Review Article

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Adolescent Idiopathic Scoliosis

Safak Ekinci [1], Omer Ersen [2]

[1] Department of Orthopedics and Traumatology
Ağrı Military Hospital
Ağrı, Turkey

[2] Department of Orthopedics and Traumatology
Erzurum Military Hospital
Erzurum, Turkey

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Corresponding author:
Safak Ekinci, MD
Department of Orthopedics and Traumatology
Ağrı Military Hospital
Ağrı, Turkey
safakekinci@yahoo.com

Abstract

Scoliosis is called idiopathic when no other underlying disease can be identified. The etiology of adolescent idiopathic scoliosis (AIS) is still unknown despite many years of research effort. Theories on AIS's etiology have included mechanical, hormonal, metabolic, neuromuscular, growth, and genetic abnormalities. Skeletally immature patients with adolescent idiopathic scoliosis are at risk of curve progression. The adolescent onset of severe idiopathic scoliosis has traditionally been evaluated using standing posteroanterior radiographs of the full spine to assess lateral curvature with the Cobb method. Scoliosis in children of school age and above primarily occurs in girls. The therapeutic goal in children is to prevent progression. In children, scoliosis of 20° or more should be treated with a brace, and scoliosis of 45° or more with surgery.

Key words: *Idiopathic scoliosis, spine, brace*

Introduction

Scoliosis is a three-dimensional deviation of the spinal axis. The Scoliosis Research Society (SRS) has defined adolescent idiopathic scoliosis as occurring in patients of 10 years or above and with an idiopathic structural lateral curve of at least 10° measured with the Cobb technique as well as vertebral rotation on a standing longitudinal radiograph of the spine combined with asymmetry on forward bending [1]. Curves smaller than this which are within normal variation tend to be asymptomatic, and are less likely to progress [2].

It has been seen that 1% to 3% of the adolescent population, more commonly in girls and as suggested by the name, has no known etiology. This definition provides a starting point for treatment decisions in the growing spine [3]. Left untreated in the growing child, numerous studies have demonstrated the negative long-term prognosis

a progressive curve fosters into adulthood, including back pain, pulmonary compromise, cor pulmonale, psychosocial effects, and even death [4-6].

Idiopathic scoliosis is a diagnosis of exclusion: this condition is diagnosed only when the history and the clinical and radiological findings do not provide clear evidence for any specific etiology. The major types of non-idiopathic scoliosis are congenital scoliosis due to malformation or faulty segmentation of the vertebrae and neuromuscular scoliosis due to muscular imbalance [7].

Curve progression is the most important factor in the natural history of idiopathic scoliosis. The risk of curve progression in idiopathic scoliosis has been associated with factors that predict potentially remaining spinal growth; therefore, skeletally immature patients with idiopathic scoliosis and major curves are at risk of progression and warrant some form of treatment [8,9].

This article is intended to give the reader an understanding of the clinical manifestations, prognosis, diagnostic evaluation, and the options for the treating of idiopathic scoliosis.

Epidemiology, Etiology and Pathogenesis

Male and female infants are equally affected by infantile scoliosis, but girls tend to be more commonly affected with increasing age, so the sex ratio from age 10 onward is already 6:1. There is a similar development with respect to the severity of curvature: boys are somewhat more likely than girls to have mild scoliosis, yet the ratio of girls to boys among children with spinal curvatures greater than 20° is 5:1, rising to 10:1 in children whose spinal curvature exceeds 30° [7].

The etiology of adolescent idiopathic scoliosis is still unknown despite many years of research effort. Theories on AIS's etiology have included mechanical, hormonal, metabolic, neuromuscular, growth, and genetic abnormalities [10].

Genetic Factors

Although the specific cause of adolescent idiopathic scoliosis (AIS) has not been established, the role of genetic or hereditary factors in its development is widely accepted [11]. Studies have documented an increased incidence of scoliosis in families [10]. Some studies have suggested autosomal dominant and X-linked modes of inheritance [12,13], while others have suggested a multifactorial or polygenic mode of inheritance to explain the wide variability in presentation of scoliosis amongst family members [14]. In a study of four multiplex families with AIS, Wise et al. found evidence of allele sharing at three loci (chromosomes 6 p, 10 q, and 18 q) in the affected individuals from two of these families [13].

Given the strong epidemiological data demonstrating familial clustering, the current opinion is in favor of AIS being a complex genetic disorder, with one or more genes interacting with the environment to result in spinal deformity [10].

Neurological Mechanisms

Dysfunction of the central nervous system can produce scoliosis. Studies involving electromyography and corticosensory-evoked potentials in patients undergoing surgery for AIS have demonstrated abnormal and asymmetrical latencies, correlating with the side and indeed the progression of the scoliosis [15]. These findings suggest a problem of the mid-brain and/or spinal cord, wherein primary neurological pathologies might cause a functional asymmetry in balance and consequently result in scoliosis [10].

Hormonal Influence

Growth hormone is normally produced by the anterior pituitary, which in turn acts via stimulation of the liver to produce somatomedins 1 and 2. Several studies have found an increase in the level of growth hormone or somatomedins in adolescent girls [16,17]. In addition, sporadic cases of a rapid increase in scoliotic curvature have been reported in patients undergoing growth hormone therapy [18]. However, Misol et al. found no differences in growth hormone levels between patients with AIS [19]. Despite these findings, the underlying reasons are still unknown, and the precise role of growth hormone abnormalities in the etiology of AIS remains uncertain [10].

Role of Melatonin

The role of melatonin in human biology concerns circadian rhythm, sleep disturbances, affective disorders, sexual maturation, aging and age-related diseases, tumor growth (malignancy), cardiovascular system, bone structure, interaction with calmodulin, and AIS [20]. The melatonin is produced mainly in the pineal gland and a small portion in the retina. The synthesis

and release of melatonin are stimulated by darkness and inhibited by light. Melatonin is rapidly metabolized, chiefly in the liver, by hydroxylation to 6-hydroxymelatonin. The urinary excretion of 6-sulfatoxymelatonin (the chief metabolite of melatonin) closely parallels serum melatonin concentrations [21]. In humans, melatonin has diurnal variations. The hormone secretion increases soon after the onset of darkness, peaks in the middle of the night, between 2 and 4 a.m., and gradually falls during the second half of the night.

Measurements of melatonin levels in patients with scoliosis have been equally controversial, with most studies showing no abnormalities in melatonin levels in adolescent patients with scoliosis [22]. The hypothesis of melatonin deficiency as a causative factor in the etiology of AIS cannot be supported by the data [20].

Role of Calmodulin

Calmodulin, a calcium-binding receptor protein, has also recently been implicated in the development of AIS. Calmodulin regulates the contractile properties of muscles and platelets. Increased calmodulin levels in platelets have been shown to be associated with curve progression [23]. Cohen et al. suggested that the platelet calmodulin level may be a better predictor for progression of the curve than the Risser sign alone [24]. Overall, while there appears to be abnormalities in growth hormone, sex hormone, melatonin, and calmodulin, their precise roles in the pathogenesis of AIS remain inconclusive [10].

Prevalence

Scoliosis is present in 2 to 4 percent of children between 10 and 16 years of age. The ratio of girls to boys with small curves of 10 degrees is equal, but increases to a ratio of 10 girls for every one boy with curves greater than 30 degrees [25]. The prevalence of curves greater than 30 degrees is approximately 0.2 percent, and the prevalence of curves greater than 40 degrees is approximately 0.1 percent [26].

Natural History and Prognosis

The three main determinants of progression are patient gender, future growth potential and the curve magnitude at the time of diagnosis. In all cases, females have a risk of curve progression which is 10 times higher than males [26].

Evaluation of growth potential is done by assessing the Risser grade. The Risser grade (zero to 5) gives a useful estimate of how much skeletal growth remains by grading the progress of bony fusion of the iliac apophysis. The iliac apophysis ossifies in a predictable fashion from anterolateral to posteromedial along the iliac crest [27]. Risser grades are as follows: grade zero signifies no ossification, grade 1 signifies up to 25 percent ossification, grade 2 signifies 26 to 50 percent ossification, grade 3 signifies 51 to 75 percent ossification, grade 4 signifies 76 to 100 percent ossification, and grade 5 signifies complete bony fusion of the apophysis. In one study, the Risser grade was directly correlated with the risk of curve progression [28]. In patients who have stopped growing, scoliosis of less than 30° can generally be considered stable, while scoliosis of more than 30° can be expected to progress at a rate of approximately 1° per year [7,29].

The effect of scoliosis on morbidity and mortality in old age has not yet been adequately studied. It was once assumed, on the basis of studies in heterogeneous patient populations, that patients with untreated adolescent scoliosis would necessarily become wheelchair-dependent in old age and were likely to die of cardiopulmonary arrest for reasons related to scoliosis [7].

History and Physical Examination

The treatment of scoliosis with longitudinal traction was first described by Hippocrates in the 5th century BC [30]. His early work was devoted to the use of traction with the Hippocratic bench for the treatment of long bone and spinal fractures [31].

Galen added direct pressure in combination with traction [32]. Variations of devices were used up until the 16th century, as alternative modalities were limited or nonexistent. The first supportive braces used to treat spinal deformity were developed by Ambrose Pare´ (1510–1590), a French army surgeon. He described a method of reducing the "dislocation" using extension and directed pressure [33].

The most important development of the study and treatment of scoliosis was Wilhelm Conrad Roentgen's 1895 discovery of an unknown type of radiation he called "x-rays" [34]. The most common etiologies of spinal deformity in the early 20th century were tuberculous and polio [35].

In 1914, Russell Hibbs performed posterior spinal

fusion to treat patients with scoliosis. In 1931, Russell Hibbs, Joseph Risser, and Ferguson published their work on the treatment of scoliosis in 360 patients using cast immobilization with posterior spinal arthrodesis [36]. In 1958, Risser described the progressive lateral to medial ossification of the iliac apophysis followed by its fusion with the ilium and the correlation of this with spinal skeletal growth [37].

In 1950, Ponseti and Friedman reported on the natural history of idiopathic scoliosis in 394 patients [38]. They found that curve pattern, age at onset, and rapidity of curve progression were important factors to consider in determining prognoses of patients with scoliosis. Ponseti and Friedman's findings helped to establish treatment guidelines for the nonoperative and surgical treatment of patients with adolescent idiopathic scoliosis.

In 1946, Walter Blount described the use of a removable cervicothoracolumbosacral orthosis (CTLSO), and published his experience of using the Milwaukee brace in the nonoperative treatment of adolescent idiopathic scoliosis with his colleagues in 1958 [39].

Symptoms and Physical Findings

The history and physical examination are intended to exclude secondary causes of the spinal deformity. The patient should be asked about a family history of scoliosis, menstrual onset, and the presence of pain and neurologic changes, including bowel and bladder dysfunction [27]. Idiopathic scoliosis only rarely causes pain in children and adolescents, and often comes to attention only because of the hunched rib cage and lumbar bulge that it causes, or because of asymmetry of the shoulders, chest or pelvis. The symptoms in adults depend on the level at which the maximal curvature is located [7]. The direction of curves in adolescent idiopathic scoliosis is remarkably consistent. The Cobb angle is considered the gold standard to evaluate the curve magnitude on radiographic examination. The main clinical parameters in assessing trunk morphology are the C7 plumb line, shoulder and hip asymmetry [40]. Spinal curvatures in skeletally immature patients with nonossified iliac apophysis have been found to progress in 65% with curves between 20° and 30° and in nearly all patients with a curvature greater than 30° [41].

Any abnormal neurologic findings should raise concern for spinal cord pathology. Although there is no ideal screening test, Adam's forward bend test requires no additional equipment and can help to identify scoliosis [25]. The child bends forward at the waist until the spine becomes parallel to the horizontal plane, while holding palms together with arms extended (Figure 1). The examiner looks along the horizontal plane of the spine from the back and side to detect an asymmetry in the contour of the back known as a "rib hump".

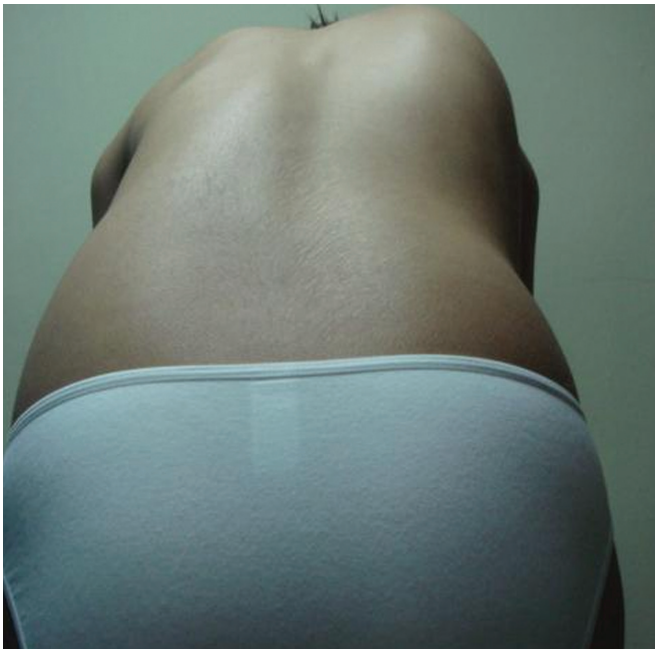

Figure 1. The Adams forward-bending test in a 12 years-old girl with right-convex adolescent idiopathic thoracic scoliosis; when she bends forward, spinal rotation becomes evident through the appearance of a hunched rib cage on the right.

Diagnostic Evaluation

The general historical interview should include specific questioning about potentially associated conditions, such as a congenital heart defect or urological problems, and may serve to identify causes of nonidiopathic scoliosis. The family history may suggest a hereditary predisposition to scoliosis. Pain and mental distress should be evaluated by specific questioning [7].

The examiner inspects the patient to assess shoulder stance and the symmetry of the chest and waist. A pelvic obliquity when the patient stands with the lower limbs fully extended and both feet planted flatly on the ground indicates an asymmetry of leg length. When the patient stands up straight with the legs fully extended, the head should be centered over the pelvis when viewed both from the front and from the side. Unusually pronounced focal growth of hair over the lumbar

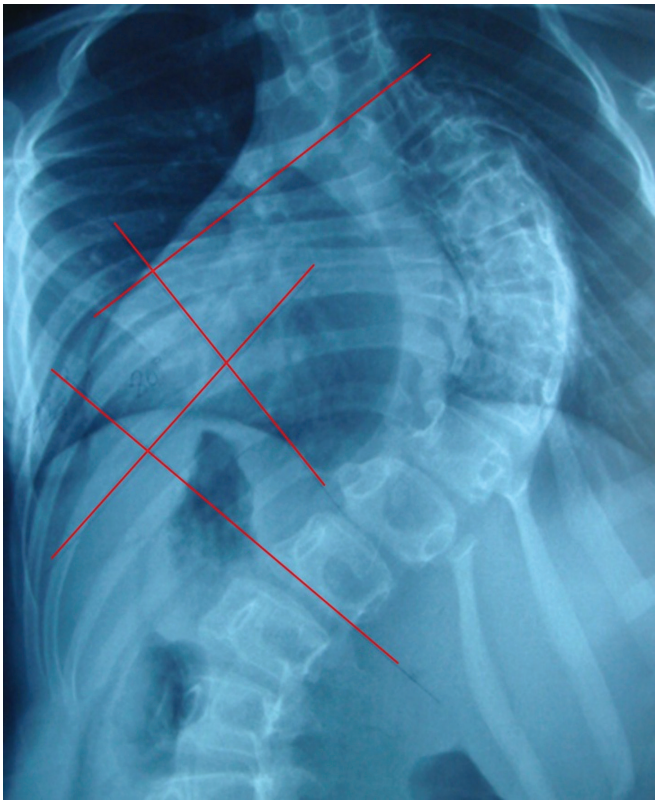

Figure 2. The Cobb method of measuring the degree of scoliosis.

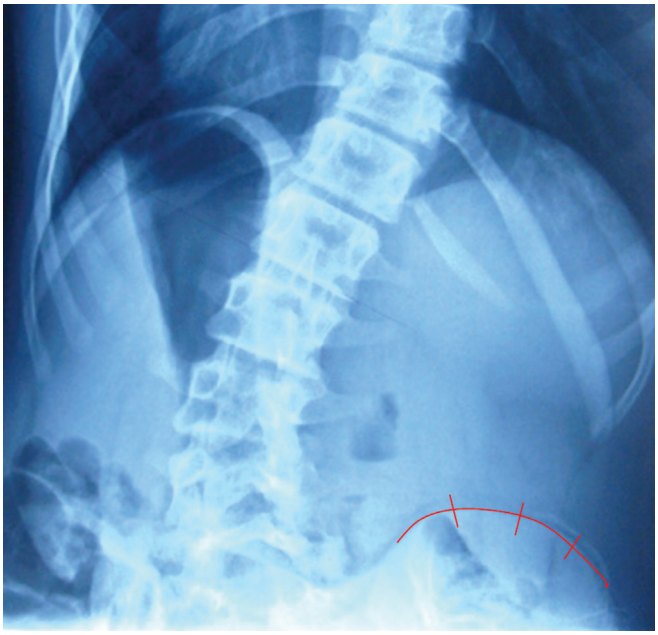

Figure 3. Risser grades zero to 5. Grade 2 ossification have been shown at the iliac apophysis.

spine frequently accompanies incomplete fusion of the laminae and can indicate a neurological cause of scoliosis, as can abnormal pigmentation, e.g., café-au-lait spots, a sign of neurofibromatosis.

Adolescent idiopathic scoliosis is by far the most common cause of spinal curvature in the teenage patient. There is a long differential diagnosis for the causes of spinal deformity (Appendix). Most of these different causes of scoliosis are easily distinguished from adolescent idiopathic scoliosis by the age of presentation, clinical history, radiographic appearance, and physical examination [2].

Abnormalities on physical examination require radiographic evaluation with standing posteroanterior and lateral radiographs to allow measurement of the curve using the Cobb method (Figure 2) and Risser (Figure 3) grading of the iliac apophysis. Magnetic resonance imaging is indicated whenever there is a left thoracic curve, unusual pain or abnormalities on neurologic examination, or other red flags, to evaluate for spondylolisthesis, tumors or syringomyelia [27,42].

The major (largest) curve in the patient with adolescent idiopathic scoliosis is often accompanied by vertebral axial rotation, will not completely correct with side-bending views, and is thus, by definition, the structural curve. Other curvatures may be inflexible, structural curves or flexible, nonstructural curves that are present to maintain truncal balance [2].

Many authors contend that routine MRI examinations are not necessary in the absence of neurologic findings when there are typical radiographic findings. A left thoracic curve, the presence of a neurologic abnormality, and presentation before the age of 11 years were significantly associated with a positive MRI examination [2].

Pain is a common symptom of adolescent idiopathic scoliosis, occurring in as many as 32% of patients in one series, although it is rarely disabling [43].

The Treatment of Adolescent Scoliosis

Treatment of adolescent idiopathic scoliosis is varied and includes observation, bracing and surgery. The choice of treatment is based on the degree of morbidity, patient factors, surgeon preference, and the risk of curve progression over time.

Scoliosis of less than 20° should be followed up in periodic outpatient visits at 4 to 6 month intervals, and new X-rays should be taken if progression is suspected [7].

Bracing has limited effectiveness and is generally recommended for scoliosis between 30° and 45° before the termination of bone growth and for scoliosis between 20° and 30° that progresses by more than 5° in 6 months. Bracing has been shown to successfully

prevent curve progression in 75% in this patient population [44]. The aim is straightening by at least 50% in the brace. Follow-up assessment of the correction is performed with a spine X-ray in the brace, which is taken a few weeks after the brace is first used to allow a period of adaptation [7]. A brace is designed to apply an external force to the trunk during the adolescent growth phase to prevent progression.

Brace types

The Milwaukee brace is a cervico-thoracic-lumbosacral orthosis developed in the 1940s. It is used for thoracic and double curves. The Wilmington brace is a TLSO (thoracic-lumbosacral orthosis) type of brace. It was designed by G. Dean MacEwen to improve patient compliance by making the brace less bulky and more lightweight, as compared with the Milwaukee brace.

The Boston brace was developed in the 1970s. It is also a TLSO-type brace and is made from prefabricated polypropylene pelvic module with a soft foam polyethylene lining. The Boston brace is a full-time brace and can be used to treat all scoliosis [3].

The Dynamic Spine-Cor brace, developed in 1992–1993, uses a specific Corrective Movement which is dependent on the type of curve. To be effective and to obtain a neuromuscular integration, the brace must be worn 20 hours a day for a minimum of 18 months. Generally, the brace is stopped at skeletal maturity (at least Risser 4) [3].

The Charleston brace is a custom-molded spinal orthosis that holds the patient in an overcorrected position. This brace is a nighttime brace only.

The Providence brace was developed when it was observed that substantial correction of scoliotic curves could be achieved using an acrylic frame to apply direct corrective forces to the patient. The brace can be used to treat all single and double curves. The brace is now fabricated using computer-aided design and manufacturing techniques. The Providence brace is a nighttime-only type of brace [3].

The recommendations for bracing study inclusion were patients of 10 years or above, a Risser sign of 0 to 2, initial curve magnitude of 25° to 40°, and no prior treatment at the initiation of brace treatment. The outcome data should be determined from the percentage of patients with: less than 5° or greater than 6° of progression at maturity, curves exceeding 45° at maturity, and progression resulting in the recommendation for surgery [3].

Multiple factors can be obstacles to successful brace treatment. Poor compliance with wear schedules is a major recurring theme in the braced patient, particularly males.

Braces are usually worn 18 to 23 hours a day, although evidence exists demonstrating the effectiveness of part-time or nighttime bracing to address patient compliance issues. Part-time or nighttime bracing (Charleston, Providence) may be effective for curves less than 35 degrees; however, curves greater than 35 degrees often require full-time bracing to reliably limit curve progression.

Surgery is generally the preferred option for a skeletally immature patient with a progressing 40° scoliosis or a skeletally mature patient with a painful or progressive curve greater than 45°. Skeletally immature patients continue to grow anteriorly after a posterior fusion. This may result in a rotational deformity, often with a stable Cobb angle, known as the "crankshaft" phenomenon [45]. Clinically, this often manifests as an increased rib hump in the postsurgical patient. The surgeon may include an anterior fusion in these patients to prevent this occurrence. The goals of surgery include restoration of truncal balance; a stable, pain-free spinal fusion; and improved cosmesis, including rib hump, shoulder, and hip symmetry. The goal is not necessarily to completely straighten the spine. In fact, the degree of deformity correction does not correlate with clinical outcome. Overcorrection of curves may lead to truncal imbalance or asymmetry of the shoulders. The cosmetic appearance of the trunk in a patient with scoliosis does not depend solely on the magnitude of the Cobb angle, but rather on frontal trunk balance, thoracic hypokyphosis, frontal rib cage deformity, rib hump, waist asymmetry, and trunk rotation [40].

Modern instrumentation systems enable 60% to 80% correction as well as immediate ambulation without the need for a brace after surgery. The rate of neurological complications varies from 0.2% to 1.8% depending on the extent of the procedure; most such complications are transient [46].

Physiotherapy can be initiated even when the scoli-

osis is only mild. The most popular type of physiotherapy for scoliosis is Schroth therapy [7].

Prevention

The development of scoliosis cannot be prevented. Thus, attention is currently being focused on early detection so that timely treatment can be provided [7].

Conflict of interest statement

The authors have no conflicts of interest to declare.

APPENDIX: Causes of Scoliosis

1. ***Idiopathic***
   Adolescent, juvenile, infantile
2. ***Degenerative neuromuscular***
   - *Neuropathic*
     Spina bifida, cerebral palsy
   - *Musculopathic*
     Muscular dystrophy
3. ***Congenital***
   - *Anomalous formation*
     Hemivertebra, wedge vertebra
     Failure of segmentation
     Unilateral bar or vertebral fusion
   - *Neurogenic*
     Chiari malformation, syrinx, tethered cord, di astematomyelia
4. ***Developmental***
   - *Skeletal dysplasias*
     Achondroplasia, mucopolysaccharidoses
   - *Skeletal dysostoses*
     Neurofibromatosis, osteogenesis imperfecta, Marfan, Ehlers-Danlos
5. ***Secondary***
   Tumors, infection, trauma

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