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Cervical Spondylosis-A Review

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ABSTRACT

Cervical pain is a very common complain encountered in ENT outdoors. Cervical spondylosis is one of the leading causes presenting with cervical pain. The different possible presentations, evaluation, pathophysiology and treatment of this disease are discussed in brief.

Keywords: *Spondylosis, Cervical.*

INTRODUCTION

Degenerative changes in amphiathrodial joints and zygoapophyseal joints of cervical spine leads to an array of symptoms. This disease is named cervical spondylosis or cervical osteoarthiritis[1]. A lot of patients present in ENT outpatient department with various complaints pertaining to the disease. It affects 90% of population greater than 65 years of age[2]. In this article we review the presentation, evaluation, pathophysiology and management of the disease briefly from an ENT perspective.

Presentation

Most cases are asymptomatic[3]. They are usually presented as cervical pain, radiculopathy or myelopathy[2]. Cervical pain is mainly carried by supply of sinuvertebral nerve and posterior ramus branches[3,4]. Radiculopathy is due to compression of nerve roots. It may be sensory, motor or autonomic. Sensory involvement manifests as pain, paresthesia or hyperesthesia. Pain can be in neck, upper limbs, scapular region, head, in both arms upto fingers. Pain can be neuralgic which is sharp shooting in nature or myalgic, a dull pain in the corresponding myotome. Paresthetia follows the dermatome of the cervical nerve involved.

Myelopathy is due to compression of long tracts[5]. Motor symptoms range from stiffness, paraparesis to foot drop. Initial symptoms are upper motor neuron type while later symptoms are mostly lower motor neuron type[6-11]. Sensory symptoms range from headache, rotator vertigo, numbness, myoclonic jerks to hiccups[1].

Evaluation

Physical examination shows limited and painful rotation, lateral flexion and extension of neck. Palpation of cervical vertebrae may elicit tenderness and irregular bony projections. Spasm of

paraspinal muscles is a very common finding. Radiological investigation is the mainstay in suspected cases. Static and flexion-extension x-rays in antero-posterior and lateral views are the initial investigation. X-rays may show loss of intervertebral space height, kyphotic changes, osteophytes, facet arthropathy, and fusion of adjacent vertebrae[6, 7]. CT scans are used for the evaluation of spinal canal's shape and size, transverse foramina and joint spaces[2, 8, 9]. While MRI gives the better picture of the spinal canal dimensions, the spinal cord, intervertebral discs, and cervical soft tissues [2,10-11].

Pathophysiology

Three consequential general events were described by Sokoloff [1]. Initially there is a degeneration of the articular cartilage leading to denudation of the joint. Fibrillation of articular cartilage and remodeling of the periarticular structures including bone follows. The stiffness of the subchondral bone increases which leads secondarily to changes in the cartilage. Mechanical factors involved in the pathogenesis of cervical spondylosis have been categorized into two groups as static and dynamic factors[14] (Table 1).

Table 1: Mechanical factors in pathogenesis of spondylosis.

Static	Congenital canal stenosis, disc herniation, degenerative osteophytic growth, hypertrophy of the ligamentum flavum, calcification of PLL.
Dynamic	Abnormal forces on the spinal column and cord during movement.

The first event in the pathogenesis is decreased in concentration of proteoglycan of hyaline cartilage[13, 15]. Which results in shrinkage and loss of elasticity of nucleus pulposus and loss of disc height and loss of cervical lordosis. Loss of disc space is more pronounced in the ventral part[15, 16]. This in turn results in the additional pressure on dorsal part and ultimately kyphotic changes[15]. These lead to the reduction in sagittal spinal canal diameter which is the main pathophysiological change[3].

The other mechanism of reduction of spinal canal diameter is disc herniation. The degenerated disc usually herniates through the dorsal weak annulus fibrosis[17, 18]. Reactive bone formation or osteophytes projection into the spinal canal is the third contributing factor. This bone formation starts in the bare bone area where the posterior longitudinal ligament has peeled off the vertebral body edge[18, 19]. Mostly osteophytic formation is prominent in the area of C5-C6 and C6-C7 where cervical movements are maximum[17]. A fourth factor which often results in the decreased diameter is the congenital spinal canal stenosis[6]. This magnifies the effect of the degenerative changes and leads to early onset myelopathy.

Spinal cord compressions may be due to osteophytes or hypertrophied ligamentum flavum[14, 19]. Vascular compression symptoms may also surface due to compressions of anterior and posterior spinal arteries and other local vessels[6, 15].

Treatment

The disease is usually progressive in nature and the first intervention should be educating the patient about the natural course of the disease. Physical therapy like heat, ultrasound, heat pads, infrared lamps can be used to reduce the pain[1]. Analgesics, anti-inflammatories and muscle relaxants can be used depending on the intensity of pain. Stretching and range-of-motion exercises including cervical, thoracic and lumbar areas should be advised. In cases of patients with complications such as radiculopathy, myelopathy, vascular and compressive symptoms neurologic and orthopaedic consultations should be done urgently.

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