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An Overview about Spinal Cord Lesions

Mostafa Mohy El Deen, Atef El Kelany, Sherif Abd El Raaof, Ahmed Esam Nasr Mohamed

Department of Neurosurgery, Faculty of Medicine, Zagazig University, Egypt.

Corresponding author: Ahmed Esam Nasr Mohamed

Email: ahmedelzorokany@gmail.com

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Abstract: Background: The spinal cord connects the brain and peripheral nervous systems, processing and transmitting motor and sensory information, and modulating reflex responses. Spinal cord damage through compression or other mechanisms causes dysfunction in the form of pain, limb weakness, numbness, bladder/bowel dysfunction, and irregular gait. Clinical outcomes vary with magnitude, type of injury, severity, location and biomechanical origin. There is a lack of data describing the epidemiology and aetiology of NTSCI. This is likely to be due to the wide range of causes, the lack of national registries, and the secondary nature of NTSCI leading to patients not always being admitted to specialist spinal cord injury units. The prevalence of NTSCI has been estimated to be 1120 per million and 2310 per million in the only two studies undertaken. Annual incidence is estimated to be between 6 and 76 cases per million. Since the incidence of traumatic injury is estimated to be between 10 and 40 cases per million per year, NTSCI affects the same number or more people annually than traumatic injury. Demographic studies have identified that NTSCI patients have median ages of 62 to 67 years old; with an even gender divide. In TSCI, approximately 80% of patients are males and the mean age is between 38 and 52 years old

Keywords: *Spinal Cord Lesions*

Introduction

The spinal cord connects the brain and peripheral nervous systems, processing and transmitting motor and sensory information, and modulating reflex responses. Spinal cord damage through compression or other mechanisms causes dysfunction in the form of pain, limb weakness, numbness, bladder/bowel dysfunction, and irregular gait. Clinical outcomes vary with magnitude, type of injury, severity, location, and biomechanical origin [1].

Spinal cord injuries can be classified by velocity. High-velocity insults, up to 4.5 m.s-1, cause traumatic spinal cord injuries (TSCI). Conversely, slow-velocity insults where trauma is not involved, with development over a timescale between months to years, can be classified as non-traumatic spinal cord injuries (NTSCI). NTSCI are predominantly caused by chronic illness and disease, with 50% caused by degenerative diseases and cancers. The key features which distinguish NTSCI from TSCI, as determined by [2], are:

No acute mechanical insult, slow velocity compression

Absence of gross hemorrhagic necrosis
Chronic and progressive nature (likely induces compensatory mechanisms)
There is a lack of data describing the epidemiology and etiology of NTSCI. This is likely due to the wide range of causes, the lack of national registries, and the secondary nature of NTSCI leading to patients not always being admitted to specialist spinal cord injury units. The prevalence of NTSCI has been estimated to be 1120 per million and 2310 per million in the only two studies undertaken. Annual incidence is estimated to be between 6 and 76 cases per million [3].
Since the incidence of traumatic injury is estimated to be between 10 and 40 cases per million per year, NTSCI affects the same number or more people annually than traumatic injury. Demographic studies have identified that NTSCI patients have median ages of 62 to 67 years old, with an even gender divide. In TSCI, approximately 80% of patients are males and the mean age is between 38 and 52 years old [4].
Spinal cord injuries are a complex clinical problem, affecting quality of life and lifespan. Alongside the damage caused by injury, complications and secondary illnesses occur frequently in spinal cord injury, and vary based on the type of injury. Urinary tract infections, spasticity, and pressure ulcers affect at least 20% of NTSCI patients [5].
Mortality in all patients with spinal cord injury is up to three times greater than the general population, with significantly higher levels of mortality in NTSCI patients than TSCI patients. This difference in patients with NTSCI may be due to the increased age of NTSCI patients, as well as an increased rate of comorbidities in such populations. In patients with NTSCI, treatment is limited and predominantly involves surgical decompression and fixation. These interventions may arrest injury progression, but neurological deficits remain, and one in twenty patients are at risk of severe neurological complications [6].
Differences between Traumatic and Non-Traumatic SCI
The demographic and clinical pattern of neurological damage in patients with NTSCI differs considerably from those with TSCI. For example, TSCI occurs predominantly in young men between the ages of 16–30 years with almost equal numbers sustaining tetraplegia/paraplegia and complete/incomplete injuries. More men sustain TSCI than women (about 21% are female) [7].
NTSCI tends to affect older adults with a more even gender distribution. Injury is usually incomplete and more likely to cause paraplegia than tetraplegia. NTSCI is associated with reduced secondary complications such as spasticity and deep vein thrombosis, but among older adults, other illnesses and conditions, and general deconditioning can seriously affect functional outcome [8].
This has implications for patients' management and rehabilitation so that their individual needs are assessed adequately, and their rehabilitation program is tailored to them. An understanding of the disorders and diseases that can cause SCI (Table 1) and their likely progress and outcomes will enable members of the rehabilitation team to meet patients' needs. There is a wide range of causes of NTSCI [5].
Individuals with NTSCI are more likely to be older, female, married, and retired when compared to those with TSCI. The etiologic presentation of NTSCI may assist in explaining these differences, as spinal stenosis and cancerous compression of the spinal cord more commonly involve individuals in the fifth decade and beyond. Cancer-related SCI has a peak incidence between 50–70 years of age. SCI in persons younger than 50 years of age is more commonly due to traumatic etiology [9].
Given the likelihood of NTSCI to affect older individuals, factors such as demographics, neurological presentation, concomitant illness, and rehabilitation functional outcome become important issues for consideration. Older age has been shown to affect rehabilitation outcome after SCI. Elderly individuals with NTSCI may present with associated medical complications, such as cardiopulmonary disease or diabetes, which could adversely affect medical and functional outcomes [10].
Additionally, the use of medications in older individuals, who may have decreased tolerance, must be closely monitored to prevent side effects. Medications such as those utilized to control spasticity or pain can lead to

sedation. Memory and retention may be worse in older individuals and could result in diminished rehabilitation efficiency and decreased functional improvement [11].

The Vertebral Column

The vertebral column is an aggregate of articulated, superimposed segments, each of which is a functional unit. The function of the vertebral column is to support a man in an upright position, mechanically balance to conform to the stress of gravity, permit locomotion, and assist in purposeful movements [12].

The head is located over the body of the sacrum, and the spine in an upright manner bears an axial load to support the body. Loss of this spinal balance produces a position that is at a biomechanical disadvantage. To stand upright and look forward while standing and walking, the patient with sagittal plane imbalance causes back muscle to strain to successfully or unsuccessfully reduce a patient's sideways tilt [13].

The additional energy expenditure associated with standing and walking in patients with a spinal deformity leads to reduced functional capacity, including pulmonary function and a poorer quality of life. The definition and scope of spinal deformity continue to evolve. Certainly, the term spinal deformity includes conditions such as idiopathic adolescent scoliosis, congenital scoliosis, post-traumatic deformities, and other adult spinal deformity, including post-infective kyphosis [14].

Kyphosis

Kyphosis is an increase in the forward curvature of the spine seen in the sagittal view . Kyphosis is a physiologic curvature of the thoracic spine, and its connection with cervical and lumbar lordosis curves above and below make up the length of the spine above the sacrum. In the sagittal view, thoracic kyphosis normal angular range falls between 20° and 40°. Hyperkyphosis is defined as a Cobb angle greater than 50° [15].

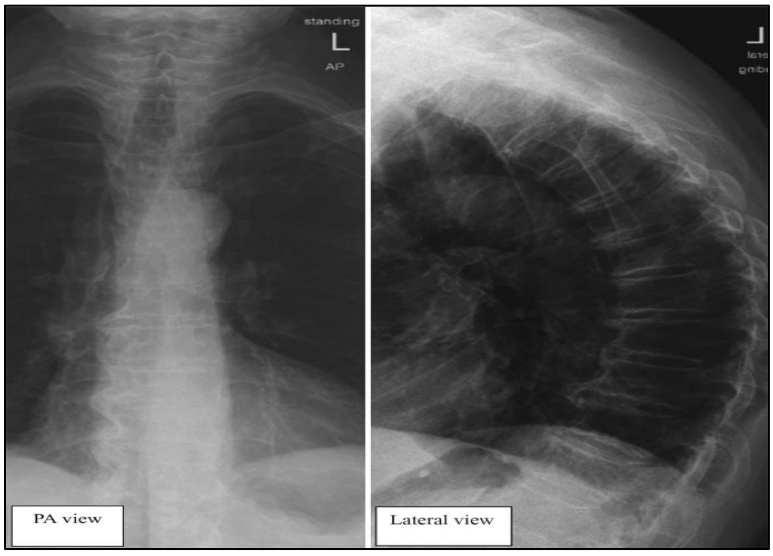

Figure (1): PA and lateral views of Spine. On PA view, no lateral deformity is noted, but the lateral views demonstrate kyphosis [15].

The thoracic spine is less flexible as compared to the cervical and lumbar spine where its attachment to the ribcage is a source for its immobility. The spine comprises 7 cervical vertebrae, 12 thoracic vertebrae, 5 lumbar vertebrae, a fused sacrum, and fused coccyx. From the age of 40, physiologic kyphosis progresses, and the kyphotic angle increases with age [16].

Hyperkyphosis is prevalent in 20–40% of the population after age 60. It affects women more than men and progresses more in women of menopausal age. Menopause accelerates bone turnover, and low bone mineral

density is a risk factor for fracture and kyphotic progression. About a third of patients with osteoporosis have compression fractures [15].

Compression fractures cause wedging of the vertebral body, putting pressure on additional segmental levels in the spine and increasing the risk of future fractures and progression of the kyphotic angle. Each vertebral fracture can increase the kyphosis angle by 3.8°. Conversely, nonmetabolic kyphosis may not be a risk fracture for compression fractures [17].

In kyphosis, postural imbalance with anterior displacement of the center of mass increases the risk of falls and non-spine fractures. Other risk factors include degenerative disk disease, weakness of back extensor muscles, older age, and a family history of hyperkyphosis (Table 2) [18].

Postural kyphosis is related to adolescent slouching and forward head tilt, creating weakening of the extensor muscles of the upper back. The vertebral structures are normal in shape and form in this diagnosis. As the spine maintains flexibility, this deformity can be corrected [19].

Juvenile kyphosis, also known as Scheuermann's disease, is a thoracolumbar structural deformity occurring before puberty. The vertebral structures demonstrate anterior wedging due to aberrant bone mineralization. This spine is rigid compared to postural kyphosis and restrictive to correction [20].

Congenital kyphosis from anomalies is rare, however disabling and rapidly progressing. The result of either a lack of spinal segmentation or failure of segmental development is usually diagnosed during childhood [21].

Adult kyphosis can be from degenerative, metabolic, iatrogenic, and post-traumatic, autoimmune from ankylosing spondylitis, neurologic from neurofibromatosis, and infectious from tuberculosis. Metabolic causes are kyphosis from osteoporosis. Degenerative and metabolic etiologies comprise a majority of adult-related kyphosis [22].

Potts kyphosis is vertebral infection from tuberculosis resulting in segmental damage causing severe thoracic or thoracolumbar kyphosis. Segmental junctional kyphosis is an iatrogenic kyphosis after spinal fusion surgery causing collapse of the proximal vertebral segments [23].

Complications of hyperkyphosis include pulmonary compromise with restricted vital capacity, putting patients at risk of pneumonia and COPD. Kyphosis angulation increases energy expenditure, making household chores and activities of daily living (ADL) cumbersome. Neurologic compromise can be expected in severe kyphotic angles >90°, and in some cases as early as beyond 50° [24].

Symptoms

Pain and stiffness are common symptoms from spinal stress due to kyphotic changes. Shortness of breath may be present due to reduced capacity, limited lung expansion, and rib immobility. Fatigue is common from increased energy expenditure due to kyphotic angulation. Cord compression should be suspected in cases of hyperkyphosis with bowel incontinence, bladder retention, and progressive gait deficits [25].

Physical Examination

a) Inspection

Patients present with a forward head and neck tilt with a rounded back or humpback deformity. Increased lumbar lordosis and forward pelvic tilt are also present. Also, true height is reduced from forward riding head tilt, and neck extension may be limited due to muscle weakness [26].

b) Musculoskeletal Examination

Muscle examination would reveal tight hamstrings and hip joint contractures with pelvic obliquity due to compensation efforts to correct sagittal imbalance. Patients would demonstrate tenderness to palpate the thoracolumbar paraspinal muscles, including facet joints above and below the apex of the curve. Belly protrusion from weak abdominal muscle and chest wall collapse may be evident on observation [27].

c) Special Tests

Special tests to differentiate rigid and flexible kyphosis would be laying the patient in the supine position and observing flattening and correction of flexible kyphosis and retention of curvature for rigid kyphosis [25].

Diagnostic Studies

a) Plain Films

Standing X-ray in the lateral view is the gold standard for assessing kyphosis using imaging. The Cobb angle is the gold standard to measure the degree of kyphotic angle and wedging between vertebral segments. It is measured from the superior endplate of T2 to the inferior endplate of T12. Serial X-rays are useful to monitor the immature adolescent spine, especially during growth spurt for kyphosis correction. Advanced imaging modalities are considered if complications are suspected [28].

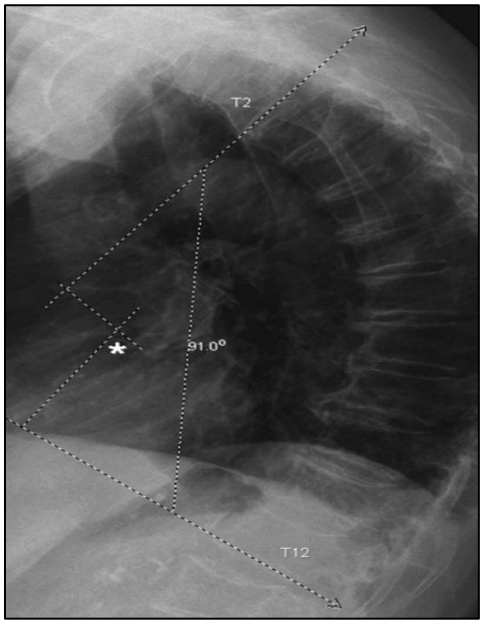

Figure (2): Cobb angle measurement (Star) in a patient with kyphotic deformity[28].

b) MRI/CT

MRI with and without contrast or CT with myelogram (for MRI incompatibility) are considered if cord compression is suspected or for infectious etiologies such as Potts kyphosis. Advanced MRI or CT imaging is also useful if surgical correction is being considered. Pulmonary function tests help measure lung compromise from the restrictive effect of progressing kyphosis [29].

Treatment

a) Medical Management

Bracing is appropriate for the adolescent child with kyphosis, where corrective bracing can retard its progression. The Milwaukee brace is commonly worn for extended daily periods until skeletal maturity is reached. If kyphosis fails bracing, surgical correction may be required [30].

Modalities and TENS can assist in desensitizing pain. Nonsteroidal anti-inflammatory drugs (NSAIDs) and Tylenol are first-line oral pain medications for general pain conditions and can be trialed for kyphosis-mediated pain. Topical lidocaine and Flector patches at the site of maximal tenderness can help reduce nociceptive-mediated pain. Opioids are not recommended due to long-term unfavorable sequelae from opioid dependence, and further compromise of respiratory function [31].

Bisphosphonate management is recommended for patients with osteoporosis and kyphosis. Bisphosphonates stall osteoporosis progression, thereby stalling kyphosis progression and fractures. In addition, calcium and vitamin D reduce the risk of osteoporosis-related fractures [32].

b) Rehabilitation

Physical therapy emphasizes improving flexibility and postural alignment and strengthening weak back extensors. In patients with osteoporosis, flexion exercises should be avoided as they could potentiate fractures. Studies suggest strengthening rather than stretching exercises are beneficial for kyphosis correction. Prone-based exercises, which strengthen the middle and lower trapezius, and scapular exercises including scapular retraction and trunk stabilization are emphasized. Studies suggest 2–3 physical therapy sessions per week for 8–12 weeks [33].

Balance and gait training are integral as falls are a common predisposition in this population. Restrictive lung compromise is prevalent in advanced kyphosis, and diaphragmatic breathing should be encouraged to improve pulmonary function. Modern digital technologies with phone apps are useful for short exercise video clips for spine strengthening and posture at the convenience of home [34].

c) Procedures

Radiofrequency (RF) Ablation

Progressive segmental degeneration is present with neck and upper back pain from facet joints. If cervical and thoracic facet is suspected as a source of pain, a medical branch block can aid with diagnoses, and treatment, including RF ablation, could be offered [35].

Trigger Point

Myofascial pain in the cervical or thoracic region from compensation could be addressed with muscle relaxants and trigger point injections [36].

Bone Augmentation

Acute vertebroplasty or kyphoplasty are indicated for acute pain management in the setting of a recent compression fracture that may lead to exaggerated kyphosis. Kyphoplasty improves pain and may prevent further kyphotic angulation due to progressive anterior vertebral collapse [37].

d) Surgery

Age and comorbidities are factored into surgical considerations. Some indications in the juvenile and adolescent population include limited conservative relief for at least 6 months, progressive deformity and debility, kyphosis progression despite corrective bracing, and curves greater than 70° [30].

Corrective surgery is not recommended in the elderly due to comorbidities and an unfavorable risk-safety profile, however, is considered on an individual basis. Other surgical indications include debilitating pain, severe disability from cardiopulmonary compromise, and progressing neurological deficits [30].

Surgery involves pedicle screw fixation and osteotomy using a posterior, anterior, or combined approach. As long-term stabilization is the goal, these surgeries typically involve spinal fusion. The surgeries can be performed in single- or two-stage approaches for correction of deformity. They involve restoration of segmental alignment, sagittal rod contouring, and osteotomies [34].

Scoliosis

A normal spine is generally straight and has no appreciable lateral curve on coronal radiographs. A lateral curvature of the spine is considered scoliosis. A leftward curved spine is levoscoliosis, and a rightward curved spine is dextroscoliosis. A deviation from a vertical line could also be combined with a spinal rotation defined as scoliosis with a rotatory component. In some patients, segmental lateral listhesis is noted in addition to the abnormal scoliotic curve [38].

Scoliosis can develop in any segment of the spine, although thoracic and lumbar scoliosis is more often recognized by clinicians. Patients may have an isolated levoscoliosis or isolated dextroscoliosis, but often on X-rays, a combination of levoscoliosis and dextroscoliosis is seen in the same patient as spine curves in one direction proximally and in the opposite direction distally (S or reversed S shape). According to the Scoliosis Research Society, at least 10° of scoliosis should be notable on the PA radiograph to be considered diagnostic 39].

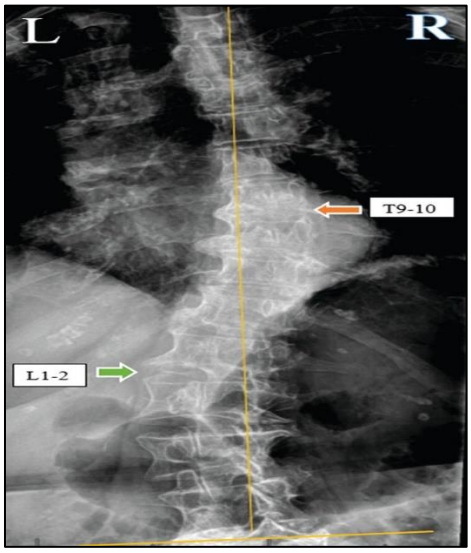

Figure (3): Reversed S scoliotic curve with dextroscoliosis centered at T9–10 disk space and levoscoliosis centered at L1–2 disk space [40].

In general, scoliosis is either congenital, idiopathic, secondary to other conditions, or degenerative (Table 3). In congenital cases, it is related to abnormally developed vertebrae resulting in a scoliotic curve. Secondary causes of scoliosis could be related to neuromuscular/neurological disorders such as cerebral palsy, muscle abnormalities as in Duchenne muscular dystrophy, and some genetic disorders including Marfan syndrome and neurofibromatosis [40].

Idiopathic scoliosis is by far the most commonly seen type in MSK clinics, and there is no underlying etiology for this type of scoliosis. Juvenile idiopathic scoliosis in ages 4–10 comprises 10–15% of all the idiopathic scoliosis in children, and if untreated may have cardiopulmonary consequences. Furthermore, curvature of 30° or more in this age population tends to progress, of which 95% would likely require surgical intervention [41]. Scoliosis has a genetic component in which siblings have up to 7 times more frequency of developing scoliosis, children of patients with scoliosis have 3 times more prevalence of having scoliosis, and counseling pregnant females with scoliosis should include education on potential scoliosis and their offspring [42].

Pediatric orthopedist/physiatrist closely monitors the progression of the scoliosis in children, and clinical determination is made to monitor, brace, or operate depending on the degree of curvature, the rapidity of the progression, the patient's age, and the Tanner stage. Degenerative scoliosis often develops in the 6–8th decade of life, due to osteoarthropathy of the facet joints, vertebral compression fracture, and desiccated disk spaces with asymmetrical intervertebral spacing [43].

Clinical Presentation

Scoliosis is often symptom-free but can be associated with complaints such as asymmetrical shoulders, asymmetrical pelvis, leg length discrepancy, chest wall/breast asymmetry, back asymmetry, unilateral scapular protuberance, and in degenerative scoliosis, a complaint of getting shorter [44].

Presenting symptoms may be pain at the apex of the scoliosis convexity. If a patient has an S-shaped scoliosis, pain may be on either of the two convexities of the curve. Unless the scoliosis is associated with severe rotation or lateral listhesis, numbness, tingling, or weakness is not a presenting symptom. In a severe scoliotic curve, when cardiopulmonary function is compromised, shortness of breath and early fatigue with activities could be a presenting symptom, which can be validated by pulmonary function testing [45].

Physical Examination

A thorough musculoskeletal examination is necessary. The height of the patient and the Tanner stage should be documented and followed in time as they are part of monitoring skeletal growth and the risk of curve

progression. Examination includes detection of out-of-norm findings, including excessively tall patients with perhaps long fingers (signs of Marfan), any skin abnormalities that may relate to secondary scoliosis (such as neurofibromatosis), abnormal joint laxity (Ehlers–Danlos syndrome), evaluation of gait, and evaluation of the foot and its arch, which may point to genetic causes of scoliosis [46].

In patients with severe scoliosis, auscultation of the heart and lungs at both apex and distal lung is indicated, and ventilation in all quadrants of the lung should be documented. A thorough sensory/motor/reflex testing should be performed. Expectations are normal findings in scoliosis, unless there is any particular nerve root impingement in which dermatomal/myotomal deficits may be noted [47].

Skeletal examination includes identifying asymmetry in both coronal and sagittal planes. In the coronal plane, asymmetrical shoulders, asymmetrical pelvis, and asymmetry in the spine either S or reversed S curve are noted. In the sagittal plane, attention should be made to any rotation of the spine, and any asymmetry in the chest wall, breast, scapular protuberance, and rib cage, all should be documented and tracked in serial examinations [48].

Observation should include side-to-side asymmetry in the anterior superior iliac spine (ASIS)/posterior superior iliac spine (PSIS), which leads to leg length discrepancy. Leg length discrepancy should be measured to aid with shoe lift prescription (measurement between ASIS and medial malleoli) [43].

Diagnostic Workup

a) Plain Films

Plain radiograph is a standard diagnostic test for scoliosis. Scoliosis series include full-length anteroposterior (AP) and full-height left lateral spine views. It could be done in a standing or decubitus position. The Cobb angle is measured when the patient is followed over time. Repetitive Cobb angle measurement determines the progression of the curve. The Risser sign (ossification of the iliac apophysis) should be documented on X-ray as it identifies the degree of spinal skeletal maturity, which is the clinical information for the management of adolescent scoliosis [49].

b) CT Scan

This modality requires high-dose radiation. CT is reserved for patients prior to surgical planning, potential congenital anomalies, and in cases with spondylolisthesis and a rotatory component. Sagittal and coronal reformatting would add additional value. In a post-fusion state, if there is concern regarding nonfusion, CT with 2D coronal and sagittal reconstruction may be utilized [50].

c) Ultrasound

There is no indication for diagnostic ultrasound in scoliosis; however, it may avoid inadvertent pneumothorax while performing trigger point injections in the paraspinal muscles through visualization of the needle tip within muscle layers, in patients with significant rotatory scoliosis of the thoracic spine [51].

d) MRI

In degenerative spine with scoliosis, additional findings, including stenotic central canal and foraminal impingement, can be identified, and the extent of degenerative disk disease could be fully evaluated [52].

Table 3: (Please provide details about Table 3 here, such as a description of what it contains or a link to the table.)

Treatment

a) Medical Management

In degenerative scoliosis, facet arthropathy and asymmetrical muscle firing may lead to symptomatic scoliosis, including pain. In this group, in particular, anti-inflammatories (oral or topical) and muscle relaxants may be used. Latter treatment options include spinal manipulation, utilization of a TENS unit, therapeutic massage, and rehabilitation efforts. Opioids are generally not indicated [53].

b) Bracing

The goal of bracing in scoliotic patients is to halt the progression of the curve before reaching skeletal maturity. Other goals include improving cosmetically and reducing the risk of prolonged symptomatic scoliosis (cardiopulmonary and pain). Examination, Cobb angle, Risser sign of skeletal maturity, and Tanner stages all play a role in the determination of bracing and the timing of it (Table 4) [54].

The effectiveness of bracing in early nonoperative management has been established by the BrAIST RTC study, level 1 research. It was demonstrated that 18 hours/day bracing was approximately 72% successful in halting curve progression as compared to 48% in the control arm that received no brace and was simply observed. There was a significant positive association between hours of brace wear and the rate of treatment success [55].

Braces are customized to the patient. Brace type and extent of bracing depend on the type of curvature (single versus L-shaped and with the segment of spine). The most common type of brace is a thoracolumbosacral orthosis (TLSO), appropriate for thoracolumbar S-shaped curvature. Several types are available, and research projects have been done to compare efficacy. These braces include the Boston brace, the Wilmington brace, Charleston brace, and the Providence brace. Ultimate success will lie on proper customization/fitting, proper monitoring, and compliance of the patient with the brace [56].

Table 4: (Please provide details about Table 4 here, such as a description of what it contains or a link to the table.)

c) Rehabilitation

Exercises are individualized according to patient needs, curve pattern, and treatment phase. Engagement in frequent and regular sports activity has been confirmed as a positive component in helping to minimize curves in young patients [57].

Physiotherapy Scoliosis Specific Exercises (PSSEs) have been used in the USA in conjunction with spinal orthotic management in the treatment of progressive idiopathic scoliosis. The combination of the two modalities may offer advantages over more simplified treatment plans. PSSEs have also been applied for adult patients with pain associated with scoliosis [58].

According to the 2016 Society on Scoliosis Orthopaedic and Rehabilitation Treatment (SOSORT) consensus and guidelines, the common principles of PSSE involve autocorrection, elongation, and chest wall expansion with the integration of the “corrected” posture into daily life activities. SOSORT also endorses the usage of exercises in the postsurgical rehabilitation period. It has been reported that patients who experience pain 10 or more years after scoliosis surgery can reduce the pain frequency through a multimodal treatment, including stabilizing postural and respiratory exercises [59].

Other “schools” of scoliosis physiotherapy have evolved, the Schroth technique, Schroth-Barcelona School, SEAS in Italy, and DoboMed and FITS in Poland, all of which incorporate exercise-based treatment [57].

d) Procedures

Myofascial pain in the paraspinal muscles adjacent to the scoliotic region could be addressed with trigger point injections. Pain from degenerative facet in the context of scoliosis can be treated with standard treatments for facet-mediated pain, including RF ablation of medial branches [60].

e) Surgery

Severe and/or progressive scoliosis often needs surgery. Criteria for surgical care are outlined in the Scoliosis Research Society Recommendations. This includes curves greater than 50° progressing even after skeletal maturity, curves of greater magnitude causing loss of pulmonary function, and much greater curves causing respiratory failure [61].

Posterior fusion with instrumentation has been a standard surgical treatment for scoliosis using Harrington rods. In modern instrumentation systems, anchors are used to connect the rod and the spine, resulting in better correction and less frequent implant failures. Segmental pedicle screw constructs or hybrid constructs using pedicle screws, hooks, and wires are commonly done today [62].

Spondylolisthesis

Spondylolisthesis is a condition in which one vertebral body slips in relation to another. There are two common types of spondylolisthesis as it relates to lower back pain and leg pain: degenerative spondylolisthesis and isthmic spondylolisthesis. Other causes of spondylolisthesis include congenital, traumatic, pathologic, and postsurgical. In degenerative spondylolisthesis, the bones gradually start to slip in relation to one another. It is most common in people over age 50, more common in women (by a rate of about 3:1), and it most commonly occurs at L4–L5 [63].

In isthmic spondylolisthesis, a stress fracture in the spine at the bilateral pars interarticularis allows the vertebral bodies to slip in relation to one another. The pars interarticularis is critical to maintaining the integrity of the spinal alignment because it connects the facet joint above to the facet joint below. The facet joints prevent anterior-posterior translation of the bones, and so loss of the integrity of this unit allows the bones to start to shift [64].

Isthmic spondylolisthesis most commonly occurs at L5–S1 and is more common in males. Fractures of the pars interarticularis tend to occur in young athletes who participate in sports that involve repetitive extension such as gymnastics, ballet, volleyball, rowing, diving, and football. Approximately 8–15% of asymptomatic adolescents have been reported to have pars interarticularis stress fractures (spondylolysis) [65].

In adolescents with lower back pain, the incidence of spondylolysis has been reported to be as high as 47%. For fractures of the pars interarticularis to lead to an isthmic spondylolisthesis, it generally (though not always) involves a bilateral fracture. The degree of spondylolisthesis is graded based on the degree of slippage of the vertebral bodies [66].

It is important to realize that most people with spondylolisthesis have no symptoms. Some reports estimate that as many as 80% of people with a spondylolisthesis have no pain. However, spondylolisthesis can cause symptoms. When symptomatic, patients generally present with extension-based lower back pain. Sometimes, the pain will radiate into the legs and may be accompanied by radicular symptoms, such as numbness, tingling, and/or weakness [67].

Grades I and II spondylolisthesis are very common. Grades III and IV spondylolisthesis are rare (especially in the absence of significant trauma), accounting for less than 2% of all cases, and an asymptomatic grade III or IV spondylolisthesis is exceedingly rare (though not unheard of). An asymptomatic spondylolisthesis does not require treatment, although grades III and IV asymptomatic cases should be evaluated closely for neurologic compromise and followed for progression of the listhesis or the development of neurologic compromise [68].

If a spondylolisthesis is present in a symptomatic patient, flexion and extension views of the lumbar spine are often obtained in order to evaluate for movement (instability) of the lumbar spine. If movement is present, this indicates some degree of instability and additional chronic mechanical stresses being placed on the spine and generally indicates a more difficult treatment course. Ultimately, movement on flexion and extension radiographs makes a good physical therapy program all the more important in order to use the muscles to help support the spine [69].

When a spondylolisthesis is suspected of causing axial lower back pain, initial conservative care is appropriate. Physical therapy that focuses on lumbar stabilization exercises, hip flexor, and knee extensor stretching can be helpful. Physical therapy will sometimes incorporate passive modalities, such as soft tissue mobilization, electrical stimulation, and ultrasound to help with the pain and inflammation [70].

If symptoms persist, and interventional procedures are being considered, then the first mode of diagnosis and treatment is to rule out the more common causes of symptoms, including the disc, facet joint, and sacroiliac joints. These should be systematically interrogated with fluoroscopically guided diagnostic blocks [71].

While the disc is the more common source of pain epidemiologically, it is reasonable to start by blocking the facet joints, as this is both a more conservative injection and, in the presence of a spondylolisthesis, it seems

reasonable (though the data here is lacking and so the reader should take this point as conjecture) that it is more common to cause back pain than in patients without a spondylolisthesis [72].

If other more common causes of lower back pain have been ruled out and the history, physical examination, and imaging studies are consistent with spondylolisthesis causing the pain but the pain is not improving with aggressive conservative care, then surgical alternatives may include a laminectomy but generally involves fusion surgery in order to stabilize the spine [67].

When leg symptoms, including pain, numbness, tingling, and/or weakness, is a prominent component of the problem, and spondylolisthesis is present on imaging studies, then the spondylolisthesis is generally contributing to irritation of the affected nerve root. In this instance, treatment involves addressing the resultant radiculopathy. This generally begins, as with axial lower back pain, with conservative care including physical therapy [73].

If symptoms persist, an epidural steroid injection in which the steroid is delivered to the affected nerve root may be performed. When an injection is used, it is generally coupled with physical therapy with the intent that the injection alleviates the swelling and inflammation around the nerve root and the therapy exercises help support the spine so that the inflammation does not return. If radicular symptoms persist, as with spondylolisthesis causing only lower back pain, surgical alternatives may include a laminectomy but generally involves fusion surgery in order to stabilize the spine [74].

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