Cervical spondylosis: a literature review with attention to the African population

Degefe A. Belachew, Bernhard J. Schaller, Zenebe Guta

Department of Neurology, Faculty of Medicine, Addis Ababa University, Ethiopia

Submitted: 11 August 2007
Accepted: 19 September 2007

Arch Med Sci 2007; 3, 4: 315-322
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Corresponding author:
Degefe Belachew, MD
Department of Neurology
Faculty of Medicine
Addis Ababa University
Ethiopia
E-mail: belachewda@yahoo.com

Abstract

Cervical spondylosis, a degenerative disease of the cervical spine, intervertebral discs, ligaments and cartilaginous material, is commonly seen in individuals after the age of 40 years and is believed to be part of the normal aging process of the vertebral column. It is seen in up to 19% of asymptomatic individuals. The major clinical presentations of cervical spondylosis can be subdivided into: (i) cervical spondylotic myelopathy, (ii) cervical radiculopathy, and (iii) neck pain syndromes. There are only a few studies showing that the disease is similarly present in Africa compared to North America or Europe and that the African lifestyle may predispose to early clinical manifestations.

Medline and Embase databases were searched for trials, case series or case reports published between 1966 and March 2007 with the objective of updating current knowledge about cervical spondylosis with special reference to the population in Africa.

Cervical spondylosis is one of the most common causes of cervical radiculopathy, myelopathy and chronic neck pain syndromes. A lot of studies have been conducted with regard to the pathophysiology, risk factors and treatment of the disease since the early 1990s. In Africa, there exist only a few studies on this topic indicating that there are some risk factors which are somewhat different from the developed nations and which may lead to early clinical manifestation. These factors embrace mainly load carrying and fluorosis, which are prevalent in the Rift Valley areas of Africa.

Key words: review, cervical spondylosis, myelopathy, surgery, spine, Africa.

Introduction

Cervical spondylosis, a degenerative disease of the cervical spine, intervertebral discs, ligaments and cartilaginous material, is commonly seen in individuals after the age of 40 years and is believed to be part of the normal aging process of the vertebral column. It was shown by radiological investigation of asymptomatic individuals that spondylotic changes increase with each decade of life: 5-10% by the age of 20-30 years, >50% by age 45 years, and up to >90% by 60 years of age.

Boden et al. reported about the magnetic resonance imaging (MRI) scans of sixty-three volunteers who had no history of symptoms indicative of cervical disease. The scans demonstrated an abnormality in 19% of the asymptomatic subjects: 14% of those who were less than forty years old and 28% of those who were older than forty [1]. Of the volunteers who

were less than forty years old, 10% indicated a herniated nucleus pulposus and 4% had foraminal stenosis. Of the volunteers who were older than forty years herniated nucleus pulposus (5%) and bulging of the disc (3%) were minor findings in contrast to 20% with foraminal stenosis [1]. The prevalence of abnormal MRI of the cervical spine as related to age in asymptomatic individuals emphasizes therefore the dangers of predicting operative decisions on diagnostic tests without precisely matching those findings with clinical signs and symptoms [1].

Hayashi et al. in their radiological evaluation of cervical spondylotic myelopathy in elderly people studied 42 individuals over the age of 60 years and compared them with those of younger patients [2]. Myelography and post-myelography computed tomography (CT) showed multi-segmental lesions (average: 3.0 lesions) that contributed to the various clinical features of myelopathy [2]. The upper disc levels of C3-4 and C4-5 were predominantly involved in the aged patients [2]. Disc protrusion, posterior osteophyte and retrolisthesis, in addition to ligamentous entrapments, were the primary aetiological factors of myelopathy in this patient subgroup [2].

There are several main clinical syndromes which are described in relation to cervical spondylosis: (i) cervical spondylotic myelopathy, (ii) cervical radiculopathy, and (iii) neck pain syndromes.

In this review we will try to look into myelopathy in more detail and give an overview about the actual knowledge of this disease in the African continent with special reference to potential differences in the current knowledge of the disease. This is the reason why we will not focus on epidemiology and aetiology of cervical spondylosis in this review.

Methodology

Medline and Embase databases were searched for trials, case series or case reports published between 1966 and March 2007. The terms used included: "cervical spondylosis", "cervical spondylotic myelopathy", "cervical radiculopathy", and "neck pain". More recent publications were used in preference to older ones, as the purpose of the review is to give an up-to-date review of the current knowledge. Older publications were retrieved if they were cited in the selected publications and were deemed significant for the purpose of this review.

Pathophysiological background

According to Stookey, the spinal cord disorder associated with cervical spondylosis (cervical spondylotic myelopathy) was originally attributed to compression of the cord by cartilaginous nodules of degenerate disc material [3]. Because these nodules are small compared with spinal tumours causing similar disability, it was suggested that compression by these alone could not explain the disorder. The rarity of the condition when compared with the frequency of the skeletal changes, the lack of correlation between the severity of the spondylosis and the severity of the paraparesis, and the disappointing results of surgical removal of the nodules further undermined the theory that compression of the spinal cord was not the sole explanation [4].

A progressive, age-related degeneration of intervertebral discs in the cervical spine is followed by a decrease of the intervertebral space. Clinical studies indicated that subperiosteal bone formation occurs next, forming osteophytic bars that extend along the ventral aspect [5, 6]. Uncinate process hypertrophy also occurs, often encroaching upon the ventrolateral portion of the intervertebral foramina. Nerve root irritation also may occur as intervertebral disc proteoglycans degrade. Ossification of the posterior longitudinal ligament can occur with cervical spondylosis and can be an additional contributing source of severe anterior cord compression. Age-related hypertrophy of the ligamentum flavum and thickening of bone may result in further narrowing of the spinal cord space.

The consecutive myelopathy of the cervical spine may be due to direct spinal cord compression, ischaemia due to compression of related vascular structures, repeated local trauma to the spinal cord by physiological movements in the presence of osteophytic bars, or a combination of these factors [7]. Different investigators suggested the importance of interference with the blood supply of the cord [8-11]. In 1972, Nurick – for example – studied the importance of compressive factors and assessed the importance of ischaemia of the cord caused by spondylosis but arising independently of compression [12]. Cervical spinal myelopathy (CMS) patients have a significant reduction of the spinal canal's sagittal diameter, which has been correlated with increased neurological deficits [12]. However, generalized vascular disease had no apparent effect on the severity of CSM [12]. Additionally, degenerative kyphosis and subluxation are fairly common findings that may further contribute to spinal cord compression in patients with cervical spondylotic myelopathy. Free radical- and cation-mediated cell injury, glutamatergic toxicity and apoptosis may be of additional relevance to the pathophysiology of cervical spondylotic myelopathy [7]. The pathological course of cervical spondylotic myelopathy is therefore characterized by early involvement of the corticospinal tracts, and relative preservation of anterior columns.

A more recent review evaluated current evidence for CSM and evaluated the works of different investigators demonstrating ischaemia as one of

Table I. Cervical cord compression

• Disc herniation alone
• Degenerative changes that occur in the spine such as degeneration of the joints, intervertebral discs, ligaments and connective tissue of the cervical vertebrae
• Bone spur growth in the spinal canal (spondylosis)

Table II. The pathophysiology of cervical myelopathy

• Static factors: which result in acquired or developmental stenosis of the cervical canal
• Dynamic factors: which involve repetitive injury to the cervical cord

the major underlying factors in patients with CSM [13]. Anterior compression compromises perfusion through the transverse arterioles arising from the anterior sulcal arteries, while posterior cord compression works to reduce perfusion to the intramedullary branches of the central grey matter.

More recently, shearing forces have been theorized to be important factors in the pathophysiology of cervical spondylotic myelopathy. Narrowing of the spinal canal and abnormal or excessive motion may result in shear forces that cause axonal injury in the cervical cord, where changes seen in the cord may actually be a form of stretch injury [14]. Although the exact pathophysiology underlying cervical spondylotic myelopathy remains uncertain, it is largely accepted to be a disorder that involves compressive forces on the spine, likely due to multiple factors (Tables I and II).

These mechanical factors in turn result in direct injury to neurons and glia as well as secondary cascade of events including ischaemia, excitotoxicity and apoptosis.

Cervical spondylotic myelopathy

Cervical spondylosis is the commonest cause of spinal cord disorders in patients over the age of fifty years [15]. Because its presentation may be subtle, involves elderly people and there are no pathognomic findings, diagnosis may be difficult. Cervical spondylotic myelopathy is part of the spectrum of degenerative disorders of the cervical spines and is the most devastating. There can be considerable overlap of the presentations of these disorders.

There are five distinct syndromes isolated on the basis of clinical presentations [16]:

1) *lateral or radicular syndrome* – where nerve root symptoms (radicular pain or neurological deficits) predominate;
2) *medial or myelopathic syndrome* – characterized by long tract signs and symptoms;
3) *a combined syndrome* – which includes both root and long tract signs and symptoms and is the commonest form;
4) *a vascular syndrome* – may not present any clear sensory or motor symptoms because of the variable injury to the cord resulting from vascular ischaemia;
5) *anterior syndrome* – painless weakness in upper extremities without accompanying weakness in the lower extremities. This is attributed to pressure affecting only the anterior horns of the grey matter of the spinal cord.

Time course of symptoms

The narrowing of the spinal canal itself does not usually cause any symptoms. It is when inflammation of the nerves occurs at the level of increased pressure that patients begin to experience clinical problems.

One of the first symptoms and also signs of cervical myelopathy is gait disturbance, especially in dark surroundings, when the optical control should be compensated for by the proprioceptive receptors in the feet. Cervical pain and mechanical signs are uncommon in cases of myelopathy. The onset of cervical spondylotic myelopathy is usually insidious and progressive, but may be step-like, with multiple remissions leading to an initial phase of deterioration followed by a stabilization that typically lasts for years and may not change thereafter in a small number of patients [17]. Thus, the degree of disability is established early in the course of cervical spondylotic myelopathy. In most cases the disability is mild, and the prognosis for these is good.

Clinical manifestations

The clinical definition of a myelopathy is the presence of long tract signs, which are the result of the spinal afferent or efferent (pyramidal) nerve tracts. Some myelopathic signs include hyperreflexia of deep tendon reflexes of the upper and lower extremities, increased muscle tone or clonus, and the presence of pathological reflexes, including Babinski's sign (plantar reflex) and/or Hoffman's sign.

The upper extremities may be affected with compression of the cervical spinal cord. Hoffman's sign is referred to as the "upper extremity Babinski's sign". It is elicited by stimulating the extensor tendon

to the third digit by forcible flexion of the distal phalanx, followed by a sudden release, resulting in a flexion and adduction of the thumb and concurrent flexion of the index finger. Sometimes there is flexion of the other fingers as well. The sign is incomplete if only the thumb or only the index finger responds. Hoffman's sign has a significant false positive rate [18], particularly in young women, and care must be taken with interpreting this sign [19].

There may be weakness and wasting of the hand muscles. Slow, stiff opening and closing of the fists may be seen [20]. It is not always appreciated, however well described by Ebara et al., that some patients with spinal cord compression will present with atrophy of the small muscles of the hands, described as "myelopathic hand", as a result of segmental anterior horn cell necrosis [17, 21].

There is often proximal weakness of the lower extremities (mild to moderate iliopsoas muscle weakness occurs in about 50%) and a spasticity of the lower extremity. Severe cases of cervical spinal canal stenosis can also cause paraplegia, and significant loss of function also rarely, if ever, occurs.

In more than 70% of the cases, the reflexes are hyperactive at a varying distance below the level of cervical spinal canal stenosis; clonus or Babinski's reflex may also be present. Additional signs of CSM include:

- *inverted radial reflex* – tested by stimulating the distal brachioradialis tendon through gentle percussion, producing hyperactive finger flexion;
- *finger escape sign* – provoked by placing the patient's arms forward with the elbow pronated. A positive sign is noted if the patient is unable to maintain their hands in an extended position with the third to fifth digits abducted;
- occasionally, patients describe an electric shock like sensation shooting down the spine, with flexion of the neck, known as *Lhermitte's sign*.

Myelopathy may also manifest as a loss of proprioception (80%), and, more commonly, gait or fine motor dysfunction, such as difficulty buttoning one's shirt or change in handwriting [15]. The early phase of CSM is also characterized by clumsiness and unsteadiness with gait. Severe muscle atrophy caudal to the level of stenosis is uncommon with spondylotic myelopathy, unless it is detected in much later stages. Therefore if atrophy is present, physicians must evaluate for fasciculations, particularly proximal to the level of stenosis (i.e. tongue), and exclude the possibility of amyotrophic lateral sclerosis.

Bladder sphincter symptoms are common (usually urgency), with anal sphincter disturbances being rare.

Differential diagnosis

Diagnosing degenerative disorders of the cervical spine starts with a good history and physical examination. Typically, patients demonstrate neck pain. This is the most common complaint. Unfortunately, neck pain is also a common complaint in the vast majority of people who have nothing more than a stiff neck. It is important to differentiate neck pain related to degenerative spinal disorders from other ailments (Table III). Some of these (e.g. spinal cord tumour) may be demonstrated radiologically. Asymptomatic cervical spondylosis is very common, and about 10% of cases of cervical spondylotic myelopathy are later found to be due to another disease process including: amyotrophic lateral sclerosis, multiple sclerosis, or subacute combined system disease [22].

The diagnosis is based on observation of the aforementioned neurological symptoms, a detailed physical examination and X-rays. A CT scan, MRI or a myelogram is necessary to confirm the diagnosis. This imaging studies are followed in some cases by

Table III. Main differential diagnosis of myelopathy

• **Congenital** – Arnold-Chiari malformation, tethered cord, syringomyelia, neuroenteric cyst
• **Acquired** – cervical or thoracic spinal stenosis, traumatic, herniated intervertebral disc, kyphosis, extramedullary haematopoiesis, epidural lipomatosis
• **Neoplastic** – spinal cord tumours, carcinomatous meningitis, paraneoplastic syndrome
• **Vascular** – haematoma, spinal cord infarction, spinal cord AVM, spontaneous thrombosis, radiation myelopathy
• **Autoimmune** – demyelating, multiple sclerosis, Devic syndrome, post-viral (or post-vaccination)
• **Metabolic/toxic** – combined system disease, local anaesthetics
• **Infectious** – (para)spinal abscess, osteitis/osteomyelitis, pyogenic discitis, AIDS-related myelopathy, tuberculosis, spinal meningitis, viral and syphilitic involvement
• **Peripheral neuromuscular disorders** – Guillain-Barre syndrome, myopathies
• **Degenerative** – amytrophic lateral sclerosis

AVM – arterio-venous malformation

other diagnostic studies such as laboratory investigations, neurophysiological or cerebrospinal fluid examinations for further differential diagnostic work-up. In this context, it must be emphasized that some degree of spondylotic changes is seen in 25-50% of the population over the age of 50 years, and in 75% of people over 75 years. Obviously, most people do not develop symptoms from these degenerative processes.

Management of cervical spondylosis

Medical management of cervical spondylotic syndromes, including axial neck pain, radiculopathy and myelopathy, typically includes pharmacological and rehabilitation components [23]. Non-steroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, analgesics, antidepressants and anticonvulsants are frequently used in non-operative management of these conditions.

Non-operative treatment option

A thorough understanding of the natural history of degenerative disorders of the cervical spine will allow appropriate treatment to be carried out. The natural history of untreated axial neck pain from cervical spondylosis is not known. With non-operative treatment, approximately 75% of patients have complete or partial, but significant, relief of symptoms. Treatment studies of patients with cervical spondylosis with mixed symptoms of axial neck pain, radicular symptoms, or both, treated non-operatively, suggest that 45 to 60% of patients have good resolution of symptoms, with the remainder continuing with moderate to severe residual pain [24, 25].

Non-operative treatment of spondylosis with radiculopathy has not been compared with surgical therapy in randomized trials [23]. A large epidemiological survey of cervical radiculopathy in Rochester, Minnesota, found that 75% of patients improved with conservative care and 20% were treated surgically. After 6 years of follow-up, 90% of patients were doing well [26]. In addition, Kadaňka et al. compared in a randomized study conservative and surgical treatment of spondylotic cervical myelopathy to establish predictive factors for outcome after conservative treatment and surgery [27]. The clinical, electrophysiological and imaging parameters were examined to reveal how they characterized the clinical outcome. The patients with a good outcome in the conservatively treated group were of older age before treatment, had normal central motor conduction time (CMCT), and possessed a larger transverse area of the spinal cord [27]. The patients with a good outcome in the surgically treated group had a more serious clinical picture. Patients should preferably be treated conservatively if they have a spinal transverse area larger than 70 mm^2, are of older age and have normal CMCT. Surgery is therefore more suitable for patients with clinically worse status and a lesser transverse area of spinal cord [27].

The natural history suggests that for the most part, patients with axial symptoms are best treated without surgery, while some patients with radiculopathy will continue to be disabled by their pain, and may be candidates for surgery. Myelopathic patients are unlikely to show significant improvement, and in most cases will show stepwise deterioration. Surgical decompression and stabilization should be considered in these patients.

With non-surgical therapy only 30-50% of patients are expected to stabilize (Table IV). However, there are a number of ways in which we can treat cervical degenerative disease leading to spinal stenosis without surgery. No carefully controlled trials have compared these modalities, so these therapies often are initiated based on a clinician's preference or specialty. Comparing the efficacy of these non-operative treatment options against no treatment at all is difficult.

In many cases, non-surgical treatment does not treat the conditions that cause the degenerative disorder of the spine; however, they might temporarily relieve pain. Severe cases of cervical stenosis often require surgery.

Surgical management

Operative management is indicated for progressive neurological deterioration, intractable pain and documented compression of nerve roots or of the spinal cord that leads to progressive symptoms [16]. Surgery has not been proven to help neck pain and/or suboccipital pain. Several approaches to the cervical spine have been proposed. The approach selected is determined by the type and location of pathology and by the surgeon's preference. Indications for surgery are primarily: (i) patients with progressive myelopathy who may be neurologically stabilized with surgery, and (ii) patients with pain.

Table IV. Cervical spondylotic myelopathy: main elements of conservative treatment

• **Immobilization** by a collar or brace (especially during the night; if possible <2 months)
• **Medications:** antiphlogistic (in the acute phase of the disease, 1-4 weeks), analgesic (up to 4 weeks), muscle relaxant
• **Physical therapy**

Cervical radiculopathy can be approached either via the anterior approach or by the posterolateral approach. The general sentiment is to treat anterior disease (e.g. osteophytic bar) with an anterior approach. Using this approach, the compressive factors should not exceed 2-3 disc levels. An anterior approach is technically more demanding, carries a higher risk, and often requires fusion. For performing a decompressive cervical laminectomy/laminotomy ("posterior approach"), the compressive changes should be present in more than 2-3 disc levels. So called "keyhole foraminotomies" are carried out at levels involved with radiculopathy. Intraoperative violation of the facet joints can result in the postoperative development of a so-called "swan-neck deformity" [28]. The posterior approach to cervical radiculopathy has similar results as the anterior approach when used for the proper indications. Surgical intervention for cervical myelopathy is controversial. Once moderate neurological signs and symptoms develop, surgical intervention is likely to be beneficial over further medical treatment.

However, the outcome of the surgical treatment is dependent on many factors and is often disappointing [29], even excluding cases that are later proven to have demyelinating disease. Most frequently discussed as potentially predictive factors for the outcome of surgery for CSM are age, duration of symptoms, preoperative clinical status, anteroposterior diameter of the canal and area of the spinal cord at the level of the maximal compression, findings of hyperintense areas in the spinal cord, one level or multilevel compression, congenital diameter of the spinal canal (expressed as Pavlov's index), and chosen method of decompression [27].

It is often contended that progression of myelopathy can be arrested by surgical decompression. This is not always true, and conservative treatment may be as effective as surgery, with laminectomy yielding improvement in 55%, change in 25%, and worsening in 20%, which is similar to results with conservative treatment [30, 31]. As discussed earlier, in the natural course of cervical spondylotic myelopathy, most of the deficit occurs early and then stabilizes in a considerable number of cases. For this reason, one may suggest conservative treatment in oligosymptomatic patients [29].

The situation in Africa

There are only a few studies in Africa on cervical spondylosis and its management. Taking into consideration age as one of the most important predictors of cervical spondylosis, the prevalence of cervical spondylosis does not seem to differ from that in other parts of the world. However, patients are often diagnosed very late in the course of their illness when irreversible changes and progressive neurological deterioration have already occurred. This may be due to the lack of diagnostic facilities and inadequate suspicion on the part of the care takers.

Pathophysiology

Daily life is different in Africa compared to Europe or North America. For example, carrying objects on the head is a very common practice in some African countries [32]. Taitz studied therefore the difference between cervical spines in cadavers of South African blacks and whites and found that the cervical vertebrae of blacks were significantly less affected than their white counterparts [33]. In addition, the distribution of osteophytosis in blacks is different from whites; osteophytes appear to affect either the vertebral body or apophysial joint facets in the cervical vertebrae of the blacks. In the whites, in sharp contrast, both sites are often affected on the same vertebra, which in life may result in a pincer-like entrapment of the spinal nerve root and/or vertebral artery [34]. But when we see lifestyles of Africans and other developing nations, we find some other risk factors for early vertebral degenerative changes. Jäger et al. evaluated in a case control study the relationship between load-carrying on the head and the development of degenerative changes in the cervical spine of 70 Ghanaians [35]. In 31 of the 35 (89%) carriers degenerative change was found in the cervical spine, but only in 8 of the 35 (23%) non-carriers, leading to the conclusion that the axial strain of load-carrying on the head exacerbates degenerative change in the cervical spine [35], with consecutive narrowing of the medullary canal [14]. Age and the weight carried seem to be important factors in the development of degenerative changes in this subpopulation [35].

Clinical manifestation

Ossification of the posterior longitudinal ligament at the cervical level is a rare condition in Caucasian people. The incidence of such endemic fluorosis in Africa is not known, but it seems that a substantial proportion of patients with cervical spondylosis are based on this origin. Haimanot et al. therefore studied the skeletal changes in fluorosis in Ethiopians living in the Rift Valley areas of the country, demonstrating that the changes are more severe in the cervical region and the clinical picture is similar to that of "classical" cervical spondylosis [36]. Neurological manifestations in the forms of myelopathy with and without radiculopathy (respectively 72% and 28%) occurred after exposure to high fluoride (>4 ppm) for longer than 10 years. So, fluorosis may complicate the aging changes in the vertebral columns and worsens the clinical picture.

Before the onset of overt neurological symptoms, a person with skeletal fluorosis develops progressive bone pains and stiffness of the whole body.

Squatting, particularly when defecating, becomes very troublesome. Standing up from a sitting position and sitting up in bed become increasingly difficult. At the stage when neurological symptoms and signs become manifested, the affected persons have marked kyphosis, with stiffness and restriction of movement of the whole body. Flexion deformity of the hip and knees and fixation of the chest wall are typically seen. In the progressive cervical radiculomyelopathy that develops insidiously, the common predominant neurological picture usually consists of marked wasting and atrophy of the small muscles of the hands, in addition to spastic paraparesis or quadriparesis, often in flexion. To a lesser extent, similar atrophies are seen in the lower limbs. In some patients, widespread fasciculation may be confused with motor neuron disease. The pathogenesis of the neurological sequelae of osteofluorosis is due to fluorotic changes resulting in considerable encroachment on the diameter of the intervertebral foramina and spinal canal, which are most marked in the cervical region [36].

The clinical signs in the case of load carrying seem to be similar to those described for fluorosis. But listhesis is seen in this subpopulation in up to 21% compared to 2% in controls, with spinal level C 4 affected in 60% of the cases [37]. Kopacz et al. [38] reported an incidence of 5% in asymptomatic (African) women with a homogeneous distribution at all levels.

Surgical management

Because of the lack of knowledge of this diseases in wide areas of Africa, there is little known about the conservative treatment of cervical spondylosis. But in a surgical treatment study, Loembe et al. reported the surgical outcome of 18 patients in Gabon. All patients presented various progressive spinal cord lesions (tetraparesis – 13, paraparesis – 4, tetraplegia – 1) and with spasticity [33]. The anterior surgical approach was ued in all 18 cases with an average follow-up of 6.9 years [33]. All patients obtained fusion, and stability was achieved after 3-5 months on average [33]. However, neurological recovery was complete in 10 cases, partial in 7 cases and unchanged in one case [33].

Conclusion

Not only in African patients, but also in all cases of cervical spondylosis, the decision making regarding the different treatment options is multifactorial [39]. The different origin of the disease in Africa also sheds light on the different treatment of these patients: in African countries, women may develop degenerative disc disease at the upper levels of the cervical spine, with increased incidence of listhesis at a younger age [37]. A better understanding of cervical spondylosis as presented in Africa will help therefore to determine which patients are suitable candidates for surgical intervention. There is a need for simple clinical and laboratory algorithms to assist in the identification of patients with a high likelihood of cervical spondylosis in Africa.

In conclusions, cervical spondylosis represents a degenerative disease of the cervical vertebrae, intervertebral discs and ligaments. In the majority of cases the degenerative process may not lead to clinical symptoms. But, it also causes disabling clinical problems such as myelopathy, radiculopathy and neck pains.

There are different options for managing the clinical problems, which can broadly be divided into conservative and surgical methods. Milder clinical conditions and those who cannot tolerate surgery are treated conservatively and those who are not responsive to the medical treatments and those with disabling neurological syndromes are treated surgically.

In Africa, the prevalence of the condition seems to be similar to that of other continents but patients may manifest early because of their lifestyles. Otherwise, treatment outcomes are similar.

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