Rheumatoid Arthritis of the Cervical Spine

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Abstract

Cervical involvement in patients with rheumatoid arthritis occurs primarily in the upper cervical spine. The characteristic deformities are atlantoaxial subluxation, vertical settling, and subaxial subluxation. The typical patient complaints are neck pain and occipital pain. Subtle signs of myelopathy may also be present. Useful radiologic studies include plain radiography, tomography, and functional magnetic resonance imaging. The most helpful radiographic measurements are the anterior atlantodens interval, the posterior atlantodens interval, and assessment of vertical settling. Atlantoaxial subluxation greater than 9 mm with vertical settling and a posterior atlantodens interval less than 14 mm correlate with neurologic deficit. Nonoperative management does not change the natural history of cervical disease. Traditional surgical indications include intractable pain and neurologic deficit. The author discusses more controversial indications and proposes a rationale and protocol for treatment. The primary surgical objectives are to achieve stabilization of the affected segments and to relieve neural compression by reduction of subluxations or direct decompression. Arthrodesis provides reliable pain relief. Neurologic recovery occurs more consistently in patients with lower grades of preoperative myelopathy.

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Rheumatoid arthritis (RA) causes synovitis and pannus formation, which can lead to the destruction or incompetence of involved joints, ligaments, and bones. In the cervical spine, this may lead to subluxation, instability, and neural compression.

The frequency of cervical involvement in patients with RA has been reported to range from 34% in a general rheumatology clinic population to as high as 86% in a seropositive hospital-based population.[1,2] The determination of the frequency of cervical involvement depends on the patients studied, the radiographic techniques employed, and the radiographic criteria used. The clinical variable that correlates most closely with cervical involvement in a patient with RA is the presence of deformities of the hand, particularly metacarpophalangeal subluxations.[3-7]

When the cervical spine is involved, the anatomic manifestations and clinical symptoms and signs are varied. Although subluxations can involve any level, the upper cervical spine is the most frequently involved and the most clinically significant region. The occiput-C1 and C1-C2 articulations are exclusively synovial and are thus a primary target for rheumatoid involvement. The C1 and C2 facets are oriented in the axial plane; therefore, there is no bone interlocking to prevent subluxation. Stability at this level is dependent primarily on the ligamentous and capsular structures.

Three rheumatoid deformities of the cervical spine occur most commonly: (1) Subluxation of C1 on C2 can be the result of either an incompetent transverse ligament or erosion of the dens. This occurs usually as an anterior subluxation, but may be in a posterior or lateral direction. (2) A decrease in the longitudinal distance between the dens and the brainstem may be caused by erosion of the occiput-C1 or C1-C2 joint or both. The resultant deformity is variably described as pseudobasilar invagination, vertical settling, atlantoaxial impaction, cranial settling, or superior migration of the odontoid. (3) Subaxial subluxations (i.e., at levels below C2) are caused by facet joint erosion and ligament incompetence and can occur at one or more levels.

In addition to causing specific deformities, pannus also causes symptoms without deformity. The usual sites of involvement include the facet joints where the anterior

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aspect of the dens articulates with the anterior portion of the ring of C1, the bursae at the tip of the dens, and the bursae at the posterior portion of the dens anterior to the transverse ligament. The development of hypertrophic pannus at this level may compound any neurologic compression caused by instability. Inflammatory changes at these locations can lead to the development of instability at C1-2 due to either incompetence of the transverse ligament or erosion of the dens with subsequent loss of osseous integrity.

The most dangerous consequence of these rheumatoid deformities is brainstem or spinal cord compression. Steel's "rule of thirds" divides the anteroposterior diameter of the ring of C1 (approximately 3 cm) into three parts: one third is occupied by the dens, one third is occupied by the spinal cord, and one third is available space in which large motions can occur.[8] Neural compression may be caused by anterior or posterior subluxation or vertical settling of vertebrae, which compromise the available space. Cord compression between the C1 lamina and the dens can occur with anterior subluxation of C1 on C2. Compression may also occur without subluxation as a result of pannus compressing the anterior aspect of the cord, usually associated with posterior erosion of the dens.

Epidemiology and Natural History

Progressive neurologic deficits have been reported to be present in 15% to 36% of patients with RA.[9,10] The prevalence of progressive cervical subluxations is greater, reportedly ranging from 43% to 80% on the basis of findings in radiologic surveys.[11-13] Thus, many patients with radiographic evidence of instability do not have neurologic deficits. Therefore, it is necessary to understand the natural history of cervical rheumatoid disease to determine the probability of subsequent neurologic deterioration. This information is one factor in the decision as to whether surgical intervention is necessary.

Davis and Markley[13] first documented death as a consequence of medullary compression caused by atlantoaxial subluxation due to RA. In a later postmortem study of 104 patients with RA, Mikulowski et al[14] identified 11 (11%) with atlantoaxial subluxation and cord compression. Sudden death secondary to a hyperflexion event was documented in 7 of the 11 cases. In all 7, the postmortem demonstrated that medullary compression by the odontoid was the proximate cause of death.

Boden et al[15] evaluated the data on 73 patients with established rheumatoid cervical spine involvement who were followed up for 20 years. Neurologic deficit developed in 42 patients (57%) (Ranawat class II in 11 and class III in 31). Of the 7 patients treated nonoperatively, all had an increase in the severity of paralysis. Marks and Sharp[16] found that more than 50% of their patients with documented cervical myelopathy died if they did not undergo surgical stabilization. Therefore, patients with rheumatoid cervical myelopathy are at risk for premature death but not necessarily due to a direct neurologic cause. Only operative intervention favorably affects survival rate.[15,16]

Clinical Manifestations

A careful history is necessary to establish the presence or absence of symptoms that may result from rheumatoid cervical involvement. Neurologic symptoms may include occipital neuralgia caused by compression of the greater occipital branch of C2. This typically causes patients to complain of occipital headaches when upright that are relieved by recumbency. Ear pain may result from involvement of the greater auricular branch of C2; facial pain, as a result of impingement of the trigeminal nucleus (associated with vertical settling). Visual and equilibrium disturbances may be caused by vertebrobasilar insufficiency; paresthesia, by spinothalamic tract compression. Urinary retention, followed by incontinence, is caused by bulbar involvement. Patients sometimes complain of electric shock–like sensations in the torso or extremities (Lhermitte's sign) associated with neck motion if there is a mobile subluxation. Vertical nystagmus and Cheyne-Stokes respirations are late signs of brainstem compression.

The examination of patients with RA may be difficult to perform and interpret because of the presence of other neuromusculoskeletal manifestations of the disease. Patients frequently have muscle atrophy due to either rheumatoid disease or prolonged corticosteroid therapy. Multiple peripheral joint deformities, tendon subluxation, and tendon ruptures may make it difficult to evaluate the function of a specific muscle group. Peripheral entrapment neuropathies often develop as a result of joint subluxations or the presence of inflammatory tissue adjacent to a nerve. Pathologic changes in reflexes resulting from pyramidal tract involvement (Hoffmann and Babinski) can be difficult or impossible to elicit because of joint ankylosis or tendon ruptures.

Because of these complexities, a gradual loss of function is sometimes attributed to progressive peripheral joint disease, when in fact it may be the result of progressive myelopathy. Electrophysiologic studies, particularly somatosensory evoked potential (SSEP) testing, may be helpful in determining the presence of a myelopathy; however, SSEP testing is useful only if the results are positive.[17] The classification of rheumatoid myelopathy developed by Ranawat et al[18] (Table 1) is helpful in characterizing deficits and arriving at a prognosis.

Table 1
Classification of Rheumatoid Myelopathy Developed by Ranawat et al[18]

Class	Clinical Characteristics
I	No neural deficit
II	Subjective weakness with hyperflexia and dysesthesia
IIIA	Objective weakness and long-tract signs; ambulatory patient
IIIB	Objective weakness and long-tract signs; nonambulatory patient

Radiologic Evaluation

Radiologic techniques can be helpful in defining rheumatoid cervical deformities and in helping to identify patients at risk for neurologic deficit.[15,19]

The lifetime risk of having a radiologically demonstrable progressive deformity has been reported to be between 43% and 80% for all patients with RA.[11,12] This range of risk of progression is so wide because many investigators have evaluated only atlantoaxial subluxations and have therefore failed to recognize the potentially more serious deformity of vertical settling, which can occur alone or in combination with atlantoaxial subluxation.

Plain Radiography

The radiographic evaluation should include lateral cervical spine films in the flexed and extended positions to assess the presence and potential reducibility of any subluxation (Fig. 1). Several measurements made on plain films are useful in planning clinical management and defining operative indications, most notably (1) the anterior atlantodens interval (AADI), (2) the posterior atlantodens interval (PADI), (3) the McGregor line, (4) the Ranawat measurement, and (5) the Redlund-Johnell measurement.

The AADI is measured on a lateral radiograph from the anterior aspect of the dens to the posterior aspect of the midportion of the anterior ring of C1 (Fig. 2, A). The normal value for this is 3 mm in the neutral position in the adult.[20] The PADI is the distance between the posterior surface of the dens and the anterior edge of the posterior ring of C1 as seen on a lateral radiograph.

A B

Fig. 1 Reducible atlantoaxial subluxation in a patient with occipital pain and no myelopathy. **A,** Flexion radiograph shows an anterior atlantodens interval of 11 mm. **B,** Extension reduction to normal position.

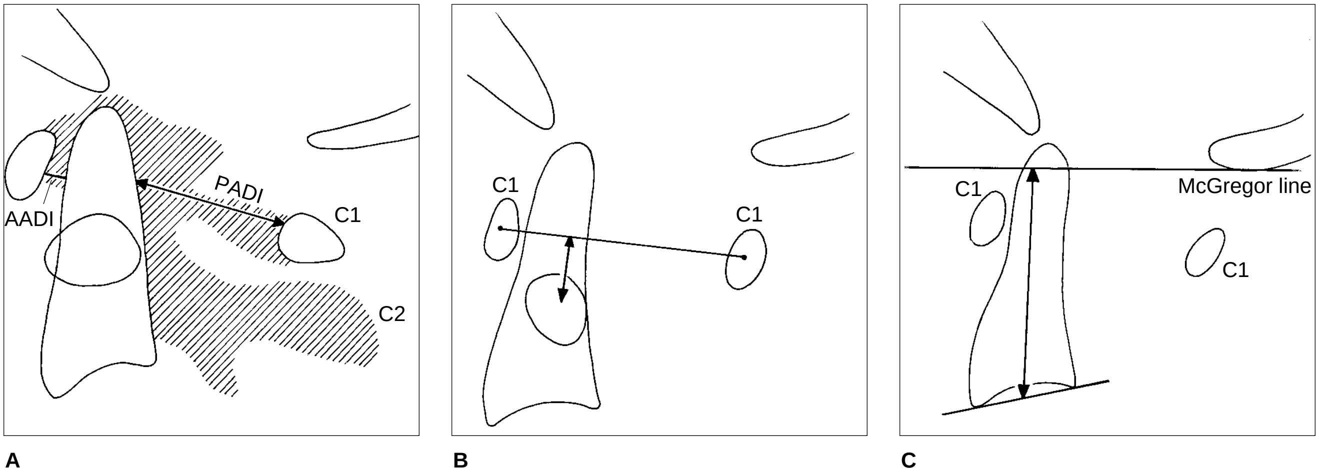


Fig. 2 **A,** Measurement of AADI and PADI. **B,** The Ranawat method for measurement of vertical settling. **C,** The Redlund-Johnell method for measurement of vertical settling.

The subaxial canal diameter is a similar measurement made on a lateral radiograph of the subaxial (i.e., below C2) spine. It is measured from the posterosuperior aspect of a vertebral body to the spinolaminar junction of the vertebra above.

A variety of radiographic lines (e.g., McGregor, McRae, Chamberlain, Wackenheim, Fischgold-Metzger) have been used to measure vertical settling between the occiput and the atlantoaxial complex. The McGregor line, probably the most commonly used, is drawn from the posterior edge of the hard palate to the most caudal point of the occipital curve. These landmarks are readily identified on a routine lateral cervical radiograph. Vertical settling is defined as migration of the tip of the odontoid more than 4.5 mm above the McGregor line. The disadvantage of these measurements is that the osseous landmarks are often obliterated (e.g., the tip of the dens can be completely eroded) or indistinct.

Two additional useful measures of vertical settling, the Redlund-Johnell and Ranawat measurements, can be used together to delineate the articulation (occiput-C1 or C1-C2 or both) involved in vertical settling. The Ranawat value is determined on a lateral cervical spine radiograph (Fig. 2, B). The coronal axis of C1 is marked by connecting the center of the anterior arch of C1 with the center of the posterior arch of C1. A second line starts at the center of the sclerotic ridge of C2 (the pedicles) and extends superiorly along the vertical axis of the odontoid until it intersects the first line. A value of 13 mm or less signifies vertical settling.

The Redlund-Johnell measurement is the minimum distance between the McGregor line and the midpoint of the inferior margin of the body of the axis (C2) (Fig. 2, C). A value less than 34 mm in men and less than 29 mm in women signifies vertical settling.

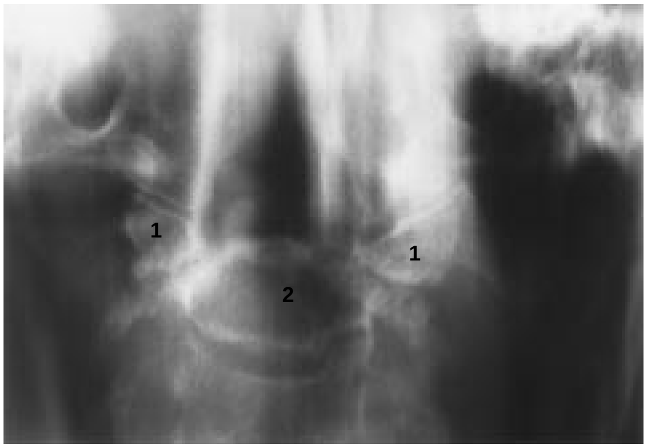
Tomography

Radiographic interpretation may be difficult because of osteopenia, deformity, and obliteration of joints, which make identification of anatomic landmarks difficult. Temporomandibular joint involvement may make an open-mouth view difficult or impossible to obtain. In these instances, anteroposterior and lateral tomography is very helpful in delineating the osseous anatomy (Fig. 3).

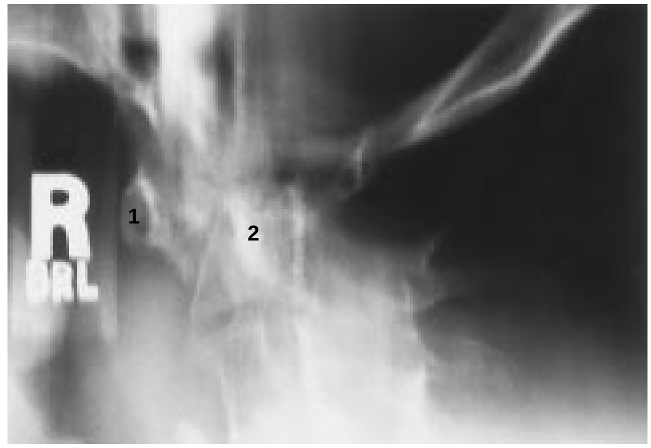
Computed Tomography

Computed tomography (CT) can provide valuable information regarding osseous detail and extent of erosions. Because it is performed in the axial plane, which is the plane of the deformity, it is less helpful in defining axial and sagittal relationships. However, sagittal and coronal reconstructions may be helpful. Fairly fine sections (1 to 2 mm) are needed to ensure that the spatial resolution of sagittal reconstruction is sufficient for adequate visualization. The use of intrathecal contrast material enhances the ability of CT to demonstrate areas of neural compression.

The role of myelography in combination with CT in identifying neural compression has mostly been supplanted by the development of magnetic resonance (MR) imaging. The combination of myelography and CT is most useful in patients whose condition presents a specific



A



B

Fig. 3 **A,** Anteroposterior tomogram shows destruction of C1-2 articulation and erosion of the dens in a patient with class IIIB myelopathy. **B,** Lateral tomogram of same patient shows erosion of the dens. AADI = 10 mm; PADI = 10 mm. Vertical settling is evident; Ranawat value = 10 mm.

contraindication to MR imaging or who have hardware in the cervical spine that will degrade the MR image and obscure anatomic areas of interest.

Magnetic Resonance Imaging

Magnetic resonance imaging is extremely useful in identifying the presence and etiology of cord compression and whether it arises from the osseous elements or from the presence of granulation tissue. This modality has the advantage of requiring no contrast medium. Furthermore, images can be reformatted in any plane. The craniomedullary junction and the entire length of the cervical cord can be visualized. Functional MR imaging, whereby images are obtained in the flexed and extended positions, is now gaining utility.[21-23] If neural compression is present in only one of these positions, it might otherwise be missed. Therefore, MR imaging allows evaluation of not only cord compression but also cord configuration (Fig. 4).

One measure of cord distortion is the cervicomedullary angle, which is measured on a midsagittal cervical MR image. The angle is formed by a line drawn parallel to the anterior aspect of the cervical cord and a line drawn along the anterior aspect of the medulla. The normal angle is 135 to 175 degrees. An angle of less than 135 degrees is a sign of vertical settling and has been correlated with clinical evidence of myelopathy.[19,23,24]

An MR imaging study should be obtained if there are neurologic signs or symptoms. A PADI or subaxial sagittal canal diameter less than 14 mm on plain radiographs is an indication for a flexion-extension MR imaging study to evaluate the true space available for the cord. In patients with vertical settling, an MR study obtained in flexion is advisable to quantitate the amount (if any) of spinal cord compression.

Predictive Value of Imaging Techniques

Despite the fact that there is a greater incidence of radiographic evidence of progression than of neurologic progression, there are certain radiographic measurements that predict the risk of neurologic compromise. The AADI, which has been used traditionally, does not correlate with paralysis. However, vertical settling in association with an increased AADI increases the risk of cord compromise.[9]

Fig. 4 Midsagittal cervical MR image shows erosion of dens with vertical settling. Cervicomedullary angle is 125 degrees. Cord compression is seen between dens and posterior ring of C1, as well as at the top of the dens.

More accurate and reliable predictors of whether a patient will have neurologic compromise are the PADI and the subaxial sagittal canal diameter. In a study of rheumatoid patients with long-term follow-up, Boden et al[15] found that a subaxial sagittal canal diameter or PADI of 14 mm or less was 97% sensitive in predicting the presence of a neurologic deficit. Just as important, a patient with a canal diameter or PADI of 14 mm or more had a 94% chance of being neurologically intact. It is important to remember that the PADI and the spinal canal diameter are measured on plain radiographs and are not equivalent to the space available for the cord (seen on MR images) because the thickness of synovial pannus is not visible (hence, is not measurable) on plain films. The PADI has also been found to be a good predictor of postoperative neurologic recovery. In the study by Boden et al, no neurologic recovery was seen in patients with a preoperative PADI of 10 mm or less, whereas neurologic recovery of at least one class occurred in patients with a preoperative PADI of 10 mm or more. If basilar invagination was part of the deformity, neurologic recovery occurred only if the PADI was greater than 13 mm.

Treatment Goals and Options

The goals of management are to avoid the development of an irreversible neurologic deficit, to avoid sudden death secondary to unrecognized cord compression, to relieve pain, and to avoid unnecessary surgery. The options for treatment of structural abnormalities caused by RA in the cervical spine are limited. Medical management, physical therapy, and use of orthotic devices do not affect the development or progression of the structural abnormality and often do not relieve symptoms.[11] Orthotic management may be difficult due to problems with initial fitting because of the micrognathism and the short necks of many patients. Use of orthotics may also cause secondary problems, such as the development of temporomandibular joint syndrome. In addition, rheumatoid patients often have very friable skin, which tolerates orthotics poorly.

Operative Treatment

Indications and Techniques

Surgical intervention is considered by many to be appropriate for patients who have intractable pain or neurologic deficits. Indications for surgical intervention in patients without neurologic compromise but with radiographic evidence of instability or structural abnormalities are more controversial. On the basis of an understanding of the natural history of the disease process and the radiographic risk factors for neurologic deficit, reasonable recommendations for surgical intervention can be made for this group of patients. The following scheme for surgical intervention addresses these indications according to the specific anatomic abnormality present.

If surgery is selected, the goals include reduction of subluxations, decompression of compromised neural tissue, and stabilization to maintain realignment and relieve pain. The use of continuous preoperative traction with skull tongs may be beneficial in reducing subluxations and relieving cord compression in patients with myelopathy. This should be done with careful radiographic and neurologic monitoring. At the time of surgery, fiberoptic intubation should be performed with the patient awake to minimize uncontrolled cervical motion. Manipulation or attempted reduction in the operating room should be avoided.

Patients with pain and a reducible atlantoaxial subluxation but without neurologic deficits may benefit from a posterior C1-C2 fusion. Techniques to accomplish stabilization include the use of Gallie or Brooks wiring configurations, a Halifax clamp, or transarticular screws.[25-28] Placement of transarticular screws is technically demanding.[26] Our preferred technique is modified Gallie wiring with the use of 18- or 20-gauge wire and autologous grafting.

Patients with a fixed atlantoaxial subluxation and neurologic deficit or a partially reducible subluxation and a PADI of 14 mm or less require decompression. This is accomplished by a C1 laminectomy, performed with use of a high-speed diamond burr, and stabilization of C1 and C2 with transarticular screws and autologous grafting.[26]

Patients with both atlantoaxial subluxation and vertical settling should be considered for surgical stabilization because of the higher risk of neurologic compromise that may not be reversible and can be fatal.[15,16] Fusion should extend from the occiput to C2 if vertical settling is present. Preoperative radiographs should be carefully evaluated for subluxation of adjacent segments, and the fusion should extend caudad to include all subluxated segments (i.e., they should be fused to a normal level). In patients with a PADI of 14 mm or less or symptomatic cord compression, a C1 laminectomy should be performed in addition to the occipital cervical fusion. Continued observation may be appropriate for patients who have isolated and fixed basilar invagination but no symptoms and no evidence of cord compression.

The techniques for performing a posterior occipitocervical fusion include wiring with shaped iliac-crest bone grafts, occipitocervical plating, and the use of rod-loop constructs with wiring.[29,30] Wiring constructs have been successful in achieving solid fusion in these patients, who often have very poor bone stock[29] (Fig. 5). They require postoperative immobilization with an external orthosis, such as an extended Philadelphia collar or a halo vest. Occipitocervical plating provides a more rigid construct but requires adequate bone stock for good purchase of the screws.[30] It is a technically demanding technique and requires a thorough knowledge of upper cervical and occipital anatomy for safe screw placement.[31] Patients with plating and a stable construct need only a soft collar postoperatively. Thus, occipitocervical plating can be useful if the need for a halo vest is to be avoided postoperatively.

Fig. 5 Lateral radiograph obtained after solid posterior fusion from occiput to C2 for atlantoaxial subluxation and vertical settling. Wiring and onlay graft were used.

Soft-tissue pannus that causes neurologic compromise frequently resolves after a solid posterior fusion has been obtained.[32] However, patients with vertical settling and a solid posterior fusion who have a persistent neurologic deficit and symptomatic cord compression from the odontoid may be considered for an anterior odontoid resection. This can be done through a transoral or high retropharyngeal approach.[33] The results of anterior resection of the odontoid have been mixed,[15,34] which may be due to the severity of the preexisting disease rather than to the procedure itself.

Subaxial subluxations should be evaluated by assessing the subaxial canal diameter and the space available for the cord. If plain radiographs demonstrate a canal diameter of 14 mm or less and flexion-extension MR images show the space available for the cord to be 13 mm or less or if a patient has intractable pain, a posterior fusion should be considered because of the documented risk of neurologic deficit in patients with these particular characteristics.[15] A decompressive laminectomy may be added if necessary on the basis of the neurologic symptoms.

Anterior decompression should be performed only in selected cases, because it may lead to increasing kyphosis and failure of the anterior fusion construct.[18,33] This is attributable to osteoporosis of the vertebral bodies and the difficulty of obtaining solid fusion anteriorly. If an anterior procedure is contemplated, strong consideration should be given to the addition of a posterior stabilization procedure to minimize the risk of failure of the anterior construct. Anterior decompression and fusion may be considered for patients with residual anterior neurologic compression and persistent symptoms after either preoperative traction or a previous stabilization.

Outcomes

Surgical fusion with decompression when necessary has been very reliable in providing relief of neck and occipital pain. Modern techniques, with their improved internal fixation, result in pain relief for 85% to 100% of patients.[18,29,32,34-37] However, surgical intervention is much less reliable in providing neurologic improvement.[15,18,34] Most studies have shown that the degree of neurologic improvement is related to the initial deficit.[15,18,37] If one looks at recovery by specific class of myelopathy, 60% to 100% of class II patients will show improvement, compared with only 20% to 60% of class III patients.[15] Studies have reported 40% to 50% improvement in the neurologic function of myelopathic patients after surgery.[15,18,34]

One study has shown a correlation between neurologic recovery and the PADI.[15] A preoperative PADI of 10 mm or more is predictive of neurologic recovery from atlantoaxial subluxation, and a preoperative PADI of 14 mm or more is predictive of recovery if there was any associated vertical settling. These studies emphasize the importance of recognizing the development of neural compression in its earliest stages to optimize the outcome of surgical intervention.[18,33]

Complications

Historically, there has been a high incidence of perioperative complications in patients with RA.[36,38] This is related to several factors; probably the most important is that only those patients with severe myelopathy were treated surgically. Earlier recognition of neurologic compromise, operative stabilization before progression of neurologic deficit, and modern anesthetic techniques have all contributed to lowering the perioperative mortality to between 2% and 10%.[15,18,30]

Other complications include infection, wound dehiscence, and loss of reduction. The incidence of pseudarthrosis has been reported to range from 0% to 50%. The use of grafting with rigid fixation techniques has resulted in better fusion rates.[15,18,30,36,37] Halo vests have been helpful in leading to better fusion rates, although they are not a panacea. Late subaxial subluxations can occur below a fused segment.[39] Because of this risk, all anatomically involved levels should be incorporated in the fusion even if they are distal to the primary area of pathologic change.

Summary

Cervical spine involvement is not uncommon in patients with rheumatoid arthritis. A careful history and physical examination are important, with special attention to subtle signs of myelopathy. Lateral cervical radiographs can be helpful in evaluating the presence of deformities. Useful measurements made on plain films include the AADI, the PADI, and the Ranawat and Redlund-Johnell measurements. Magnetic resonance imaging is helpful in identifying the presence and etiology of cord compression.

Nonoperative treatment does not affect the natural history of the disease process. Decompression when appropriate and fusion at unstable levels can give reliable pain relief. However, the likelihood of neurologic improvement is dependent on the grade of preoperative myelopathy.

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