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# Comparing SSEPs and MRI as Diagnostic Tools for Evaluating Compressive Myelopathies Caused by Disc Herniation

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**Abstract**: Compressive myelopathy is a neurological deficit that results from compression of the spinal cord and leads to a variety of symptoms and potentially serious consequences. Magnetic resonance imaging (MRI) is the gold standard for diagnosing compressive myelopathy as it provides high-resolution anatomical images and is the preferred method for evaluating the underlying pathology, but somatosensory evoked potentials (SSEPs) have also been used as a diagnostic tool as it provides information about the functional integrity of the spinal cord and assess the severity and extent of the damage. This article presents a comparative analysis of MRI and SSEPs in the diagnosis of compressive myelopathy and discusses the complementary role they can play in diagnosis and monitoring of the disease.

Seventy four subjects were involved in the study, divided into controls (35) and cases (39) of myelopathies due to disc herniation. Severity of myelopathy for cases group were classified according to modified Japanese Assessment scale (mJOA) into; mild, moderate and severe. SSEPs showed more correlation with severity of myelopathy more than MRI (r=0.948, 0.599 respectively) and higher sensitivity of SSEPs for detecting severe myelopathies (91.7%) while MRI sensitivity was only (16.7%) sensitive.

**Keywords**: SSEPs, spine MRI, Electrodiagnostics, compressive myelopathy.

## 1. Introduction

### 1.1 Definition

Compressive myelopathy refers to any neurological deficits that result from spinal cord compression and most commonly affects cervical spine segments. Causes could result sometimes in spinal canal stenosis including disc herniation as the most common cause followed by osteophytes, spinal osteochondroma, extradural mass, and/or paravertebral ligamentous ossification, trauma and aretiovenous malformation (AVM). Symptoms are variable according to lesion site and severity but mostly include motor deficit, pain/ sensory deficit, unbalance/ataxia and uncontrolled sphincters ([1]).

### 1.2 Diagnosis

A thorough physical exam and a detailed history is the first step in diagnosis. Myelopathy symptoms are not exclusive. Spine MRI is the gold standard modality for diagnosis of myelopathy because of its superior soft-tissue resolution and multiplanar capability, making it ideal for evaluation of the spinal canal and its contents as well as the surrounding bony and soft-tissue structures to evaluate compressive pathologies ([2, 3]).

Electrodiagnostics are helpful to determine the precise nerve root that is involved in pathogenesis. Somatosensory evoked potentials (SSEPs) have been routinely used over the years to evaluate the somatosensory pathway and thereby supplement the diagnostic process when the history, neurologic examination, and imaging were not fully conclusive and for localization of lesion site ([4]).

### 1.3 Clinical assessment

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There are several functional disability measures can be used to assess severity of myelopathies such as Cooper myelopathy Scale, Nurick Scale, European Myelopathy Score (EMS), modified Japanese Orthopedic Association Score (mJOA), Myelopathy Disability Index (MDI) and other scores.

These scores involve the assessment of motor function of upper and lower limbs, sensory level and sphincter control with grading system, the lower the grade, the more severe deficit.

The mJOA score is the score most often used in published literatures. The Nurick score mainly emphases on gait dysfunction ([5]).Mild myelopathy is defined as a mJOA sore ranging from 15 to 17, moderate myelopathy from 12 to 14 and severe myelopathy as a mJOA from 0 to 11 ([6]).

## 2. Materials and Methods

Our study involved 74 subjects; 35 healthy controls (their mean age 37.74 ± 15.84) and 39 patients (their mean age 42.02 ±15.24). Patients were classified according to mJOA scoring system into mild, moderate and severe.

Whole spine MRI and SSEPs (median and tibial SSEPs bilaterally) were done for all subjects and their results were compared according to their relation to mJOA score. We used grading system to evaluate spinal cord compression in disc herniation; grade 0: no compression, grade 1: slight compression, cord width decrease by <1/3, grade 2: moderate compression, cord width decrease by >1/3, grade 3: severe compression, cord width decrease by >2/3 ([7]).

NCS and EMG tests were performed to exclude peripheral neuropathy that either could affect the diagnosis or SEPPs results. Control group were examined to establish normative laboratory data for SSEPs results. Blood investigations such as complete blood test, thyroid function test, A1C, Erythrocyte sedimentation rate (ESR), C-Reactive protein (CRP) to exclude any chronic disease might affect peripheral nerves. CT spine and myelography were also done to confirm myelopathy for some cases. Brain imaging was performed to exclude concomitant cerebral lesion.

## 3. Results

This study included 74 subjects; 35 controls (19 males and 16 females) and 39 cases (25 males and 14 females) as shown in table 1.

**Table 1: Demographic features of case and control groups.**

| parameters | | Cases (no. = 39) | Controls (no. = 35) |
|---|---|---|---|
| Sex | male | 25 (53%) | 19 (56%) |
| | female | 14 (47%) | 16 (44%) |
| Age (years) | | 42.02 ±15.24 | 37.74 ± 15.84 |

Both age and disease duration in table (2) showed high significant difference among mJOA categories (p-value= 0.012, 0.027 respectively, since severe myelopathies affected much older patients and had more chronic disease course.

**Table 2: results of age and disease duration for patients according to mJOA severity.**

| Parameters | | Age | Disease duration |
|---|---|---|---|
| Mild N=8 | Mean | 31.25 ± 14.310 | 0.77 ± 0.619 |
| | Median | 23.50 | 9 months |
| | Minimum | 18 | 4 weeks |
| | Maximum | 55 | 2 years |
| Moderate N= 19 | Mean | 46.05 ± 11.173 | 1.978421 ± 1.022 |
| | Median | 48.00 | 1 year |

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| | | | |
|---|---|---|---|
| | Minimum | 24 | 2 months |
| | Maximum | 69 | 4 years |
| Severe N= 12 | Mean | 53.17 ± 14.825 | 2.916667 ± 2.83 |
| | Median | 52.00 | 2.5 years |
| | Minimum | 20 | 2 months |
| | Maximum | 73 | 10 years |
| P= value | | 0.012 | 0.027 |

**Non-parametric two sample Kruskal–Wallis test for continual data and significant difference is considered if p<0.05.**

Figure (1) displays relationship between site of herniated disc site and mJOA severity, where only 8 patients with disc herniation had mild score and 19 patients had moderate mJOA score while 12 patients had severe mJOA.

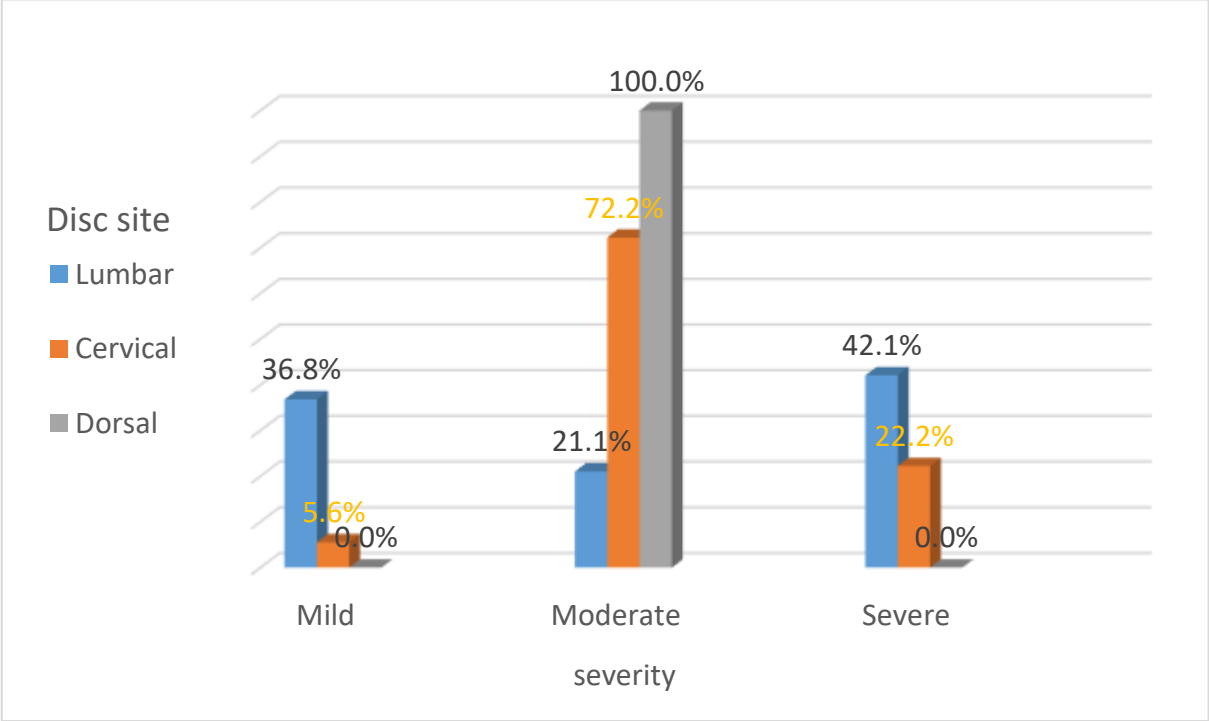


**Figure 1: Distribution of site of herniated discs according to mJOA.**

On spine MRI, 5 cases of mild mJOA were identified as grade 0 (62.5%) and 3 cases as grade 1 (37.5%) as shown in figure 2, meanwhile 10 cases of moderate mJOA had grade 1 (52.6%) and 9 cases had grade 2 (47.4%). MRI was less consistent with mJOA in severe cases; 33.3% as grade 1, 50% as grade 2, and only 16.7% as grade 3 (severe compression).

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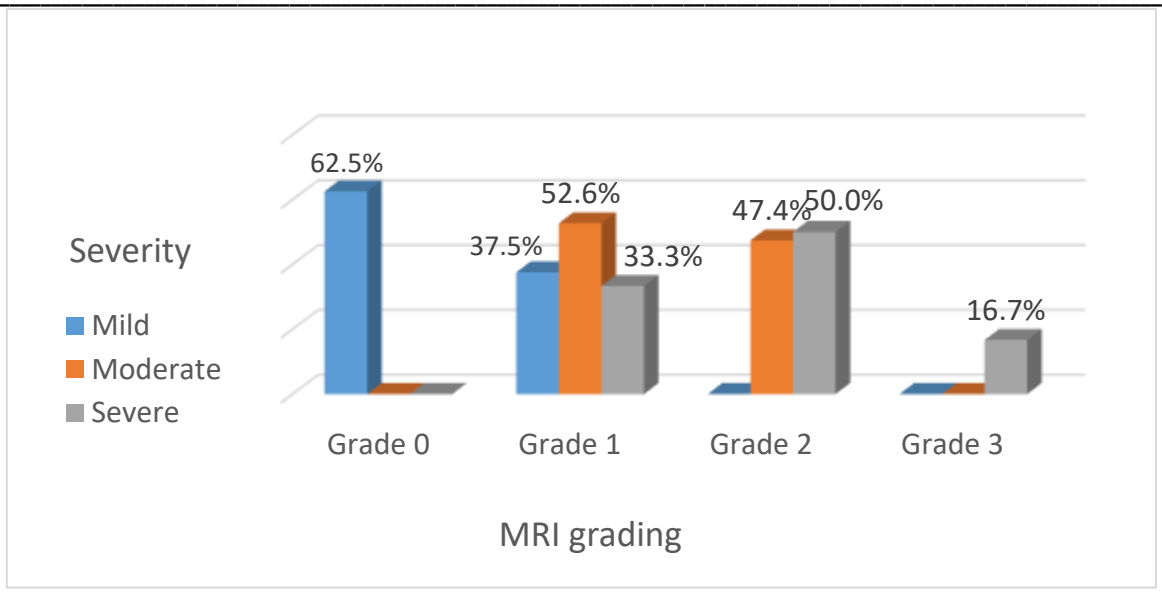


**Figure 2: Distribution of MRI grading in patients with disc herniation according to mJOA. Grade 0: no compression, grade 1: slight compression, grade2: moderate compression and grade 3: severe compression.**

In our study, SSEPs conduction delay is classified into two categories; 1. Prolonged conduction, 2. Absent/ poor waveform (Ab/ PWF). Latencies and interpeak latencies considered prolonged when exceed mean+ 3SD of normative laboratory data. Ab/PWF considered when the latencies corresponding to the clinical lesion site is absent or exceed the analysis time set on machine. Difference in latencies noticed during the study for patients with moderate and severe myelopathy. N20 for cervical lesions on median nerve SSEPs showed highly significant difference (p-value< 0.05) between these latencies and mJOA severity (tables 3). For mild cases, median =24.4ms, min.= 24.4ms and max.= 24.4ms; for moderate cases, median=28.02ms, min.= 24.8ms and max.= 29.2ms while in severe cases, median= 37.4ms, min.=34.4ms and max.=39.2ms; p=0.001.

**Table 3: Comparing N20 latency by median SSEPs with mJOA severity**

| mJOA | N20 | | | | | p-value |
|---|---|---|---|---|---|---|
| | N | Mean ± SD | Median | Min. | Max. | |
| **Mild** | 8 | 24.4 | 24.40 | 24.40 | 24.40 | 0.001 |
| **Moderate** | 19 | 27.71± 1.56 | 28.05 | 24.8 | 29.2 | |
| **Severe** | 12 | 36.20 ± 2.94 | 37.40 | 34.40 | 39.20 | |

**Non-parametric, Kruskal–Wallis test for continual data and significant difference is considered if p<0.05.**

P37 latency (table 4) was obtained by tibial nerve SSEPs (for mild cases, median= 38.9ms, min. = 36.7ms and max. = 40.3ms; for moderate cases, median= 48.6ms, min. = 44.3ms and max. = 55ms while in severe cases, median= 62.9ms, min= 58ms and max. = 67.4ms; p=0.0001).

**Table 4: Comparing P37 latency by tibial SSEPs with mJOA severity.**

| mJOA | P37 | | | | | P-value |
|---|---|---|---|---|---|---|
| | N | Mean ± SD | Median (ms) | Min. (ms) | Max. (ms) | |
| **Mild** | 8 | 38.89 ± 1.37 | 38.90 | 36.70 | 40.30 | 0.0001 |
| **Moderate** | 19 | 47.59 ± 2.29 | 48.60 | 44.30 | 50.00 | |
| **Severe** | 12 | 62.68 ± 2.75 | 62.90 | 58.00 | 67.40 | |

**Non-parametric, Kruskal–Wallis test for continual data and significant difference is considered if p<0.05.**

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SSEPs was least consistent (in table 5) with mJOA in mild cases (diagnosed only 12.5% as prolonged conduction) while 100% of moderate cases had prolonged conduction and in severe cases, SSEPs was more consistent with mJOA, 91.75% diagnosed as Ab/PWF.

**Table 5: Relationship between myelopathy severity and SSEPs conduction type.**

| SSEPs conduction severity | mJOA (clinical assessment) | | |
|---|---|---|---|
| | Mild (n=8) | Moderate (n=19) | Severe (n=12) |
| Normal | 7 (87.5%) | 0 | 0 |
| prolonged | 1 (12.5%) | 19 (100.0%) | 1 (8.3%) |
| Ab/PWF | 0 | 0 | 11 (91.7%) |

***Percentage from column total**

By using spearman's correlation (table 6) for all patients, SSEPs showed more positive correlation with mJOA rather than MRI (r=0.948, 0.599 respectively). SSEPs showed higher sensitivty in detecting severe myelopathies (91.7%) in comparison with MRI only (16.7%)

**Table 6: Correlation of mJOA with MRI and SSEPs severity**

| Study tools | mJOA | |
|---|---|---|
| | R | P-value |
| MRI | 0.599 | 0.0001 |
| SSEPs | 0.948 | 0.0001 |

[*] **Spearman's correlation coefficient**

## 4. Discussion:

The most frequent cause of compressive myelopathy is related to degenerative diseases of the spine and the subsequent common etiologies are trauma and spinal cord compression due to extradural masses caused by metastatic disease to bone or primary neoplastic ([8]). Myelopathy had variable mJOA score depending on compression severity and clinical presentation.

Age and disease duration were significantly different among patients and affecting mainly severe myelopathies, probably due to the chronicity of illness that leads to spinal cord edema and ultimately to spinal cord deformity therefore with time, myelopathies become more severe since the spinal cord has limited neuroplasticity compared to the brain. Unlike the brain, which has a high degree of plasticity and can reorganize itself in response to new experiences or injuries, the spinal cord is more fixed in its organization and has limited ability to regenerate damaged nerve cells or rewire neural circuits. While there is some evidence that the spinal cord can undergo some degree of plasticity, such as changes in the strength of synaptic connections between neurons, these changes are generally limited and are unlikely to result in significant functional recovery following injury. ([9,10]). MRI was least feasible for patients with disc herniation since only 16.7% of severe myelopathy was classified as severe (grade 3). This is could be attributed to the technical sections of the MRI that may miss some parts of the herniated discs. ([11]).

SSEPs sensitivity studies have been conducted for a variety of illnesses, and aberrant conduction has been addressed as either prolonged conduction or Absent/poor waveform (Ab/PWF). In our research, when compared to mild and moderate myelopathies, severe myelopathies showed a noteworthy pattern of extremely delayed latencies. SSEPs showed abnormality in only 12.5% of mild myelopathies while all moderate and severe myelopathies were abnormal on SSEPs (either median or tibial or both). Probably due to that the mild compression of herniated disc not affecting the spinal cord to the degree that can be detected by SSEPs. Whereas all moderate myelopathies had prolonged conduction (100%) and 91.7% of severe cases

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had Ab/PWF. A study enrolled in Baghdad over 22 patients with Spondylotic cervical myelopathy (SCM), 86.36% of patients had abnormal SSEPs study (either tibial or median or both tests), in which absent or prolonged N20 and N13-N20 were the most recurrent abnormalities for median SEPPs and absent or prolonged P37 and LP-P37 were most common abnormalities in tibial SSEPs. Results showed that normal SSEPs findings mostly interrelated with mild and early myelopathy (grade-1 and grade-2 Nurick) ([12]).

Table 6 shows the overall correlation between MRI, SSEPs and mJOA by using spearman's correlation test (r=0.948, 0.599 respectively). After conducting numerous correlation investigations, Feng et al., 2020 ([13]) found a substantial (r = 0.94) correlation between the incidence of progressive myelopathy and the SEPPs classification for the upper SEPPs. While MRI is generally the imaging of choice for compressive lesions, Nardone et al., 2016 ([14]) stated that neuroimaging is not always completely accurate for establishing a diagnosis, and the functional involvement of the spinal cord cannot be assessed by MRI.

## 5. Conclusion

Myelopathy is a diagnostic dilemma that necessitates several investigation tools. Both imaging techniques and neurophysiological diagnostic tools are evolving and together can assist for further analysis of site and severity of the spinal cord lesion. In our study, SSEPs showed prolonged poor/absent waveform in 91.7% of severe myelopathies while MRI identified severe compression in only 16.7% of severe myelopathies and SSEPs correlated more with clinical severity rather than MRI since imaging detects structural abnormalities and does not necessarily correlate with functional disability while SSEPs detects functional abnormality and till nowadays no studies established to categorize conduction delay in SSEPs.

We recommend to perform SSEPs for all patients with compressive myelopathies along with radiological modalities especially for patients with disc herniation and scheduled for surgery.

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