myelitis (8) & infections (9) while causes of CM were probable or possible sarcoidosis (7), infections (9), Vitamin B12 deficiency (4) & radiation (11).. No etiology could be found in 48 (31.8%) patients (34 ASM and 14 CM). In 21/96 (21.9%) patients of ASM, acute transverse myelitis was idiopathic based on current diagnostic criteria.

Kamble et al.,[11] studied 80 patients of non-compressive myelopathies, out of which 44 had acute-subacute and 36 had chronic myelopathies. Etiological spectrum of acute to sub-acute myelopathies suggested post infectious myelitis to be the most common, followed by NMO spectrum disorders (NMOSD) and Multiple sclerosis (MS) where as in chronic myelopathies, vitamin B12 deficiency was most often seen. A noteworthy finding in this study was of the large number of cases of post-infectious myelitis due to dengue virus.

Myelopathy can cause considerable morbidity due to either paraparesis or quadriparesis. Magnetic resonance imaging (MRI) is the mainstay in evaluation of myelopathy. It has improved imaging of the spinal cord lesions to a point that reliable diagnosis of even a non-expansible lesion is routinely possible.[12]

The management strategies between compressive and non-compressive myelopathies differ dramatically, as compressive lesions usually require urgent neurosurgical intervention and decompression of the spinal cord, whereas non compressive myelopathies are usually amenable to medical treatment itself.[13,14]

CONCLUSION

In cases of non-compressive myelopathies, detailed history and clinical examination coupled with neuroradiological imaging and serological, CSF markers have increased our diagnostic accuracy to find the cause. Majority of these conditions are managed medically but have relapsing remitting sometimes progressive course if not treated early; hence early and efficient diagnosis of these myelopathies is important.

REFERENCES

1. Prabhakar S, Syal P, Singh P, Lal V, Khandelwal N, Das CP. Non‐compressive myelopathy: Clinical and radiological study. Neurol India 1999; 47:29‐9.
2. Mohan PK, Jaya KS, Anvitha M, Jaya TC. A study to find the various causes for compressive myelopathy. Int J Med Res Rev. 2020;8(3):247-251.
3. Kranz PG, Amrhein TJ (2019) Imaging approach to myelopathy: acute, subacute, and chronic. Radiol Clin North Am 57(2):257–279
4. Granados AM, Garcia L, Ortega C, Lopez A. Diagnostic approach to myelopathies. Rev ColombRadiol2011;22:1-21.
5. Lee MJ, Aronberg R, Manganaro MS et al (2019) Diagnostic approach to intrinsic abnormality of spinal cord signal intensity. Radiographics 39:1824–1839
6. Pekcevik Y, Mitchell CH, Mealy MA, Orman G, Lee IH, Newsome SD, et al. Differentiating neuromyelitisoptica from other causes of longitudinally extensive transverse myelitis on spinal magnetic resonance imaging. Mult Scler. 2016;22(3):302-11.
7. Singh R, Prasun N, Ahmad S. An evaluation of etiological and radiological profile of patients of non-compressive myelopathy in a neurological institute of Eastern India. Int J Med Sci Public Health 2017;6(10):1462-1466.
8. Yavnika Jain, Rajendra Singh Jain, Trilochan Srivastav, Etiologic Profile of Non-Compressive Myelopathy in a Large Tertiary Care Centre of North West Part of India, International Journal of Science and Research (IJSR), Volume 10 Issue 2, February 2021
9. M. Thangaraj, Jayasankar V. R. A study on etiological profile of non-compressive myelopathies in a tertiary care hospital in central Tamilnadu. International Journal of Contemporary Medical Research 2019;6(9):I4-I7
10. Kayal AK, Goswami M, Das M, Basumatary LJ, Bhowmick SS, Synmon B. Etiological profile of noncompressive myelopathies in a tertiary care hospital of Northeast India. Ann Indian AcadNeurol2017;20:41-50.
11. Kamble S, Sardana V, Maheshwari D, Bhushan B, Ojha P. Etiological Spectrum of Non Compressive Myelopathies in Tertiary Care Centre. J Assoc Physicians India 2019; 67:14-16.
12. Arjunprakash, Poojary SR, Ranganath R, Vedaraju KS. Evaluation of non-traumatic myelopathy using magnetic resonance imaging. IJRDI 2020;3:270-6.
13. Jankowitz BT, Gerszten PC. Decompression for cervical myelopathy. Spine Journal. 2006.

14. Schmalstieg WF, Weinshenker BG. Approach to acute or subacute myelopathy. Neurology. 2010;