A Review Paper

Infantile Idiopathic Scoliosis

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Abstract

This review of infantile idiopathic scoliosis highlights the clinical features, etiology, epidemiology, and treatment considerations that clearly distinguish this entity from the more common diagnosis of juvenile and adolescent idiopathic scoliosis. A comprehensive understanding of infantile idiopathic scoliosis provides the basis for reliable prediction of those curves that are likely to spontaneously resolve and those that will relentlessly progress if left untreated.

Structural scoliosis during infancy was first described by Harrenstein[1] in 1929. In a series of 46 young children, Harrenstein proposed rickets as the likely etiology in the majority. Two decades elapsed, however, until the distinct diagnosis of infantile idiopathic scoliosis (IIS) was recognized and published by James[2] (1951). James found no association with rickets (or any other identifiable etiology) in his group of 33 infants and therefore included the term idiopathic in the diagnosis. James highlighted several characteristic features of IIS that were dissimilar to those of adolescent scoliosis, including onset of deformity before age 3 years, predominance of male gender, and high incidence of left-sided thoracic curves. He also noted occasional complete resolution of the curve, although the majority in his series demonstrated increasing spinal deformity over time. Scott and Morgan[3] affirmed that IIS was unique from adolescent idiopathic scoliosis and from other etiologies of spinal deformity in the very young (eg, congenital scoliosis) in an additional series of 28 patients published in 1955.

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Clinical Features

The clinical features of IIS that clearly distinguish this diagnosis from the more common juvenile and adolescent idiopathic scoliosis are listed in Table I. IIS is identified at birth in at most 5% of cases.[4,5] Rather, the diagnosis is more typically made between 6 months and 1 year of age.[6] By definition, curve onset occurs before age 3 years. Males and females are affected in a 3:2 ratio.[4,7] The diagnosis presents as a fixed structural spinal deformity that is not eliminated by axillary suspension and that is located in the thoracic spine in 85% to 100% of published cases.[3,6,7] The apical vertebra is typically at the ninth thoracic vertebra.[6] Lumbar curves are usually seen as a secondary curve below the primary thoracic-level deformity. In stark contrast to adolescent idiopathic scoliosis, in IIS the convexity of the thoracic curve is left-sided in the majority. In various series, 76% to 93% of IIS cases demonstrate such a left-sided pattern.[3-7]

Alternative etiologies for a structural spinal deformity must be excluded before the diagnosis of IIS is made. Neuromuscular, syndromic, and congenital causes for the scoliosis must be specifically eliminated. A thorough physical examination is necessary to help reject such other possibilities. A radiograph of the entire spine should be taken to exclude congenital scoliosis.

Several other anomalies have been reported to be commonly associated with IIS (Table II). Plagiocephaly has been reported in many series to be present in 80% to 100% of cases.[6-8] In addition, both Wynne-Davies[7] and Conner[9] found a high correlation between mental retardation and progressive curves in infants with idiopathic scoliosis. Furthermore, a 5- to 10-fold increased incidence of developmental dislocation of the hip has been recorded,[7,10] with dysplastic changes seen in 1 of 4 infants.[8] No correlation between the side of hip dysplasia or dislocation and the convexity of the curve has been demonstrated. Based on these findings, a pelvic radiograph should be standard during the early evaluation of an infant with idiopathic scoliosis.

Table I. Distinguishing Clinical Features of Infantile Idiopathic Scoliosis

<3 years old at onset
Male predominance (3:2)
Left-sided thoracic curve predominance (75%–93%)
Spontaneous resolution common (80%–90%)

Table II. Anomalies Commonly Associated With Infantile Idiopathic Scoliosis

Plagiocephaly
Mental retardation
Developmental hip dysplasia
Neural axis abnormalities

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Of additional particular note is the apparently common association of neural axis abnormalities in infants with idiopathic scoliosis. Such abnormalities have been shown to be present despite entirely normal neurologic function and examination. In 1992, Lewonowski and colleagues[11] published a series of 4 infants with normal neurologic function studied by magnetic resonance imaging (MRI), with 2 of these 4 found to have abnormal scans. Gupta and colleagues[12] repeated this investigation in 1998 with prospective MRI in an additional 6 infants. Once again, a 50% incidence of neural axis abnormalities was seen. In 2002, Dobbs and colleagues[13] found a lower but still significant 21.7% rate in a series of 46 infants with idiopathic scoliosis. Arnold-Chiari malformation with an associated cervicothoracic syrinx was most commonly discovered. Additional abnormalities found included diffuse dural ectasia, low-lying conus, and brainstem tumor. Many of these abnormalities required neurosurgical care. In light of these 3 series, routine MRI of the brain and spine is recommended for all infants with idiopathic curves of 20° or more.[13]

One last clinical feature unique to IIS as compared with the juvenile and adolescent types is the high rate of spontaneous resolution of the deformity without treatment. Although early on James[2] suggested that most curves in his series progressed over time, in subsequent series resolution has occurred in the majority. In fact, spontaneous resolution is to be anticipated in 80% to 90% of IIS cases.[5,6] Most resolving curves will have done so by age 3 years, although some linger into later childhood before complete resolution.[6,9,14]

Etiology and Epidemiology

Prenatal and postnatal factors have been proposed as potential etiologies leading to IIS. Lloyd-Roberts and Pilcher[6] argued in 1965 that intrauterine molding was most likely the cause of the spinal deformity, given the observed high incidence of plagiocephaly and rib deformity despite a lack of vertebral rotation on early radiographs. Others have implicated postnatal environmental effects, such as the custom of supine positioning of infants—previously common in Europe.[15] Mau[16] cited a variety of associated abnormalities he felt were related to external forces, including plagiocephaly, unilateral pelvic flattening, fixed thoracolumbar kyphosis, torticollis, hip dysplasia, limited abduction of the contralateral hip, and calcaneous foot deformity. Wynne-Davies[7] suggested a multifactorial etiology whereby a genetic tendency for infantile scoliosis is "triggered" by medical, genetic, and social factors.

Postnatal environmental factors, with or without a genetic predisposition, seem likely to contribute to development of IIS when past and current prevalence of the diagnosis is examined. Before 1980, IIS was a predominant diagnosis in the United Kingdom and Europe.[4,14,17] In 1975, James[14] reported the age distribution of 200 consecutive cases of newly diagnosed idiopathic scoliosis: 82 cases occurred in patients under age 3 years (infants), 34 in patients age 3 to 9 years (juveniles), and 84 in patients age 10 years or older (adolescents). Interestingly, a survey conducted by McMaster[17] in 1979 revealed that IIS comprised 50% of all scoliosis cases in the United Kingdom, whereas a concurrent survey in North America demonstrated only a 0.5% incidence. This disparity was seen despite a common genetic stock.[7] Mau[15,16] strongly implicated the supine positioning of infants in the United Kingdom and Germany as a critical influence (as opposed to prone positioning of infants—a North American practice). Indeed, the incidence of both resolving and progressive forms of IIS declined dramatically from 41.75% of cases in 1968–1972 to a mere 4% of cases in 1980–1982, when prone positioning of infants gained popularity in the United Kingdom.[17] A similar precipitous drop was demonstrated in Germany.[16] This led McMaster and others to conclude that IIS is largely a preventable deformity.

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Management

Determine Curve Behavior

The first step in managing infants with idiopathic scoliosis is determining whether the curve is likely to spontaneously resolve or continue to progress. Before the

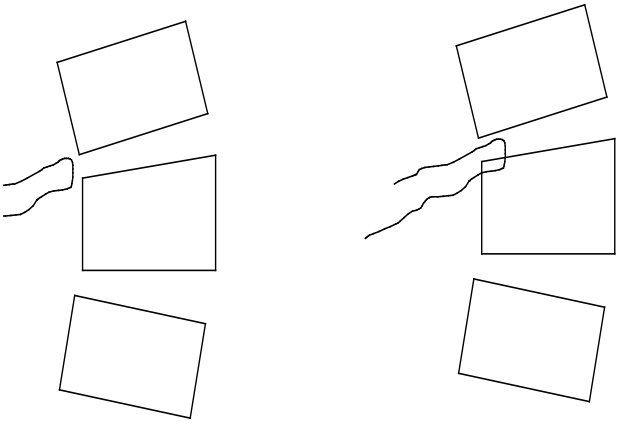

Figure 1. The relationship of the apical ribs with the apical vertebra as seen on a coronal radiograph is described as either phase 1 (rib head does not overlap the apical vertebra) or phase 2 (the apical rib head does overlap the apical vertebra).

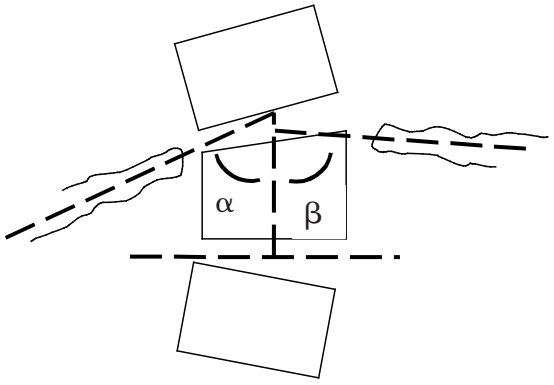

Figure 2. The rib vertebral angle (RVA) is the angle formed between a perpendicular drawn to either the upper or lower endplate of the apical vertebra with a second line drawn from themidponit in the rib head to the midpoint in the rib neck just medial to the region where the neck widens into the shaft of the rib. The RVA difference (RVAD) is the difference of the RVA on the concave and convex sides of the curve, and is useful in distinguishing resolving versus progressive curves while in phase 1.

landmark publication by Mehta[18] in 1972, the behavior of IIS was not predictable early in the patient's clinical course. Initial reports suggested that shorter curve length, initial curve magnitude of more than 35°, younger age at onset, increased degree of rotation, presence of additional developmental abnormalities, and presence of a compensatory curve correlated with the progressive variety of IIS.[3,5,9] Resolving curves were noted to be predominantly thoracic or thoracolumbar, while progressive curves were more likely to be a double major curve.[8] Isolated lumbar curves were believed to have a better prognosis than isolated thoracic curves.[5] These same authors admitted, however, that there was no discernable distinction between resolving and progressive forms of IIS and that in many cases their early presentations were identical.[5] In a later series (1980), Ceballos and colleagues[8] found these distinguishing criteria to be unreliable in predicting later curve behavior. Even James[4] and James and colleagues,[5] who in their early investigations declared that the presence of a compensatory curve "seems to be an absolute indication of the permanency" of the curve, subsequently identified 2 cases in which double curve patterns spontaneously resolved.

Just 1 year after James[4] declared no clinical features could reliably distinguish resolving and progressive curves in their early stages, Mehta[18] introduced new radiographic criteria based on the relationship of the apical ribs with the apical vertebra (this relationship has been repeatedly validated as a means to predict curve behavior). Mehta defined 2 phases of curve patterns (Figure 1) based on whether the rib head is distinct from the upper corner of the apical vertebra on a coronal radiograph (phase 1) or the rib head overlaps the upper vertebral corner (phase 2). In her series of 138 cases from a registry of 361 cases, Mehta recognized that all phase 2 patterns correlated with definitive progression. A transition from phase 1 to phase 2 was also invariably associated with future progression.

Mehta[18] recognized that the earliest observable radiologic finding was an increased downward slope of the ribs on the convex side of the curve with the maximum at the apex of the curve and that measurement of the angle formed between the rib and the apical vertebra could further differentiate progressive and resolving curve types while the curve was still in phase 1. The rib vertebral angle (RVA) was defined as the angle formed between a perpendicular drawn to either the upper or lower endplate of the apical vertebra with a second line drawn from the midpoint in the rib head to the midpoint in the rib neck just medial to the region where the neck widens into the shaft of the rib. Furthermore, the RVA difference (RVAD) was defined as the difference of the RVA on the concave and convex sides of the curve (Figure 2).

Mehta[18] found the RVAD useful in distinguishing resolving and progressive curves while in phase 1. A 20° RVAD was established as a relative tidemark for curve prognosis. Mehta found that 80% of all resolving curves had an initial RVAD of less than 20°, and that the remaining demonstrated a decreased RVAD on radiographs 3 months later. In contrast, the RVAD was 20° or more in 80% of all progressive curves on initial radiographs, and this value remained unchanged or increased on radiographs obtained 3 months later. Given these findings, phase 1 curves with an initial RVAD of 20° or more were likely progressive, although a definite conclusion was not possible until a comparison radiograph was obtained after another 3 months. The conclusions were found to be valid even in cases in which the Cobb angle increased over 3 months while the RVAD decreased (thus indicating a resolving rather than progressive curve, despite the increase in measured curve magnitude). Mehta's criteria were confirmed by all subsequent published investigations.[8,19,20]

There is one caveat to the guideline that a <20° RVAD portends a favorable outcome: A finding of a downward slope of the twelfth rib on the concave side indicates an early double curve and produces a deceptively low or even negative RVAD of the thoracic curve.[18] With recognition of this pattern, an erroneous conclusion (that the curve is likely to resolve) can be avoided.

In a retrospective study of 169 children, Kristmundsdottir and colleagues[21] proposed a simplification of Mehta's method. They found that measurement of the RVA on the convex side of the curve alone was equally accurate in predicting phase 1 curve behavior. Both a convex RVA of 68° or more and Mehta's RVAD of less than 20° correlated with 95% of all resolving curves. A convex RVA of more than 68° or a RVAD of more than 20° correlated with progression in 60% of cases in the investigation by Kristmundsdottir and colleagues.

Resolving Curves

No treatment is necessary for infants with resolving idiopathic scoliosis. As mentioned, the majority of curves resolve by age 3 years. Of 34 of 42 patients available at a follow-up of more than 25 years, few had back pain, and none experienced any interference in employment or social activities.[22] None had demonstrated a significant progression in scoliosis during the subsequent adolescent growth spurt.

Progressive Curves

Unlike the prognosis for infants with a resolving curve, the prognosis for infants with a progressive idiopathic curve is dismal. James and colleagues[5] emphasized that most curves will reach a magnitude of 100° or more if left untreated. Scott and Morgan[3] described 14% mortality in their series of 28 patients and cited cardiorespiratory failure the most common cause of death. Morgan and Scott[23] also cited the excess mortality rate associated with severe thoracic deformity. A more recent evaluation of respiratory function in patients treated both nonsurgically and surgically for progressive curves found parameters that correlated with the severity of the eventual deformity.[24] In that study, patients who underwent early fusion but then had recurring deformity had pulmonary function values a mere 41% of predicted.

Immediate nonsurgical measures are indicated once the progressive nature of an infant's scoliosis is recognized. In this young population, initial casting under anesthesia is typically recommended, followed by a custom brace when the child is large enough.[4,5,25-27] A Milwaukee brace can be fitted as early as 18 months in many cases; this brace has been preferred over a custom-molded thoracolumbosacral orthosis for not compromising respiratory function.[25] Surgery is recommended when curves progress despite appropriate bracing.

Initial reports of surgical fusion for severe progressive scoliosis in children were discouraging. In 1956, Morgan and Scott[23] published a long-term follow-up study that indicated poor results after spinal fusion regardless of the child's age at surgery. Children age 5 to 7 years with preoperative curves averaging 53° were found to have postoperative progression to a mean of 115° by age 20. Children age 12 to 14 years with preoperative curves averaging 94° were found to have postoperative progression to a mean of 120° by age 20. Morgan and Scott questioned the wisdom of surgical fusion and instead emphasized a program of generalized respiratory strengthening in an attempt to postpone premature cardiorespiratory demise.

James and colleagues[5] provided a more optimistic outlook when they formalized a surgical treatment algorithm in 1959. This approach became the standard method for treating progressive curves for the next 2 decades. Body casting and Milwaukee bracing were used until the child was deemed old enough for a posterior spinal fusion. This surgical procedure was not recommended for children younger than 10 years.[4] With the introduction of Harrington[28] spinal instrumentation in 1962, the rate of favorable outcomes improved. A series of 6 patients with progressive IIS who were treated with plaster casting, bracing, and subsequent fusion demonstrated a decrease in mean curve magnitude from 66° before treatment to 47° at maturity[14]; only 1 child experienced curve progression over this follow-up period, and only from 54° before treatment to 70° at maturity. Using a similar technique, McMaster and Macnicol[27] reported a mean correction of 40% of the initial curve, although some loss in correction was noted over time.

Hefti and McMaster[29] reported a similar loss in correction despite achieving a solid posterior fusion. Such correction loss is the likely sequela of the crankshaft phenomenon described by Dubousset and colleagues.[30] To minimize this effect, an anterior–posterior combination is now favored when fusion is performed at a young age. Traditional assessment of maturity has largely relied on Risser[31] staging and menarchal status, although additional attention to the triradiate cartilage growth plate and peak growth velocity may provide improved selection of children who would be best served by an anterior, as well as posterior, spinal fusion.[32,33]

Luque[34] first popularized posterior instrumentation of the spine without fusion in an attempt to provide corrective control of progressive curves yet allow for continued longitudinal spinal growth. Use of the Luque trolley technique has provided mixed results. Both Patterson[35] and Pratt and colleagues[36] reported improved correction when this method was combined with an anterior apical fusion, although the Luque trolley alone did not prevent progression. In a series involving this technique, Rinsky and colleagues[37] maintained correction in only 4 of 9 patients and recommended against its use. Subcutaneous rodding is an alternative, but not enough cases of its use in IIS have been reported to assess its efficacy. A high rate of complications, including premature rod failure, rod prominence, sinus tract formation, infection, junctional kyphosis, and unintentional spontaneous fusions, have been recorded using these techniques.[36-38]

The mixed clinical results of the initial methods of spinal instrumentation of the spine without fusion have led to technique changes that are hoped to improve surgical outcome.[39] The use of dual rod posterior spinal constructs anchored more securely at both the cranial and caudal ends of the deformity may decrease the rate of mechanical failure and hook pullout. With such methods, however, there are still concerns regarding possible spinal fibrosis, curve stiffening, and premature fusion. Until further convincing prospective studies are published, instrumentation without fusion remains controversial.

Despite these cautions, a growing body of evidence supports the importance of maintaining thoracic spinal growth during a child's early years (up to age 8) to maximize thoracic dimensions and the space available for the lungs. The groundbreaking work of Campbell and colleagues[40-43] in treating thoracic insufficiency syndrome highlights a

potential new device—the vertical expandable prosthetic titanium rib—that appears to support more normal lung and spinal growth while adequately controlling spinal deformity in infants and young children. Although the device is promising, the US Food and Drug Administration approves its use for children with IIS only with a compassionate use exemption. Campbell and colleagues' results suggest that this expandable rib device stabilizes, if not increases, respiratory function by improving the chest wall and spinal deformity. Consequently, such an approach could eliminate the possible unintended restrictive effect of early spinal fusion on thoracic dimensions and lung volumes.

Additional surgical procedures, such as apical rib resection, costodesis with contralateral rib release, and convex anterior and posterior epiphysiodesis, have been unsuccessfully used in attempts to control progressive infantile curve types.[44-46] These methods have not demonstrated favorable outcomes and are not advised.

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