Cervical vertebral stenotic myelopathy (CVSM) is characterized by ataxia and weakness, caused by narrowing of the cervical vertebral canal in combination with malformation of the cervical vertebrae. Stenosis of the vertebral canal, anywhere from the first cervical vertebral body (C1) to the first thoracic vertebral body (T1), results in intermittent or continuous compression of the spinal cord and subsequent neurologic disease, is the most important abnormality in CVSM. Because malformation, malarticulation, or malalignment of one or more cervical vertebral bodies often leads to CVSM, the disease is also known as cervical vertebral malformation (CVM) or cervical vertebral instability (CVI).

CVSM is the leading cause of non-infectious spinal cord ataxia in the horse and is estimated to affect 2% of Thoroughbred horses. Two forms of spinal cord compression, dynamic or static, which were derived from three original classifications, Type I-III described by Rooney in 1969. In horses with dynamic stenosis compression of the spinal cord during myelography is recognized only during movement of the neck, particularly during flexion. Cervical static stenosis is defined by narrowing of the vertebral canal with subsequent compression of the spinal cord during myelography, regardless of the position of the neck. Although several sites are predisposed to the development of CVSM, narrowing of the cervical vertebral canal can occur at any site within the vertebral column, and stenosis at more than one site is not uncommon.

The pathogenesis of CVSM appears to be multifactorial. Etiologic factors such as genetic predisposition, hormonal changes, nutrition, trauma, and exercise are the most investigated. There has not been general agreement on the importance of degenerative joint disease of the cervical vertebrae in the pathogenesis of CVSM. In contrast to young horses in which the role of degenerative joint disease in the pathogenesis of CVSM appears controversial, in older horses (> 4 years) CVSM is generally associated with significant arthropathies of the caudal cervical articular processes leading to static or sometimes dynamic compression of the spinal cord. In these horses the compression of the spinal cord can be attributed to the bony and soft tissue proliferation at the affected articular processes. The genesis of the degenerative joint disease in older horses with CVSM could be caused by trauma, although may be related to long standing developmental problems.

CVSM has been reported in most light breeds, but is most commonly seen in Thoroughbreds, Quarter Horses, Warmbloods and Tennessee Walking Horses. CVSM has been reported to be more common in male horses. Young horses will generally present for acute onset of ataxia or gait abnormalities, however, mild ataxia and clumsiness may often go unnoticed. Owners often report affected horses are growing rapidly, well-fed, and large for their age. Physical examination may reveal abrasions around the heels and medial aspect of the thoracic limbs due to interference, and short, squared hooves due to excessive toe-dragging. Many young horses affected with CVSM have signs of developmental orthopedic disease such as physitis or physeal enlargement of the long bones, joint effusion secondary to osteochondrosis, and flexural limb deformities.