Case Report

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Cervical degenerative espondilopathy in post-menopausal women

Abstract

Cervical syndromes comprise a set of clinical conditions that have pain (cervicalgia) in common, to which are added muscular, articular, neurological and even vascular manifestations, with cephalic (cervical cephalic syndrome) and brachial (cervical brachialgic syndrome) irradiations, whose The origin is degenerative spondylodiscopathy or cervical spondylosis. It is a very common entity with an incidence of 85% in people over 60 years of age and onset at an early age, with a high incidence in women. The most common etiology is degenerative: spondylo-disc-uncus-arthrosis constituting 90% of cases, the rest would be inflammatory, tumorous, septic and traumatic, being rare.

Keywords: spondylopathy, discopathy, osteophytosis, spondylosis, Grit sign

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Edison Edgardo Romero-Galván
Diploma in Bone and Metabolic Diseases, Autonomous University of Baja California (UABC), Uruguay

Correspondence: Edison Edgardo Romero Galván, MD, Diploma in Bone and Metabolic Diseases, Autonomous University of Baja California (UABC), Private Medical Office in Tacuarembó/Uruguay, Email romlab@hotmail.com

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Introduction

The cervical spine forms a very mobile anatomical sector of the vertebral column, it is crossed vertically by three fundamental structures: spinal cord, vertebral arteries and sympathetic system, transversely by the spinal roots which are shorter and horizontal so They are more vulnerable to traction.

It is very flexible, fragile and mobile, it is susceptible to minimal trauma, to chronic suffering that results in degenerative disc processes (discopathy), in vertebral bodies and apophyseal structures that are externalized through posterior muscular compromise that are powerful, powerful with outstanding tonic and dynamic action expressing itself with pain due to contractures with neurological compromise (neuropathic pain).

Incidence

30% of the population presents pain at the cervical level with a maximum incidence between 30 and 60 years, it predominates in women, it can be acute or chronic forms, with or without root irradiation. The cervical vertebrae have the uncinate processes or uncinate process described by Hubert von Luschka in 1858, in whose vicinity is the vertebral artery relating to the nerve root.

The intervertebral disc is made up of a fibrous ring formed by concentric layers of fibrous lamellae with a helical arrangement and type I collagen fibers. Denser and thicker in the anterior part; It is inserted into the vertebral bone by Sharpey's fibers (William) with a gradual transition towards the nucleus pulposus Figure 1. The annulus is less thick towards the back, being one of the factors responsible for the predominance of the posterior protrusions of the nucleus pulposus, although in In exceptional cases there may be anterior protrusions being described for the first time by Batts in 1939, three cases in 50 pieces of corpses.[1]

From 1929 to 1932 Junghans H., Schmorl G. and Rudolf Andrae described the anatomopathological characteristics of intervertebral disc herniation, but for many more years the term "chondroma" continued to be used to refer to this pathology, described for the first time by Stookey in 1928 as "Ventral extradural cervical chondromas"; Only in 1940 did the author himself recognize that they were herniated discs that compressed the spinal cord.[2]

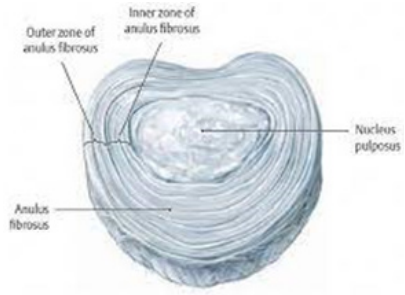

Figure 1 Fissured fibrous annulus and elastic, gelatinous nucleus pulposus. In the posterior area the ring is thinner than in the anterior part, which predisposes to Protrusions.

In 1930 French authors Alajouanine and Petit-Dutaillis suggested that the lesion, previously identified as a chondroma, was actually a hernia of the nucleus pulposus.[3]

The termdisc or cervical herniationdisc herniation (CDH) is used today to refer to the injury of the intervertebral disc in which the nucleus pulposus leaks outward, this term was established in 2013 by the Nomenclature and Classification of Disc Pathology (NASS) through radial fissures and ring tears.

Schmorl and Junghanns (1968), Schaffer himself (1930) in autopsy studies found degenerative signs from the age of 30, cellular degeneration, fibrous disintegration, structural changes in the macromolecules that maintain disc hydration.The cellularity is very scarce, it has fibroblasts and chondrocytes that decrease with age. The water in the disc is reversibly ionically bound to macromolecules such as different mucopolysaccharides and proteoglycans such as Chondroitin and Keratan sulfate. As these molecules depolymerize and the loss of Chondroitin sulfate together with the disoriented collagen, the disc dehydrates, altering the function of the gel, which affects the osmotic and hydrostatic pressures of the nucleus. These macromolecules synthesized by chondrocytes have a half-life between 3 and 14 days; They form a network that gives elasticity and viscosity to the disc due to its ability to fix wáter.[4,5]

The nucleus pulposus, enclosed in the annulus fibrosus, acts as a semipermeable membrane, exchanging liquids and nutrients; It is subject to hydrostatic pressure due to the load transmitted by the

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bodies; which varies with the position and the weights the person carries. The function of the disc is to cushion and transmit loads between vertebrae, the nucleus pulposus moves with movements; It has a colloid-osmotic pressure that depends on the macromolecules that attract water to maintain the turgor pressure, which is the pressure against resistance of a body being able to expand by absorbing water. Oncotic pressure is the sum of the two (turgor or colloidosmotic pressure and hydrostatic pressure). When the nucleus pulposus is subjected to a hydrostatic pressure (load pressure) greater than 80 kPa (kilopascals) equal to 800 Newton, it expels water and the disc loses height, decreasing the interdiscal space. The loss of water increases the osmotic pressure reaching a point where water extrusion stops. When the hydrostatic pressure falls below 80 kPa the nucleus expands due to the entry of water that dilutes the macromolecules, the osmotic pressure falls and the entry of water ceases; This process is faster in young discs. In the morning we are taller than at night, standing increases disc pressure and losing water decreases height, especially in overweight patients.

The process of disc degeneration begins with a decrease in the metabolic activity of the disc cells, a decrease in the synthesis of macromolecules in the nucleus and an alteration in the fibers of the annulus fibrosus. The fibers bulge, they are subjected to the pressure of the nucleus, so they begin to crack and through these, fragments of the nucleus pulposus migrate giving a PROTUSION, a term suggested by Schmorl without being considered for a long time by other authors. If the fissure becomes larger, the contents come out, constituting the HERNIATED disc Figure 2 and Figure 3 with extrusion and migration (sequestration) that can be cephalic or caudal, were described for the first time by Charles Bell (1824), Henley (1856) and Virchow (1857) at the cervical level with spinal cord compression.

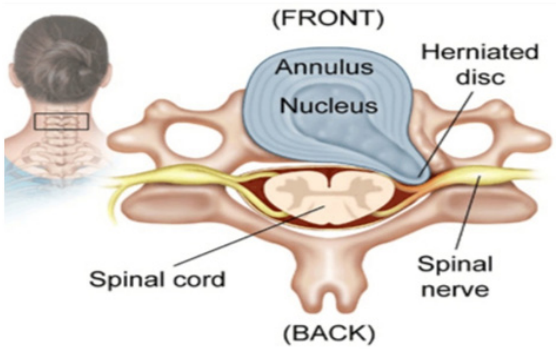

Figure 2 Herniated disc compressing neurological root.

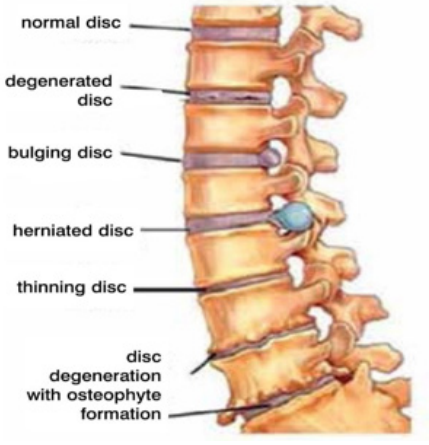

Figure 3 Different evolutionary stages of discopathy until marginal osteophytosis.

In the posterior part it comes into contact with the posterior common ligament, irritation of its innervation causes symptoms, but its arrangement favors the protrusions being lateral towards the neuroforamina. Changes in the nucleus pulposus with loss of turgor and crushing of the annulus produce instability, which pulls on the anterior longitudinal ligament and the vertebral edge, causing osteophytes to appear, designated as "marginal spondylosis" by Borak in 1947, causing symptoms if they invade the canal (osteophytosis). intracanalicular or stenotic spondylosic duct) popularly known as parrot beaks Figure 4 producing its narrowness with risks of spinal compression when it is less than 13 mm. They appear above the age of 30 and by age 40 most people have some degree of marginal osteophytosis.

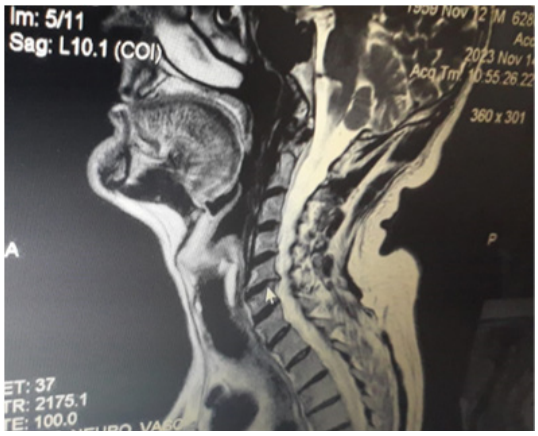

Figure 4 62-year-old patient with cervico-brachialgic symptoms, an intracanalicular osteophyte is observed (arrow/cursor) that produces stenosis of the spinal canal (November 2023).

Above the age of 60, the disc becomes fibrous, signs of emptiness with disc desiccation, the osteophytes can fuse (pontic osteophytosis) and the vertebral segments lose mobility Figure 5 giving spinal rigidity at advanced ages, the symptoms come from osteoarthritis of the posterior arch. Osteophytes at the cervical level can narrow the orifice of the vertebral artery Figure 6 giving vertebrobasilar insufficiency described by Dutton CB and Riley LH in 1969; in the neuroforamina they compress the neurological roots giving a clinical picture similar to that of disc protrusions and herniations.

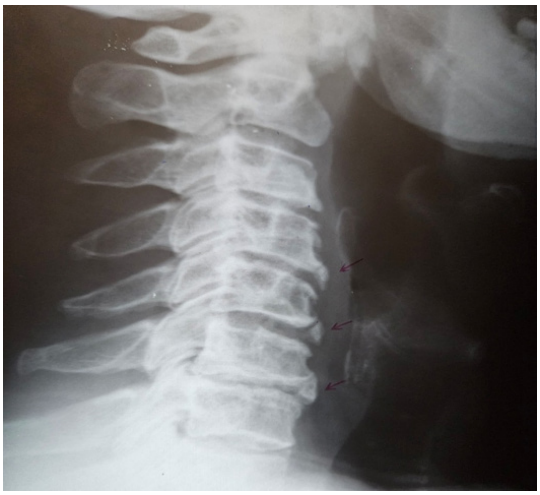

Figure 5 Profile x-ray of the cervical spine, 62-year-old patient. A rectification of her cervical lordosis is observed, a decrease in vertebral height due to postmenopausal demineralization, multiple impingements due to degenerative disc disease and the arrows mark the marginal osteophytes C3-C4-C5 (Personal case Romero Galván EE MD treated in 1985).

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Figure 6 Marked marginal osteophytosis of the cervical vertebra of a woman who died at 77 years of age.

In the transverse process, the passage hole of the vertebral artery is covered with a sleeve of sympathetic neurological fillets, posterior sympathetic of Lazorthes. The figure shows how the osteophytes narrow the passage hole of the vertebral artery (C2 to C6), right of the figure, compromising the vascular and sympathetic chain, as well as intracanicular osteophytes stenosing the spinal canal.

Semiology

Upon inspection, neck stiffness is noted, rotation movements limited by pain are avoided. The patient must perform flexion-extension movements, in flexion the chin must contact the sternal fork and in extension separate them by about 20 cm; Failure to reproduce this situation indicates upper cervical involvement C1 to C3, if there is difficulty in lateralization it is due to lower cervical involvement C4 to C8. Perform palpation of the spinous and transverse processes, remembering that C3 is in the same horizontal plane as the hyoid bone, C4 corresponds to the thyroid cartilage, C5 to the lower portion of the same, and C6 to the cricoid. Palpate the entire muscular complex of the neck looking for tension and contractures. Perform the Jostes and Spurling maneuvers described below and finish with a neurological examination of both upper limbs, looking for sensory deficits in the C4-C5-C6-C7-C8 dermatomes, in the pads of the fingers: "Grit sign", expressed as cervicobrachialgia.

Clinic

The most frequent symptom is local or radiating PAIN, it is produced by irritation of the nerve endings such as the Luschka nerve, which is an anterior branch of the spinal nerve, which innervates the posterior common longitudinal ligament; Its posterior branch innervates the joint capsule. The pain has mechanical or biochemical origin from the degradation products of the disc. The root is very sensitive to mechanical irritation due to compression or traction of the dural or thecal sac and is characterized by radiating along the dermatomes Figure 7; Muscle atrophy corresponding to the affected root can occur if the compression persists for a long time. Radicular pain due to disc compression (protrusion or herniation) is modified with changes in posture that decompress the root or we can reproduce the pain through the Spurling Roy Glen maneuver described in 1944[6] Figure 8 or foraminal compression test with a specificity of 95% and consists of extending the head to the side ipsilateral to the pain, exerting downward force on the head, reproducing the pain radiating to the affected upper limb; The reverse maneuver is the Jostes maneuver that calms cervical headaches by pulling the skull upward with both hands, taking it by the lower jaw and the occipital.[7] Neuropathic radicular pain increases with Valsalva maneuvers, sneezing, coughing, or bowel movements.

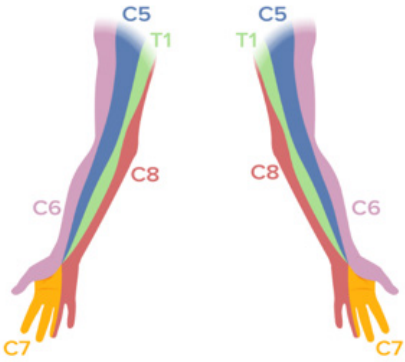

Figure 7 C5-C6-C7-C8 root dermatomes. The fingers of the hands correspond to C 6-C7-C8 and identify the "Grit Sign"; C5-C6 correspond to the anteroexternal surface of the arm and forearm and C8 to the internal surface of the forearm.

Figure 8 Spurling maneuver reproducing radicular pain radiating to the affected upper limb caused by a disc protrusion or herniation.

The patients present sensory and motor deficits in the upper limbs. With respect to the first, personally in my 40 years of experience I have discovered a clinical sign which I have called "Grit sign" which consists of sliding the pad of the thumb over the pads of the other fingers with both hands simultaneously, the Patients report a sensation of fine sand or roughness in one of their hands and upon completing the sensitive neurological examination of the upper limbs, a tactile deficit (cotton, brush, sensation of roughness), thermal deficit (the neurological hammer is less cold) and pain in the upper limbs will be found. ipsilateral forearm with decreased deep tendon reflexes, confirming disc disease C 6-7-8 with disc protrusions that compromise the neurological roots of that same side of the "grit" corresponding to dermatomes C 6, C7 and C8 due to foraminal involvement of said roots Figure 7; constituting a cervico-brachialgic syndrome, it is common in people under 40 years of age and the pain has a nocturnal component "nocturnal paraesthetic brachialgia".

Above the age of 45, degenerative phenomena appear: marginal osteophytosis, a term coined by Collins DH in 1950 and even intracanalicular causing stenosis of the spinal canal, to which is added hypertrophy of the yellow ligament; the marginal ones can form osteophyte bridges Figure 5, posterior interapophyseal osteoarthritis, as well as osteoarthritis and osteophytes of the uncinate processes that will compress the exit of the root and the vertebral artery with its sympathetic manifestations, of slow and chronic evolution.

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In cases of large osteophytes or anterior marginal spurs at the C5 and C6 level, they can produce dysphagia Figure 5 and Figure 9 as has been described by several authors such as Heck CV (1956), Meeks and Renshaw (1973)[8,9] by extrinsic compression of the esophageal wall; When faced with this symptom, first rule out organic esophageal tumor pathology.

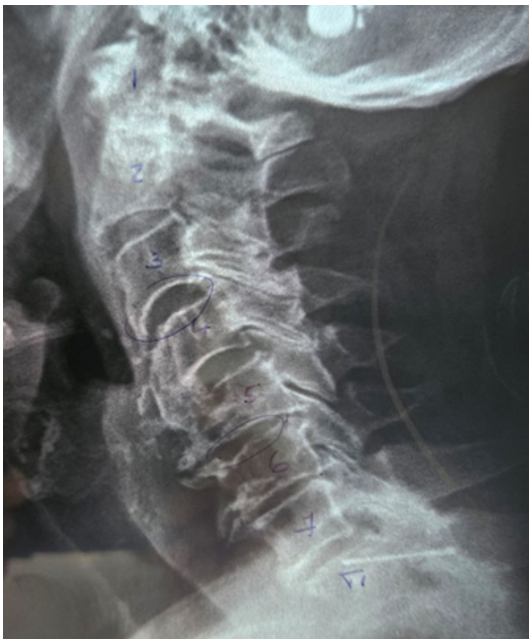

Figure 9 Thick marginal osteophytes C3 to C7 with a tendency to form osteophyte bridges, 85-year-old patient (March 2024).

This is a process of progressive onset, at an early age, with pain that increases with movement. If bone compression predominates, there is nocturnal radicular pain that radiates to the entire affected upper limb, with paresthetic sensations such as burning, burning, tingling, hypoesthesia, acroparesthesias with loss of strength in the ipsilateral hand and sometimes with a sensation of cold, sweaty hands due to sympathetic involvement.

More than 40% have a sensory deficit (Grit Sign) and/or motor deficit due to the direct connection of the cord and roots that, when passing through the conjugation foramen, come into close contact on the anterior side with the cervical discs, with the uncovertebral joint or Luschka (spondyl disc unco osteoarthritis) and on its posterior surface with the interapophyseal joint that is often compromised by osteoarthritis.

These lower cervical syndromes can cause omalgia, simulating shoulder periarthritis, so the differential diagnosis must be made by applying correct semiology, ruling out involvement of the tendon of the long head of the biceps and those of the cap of the short rotators (supraspinatus, infraspinatus, subscapularis). And teres minor); They may have sympathetic expressions due to irritation of sympathetic nerve bundles that join the cervical roots emerging from the neuroforamina.

They are also accompanied by paravertebral pain due to contractures with a sensation of rigidity, especially in prolonged flexion positions when reading, or in office work, and in these modern times due to the excessive use of cell phones from an early age that leads to a rectification of physiological lordosis.

One of the most frequent symptoms of spondylocervicopathy is headache or headaches present in 80% of cases, constituting the "cervicocephalic syndrome" due to involvement of the first two roots and especially C2 or Arnold's suboccipital nerve (Philipp Friedrich) giving rise to the "Arnold-ophthalmic syndrome" with retro-ocular pain due to anastomosis in the frontal region with supraorbital branches of the facial nerve, pain in the neck (nucalgia), hemicranium and basifrontal due to involvement of the occipitoatloaxial sector with irradiations to the vertex , to supra and retroorbital region. It is an acute, persistent, continuous pain that has little response to analgesics and can be accentuated by lying down without tolerating pillows. "Pillow sign." It is accentuated if we percuss the vertex and is transmitted to the base of the skull, increasing with cervical mobility. In 1984 Gutmann G. and Biedermann H. described a "school headache" caused by permanent and continuous flexion of the head at the end of school classes caused by an insufficiency of the transverse ligament of the atlas (atlantotransverse)[10] and that Currently I would call it "cellular headache", due to the excessive use of mobile phones in constant flexion by the new generations and even an occipital exostosis found in 218 cases to be able to support the weight of the head, hypertrophy of the external occipital protuberance Figure 10.[11]

One characteristic is sensory disorders of the scalp, tingling paresthesias or hyperesthesias that patients cannot comb their hair: "Comb sign" or dysesthesias such as a burning, burning or cold sensation; These symptoms are frequent reasons for consultation, so we must know them and know how to interpret patients. They are often associated with ocular symptoms such as photophobia, decreased visual acuity, cloudy or blurred vision, phosphenes, ocular burning, foreign body sensation, retro-ocular pain and even eyelid edema; These symptoms frequently lead to ophthalmological consultation and are typical of high spondylocervicopathy.

Vertigo or a feeling of instability are common, the second most common symptom after headaches. They appear when making a sudden movement of the head on the neck, when getting up in the morning or when standing up after bending over; They may be accompanied by latero or retropulsion, with nausea and vomiting of short duration. They are considered vestibulocochlear symptoms that are associated with tinnitus in a third of patients and even decreased hearing, of neurovegetative origin, which can even lead to sweating attacks reaching lipothymia. The first authors to describe this clinical condition with headaches and vestibulocochlear symptoms were Jean Alexandre Barré in 1925 and his disciple Lieou in 1928. They considered it to be alterations of the posterior cervical sympathetic perivascular of the vertebral artery and called it "Posterior Cervical Sympathetic Syndrome" (SSCP) and which later bears their names: "Barré-Lieou Syndrome",[12] being more common in women over 50 years of age. We have seen patients with cervical otalgia of retroauricular neuralgic origin innervated by the lesser occipital nerve and the greater auricular nerve that come from C2 and C3.

Paraclinical studies

The radiological studies to request are frontal, profile Figure 10 and oblique to see the neuroforamina; confirm degenerative cervicopathy with the presence of marginal osteophytes, decreased intervertebral spaces due to disc involvement (impingements), posterior interapophyseal osteoarthritis and the change in physiological lordosis, rectification and even inversion of the same in severe cases. Many times there is no correlation between the clinical picture, patient symptoms and radiology findings. In these studies (x-rays) in cases of disc protrusions we see the decrease in the intervertebral space and the rectification of the physiological lordosis above the injured disc, known as "Güntz's Sign", with few degenerative changes as it occurs more in young women before of 40 years.

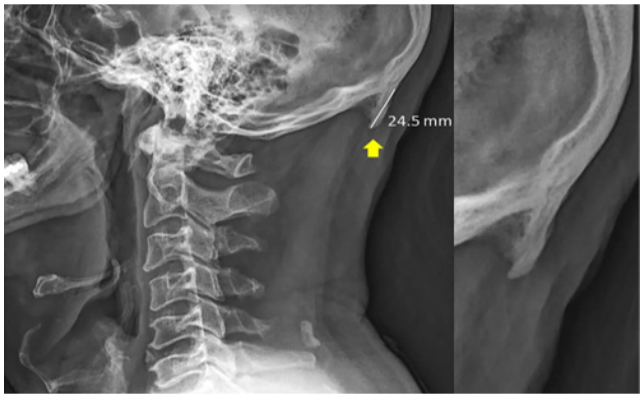

Figure 10 Exostosis of the External Occipital Protuberance and rectification of cervical lordosis due to excessive cell phone use (Taken from Shahan D. and Sayers Mark GL 2019).

The other gold standard study is the MRI of the cervical spine Figure 11 where the disc compromise, the signal changes due to disc dehydration (in T2) leading to a decrease in disc height and the different degrees of protrusions through the discs are directly observed. fissures of the annulus fibrosus, contacting the dural or thecal sac, producing stenosis of the spinal canal and root involvement at the level of the different neuroforamina. Also reported in the Resonances are "disc vacuum phenomena" described by Knittson in 1942[13] and by Gershon-Cohen in 1946[14] who observed radiolucent striae in the nucleus pulposus with the annulus fibrosus intact without protrusions or hernias and attributed to degeneration of collagen fibrils.

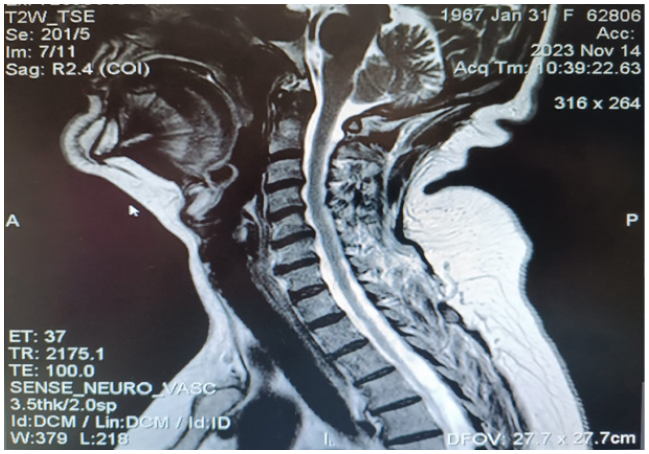

Figure 11 Resonance of the cervical spine, sagittal section of a 56-year-old patient with right cervicobrachialgic syndrome and "grit sign" present in the right hand (Personal case Romero Galván, EE MD treated in November 2023). Disc protrusions are observed from C2 to C6, with dehydration and narrowing of the spinal canal.

Differential diagnoses

Despite these being frequent clinical conditions, it is necessary to carry out differential diagnoses with other pathological entities such as vertebral secondary diseases in neoplastic patients known or not previously known, also differentiating with benign tumors such as neuronomas or spinal meningiomas, with chronic inflammatory pathologies such as ankylopoietic spondylitis Bechterew) and with infectious discitis. Headaches can be differentiated from other causes such as high blood pressure, headaches due to intracranial expansive processes, and migraines themselves. Vertigo can be differentiated from that caused by Ménière's Disease.

Treatment

Cervical syndromes of degenerative origin, with or without cephalic or brachial projection, are produced by the sum of various factors, which cause, as a final common pathway, the irritation or compression of the cervical neurological roots and the sympathetic plexuses associated with other states such as muscle contractures and inflammatory processes; In these senses we must act therapeutically. Most of the time the treatment is medical, physiatric and eventually neurosurgical, aiming to calm musculoskeletal and neuropathic pain, overcome paravertebral contractures, suppress inflammatory processes, reduce psycho-emotional tensions, stop degenerative events and recover root trophism to attenuate deficit neurological symptoms.

To relieve pain, major analgesics (Morphine, Tramadol, Codeine) are used exceptionally; minor ones, non-steroidal anti-inflammatory drugs (NSAIDs) and COX 1 inhibitors (Diclofenac, Ketoptofen, Ibuprofen, among others) are used more frequently with the precaution of be gastrolesional, gastric protection with proton pump inhibitors (Omeprazole, Lanzoprazole, Pantoprazole) is necessary for the duration of treatment. You can use COX2 inhibitors that do not damage the gastric mucosa, such as Celicoxib 200 mg or Eterocoxib 90 to 120 mg and even Meloxicam 15 mg. These drugs can be associated with Paracetamol 1000 mg every 6 hours and muscle relaxants such as Orphenadrine, Tizanidine 2 to 4 mg or Cyclobenzaprine 5 to 10 mg, indicating them at night with the purpose of treating muscle contractures, reducing hypertonia during sleep, Do not indicate them during the day to avoid drowsiness. Corticosteroids would not be indicated because the inflammation of mechanical origin is of low intensity and can be decreased with the NSAIDs described.

To cushion degenerative events, the best option is to indicate Chondroitin Sulfate between 800 and 1200 mg daily, remembering its function at the disc level to maintain its hydration; It has no side effects and no drug interactions. For neurological trophism, vitamins B1, B6 and B12 (Thiamin, Pyridoxine, Cyanocobalamin: TPC) are indicated with a dose of 5000 IU with a very good response in sensory deficit disorders, the "grit sign" disappearing, normalizing the neurological examination. They have antinociceptive, neuroprotective and neuromodulatory effects,[15] improving the production of neurotransmitters of the descending modulatory pathway of neuropathic pain.[16,17]

They are also relieved by giving them anti-neuralgics, all anticonvulsants have these effects from Carbamazepine to the latest molecules such as Pregabalin (600 mg maximum doses), Lamotrigine 100 mg and Gabapentin 400 mg, acting on the modulation of ion channels, blocking those of Sodium for Carbamazepine and those of Calcium for Gabapentin. They are adjuvants in analgesic therapy, as are antidepressants such as MAO inhibitors, tricyclics and the most recent Serotonin reuptake inhibitors, they change the pain threshold by dampening it.

Physiotherapy is very effective to restore function and spinal hygiene exercises to avoid inappropriate postures and be able to correct the rectification of cervical lordosis as well as the use of Philadelphia Collarete, use it in fractions of time to avoid muscle atrophy.

Patients in whom extruded hernias are confirmed should consult with Neurosurgery.

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Conflicts of interest

The author declares that there is no conflicts of interest.

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