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Chapter

Conservative Treatment of Degenerative Lumbar Scoliosis



Degenerative lumbar scoliosis (DLS) is commonly seen in people over the age of 50 years. The prevalence increases with age. Patients with DLS often complain of low back pain and radiculopathy. Neurological complaints are rare. Current treatments are generally targeted at pain relief. Effects are temporary; this is understandable as the spinal deformities which are the cause of the pain are not addressed. A few studies have shown that scoliosis specific exercises and lordotic bracing stabilize or reduce the rate of curve progression in patients with DLS. Patients should also be instructed in performing corrective movements in daily activities. In the presence of sarcopenia or decreased bone mineral density (BMD), resistance exercises and nutritional supplements should also be prescribed, as reduction in paraspinal muscle mass and BMD are risk factors of DLS. In the presence of neurological involvement or when the symptoms are refractory to conservative treatment, referral for surgery is required.

Keywords: degenerative lumbar scoliosis, adult scoliosis, scoliosis-specific exercise, spinal brace, sarcopenia, osteoporosis

1. Introduction

Adult scoliosis includes adult idiopathic scoliosis as well as degenerative lumbar scoliosis (DLS). DLS is a de novo scoliosis. It is defined as a lateral curvature of the spine in excess of 10° and a sagittal vertical axis (SVA) of more than 50 mm. in an adult over 50 years of age [1]. The sagittal vertical axis is the horizontal distance from the vertical plumbline dropped from the centroid of C7 to the posterosuperior corner of the sacral end plate.

The prevalence of DLS varies with different studies. The reported rate ranges from 8.3–68% [2–5]. The marked variation in prevalence reported is possibly a result of the differences in inclusion criteria of the different studies. Kilshaw et al. [6] evaluated the prevalence of lumbar scoliosis using abdominal and kidney–ureter-bladder radiographs on patients over the age of 20 years [6]. The study showed that lumbar scoliosis is more prevalent in women and increases with age [6]. At the ages of between 60 and 69 years, the prevalence was 6%, whereas at the age of 90 years, the prevalence was over 30% [6]. It has, however, to be noted that the study did not differentiate between adult idiopathic scoliosis and DLS and that the radiographs employed were supine films, which might underestimate the Cobb angle and thus the prevalence of lumbar scoliosis. Xu et al. [7] evaluated 2395 Han Chinese over 40 years of age for the presence of DLS, using dual energy X-ray absorptiometry (DEXA) images. They reported a prevalence of 13.3%, with females more commonly

affected than males. Also, the prevalence increased with age [7]. Prevalence was reduced with increasing curve severity; over 80% of the patients with DLS had curves in between 10° and 20° [7].

2. Etiopathogenesis

The causes of adult scoliosis are many. Aebi [1] classified adult scoliosis into four different groups, based on their etiologies. Type 1 refers to primary or de novo degenerative lumbar scoliosis. Type 2 refers to adult idiopathic scoliosis (ADIS), and type 3 refers to adult curves with other primary causes. The last type includes two subgroups. Type 3a refers to adult scoliosis caused by spinal or extra-spinal factors, while type 3b refers to those caused by metabolic bone diseases [1]. Thus adult scoliosis patients are not a homogeneous population group. Our present discussion would focus on DLS which is more prevalent than other types of adult scoliosis.

The pathomechanisms of DLS have not been entirely elucidated, though vertebral instability has been proposed to play a role in its pathogenesis [8, 9]. Kobayashi et al. [4], in a study of the prevalence of DLS, proposed that lateral osteophytes present at the endplate which are in excess of 5 mm together with an asymmetric tilt of disc space >3° are risk factors for the development of DLS [4].

The factors initiating the vertebral instability, however, are unknown. Lumbar paraspinal muscle atrophy; facet tropism, which is defined as the angular asymmetry between the left and right facet joint orientation; and osteoporosis have all been implicated in the pathogenesis of the condition [7, 10, 11].

Lumbar multifidus muscle atrophy (LMA) has also been postulated to contribute to vertebral instability [10]. The multifidus muscle is the deepest and most medial paraspinal muscle, adjacent to the facet joint. LMA is common in DLS, particularly on the concave side of the lumbar scoliosis (**Figure 1**) [12, 13]. Conversely, hyperplasia of the multifidus muscle is evident on the convex side of DLS [14]. Sun et al. [10] investigated the relationship between LMA and various coronal and sagittal radiographic parameters in 144 patients with DLS [10]. They showed that the LMA in the upper and lower vertebral levels adjacent to the apex on the concavity of the lumbar scoliosis correlated positively with the Cobb angle [10]. Conversely, the LMA on the convex side correlated negatively with the lumbar Cobb angle [10]. Sun et al. [10] postulated that LMA may cause vertebral instability and subsequent degenerative changes of lumbar facet joints. Remodeling of articular processes, which includes cartilage degeneration and bone erosion, generally lags behind LMA [10].

Facet tropism has also been postulated to increase the risk of vertebral rotatory olisthesis (VRO) and degenerative lumbar scoliosis [11, 15, 16]. Vertebral rotatory olisthesis refers to lateral and rotatory vertebral translation. Facet joints were found to be more angled in a coronal plane on the convex side of VRO than those of the control subjects without VRO [11]. More severe facet tropism is associated with a higher incidence of VRO [11]. The asymmetric facet orientation causes uneven stress distribution across the zygapophyseal tissues and brings about degenerative changes and segmental instabilities [11]. An intraoperative biomechanical study demonstrated that facet tropism contributed to lumbar vertebral instability [17].

The role of osteoporosis in DLS has been controversial, with some studies showing that osteoporosis contributed to DLS, a number showing that DLS caused the osteoporosis, with others showing no correlation between the two [7]. The lumbar scoliosis brought about by vertebral instability may stabilize or progress [8, 9]. In the presence of marked scoliotic wedging of one disc in the early phase of DLS, adjacent discs may compensate by wedging in the other direction to maintain balance, with resultant stabilization or even regression of the lumbar scoliosis (**Figure 2**) [8].

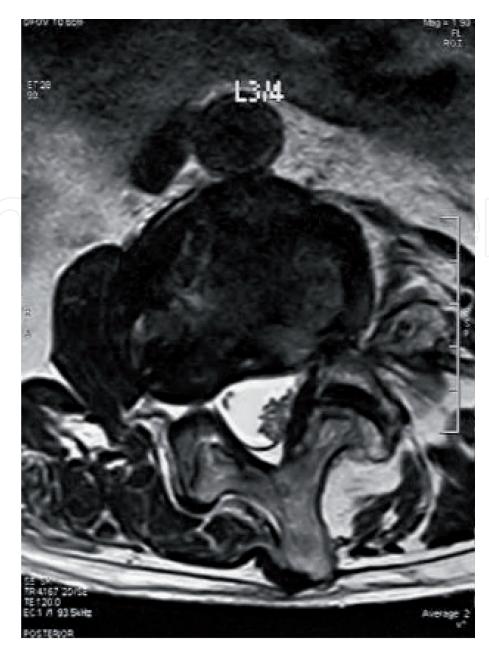


Figure 1.Lumbar multifidus atrophy. From the MRI, it is evident that there was marked asymmetric lumbar multifidus atrophy at the level of L3/L4. The fatty infiltration area in the left multifidus was significantly larger than that in the right multifidus.

In other cases, degenerative scoliosis may progress. The increased pressure and shear stress on the facet joints cause alterations within the synovial surfaces of the articular processes with subsequent facet hypertrophy, capsular degeneration, and ligamentous hypertrophy [18]. Also, asymmetric loading of the lumbar facet joints and intervertebral discs may result in spinal deformities occurring in three planes [19, 20], particularly in the presence of decreased bone density. Depending on the number of segments involved, this can also cause segmental or multi-segmental vertebral instabilities. Further instability in the sagittal and coronal planes may result in degenerative spondylolisthesis and rotatory olisthesis, respectively [21]. It has to be noted that rotatory olisthesis is present even in mild lumbar scoliosis of less than 20° [21].

Lumbar VRO is prevalent in L3–L4, followed by L2–L3 and L4–L5. Of all the VRO, L3–L4 laterolisthesis contributes around half of the prevalence [11, 22, 23]. Watanuki et al. [24] proposed that this was related to the mechanical stress at the L3–L4 levels, as the lower lumbar levels are more fixed and the upper lumbar

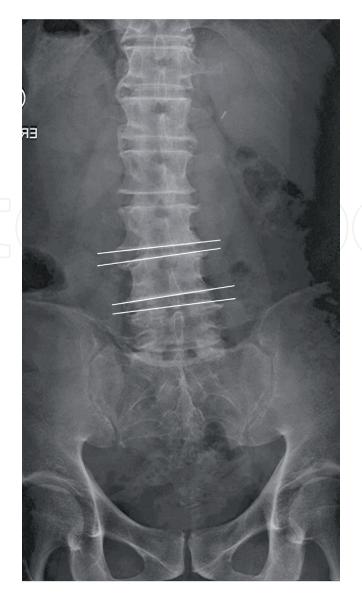


Figure 2.Mild intervertebral disc wedging in one level is compensated by wedging in the opposite direction to maintain coronal balance. Mild disc wedging was evident in L4/L5 level. The wedging was compensated by disc wedging above (L3/L4) in the opposite direction, balancing the spine.

segments are more mobile [24]. The smaller size of the L4 vertebral body may also contribute to the higher incidence of laterolisthesis at L3–L4, as a reduction of 25% of the vertebral cross-sectional area increases mechanical stress by 30% with an applied load, contributing to vertebral instability [25].

To reduce the instability, the body reacts by growing osteophytes (**Figure 3**). The spondylosis (osteophytes by the end plates) and the spondyloarthritis (degenerative changes of the facet joints) that result, together with the ligamentous hypertrophy, compromise the central spinal canal and the lateral recess and may bring about claudication and nerve root compression symptoms [1].

Apart from bone and articular involvement, paraspinal muscle atrophy is prevalent in DLS. Sarcopenia, which is a reduction in skeletal mass, is commonly seen in patients with DLS. Eguchi et al. [26], using DEXA scans to assess the appendicular and trunk skeletal muscle mass, showed that sarcopenia was present in 46.6% of the DLS patients [26]. Sarcopenia is defined as the appendicular skeletal mass index of less than 5.46 kg/m² [27]. The appendicular skeletal mass index (ASMI) is the sum of the arm and leg lean mass (kg) divided by square of the height (m²) [27]. Studies have also shown that ASMI negatively correlated with pelvic tilt [26], whereas trunk skeletal mass index (Trunk SMI) which is defined as trunk lean mass



Figure 3.The osteochondrosis at L4/L5 intervertebral level, together with the bridging osteophyte in the left of L3/L4, stabilized the mild scoliosis curve and maintained coronal balance in this man aged 63 years.

divided by height² (m²) significantly correlated with the sagittal vertical axis, pelvic tilt (PT), and lumbar scoliosis [26]. Moreover, trunk SMI correlated positively with bone mineral density (BMD), suggesting that reduction in trunk muscle mass was associated with osteoporosis and sagittal imbalance [26], which is prevalent in patients with DLS.

3. Clinical presentation

Patients are generally over 50 years of age. Clinical presentation is variable. Onset is generally gradual, though it can be sudden, after a day's work, repetitive bending of the low back, poor sitting posture, or prolonged standing.

Most of the patients complain of low back pain, radiculopathy, and claudication [28]. Liu et al. [28], in a study of the clinical features of 112 patients with DLS treated surgically, found that 77% of them complained of low back pain, 90% complained of radiculopathy, and 48% complained of neurogenic intermittent claudication. Only 3% of them had neurological symptoms [28]. The symptoms can present singly or in combination [28].

3.1 Low back pain

Low back pain is generally diffuse. It is often located in the apex and concavity of the curve and at the junction between two curves [28]. The severity of the pain varies with different curve types, with thoracolumbar, lumbar, and lumbosacral curves being more painful than thoracic curves. A compensatory hemicurve is the least painful, except for the left compensatory lumbosacral hemicurve [29–31]. Pain is also localized on the iliac crest and the coccyx, where the tendons of the lumbar paraspinal muscles insert [1]. Rarely, the lowest ribs impinge on the iliac crest and cause pain [1]. In the presence of a reduced lumbar lordosis or a complete loss of the natural lumbar curve, the muscle pain is generally greater. This is not unexpected as the lumbar paraspinal muscles have to contract continuously to maintain coronal and sagittal spinal balance.

Whether the extent of the pain is related to the magnitude of the curve and coronal balance has not been clearly elucidated as yet [29, 32]. A number of studies have shown that Cobb angles in excess of 45° are associated with more pain [33].

Other studies, however, have shown that the magnitude of the curve was not related to the pain [29, 34].

The impact of coronal balance on low back pain is likewise controversial. Some studies showed that a coronal imbalance in excess of 4–5 cm. is associated with more pain and reduction in function in un-operated scoliosis patients [32, 35]. Further trunk shift is a predictor of surgery in patients with DLS [35]. Other studies, however, did not show such an association [36].

3.2 Radiculopathy

Radiculopathy is common in patients with DLS. Many studies have attempted to investigate the relationship between the scoliosis curve, VRO, and the nerve roots involved [28, 37, 38]. In a study evaluating 47 male and 65 female DLS patients with a mean age of 54.7 years, Liu et al. [28] showed that L3 and L4 nerve roots are generally compressed on the concave side of the scoliosis [28]. Conversely, L5 and S1 nerve roots are more commonly afflicted on the convex side of the scoliosis [28].

Liu et al. [37] evaluated the site of nerve root irritation in 22 DLS patients [37]. They identified three zones (**Figure 4**) where the nerve root could be compressed or irritated. These included the lateral recess zone, the foraminal zone, and the extraforaminal zone [37]. The lateral recess zone refers to the zone where the nerve root passes from the thecal sac to the entrance of the foramen; the foraminal zone refers to the interval canal beneath the pedicle, and the extra-foraminal zone refers to the zone outside the lateral border of the pedicle [37]. They found that the L3 and L4 nerve roots are more commonly compressed in the foraminal and extra-foraminal zones in the concavity of the scoliosis curve. Conversely, L5 and S1 nerve roots are more commonly affected by a lateral recess stenosis on the convex side [37].

Gardner et al. [38] evaluated different patterns of lumbar spinal stenosis with lateral subluxation in patients with DLS and had similar findings [38]. They showed that the pattern of nerve root compression varies with the types of lateral subluxation, viz., the open subluxation and closed dislocation. Open subluxation refers to subluxation where the disc is open on the side where the vertebra above is slipping. The wedge is open on the convexity of the curve (**Figure 5**). Conversely, closed dislocation is present when the disc is closed on the side where the vertebra above is slipping [23]. Gardner et al. [38] showed that open subluxation commonly affects L3 and L4 levels. When present, it causes contralateral lateral recess and foraminal

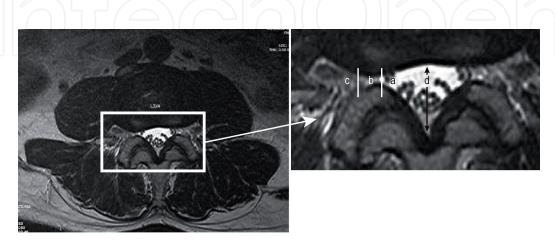


Figure 4.

The magnified view of the spinal canal and the intervertebral foramina. Nerve root irritation can occur in (a) the lateral recess zone, (b) the foraminal zone, and (c) the extra-foraminal zone; (d) is the sagittal diameter of the spinal canal. Spinal stenosis can result from narrowing of the sagittal diameter of the spinal canal or that of the lateral recess, when they are known as lumbar spinal stenosis and lateral lumbar spinal stenosis, respectively.



Figure 5.VRO was evident at Ll, L2, and L3 levels. L1 translated tangentially to the right, with no disc wedging. L2 similarly translated to the right though to a smaller extent, with disc closing on the right, which was the concavity of the lumbar curve. This is defined as closed dislocation. L3, on the other hand, slipped to the left. The disc wedged open on the side of curve convexity. This is termed open subluxation.

stenosis. Closed dislocation, on the other hand, is generally seen on the concavity of the curve, causing an ipsilateral pattern of stenosis [38, 39]. L1 and L2 are the most frequently involved [38].

In a study of a cohort of 78 patients with DLS and spinal canal stenosis, Ferrero et al. [39] demonstrated that foraminal and lateral stenosis were most frequently observed on the concavity of the distal lumbosacral curve. L5 radicular pain was significantly more frequent in the presence of compensatory lumbosacral hemicurve [39].

In view of the different patterns of vertebral instability and compensatory curve patterns, it is understandable that the clinical presentation of DLS varies. Nerve root irritation may be single or multilevels, causing pain in different dermatomes [40, 41].

It is interesting to note that the side of radicular pain frequently corresponded to the side of coronal shift. Patients with right truncal coronal shift more frequently present with right radicular pain; similarly, patients with left coronal shift more commonly present with left radicular pain [39]. The mechanism involved was not clear, though it was found that in 69% of the cases, the truncal coronal shift was associated with the side of the lumbosacral counter-curve (i.e., C7 is shifted to the convex side of the main lumbar curve) [39].

3.3 Claudication

With progression of the condition, pain generally involves the buttock as well as the leg, causing neurogenic claudication symptoms which are worse with standing and walking and are relieved with sitting or stooping. The condition is a result of lumbar spinal stenosis brought about by impingement of nerves emerged from the spinal cord. Studies have shown that the symptoms vary over time in different patients. Symptoms tended to improve in 15% of the patients. In 40% of the patients, the condition tended to deteriorate during the initial 2–3 years of follow-up, and in 45% of the patients, the condition remained stable [42, 43].

3.4 Neurological symptoms

Compression of nerve roots is common, with reported incidence varying from 47–78% [44, 45]. The incidence of cauda equina compression with apparent bladder and rectal sphincter problems, however, is low [1].

Central spinal stenosis is generally seen at the junction between two curves. In the study by Ferrero et al. [39], 70% of the cohort had central stenosis at the junction between the main lumbar curve and the lumbosacral hemicurve. Central spinal stenosis also occurs in the concavity of the main lumbar curve and at the junction between the main thoracic curve and the lumbar curve [39].

3.5 Curve progression

Many studies have shown adult scoliosis tends to progress, with the rate of progression higher in DLS than ADIS [46].

DLS tends to progress irrespective of the magnitude of the curve (**Figure 6**) [46]. A study reported the rate of progression of scoliosis in patients with DLS to be 1.64° per year $(0.77-3.82^\circ)$ [7], while another study reported an increase of 3° per year in a cohort of 200 people over the age of 50 years [47]. The radiographic risk parameters are similar to that of ADIS and include apical vertebral rotation \geq III, a Cobb angle $>30^\circ$, lateral vertebral translation >6 mm., and L5 above the intercristal line, which is the line joining both iliac crests [9, 47].

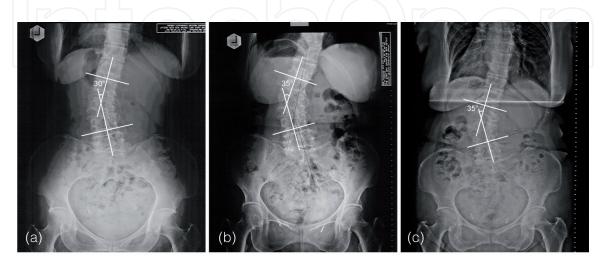


Figure 6.Progression of degenerative lumbar scoliosis in a postmenopausal woman 70 years of age. In the year 2003 (a), the left lumbar scoliosis measured 30°. It increased to 35° in year 2009 (b). After the patient was prescribed Fosamax by his medical practitioner, the curve stabilized, as can be seen in the radiograph in 2017 (c).

4. Physical examination

The physical evaluation aims at the differential diagnosis of the condition as well as to identify the nerve root(s) involved. The findings depend on the severity of the condition and if there are signs of neurological involvement. In the presence of neurological claudication, patients generally walk with an antalgic gait (gait to avoid or reduce pain), with the trunk listing forward to widen the spinal canal and to reduce the compression on the nerve roots. In more severe cases, the patients may walk with flexed hips and knees [48]. With progression of the condition, the walking distance reduces. Not uncommonly, the patient reports a reduction in height, which averages 4–24 cm. in 1–22 years [49].

Inspection from the back generally shows a hump in the low back with the concavity on the opposite side. Generally pelvic obliquity occurs; Radcliff et al. [50] reported a pelvic tilt in 87% of patients with DLS [50, 51]. Patients with a single lumbar curve were more likely to have a higher pelvis contralaterally (79%), as a compensatory mechanism to maintain coronal trunk balance [50]. Patients with a lumbar curve and a compensatory lumbosacral hemicurve did not display consistent pelvic obliquity compensatory patterns [50].

In the presence of marked pelvic obliquity or pelvic tilt, apparent leg length discrepancy becomes evident, with the leg ipsilateral to the lumbar convexity appearing shorter [50] and the posterior superior iliac spines being unlevel. The coronal spinal imbalance can be determined by measuring the distance from C7



Figure 7.

Coronal imbalance and sagittal imbalance are evident in this man aged 62 years old. He complained of radiculopathy localized to the right anterior thigh. The radiograph (a) showed a right lumbar scoliosis with a mild compensatory left thoracolumbar scoliosis. Though the sagittal imbalance was not significant (b), there was a reduction in sacral slope and thoracolumbar lordosis.

to the vertical line extended from the gluteal cleft. The distance measured in mm. represents the coronal shift (**Figure 7**). In the presence of a single thoracolumbar or lumbar curve, the spine is generally decompensated to the side of lumbar convexity.

In patient with DLS, sagittal imbalance is more significant clinically than coronal imbalance [52]. Loss of lumbar lordosis is generally evident with patients leaning forward [48]. In cases with spinal stenosis, patients may flex their hips and knees to compensate for the sagittal spinal imbalance. In long standing cases, contracture of the hips may result, which can be assessed by the Thomas leg raise test [53].

The physical examination can also be used to identify the pain driver. Tenderness is generally elicited at the junction between two major curves, including the junction between the thoracic and lumbar curves and between the lumbar curve and the compensatory lumbosacral hemicurve. Also, pain can be elicited at the apex of the thoracolumbar or lumbar curves [33] and on the iliac crest where the tendons of the lumbar paraspinal muscles attach [1]. A neurological examination which consists of the assessment of motor strength, reflexes, sensation, and gait also needs to be performed, to assess the extent of neurological involvement and to rule out other possible causes of back pain.

5. Radiographic examination

Radiographic examination usually involves taking erect posteroanterior and lateral full spine radiographs. This enables the evaluation of the regional spinopelvic parameters as well as global spinal balance. Sagittal spinal balance has been reported to be positively associated with health-related quality of life (HRQOL) [32, 35].

5.1 Frontal spinal radiograph

A frontal radiograph generally depicts a thoracolumbar or lumbar scoliosis, which is generally shorter than that seen in ADIS, involving only a few vertebral segments. Additionally, vertebral body deformities are less severe than that of ADIS [54]. Of interest is that the majority of lumbar curves with a convexity to the right had apexes above L2 and those with convexities to the left had apexes below L2 [55]. The authors, however, did not offer any explanation for the findings [55].

Depending on the degree of the DLS, radiographic features differ. In the early stages, mild lumbar intervertebral wedging may be present, and compensation in the form of wedging to the opposite direction may be seen in the upper vertebral levels [8].

With progression of the condition, vertebral instability in the form of a translatory shift may be evident [39]. Very often, lateral vertebral translation or laterolisthesis is accompanied by vertebral rotation, when it is known as VRO. VRO most often affects the L3 and L4 levels and less commonly L2–L3 and L4–L5. Of note is that 50% of the VRO occurs at the junction between the main lumbar curve and the compensatory lumbosacral hemicurve [39]. VRO also occurs at the apex of the main lumbar curve and at the junction between the thoracic curve and the lumbar curve [39]. Open subluxation tends to occur on the convexity of the main curve, while closed dislocation tends to occur at the junction between the scoliosis curves [39].

In late stage DLS, osteophytosis may be seen in the vertebral end plates in the concavity of the lumbar scoliosis. Large bridging osteophytes provide stability to previously unstable vertebrae. Also evident are signs of disc degeneration, facet arthrosis, and spinal stenosis [4]. The possibility of lateral recess stenosis and central spinal stenosis may also be discerned from the frontal radiographs. The Cobb

angle and the apical vertebral rotation need to be measured, as they are related to the risk of curve progression and back pain. A lumbar scoliosis in excess of 30°, an apical vertebral rotation in excess of 33%, and lumbarization increase the propensity for curve progression and the incidence of back pain [34].

Pelvic obliquity is common in DLS [50]. From the radiograph, the coronal balance may also be determined. It is the distance between the vertical lines extended from the mid sacrum (central sacral line, CSL) to mid C7. When it is in excess of 4 cm, it is associated with deterioration of pain and function scores in adult scoliosis patients [32, 35]. Of importance is that Ferrero et al. [39] reported that the side of radicular pain corresponded to the side of coronal shift in 70% of the subjects [39].

5.2 Lateral spinal radiograph

Lateral spinal radiography generally reveals a reduction of lumbar lordosis and sagittal imbalance. This is important as regional spinopelvic parameters and global spinal balance have been found to be associated with clinical outcome. A study showed that pelvic incidence-lumbar lordosis (PI-LL) mismatch \geq 10° and pelvic tilt \geq 22° were reported to correlate with disability [56].

Sagittal spinal imbalance is common in patients with DLS. One of the commonly used parameters is the sagittal vertical axis, which is the distance between the vertical line dropped from C7 and the posterosuperior angle of the sacrum. It is noteworthy that a SVA ≥ 7 cm. is associated with clinical symptoms [32]. The finding was supported by other studies [36, 56]. In mild and moderate spinal malalignment, patients with DLS tend to incline the trunk forward and tend to develop a posterior pelvic shift to maintain balance and to provide relief from neurologic symptoms, especially in the presence of concomitant degenerative spondylolisthesis [48].

6. Advanced clinical imaging

In the presence of claudication and neurological symptoms, computed tomography and magnetic resonance imaging (MRI) may be indicated for diagnosis, monitoring, and follow-up. When decreased BMD is suspected, bone density measurement using a DEXA scan or radiofrequency echographic multi spectrometry (REMS) method is indicated. Computed tomography generally shows signs of facet arthropathy and spinal stenosis, including central spinal stenosis, lateral recess stenosis, and foraminal stenosis.

Magnetic resonance imaging of the lumbar spine is used to assess the soft tissues of the spine, including the spinal cord and tissues within the spinal canal. It is also useful for the assessment of the degenerative changes of disc and facets as well as to assess the extent of spinal stenosis. Recent studies have shown that bone marrow edema was associated with low back pain [57, 58]. In a study of 120 DLS patients, Nakamae et al. [57] found that bone marrow edema was associated with low back pain (**Figure 8**) and that the bone marrow edema score was positively associated with low back pain severity [57]. Bone marrow edema was often seen in the concavity of the curve [57]. Buttermann et al. [58] found that the painful scoliosis which was located at the apex of the scoliosis curve or at the lumbosacral junction was associated with a higher frequency of end plate inflammatory changes [58]. The study showed that the end plate changes demonstrated a bimodal distribution, with peaks at L2–L3 and L5–S1 [58].



Figure 8.Bone marrow edema is evident just below the inferior end plate of L2 and superior end plate of L3 in the left.

MRI may also reveal a reduction in muscle mass in the lumbar paraspinal muscles in patients with DLS [59, 60].as paraspinal muscles are involved in the stability of the lumbar spine; Barker et al. [59] suggested that their atrophy was associated with lumbar instability [59]. The percentage of fat infiltration areas in paraspinal muscles was significantly higher on the concave side than the convex side. Further the asymmetry of the multifidus muscle change is positively correlated with the lumbar curvature, lateral vertebral translation, and apical vertebral rotation [60].

Studies showed that BMD was lower in DLS patients than normal controls [7]. Also, BMD was found to correlate negatively with the Cobb angle [61]. Patients with DLS and Cobb angle \geq 20° had lower BMD than those with curves less than 20° [7]. A low BMD was associated with increased risk of curve progression. Thus assessment of BMD is of importance in DLS patients.

BMD can be assessed using either the DEXA or the REMS methods. Though DEXA is the gold standard in the assessment of BMD, it has to be noted that DEXA is prone to errors, which includes wrong inclusion of vertebrae and positioning of patient [62]. In the presence of DLS, the spinal BMD could be falsely elevated [62],

as the degenerative changes, such as aortic calcification, vertebral osteophytes, facet degeneration, end plate sclerosis, and vertebral rotation, may all have artificially elevated readings obtained from a standard anteroposterior lumbar DEXA scan [63], causing errors in clinical management. A study by Pappou et al. [62] study showed that the falsely elevated scores increased with Cobb angles in excess of 22.5° [62]. The viable alternative for conducting a BMD evaluation of patients with DLS are the hip DEXA values [62]. Alternately, REMS measurement can be used. It relies on a machine algorithm and takes into consideration the entire bony profile including the vertebral microarchitecture, compact bone to trabecular bone mineral density ratio, and collagen index, thus reducing the many errors that are associated with the DEXA measurement [64, 65].

7. Body composition assessment

The body composition of the patient needs to be evaluated, when sarcopenia or loss of muscle mass with aging is suspected. Recent studies have shown that 46.6% of patients with DLS had reduced muscle mass involving the extremities and the trunk [26]. The trunk SMI was found to be significantly negatively correlated with sagittal vertical axis, pelvic tilt (PT), lumbar scoliosis, and apical vertebral rotation, suggesting that the reduction in trunk muscle mass was related to the stooped posture, pelvic retroversion, and lumbar scoliosis [26].

8. Treatment

Patients with DLS generally seek treatment for pain and disabilities, instead of deformities [52]. Conservative treatment is generally indicated, and this often involves methods to control or relieve pain, such as epidural injection, non-steroidal anti-inflammatory drugs, analgesics, traction, electrotherapies, dry needling, manipulation, mobilization, and deep tissue massage. These methods can generally provide relief, though temporarily [66, 67]. A systematic review concluded that there was only level IV evidence in support of the effectiveness of physical therapy, chiropractic care, and bracing in the treatment of adult scoliosis patients and level III evidence for steroid injection [66]. The long-term successful rate of conservative treatment of symptomatic adult scoliosis was only 27% [68, 69].

The poor outcomes of the above interventions are not unexpected, as the treatments were directed towards pain relief, but not the deformities and the global imbalance that are causing the symptoms [69]. Treatment approaches that target spinal deformities yielded better results in terms of reduction in pain and disability ratings in ADIS patients [70–76]. Yet, it has to be noted that many of the studies targeted younger cohorts who suffered from ADIS rather than DLS. Further for patients who are in pain or have difficulties performing exercises, a spinal brace may be indicated. It stabilizes the spine, improves the sagittal imbalance, and reduces the load in the lumbar spine. de Mauroy et al. [77] have shown that a spinal brace is able to stabilize progressive curves in 80% of the adults with scoliosis [76].

8.1 Scoliosis-specific exercises

Many case reports and case series studies have reported that scoliosis-specific exercises (SSE) and multi-modal rehabilitation reduce pain, disability, and curves in patients ADIS [70–76]. Yet, only a few studies have targeted patients with DLS. Daily

side plank exercises on the side of curve convexity for 3–22 months were reported to reduce the curves significantly in 30% of the patients with ADIS and DLS [70]. The study, however, did not evaluate the impact of the exercises on pain and disabilities [70]. A prospective pilot study by Ng et al. [72] showed that 9 months of scoliosis-specific exercises at home reduced the thoracolumbar or lumbar curves in over 30% of the ADIS and DLS subjects [72]. Also, our unpublished study showed that 6 weeks of SSE reduced pain and disability ratings of subjects with ADIS and DLS.

While many studies have addressed the coronal curves in ADIS and DLS patients, very few studies have addressed the impact of SSE on the sagittal profile of patients [72]. The effects of SSE on the sagittal profile of this group of patients are thus uncertain. Additionally while SSE may be indicated in the management of patients with DLS, our experience has shown that many older patients had difficulties in mastering the Schroth exercises or the scientific exercises approach to scoliosis (SEAS). They had difficulties in coordinating breathing together with the corrective movements needed. A number of patients encountered problems holding the spine in an erect position, while other patients had increased low back pain soon after the exercises, despite normal spine DEXA scores. This was possibly a result of the DEXA over-estimating the spinal BMD scores when the patient was actually osteopenic. Instead of focusing on corrective exercises, the patients may need to be instructed to adopt corrective postures during daily activities as they are easier to master.

In the presence of a left lumbar curve, the patient can stand, with his or her right knee flexed to lower the right pelvis. Alternately, the patient can raise the left heel. This raises the left pelvis [78]. Either way, this lowers the right sacrum, in relation to the left, and reduces the lumbar scoliosis. This may enable the patient to stand longer. To further reduce the left lumbar curve or reverse the curve, the patients could side shift to the left [78]. Conversely, in the presence of a right lumbar curve, the patient should reverse the above postures.



Figure 9.Contraction of the gluteus medius would level the pelvis. (a) The patient was standing naturally. The right pelvis can be seen shifted to the right and was higher, with pelvis obliquity. (b) Contraction of the right gluteus medius leveled the pelvis. The patient was instructed to learn walking in this corrected position.



Figure 10.
This patient with left thoracolumbar scoliosis can derotate the left lumbar spine forward during daily activities.

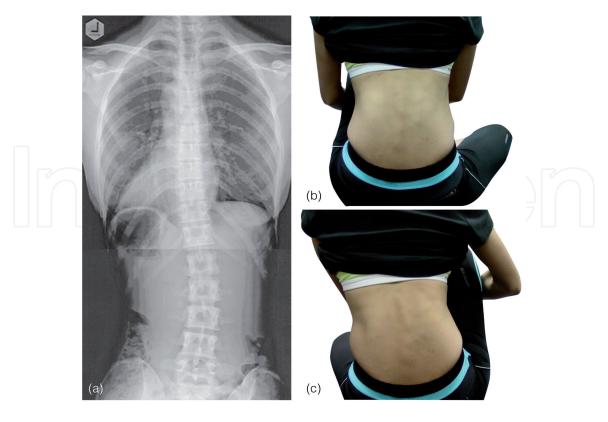


Figure 11.
Patient should refrain from faulty habitual postures, which would aggravate the scoliosis. (a) Frontal lumbar radiograph showed a right lumbar scoliosis, with apex at L2 in a female patient with ADIS. (b) When sitting on the floor, he habitually flexed her left hip and knee, increasing the right lumbar scoliosis. (c) When she flexed her right hip and knee, however, the lumbar curve reduced. Yet, the latter posture should also be discouraged, as lumbar lordosis was not maintained.

Yet, it is difficult to maintain the correct standing posture during ambulation, unless the patient learns how to level the pelvis. Patients with a left thoracolumbar or lumbar curve needs to contract the right hip abductor to bring the pelvis to the midline and level it (**Figure 9**) while derotating the left lumbar curve forward [51] (**Figure 10**). Similarly, patients with right thoracolumbar or lumbar curves need to derotate the right lumbar curve forward while contracting the left hip abductors [51]. The patient then learns to walk with the gluteus medius contracted.

When sitting, the patient needs to maintain the lumbar lordosis, as forced thoracolumbar lordosis was found to reduce double major curves [79]. In the presence of a loss of lumbar lordosis, the patient may be advised to wear a wearable lumbar cushion at all times, though its effects in single thoracolumbar or lumbar curve have not to date been investigated. It is also crucially important that the patient refrains from adopting postures or activities that reinforce the faulty scoliosis pattern (**Figure 11**).

8.2 Sole lift

Functional leg length discrepancy is common, as compensation in patients with DLS. Prescription of a sole lift, in the presence of an apparent LLD, but not anatomical LLD, may induce a compensatory lumbosacral hemicurve, instead of reducing the main lumbar curve [51]. Patients should preferably be advised to contract the gluteus medius on the side of higher pelvis to level the pelvis [51], to flex the knee on this side to lower the pelvis or to raise the heel of the leg ipsilateral to the convexity of the lumbar curve [78] to raise the pelvis.

8.3 Spinal bracing

Spinal bracing has been advocated in the management of adult scoliosis, to halt progression of curves, restore sagittal balance, and treat pain and disability. The effectiveness of braces, however, has been controversial [77, 80]. A number of studies opined that spinal braces do not halt curve progression. Any benefits of pain relief are offset by the deconditioning of the lumbar paraspinal muscles [80].

Recent studies, however, have shown that spinal bracing is effective in reducing pain and halting curve progression (**Figure 12**) [77, 80]. A study which used a lordosing bivalve polyethylene overlapping brace to treat 158 adults with spinal deformities for over 5 years showed that 24% of the curves improved by \geq 5°, 56% of the curves stabilized, and 20% worsened by \geq 5° [77]. The findings were supported by a long-term follow-up study of 22 years [80]. It was shown that brace wear reduced the progression of curves in both ADIS and DLS patients [80]. The yearly progression for curves in patients with DLS reduced from 1.47° to 0.24° per year [80]. de Mauroy [77] suggested that the brace treatment not only is palliative but also helps to stabilize the lumbar spine in lordosis [77].

8.4 Increase paraspinal muscle mass

Apart from SSE to reduce the scoliosis angles, patients should be encouraged to perform exercises to improve muscle mass, as sarcopenia is prevalent in patients with DLS [26].

Many studies have shown that physical exercises, proper nutrition, and optimal hormonal homeostasis are the three pillars to fight or treat (pre)-sarcopenia [81, 82]. Physical exercises should consist of resistance and endurance exercise



Figure 12.

The man aged 73 years of age complained of right anterior thigh pain with intermittent claudication. The lumbar radiograph and MRI (a) showed a right thoracolumbar scoliosis (b) with a reduction in thoracolumbar lordosis (c) and mild sagittal imbalance. The patient was treated by exercises that increased the thoracolumbar lordosis and a lordotic spinal brace. (d) Despite that the patient was non-compliant and wore the brace only at home for 4 hours daily, the brace treatment increased the walking distance from 10 minutes to around 30 minutes.

training (50% resistance training and 50% endurance training). They should be performed at least three times a week [83]. Resistance exercise training aims at improving muscle strength, muscle mass, and BMD and optimizing the hormonal milieu [81], whereas endurance exercise training targets at improving the cardio-vascular function, increasing the insulin sensitivity and the anti-inflammatory

effects, as well as maintaining the endocrine milieu [81, 83]. Thus patients with DLS should also be encouraged to take up a regular exercise program. Nourishment with optimal protein intake is also important. Patients should take 25–30 g of protein with essential amino acids daily [82]. Supplements should include long-chain omega 3 fatty acids and antioxidants (e.g., polyphenols such as hydroxytyrosol, resveratrol, epigallocatechin 3 gallate, curcumin, quercetin) and vitamin D [81, 84]. Ideally, vitamin D should be dosed to attain a serum level of 30 ng/L [82]. Depending on the hormonal level, testosterone and creatine may also be prescribed to treat the (pre)-sarcopenia [82].

8.5 Osteoporosis management

Reduction in BMD is common in patients with DLS. A study by Eguchi et al. [26] showed that trunk skeletal muscle mass correlated positively with BMD [26]. The presence of sarcopenia would thus be indicative of osteoporosis [26]. Depending on the BMD, treatment by medication and/or nutritional supplementation may be required. Pharmacological agents are indicated in the presence of a moderate or high risk of fracture. Common medications prescribed for postmenopausal osteoporosis include estrogen, estrogen + progestin, bisphosphonates, selective estrogen receptors modulators (SERMS), the denosumab, calcitonin, and teriparatide. Each of them has different indications and contraindications [85]. Whether these medications help stabilize or halt the progression of DLS has however not been studied to date. Clinically, however, the author has seen cases of rapidly progressing DLS controlled by administration of bisphosphonates.

Together with pharmacological agents, nutritional supplements such as calcium, vitamin D3, vitamin K2, and silica and abstinence from alcohol and smoking are indicated [86]. Recent studies have demonstrated that calcium supplementation is associated with a low bone calcium content with a parallel increase in vascular calcium content [86] and that low BMD is correlated with an increased cardiovascular mortality [87, 88]. The calcium paradox is speculated to be related to vitamin K2 deficiency [89]. It is thus prudent to advise patients with DLS and osteoporosis to take vitamin K2 along with a calcium supplement.

8.6 Surgery

When conservative treatment fails to provide pain relief or control the symptoms, the patient needs to be referred for surgery, particularly in the presence of neurological signs and symptoms [90], as the outcome of surgery has been reported to be superior to conservative treatment [48], albeit with a much higher risk of complications.

9. Conclusions

When treating patients with DLS, we should not only target symptomatic relief, but it is also necessary to address the underlying aggravating or risk factors of the condition. Physiotherapy, manipulation, and needling can be used to treat pain, together with spinal bracing. Scoliosis-specific exercises should be prescribed, and corrective postures should be encouraged during daily activities to improve the sagittal and coronal spinal imbalances. In the presence of sarcopenia and decreased BMD, resistance exercise training and nutritional supplements are also indicated.

Conflict of interest

The author declares no conflict of interest.





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References

- [1] Aebi M. The adult scoliosis. European Spine Journal. 2005;**14**:925-948
- [2] Hong JY, Suh SW, Modi HN, Hur CY, Song HR, Park JH. The prevalence and radiological findings in 1,347 elderly patients with scoliosis. Journal of Bone and Joint Surgery. British Volume (London). 2010;**92**:980-983
- [3] Kebaish KM, Neubauer PR, Voros GD, Khoshnevisan MA, Skolasky RL. Scoliosis in adults aged 40 years and older: Prevalence and relationship to age, race, and gender. Spine (Phila Pa 1976). 2011;36(9):731-736
- [4] Kobayashi T, Atsuta Y, Takemitsu M, Matsuno T, Takeda N. A prospective study of de novo scoliosis in a community based cohort. Spine (Phila Pa 1976). 2006;31:178-182
- [5] Schwab F, Dubey A, Pagala M, Gamez L, Farcy JP. Adult scoliosis: A health assessment analysis by SF-36. Spine (Phila Pa 1976). 2003;28(6):602-606
- [6] Kilshaw M, Baker RP, Gardner R, Charosky S, Harding I. Abnormalities of the lumbar spine in the coronal plane on plain abdominal radiographs. European Spine Journal. 2011;**20**:429-433
- [7] Xu L, Sun X, Huang S, Zhu Z, Qiao J, Zhu F, et al. Degenerative lumbar scoliosis in Chinese Han population: Prevalence and relationship to age, gender, bone mineral density, and body mass index. European Spine Journal. 2013;**22**(6):1326-1331
- [8] Murata Y, Takahashi K, Hanaoka E, Utsumi T, Yamagata M, Moriya H. Changes in scoliotic curvature and lordotic angle during the early phase of degenerative lumbar scoliosis. Spine. 2002;**27**(20):2268-2273

- [9] Pritchett JW, Bortel DT. Degenerative symptomatic lumbar scoliosis. Spine (Phila Pa 1976). 1993;18(6):700-703
- [10] Sun XY, Kong C, Lu SB, Wang W, Cheng YZ, Sun SY, et al. The parallelogram effect of degenerative structures around the apical vertebra in patients with adult degenerative scoliosis: The influence of asymmetric degeneration and diagonal degeneration on the severity of deformity. Medical Science Monitor. 2019;25:3435-3445
- [11] Bao H, Zhu F, Liu Z, Bentley M, Mao S, Zhu Z, et al. Vertebral rotatory subluxation in degenerative scoliosis: Facet joint tropism is related. Spine (Phila Pa 1976). 2014;39(26 Spec No.):B45–B51
- [12] Yagi M, Akilah KB, Boachie-Adjei O. Incidence, risk factors and classification of proximal junctional kyphosis: Surgical outcomes review of adult idiopathic scoliosis. Spine (Phila Pa 1976). 2011;36(1):E60-E68
- [13] Shafaq N, Suzuki A, Matsumura A, Terai H, Toyoda H, Yasuda H, et al. Asymmetric degeneration of paravertebral muscles in patients with degenerative lumbar scoliosis. Spine. 2012;37(16):1398-1406
- [14] Kim H, Lee CK, Yeom JS, Lee JH, Cho JH, Shin SI, et al. Asymmetry of the cross-sectional area of paravertebral and psoas muscle in patients with degenerative scoliosis. European Spine Journal. 2013;22(6):1332-1338
- [15] Poureisa M, Behzadmehr R, Daghighi MH, Akhoondzadeh L, Fouladi DF. Orientation of the facet joints in degenerative rotatory lumbar scoliosis: An MR study on 52 patients. Acta Neurochirurgica. 2016;158(3):473-479

- [16] Gao T, Lai Q, Zhou S, Liu X, Liu Y, Zhan P, et al. Correlation between facet tropism and lumbar degenerative disease: A retrospective analysis. BMC Musculoskeletal Disorders. 2017;18(1):483
- [17] Hasegawa K, Shimoda H, Kitahara K, et al. What are the reliable radiological indicators of lumbar segmental instability? Journal of Bone and Joint Surgery. British Volume (London). 2011;93:650-657
- [18] Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reilly J. Pathology and pathogenesis of lumbar spondylosis and stenosis. Spine (Phila Pa 1976). 1978;3:319-328
- [19] Kotwal S, Pumberger M, Hughes A, et al. Degenerative scoliosis: A review. HSS Journal. 2011;7:257-264
- [20] Tempel ZJ, Gandhoke GS, Bonfield CM, et al. Radiographic and clinical outcomes following combined lateral lumbar interbody fusion and posterior segmental stabilization in patients with adult degenerative scoliosis. Neurosurgical Focus. 2014;36:E11
- [21] Korovessis P, Piperos G, Sidiropoulos P, Diures A. Adult idiopathic lumbar scoliosis. A formula for prediction of progression and review of the literature. Spine. 1994;**19**(17):1926-1932
- [22] Freedman BA, Horton WC, Rhee JM, et al. Reliability analysis for manual radiographic measures of rotatory subluxation or lateral listhesis in adult scoliosis. Spine (Phila Pa 1976). 2009;34:603-608
- [23] Trammell TR, Schroeder RD, Reed DB. Rotatory olisthesis in idiopathic scoliosis. Spine (Phila Pa 1976). 1988;13:1378-1382

- [24] Watanuki A, Yamada H, Tsutsui S, Enyo Y, Yoshida M, et al. Radiographic features and risk of curve progression of de-novo degenerative lumbar scoliosis in the elderly: A 15-year follow-up study in a community-based cohort. Journal of Orthopaedic Science. 2012;17(5):526-531
- [25] Jimbo S, Kobayashi T, Aono K, Atsuta Y, Matsuno T. Epidemiology of degenerative lumbar scoliosis: A community-based cohort study. Spine (Phila Pa 1976). 2012;37(20):1763-1770
- [26] Eguchi Y, Suzuki M, Yamanaka H, Tamai H, Kobayashi T, Orita S, et al. Associations between sarcopenia and degenerative lumbar scoliosis in older women. Scoliosis and Spinal Disorders. 2017;12:9
- [27] Sanada K, Miyachi M, Tanimoto M, Yamamoto K, et al. A cross sectional study of sarcopenia in Japanese men and women reference values and association with cardiovascular risk factors. European Journal of Applied Physiology. 2010;**110**:57-65
- [28] Liu W, Chen XS, Jia LS, Song DW. The clinical features and surgical treatment of degenerative lumbar scoliosis: A review of 112 patients. Orthopaedic Surgery. 2009;1(3):176-183
- [29] Briard JL, Jegou D, Cauchoix J. Adult lumbar scoliosis. Spine. 1979;4(6):526-532
- [30] Kostuik JP, Bentivoglio J. The incidence of low-back pain in adult scoliosis. Spine (Phila Pa 1976). 1981;**6**(3):268-273
- [31] Simmons EH, Jackson RP. The management of nerve root entrapment syndromes associated with the collapsing scoliosis of idiopathic lumbar and thoracolumbar curves. Spine (Phila Pa 1976). 1979;4(6):533-541

- [32] Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, Schwab F. The impact of positive sagittal balance in adult spinal deformity. Spine. 2005;**30**(18):2024-2029
- [33] Jackson RP, Simmons EH, Stripinis D. Incidence and severity of back pain in adult idiopathic scoliosis. Spine (Phila Pa 1976). 1983;8(7):749-756
- [34] Weinstein SL, Ponseti IV. Curve progression in idiopathic scoliosis. The Journal of Bone and Joint Surgery. American Volume. 1983;**65**(4):447-455
- [35] Ploumis A, Liu H, Mehbod AA, Transfeldt EE, Winter RB. A correlation of radiographic and functional measurements in adult degenerative scoliosis. Spine (Phila Pa 1976). 2009;34(15):1581-1584
- [36] Lafage V, Schwab F, Patel A, Hawkinson N, Farcy JP. Pelvic tilt and truncal inclination: Two key radiographic parameters in the setting of adults with spinal deformity. Spine (Phila Pa 1976). 2009;34(17):E599-E606
- [37] Liu H, Ishihara H, Kanamori M, Kawaguchi Y, Ohmori K, Kimura T. Characteristics of nerve root compression caused by degenerative lumbar spinal stenosis with scoliosis. The Spine Journal. 2003;**3**(6):524-529
- [38] Gardner RO, Torrie PA, Bertram W, Baker RP, Harding IJ. A radiological evaluation of lateral vertebral subluxation associated with spinal stenosis in the lumbar spine in degenerative scoliosis. Spinal Deformity. 2013;1(5):365-370
- [39] Ferrero E, Khalif M, Marie-Hardy L, Regnard N, et al. Do curve characteristics influence stenosis location and occurrence of radicular pain in adult degenerative scoliosis? Spine Deformity. 2019;7:472-480

- [40] Epstein JA, Epstein BS, Jones MD. Symptomatic lumbar scoliosis and degenerative changes in the elderly. Spine. 1979;4:542-547
- [41] Grubb S, Lipscomb H. Diagnostic findings in painful adult scoliosis. Spine. 1992;**17**(5):518-527
- [42] Vanderpool DW, James JI, Wynne-Davies R. Scoliosis in the elderly. The Journal of Bone and Joint Surgery. American Volume. 1969;**51**(3):446-455
- [43] Amundsen T, Weber H, Nordal HJ, Magnaes B, Abdelnoor M, Lilleås F. Lumbar spinal stenosis: Conservative or surgical management? A prospective 10-year study. Spine. 2000;25(11):1424-1435
- [44] Smith JS, Fu KM, Urban P, Shaffrey CI. Neurological symptoms and deficits in adults with scoliosis who present to a surgical clinic: Incidence and association with the choice of operative versus nonoperative management. Journal of Neurosurgery. Spine. 2008;9:326-331
- [45] Fu KM, Rhagavan P, Shaffrey CI, Chernavvsky DR, Smith JS. Prevalence, severity, and impact of foraminal and canal stenosis among adults with degenerative scoliosis. Neurosurgery. 2011;69:1181-1187
- [46] Marty-Poumarat C, Scattin L, Marpeau M, Garreau de Loubresse C, Aegerter P. Natural history of progressive adult scoliosis. Spine. 2007;32(11):1227-1234
- [47] Faraj SS, Holewijn RM, van Hooff ML, de Kleuver M, Pellisé F, Haanstra TM. De novo degenerative lumbar scoliosis: A systematic review of prognostic factors for curve progression. European Spine Journal. 2016;25(8):2347-2358
- [48] Buckland AJ, Vira S, Oren JH, Lafage R, Harris BY, et al. When is

- compensation for lumbar spine stenosis a clinical sagittal plane deformity? The Spine Journal. 2016;**16**:971-981
- [49] Stagnara P. Scoliosis in adults. Consequences of idiopathic scoliosis in adulthood. Chirurgie. 1982;**108**(4):356-363
- [50] Radcliff KE, Orozco F, Molby N, Chen E, Sidhu GS, Vaccaro AR, et al. Is pelvic obliquity related to degenerative scoliosis? Orthopaedic Surgery. 2013;5(3):171-176
- [51] Lehnert SC. Three-Dimensional Treatment for Scoliosis. A Physiotherapeutic Method for Deformities of the Spine. Palo Alto, CA: Martindale Press; 2007
- [52] Acaroğlu RE, Dede O, Pellisé F, Güler UO, Domingo-Sàbat M, et al. Adult spinal deformity: A very heterogeneous population of patients with different needs. Acta Orthopaedica et Traumatologica Turcica. 2016;50(1):57-62
- [53] Ames CP, Smith JS, Scheer JK, et al. Impact of spinopelvic alignment on decision making in deformity surgery in adults: a review. Journal of Neurosurgery: Spine. 2012;**16**(6):547-564
- [54] Grubb SA, Lipscomb HJ, Coonrad RW. Degenerative adult onset scoliosis. Spine (Phila Pa 1976). 1988;**13**(3):241-245
- [55] de Vries AAB, Mullender MG, Pluymakers WJ, Castelein RM, van Royen BJ. Spinal decompensation in degenerative lumbar scoliosis. European Spine Journal. 2010;**19**:1540-1544
- [56] Schwab FJ, Blondel B, Bess S, Hostin R, Shaffrey CI, et al. Radiographical spinopelvic parameters and disability in the setting of adult spinal deformity: A prospective

- multicenter analysis. Spine (Phila Pa 1976). 2013;**38**(13):E803-E812
- [57] Nakamae T, Kiyotaka Yamada K, Takuro Shimbo T, Toshikatsu Kanazawa T, et al. Bone marrow edema and low back pain in elderly degenerative lumbar scoliosis a cross-sectional study. Spine. 2016;41(10):885-892
- [58] Buttermann GR, Mullin WJ. Pain and disability correlated with disc degeneration via magnetic resonance imaging in scoliosis patients. European Spine Journal. 2008;**17**(2):240-249
- [59] Barker KL, Shamley DR, Jackson D. Changes in the crosssectional area of multifidus and psoas in patients with unilateral back pain: The relationship to pain and disability. Spine. 2004;**29**(22):E515-E519
- [60] Xie D, Zhang J, Ding W, Yang S, et al. Abnormal change of paravertebral muscle in adult degenerative scoliosis and its association with bony structural parameters. European Spine Journal. 2019;28(7):1626-1637
- [61] Ding WY, Yang DL, Cao LZ, Sun YP, Zhang W, Xu JX, et al. Intervertebral disc degeneration and bone density in degenerative lumbar scoliosis: A comparative study between patients with degenerative lumbar scoliosis and patients with lumbar stenosis. Chinese Medical Journal. 2011;124(23):3875-3878
- [62] Pappou IP, Girardi FP, Sandhu HS, Parvataneni HK, Cammisa FP Jr, Schneider R, et al. Discordantly high spinal bone mineral density values in patients with adult lumbar scoliosis. Spine (Phila Pa 1976). 2006;31(14):1614-1620
- [63] Rand T, Seidl G, Kainberger F, et al. Impact of spinal degenerative changes on the evaluation of bone mineral density with dual energy X-ray

- absorptiometry (DXA). Calcified Tissue International. 1997;**60**:430-433
- [64] Casciaro S, Conversano F, Pisani F, Muratore M. New perspectives in echographic diagnosis of osteoporosis on hip and spine. Clinical Cases in Mineral and Bone Metabolism. 2015;12(2):142-150
- [65] Cavalli L, Arioli G, Bianchi G, Caffarelli C. Radiofrequency echographic multi spectrometry (REMS) for the non-ionising diagnosis of osteoporosis at femoral neck: Results of a multicenter clinical study comparing rems and dxa. Annals of the Rheumatic Diseases. 2018;77(Suppl 2):811.1
- [66] Everett CR, Patel RK. A systematic literature review of nonsurgical treatment in adult scoliosis. Spine (Phila Pa 1976). 2007;32(19 Suppl):S130-S134
- [67] Kluba T, Dikmenli G, Dietz K, Giehl JP, Niemeyer T. Comparison of surgical and conservative treatment for degenerative lumbar scoliosis. Archives of Orthopaedic and Trauma Surgery. 2009;**129**:1-5
- [68] Savage JW, Patel AA. Fixed sagittal plane imbalance. Global Spine Journal. 2014;4:287-296
- [69] Ryan DJ, Protopsaltis TS, Ames CP, Hostin R, Klineberg E, Mundis GM, et al. T1 pelvic angle (TPA) effectively evaluates sagittal deformity and assesses radiographical surgical outcomes longitudinally. Spine (Phila Pa 1976). 2014;39(15):1203-1210
- [70] Fishman M, Groessl EJ, Sherman KJ. Serial case reporting yoga for idiopathic and degenerative scoliosis. Global Advances in Health and Medicine. 2014;**3**(5):16-21
- [71] Negrini A, Negrini MG, Donzelli S, Romano M, Zaina F, Negrini S. Scoliosis-specific exercises

- can reduce the progression of severe curves in adult idiopathic scoliosis: A long-term cohort study. Scoliosis. 2015;**10**:20
- [72] Ng SY, Chan WY, Ho TK, Ng YL. A pilot study on the effect of outpatient Schroth exercises on thoracolumbar and lumbar curves in adult scoliosis patients. In: Bettany-Saltikov J, Schreiber S, editors. Innovations in Spinal Deformities and Postural Disorders. Rijeka: IntechOpen; 2017. pp. 93-112
- [73] Monticone M, Ambrosini E, Cazzaniga D, Rocca B, Motta L, Cerri C, et al. Adults with idiopathic scoliosis improve disability after motor and cognitive rehabilitation: Results of a randomised controlled trial. European Spine Journal. 2016;25(10):3120-3129
- [74] Barrios C, Lapuente JP, Sastre S. Treatment of chronic pain in adult scoliosis. Studies in Health Technology and Informatics. 2002;88:290-303
- [75] Morningstar MW. Outcomes for adult scoliosis patients receiving chiropractic rehabilitation: A 24-month retrospective analysis. Journal of Chiropractic Medicine. 2011;**10**:179-184
- [76] Harrison DE, Oakley PA. Scoliosis deformity reduction in adults: A CBP[®] Mirror Image case series incorporating the 'non-commutative property of finite rotation angles under addition' in five patients with lumbar and thoraco-lumbar scoliosis. Journal of Physical Therapy Science. 2017;29(11):2044-2050
- [77] de Mauroy JC, Lecante C, Barral F, Pourret S. Prospective study of 158 adult scoliosis treated by a bivalve polyethylene overlapping brace and reviewed at least 5 years after brace fitting. Scoliosis and Spinal Disorders. 2016;11(Suppl 2):28

- [78] Maruyama T, Kitagawal T, Takeshita K, Nakainura K. Side shift exercise for idiopathic scoliosis after skeletal maturity. Studies in Health Technology and Informatics. 2002;**91**:361-364
- [79] van Loon PJ, Kühbauch BA, Thunnissen FB. Forced lordosis on the thoracolumbar junction can correct coronal plane deformity in adolescents with double major curve pattern idiopathic scoliosis. Spine (Phila Pa 1976). 2008;33(7):797-801
- [80] Palazzo C, Montigny JP, Barbot F, Bussel B, Vaugier I, Fort D, et al. Effects of bracing in adult with scoliosis: A retrospective study. Archives of Physical Medicine and Rehabilitation. 2017;98:187-190
- [81] Sgrò P, Sansone M, Sansone A, Sabatini S, Borrione P, Romanelli F, et al. Physical exercise, nutrition and hormones: Three pillars to fight sarcopenia. The Aging Male. 2019;**22**(2):75-88
- [82] De Spiegeleer A, Petrovic M, Boeckxstaens P, Van Den Noortgate N. Treating sarcopenia in clinical practice: Where are we now? Acta Clinica Belgica. 2016;71(4):197-205
- [83] Landi F, Marzetti E, Martone AM, Bernabei R, Onder G. Exercise as a remedy for sarcopenia. Current Opinion in Clinical Nutrition and Metabolic Care. 2014;17(1):25-31
- [84] Driss F, El-Benna J. Chapter 143—Antioxidant effect of hydroxytyrosol, a polyphenol from olive oil by scavenging reactive oxygen species produced by human neutrophils. In: Olives and Olive Oil in Health and Disease Prevention. San Diego, USA: Acad Press; 2010. pp. 1289-1294
- [85] Gallagher JC, Tella SH. Prevention and treatment of postmenopausal osteoporosis. The Journal of Steroid

- Biochemistry and Molecular Biology. 2014;**142**:155-170
- [86] Persy V, D'Haese P. Vascular calcification and bone disease: The calcification paradox. Trends in Molecular Medicine. 2009;**15**:405-416
- [87] Von der Recke P, Hansen MA, Hassager C. The association between low bone mass at the menopause and cardiovascular mortality. The American Journal of Medicine. 1999;**106**:273-278
- [88] Farhat GN, Newman AB, Sutton-Tyrrell K, Matthews KA, Boudreau R, Schwartz AV, et al. The association of bone mineral density measures with incident cardiovascular disease in older adults. Osteoporosis International. 2007;18:999-1008
- [89] Wasilewski GB, Vervloet MG, Schurgers LJ. The bone—Vasculature axis: Calcium supplementation and the role of vitamin K. Frontiers in Cardiovascular Medicine. 2019;**6**:6
- [90] Neuman BJ, Baldus C, Zebala LP, Kelly MP, Shaffrey C, et al. Patient factors that influence decision making: Randomization versus observational nonoperative versus observational operative treatment for adult symptomatic lumbar scoliosis (ASLS). Spine (Phila Pa 1976). 2016;41(6):E349-E358