



Characteristics and treatment of dynamic sagittal imbalance in adult spinal deformity

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Received: 12 November 2019 / Accepted: 10 May 2020 / Published online: 2 June 2020
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Abstract

Objective To raise the diagnostic criteria, classification and treatment strategy of dynamic sagittal imbalance (DSI).

Methods One hundred thirty-three adult spinal deformity (ASD) patients with stooping and back pain after walking were retrospectively analyzed. Based on the radiographic parameters and Oswestry Disability Index (ODI) scores, the diagnostic criteria of DSI were raised. DSI patients received nonoperative treatment and (or) surgery. Radiographic parameters and health-related quality of life (HRQOL) outcomes would be measured and compared between prewalk and postwalk and among each subgroup.

Results One hundred thirty-three ASD patients with stooping and back pain after walking were enrolled in our study. The quantitative diagnostic criteria was prewalk SVA < 40 mm and postwalk SVA-prewalk SVA ≥ 20 mm after 10-min walk. Based on the quantitative diagnostic criteria of DSI raised by our team, DSI patients could be classified into three groups: 20 mm ≤ ΔSVA < 60 mm (mild, 31.0%), 60 mm ≤ ΔSVA < 100 mm (moderate, 42.1%) and ΔSVA ≥ 100 mm (severe, 27.0%). After nonoperative treatment, the ΔSVA in mild and moderate group was prominently decreased ($P < 0.001$) with the significant improvement of HRQOL outcomes ($P < 0.001$), while there was no significant change in ΔSVA and clinical outcomes in group C ($P > 0.05$). Patients who received the operative treatment showed prominent improvement in ΔSVA and clinical outcomes ($P < 0.001$).

Conclusion Our study proposed a quantitative diagnostic criteria and novel classification of DSI. Nonoperative treatment is effective for most DSI patients with ΔSVA < 100 mm, while the majority of DSI patients with ΔSVA ≥ 100 mm need operative intervention.

Keywords Adult spinal deformity · Dynamic sagittal imbalance · Diagnostic criteria · Classification · Nonoperative or operative treatment

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Introduction

Adult spinal deformity (ASD) is an increasingly prevalent and costly problem which affects 32 to 68% of population older than 65 years all over the world [1, 2]. ASD could lead to severe back pain with neurogenic claudication, radiculopathy and walking intolerance, as well as psychological burden [3]. For ASD patients demanding operative intervention, how to correct coronal imbalance used to be a key problem surgeons dedicated to study [4]. In the last decade, more and more surgeons focused on the solution of sagittal imbalance [5–7]. Degenerative flat back is a subtype of ASD which is more common in Asian countries, especially China, Japan and Korea [2]. Those patients, mostly in older women, usually have difficulty in walking with stooping trunk. The precise pathophysiology of degenerative flat back is unclear,

but extensive degeneration and weakness of back extensor muscles are considered to be the most important influence factors [8, 9].

Quite a lot of ASD patients with flat back show a normal SVA (< 40 mm), which could be easily thought as sagittal balance [10]. In fact, a series of compensatory mechanisms, such as contraction of lumbar extensor muscles and pelvic retroversion have been recruited to prevent the tendency toward forward imbalance [11]. Once the compensatory mechanisms exhaust, even after a short walk, the standing SVA would significantly worsen, suggesting its dynamic nature. It is generally called dynamic sagittal imbalance (DSI) [12] (Fig. 1). However, at present, no one had raised a definite evaluation criterion of DSI which could better guide the treatment of this special disease. Routine static radiographs often fail to unmask the potential DSI. A previous study reported a novel and simple approach on which patients were asked to take a full-spine radiograph at initiation and take the second one after 10-min walk [13]. It would be more convenient for clinicians to detect the masked DSI in our outpatients.

In general, nonoperative management including physical therapy, steroid injection and medications is regarded as the first-line treatment for ASD patients, despite the absence

of strong supporting evidence [2, 14]. Operative treatment may be considered if patients are unsatisfied with the effect of nonoperative treatment or even neurological symptoms come out. Compared with nonoperative treatment, surgical intervention can provide significant improvement of spinal alignment and clinical outcomes [15, 16]. However, for DSI, little is known about the effect of conservative or operative treatment. The purpose of this study is to analyze the characteristics of DSI and clarify the effect of nonoperative and operative treatment for DSI.

Materials and methods

Study design

Starting in 2015, a selected group of 133 ASD patients with the complaint of stooping and back pain after walking were assessed in our center. All patients were enrolled through institutional review board-approved protocol. Enrollment criteria include: a coronal deformity (Cobb angle $< 30^\circ$) and loss of lumbar lordosis (“flat back,” PI-LL $> 10^\circ$). Those patients were asked to take two sets of anterior–posterior and lateral full-spine radiographs at initiation and after a 10-min walk to evaluate how sagittal alignment changed after walking. According to a previous finding, a 10-min walk was thought to be sufficient and could be tolerated by most patients in the outpatient [13]. Patients were excluded if ASD was due to one of the following causes: spondylolysis/spondylolisthesis, congenital or neuromuscular scoliosis, hip pathology, compression fracture, symptomatic lumbar stenosis, Scheuermann kyphosis and Cobb angle $> 30^\circ$.

Slobodyanyuk et al. [17] put forward the equation to evaluate the minimum clinically important difference (MCID) deviation of each patient from normative values. In our study, we chose ODI for further study. So, the equation was described as such: $\text{Diff}_{\text{domain}} = (\text{ODI}_{\text{patient}} - \text{ODI}_{\text{normative}}) / \text{MCID}_{\text{ODI}}$. The MCID_{ODI} and $\text{ODI}_{\text{normative}}$ used in our study were -8 points and 9.05 points [18, 19]. According to previous studies by Moal et al. [20], patients were categorized into four clinical severity groups for the $\text{Diff}_{\text{domain}}$ scores as follows: (1) worst, $> 4\text{MCIDs}$ below normative; (2) severe, $2\text{--}4\text{ MCIDs}$ below normative; (3) poor, $1\text{--}2\text{ MCIDs}$ below normative and (4) asymptomatic, $< 1\text{MCID}$ below normative or above normative. Based on the radiographic parameters and ODI scores, our team raised the diagnostic criteria of DSI.

Then, 126 DSI patients were firstly recommended to receive standard nonoperative treatment. In our study, nonoperative treatment mainly included exercises (hip bridge and hyperextension, four times a week for 40 min, 3 months), celecoxib (200 mg, twice daily, 3 weeks) and physical therapy (three times a week, 3 months).

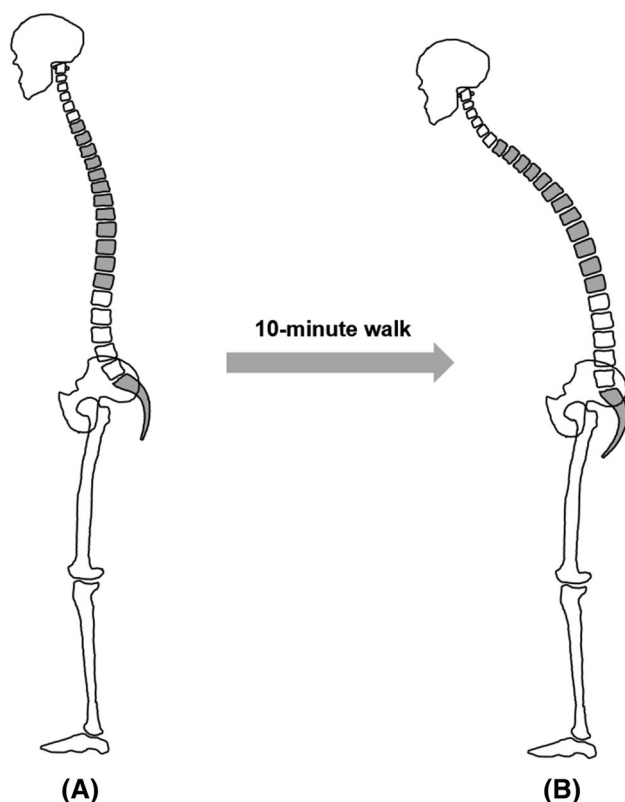


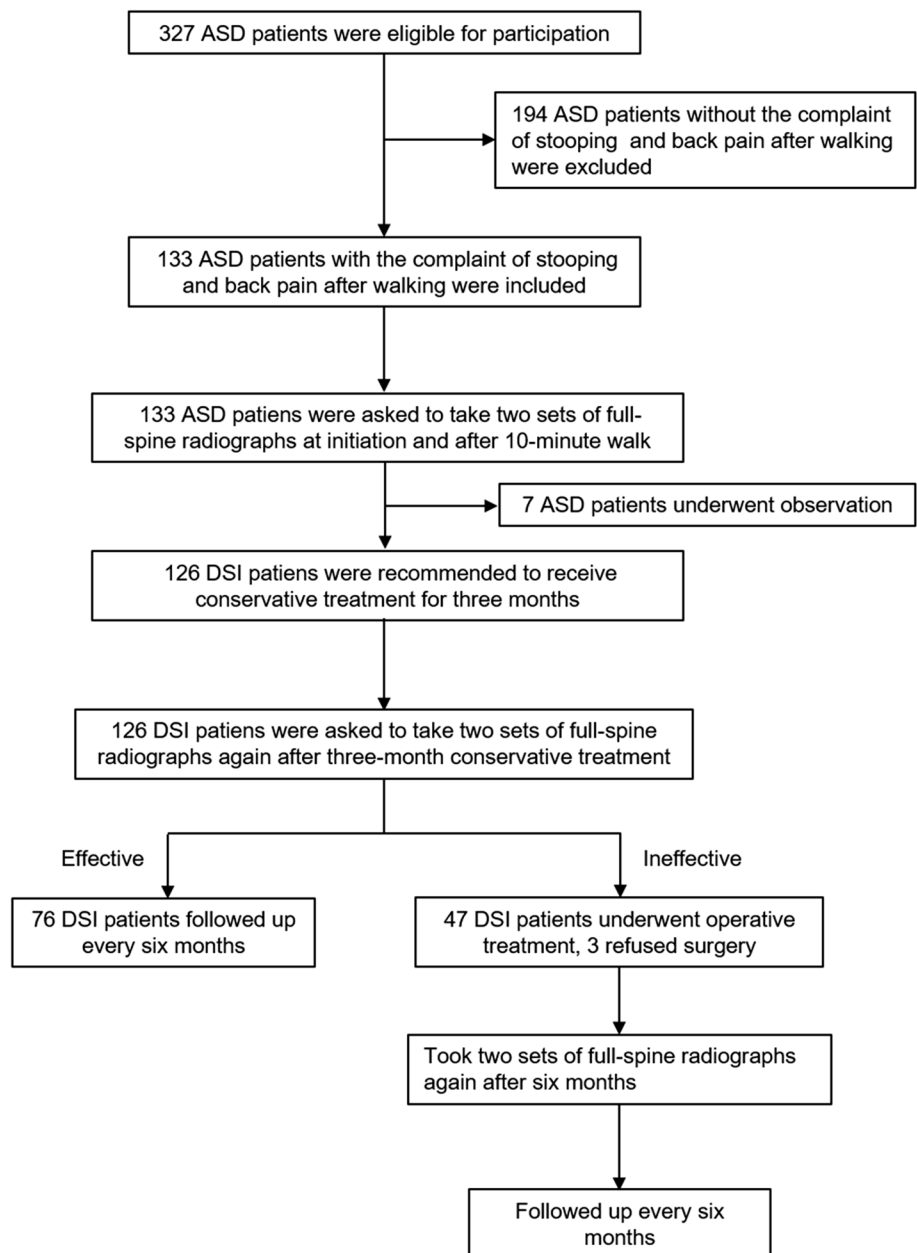
Fig. 1 **a** At initiation, patients with DSI generally show normal sagittal balance. **b** After 10-min walk, patients with DSI would show severe sagittal imbalance

Operative treatment was only performed on patients who were ineffective in strict conservative treatment for more than 3 months. All surgeries were performed by a same team. The outcomes (effective or ineffective) were evaluated based on the HRQOL measures [mainly Oswestry Disability Index (ODI) for convenience in our study] of the patients. Effective outcome was termed as at least 8 points of improvement in ODI after nonoperative or operative treatment, and ineffective outcome as smaller than 8 points improvement or deterioration in ODI [18, 21] (Fig. 2).

Data collection

All analyses were based on standing anterior–posterior and lateral 36-inch radiographs of the spine that extended from the proximal femur to the base of the skull. All patients were asked to look forward to maintain a horizontal gaze and with their knees extended, arms flexed and hands placed on their clavicles before taking radiographs. Once the first radiograph was completed, the patients were immediately asked to walk at their usual speed for 10 min without resting neither on a chair nor the walls. Our research assistant would supervise each patient for the entirety of the 10-min walk. Immediately after 10-min walk, the repeated radiograph

Fig. 2 Flow of participants from screening and enrollment through 18-month follow-up. Follow-up time points indicate the time since first treatment occurred



would be taken. Then those patients would return to consulting room. Nonoperative treatment would be firstly recommended. After three-month nonoperative treatment, patients would receive evaluation at initiation and after 10-min walk again. Those who were ineffective with nonoperative treatment would receive operative intervention and be assessed with the same protocol further.

Radiographic parameters included: thoracic kyphosis (TK), thoracic lumbar kyphosis (TLK), lumbar lordosis (LL), sagittal vertical axis (SVA), pelvic tilt (PT), sacral slope (SS), pelvic incidence (PI), mismatch between PI and LL (PI-LL) (Fig. 1). All radiographs were digitized, and two independent observers measured all radiographic parameters. The following health-related quality of life (HRQOL) questionnaires, completed at initiation and after 10-min walk, were assessed for patients in this study: Oswestry Disability Index (ODI), the Scoliosis Research Society instrument (SRS-22) and Numeric Rating Scales (NRS, 0–10) for back and leg pain (Fig. 3).

Statistical analysis

Statistical analyses were performed using SPSS 17.0 statistics software (SPSS Inc, Chicago, IL). Descriptive statistics were listed in the form of mean and standard deviation. For categorical variables (e.g., sex), cross-tabulations were generated, and the Fisher exact or Pearson χ^2 tests were used to compare distribution. Differences of other demographic variables [age, body mass index (BMI) and bone mineral intensity (BMD)] were assessed by using analysis of variance. Unpaired *t* tests were used to evaluate differences of changes in parameters (radiographic evaluation and HRQOL scores) at different time points between subgroups. Differences of each subgroup between prewalk and postwalk were compared by using a paired *t* test. Significance for all statistical tests was set at $P < 0.05$.

Results

Definition of threshold values of DSI

One hundred thirty-three ASD patients with the complaint of stooping and back pain after walking were classified into seven subgroups according to evenly spaced Δ SVA (20 mm): 0–20 mm, 20–40 mm, 40–60 mm, 60–80 mm, 80–100 mm, 100–120 mm and ≥ 120 mm. The proportions of each subgroup were separately 5.3% (7/133), 12.0% (16/133), 17.3% (23/133), 21.1% (28/133), 18.8% (25/133), 13.5% (18/133) and 12.0% (16/133). Moreover, with the growth of Δ SVA, the mean postwalk ODI

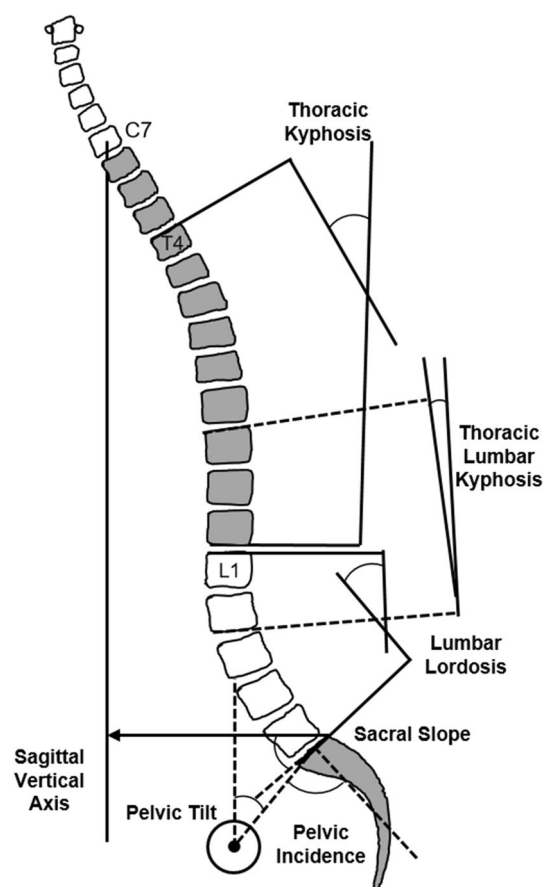


Fig. 3 Sagittal spino-pelvic radiographic parameters included TK, TLK, LL, PI, PT, SS and SVA. *TK* thoracic kyphosis, *TLK* thoracic lumbar kyphosis, *LL* lumbar lordosis, *PI* pelvic incidence, *PT* pelvic tilt, *SS* sacral slope, *SVA* sagittal vertical axis

of subgroups increased from 16.8 ± 10.3 to 54.6 ± 15.7 points. The $\text{Diff}_{\text{domain}}$ scores reflected the minimum clinically important difference (MCID) deviation of each patient from normative values. The $\text{Diff}_{\text{domain}}$ scores of each subgroup were 0.969, 1.331, 1.931, 2.844, 3.906, 4.819 and 5.694 (Table 1). The $\text{Diff}_{\text{domain}}$ scores of the first subgroup (0–20 mm) was 0.969 MCID which was classified into asymptomatic group (< 1 MCID) indicating no significant clinical difference. Whereas the $\text{Diff}_{\text{domain}}$ scores of other six subgroups were all larger than 1 MCID. So the proximate threshold value of Δ SVA for diagnosing DSI was ≥ 20 mm. The $\text{Diff}_{\text{domain}}$ scores of the second and third subgroups were 1.331 MCIDs and 1.931 MCIDs which were categorized into poor group (1–2 MCIDs). Therefore, correspondingly, the proximate threshold values of Δ SVA were 20–60 mm, which was defined as group A (mild) (Fig. 4). Similarly, the proximate threshold values of Δ SVA for group B (moderate) (Fig. 5) and group C (severe) (Fig. 6) were 60–100 mm and ≥ 100 mm (Table 2).

Table 1 Distribution, postwalk ODI and diffdomain of each Δ SVA subgroup among 133 ASD patients

	Δ SVA subgroup						
	0–20 mm	20–40 mm	40–60 mm	60–80 mm	80–100 mm	100–120 mm	≥ 120 mm
Cases	7	16	23	28	25	18	16
Proportion	5.3%	12.0%	17.3%	21.1%	18.8%	13.5%	12.0%
ODI _{postwalk}	16.8 ± 10.3	19.7 ± 13.2	24.5 ± 16.4	31.8 ± 21.6	40.3 ± 19.5	47.6 ± 18.1	54.6 ± 15.7
Diff _{domain}	0.969	1.331	1.931	2.844	3.906	4.819	5.694

SVA sagittal vertical axis, Δ SVA SVA after 10-min walk minus SVA at initiation. MCID indicates minimal clinical difference

Diff_{domain} indicates MCID deviation of each patient from normative values. $= (\text{ODI}_{\text{patient domain}} - \text{ODI}_{\text{normative domain}}) / \text{MCID}_{\text{ODI}}$

The normative ODI score used in our study was 9.05 points obtained from Tonosu et al.

The MCID value of ODI used in our study was -8 points

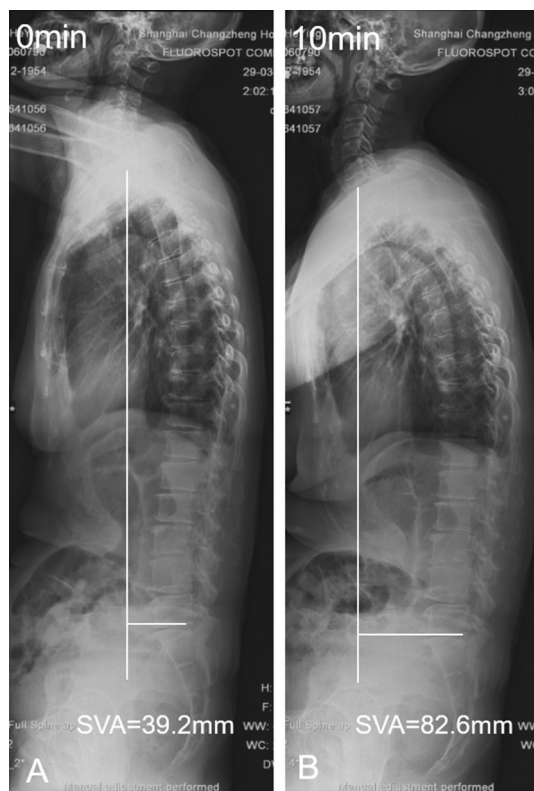


Fig. 4 Representative radiographs of a patient in Group A (mild, $0 \text{ mm} < \Delta\text{SVA} < 60 \text{ mm}$). Note that initially sagittal balanced spine (a) became imbalanced (b) after 10-min walk. Δ SVA of the patient was 43.4 mm. SVA indicates sagittal vertical axis

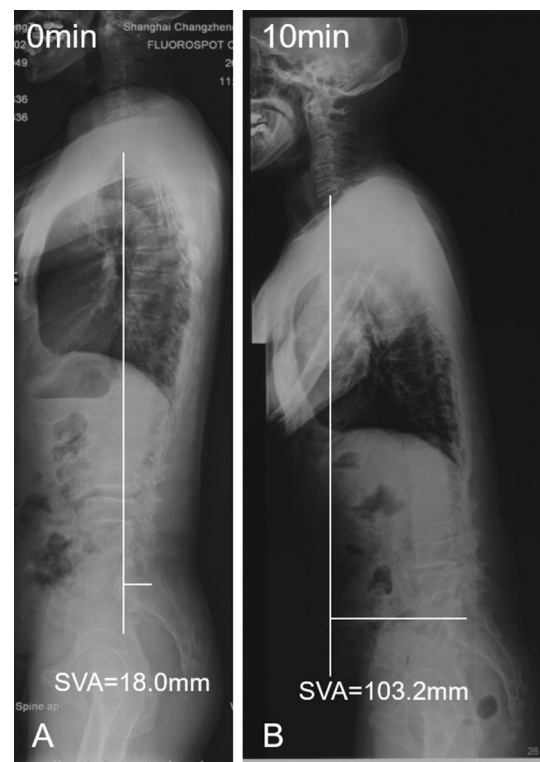


Fig. 5 Representative radiographs of a patient in Group B (moderate, $60 \text{ mm} \leq \Delta\text{SVA} < 100 \text{ mm}$). Note that initially sagittal balanced spine (a) became imbalanced (b) after 10-min walk. Δ SVA of the patient was 85.2 mm. SVA indicates sagittal vertical axis

Demographic data of DSI patients

A total of 126 patients with DSI were included in this study (Male: 14 and Female: 112). The mean age of patients was 61.0 ± 7.4 years old. Mean BMI was 25.0 ± 3.9 , and mean BMD was -1.6 ± 1.9 . All three subgroups would be

classified as osteopenia. There were 39 patients (31.0%, 39/126) in group A (mild), 53 patients (42.1%, 53/126) in group B (moderate) and 34 patients (27.0%, 34/126) in group C (severe). There was no significant difference among the three subgroups on demographic data ($P > 0.05$). The demographic characteristics of patients were summarized in Table 3.

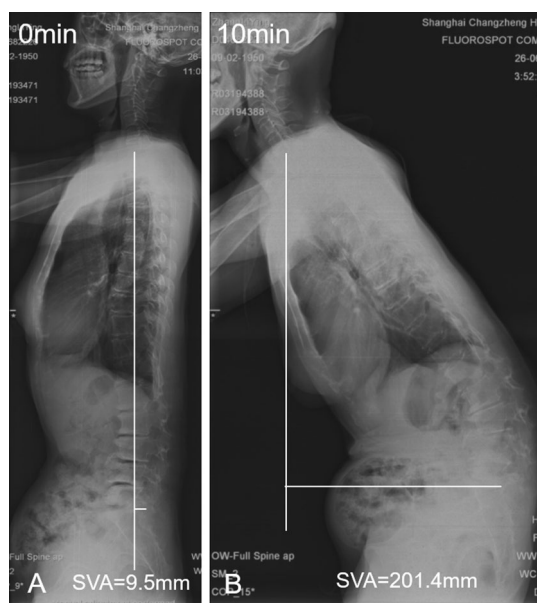


Fig. 6 Representative radiographs of a patient in Group C (severe, $\Delta\text{SVA} \geq 100$ mm). Note that initially sagittal balanced spine (a) became imbalanced (b) after 10-min walk. ΔSVA of the patient was 191.9 mm. SVA indicates sagittal vertical axis

Changes in radiographic and clinical outcomes parameters in DSI

Prewalk and postwalk radiographic and HRQOL outcomes of each subgroup were presented in Table 4. For group A, average prewalk SVA was 24.9 ± 15.3 mm and prewalk PI-LL was $16.9 \pm 12.5^\circ$. Average prewalk SVA was 30.5 ± 19.4 mm and prewalk PI-LL was $20.9 \pm 13.7^\circ$ in group B. For group C, average prewalk SVA was 32.2 ± 21.6 mm and prewalk PI-LL was $19.3 \pm 14.7^\circ$.

Average prewalk SVA of each subgroup was smaller than 40 mm that would be usually thought as a symbol of sagittal balance in ASD. Each subgroup had average prewalk $\text{PT} < 30^\circ$. There was no significant difference among subgroups on prewalk TK, TLK and LL. In addition, no significant difference was observed on ODI, SRS-total and NRS at initiation. After 10-min walk, significant deteriorations gradually emerged in each subgroup ($P < 0.001$). In group A, average SVA increased to 55.9 ± 32.6 mm (prewalk vs postwalk, $P < 0.001$), which was accompanied by significant decreases in average LL (8.4° , $P < 0.001$), while increases in average TLK (2.6° , $P < 0.001$) and PI-LL (8.3° , $P < 0.001$). For group B, average postwalk SVA was 109.2 ± 65.7 mm (prewalk vs postwalk, $P < 0.001$), accompanied by significant decreases in average LL (20.0° , $P < 0.001$), while increases in average TK (2.3° , $P < 0.001$), TLK (3.4° , $P < 0.001$) and PI-LL (19.8° , $P < 0.001$). Average postwalk SVA was 184.5 ± 103.5 mm in group C (prewalk vs postwalk, $P < 0.001$). This was accompanied by significant decreases in average LL (38.9° , $P < 0.001$). Also, significant increases were observed in average TK (3.2° , $P < 0.05$), TLK (13.0° , $P < 0.001$) and PI-LL (39.4° , $P < 0.001$). Postwalk average PT in groups B and C was larger than that before 10-min walk ($P < 0.05$, $P < 0.05$). Postwalk average ODI and NRS in subgroups were both larger than that at initiation ($P < 0.001$, $P < 0.001$), while postwalk SRS-total was smaller ($P < 0.05$). Group A had the smallest SVA change (ΔSVA), whereas group C had the largest ΔSVA , accompanied by the same distribution of ΔTK change (ΔTK) and TLK change (ΔTLK), especially LL Change (ΔLL). Furthermore, average ODI change (ΔODI) and NRS change (ΔNRS) increased with the growth of ΔSVA and SRS-total change ($\Delta\text{SRS-total}$) decreased with the growth of ΔSVA .

Table 2 Determination of threshold values of DSI

	ΔSVA subgroup			
	0–20 mm	20–60 mm (group A, mild)	60–100 mm (group B, moderate)	≥ 100 mm (group C, severe)
$\text{Diff}_{\text{domain}}$ (MCID)	0.969	1.331–1.931	2.844–3.906	≥ 4.819
Criteria (MCID)	< 1	1–2	2–4	> 4

MCID minimal clinical difference, $\text{Diff}_{\text{domain}}$ MCID deviation of each patient from normative values

Table 3 Comparison of demographic data in three groups of DSI with different SVA change

	$20 \leq \Delta\text{SVA} < 60$ mm (A, $n = 39$)	$60 \text{ mm} \leq \Delta\text{SVA} < 100$ mm (B, $n = 53$)	$\Delta\text{SVA} \geq 100$ mm (C, $n = 34$)	P value
Age (y)	60.1 ± 6.1	61.2 ± 6.9	61.8 ± 7.2	0.365
Sex (Man/Women)	3/36	6/47	4/30	0.809
BMI (kg/m^2)	25.2 ± 7.3	24.2 ± 6.8	26.0 ± 7.5	0.461
BMD	-1.3 ± 1.1	-1.7 ± 1.6	-1.9 ± 1.6	0.105

BMI body mass index, BMD bone mineral density

Table 4 Comparison of radiographic and clinical outcome parameters from each subgroup at initial presentation and after 10-min walk

	SVA change			P value		
	20 ≤ Δ SVA < 60 mm (A, n = 39)	60 mm ≤ Δ SVA < 100 mm (B, n = 53)	Δ SVA ≥ 100 mm (C, n = 34)	A versus B	A versus C	B versus C
TK (°)						
Prewalk	22.6 ± 4.7	21.5 ± 5.0	20.0 ± 7.9	0.288	0.099	0.328
Postwalk	23.4 ± 6.9	23.8 ± 5.2	23.2 ± 6.4	0.752	0.899	0.633
Change	0.8 ± 4.8	2.3 ± 4.1	3.2 ± 6.3	0.110	0.069	0.463
P value	0.235	< 0.001*	0.043*			
TLK (°)						
Prewalk	0.2 ± 4.6	0.8 ± 5.4	2.3 ± 5.8	0.577	0.089	0.223
Postwalk	2.8 ± 5.0	4.2 ± 6.2	15.3 ± 9.5	0.249	< 0.001*	< 0.001*
Change	2.6 ± 3.7	3.4 ± 4.8	13.0 ± 8.7	0.388	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	< 0.001*			
LL (°)						
Prewalk	− 31.5 ± 13.7	− 29.6 ± 14.5	− 27.1 ± 13.5	0.527	0.172	0.423
Postwalk	− 23.1 ± 15.0	− 9.6 ± 12.0	11.8 ± 16.3	< 0.001*	< 0.001*	< 0.001*
Change	8.4 ± 9.7	20.0 ± 16.8	38.9 ± 24.3	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	< 0.001*			
PT (°)						
Prewalk	17.8 ± 7.8	20.4 ± 11.0	22.5 ± 12.7	0.188	0.067	0.416
Postwalk	20.2 ± 10.7	24.0 ± 9.9	28.9 ± 9.2	0.082	< 0.001*	0.023
Change	2.4 ± 5.8	3.6 ± 6.0	6.4 ± 8.2	0.339	0.021	0.091
P value	0.117	0.031*	0.047*			
SS (°)						
Prewalk	30.6 ± 8.5	30.1 ± 11.7	23.5 ± 10.2	0.813	0.002*	0.008*
Postwalk	28.1 ± 9.5	26.3 ± 9.4	17.6 ± 10.8	0.369	< 0.001*	< 0.001*
Change	− 2.5 ± 5.0	− 3.8 ± 6.5	− 5.9 ± 7.5	0.300	0.029	0.170
P value	0.205	0.039*	0.029*			
PI-LL (°)						
Prewalk	16.9 ± 12.5	20.9 ± 13.7	18.9 ± 14.7	0.155	0.453	0.607
Postwalk	25.2 ± 13.1	40.7 ± 13.2	58.3 ± 15.2	< 0.001*	< 0.001*	< 0.001*
Change	8.3 ± 8.5	19.8 ± 15.6	39.4 ± 21.5	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	< 0.001*			
SVA (mm)						
Prewalk	24.9 ± 15.3	30.5 ± 19.4	32.2 ± 21.6	0.139	0.106	0.704
Postwalk	55.9 ± 32.6	109.2 ± 65.7	184.5 ± 103.5	< 0.001*	< 0.001*	0.001*
Change	31.0 ± 18.4	78.7 ± 20.5	152.3 ± 45.2	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	< 0.001*			
ODI						
Prewalk	10.3 ± 10.4	11.9 ± 11.0	14.0 ± 9.7	0.482	0.122	0.366
Postwalk	21.9 ± 10.8	35.0 ± 18.5	50.9 ± 20.7	< 0.001*	< 0.001*	< 0.001*
Change	11.6 ± 5.5	23.1 ± 10.7	36.9 ± 13.0	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	< 0.001*			
SRS-22 total						
Prewalk	4.1 ± 0.6	3.9 ± 0.7	3.9 ± 0.6	0.154	0.160	1.000
Postwalk	3.9 ± 0.8	3.3 ± 0.9	2.8 ± 0.9	0.001*	< 0.001*	0.013*
Change	− 0.2 ± 0.2	− 0.6 ± 0.4	− 1.1 ± 0.7	< 0.001*	< 0.001*	< 0.001*
P value	0.031*	0.009*	< 0.001*			
NRS						
Prewalk	1.3 ± 1.2	1.6 ± 1.6	1.7 ± 1.5	0.328	0.210	0.771
Postwalk	2.7 ± 1.3	5.0 ± 1.7	8.0 ± 1.8	< 0.001*	< 0.001*	< 0.001*
Change	1.4 ± 1.2	3.4 ± 1.9	6.3 ± 2.3	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	< 0.001*			

Table 4 (continued)

TK thoracic kyphosis, *TLK* thoracic lumbar kyphosis, *LL* lumbar lordosis, *PT* pelvic tilt, *SS* sacral slope, *PI-LL* mismatch between pelvic incidence and lumbar lordosis, *SVA* sagittal vertical axis, *ODI* Oswestry Disability Index, *SRS-22 total* total score of Scoliosis Research Society instrument (SRS-22), *NRS* Numeric Rating Scales

*Indicates significant

Table 5 Comparison of changes in SVA and clinical outcomes parameters from each subgroup after 3-month nonoperative treatment

	SVA change			P value		
	20 ≤ ΔSVA < 60 mm (A, n = 39)	60 mm ≤ ΔSVA < 100 mm (B, n = 53)	ΔSVA ≥ 100 mm (C, n = 34)	A versus B	A versus C	B versus C
SVA change (mm)						
Pre-N	31.0 ± 18.4	78.7 ± 20.5	152.3 ± 45.2	< 0.001*	< 0.001*	< 0.001*
Post-N	16.3 ± 16.5	50.6 ± 28.7	145.1 ± 40.7	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	0.087			
ODI change						
Pre-N	11.6 ± 5.5	23.1 ± 10.7	36.9 ± 14.0	< 0.001*	< 0.001*	< 0.001*
Post-N	3.1 ± 4.6	15.0 ± 7.8	33.2 ± 10.6	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	0.003*	0.102			
SRS-22 total change						
Pre-N	− 0.3 ± 0.2	− 0.6 ± 0.4	− 1.1 ± 0.7	< 0.001*	< 0.001*	< 0.001*
Post-N	− 0.1 ± 0.2	− 0.3 ± 0.3	− 1.0 ± 0.5	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	0.184			
NRS change						
Pre-N	1.4 ± 1.2	3.4 ± 1.9	6.3 ± 2.3	< 0.001*	< 0.001*	< 0.001*
Post-N	0.6 ± 1.1	1.8 ± 1.5	5.5 ± 1.8	< 0.001*	< 0.001*	< 0.001*
P value	< 0.001*	< 0.001*	0.116			

SVA change change of sagittal vertical axis, *ODI change* Oswestry Disability Index, *SRS-22 total change* change in total score of Scoliosis Research Society instrument (SRS-22), *NRS change* change in numeric rating scales. *Pre-N* before nonoperative treatment; *Post-N* 3-month after nonoperative treatment

*Indicates significant

Nonoperative treatment for DSI

One hundred twenty-six DSI patients were recommended conservative treatment firstly. Prenonoperative and postnonoperative treatment radiographic and HRQOL outcomes of each subgroup were presented in Table 5. After nonoperative treatment, for group A, significant decrease was noted in average ΔSVA (14.7 mm, $P < 0.001$), accompanied by significant decrease in average ΔODI (8.5, $P < 0.001$) and ΔNRS (0.8, $P < 0.001$), whereas significant increase in ΔSRS-total (0.2, $P < 0.001$). In addition, similar changes were observed in group B: average ΔSVA (28.1 mm, $P < 0.001$), ΔODI (8.1, $P = 0.003$) and ΔNRS (1.6, $P < 0.001$) significantly decreased and ΔSRS-total increased (0.3, $P < 0.001$). However, for group C, there were no significant difference in ΔSVA, ΔODI, ΔSRS-total or ΔNRS ($P > 0.05$) between prenonoperative and postnonoperative treatment.

According to our evaluation criteria of treatment effect, for group A, nearly all the patients got significant improvement (36/39, 92.3%). 73.6% (39/53) of patients in group B

Table 6 Effect of 3-month nonoperative treatment for each subgroup of DSI

		Effective	Noneffective
SVA change (mm)	20–60 mm	36/39 (92.3%)	3/39 (7.7%)
	60–100 mm	39/53 (73.6%)	14/53 (26.4%)
	≥ 100 mm	1/34 (2.9%)	33/34 (97.1%)

SVA change change of sagittal vertical axis

got improved, while 26.4% reported no improvement. However, for group C, only 2.9% (1/34) of the patients reported improvement, whereas 97.1% (33/34) got no improvement (Table 6).

Operative treatment for DSI

Fifty DSI patients got no significant improvement with conservative treatment. Forty-seven patients (14 from group B, 33 from group C) received operative treatment and three

patients from group A refused operation (Fig. 7). After operation, average Δ TK, Δ TLK, Δ LL, Δ PI-LL and Δ PT all significantly decreased ($P < 0.05$). In addition, after operation, average Δ SVA significantly decreased ($P < 0.001$), accompanied by significant decrease in average Δ ODI ($P < 0.001$) and Δ NRS ($P < 0.001$), whereas significant increase in Δ SRS-total ($P < 0.001$). There was no significant difference in Δ SVA and Δ ODI after operation between group B (14 patients) and group C (33 patients) (Table 7).

Discussion

Bae et al. [13] firstly introduced the method of taking full-spine radiograph again after 10-min walk for assessing how sagittal balance changed with activity. In their study, ASD patients were classified into two groups: “compensated (prewalk SVA < 40 mm)” and “decompensated (prewalk SVA ≥ 40 mm).” Our study focused on the “Compensated” group, which would hold more relevance for surgical decision making. Beside dynamic changes in radiographic parameters, we add HRQOL data in our study to assess clinic outcome objectively. Furthermore, the effect of nonoperative and operative treatment for DSI was explored comprehensively. Our studies suggest that DSI is not rare in old ASD patients, usually osteopenia or osteoporosis, especially in females. According to Δ SVA, DSI patients were stratified into three groups:

$20 \text{ mm} \leq \Delta$ SVA < 60 mm, $60 \text{ mm} \leq \Delta$ SVA < 100 mm and Δ SVA ≥ 100 mm. With the gradual increase in Δ SVA from group A to C, Δ TK, Δ TLK, Δ LL and Δ PI-LL increased consistently, whereas HRQOL scores gradually deteriorated. Bae et al. also showed that Δ SVA correlated with Δ TK, Δ LL, Δ PI-LL and Δ PT [13]. At the same time, lots of studies indicated change in PT, SVA and PI-LL could significantly affect HRQOL scores [22–24]. Our results are similar to those results mentioned. DSI patients are characterized by normal SVA (SVA < 40 mm) at initiation, and prominent increase in SVA (SVA ≥ 40 mm) after activity with their trunks markedly inclined. Only taking full-spine radiograph again after 10-min walk could sagittal balance be masked, and then, doctors could give the most suitable suggestions.

Dubousset firstly raised the theory of “cone of economy.” It described a range in which one’s body can keep postural balance without significant energy expenditure. Once outside of this “zone,” a series of compensatory mechanisms are recruited for keeping upright [24]. Barrey et al. [25] concluded: Spinopelvic-related compensatory mechanisms included cervical hyperlordosis, reduction in TK, retrolisthesis or hyperextension in lumbar spine, pelvic retroversion, while lower extremities-related compensatory mechanisms included flexion of knees and extension of the hips and ankles. When those compensatory mechanisms effectively work, sagittal balance would be maintained persistently. However, for quite a few of ASD patients, once any

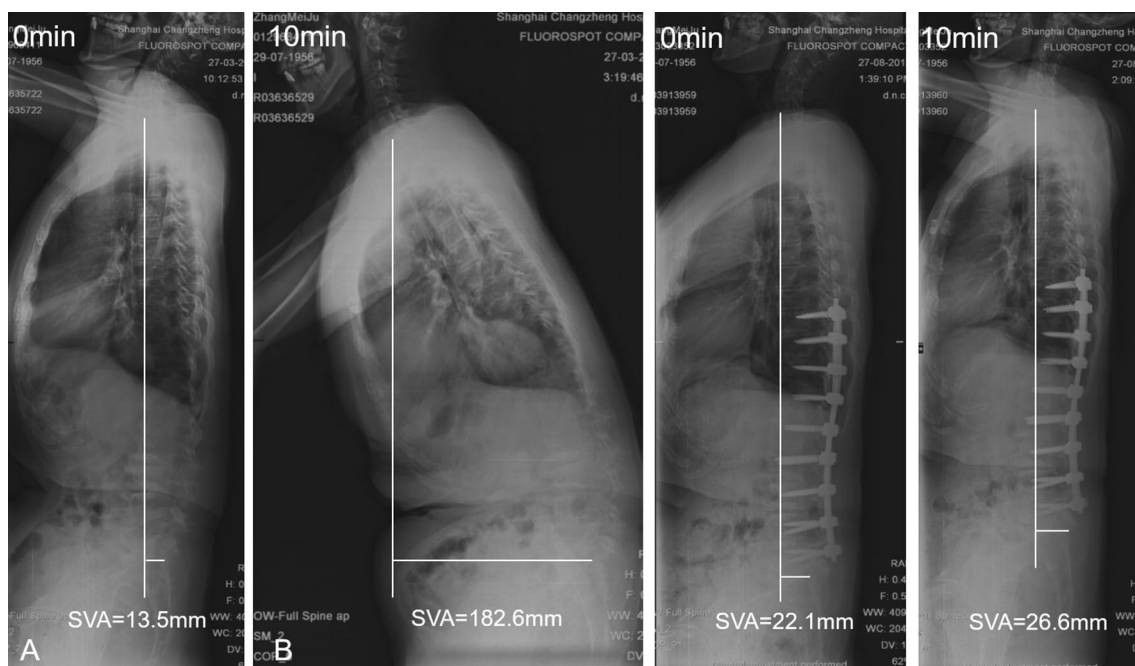


Fig. 7 Representative radiographs of a patient in Group C (severe, Δ SVA ≥ 100 mm). Note that initially sagittal balanced spine (a) became imbalanced (b) after 10-min walk. Δ SVA of the patient was 169.1 mm. Then the patient received a posterior spinal fixation from

T9 to L5 combined with posterior L4/5 interbody fusion. At 1-year follow-up, Δ SVA of the patient was 4.5 mm after 10-min walk (c, d). SVA indicates sagittal vertical axis

Table 7 Comparison of changes in sagittal radiographic parameters and clinical outcomes parameters from each subgroup 1 year after operative treatment

	60 mm \leq Δ SVA < 100 mm (B, N = 14)	Δ SVA \geq 100 mm (C, N = 33)	P value
TK change (°)			
Pre-O	2.6 \pm 2.2	3.1 \pm 5.8	0.671
Post-O	1.0 \pm 0.8	1.3 \pm 0.9	0.287
P value	< 0.001*	< 0.001*	
TLK change (°)			
Pre-O	3.9 \pm 2.5	13.1 \pm 8.0	< 0.001*
Post-O	0.4 \pm 0.2	0.5 \pm 0.3	0.260
P value	< 0.001*	< 0.001*	
LL change (°)			
Pre-O	23.4 \pm 15.7	39.2 \pm 22.5	0.021*
Post-O	7.8 \pm 4.3	9.5 \pm 5.1	0.281
P value	< 0.001*	< 0.001*	
PI-LL change (°)			
Pre-O	23.3 \pm 15.4	39.5 \pm 21.9	0.016*
Post-O	7.6 \pm 4.2	9.4 \pm 5.3	0.266
P value	< 0.001*	< 0.001*	
PT change (°)			
Pre-O	4.7 \pm 3.1	6.5 \pm 6.0	0.184
Post-O	2.9 \pm 2.0	3.8 \pm 2.5	0.239
P value	0.023*	0.008*	
SVA change (mm)			
Pre-O	85.2 \pm 13.5	153.7 \pm 43.8	< 0.001*
Post-O	29.5 \pm 16.3	38.3 \pm 24.1	0.219
P value	< 0.001*	< 0.001*	
ODI change			
Pre-O	29.5 \pm 9.2	37.3 \pm 11.4	0.029
Post-O	5.7 \pm 3.8	7.9 \pm 4.6	0.123
P value	< 0.001*	< 0.001*	
SRS-22 total change			
Pre-O	-0.8 \pm 0.3	-1.2 \pm 0.6	0.004*
Post-O	-0.2 \pm 0.1	-0.3 \pm 0.2	0.028*
P value	< 0.001*	< 0.001*	
NRS change			
Pre-O	4.3 \pm 1.5	5.6 \pm 2.0	0.035*
Post-O	1.1 \pm 0.9	1.9 \pm 1.6	0.036*
P value	< 0.001*	< 0.001*	

TK change change in thoracic kyphosis, TLK change change in thoracic lumbar kyphosis, LL change change in lumbar lordosis, PI-LL change change in mismatch between pelvic incidence and lumbar lordosis, PT change change in pelvic tilt, SVA change change in sagittal vertical axis, ODI change change in Oswestry Disability Index, SRS-22 total change change in total score of Scoliosis Research Society instrument (SRS-22); NRS Change, change in Numeric Rating Scales. Pre-O indicates before operative treatment; Post-O indicates 1 year after operative treatment

*Indicates significant

one of compensatory mechanisms fatigues after activity of certain intensity, sagittal malalignment would appear with worsening pain and disability [26]. All those repositioning mechanisms depend on contraction and interaction between quadriceps, hamstrings, hip flexors and paraspinal muscles. A paper by Katsu et al. [8] studied paraspinal muscles of 110 ASD patients with magnetic resonance imaging (MRI). They found the relative muscle cross-sectional areas (rmCSA) of ASD, which were important measure gauges of muscle strength, were significantly smaller than that of control. Moreover, rmCSAs of multifidus and erector muscles in ASD correlated with PT and ODI negatively, which indicated that enough paraspinal muscle strength could prevent the trend of stooping and reduce the demand of other compensatory mechanisms and then improve ODI. Shahidi et al. [27] studied the fat infiltration of paraspinal muscles in adult patients scheduled for lumbar spine surgery. They found there was an increase in fatty infiltration with age in erector and multifidus muscles in both sexes. As we all know, fatty infiltration is one of the most important evaluation criteria of muscle quality. A decrease in the quantity (rmCSA) and alterations in the quality (fat infiltration) can significantly weaken strengths and endurance of paraspinal muscles, which indicate the reduced ability of spinopelvic compensatory mechanisms. In addition, anterior translation of the gravity line due to stooping trunk leads to the fatigue of back extensor muscles, which further accelerates sagittal imbalance [28, 29]. Therefore, at initiation in our study, the performance of spinopelvic related muscles, especially paraspinal muscles, were sufficient to keep sagittal balance. However, on account of the significant reduced endurance and strength of paraspinal muscles, just after 10-min walk, paraspinal muscles quickly fatigue. Then, SVA, TLK and PI-LL significantly increased with the reduction of LL or even lumbar kyphosis.

Previous studies reported that after three to six months of strict nonoperative treatment, a few of ASD patients would get relieved [2]. Slobodyanyuk et al. [17] studied the effectiveness of conservative treatment among 189 ASD patients. They found up to 24% of nonsurgical patients demonstrated marked improvement at 1-year follow-up with nonoperative treatment. The total effective rate of nonoperative treatment was 60.3% (76/126) in our study, significantly larger than that reported by Virginie et al. In our opinion, one reason for that was the difference in evaluation criteria for treatment effect, the other was the heterogeneity between the objects of study and lack of longer follow-up. From the finding by Jun et al. [29], muscle-strengthening exercises could serve to maintain the quality of spinopelvic muscles and thus prevent spinal sagittal imbalance. Therefore, in our study, for the combination of conservative treatment, we put the emphasis on the spinopelvic muscle-strengthening exercise such as hip bridge and hyperextension. Once

the patients had no back or leg pain, they were asked to take muscle-strengthening exercise gradually. Our study suggested that for nearly all the patients ($36/39, 92.3\%$) in mild DSI group ($20 \text{ mm} \leq \Delta\text{SVA} < 60 \text{ mm}$), nonoperative treatment was effective. For moderate DSI group ($60 \text{ mm} \leq \Delta\text{SVA} < 100 \text{ mm}$), most of the patients ($39/53, 73.6\%$) could get relieved after conservative treatment, while 26.4% did not improve. In addition, nonoperative treatment was effective in only a few of patients with severe DSI ($\Delta\text{SVA} \geq 100 \text{ mm}$). Previous studies were unable to determine a causal relationship between sagittal imbalance and the weakening of paraspinal muscles [28–31]. But according to our study, after 3-month nonoperative treatment, focused on spinopelvic muscle-strengthening exercise, quite a lot of DSI patients showed no aggravating sagittal imbalance dynamically. So, we could speculate that the weakening of paraspinal muscles leads to the occurrence of dynamic sagittal imbalance.

Most recent studies usually study dynamic status of ASD patients with three-dimensional motion analysis system (3D-MAS) and reported that corrective surgery could efficiently improve the stooping and HRQOL outcomes in DSI patients ineffective of nonoperative treatment [15, 16, 32]. A study by Bailey et al. [1] found that after realignment surgery, standard radiographic and HRQOL outcomes significantly improved. From biomechanical metrics, with the decrease in peak SVA, biomechanical loads and muscular forces on the lumbar spine significantly reduced, which would greatly cut down the burden of fatigue paravertebral muscles. In our present study, we did not use the gait analysis with 3D-MAS because it was inconvenient for patients to receive gait analysis in the busy outpatients. In addition, with the simple method of taking full-spine photograph, the potential sagittal imbalance of ASD patients could be unmasked as well. Arima et al. [15] also demonstrated that corresponding to how much correction of the sagittal plane deformity was achieved, postoperative posture during gait in ASD patients improved. From our results, the trunk forward tilt was remarkably improved, which was similar to the findings previously mentioned. However, Lee et al. [12] hold the view that corrective surgery was not effective for all DSI patients. They found that surgery only improved the stooping in the patients with posterior pelvic tilt. For the patients with anterior pelvic tilt, postoperative persistent stooping would still exist. But in our study, persistent stooping of DSI patients all remarkably improved after corrective surgery. One of the most important reasons for that was postwalk PT in groups B and C was significantly larger than that before 10-min walk. Left the effect of lower extremities not considered, the muscular factors that affect dynamic sagittal balance mainly included spinal extensor muscles and pelvic extensor muscles. Spinal extensor muscles and pelvic extensor muscles work together to maintain dynamic

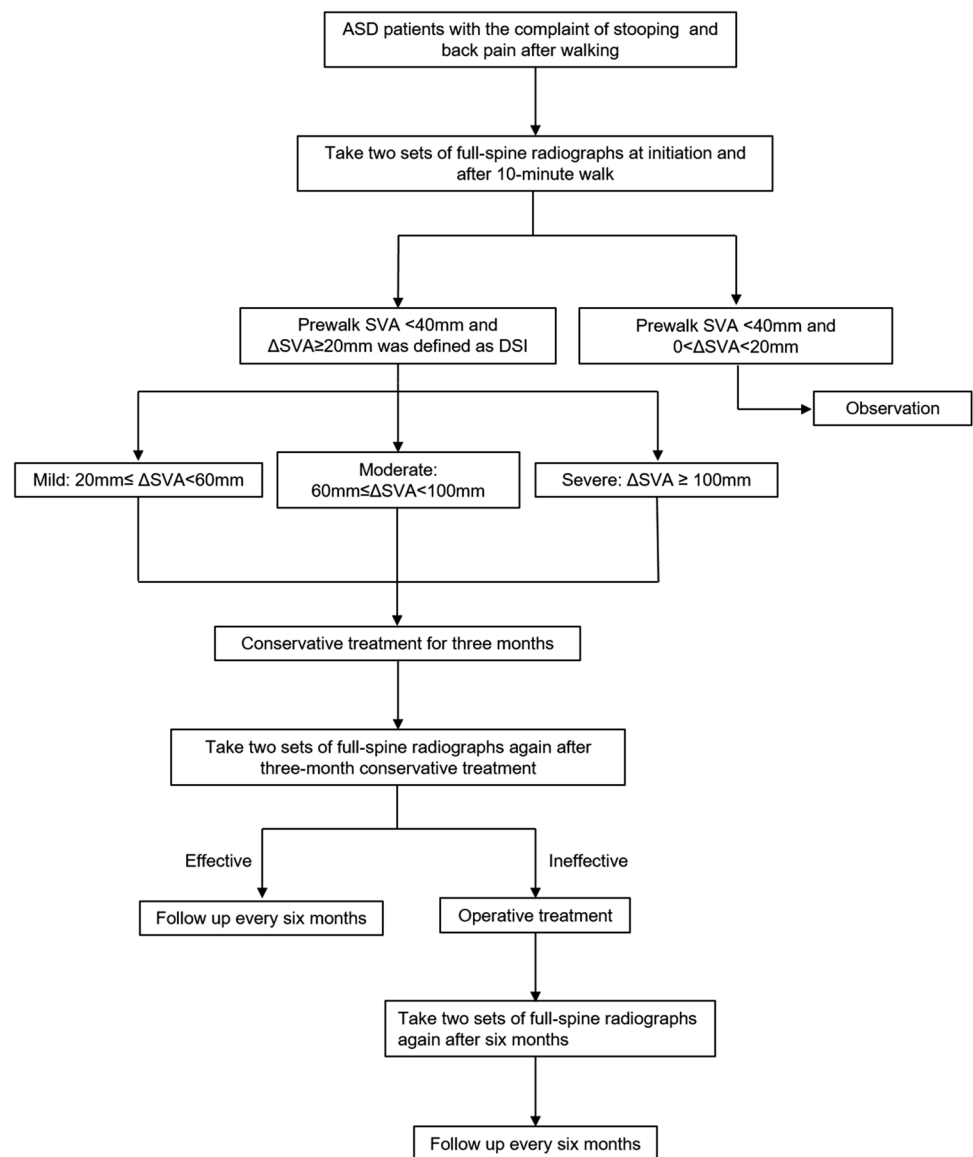
sagittal balance. If the strength of spinal extensor muscles was weakened, then postwalk TK and TLK may increase as well as postwalk LL may decrease, leading to anterior trunk tilt. Similarly, if the strength of pelvic extensor muscles was weakened, postwalk PT could remain unchanged or even decrease, which would also cause anterior trunk tilt. In our study, postwalk PT of patients undergoing surgery was all significantly larger than prewalk PT, indicating that the compensatory capacity of pelvic extensor muscles was relatively strong, so those patients after long segmental fusion and fixation could maintain dynamic sagittal balance well. However, in the study of Lee et al. mentioned above, postwalk PT of some patients was significantly smaller than prewalk PT, indicating that the function of pelvic extensor muscles was decompensated, so even if they received surgical treatment, there would be anterior trunk tilt centered around the hip joint after operation. Hence, it was necessary for surgeons to evaluate prewalk and postwalk PT before decision making.

The present study had some limitations that need further discussion and investigation. First, our sample was relatively small and postoperative follow-up was short. Future work will collect a larger sample to explore the characteristics of DSI patients and the effectiveness of nonoperative and operative treatment at long-term follow-up. Second, it is best to provide an age- and sex-matched control group of patients with normal sagittal alignment to establish baseline values for the change in DSI patients. Third, the relevant inspection items for the diagnosis of sarcopenia had not been carried out in our hospital; hence, we could not measure the amount of sarcopenia in our study, which would lead to mixed results. Fourth, we did not take compensatory mechanisms of lower extremities into account in the present study. Full-body radiographs are needed to evaluate the effect of lower limbs in further plan. Fifth, we just assess the effectiveness of nonoperative and operative treatment with ODI solely. Thus, we will introduce SRS-22 or NRS to evaluate the effectiveness of treatment and determine which is the most suitable.

Conclusions

The diagnostic criteria for DSI were prewalk SVA $< 40 \text{ mm}$ and postwalk SVA-prewalk SVA $\geq 20 \text{ mm}$ after 10-min walk. Based on the quantitative diagnostic criteria of DSI raised by our team, DSI patients could be classified into three groups: $20 \text{ mm} \leq \Delta\text{SVA} < 60 \text{ mm}$ (mild), $60 \text{ mm} \leq \Delta\text{SVA} < 100 \text{ mm}$ (moderate) and $\Delta\text{SVA} \geq 100 \text{ mm}$ (severe). For nearly two-thirds of DSI patients, nonoperative treatment focused on spinopelvic muscle-strengthening exercise is effective while only about one-third need operative intervention. Operative treatment could effectively restore the dynamic sagittal imbalance and improve HRQOLs of patients (Fig. 8).

Fig. 8 Flow of diagnosis and treatment for ASD patients with the complaint of stooping and back pain after walking



Funding This research has not received any funding.

Compliance with ethical standards

Conflict of interest All authors declared that they have no potential conflict of interest.

Ethical approval The research project was approved by the ethics department of Shanghai Changzheng Hospital, Shanghai. We have consensus with all participants. All the procedures were done under the Declaration of Helsinki and relevant policies in China. The Manuscript submitted does not contain information about medical device(s)/drug(s).

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