AIS and spondylolisthesis

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Abstract

Introduction The association of scoliosis and spondylolisthesis is well documented in literature; the nature and modalities of the relationship of the two pathologies are variable and not always clear. Also, etiologic particulars of scoliosis associated with spondylolisthesis are not well defined, even in cases where scoliosis is called idiopathic. In this paper, we review previous literature and discuss the different aspects of the mutual relationship of scoliosis and spondylolisthesis in the adolescent age.

Materials and methods It is a common notion that the highest occurrence of scoliosis associated with spondylolisthesis is at the lumbar level, both in adolescent and in adult patients. It is probable that the scoliosis that is more heavily determined by the presence of spondylolisthesis is at the lumbar level and presents curve angle lower than 15° Cobb and mild rotation. The scoliosis with curve value over 15° Cobb that is present at the lumbar level in association with spondylolisthesis probably is not prominently due to spondylolisthesis: in these cases, spondylolisthesis is probably only partially responsible for scoliosis progression with a spasm mechanism and/or due to rotation of slipping “olisthetic” vertebra.

Discussion We think that the two pathologies should be treated separately, as stated by many other authors, but we would highlight the concept that, whatever be the scoliosis curve origin, spasm, olisthetic or mixed together, this origin has no influence on treatment. The curves should be considered, for all practical effects, as so-called idiopathic scoliosis. We think that generally patient care should be

addressed to treat only spondylolisthesis or only scoliosis, if it is necessary on the basis of clinical findings and therapeutic indications of the isolated pathologies, completely separating the two diseases treatments.

Conclusions Scoliosis should be considered as an independent disease; only in the case of scoliosis curve progression over time, associated scoliosis must be treated, according to therapeutic principles of the care of any so-called idiopathic scoliosis of similar magnitude, and a similar approach must be applied in the case of spondylolisthesis progression or painful spondylolisthesis.

Keywords Spondylolisthesis · Spondylolysis · Adolescent idiopathic scoliosis · Olisthetic scoliosis

Introduction

The association of scoliosis and spondylolisthesis is well documented in literature, with percentage that ranges between 15 and 48 % [1, 2]. Only Fisk et al. [2] and Arlet et al. [3] and Seitsalo et al. [1, 4] report studies of the association of scoliosis with spondylolisthesis in young patients while all other studies group both adolescent and adult patients together. The nature and modalities of the relationship of the two pathologies are variable and not always clear. Also etiologic particulars of scoliosis associated with spondylolisthesis are not well defined, even in cases where scoliosis is referred to as idiopathic. Idiopathic scoliosis has a complex genetic etiology, involving biomechanical aspects of disks, ligaments and bone that predispose such patients to develop scoliosis, and patients with this condition have often a family history of other cases [5–8]. It is, however, questionable to define a scoliosis associated with spondylolisthesis “idiopathic” only when the anamnesis alone reveals other scoliosis cases within the same

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family. The term idiopathic only serves to demonstrate our poor knowledge of the etiology of scoliosis and the continuous advances of our understanding of molecular and genetic basis of this disease in addition to the development of more refined imaging systems (i.e. evidencing the presence of over looked bone deformities or neurologic anomalies in patients with scoliosis previously classified as idiopathic) continue to reduce the percentage of idiopathic cases, creating a better defined etiology for such cases of scoliosis. In the near future, molecular and genetic tests will be available to screen patients at risk of developing scoliosis and to clarify the scoliosis etiology [9–14] including when it is present together with spondylolisthesis. When spondylolisthesis and adjacent scoliosis are reported together in a patient without family history of scoliosis, we could reasonably enquire whether spine curve was caused by vertebral abnormality, or by other factors that produce curves. This occurrence could be related to a general collagen laxity in patients without an evident or defined pathologic laxity, i.e. true Ehlers–Danlos syndrome [15, 16] but in which a condition of this nature has been overlooked. Such patients are predisposed to develop scoliosis and the same tissue laxity could explain the presence of active spondylolisthesis or the predisposing to develop spondylolisthesis. In this paper, we will analyze existing literature and discuss the different aspects of the mutual relationship of scoliosis spondylolisthesis in the adolescent age.

Review

Fisk, Moe et al. in an extensive study [2] analyzed 500 patients with idiopathic scoliosis and a smaller group of 39 patients affected by spondylolisthesis and found that up to 48 % of children with spondylolisthesis developed at least 5° Cobb of scoliosis. In terms of etiology, the majority of literature similarly defines three main categories of spinal curvatures occurring concomitantly with spondylolisthesis [1–4, 15, 17–23]:

1. Idiopathic scoliosis in patients with positive family anamnesis presenting a curvature of the upper spine that is unlikely to be related with the olisthetic defect.
2. Spine curvature of “sciatic” type in which irritation associated with the olisthetic defect induces deformity by muscle spasm.
3. Curvature associated with an “asymmetric olisthetic defect”. In this case, the displaced vertebra is translated in both the sagittal and coronal planes and rotated around the vertical axis, thereby creating an asymmetric foundation that leads to a rotatory deformity of the spine above.

The case of an idiopathic scoliosis synchronous with spondylolysis occurs in 6.2 % of patients with a scoliosis in

a positive familiar anamnesis, according to Fisk et al. [2] and Rick et al. [24]. This value is only slightly higher to the incidence of spondylolysis in general population (4–5 %) without scoliotic curves. In latter cases, the two pathologies are probably unrelated and it is reasonable to think that these should be treated separately.

The “sciatic” curvature is associated with symptomatic spondylolisthesis in patients without any anamnestic evidence of familiar scoliosis, at least as far as it is known, and the condition is related to spine decompensation caused by muscle spasm. Generally, an antero-posterior radiographic exam of the spine shows no pedicle rotation: this aspect is similar in other lumbar scoliosis caused by spasm in other spine diseases, e.g. disk hernia. If symptomatic spondylolisthesis is treated before spine deformity becomes structured, the ending of the muscle spasm can reduce or resolve the deformity in the majority of the cases.

In the case of spine curvature associated with an asymmetric olisthetic defect, the curve demonstrates more rotation than is usual in an idiopathic scoliosis curve of similar magnitude [18]. The spondylolytic, and not the apical, vertebra has the maximal torsion, as would be the case in idiopathic scoliosis [18, 19]. Two mechanisms may be responsible for this association: first, asymmetric olisthesis may be more likely to trigger muscle spasm via tissue irritation inducing sciatic scoliosis; indeed lumbar curves with rotatory olisthesis are more likely to be associated with irradiating pain. Second, the asymmetric olisthesis may create an asymmetric foundation, which causes the vertebrae above the slip to rotate into a torsional lumbar scoliosis. This etiology has been explained by Tojner [19]: he described, in a spine having bilateral spondylolysis, the slipping of the lytic vertebra with its contemporary rotation on the narrower spondylolysis “gap” (Fig. 1). According to Tojner, this slipping has two consequences: the rotation causes the lateral shift of the body of the olisthetic vertebra, since the axis of rotation is excentric, and the rotation exerts a traction on the intervertebral disk, particularly on the side opposite to the axis of rotation. In consequence, the vertebral body can also “sink” [25] on the side opposite to the point of rotation. This “sinking” is responsible for the loss of static balance in the upper spine, which develops a compensatory curve: the inclination and rotation of upper endplate of olisthetic vertebral body creates a rotated “foundation” of the upper lumbar spine that causes the loss of static balance and the development of a “compensatory” scoliosis. Tojner included in the definition “olisthetic scoliosis” all forms of torsion scoliosis in the lumbar spine starting at the site of the olisthetic or lytic vertebra and decreasing upwards, if no other causes of scoliosis were demonstrable. In a group of 237 patients with spondylolysis and/or spondylolisthesis, he found an incidence of olisthetic scoliosis of about 30 %. It is important to

Fig. 1 Male, 15 years old. **a** Scoliosis in L5 spondylolisthesis, maximum olisthetic rotation at L5 level, **b** Scoliosis in L5 spondylolisthesis, maximum olisthetic rotation at L5 level, associated with

high idiopathic scoliosis. 1-AP X-rays projection 2-LL X-rays projection 3-CT scan, showing asymmetric “gap”

emphasis that Tojner found this high percentage of olisthetic scoliosis in a cohort including only three adolescent patients (aged 15 years), and overall they showed the most severe grade of spondylolisthesis (grade IV). The majority of patients with the other three grades spondylolisthesis was formed by people aged between 28 and 44 years and since they were adult, it is reasonable to think that most of them were affected by degeneration of zygapophyseal

joint, ligaments and disks, a degeneration that could cause itself scoliosis.

The “olisthetic” rotation hypothesis has been proposed for a long time: according to Tojner, in 1888 Neugembauer was the first to observe the torsion scoliosis of the lumbar spine which he related to unilateral spondylolisthesis and report on similar cases were published by Diessl [26]. The mechanism of this scoliosis was first described in detail by

Glorieux and Roederer [25]: they illustrated the case of spondylolysis at L5 level, in which the anterior vertebral segments slip over the sacral bone, rotating at the same time on the narrower spondylolytic gap. All these authors share the merit of understanding olisthetic scoliosis etiology without the support of modern imaging as CAT or MRI, but only studying anatomy and with the use of plain X-rays. The former authors' theory has been confirmed by more recent studies performed with the support of CT scan. Peterson et al. [15] in CT studies found, in patients with bilateral pars interarticularis defect associated with grade I–II spondylolisthesis of L5 on S1, an asymmetric rotational slippage of the olisthetic vertebra with the pars defects on one side greater than on the other; they refer to this pattern of CT imaging as “asymmetric ring”. However, frequently lumbar scoliosis may be related to a rotational foundation even without the presence of spondylolysis, or without the presence of “true” spondylolysis, as in the presence of isthmus asymmetric “elongation” or, as is more often likely observed, due to a constitutional defective zygapophysis articulation orientation in lumbar–sacral spine. In the case of curve associated with asymmetric olisthesis often, an in situ fusion of the affected level, that should reduce antalgic curve, is unable to correct the deformity or even to halt the curve progression. According to Peterson et al. [15], there are two explanations for the progression of scoliosis after intervention:

1. The vertebrae may have been fused in situ into a position of permanent asymmetry. Hence the suspect force driving the scoliosis, an asymmetric “foundation” of upper lumbar spine, may have remained postoperatively. Seitsalo et al. [1] is of the same opinion that spondylolisthesis fusion often fails to correct the scoliosis when a significant rotatory component is present.
2. Alternatively, the patient could have a progressive scoliosis synchronous with and independent of symptomatic olisthesis.

Therefore, spondylolisthesis could be considered as the initial cause of a “potential” scoliosis; sometimes spondylolisthesis exacerbates the developing scoliosis, but sometimes spondylolisthesis can have rebalancing effect and so could cause reduction of scoliosis curve progression, because it is possible to observe compensative lumbar–sacral curves in the opposite direction to primary curve, particularly if olisthesis is in L5. In this relationship, the iliolumbar ligaments play an important role, by stabilizing the olisthetic L5, halting its slipping together with other elements such as pelvic incidence etc., making the L5–S1 passage a single part, especially if L5 is deep below the iliac crests. This action initiates the opposite lumbar–sacral curve in an attempt to realign spine (Fig. 2); this

mechanism has been similarly described by Mau [20]. Confirming the stabilization role of the iliac–lumbar ligaments, Mc Phee et al. [21] found that the incidence of scoliosis associated with spondylolisthesis is much greater when the pars defect is at the L4–L5 level rather than at the L5–S1 level. These authors found that eight out of nine patients with L4–L5 spondylolisthesis also had scoliosis. They attributed the higher incidence of scoliosis in these patients to the absence of stabilizing ligaments, such as the iliolumbar ligament, at the L4–L5 level.

Lisbon et al. [17] first compared the presence of scoliosis in patients with symptomatic and asymptomatic spondylolisthesis. They performed a study on a large cohort of 1743 male Israeli soldiers aged 18–30 years. They found that incidence of scoliosis in patients without spondylolysis was 6.65 % in asymptomatic patients, while this rose to a significant 18.3 % in patients reporting back pain. In patients with spondylolysis/spondylolisthesis, the incidence of scoliosis was higher, but again in symptomatic patients the incidence of scoliosis was significantly higher than in asymptomatic group: asymptomatic spondylolisthesis showed a scoliosis incidence 23.8 % that rose to 43.1 % in symptomatic spondylolisthesis. The authors explained this figures by postulating that the majority of scoliosis associated with symptomatic spondylolisthesis is of sciatic origin and could be compared with spasm scoliosis associated with other pathologies causing lumbar pain.

Materials and methods

From 1995 to 2009, we treated 113 patients with spondylolisthesis, in this group 50 patients presented scoliosis (45 %) Only 78 patients had a complete dossier, with a minimum follow-up of 2.6 years and a maximum follow-up of 15 years. 65 % of patients were female and 35 % male. In 37 cases (47 %), spondylolisthesis was associated with scoliosis. Minimum scoliosis curve angle considered was 5° Cobb, while maximum scoliosis angle measured was 83° Cobb. Mean patient age was 14 years (10 years–16 years 8 months). Patients have been divided into three groups:

Group A 33 cases of spondylolisthesis, 60 % female, 40 % male, mean age 13 years and 4 months (range 11 years and 3 months–15 years and 4 months), where there was antalgic treatment of spondylolisthesis in some cases, no treatment of spondylolisthesis in other cases and treatment of associated scoliosis if it was necessary. Spondylolisthesis grade according to Meyerding classification was 1st grade in 27 cases and 2nd grade in 6 cases. All spondylolisthesis were lytic, except two cases (Meyerding 1°) dysplastic in Newmann-Wiltse [27] classification, developmental in Marchetti-Bartolozzi [28] classification and I type in Hermann-Pizzutillo classification [29]. In 13 cases, there was

Fig. 2 Female, 16 years old. Spondylolisthesis L5 II grade associated with opposite scoliosis curve at upper level, 47° Cobb, with opposite rotation. L5 is below iliac crests line, as it is easy to note in the

particular. The *arrows* show the different rotation in the spine. **a** AP X-rays projection standing, **b** AP X-rays projection standing particular, **c** LL X-rays projection standing

associated scoliosis (in 9 cases, curves angle was between 5° and 15° Cobb, comprising 5 lumbar curves (Lenke 5), 2 thoracic-lumbar curves (Lenke 6) and 2 thoracic curves (Lenke 1); in 4 cases, curves angle was over 15° Cobb, comprising 2 lumbar curves (Lenke 5), 1 thoracic lumbar curve (Lenke 6) and 1 thoracic curve (Lenke 1): 3 of these cases have been treated conservatively with curve stability at 2 years and 6-month follow-up).

Motivation for orthopedic consult in the 33 patients has been:

- (a) for 20 cases (60 %) scoliosis, and spondylolisthesis evidence has been collateral,
- (b) for 13 cases (40 %) lumbar pain and all have been treated for pain with analgic therapy (braced for 4–8 weeks).

Three patients showed scoliosis curves reduction/resolution (all three cases had scoliosis curve under 15°) and have been treated beginning 30 days from the reported symptoms presentation. In ten patients, there were no significant curve changes (5 cases with curves below 15° Cobb and 5 cases with curves over 15° Cobb).

In 10 cases (80 %), after mean 6 weeks (range 4–8 weeks) of treatment, pain resolved completely. In two cases, pain resolved partially with no further analgic treatment and no further continuative treatment for scoliosis. One patient with associated scoliosis with olisthetic curve according to Tojner [19] was operated by Wiltse procedure obtaining curve and pain resolution.

Group B 25 cases with spondylolisthesis grade II–III–IV in Meyerding classification, 20 females (80 %) and 5 males (20 %), mean age 15 years (range 10 years–17 years and 8 months), mean follow-up 8 years and 8 months (range 30–150 months). All these patients had lumbar pain, 80 % (20 cases) complained irradiated pain, 68 % (17 cases) complained lower limbs paresthesia, one patient with IV grade dysplastic spondylolisthesis had bladder impairment. In 6 cases only (24 %), we observed associated scoliosis; 2 cases had curve value over 25° Cobb (30° and 35° Cobb), both associated with spondylolisthesis grade IV Meyerding, and presented thoracic lumbar curves (Lenke 6) treated by bracing. The other four cases had thoracic lumbar curves (Lenke 6) below 25° Cobb (15° and 22° Cobb) and have not been treated. In no case, we observed curve reduction or resolution after spondylolisthesis treatment.

Spondylolisthesis treatment in these 25 patients comprised:

- 15 posterior fixation plus PLIF (with cages) (13 grade III Meyerding, 2 grade IV Meyerding),
- Four anterior approach and anterior fusion plus posterior fixation and posterolateral fusion (all cases grade IV Meyerding) (Fig. 3),
- Two posterior fixation and arthrodesis, posterolateral plus interbody bone grafting without cages (all cases grade III Meyerding),
- Four posterior fixation with dynamic instrumentation and posterolateral arthrodesis (all cases grade II Meyerding).

In these operations, we experienced these complications:

- One case of spondylolisthesis one level above surgery (L5 spondylolisthesis grade IV dysplastic),

- Two cases of screws rupture without loss of correction (sacral screws),
- Four cases of hyperlordosis above operated spondylolisthesis due to incorrect sagittal balance.

Group C 20 cases of asymptomatic L5 spondylolisthesis associated with scoliosis over 40° Cobb (mean curve value 62°, range 44° to 83° Cobb). Patients' gender in this group was 16 females and 4 males, mean age 14 years and 4 months (range from 12 years and 7 months to 16 years and 9 months). Spondylolisthesis grade was 7 cases I Meyerding, 9 cases II Meyerding and 4 cases III Meyerding. Associated scoliosis curves were thoracic lumbar (Lenke 5) in 14 patients, thoracic (Lenke 1) in one patient and double curves (Lenke 2) in five patients. No patient showed spondylolisthesis mobility in dynamic X-rays taken before surgery. No patient had fusion instrumentation below L5 (Fig. 4). Inferior arthrodesis limit in scoliosis

Fig. 3 Female, 13 years old. **a** Scoliosis associated with spondylolisthesis I grade, **b** after correction with arthrodesis down to L4, **c** AP and LL X-rays standing at follow-up 8 years and 3 months after operation, stable scoliosis correction without spondylolisthesis worsening

correction has been L1 in 1 patient, L3 in 10 patients and L4 in 9 patients. Mean follow-up was 5 years and 3 months (range from 2 years and 6 months to 15 years). At follow-up, we had no case of spondylolisthesis progression or lumbar pain and no necessity of spondylolisthesis treatment. In one case, we experienced rod rupture in arthrodesis without any worsening in spondylolisthesis.

Discussion

As reported in the previous studies, the majority of spondylolisthesis cases, including those with symptomatic

spondylolisthesis, are not associated with scoliosis (Fig. 5). Seitsalo et al. [1] found the highest figure for the association of scoliosis with spondylolisthesis in their study on young patient: 48 % scoliosis cases out of 190 spondylolisthesis.

It is a common notion that the highest occurrence of scoliosis associated with spondylolisthesis is at lumbar level, both in adolescent and in adult patients: these scoliosis curves show low angular and rotation value. The lowest occurrence of scoliosis associated with spondylolisthesis is at thoracic and thoracic-lumbar levels: these scoliosis generally show higher curve angle and rotation than lumbar curve and often need surgical treatment, and are still identified with the term “idiopathic”. It is probable

Fig. 4 Female, 13 years and 6 months old. **a** Spondylolisthesis L4 IV grade without scoliosis AP and LL X-rays standing, **b** particular of spondylolisthesis, **c** MRI view, **d** first stage: anterior approach, bone grafting, rebalance tilting L5, **e** second stage: posterior approach and

instrumentation stabilization, **f** follow-up 2 years after operation, correction stability, **g** follow-up 4 years after operation, control 2 years after instrumentation removal, complete fusion with stability

Fig. 5 Female 10-year-old spondylolisthesis IV grade, symptomatic lumbar sciatic pain **a** Spine LL X-rays projection standing, **b** lumbar spine MRI, **c** spine AP X-rays projection standing no scoliosis,

d picture of the patient with antalgic posture, lumbar–sacral tract kyphosis, lordosis in the upper spine tract; hips and knees bent

that the scoliosis that are more heavily determined by the presence of spondylolisthesis are at lumbar level and they present a curve angle lower than 15° Cobb and have little rotation. Similarly, on the other hand, also low-grade idiopathic scoliosis often has little rotation. The scoliosis with curve value over 15° Cobb that is present at lumbar level in association with spondylolisthesis probably is not predominantly due to spondylolisthesis; in these cases, spondylolisthesis is probably only partially responsible for scoliosis progression. It is, therefore, possible to define two types of scoliosis associated with spondylolisthesis that could be mixed together with different expression:

1. idiopathic scoliosis, generally with higher curve value,

2. spasm/antalgic scoliosis, with lower curve value.

the latter group of scoliosis could be further divided into:

- pure spasm scoliosis, where scoliosis pattern is similar to scoliosis associated with other painful spine pathology like disk herniation, osteoid osteoma etc., with low angular and rotation value,
- spasm scoliosis combined to olisthetic scoliosis, as described by Tojner, with more rotation in the olisthetic vertebra.

In our opinion, this subsequent distinction is necessary, because when we observe a lumbar curve presumably caused

by spondylolisthesis, it is difficult to judge how much of the curve is due to muscular spasm and how much is due to the rotation “foundation” described by other authors. The vertebra rotation described by Tojner and Peterson [15, 19], and more so if joined to “sinking” vertebra, is the pathological movements most probably associated with rupture of intervertebral disk; such probable disk rupture necessarily causes severe pain and antalgic muscles spasm, that can cause a compensatory antalgic scoliosis identical to the antalgic scoliosis provoked by other pain causing lumbar pathologies, such as herniated disk. Seitsalo and Peterson [1, 15] described cases of lumbar scoliosis resolution after in situ fusion of olisthetic level. In these cases, perhaps, the pain resolution has solved antalgic scoliosis because muscle spasm mechanism was prominent on olisthetic “foundation” mechanism in the genesis of spine curve. This consideration could explain why the fusion succeeded in resolving spine curve even if the intervention did not restore the symmetry of the “ring” described by Peterson [15], because the vertebrae have been fused in the “olisthetic” rotation position. On the other hand, in the cases where the scoliosis remains unaffected by fusion, we could consider that curve was like idiopathic curve and muscular spasm has only exacerbated a pre-existent pathology, not observed during previous examinations. This approach better comprises and actualizes the classification of scoliosis associated with olisthesis proposed by Mau [20] that can be resumed as structural and functional scoliosis.

The structural group curve should be considered as the scoliosis group where spasm/olisthetic rotation worked like enhancement factor and it is the group where scoliosis behaves in a way similar to so-called “idiopathic” scoliosis, even if the curve develops on a defective “foundation”. These scoliosis can present either a single curve or combined curves, with opposite lumbar–sacral curve with a different grade of rotation.

The functional group comprises scoliosis associated with spondylolisthesis where muscle spasm mechanism is the main cause of the curve with low angular grade that maintain this aspect in time.

In our opinion, the great majority of scoliosis cases, particularly those with a higher curve value are totally independent or only partially dependent on spondylolisthesis. Our experience, in accord with Arlet et al. [3], showed that in cases with adolescent scoliosis with lumbar or thoracic-lumbar curves of surgical magnitude associated with an asymptomatic spondylolisthesis, in which we treated only the scoliosis by instrumented arthrodesis, after a 5.3 years mean follow-up we never observed evolutive olisthesis at L5–S1 level. How it is possible to explain this good correction, stable over time, since we did not observe olisthesis worsening despite the bearing of upper arthrodesis (sometimes extended to one vertebra above olisthetic level)? Why is there this stability of

both scoliosis and spondylolisthesis? Why should an arthrodesis bearing on a potentially unstable vertebra not worsen this instability? Why a vertebra, that in some cases triggers scoliosis (and in this case also all authors in previous literature report low-grade scoliosis curves in their series), when it is left free from arthrodesis, should not interfere with scoliosis evolution, i.e. causing loss of balance and/or curve worsening after scoliosis correction? The only explanation is that olisthesis, not only in the cases previously reported after scoliosis correction, is not the primary cause of scoliosis, but, at least, a contributing factor in scoliosis curve progression.

The iliolumbar ligaments stabilize the L5–S1 level, but particularly in young patients in whom these ligaments have not yet acquired the fibrotic rigidity that is their character in adult subjects [30], they should not be able to block an olisthetic vertebra below the arthrodesis area, if that vertebra is not itself stable. It is evident that olisthetic vertebra stability is determined by other elements, aside from iliolumbar ligaments rigidity, e.g., by pelvic incidence, by low sacral slope with impingement effect or by low sacral slope with traction on pars interarticularis [31].

It is a common opinion that in an association of thoracic scoliosis and lumbar spondylolisthesis the two pathologies are not dependent on each other (Fig. 6) while the more scoliosis curve is near to spondylolisthesis area the more this curve is dependent on spondylolisthesis.

Still it is difficult to understand why in some cases a slipped vertebra, so intrinsically unstable, is not the triggering event in the curve formation and evolution, while in other cases the slipped vertebra is the triggering event causing scoliosis curve progression. So also in the cases with lumbar curves, it is an association of an idiopathic scoliosis with spondylolisthesis. It is reasonable thinking that scoliosis curve with low angular value at lumbar level could be caused *mainly by spondylolisthesis*, but still their behavior is the same of idiopathic scoliosis of similar value and they should be considered as idiopathic; lumbar curves showing a higher angular value should be considered pure idiopathic, in which origin olisthesis has a *minor role*.

We should also consider the opposite association, i.e. spondylolisthesis caused by scoliosis.

In literature, spondylolisthesis below the fusion area caused by rigid arthrodesis overload has been reported: underlying areas are in unstable equilibrium or are predisposed in developing olisthesis [32–34]. It is reasonable to assume that, because the majority of scoliosis, even the scoliosis that have not been corrected by arthrodesis, does not cause spondylolisthesis, the scoliosis that develop an underlying olisthesis, this last was predisposed and the arthrodesis only accelerated the evolution. This occurrence seems to be confirmed by the reports of cases of patient treated by instrumented arthrodesis for scoliosis in osteogenesis imperfecta [32]. In these cases, the intrinsic bone fragility of patient predisposes to stress

Fig. 6 Female 15 years 8 months old. **a** Spine AP X-rays projection standing scoliosis in high thoracic area and straight lumbar spine, **b** spine LL X-rays projection particular showing spondylolisthesis I grade

fracture. When instrumentation and fusion are extended to the lower lumbar area, the long lever arm created by the fused segment and the spine may put an undue stress on the already vulnerable segment trapped between the sacrum and the fusion, leading to olisthesis. According to Mau [20], this olisthesis worsening is observed associated with symptomatic scoliosis, where symptomatic scoliosis means a scoliosis associated with particular diseases, and is not a scoliosis associated with pain.

Conclusions

Our opinion is that the association of scoliosis with spondylolisthesis can basically be reduced to three situations:

1. Scoliosis and spondylolysis/spondylolisthesis are undoubtedly two different pathologies having no direct relationship, and they should be treated individually.
2. Spondylolysis/spondylolisthesis causes scoliosis with mixed mechanism.
3. Pure spasm scoliosis with low-grade angular and rotation value: pain reaction explains scoliosis genesis in the case where pain resolution leads to a total or partial curve resolution.
4. *Spasm/olisthetic scoliosis* when treatment does not solve scoliosis, this could be due to a structured curve in cases where antalgic scoliosis has been long lasting and, for all practical purposes, its behavior is the same as that of idiopathic scoliosis; alternative explanation is that curve is pure so-called “idiopathic” and has been exacerbated by muscle spasm.
5. *Scoliosis causes spondylolisthesis* this forms are associated with particular diseases to diffused or regional bone tissue alterations.

In our opinion, this classification is fundamental to select the treatment for the patient who presents an association of scoliosis with spondylolysis/spondylolisthesis. We consider that the two pathologies should be treated separately, as it has been stated by many other authors [1–4, 18, 20, 22, 23, 32], but we would highlight the

concept that, whatever would be the scoliosis curve origin, spasm, olisthetic or mixed together, this origin has no influence on treatment and the curves should be considered, at all practical effects, as the so-called idiopathic scoliosis. We think that generally patient care should be addressed to treat, if necessary, or only spondylolisthesis, completely separating this treatment from scoliosis treatment (so not having scoliosis improvement as a goal, even if in some cases spondylolisthesis therapy could improve associated curve), or only scoliosis, completely separating this treatment from spondylolisthesis treatment; historic distinction spasm/olisthetic scoliosis is completely indifferent to our approach to treatment. Scoliosis should be considered as an independent disease; only in the case of scoliosis curve progression in time, associated scoliosis must be treated, according to therapeutic principles of the care of any so-called idiopathic scoliosis of similar magnitude. Moreover, the great part of the scoliosis curves that have their main cause in spondylolisthesis, both pure spasm form and spasm/olisthetic form, is lumbar, with low angle and rotation value and with low cosmetic impairment.

The most severe scoliosis curves associated with spondylolisthesis should be considered, to all purposes, the so-called idiopathic, either because these curves are true idiopathic (as pure thoracic curves associated with olisthesis without the presence of a lumbar curve; Fig. 6) or because their behavior is the same of a true idiopathic curves; so they must be treated with the same principles used in the treatment of idiopathic scoliosis. If associated spondylolisthesis is asymptomatic, scoliosis treatment must be based on general principles of conservative or surgical treatment of scoliosis curves. Even in the case of a surgical scoliosis treated by vertebral arthrodesis extended to lumbar level, in presence of spondylolisthesis L5–S1, grade I–II–III in Meyerding classification, scoliosis arthrodesis should not include olisthetic level, if this level is not included into the scoliosis curve area that has been selected for fusion in the planning of scoliosis treatment. In our experience, we treated 25 patients for scoliosis associated with spondylolisthesis, performing vertebral arthrodesis without including the olisthetic vertebra, with a mean follow-up of 5.3 years (range 2.5–15 years). According to Arlet et al. [3], we did not observe slippage progression of the olisthetic vertebra excluded by arthrodesis, or did we observe scoliosis curve progression after fusion and no patient complained of lumbar pain after surgery. In presence of spondylolisthesis grade IV of Meyerding classification associated with scoliosis of surgical interest, even if we have no experience of such cases, we think that olisthetic vertebra should be included in vertebral arthrodesis, because if the olisthetic vertebra has been left free, the arthrodesis would bear on a kyphotic, highly unstable area;

the arthrodesis would bear on an area in great part left “empty” by the slipped vertebra, enhancing, in theory, the “expulsion” of the olisthetic vertebra. In the case of asymptomatic spondylolisthesis associated with scoliosis curve without angle value of surgical interest, we suggest that the scoliosis should be treated according to principles of conservative treatment (patient aged below 15 years, Risser 2/3, curve angle value over 15° Cobb). In presence of symptomatic or unstable spondylolisthesis, we think that it is mandatory to consider spondylolisthesis only treatment, while associated scoliosis treatment should be considered on single case basis. In our experience in 25 cases of symptomatic spondylolisthesis grade II/III/IV Meyerding, treated by surgery, only in six cases (24 %) we observed associated scoliosis with a consistent angle value. In these six cases, only two patients presented a scoliosis with curve value over 25° Cobb (30° and 35°), both of them associated with grade IV Meyerding spondylolisthesis with kyphosis of the lumbar–sacral passage, and have been treated by orthosis, while the other four patients scoliosis (15°, 20°, 18° and 22° Cobb) have not been treated. In no case, we observed associated scoliosis curve reduction or resolution after surgical treatment of the spondylolisthesis, except for one patient treated by surgery for a symptomatic grade I spondylolisthesis, following a failed antalgic brace treatment. He presented an associated scoliosis curve with 7° Cobb angle value that has been resolved after spondylolisthesis arthrodesis, but this result is not relevant. However, we think that pure spasm scoliosis could be resolved only if it has been treated very early from the beginning, before the curve is structured. Our attitude toward symptomatic spondylolisthesis grade I/II, above all if it is lytic, is to provide conservative treatment with brace immobilization for 45–60 days (6–8 weeks), using the brace full time only in the first 15 days. After this immobilization time, 80 % of our patients showed complete pain resolution and just a minority of them shows resolution or reduction of associated low-grade scoliosis curves, without any worsening with at least 2-year and 6-month follow-up (in particular only 20 % of these patients showed complete resolution of scoliosis, in the other there was no change in curve value at follow-up). In the remaining, 20 % of cases pain resolution is only partial and patient treatment varies on individual case. In this view, we do not share the opinion of Petersen et al. [15], who advocate an early surgical treatment of lytic spondylolisthesis associated with spasm/olisthetic scoliosis curves, in order to resolve the lytic “gap” in the “asymmetric ring”, with the aim to avoid the development of an hypothetical scoliosis or the worsening of present curves.

Finally, we should consider the spondylolisthesis caused by scoliosis, i.e. spondylolisthesis that did not exist before

scoliosis surgical treatment and that must be considered to be an effect of scoliosis correction/curve arthrodesis in particular diseases (osteoporosis, osteogenesis imperfecta), according to Mau [20] and Barrack et al. [32]. But, perhaps, we should change our point of view and, not considering as in the past many authors did the spondylolisthesis as the cause of scoliosis, vice versa we should investigate more about scoliosis causing spondylolisthesis, starting again from Tojner's thesis on olisthetic scoliosis: "... structural scoliosis and is characterized as follows:

1. On the X-ray film, it presents itself as torsion scoliosis, the maximum torsion being at the lytic vertebra, usually the lumbo-sacral junction.
2. As a rule, it arises at the same time as the spondylolisthesis, i.e. during the growth period.
3. After its onset, it undergoes practically no changes." [19].

It is more probable that, the opposite of Tojner's opinion, idiopathic scoliosis can cause spondylolisthesis, in particular in the chronic form by stress fracture or stress reaction described as Type III in Hermann Pizzutillo classification [29] (that is the only spondylolisthesis classification regarding young patients only).

We would moreover underline that scoliosis have an high power of developing spondylolisthesis, not only due to eventual isthmus alterations, but also because they cause intervertebral disk degeneration, inducing spondylolisthesis; this fact has been enlightened by works of Danielsson and Nachemson [35] and Winter and Silverman [36], showing that patients with scoliosis, both treated by surgery or by orthosis, have a tendency to develop intervertebral disk alterations leading to spondylolisthesis.

Finally, we should ask ourselves if a mild scoliosis with poor angle and rotation value associated with a symptomatic spondylolisthesis is enough to take importance and modify our therapeutic approach. We think that low-grade spasm scoliosis associated with spondylolisthesis is just a descriptive report without any consequence on treatment. The only important pathologic effect that this scoliosis could develop in time is the degeneration mechanism: this mechanism causes more symptomatic scoliosis in adult age, but this pathologic chapter must be still explored [37, 38]. The scoliosis that are severe enough to be treated should be considered idiopathic and should be treated like idiopathic, separately from spondylolisthesis, if spondylolisthesis is asymptomatic. If spondylolisthesis is symptomatic, scoliosis should be treated together with spondylolisthesis or after spondylolisthesis treatment, according to curve magnitude [39].

Conflict of interest None.

References

1. Seitsalo S, Osterman K, Poussa M (1988) Scoliosis associated with lumbar spondylolisthesis. A clinical survey of 190 young patients. *Spine* 13(8):899–904
2. Fisk JR, Moe JH, Winter RB (1978) Scoliosis, spondylolysis and spondylolisthesis. Their relationship as reviewed in 539 patients. *Spine* 3(3):234–245
3. Arlet V, Rigault P, Padovani JP, Touzet P, Finidori G, Guyonvarch G (1990) Scoliosis, spondylolysis and lumbosacral spondylolisthesis. A study of their association apropos of 82 cases in children and adolescents. *Rev Chir Orthop Reparatrice Appar Mot* 76(2):118–127
4. Seitsalo S, Osterman K, Hyvarinen H, Tallroth K, Schlenzka D, Poussa M (1991) Progression of spondylolisthesis in children and adolescent. A long-term follow-up of 272 patients. *Spine* 16(4):417–421
5. Wang WJ, Yeung HY, Chu WC, Tang NL, Lee KM, Qui Y, Burwell RG, Cheng JC (2011) Top Theories for the etiopathogenesis of adolescent idiopathic scoliosis. *J Pediatr Orthop* 31(Suppl 1):S14–S27
6. Kouwenhoven JW, Castelein RM (2008) The pathogenesis of adolescent idiopathic scoliosis: review of the literature. *Spine* 33(26):2898–2908
7. Burwell RG (2003) Aetiology of idiopathic scoliosis: current concepts. *Pediatr Rehabil* 6(3–4):137–170
8. Ahn UM, Ahn NU, Nallamshetty L, Buchowski JM, Rose PS, Miller NH, Kostuik JP, Sponseller PD (2002) The etiology of adolescent idiopathic scoliosis. *Am J Orthop* 31(7):387–395
9. Lombardi G, Akoume MY, Colombini A, Moreau A, Banfi G (2011) Biochemistry of adolescent idiopathic scoliosis. *Adv Clin Chem* 54:165–182
10. Ogilvie J (2010) Adolescent idiopathic scoliosis and genetic testing. *Curr Opin Pediatr* 22(1):67–70
11. Moreau A, Akoumè Ndong MY, Azeddine B, Franco A, Rompré PH, Roy-Gagnon MH, Wang D, Bagnall KM, Poitras B, Labelle H, Rivard CH, Grimard G, Ouellet J, Parent S, Moldovan F (2009) Molecular and genetic aspects of idiopathic scoliosis. Blood test for idiopathic scoliosis. *Orthopäde* 38(2):114–121
12. Letellier K, Azzedine B, Blain S, Turgeon I, Wang S, Ms Boiro, Moldovan F, Labelle H, Poiras B, Rivard CH, Grimard G, Parent S, Ouellet J, Lacroix G, Moreau A (2007) Etiopathogenesis of adolescent idiopathic scoliosis and new molecular concepts. *Med Sci* 23(11):910–916
13. Miller NH (2007) Genetics of familial idiopathic scoliosis. *Clin Orthop Relat Res* 462:6–10
14. Giampietro PF, Blank RD, Raggio CL, Merchant S, Jacobsen FS, Faciszewski T, Shukla SK, Greenlee AR, Reynolds C, Schowalter DB (2003) Congenital and idiopathic scoliosis: clinical and genetic aspects. *Clin Med Res* 1(2):125–136
15. Peterson JB, Wenger DR (2008) Asymmetric spondylolisthesis as the cause of childhood lumbar scoliosis—can new imaging modalities help clarify the relationship? *Iowa Orthop J* 28:65–72
16. Hammerberg KW (2005) New Concepts on the pathogenesis and classification of spondylolisthesis. *Spine* 30(65):S4–S11
17. Lisbon E, Bloom RA, Shapiro Y (1984) Scoliosis in young men with spondylolysis or spondylolisthesis. A comparative study in symptomatic and asymptomatic subjects. *Spine* 9(5):445–447
18. Pneumatos SG, Esses SI (2003) Scoliosis associated with lumbar spondylolisthesis: a case presentation and review of the literature. *Spine J* 3(4):321–324
19. Tojner H (1963) Olisthetic scoliosis. *Acta Orthop Scand* 33:291–300
20. Mau H (1981) Scoliosis and spondylolysis–spondylolisthesis. *Acta Orthop Trauma Surg* 99(1):29–34

21. Mc Phee IB, O'Brien JP (1980) Scoliosis in symptomatic spondylolisthesis. *JBJS* 62B:155–157
22. Goldenstein L, Hake PW, Devanny JR, Chan DPK (1976) Guidelines for the management of lumbosacral spondylolisthesis associated with scoliosis. *Clin Orthop Relat Res* 117:135–148
23. Risser JC, Norquist DM (1961) Sciatic scoliosis in growing children. *Clin Orthop* 21:137
24. Rick H, Winter RB, Moe JH (1965) The lumbosacral articulation and its relationship to scoliosis. *JBJS Br* 31:45–64
25. Glorieux P, Roederer C (1937) *La Spondylolyse et ses conséquences*. Masson, Paris
26. Diessl F (1929) *Zeit Orthop* 265
27. Wiltse LL, Newman PH, Macnab I (1976) Classification of spondylolysis and spondylolisthesis. *Clin Orthop Relat Res* 117: 23–29
28. Marchetti PC, Bartolozzi P (1997) Classification of spondylolisthesis as a guideline for treatment. In: Bridwell KH, De Wald RL, Hammerberg KW (eds) *The textbook of spinal surgery*, vol 2. Lippincott-Raven, Philadelphia, pp 1211–1254
29. Hermann MG, Pizzutillo PD (2005) Spondylolysis and spondylolisthesis in the child and adolescent: a new classification. *Clin Orthop Relat Res* 434:46–54
30. Luk KDK, Hoh C, Leong JCY (1986) The iliolumbar ligament. A study of its anatomy, development and clinical significance. *JBJS Br* 68B(2):197–200
31. Roussouly P, Goolloyly S, Berthonnaud E et al (2006) Sagittal alignment of the spine and pelvis in presence of L5–S1 isthmic lysis on low grade spondylolisthesis. *Spine* 31(20):84–90
32. Barrack RL, Whitecloud T, Skinner HB (1984) Spondylolysis after spinal instrumentation in Osteogenesis Imperfecta. *South Med J* 77(11):1453–1454
33. Harris RI, Wiley JJ (1963) Acquired spondylolisthesis as a sequel to spine fusion. *J Bone Joint Surg* 45A:1159
34. Niezgoda A, Smoczynski A, Diebski J (1978) Spondylolysis of the lumbar spine after surgical treatment of lateral idiopathic scoliosis by the Harrington method. *Chir Narzadow Ruchu Orthop Pol* 43:379
35. Danielsson A, Nachemson A (2001) Radiologic findings and curve progression 22 years after treatment for adolescent idiopathic scoliosis: comparison of brace and surgical treatment with matching control group of straight individuals. *Spine* 26:516–525
36. Winter RB, Silverman BJ (2003) Degenerative spondylolisthesis at the L4–L5 in a 32-year-old female with previous fusion for idiopathic scoliosis: a case report. *J Orthop Surg* 11(2):202–206
37. Toyone T, Tanaka T, Kato D, Kaneyama R, Otsuka M (2005) Anatomic changes in lateral spondylolisthesis associated with adult lumbar scoliosis. *Spine* 30(22):E671–E675
38. Freedman BA, Horton WC, Rhee JM, Edwards CC, Kuklo TR (2009) Reliability analysis for manual radiographic measures of rotator subluxation or lateral listhesis in adult scoliosis. *Spine* 34(6):603–608
39. Koptan WMT, El Miligui IH, El Sharkawi MM (2011) Direct repair of spondylolysis presenting after correction of adolescent idiopathic scoliosis. *Spine J* 11:133–138