Section A -Research paper

Diagnosis of Cervical Spondylotic Myelopathy

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Abstract:

Cervical spondylotic myelopathy (CSM) is a disorder with an increasing prevalence. CSM is an impaired function of the spinal cord caused by degenerative changes in the cervical spine that results in compression of the spinal cord. Symptoms of CSM are the instability of gait, loss of fine motor control of the upper limbs, numbness in the hands, weakness of the hands and legs, neck pain and stiffness, and urinary emergency. The vague nature of early myelopathic symptoms is often responsible for the diagnostic delay. The diagnosis of CSM is based on the patient's history and clinical examination. Additional diagnostic studies, such as radiographs, magnetic resonance imaging, computed tomography, bone scan, electromyography, somatosensory evoked potentials, and motor evoked potentials, can help to provide further information useful for the management of the patient. The differential diagnosis includes any condition associated with neck pain, arm pain, motor-sensory-reflex changes, and signs of spinal cord dysfunction.

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Introduction:

The true natural history of cervical spondylotic myelopathy is difficult to discern. The disease process progresses in a variable and unpredictable manner. Often there is stepwise deterioration of neurologic function, with periods of stable symptoms followed by decline. The clinical course may wax and wane over a period of years. Sensory symptoms may be transient, but motor symptoms tend to persist and progress **(1).**

Clinical evaluation:

CSM may present with variable clinical findings depending on the levels affected and involvement of the neural foramina and long tracts. A variety of neurological symptoms and signs may be present **(2).**

Symptoms:

The patient may complain of one or combination of the following**:**

Difficulty with fine motor movements and tasks with hands:

Loss of the hand co-ordination that is manifested by inability to distinguish items such as coins in one pocket, or inability to perform delicate tasks such as tying shoes or buttoning a shirt. Complaint of difficulty with writing or an unexplained change in hand writing is also common. Patients may report that they cannot tell the temperature of the water, although they can tell that their hand is submersed **(2).**

Neck pain:

This may be due to local facet pain, discogenic pain or local muscular pain. Occipital headache may be present **(2).**

Gait abnormalities:

Ambulation is slow, shuffling, and the patient frequently requires the use of external supports in walking and then gait worsening occurs later in the course of the disease because of the dorsal

columns become affected. Gait changes are the most important clinical predictor **(3).**

Loss of bladder or bowel sphincter control:

In advanced cases of CSM, patients may have bladder or bowel impairment symptoms that are usually associated for poor prognosis for recovery. Urinary symptoms vary and may consist of urinary frequency, urgency, incontinence or urinary retention **(3, 4).**

Arm pain:

It is burning pain in upper extremities due to one or multiple root affection; it may give neck, shoulder and possible arm pain and numbness. Concomitant arm pain is one of the most common presenting symptoms and may be seen in more than 40 % of patients with CSM. The presence of concomitant arm pain can influence both treating decision making as well as surgical planning **(3).**

Physical evaluation:

Neck examination:

The cervical spine range of motion should be examined actively, passively and against resistance. Asymptomatic or pain-producing limitation of motion is evidence of motion segment(s) spondylosis **(5).**

Gait and balance:

It is wide-based, hesitant, stiff, or spastic. Many tests can be done for gait such as:

Toe-to-heel walk: patient has difficulty to perform it or poor balance during toe raises **(5).**

Romberg test: patient stands with arms held forward and eyes closed loss of balance occur and this is consistent with posterior column dysfunction **(5).**

Neurological evaluation:

Motor: Assessment of motor system by**;**

- **Inspection and palpation of muscles:** By completely exposing the patient while keeping the patient's comfort and dignity, look for asymmetry for both proximal and distal muscle groups, deformities, wasting or hypertrophy, fasciculation and involuntary movements such as dystonia, chorea and athetosis **(5).**
- **Assessment of tone:** Tone is the resistance felt by the examiner when moving a joint passively. This is done at both upper and lower limbs. Detect if there is hypotonia or hypertonia.
- **Hypotonia** occur in LMNL and is usually associated with muscle wasting, weakness and hyporeflexia. **Hypertonia** is of two types spasticity and rigidity **(5).**
- **Spasticity** is velocity-dependent resistance to passive movement. Detected with quick movements and is a feature of UMNL. Usually accompanied by weakness, hyperreflexia, an extensor planter response and sometimes clonus. In mild forms it is detected as a 'catch' at the beginning or end of passive movement. In severe cases it limits range of movement **(5).**
- **Rigidity** is a sustained resistance throughout the range of movement and most easily detected when the limb is moved slowly. In Parkinsonism this is classically described as 'lead pipe rigidity' **(5).**
- **In CSM,** depending on the level of the cord damage, there is hypertonia in the lower extremities in the form of spasticity (as an UMNL) and hypotonia in the upper extremities **(5).**

Testing movement and power:

Muscle strength varies with age and fitness. Cranial nerves should be intact unless there is coexisting pathology above the foramen magnum. Weakness may be present in the upper extremities, lower extremities or both. In the upper extremities, mixed upper and lower motor neuron findings may be present depending on the level of cord damage. Flaccid weakness due to upper motor neuron damage might be present at the level of the lesion whereas spastic weakness would be expected below the lesion. For example, cord compression at C5-C6 level, biceps and suppinators are flaccidly weak whereas triceps (C7) exhibit spastic weakness. Weakness of hand muscles is also common in CSM, and the fifth digit may abduct spontaneously due to intrinsic muscle weakness. Atrophy may be present if the myelopathy is long-standing. When weakness is present in the lower extremity, spastic weakness is expected due to corticospinal tract damage**(5).**

The muscles commonly to be tested for weakness in both upper limbs and lower limbs are:

Table (1): Showing movements commonly tested in both upper and lower limbs **(2).**

Movement	UMN	Root	Reflex	Nerve	Muscle
Upper limb					
Shoulder abduction	++	C5		Axillary	Deltoid
Elbow flexion		C5/6	+	Musculocutaneous	Biceps
		C6	+	Radial	Brachioradialis
Elbow extension	+	C7	+	Radial	Triceps
Radial wrist extension	+	C6		Radial	Extensor carpi radialis longus
Finger extension	+	C7		Posterior interosseus nerve	Extensor digitorum communis
Finger flexion		C8	+	Anterior interosseus nerve	Flexor pollicis longus + Flexor digitorum profundus (index)
				Ulnar	Flexor digitorum profundus (ring + little)
Finger abduction	++	T1		Ulnar	First dorsal interosseous
		T1		Median	Abductor pollicis brevis
Lower limb					
Hip flexion	++	L1/2			Iliopsoas
Hip adduction		L2/3	+	Obturator	Adductors
Hip extension		L5/S1		Sciatic	Gluteus maximus
Knee flexion	+	S1		Sciatic	Hamstrings
Knee extension		L3/4	+	Femoral	Quadriceps
Ankle dorsiflexion	++	L4		Deep peroneal	Tibialis anterior
Ankle eversion		L5/S1		Superficial peroneal	Peronei
Ankle plantarflexion		S1/S2	+	Tibial	Gastrocnemius, soleus
Big toe extension		L5		Deep peroneal	Extensor hallucis longus

The muscle tested can be classified in a certain grade which is used for grading the muscle strength.

Table (2): Grading of muscle power **(2).**

Motor grade	*Findings*
Grade 0	No muscle contraction visible
Grade 1	Flicker of contraction but no movement
Grade 2	Full range of motion (ROM) with gravity eliminated
Grade 3	Full ROM against gravity alone
Grade 4	Full ROM against partial resistance
Grade 5	Full ROM against full resistance

Sensory evaluation:

Light touch:

In cervical spondylotic myelopathy this may be affected due to involvement of ventral spino-thalamic tract **(5).**

Superficial pain:

Use a fresh neurological pin, e.g. neurotip, not a hypodermic needle and dispose of the pin after each patient to avoid transmitting infection. Map out the boundaries of any area of reduced, absent or increased sensation and compare with dermatomal map **(as in Fig. 1)** and move from reduced to higher sensibility (i.e. from hypoaesthesia to normal, or from normal to hyperaesthesia) **(5).**

In cervical spondylotic myelopathy there may be global decrease in sensation or radicular distribution in selective dermatomes **(5).**

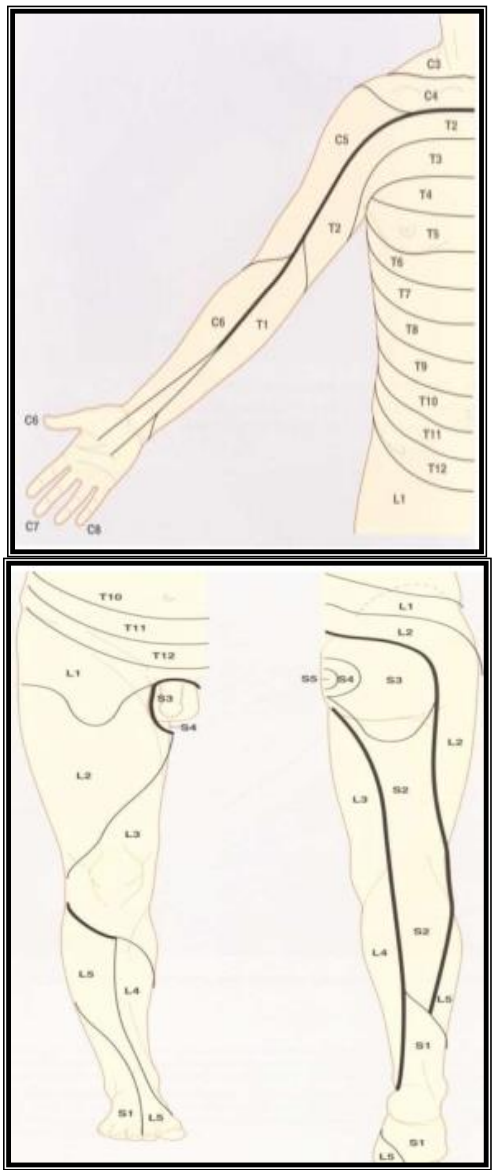

Fig. (1): Dermatomal map for both upper and lower limbs. **(2).**

Temperature:

Touch the patient with a cold metallic object, e.g. tuning fork, and ask if he feels cold **(6).**

Vibration:

Place a vibrating 128-hz tunning fork over the sternum and ask the patient if he feel it buzzing. Then place it on the tip of the great toe. If sensation is impaired, place the fork on the inter-phalangeal joint and progress proximally, to the medial malleolus, tibial tuberosity and anterior iliac spine depending on the response. Repeat the process in the upper limbs. In cervical spondylotic myelopathy this may be affected in severe cases of long standing myelopathy **(6).**

Joint position sense:

Ask the patient to close his eyes and to identify the direction of small movements in random order in the big toe. In cervical spondylotic myelopathy may be present proprioception dysfunction due to dorsal column involvement and this occurs in advanced disease and associated with a poor prognosis **(6).**

Reflexes:

Deep tendon reflexes:

A tendon reflex is the involuntary contraction of a muscle in response to stretch. It is mediated by a reflex arc consisting of an afferent (sensory) and an efferent (motor) neurone with one synapse between (a monosynaptic reflex). These stretch reflex arcs are served by a particular spinal cord segment which is modified by descending upper motor neurons **(as shown in table 3; 7).**

Table (3): Showing monosynaptic (deep tendon) reflexes and root innervations **(2).**

Reflex (muscle)	***Nerve root***
Biceps	C5
Brachioradialis	C6
Triceps	C7
Knee (patellar tendon)	L3-L4
Medial hamstring	L5
Ankle (Achilles' tendon)	S1

Ensure that both limbs are positioned identically with the same amount of stretch. Compare each reflex with the other side by checking for symmetry of response on both sides. When the reflex are tested strike the tendon not the muscle or bone. The response may be increased, normal, diminished, present only with reinforcement or absent. Hyperreflexia (abnormally brisk reflexes) is a sign of upper motor neurone lesions (UMNL). Diminished or absent jerks are most commonly due to lower motor neurone lesions (LMNL) **(7).**

Use reinforcement whenever a reflex appears absent. For knee and ankle reflexes, ask the patient to interlock the fingers and pull one hand against the other on your command, immediately before you strike the tendon (Jendrassik's manuveur). To reinforce upper limb reflexes, ask the patient to clench the teeth or to make a fist with the contralateral hand. The patient should relax between repeated attempts and strike the tendon immediately after your command to the patient **(7).**

Hoffmann's reflex:

Place your right index finger under the distal interphalangeal joint of the patient's middle finger. Use your right thumb to flick the patient's finger downwards. Look for any reflex flexion of the patient's thumb. It is evidence of an upper motor neuron lesion. In the normal patient, the Hoffman's sign is absent. Recently, a dynamic Hoffman's sign is suggested by adding a dynamic element to the Hoffman's sign, which may increase the ability of the clinician to detect early spinal cord involvement in cervical spondylosis. It was noted that if the Hoffman's sign was checked during active flexion and extension of the cervical spine as tolerated by the patient, that the sign could be elicited in subjects that did not elicit the sign in the static, resting position **(7).**

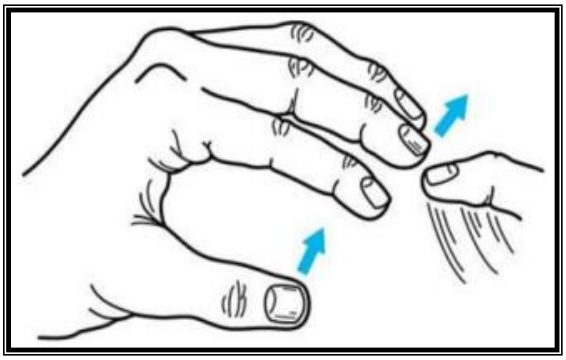

Fig. (2): Hoffmann's reflex. **(8).**

Finger jerk:

Place your middle and index fingers across the palmer surface of the patient's proximal phalanges. Tap your own fingers with the hammer. Watch for flexion of the patient's fingers **(9)**.

Superficial reflexes:

This group of reflexes is polysynaptic and elicited by cutaneous stimulation rather than stretch.

Planter response (S1-2):

Run a blunt object along the lateral border of the sole of the foot towards the little toe **(Fig. 3).** Watch both the first movement of the great toe and the other leg flexor muscles. The normal response is flexion of the great toe with flexion of the other toes **(9)**.

A true Babinski sign: involves activation of the extensor hallucis longus tendon leading to extension of the big toe and fanning of the other toes (not movement of the entire foot, a common withdrawal response to an unpleasant stimulus)**(2).**

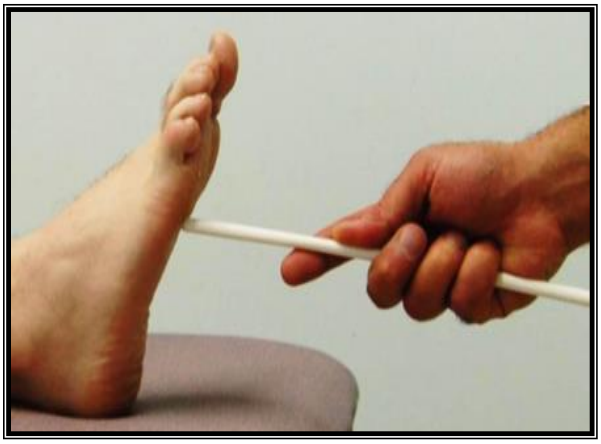

Fig. (3): Babiniski (planter) response **(2).**

Abdominal reflexes (T8-12):

The patient should be supine and relaxed. Use a blunt object and briskly, but lightly, stroke the upper and lower quadrants of the abdomen in a medial direction. The normal response is contraction of the underlying muscle, with the umbilicus moving laterally and up or down depending upon the quadrant tested toward this quadrant. For T8–10 stroke above umbilicus and for T10-12 stroke below umbilicus. Absence of this reflex may indicate UMNL **(9)**.

Cremastric reflex (L1-2): In males only

Explain what you are going to do and why it is necessary. Abduct and externally rotate the patient's thigh. Use a blunt object to stroke the upper medial aspect of the thigh. Normally the testis on the side stimulated will rise briskly **(9)**.

Myelopathic signs:

Finger escape sign:

When patient holds fingers extended and adducted, the small finger spontaneously drifts into abduction and flexion due to weakness of intrinsic muscle **(Fig. 4) (10).**

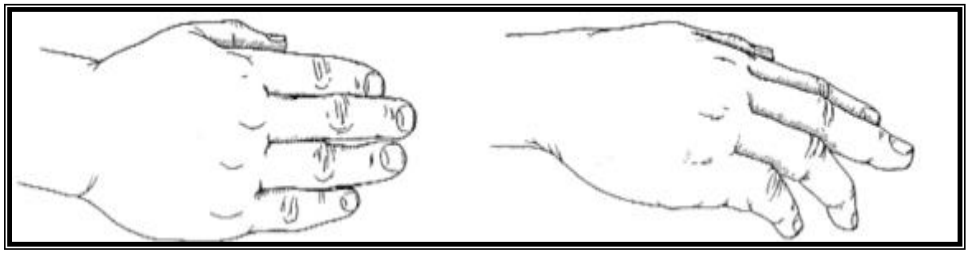

Fig. (4): Finger escape sign. **(11).**

Normally a patient can make a fist and release 20 times in 10 seconds. Myelopathic patients may struggle to do this **(Fig. 5) (10).**

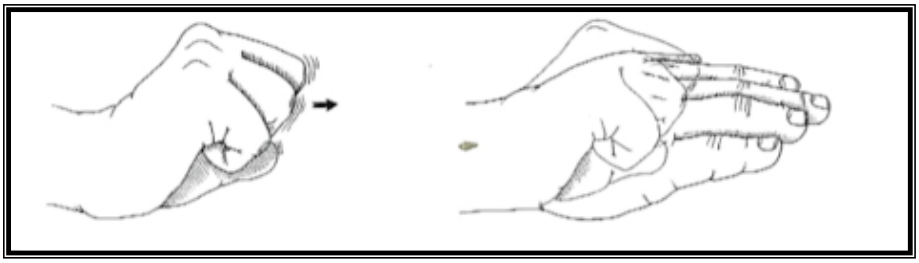

Fig. (5): Grip and release test. **(11).**

Myelopathy hand:

Ebara et al., described the myelopathy hand associated with cervical spondylotic myelopathy as one in which there is diffuse or localized muscle wasting of the extrinsic or intrinsic hand muscles **(Fig. 6).** The muscle weakness, often appearing as a deficiency of extension of the fingers, was mostly seen unilaterally. If the patient presented with bilateral involvement, there was much discrepancy between the right and left hands. Sensory findings in these patients were disproportionate to motor findings, most often being minimal or absent. The myelopathic hand patients most often had sagittal cervical spinal canal diameters below 13 millimeters and multi-segmental spondylosis **(10).**

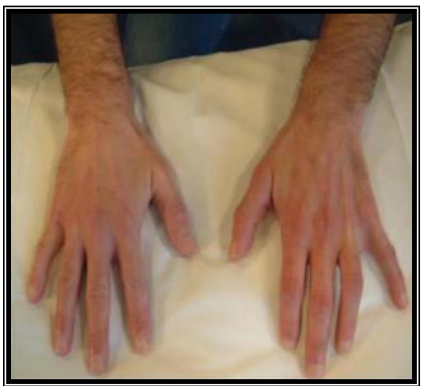

Fig. (6): Myelopathic hand **(2).**

Inverted radial reflex:

The inverted radial reflex may be present when cord and nerve root compression are present at the C5 and C6 levels. The reflex is seen during testing of the brachioradialis reflex. As the brachioradialis tendon is struck with the reflex hammer at the distal end of the radius, a diminished response is noted in the brachioradialis along with a reflex contraction of the spastic finger flexors, hence the term "inverted radial reflex" **(12).**

Sustained ankle clonus:

Support the patient's leg, with both the knee and ankle resting in 90 degree flexion. Briskly dorsiflex and partially evert the foot, sustaining the pressure. Clonus is felt as repeated beats of dorsiflexion/planter flexion. It indicates upper motor neuron damage and is accompanied by spasticity **(Fig. 7) (9)**.

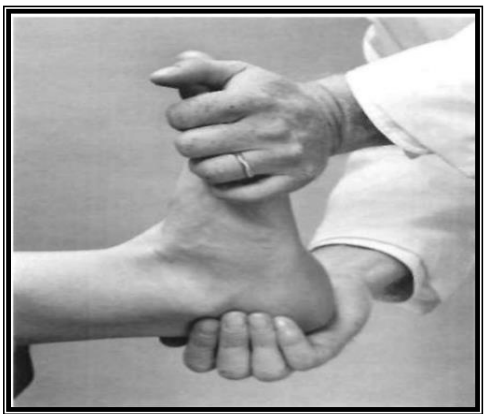

Fig. (7): Ankle clonus. **(8).**

Babiniski test:

Considered positive with extension of great toe and fanning of the other toes **(9)**.

L'hermitte's Sign:

A test is positive when extreme cervical and hip flexion in a seated patient leads to electric shock-like sensations that radiate down the spine and into the extremities **(9)**.

Grading and assessment for CSM:

Many grading systems are used for grading of CSM such as:

1. **Nurick grading:**

Based on gait and ambulatory function **(10).**

Table (4): Show Nurick grading **(13).**

Grade 0	Root symptoms only or normal
Grade 1	Signs of cord compression; normal gait
Grade 2	Gait difficulties but fully employed
Grade 3	Gait difficulties prevent employment, walks unassisted
Grade 4	Unable to walk without assistance
Grade 5	Wheelchair of bedbound

2. **Ranawat grading:**

Table (5): Show Ranawat grading **(14).**

Class I	Pain, no neurologic deficit
Class II	Subjective weakness, hyperreflexia, dyssthesias
Class IIIA	Objective weakness, long tract signs, ambulatory
Class IIIB	Objective weakness, long tract signs, non-ambulatory

3. Modified Japanese Orthopaedic Association (mJOA) functional grading scale:

A point scoring system (18 totals) based on function in the following categories:

- Upper extremity motor function.
- Lower extremity motor function.
- Sensory function in upper extremities.
- Bladder function.**(9)**

Table (6): Modified Japanese Orthopedic Association (mJOA) Criteria for the evaluation of operative results in Patients with Cervical Myelopathy **(9).**

Motor dysfunction score of the upper extremities	
0	Inability to move hands
1	Inability to eat with a spoon, but able to move hands
2	Inability to button shirt, but able to eat with a spoon
3	Able to button shirt with great difficulty
4	Able to button shirt with slight difficulty
5	No dysfunction
Motor dysfunction score of the lower extremities	
0	Complete loss of motor and sensory function
1	Sensory preservation without ability to move legs
2	Able to move legs, but unable to walk
3	Able to walk on flat floor with a walking aid (i.e., cane or crutch)
4	Able to walk up and/or down stairs with hand rail
5	Moderate to significant lack of stability, but able to walk up and/or down stairs without hand rail
6	Mild lack of stability but walks with smooth reciprocation unaided
7	No dysfunction
Sensory dysfunction score of the upper extremities	
0	Complete loss of hand sensation
1	Severe sensory loss or pain
2	Mild sensory loss
3	No sensory loss
Sphincter dysfunction score	
0	Inability to micturate voluntarily
1	Marked difficulty with micturition
2	Mild to moderate difficulty with micturition
3	Normal micturition

This scoring system differs from that of the original Japanese Orthopedic Association in that it assesses only motor dysfunction in the upper and lower extremities, sensory function in the upper extremities, and bladder function, and does not include a scale for sensory function in the trunk and lower extremities. Each scale ranges from 0 to 5, 7, 3, and 3, respectively, with a total score of 0 to 18.

Kasahata (9) defined the severity of myelopathy as mild if the mJOA score is 15 or larger, moderate if the mJOA score ranges from 12 to 14 or severe if the mJOA score is less than 12. This scale focused on the use of a spoon instead of chopsticks to evaluate motor function in the upper extremities.

4. Visual Analog Scale for assessment of axial neck pain:

Visual Analog Scale for pain is an assessment tool consisting of a 10 cm line with 0 on one end, representing no pain, and 10 on the other end representing the worst pain ever

experienced, which a patient marks to indicate the severity of his or her pain **(as in Fig. 8). (9)**.

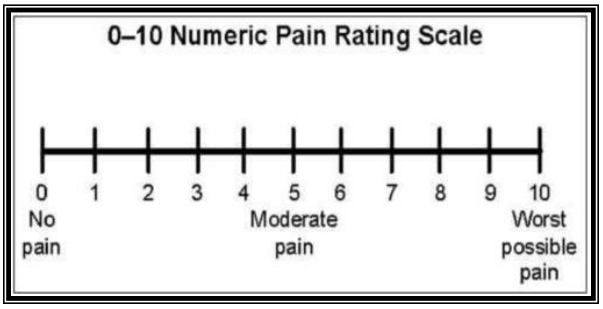

Fig. (8): Visual Analog Scale **(2).**

Investigation:

1. Plain x-ray.
2. MRI.
3. CT.
4. CT Myelography.
5. Nerve conduction study.

1. Plain x-ray:

The recommended views of the plain x-rays are anteroposterior, lateral, oblique, flexion, and extension views (dynamic view)**.** The general findings that may be found are degenerative changes of uncovertebral and facet joints, osteophyte formation and disc space narrowing**(15).**

On the lateral radiograph of the plain x-rays of the cervical spine look for:

- **Pavlov ratio:**

The antero-posterior diameter of the spinal canal in the cervical region should be approximately equal to the anteroposterior diameter of the vertebral body at that level. Thus, a normal canal should have a ratio of approximately 1:1. The ratio of the spinal canal to the vertebral body is the distance from the mid-point of the posterior aspect of the vertebral body to the nearest point on the corresponding spino-laminar junction line divided by the antero-posterior width of the vertebral body. Utilizing this method, a measurement of less than 0.80 indicates significant stenosis, as normal spines demonstrate a measurement closer to 1.00**.** So the ratio of canal size to vertebral body size useful in assessing patients at risk for spinal cord injury due to a stenotic spinal canal **(15).**

- **Sagittal alignment:**

Measuring sagittal alignment by C2 to C7 alignment which is determined by tangential lines on the posterior edge of the C2 and C7 body on lateral radiographs in neutral position **(15).**

- **Local kyphosis angle:**

The angle is measured on lateral x-rays obtained in the neutral and flexion positions between the two lines at the posterior margin of the most cranial and caudal vertebral bodies forming maximal kyphosis through C2 to C7**.** The Interpretation is normal cervical spine alignment in the sagittal plane is in lordosis, and a kyphosis angle higher than 10 degree is associated with a higher chance of cervical myelopathy **(15).**

Oblique radiograph is important to look for foraminal stenosis which often caused by uncovertebral joint arthrosis **(16).**

Flexion and extension views are important to look for angular or translational instability, compensatory sublaxation above or below the spondylotic/stiff segment **(15).**

2. Magnetic Resonance Imaging (MRI):

MRI is study of choice to evaluate degree of spinal cord and nerve root compression. MRI is of value in assessing the soft tissue structures associated with the cervical region such as cervical discs, facets, cervical tumors, intradural lesions, ossification of the ligamentum flavum, assessment of spinal cord signal intensity and assessment of anteroposterior compression ratio (APCR) **(17).**

- **Findings:**

Spinal cord signal intensity (SI) changes:

Signal intensity (SI) changes manifest as a high SI on T2- weighted images, a low SI on T1-weighted images, or both. A scale, **(presented in Table 7)** was developed by **Avadhani et al., (18).** To classify these MRI abnormalities into grades according to severity.

Table (7): Classification of intra-medullary signal intensity**(19)**

Grade	*Description*
Grade I (N/N)	Normal intensity on both T1 and T2 weighted image
Grade II (N/Hi)	No intra-medullary signal intensity abnormality on T1 weighted image, with high intra-medullary signal on T2 weighted image
Grade III (Lo/Hi)	Low intra-medullary signal intensity on T1 weighted image and high intra-medullary signal intensity on T2 weighted image

Changes in SI on either T1 or T2 weighted images are usually sufficient to confirm the diagnosis of CSM. Increased intra-medullary SI on T2 weighted images (cord malachia) represents diffuse neuronal cell loss, gliosis, edema, demyelination, axonal and spongy degeneration in the white matter, and is a sign of advanced spinal cord damage **(18).**

SI changes on MRI are associated with poorer neurological outcomes following surgery for CSM. Therefore, although this finding clarifies the diagnosis, it appears that the pathophysiological mechanisms underlying SI change are indicative of more severe, perhaps irreversible cord damage, and therefore an earlier diagnosis would be desirable. MRI evidence of spinal cord compression by indentation into the cord surface without SI changes is associated with a better outcome. Spinal cord compression without signal change was noted in 16% of cervical spine MRI scans in asymptomatic individuals under the age of 64 years, and in 26% of scans of those aged 65 years and over **(18).**

It is clear therefore that MRI cannot be relied upon in isolation. Findings on imaging must be correlated to the patient‟s clinical presentation **(18).**

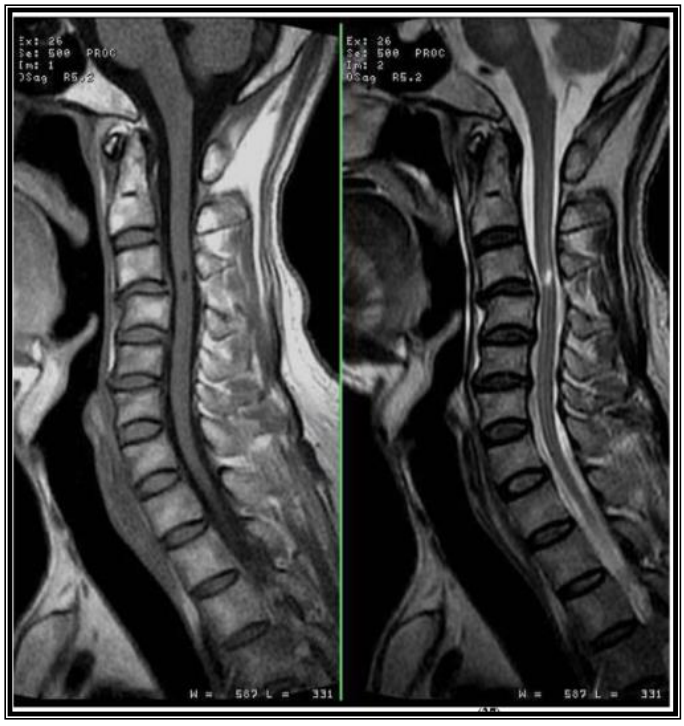

Fig. (9): T1 weighted image showing hypointense area opposite C3-C4 disc and Hyperintense in T2 weighted image (Signal intensity grade 3 on MRI) **(20).**

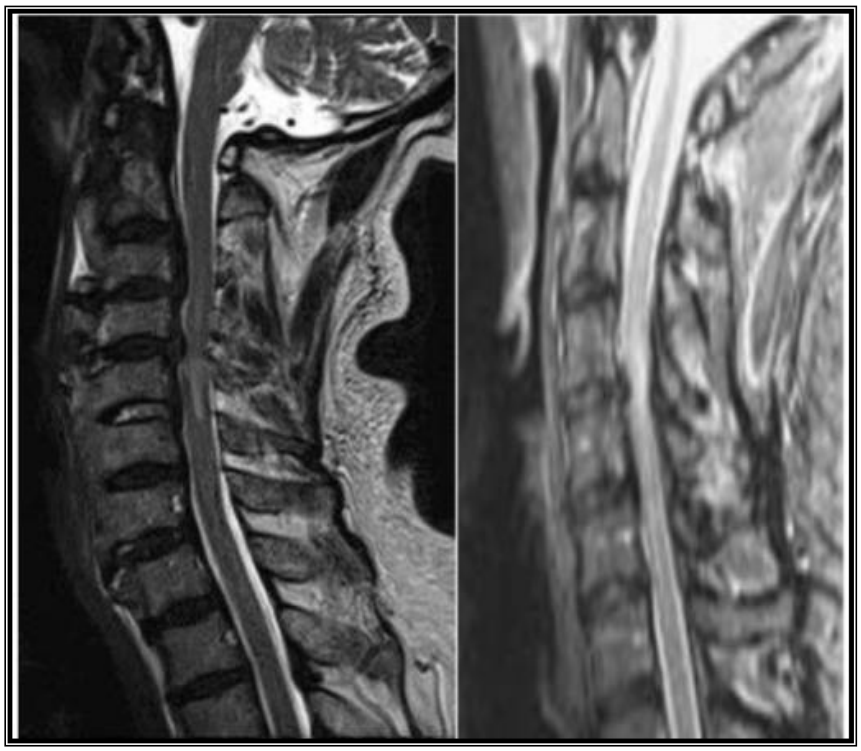

Fig. (10): Also signal intensity grade 3 on MRI. **(20).**

- **Ossification of ligamentum flavum :**

Ossification of the ligamentum flavum (OLF) is a rare disease and when causing myelopathy it affects essentially the thoracic spine. Only a few cases of symptomatic cervical disease have been reported, with all patients being Japanese **(21).**

- **Anteroposterior Compression ratio (APCR)**

The anteroposterior compression ratio (APCR) is a measurement of the cross-sectional area of the spinal cord. APCR = smallest AP diameter of cord / largest transverse diameter of cord. The normal APCR is at least 45%. Percentages for the APCR less than 40% are considered abnormal. The AP compression ratio has been studied by **Ogino**. He assessed the APCR at the most damaged spinal cord segment and found that when the APCR was 40%-45% (which is nearly in the normal range) that there was flattening of the gray matter of the spinal cord and mild, localized demyelination of the lateral corticospinal tracts. An APCR of 22%-39%, which is significantly reduced, was associated with rarefaction of the gray matter, neuronal loss in the gray matter, and diffuse demyelination of the lateral corticospinal tracts. When the APCR measured between 12% and 19% there was extensive gray matter necrosis. There was also significant necrosis and gliosis within the lateral columns and dorsal columns. So, anteroposterior compression ratio (APCR) of < 0.4 carries poor prognosis **(22).**

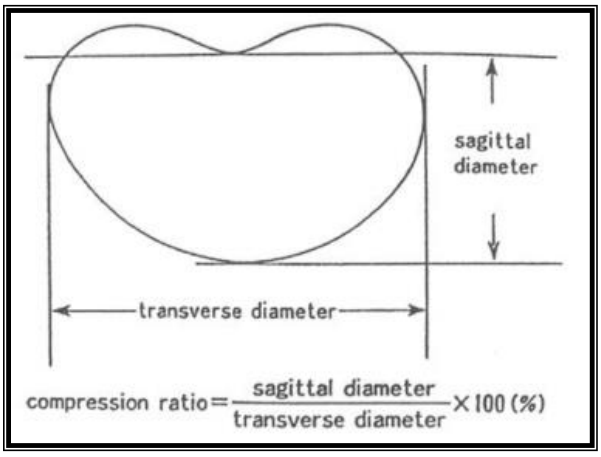

Fig. (11): Anteroposterior compression ratio (APCR). **(23).**

3. Computed Tomography (CT):

Can provide complementary information with an MRI and is more useful to evaluate OPLL, osteophytes and bony spondylotic spurs.

The slice just cranial to the disc space is the most informative slice **(24).**

Ossification of posterior longitudinal ligament (OPLL):

It is a common cause of cervical myelopathy in the Asian population. With 95% of ossification is located in cervical spine. OPLL is often associated with several other entities diffuse idiopathic skeletal hyperostosis (DISH), ossification of the ligamentum flavum (ossification of the yellow ligament (OYL) and ankylosing spondylitis **(21).**

Cervical OPLL can be classified roughly into 4 types: continuous, segmental, mixed or circumscribed (localized or others) **(21).**

4. CT Myelography:

More invasive than an MRI but gives excellent information regarding degree of spinal cord compression. Useful in patients that cannot have an MRI (pacemaker) or has artifact (local hardware). Contrast given via C1-C2 puncture and allowed to diffuse caudally or given via a lumbar puncture and allowed to diffuse proximally by putting patient in trendelenburg position**(24).**

- **Nerve conduction study:**

May be useful to distinguish peripheral from central causes.

Differential Diagnosis of CSM:

Not all patients with radiographic evidence of cervical spondylosis and a clinical syndrome suggesting involvement of the cervical cord have spondylotic myelopathy. Some may have amylotrophic lateral sclerosis, spinal cord tumor, or demyelinating diseases. Occasionally coexisting disease processes combine to produce symptoms and signs that are consistent with a single lesion**.** Because the differential diagnosis must consider these fine distinctions, patients with cervical spondylosis and neurological syndrome must be evaluated carefully and completely before therapy begins. The clinical course and its ultimate outcome are sufficiently unpredictable to warrant a cautious approach**(25).**

1. Neoplastic Disorders:

Neoplastic disorders can be classified into four categories depending upon whether the tumors are primarily extradural or intradural and upon the site of the origin**.** Primary tumors originating from vertebral bodies tend to be extradural. Benign primary tumors include giant cell tumors, osteochondromas, bone cysts and hemangiomas. The malignant bone tumors include the osteogenic sarcomas and chordomas. Metastatic cervical spine tumors occur more frequently than primary bone tumors. Metastatic tumors of the cervical spine will often present as osseous lesions of the spine with epidural extension and manifest themselves as severe pain which is constant and more intense at night, particularly when the patient assumes supine position. There may be associated tenderness over the vertebral bodies posteriorly **(26, 27).**

An intradural tumor of the cervical spine can have either an extra-medullary or intra-medullary origin. The extra-medullary intra-dural tumors include the meningiomas and benign neurofibroma or schwannomas. Intra-medullary neoplasms represent the ependymomas or gliomas of the spinal cord. Lymphosarcoma and Hodgkin's disease may present as lytic lesions of bone or an epidural mass which may rapidly progress to myelopathy**(27).**

The features which differentiate these tumors from cervical spondylosis include severe pain at rest and/or associated tenderness over the vertebral bodies with or without aggravation by cervical spine motion. On the diagnostic side, these patients will have elevated spinal fluid proteins with or without pleocytosis. The radiological studies show characteristic changes in the vertebral bodies and defects uncharacteristic for cervical spondylosis with root compression **(28).**

2. Inflammatory Disorders of the Cervical Spine:

Rheumatoid arthritis does not directly involve cervical roots. This disease causes laxity of the transverse ligaments resulting in, and thereby leading to, mal alignment of cervical vertebra, instability at the atlanto-axial junction or sub-axial levels and, thus, root impingement. Associated characteristic deformities in the limbs of rheumatoid arthritis eliminates any chance of confusing this disease with cervical

spondylosis, although the two conditions may coexist in the same patient. **(29).**

Ankylosing spondylitis involves the entire spine in an ascending fashion and result in ossification and erosion at ligamentous insertions and joints. The entire spine may become fused and osteoporotic (Bamboo spine) predisposing the patient to fracture and neurological deficit. The modified New York criteria to define ankylosing spondylitis are low back pain of 3 months' duration, lumbar stiffness, limited range of motion in frontal and sagittal planes, limited chest expansion, and bilateral or advanced unilateral sacroilitis **(30).**

Other conditions of cervical spine, which may present as a myelopathy include sarcoidosis and multiple sclerosis (unlike cervical myelopathy there is abnormal cranial nerves and jaw jerk)**(29).**

3. Bacterial, Viral, Parasitic, Fungal Infection:

They can involve the cervical spine, both bone and neural elements. The most common agent of non tuberculous osteomyelitis is staphylococcal aureus. Bacterial and fungal infection may cause epidural abscesses and discitis**.** Bacterial organisms can be introduced into the cervical spine via hematogenous or lymphatic routes, and a variety of viral disorders can directly affect the cervical nerve roots and/or spinal cord (transverse myelitis). Poliomyelitis is seldom encountered today **(31).**

In acquired immune deficiency syndrome (AIDS), a variety of spinal cord syndromes and peripheral neuropathy have been described. Cytomegalic virus (CMV) and herpes simplex virus (HSV) have been implicated as causes of myelitis in AIDS. Human T-lymphotropic virus type I (HTLV-I), a human retrovirus, has been etiologically implicated in tropical spastic paraparesis**(31).**

4. Degenerative disorders of cervical spine:

It includes many degenerative conditions such as:

Syringomyelia, hereditary spastic paraparesis, spinocerebellar atrophy and amyotrophic lateral sclerosis (ALS).

Amyotrophic Lateral Sclerosis:

Differentiating between cervical spondylitis myeloradiculopathy and amylotrophic lateral sclerosis may be especially challenging. Both tend to appear in old patients while almost all elderly patients have radiographic evidence of cervical spondylosis. Patients with amyotrophic lateral sclerosis usually can be distinguished from those with deficits from spondylosis alone on the basis of clinical findings particularly fasciculation and wasting in the lower as well as the upper extremities combined with EMG evidence of denervation **(32).**

5. Spinal cord infraction:

Its incidence is rare and most probably underrecognized and this is due to elatively rich collaterals of the spinal cord. It is often slowly progressive and disabiling with poor long term recovery **(33).**

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