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Pathophysiology, diagnosis, and management of cervical disc herniation

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ABSTRACT

Cervical disc herniation is a common cause of neck pain and neurologic symptoms with presentations ranging from asymptomatic to debilitating radiculopathy or myelopathy. Symptoms may arise when a vertebral disc herniates, causing inflammation and compression of the nerve root or spinal cord. Risk factors are multifactorial and including age, genetics, cardiometabolic and behavioral factors, occupational biomechanics, and trauma. Accurate diagnosis requires patient history, neurological findings, provocative maneuvers, and imaging. The majority of cases improve with nonsurgical care including medication, physical therapy, and activity modification. Surgical evaluation is indicated for cases involving myelopathy, progressive neurologic deficits, or chronic/treatment-refractory symptoms.

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KEYWORDS: Cervical disc herniation; Cervical radiculopathy; Cervical myelopathy; Conservative management; Neck pain

Introduction

Neck pain is among the most common causes for outpatient medical evaluation and remains a frequent source of diagnostic and therapeutic uncertainty. Although most cases are benign and self-limited, a subset reflects underlying cervical disc pathology. Cervical disc herniation is a clinically important contributor of neck and arm symptoms.[1,2] Although often asymptomatic, cervical disc herniation may produce cervical radiculopathy and/or myelopathy through inflammatory or compressive mechanisms.[1–3] Notably, neck pain may be minimal or absent, highlighting the discordance between symptoms, imaging findings, and pathology.[1,3,4]

This review provides a focused synthesis of contemporary literature on cervical disc herniation, with emphasis on pathophysiology, diagnostic evaluation, and evidence-based nonoperative management. Particular attention is given to clinical features and examination findings that distinguish disc-related pathology from nonspecific neck pain to guide appropriate escalation of care.

Methods

A structured PubMed (MEDLINE) search was performed for English-language articles published in the last 10 years. Key search terms included, but were not limited to, combinations of: "cervical disc herniation," "radiculopathy," "myelopathy," "diagnosis," "conservative management," and "imaging." Studies were included if they addressed at least one of the following domains: epidemiology, etiology, pathophysiology, diagnosis, management, or treatment of cervical disc herniation. Exclusion criteria included case reports and articles without a primary focus on cervical pathology. Titles, abstracts, and full texts were screened to determine eligibility, and selected articles were also examined to identify additional relevant material.

Background and disease overview

Epidemiology

Cervical disc herniation has a population incidence of approximately 0.8 to 1.8 per 1,000 person-years with an adult prevalence of 0.5-2%.[5] Peak occurrence is observed in the fourth to fifth decades of life with the age distribution

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spanning from the third decade onward.[1,3,6] Cervical disc herniation accounts for approximately 8% of spinal disc herniations, though true prevalence is likely higher due to asymptomatic cases.[7] Methodological heterogeneity—including variations in the diagnostic criteria, population sampling, and clinical setting—further complicates precise quantification. For instance, surgical case series typically report higher proportions, reflecting selection bias toward more severe, symptomatic presentations requiring medical intervention.

Demographically, sex and age influence the risk for cervical disc herniation.[1,8,9] In both sexes, cervical disc herniation prevalence increases with age,[1] with most diagnoses occurring between 51 and 60 years.[8,9] Nevertheless, the association between sex and cervical disc herniation remains unclear, with conflicting evidence on whether it is more common in men or women.[5,8,10]

Contemporary epidemiological understanding of cervical disc herniation is limited by the paucity of recent large-scale population studies with much of the existing literature predating recent societal and behavioral changes. Technology and shifts in physical activity patterns influence current cervical disc herniation patterns. Emerging evidence suggests that prolonged sedentary behaviors—sustained forward head posture associated with computer usage—may represent novel risk factors.[11] Evolving behavioral patterns necessitate new epidemiological investigations to accurately reflect the modern disease burden.

CLINICAL SIGNIFICANCE

- Most cervical disc herniations improve with conservative care; 80-85% resolve within 8-12 weeks without surgery.
- Diagnosis requires correlating history and examination with imaging because MRI abnormalities are frequently asymptomatic.
- Early recognition of myelopathy or progressive neurologic deficit is essential because these findings require urgent surgical evaluation.
- Stepwise nonoperative management including NSAIDs, physical therapy, and targeted injections can control radicular symptoms and restore function.

Cervical spine anatomy

The cervical spine is comprised of seven vertebrae (C1-C7) and six intervertebral discs (Fig. 1). Each disc sits between two adjacent vertebrae, acting as a cushion (Fig. 1). This cushioning allows for load sharing, shock attenuation, and multiplanar motion.

Each intervertebral disc is composed of annulus fibrosus (tough outer ring) and a nucleus pulposus (hydrated, gelatinous interior) (Fig. 2). The annulus fibrosus is composed of lamellae of collagen fibers, and encircles as well as protects the nucleus pulposus. The nucleus pulposus is composed of a loose network of fibers suspended in mucoprotein gel.

Disc displacement is either focal (herniation) or broad-based (bulge) (Fig. 3). Cervical disc herniation must be a *focal* displacement of disc material beyond the normal disc boundary. "Focal" denotes a localized outpouching (involving <25% of disc circumference—axial view) in contrast to a broad-based bulge (involving ≥25% of disc circumference). Cervical disc herniation may be morphologically classified as either a protrusion (typically contained) or an extrusion (can be contained or uncontained; if completely detached, the fragment is called a "sequestration") (Fig. 3).

Cervical disc herniation can produce cervical radiculopathy by compressing/irritating the exiting nerve root or dorsal root ganglion, and cervical myelopathy when herniated material narrows the spinal canal and compresses the cord. Cervical disc herniation most commonly occurs in the C5-C6 and C6-C7 levels.[1] In radiculopathy, if C5-C6 herniates, the C6 nerve root is compressed causing C6 symptoms.

Pathophysiology

Etiology & risk factors. Contemporary research supports a multifactorial etiology for cervical disc herniation and identified six main risk factor categories contributing to susceptibility: 1) degenerative biology,[12] 2) cardiometabolic and behavioral factors,[12] 3) occupational biomechanics of neck motion/posture,[12] 4) trauma,[1,2] 5) anthropometric characteristics,[12] and 6) comorbid medical conditions.[13,14] This model accounts for the observation that many symptomatic cervical disc herniation cases arise without identifiable precipitating events.

1) Degenerative biology

Two key degenerative risk factors for cervical disc herniation are aging and genetics. Aging drives disc degeneration through progressive biochemical and structural deterioration of the intervertebral discs.[2,12,15] Twin studies show genetics contribute 50-70% of disc degeneration phenotype, setting baseline vulnerability that environmental, behavioral, and mechanical factors later modify.[15]

2) Cardiometabolic & behavioral factors

Increased cervical disc herniation risk includes: cigarette smoking, adiposity, and diabetes mellitus.[12] These exposures aggravate a vulnerable disc rather than serving as solitary causative agents.

3) Occupational biomechanics

Historically, cervical disc herniation has been associated with repetitive neck flexion-extension or sustained non-neutral postures. Yet, large-scale prospective studies have failed to demonstrate significant associations between cumulative cervical motion and cervical disc herniation incidence.[13,16] Emerging evidence links prolonged forward-head posture during digital device use to cervical spine pathology, though the association warrants further investigation.[11]

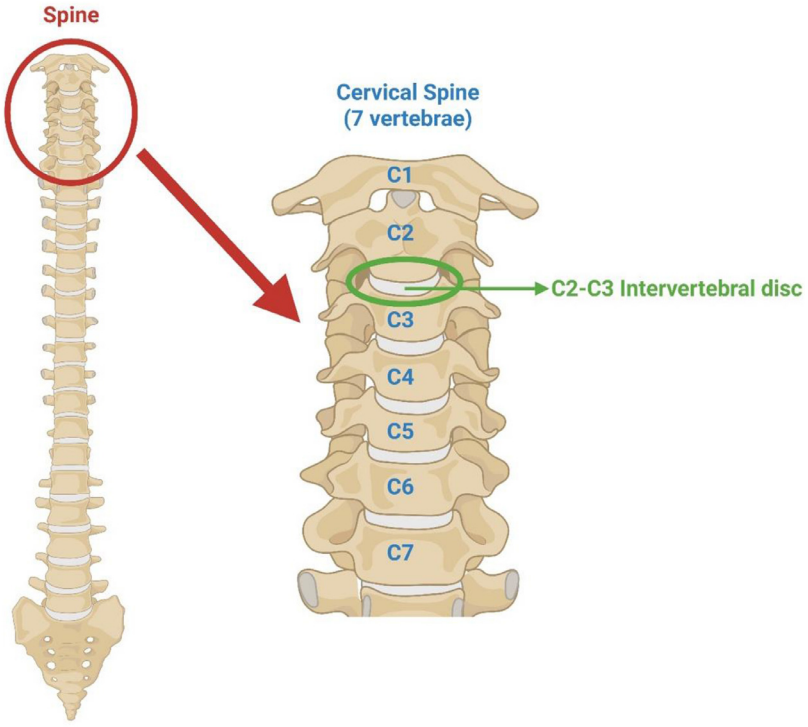

Fig. 1 The cervical region of the spine is enlarged to show the seven cervical vertebrae and the cervical intervertebral discs.

4) **Trauma**

Trauma may result in cervical disc herniation if annular disruption overwhelms the structural integrity of the annulus fibrosus and nuclear extrusion, particularly in already degenerated discs.[1,2,12] However, most cervical disc herniation occur in the absence of identifiable trauma.[17]

5) **Anthropometric/anatomic modifiers**

Anatomic variants and degenerative changes—congenitally narrow spinal canals, uncovertebral osteophytes, and spondylosis—increase the probability that a disc herniation becomes symptomatic.[4]

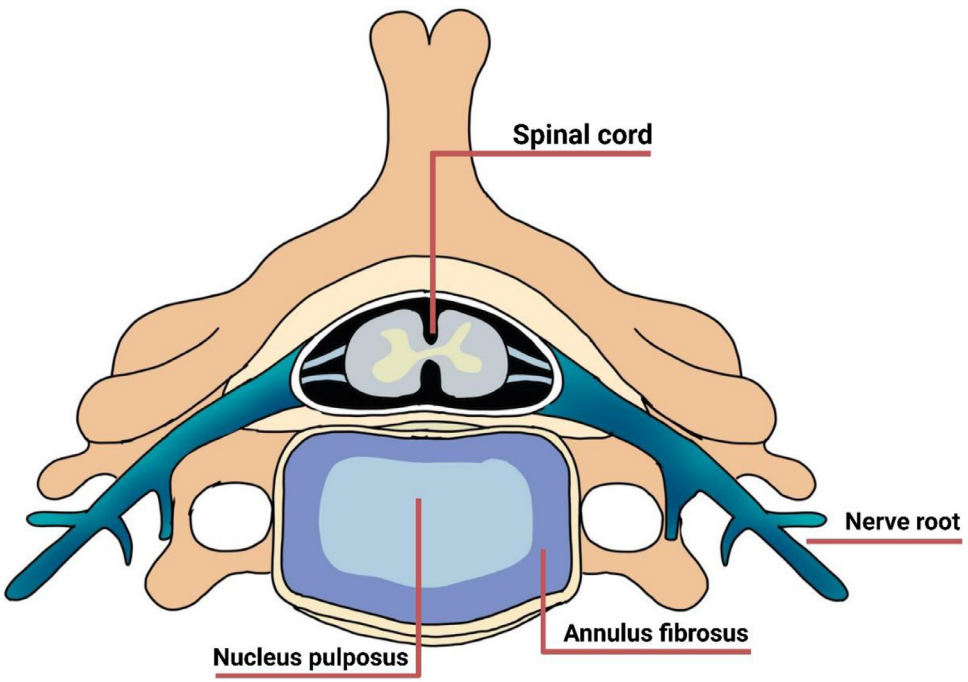

Fig. 2 Axial view of healthy cervical vertebra and intervertebral disc.

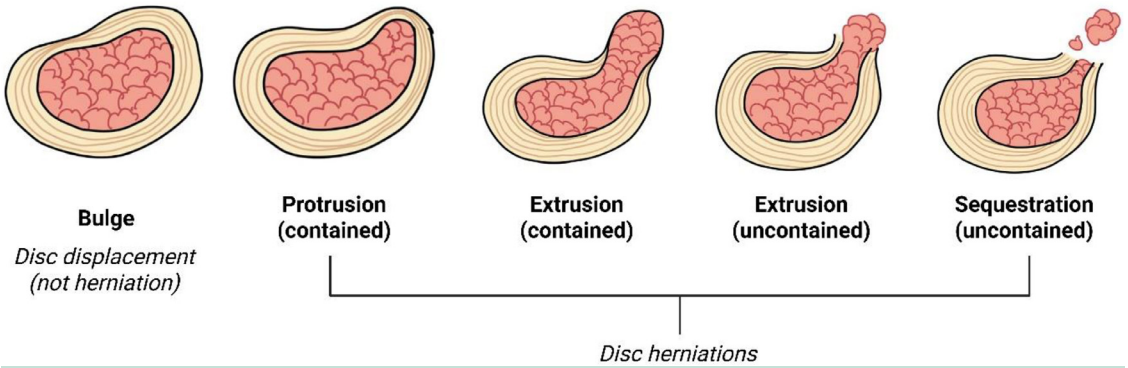

Fig. 3 Morphology of intervertebral disc displacement.

6) **Comorbid medical conditions**

Conditions affecting microvascular supply, matrix turnover, or chronic inflammation may accelerate cervical disc herniation degeneration and impair tissue repair, hindering recovery.[13,12]

Clinical manifestations: signs and symptoms

cervical disc herniation exists on a spectrum from asymptomatic to severely debilitating. The classic symptom triad consists of neck pain, radicular pain, and neurological dysfunction.[1,7] In radiculopathy, symptoms depend on disc level, herniation size, bulge direction, and affected structures.[1,2] Symptom duration varies: acute cases often resolve within 8-12 weeks, while some persist 4-6 months or become chronic, lasting over six months despite intervention.[1,17]

Most neck pain is mechanical, varying with position and load, and lacks radicular or myelopathic features.[1] Cervical disc herniation pain localizes to the posterior or posterolateral cervical region, affected by head position and activities, often accompanied by stiffness and restricted range of motion.

Radicular pain is described as sharp, shooting, or burning and radiates along the affected nerve's dermatome from the neck to the hand. The radiation pattern corresponds to the compressed nerve root (Table 1, Fig. 4). It is distinct from the dull ache of mechanical pain.[5]

Radiculopathy and radicular pain may present independently or concurrently. Cervical radiculopathy encompasses motor, sensory, and reflex abnormalities distributed in dermatomal patterns corresponding to the affected nerve root (Fig. 4).[3, 18,19] Radiculopathy often yields one-sided, sharp "electric" pain down the arm with tingling or numbness and muscle weakness. In contrast, myelopathy commonly produces clumsy hands, balance/walking problems, stiffness or spasms, and overactive reflexes. Myeloradiculopathy will typically result in combined symptoms.

Responses to cervical disc herniation vary widely, influenced by pain tolerance, psychological factors, comorbidities, and herniation anatomy.[20,13,1,2] Some patients with substantial herniations are asymptomatic, while others with minor protrusions experience severe symptoms.[2,18] This highlights the complex link between structural pathology and clinical expression.

Diagnosis

Accurate cervical disc herniation diagnosis is crucial, as symptoms overlap with conditions such as spondylosis, tumors, etc.[1] Misdiagnosis may lead to inappropriate treatments, resulting in unnecessary or untimely procedures. Accurate diagnosis typically follows a three-step process: patient history, physical exam, and imaging confirmation.[18]

History and clinical presentation

When cervical disc herniation is suspected, the history should aim to distinguish nonspecific mechanical neck pain from disc pathology involving neural elements.[20,1] Evaluation for radiculopathy or myelopathy should include a comprehensive history of comorbidities, spinal trauma or procedures, activity exposures, and previous cervical symptoms.[20,1,18] For each complaint, note onset, course, severity, functional impact, and aggravating or alleviating factors.[1,21]

To differentiate radiculopathy from nonspecific neck pain, the history should assess for sensory, motor, and reflex

Table 1 Radiation pattern corresponding predictably to the compressed nerve root.

Root	Typical pain/paresthesia	Key weakness	Reflex change
C5	Lateral neck/shoulder, upper arm	Deltoid (shoulder abduction) ± biceps	Biceps ↓ (variable)
C6	Lateral forearm, thumb/index	Biceps & wrist extension	Brachioradialis ↓ (± biceps)
C7	Posterior arm/forearm, middle finger	Triceps, wrist/finger extensors	Triceps ↓
C8	Medial forearm, ring/little fingers	Finger flexors/grip	None specific

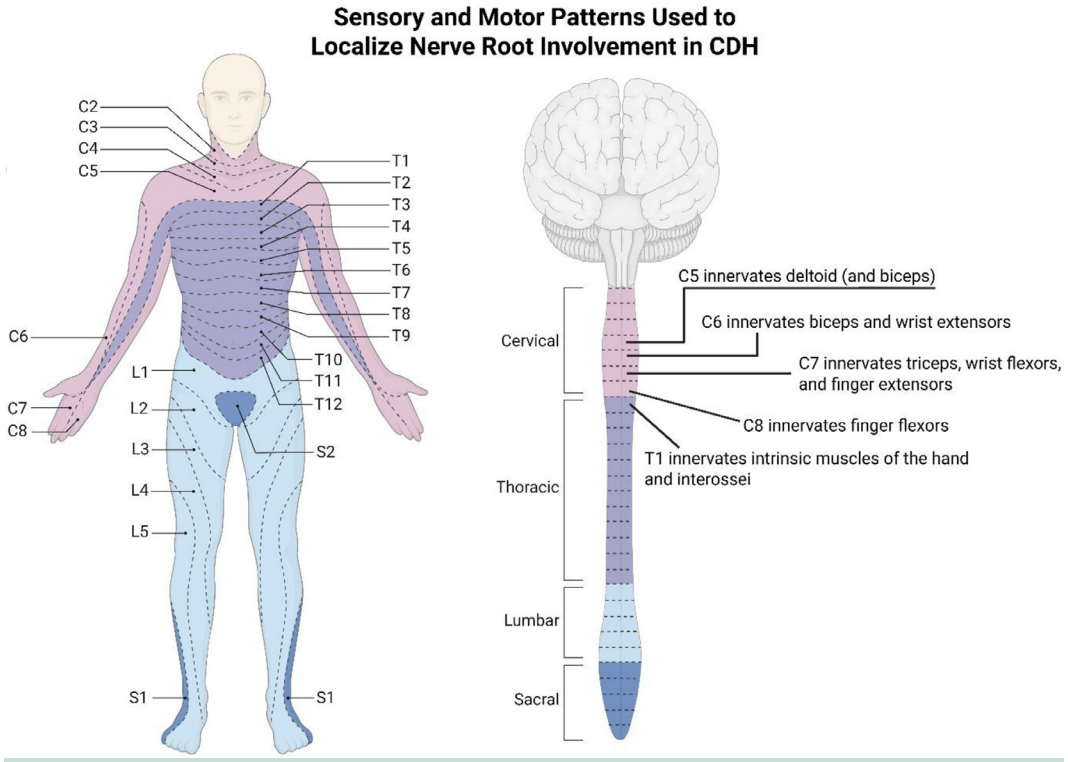

Fig. 4 Left panel depicts representative sensory dermatomes; right panel summarizes key upper-extremity myotomes relevant to CDH (C5-T1). CDH = cervical disc herniation.

symptoms, which suggest nerve root involvement (Fig. 4).[20,1,18] Assessing for myelopathy, including bowel or bladder dysfunction, is also important to determine extent of injury.[1,19] Clinicians should identify red flag features to guide diagnosis and imaging urgency (Table 2).[18,22,23]

Table 2 Neurologic red-flag screening checklist in suspected CDH with features prompting urgent or emergent evaluation.

Red-flag category	Features to inquire patient	Quick patient question (script)	Significance (What it suggests?)	Action (What to do?)
Cervical myelopathy (spinal cord compression)	Gait imbalance, falls, hand clumsiness (i.e., dropping objects); bilateral symptoms/neurological signs (both arms or arm & leg); leg stiffness or spasticity	"Any new balance issues/falls?" "Any problems holding onto objects?" "Any leg stiffness?"	Cervical spinal cord dysfunction from disc herniation or stenosis	Urgent spine/neuro evaluation with urgent cervical MRI
Bowel/bladder	New urinary retention; new urinary or fecal incontinence; new saddle-type sensory change; rapidly worsening gait; weakness with urinary changes	"Any new trouble starting urination, retaining urine? "Any numbness in your groin area?" "Any new bowel accidents?"	Advanced myelopathy/cord compression	Emergent evaluation with urgent cervical MRI
Progressive motor deficit (radiculopathy with deficit)	Progressive arm/hand weakness; objective weakness; new/worsening dropping objects from weakness; visible atrophy	"Any worsening weakness in your arm/hand?" "Any trouble extending wrist or fingers?" "Have you started dropping things from weakness?"	Nerve root compression (assess for cord involvement)	Urgent referral (same-day if worsening) with urgent MRI

Physical and neurological examination

Physical examination is conducted to obtain information regarding location of pain and extent of neurological compromise (muscle weakness or numbness, and abnormal sensations).[1,18,23] Cervical palpation may reveal tenderness, swelling, or muscle tension, while range-of-motion testing identifies mechanical restrictions or pain-provoking movements.[20,1,23]

Conversely, the neurologic exam focuses on assessing motor strength, sensory function, and reflexes to identify specific nerve root involvement.[1,18] Motor testing reveals myotomal weakness patterns (Fig. 4), and abnormal reflexes, can help localize nerve root compression.[1]

Provocative testing

Cervical radiculopathy tests help localize root compression and support cervical disc herniation diagnosis.[1,6,18,23] Key provocative tests, which enhance diagnostic accuracy, include Spurling, Shoulder Abduction, and Upper Limb Tension tests (Table 3).[18] Myelopathy is assessed via tandem gait, examination of upper motor neuron signs, and hand dexterity evaluation (Table 4).[19]

Diagnostic imaging

Imaging is not routinely required at initial evaluation, as history and examination often guide early management.[2,18] Imaging is indicated in the presence of concerning features, including prior trauma, symptoms persisting beyond six weeks, signs of myelopathy, or red flag findings suggestive of malignancy, infection, or inflammation.[18,23] In these settings, imaging confirms the diagnosis, localizes pathology, excludes alternative etiologies, and guides management.[1,2,18]

Treatment and management

The therapeutic objective in managing cervical disc herniation is to reduce neural inflammation, alleviate pain, restore function, and prevent neurologic deterioration.[1] Most patients (> 85%) improve within 8-12 weeks without treatment, and approximately 80% of herniated discs can be managed conservatively.[1,18]

Activity modification and self-care

Conservative management emphasizes activity modification and self-care.[1,5] High-impact activities, heavy lifting, sustained non-neutral cervical postures, and symptom-provoking movements should be avoided.[1,5,19] Early, pain-limited mobilization is favored over prolonged rest, with gradual activity progression as tolerated.[24] Adjuncts may include heat, cryotherapy, and short-term cervical collar use.[1,5]

Pharmacological management

Pharmacotherapy aims to reduce pain and inflammation, tailored to symptom severity.[1] Nonsteroidal anti-

Table 3 Provocative tests for cervical radiculopathy due to suspected CDH.

Test	How to perform	Positive finding significance	Interpretation in suspected CDH
Spurling Test	Seat patient upright. Gently extend the neck and laterally flex the neck toward the symptomatic side. Apply a light axial compression.	Reproduction of ipsilateral radiating arm pain or paresthesias.	Supports cervical radiculopathy from nerve root compression.
Shoulder Abduction Relief Test (Bakody sign)	Have patient flex the elbow of symptomatic arm and place hand on top of the head with shoulder abducted.	Reduction/relief of radicular arm pain or paresthesias.	Suggests cervical nerve root irritation.
Upper Limb Tension Test (median-nerve biased)	Lay patient supine. Depress and stabilize shoulder on affected side. Abduct arm 90˚ and supinate the forearm with fingers and wrist extended. Flex the elbow 90˚ degrees to enter starting position. Externally rotate the shoulder. Extend elbow until symptoms are reproduced. Laterally flex neck away from arm to further provoke symptoms if needed.	Reproduction of patient's radiating symptoms. Enhanced symptom intensity with contralateral laterally flexed neck positioning.	Supports cervical radiculopathy from nerve root compression or irritation.

Table 4 Examination maneuvers for cervical myelopathy in suspected CDH.

Maneuver	How to perform	Positive finding significance	Interpretation in suspected CDH
Tandem gait (heel-to-toe gait)	Stand by patient's side. Ask patient to walk heel-to-toe in a straight line. Observe balance and turns.	Inability to maintain tandem steps, unsteadiness, frequent corrective steps, or broad-based gait.	Suggests spinal cord myelopathy (higher urgency for referral & imaging).
Hyperreflexia (UMN sign)	Assess deep tendon reflexes for both upper and lower extremities. Perform side-to-side reflex comparison.	Brisk reflexes, asymmetrical reflexes, or pathologically increased reflexes.	Supports UMN involvement consistent with cervical myelopathy.
Hoffmann sign (UMN sign)	Have patient relax hand. Flick the distal nail of the middle finger.	Reflexive thumb flexion/adduction and/or index finger flexion.	Suggests corticospinal tract involvement. In the setting of neck/arm symptoms, raises concern for CDH-related myelopathy.
Babinski sign (UMN sign)	Stroke the lateral sole of the foot starting from the heel, moving toward forefoot.	Dorsiflexion of great toe with fanning of other toes.	Indicates UMN lesion; supports cervical myelopathy.
Clonus (UMN sign)	Seat patient. Rapidly dorsiflex the foot while stabilizing the ankle.	Rhythmic beating of foot (sustained clonus is more concerning).	Supports UMN involvement from cord compression (possible CDH-related myelopathy).
Hand dexterity assessment	Seat patient. Ask patient to rapidly tap index finger to thumb (rapid finger tapping). Ask patient to rapidly flip hand back and forth on thigh (rapid alternating movements).	Slowed finger taps, impaired rapid alternating movements.	Functional evidence of cervical myelopathy affecting hand function, which may occur with CDH-causing cord compression.

UMN = upper motor neuron

inflammatory drugs (NSAIDs) remain first-line, though prolonged use is limited by gastrointestinal, renal, and hepatic risks.[24] For inadequate response to over-the-counter agents, short courses of prescription-strength NSAIDs, oral corticosteroids, or muscle relaxants may be considered.[7] Opioid analgesics are generally avoided, reserved only for severe, refractory cases under close monitoring, as they have not demonstrated superior efficacy. [1,7, 25]

Physical therapy (PT) and rehabilitation

PT addresses biomechanical dysfunction, muscular weakness, postural abnormalities, and functional limitations through exercises and manual techniques.[1,18,23] For cervical disc herniation, it strengthens and mobilizes the neck, and improves posture.[1,5,7] PT is a core part of treatment, and long-term home exercise compliance is necessary for success and recurrence prevention.[1,7]

Interventional pain management

If pain persists beyond 4-6 weeks despite PT and medication, cervical epidural steroid injections (ESIs) may be considered to reduce neural inflammation, aid rehabilitation, avoid or delay surgical intervention, and provide short-term symptom relief.[18] Approximately 50% of patients experience relief after an ESI, which varies in duration and intensity.[26] Multiple injections may be given to maximize the effects.[7] ESIs are temporary, targeting inflammation rather than addressing the underlying disc pathology, and are not curative.[7]

Indications for surgical intervention

While the majority of cervical disc herniation cases resolve with conservative management,[1,18] surgical intervention is indicated when:

- Cervical myelopathic symptoms are present.[4,18]
- Neurological deterioration.[1,18]
- Disabling radicular pain despite 6-12 weeks of comprehensive conservative management, impairing quality of life and function.[18,27]

Conclusion

Management of cervical disc herniation is guided by a stepwise, evidence-based approach that reflects its generally favorable natural history. Initial conservative therapy (including NSAIDs, PT, and patient education) results in substantial improvement for most patients. Escalation to adjunctive pharmacologic or interventional therapies may be considered for persistent symptoms while surgical

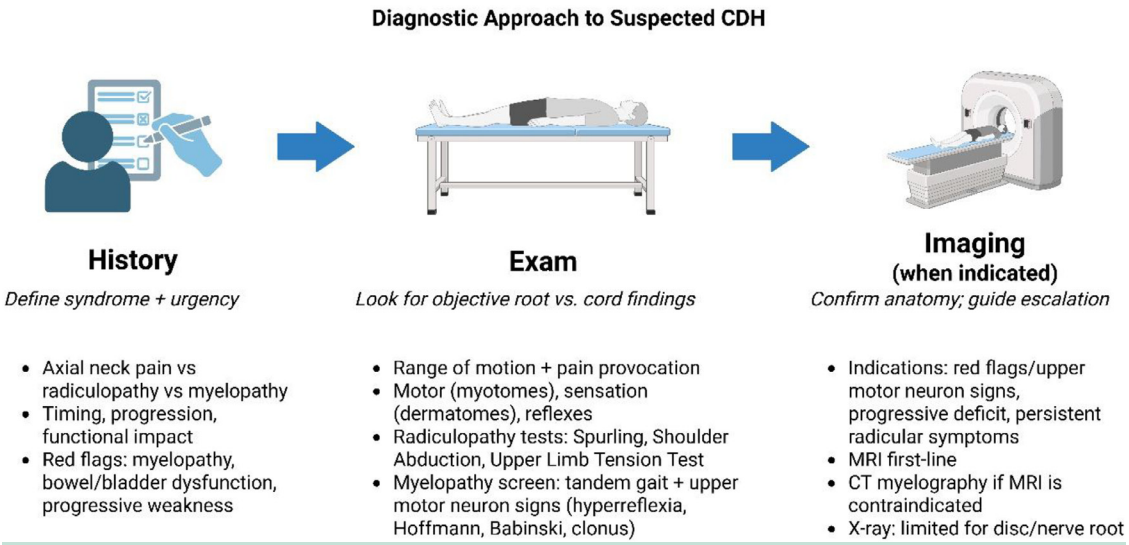

Fig. 5 A three-step workflow for evaluating suspected CDH.

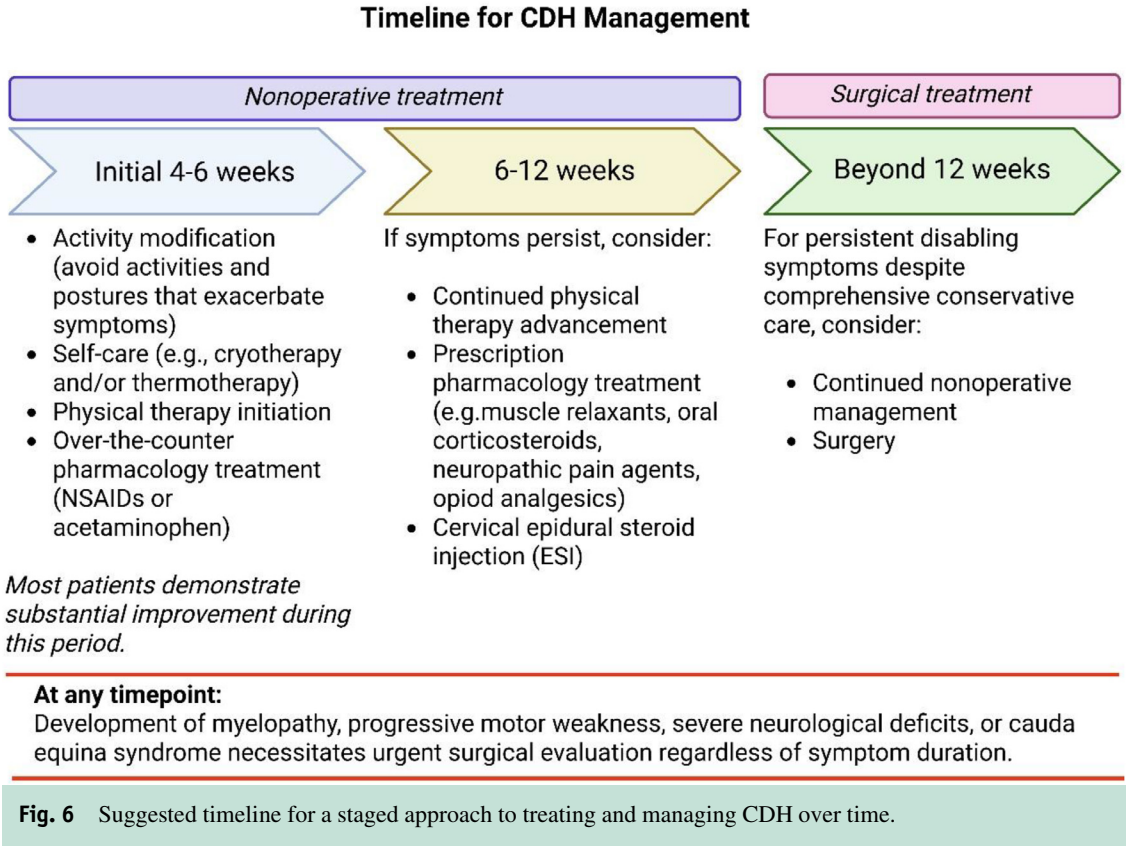

Fig. 6 Suggested timeline for a staged approach to treating and managing CDH over time.

evaluation is reserved for refractory cases or those with neurological progression. Fig. 5-6

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Declaration of competing interest

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