Alterations of gait kinematics depend on the deformity type in the setting of adult spinal deformity

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Abstract

Purpose To evaluate 3D kinematic alterations during gait in Adult Spinal Deformity (ASD) subjects with different deformity presentations.

Methods One hundred nineteen primary ASD (51 ± 19y, 90F), age and sex-matched to 60 controls, underwent 3D gait analysis with subsequent calculation of 3D lower limb, trunk and segmental spine kinematics as well as the gait deviation index (GDI). ASD were classified into three groups: 51 with sagittal malalignment (ASD-Sag: SVA > 50 mm, PT > 25°, and/or PI-LL > 10°), 28 with only frontal deformity (ASD-Front: Cobb > 20°) and 40 with only hyperkyphosis (ASD-HyperTK: TK > 60°). Kinematics were compared between groups.

Results ASD-Sag had a decreased pelvic mobility compared to controls with a decreased ROM of hips (38 vs. 45°) and knees (51 vs. 61°). Furthermore, ASD-Sag exhibited a decreased walking speed (0.8 vs. 1.2 m/s) and GDI (80 vs. 95, all $p < 0.05$) making them more prone to falls. ASD-HyperTK showed similar patterns but in a less pronounced way. ASD-Front had normal walking patterns. GDI, knee flex/extension and walking speed were significantly associated with SVA and PT ($r = 0.30$–0.65).

Conclusion Sagittal spinal malalignment seems to be the driver of gait alterations in ASD. Patients with higher GT, SVA, PT or PI-LL tended to walk slower, with shorter steps in order to maintain stability with a limited flexibility in the pelvis, hips and knees. These changes were found to a lesser extent in ASD with only hyperkyphosis but not in those with only frontal deformity. 3D gait analysis is an objective tool to evaluate functionality in ASD patients depending on their type of spinal deformity.

Level of evidence I Diagnostic: individual cross-sectional studies with consistently applied reference standard and blinding.

Keywords Adult spinal deformity · 3D gait analysis · Sagittal malalignment · Kinematics · Biomechanics

Introduction

Life expectancy has increased as medical and healthcare developments have progressed. As the population growths older, the number of pathologies multiplies [1]. Most of the time, these diseases are degenerative and occur in conjunction with wear and tear of the tissues, leading to musculoskeletal problems, particularly in the spine [2].

Adult spinal deformity (ASD) consists of a variety of postural and spino-pelvic alterations of the lumbar or thoracolumbar spine, involving one or more of the three planes[3, 4]. It is defined as the presence of pain or loss of function with an increase in one of the following radiographic parameters: Pelvic tilt (PT), sagittal vertical axis (SVA), PI-LL

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mismatch, coronal Cobb angle and thoracic kyphosis (TK) [5, 6]. ASD has a physical and mental impact on the individual. In fact, it can alter daily function and be associated to anxiety and depression in extreme cases [7, 8]. The severity of the deformity is quantified radiologically and can often guide surgical decision [9, 10].

In a standing position, the body maintains the center of gravity above the feet while holding a horizontal gaze [11]. This positioning led to the notion of sagittal alignment, an interaction among different mechanical factors. Consequently, any abnormality of the spine may result to a deviation of the body's center of gravity. In these instances, multiple mechanisms are put in place to counter-balance the changes [12]. In clinical routine, postural assessment is highly valued for better understanding of these mechanisms, using full body frontal and sagittal X-rays in standing position.

Unfortunately, these compensatory mechanisms can lead to serious implications for the patient, such as back pain, muscle fatigue, accelerated joints degeneration, and above all, restriction of many daily activities like walking. Clinicians rely mainly on health-related quality of life (HRQoL) and disability questionnaires for the assessment of deformity repercussions on functionality. These questionnaires emphasize the level of ability to perform some activities of daily living, such as walking, in preoperative and postoperative [7, 13]. However, there is a lack of objectivity and quantification in this technique.

While gait and motion analysis have been used for specific pathologies such as cerebral palsy, and Parkinson's disease, only a few projects employed this technology in ASD patients [14–16]. A recent study showed that ASD patients had walking kinematic alterations associated with a deteriorated quality of life [17]. However, it is still unknown which spinal deformity component from the ASD classification affects the gait pattern.

Therefore, the aim of this study is to evaluate 3D kinematic alterations during gait in ASD subjects with different types of spinal deformity. We hypothesized that kinematic alterations during walking in patients with ASD are mostly related to sagittal malalignment.

Methods

Study design

This is a cross-sectional study including ASD patients and asymptomatic adults. Enrolled ASD subjects were referred to our center by their physicians based on analysis of radiographs on which they presented any of the following radiological criteria: PT > 25°, SVA > 50 mm, PI-LL > 10°, Cobb angle > 20° and/or T1T12 > 60°. All ASD patients were older than 20 years and complained from pain/discomfort. Patients with gait altering disorders not explained by their spine condition or having undergone surgery of the lower limbs or spine within the past two years were excluded. Recruited controls were older than 20 years with no pain, no lower limbs or spinal surgery, no musculoskeletal system disorders and no history of degenerative joint disease. This study was approved by the Ethics Committee of our institution (CEHDF1259). All participants signed an informed consent prior to the trials.

Data acquisition

Age (years), sex (F/M), height (cm) and weight (Kg) were collected for each subject. Subjects underwent low dose full body biplanar X-rays (EOS®, EOS Imaging, Paris, France) in free standing position [18] (Fig. 1a). Three-dimensional reconstructions of the spine and pelvis were performed using Stereos® software (v1.8.99.20R, EOS imaging, Paris, France) with extraction of the following radiographic parameters: SVA (mm), center of auditory meatus to hip axis plumbline CAM-HA (mm), Pelvic incidence PI (°), Pelvic tilt PT (°), PI-LL mismatch (°), T1T12 thoracic kyphosis TK (°), L1S1 lumbar lordosis LL (°), L4S1 lumbar lordosis (°), global tilt GT (°), knee flexion KF (°) and frontal Cobb angle (°) (Fig. 1b–c). The lordosis distribution index LDI (%) was calculated as the ratio between L4S1 and L1S1 [19]. The Global Alignment and Proportion (GAP) Score was also calculated [20, 21], as a predictor of mechanical complications in ASD patients undergoing surgery, based on a pelvic incidence proportional method of analyzing the sagittal plane. For the lumbar lordosis distribution, the lumbar apex was defined as the most distant point from the line joining the inflection point and the midpoint of the sacral plate [22, 23].

All participants filled the following HRQoL and disability questionnaires [7, 8]: Visual Analog Scale for pain (VAS), Oswestry Disability Index (ODI), Short Form Health Survey (SF-36): measuring both Physical Component (PCS) and Mental Component (MCS) Summary.

Eight cameras (Vero 2.2, Vicon Motion Systems®, Oxford, UK) were used to capture full-body kinematics during gait (frequency: 200 Hz). The conventional Davis protocol was used for the lower limb marker set [24]. For the trunk and spine, markers were placed according to the Leardini protocol [25] on the following anatomical bony landmarks (Fig. 2a–b): right and left acromions, deepest point of the suprasternal notch, xiphoid process and spinous processes of C7, T2, T10, L1, L3 and L5 vertebras (Fig. 1b). Patients walked at self-selected speed on a 10-m walkway. The following joint angles were calculated in the three planes using Nexus and ProCalc (Vicon®, Oxford, UK) and normalized to the gait cycle: segmental spine motion (L3L5 relative to L1L3, L1L3 relative to T10L1, T10L1 relative to

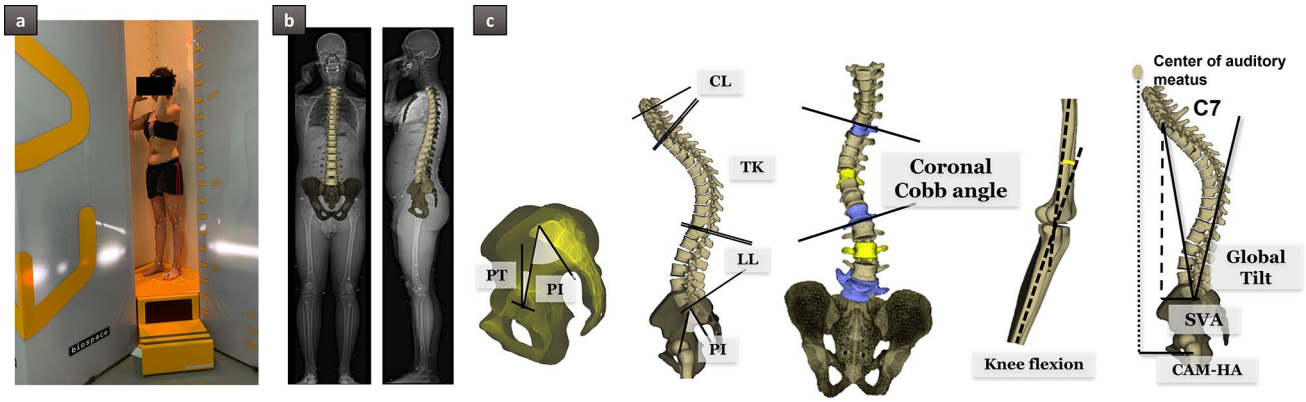

Fig. 1 **a** Subject in the free standing position during the EOS biplanar X-ray acquisition (**b**) 3D reconstruction of the spine and pelvis (**c**) Spino-pelvic and postural parameters: pelvic incidence PI (°), pelvic tilt PT (°), L1S1 lumbar lordosis (°), T1T12 thoracic kyphosis (°), coronal Cobb angle (°), sagittal vertical axis SVA (mm), distance from center of auditory meatus plumb line to hip-axis CAM-HA (mm), and Global Tilt (°)

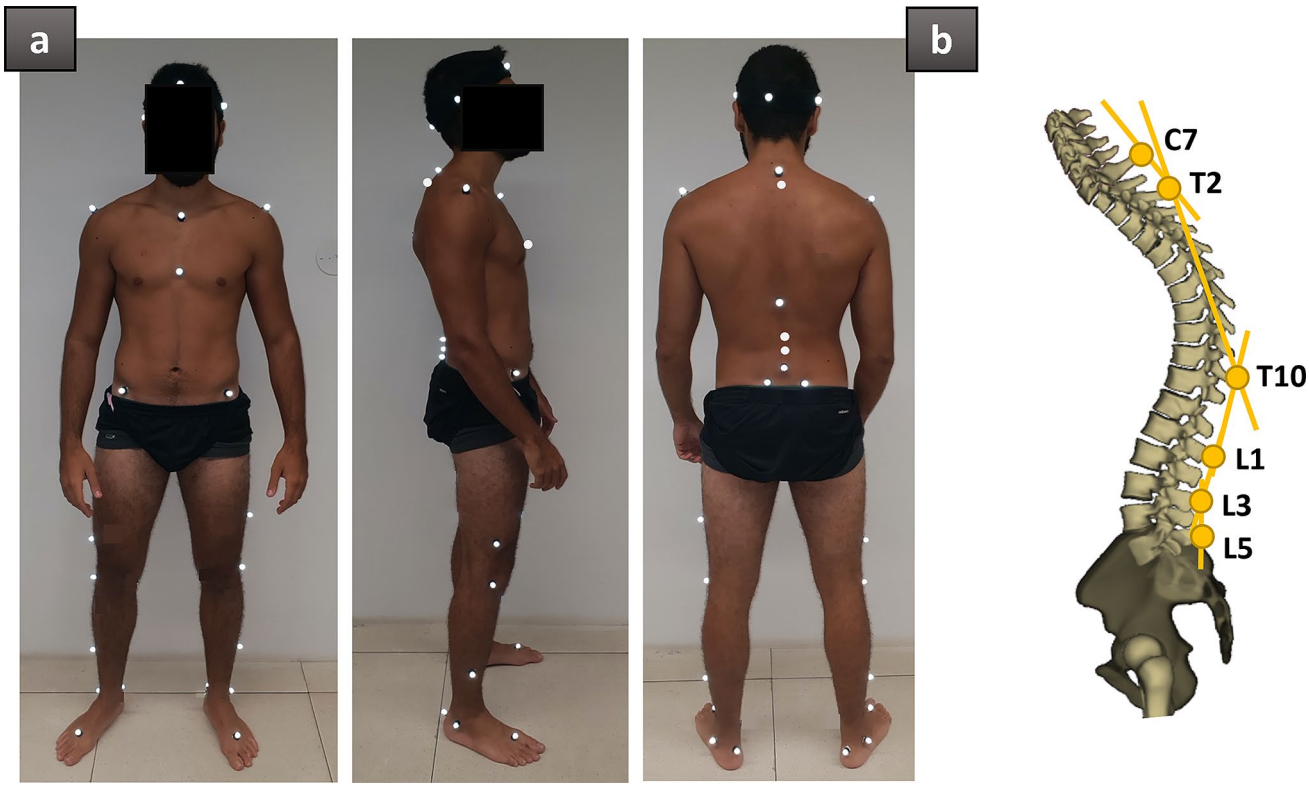

Fig. 2 **a** Positioning of markers used during gait acquisition (**b**) Representation of spine segments as described by Leardini et al.

T2T10 and T2T10 relative to T2C7), trunk (pelvis relative to thorax), pelvis (pelvis relative to global reference), pelvis-L3L5 (pelvis relative to L3L5), hip (femur relative to pelvis), knee (tibia relative to femur), ankle (foot relative to tibia) and foot (foot relative to global reference). The following spatial–temporal parameters were collected: walking speed (m/s), cadence (steps/min), time of foot off (transition from stance to swing phase, in % of gait cycle), single and double support times (s), as well as step length (m).

The gait deviation index (GDI) was also calculated [26], measuring the deviation of a specific subject's gait from a normative database, based on the pelvis and lower limb kinematics. It is scored between 0 and 100 and decreases with severity.

Statistical analysis

A comparison of demographics between ASD and controls was performed using Mann–Whitney. Sex was compared using Chi-squared test.

The ASD population was divided into three subgroups, patients in the ASD-Frontal group presenting only a coronal Cobb angle > 20 degrees, patients in the ASD-Sagittal group presenting at least one of the following parameters an SVA > 50 mm, and/or a PT > 25 degrees, and/or a PI-LL > 10 degrees, and patients in the ASD-HyperTK group presenting only a TK > 60 degrees.

HRQoL outcomes, spino-pelvic alignment, global postural parameters and kinematic parameters (mean, minimum, maximum and range of motion ROM) during the gait cycle were compared between groups using Kruskal–Wallis test [27, 28].

The relationship between altered radiographic parameters and gait changes was assessed through a univariate analysis, using Pearson's correlations.

Statistical analyses were performed using XLSTAT (version 2019, Addinsoft, Paris, France). The level of significance was set at 0.05, and Bonferroni corrections were applied when multiple correlations were computed.

Results

Demographics

In total, 119 ASD patients (51.5 ± 19.2 years [20–85]; 90 F) and 60 controls (48.6 ± 10.1 years [20–76]; 29 F) were enrolled with similar age, weight (ASD = 72.1 ± 14.5 kg vs controls = 73 ± 12.7 kg) and sex distribution (all $p > 0.05$). ASD patients were on average 4.2 cm shorter than controls (161.9 ± 9.9 cm vs 166.1 ± 8.0 cm, $p = 0.002$). Out of the 119 ASD patients, 28 were classified as ASD-Frontal, 40 as ASD-HyperTK and 51 as ASD-Sagittal.

Radiographic parameters

Only ASD-Sagittal showed increased SVA (67.7 mm vs. − 7.4 mm, $p < 0.001$), PT (27.9° vs. 11.7°, $p < 0.001$), GT (28.9° vs. 0.8°, $p < 0.001$) and PI-LL (17.2° vs. − 11.6°, $p < 0.001$) compared to controls. ASD-Sagittal also had significantly lower L1S1 lumbar lordosis (38.6° vs. 61.9°, $p < 0.001$), lower L4S1 lumbar lordosis (29.4° vs. 38.3°, $p < 0.001$) and higher knee flexion (11.6° vs. 0.3°; $p < 0.001$). ASD-Sagittal showed a decreased LDI (58.2° vs. 62.5°, $p = 0.002$) with an increased GAP score (7.6 vs. 1.7, $p < 0.001$) compared to controls. ASD-HyperTK had significantly increased TK (71.8° vs. 46.9°, $p < 0.001$), while ASD-Front had the largest coronal Cobb angle (38.2° vs. 3.6°, $p < 0.001$, Fig. 3). All subjects showed a lumbar apex at L3 or L4, except for 5 subjects in ASD-Sagittal who had a lumbar apex at L5 (3 subjects) and L2 (2 subjects).

HRQoL scores

ASD-Sagittal had significantly the lowest PCS when compared to controls (36.8 vs. 50.6, $p < 0.001$) and showed the highest disability (ODI: 38.2 vs. 14.5, $p < 0.001$); ASD-Frontal and ASD-HyperTK scored in between on the PCS (44.4 and 41.4, respectively) and the ODI (21.5 and 28.1, respectively). However, ASD-Sagittal and ASD-HyperTK had an increased VAS score (6.6 and 6.1, resp. vs. 3.3 $p < 0.001$) compared to controls.

Walking kinematics

Detailed between-group comparisons of kinematic parameters are displayed in Table 1. GDI was the most affected in ASD-Sagittal group (80.4 vs. 95.0 in controls, $p < 0.001$). ASD-Sagittal patients showed a decreased ROM of the pelvic obliquity (6.3° vs. 10.5°, $p < 0.001$) and rotation (9.8° vs. 11.8°, $p = 0.001$) compared to controls. They also had a reduced ROM of the hip flexion/extension (38.3° vs. 44.8° in controls, $p < 0.001$). Moreover, ASD-Sagittal group exhibited a lack of knee flexion in swing (55.3° vs. 61.7°, $p < 0.001$), a lack of knee extension in stance (8.6° vs. 3.5°, $p < 0.001$), thus a decreased ROM of the knee flexion/extension (51.2° vs. 61.0°, $p < 0.001$) during the whole gait cycle. Furthermore, ASD-Sagittal subjects presented an increased head extension (− 9.5° vs 2.0° in controls, $p < 0.001$) and a flexed thorax (13.2° vs. 3.8° in controls, $p < 0.001$) during walking. Moreover, ASD-Sagittal group showed a reduced dynamic lumbar lordosis L1-L3/L3-L5 during gait (− 6.9° vs -12.5° in controls, $p < 0.001$). Concerning time-distance parameters, subjects in the ASD-Sagittal group walked slowly (0.8 vs. 1.2 m/s, $p < 0.001$), with a longer double support time (0.37 vs. 0.25 s, $p < 0.001$) and shorter step length (0.49 vs. 0.64 m, $p < 0.001$) compared to controls.

As for ASD-HyperTK patients, they had no significant decrease in GDI (90.7 vs. 95.0 in controls, $p = 0.07$). ASD-HyperTK showed decrease in ROM of the pelvic obliquity (8.3° vs. 10.5°, $p = 0.003$) and rotation (9.4° vs. 11.8°, $p = 0.001$) compared to controls. ASD-HyperTK showed decreased dynamic pelvic tilt (8.3° vs. 10.5°, $p = 0.004$). ASD-HyperTK group had also a significant decreased ROM of knee flexion/extension (55.8° vs. 61.0°, $p < 0.001$). They presented with slower walking pace (1.0 vs 1.2 m/s in controls, $p < 0.001$), a longer double support time (0.29 vs. 0.25 s, $p = 0.002$) and a shorter step length (0.57 vs. 0.64 m, $p < 0.001$), but to a lesser extent than the ASD-Sagittal group.

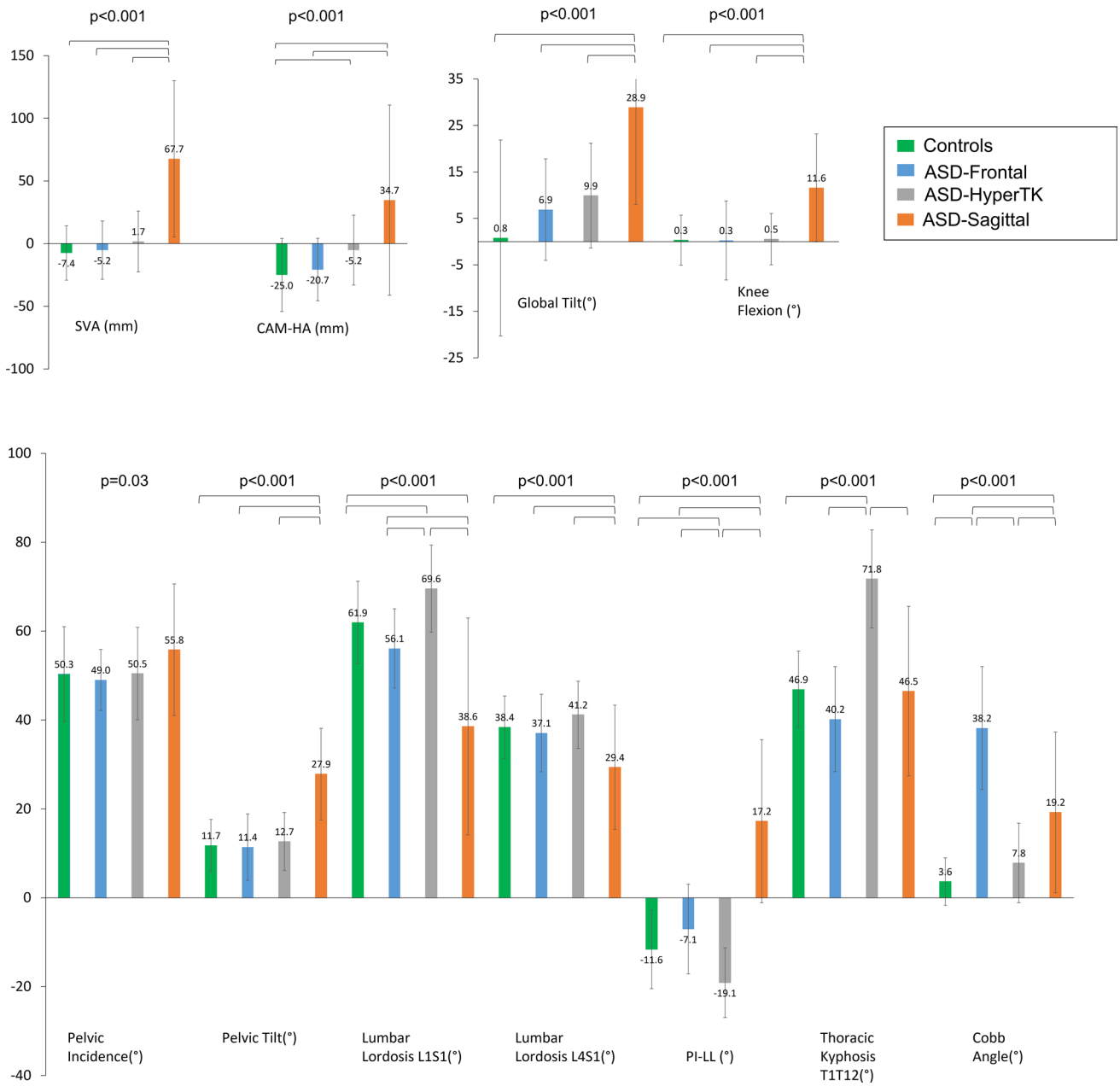

Fig. 3 Comparison of spino-pelvic and postural parameters between subgroups: Controls, ASD-Frontal, ASD-HyperTK and ASD-Sagittal

Regarding the ASD-Frontal population, they had normal GDI (94.6 vs 95.0 in controls, $p = 0.86$). They had an extended thorax attitude (− 0.9° vs. 3.8° in controls, $p < 0.01$). The remaining kinematics and time-distance parameters were similar to controls.

Kinematic waveforms that differed between subgroups are displayed in Fig. 4.

Univariate analysis

The univariate analysis showed several significant correlations between radiographic parameters, HRQoL outcomes and gait alterations (Table 2). The GDI was negatively correlated to both SVA and PT ($r = -0.39$ and $r = -0.37$, resp.). The decrease of knee flexion/extension ROM correlated with SVA, PT, LL, VAS and ODI ($r = -0.49$ to $r = -0.29$). Walking speed and step length negatively correlated with

Table 1 Comparison of gait kinematics between the 4 subgroups: controls, ASD-Frontal, ASD-HyperTK and ASD-Sagittal

	Mean ± SD												*p*-value	Controls vs. ASD-Frontal	Controls vs. ASD-HyperTK	Controls 3 vs. ASD-Sagittal	ASD-Frontal vs. ASD-HyperTK	ASD-Frontal vs. ASD-Sagittal	ASD-HyperTK vs. ASD-Sagittal
	Controls			ASD-Frontal			ASD-HyperTK			ASD-Sagittal									
Head																			
Mean head flexion/extension (°)	2.0	±	9.1	0.9	±	11.3	− 1.3	±	11.5	− 9.5	±	12.3	**< 0.001**			*		*	*
ROM flexion/extension head (°)	6.8	±	4.2	7.4	±	4.6	6.9	±	7.9	6.6	±	4.4	0.39						
Trunk																			
Mean flexion/extension thorax (°)	3.8	±	4.3	− 0.9	±	4.0	6.7	±	5.5	13.2	±	13.2	**< 0.001**	*		*	*	*	
ROM thorax flexion/extension (°)	3.2	±	1.1	3.2	±	1.1	3.0	±	1.0	3.3	±	1.7	0.81						
Mean shoulder/pelvis axial rotation (°)	0.9	±	2.5	2.6	±	3.7	0.3	±	3.5	0.8	±	4.3	0.11						
ROM shoulder/pelvis axial rotation (°)	16.1	±	4.8	16.5	±	5.7	13.8	±	5.3	12.4	±	5.0	**< 0.001**			*		*	
Mean pelvis-L3L5 flexion/extension (°)	10.1	±	5.2	12.7	±	9.8	14.1	±	7.8	9.7	±	9.8	0.08						
ROM pelvis-L3L5 flexion/extension (°)	5.9	±	2.0	6.8	±	3.9	6.2	±	3.3	5.8	±	2.6	0.80						
Spinal segments																			
Mean L1L3-L3L5 flexion/extension (°)	− 12.5	±	6.0	− 12.4	±	9.3	− 13.7	±	8.7	− 6.9	±	12.0	**0.001**			*			*
ROM L1L3-L3L5 flexion/extension (°)	8.0	±	3.3	8.5	±	5.5	9.0	±	6.2	6.0	±	4.2	**0.001**			*		*	*
Mean T10L1-L1L3 flexion/extension (°)	− 7.2	±	6.1	− 7.8	±	7.7	− 17.3	±	12.0	− 2.1	±	13.3	**< 0.001**		*		*		*
ROM T10L1-L1L3 flexion/extension (°)	5.9	±	2.3	5.8	±	5.2	6.7	±	3.8	4.7	±	4.4	**0.002**			*			*
Mean T2T10-T10L1 flexion/extension (°)	24.0	±	3.7	12.9	±	8.6	35.2	±	8.5	23.4	±	11.9	**< 0.001**	*	*		*	*	*
ROM T2T10-T10L1 flexion/extension (°)	2.9	±	1.1	2.8	±	2.5	2.5	±	1.2	2.5	±	1.9	**0.03**			*			
Mean C7T2-T2T10 flexion/extension (°)	26.7	±	5.5	27.5	±	8.2	34.9	±	7.7	30.6	±	9.0	**< 0.001**		*		*		
ROM C7T2-T2T10 flexion/extension (°)	3.4	±	1.3	3.5	±	2.2	3.5	±	2.0	3.1	±	1.8	0.11						
Pelvis																			
Mean pelvic tilt (°)	12.7	±	6.4	11.2	±	5.5	8.3	±	6.6	10.2	±	10.6	**0.04**		*				
ROM pelvic tilt (°)	3.6	±	1.3	3.7	±	1.3	3.6	±	1.1	4.2	±	2.1	0.54						

Table 1 (continued)

	Mean ± SD												*p*-value	Controls vs. ASD-Frontal	Controls vs. ASD-HyperTK	Controls 3 vs. ASD-Sagittal	ASD-Frontal vs. ASD-HyperTK	ASD-Frontal vs. ASD-Sagittal	ASD-HyperTK vs. ASD-Sagittal
	Controls			ASD-Frontal			ASD-HyperTK			ASD-Sagittal									
Mean pelvic obliquity (°)	0.1	±	1.7	0.2	±	1.8	0.2	±	1.8	− 0.3	±	3.1	0.78						
ROM pelvic obliquity (°)	10.5	±	3.5	11.1	±	4.3	8.3	±	3.5	6.3	±	3.6	**< 0.001**		*	*	*	*	*
Mean pelvic rotation (°)	1.0	±	2.9	− 0.1	±	3.5	1.0	±	2.8	0.9	±	4.2	0.28						
ROM pelvic rotation (°)	11.8	±	3.7	12.8	±	5.4	9.4	±	4.7	9.8	±	4.6	**< 0.001**		*	*	*	*	
Hip																			
ROM hip flexion/extension in stance (°)	43.3	±	5.6	41.6	±	6.7	41.3	±	5.8	37.0	±	8.6	**0.001**			*			
Max hip extension in stance (°)	− 6.3	±	8.0	− 6.7	±	7.0	− 10.0	±	8.5	− 3.2	±	13.4	**0.03**						*
ROM hip flexion/extension (°)	44.8	±	5.2	43.3	±	6.0	42.8	±	5.4	38.3	±	8.8	**< 0.001**			*		*	
ROM hip abduction/adduction (°)	15.4	±	3.9	15.2	±	3.7	13.2	±	3.6	13.1	±	4.3	**0.003**		*	*			
Mean hip internal/external rotation (°)	− 1.1	±	9.5	3.1	±	13.8	− 4.3	±	11.6	− 5.7	±	15.8	**0.01**					*	
Mean hip flexion/extension (°)	18.1	±	7.5	16.5	±	5.5	13.0	±	8.7	16.7	±	11.7	0.07						
Mean hip abduction/adduction (°)	− 0.6	±	4.1	0.8	±	3.0	0.0	±	3.5	− 0.5	±	5.0	0.36						
Knee																			
Max knee flexion in stance (°)	16.8	±	6.0	16.4	±	7.0	17.2	±	7.2	18.6	±	7.9	0.58						
Max knee extension in stance (°)	3.5	±	5.6	3.9	±	5.2	4.6	±	6.3	8.6	±	8.1	**0.001**			*		*	*
Max knee flexion in swing (°)	61.7	±	6.1	61.3	±	6.8	58.4	±	7.7	55.3	±	10.7	**0.003**			*		*	
Knee extension at initial contact (°)	4.1	±	7.2	8.0	±	8.9	6.7	±	7.6	9.7	±	10.5	**0.04**			*			
ROM knee flexion/extension (°)	61.0	±	6.4	59.6	±	6.9	55.8	±	7.6	51.2	±	10.9	**< 0.001**		*	*	*	*	
Mean Knee flexion/extension (°)	21.1	±	4.5	21.2	±	5.0	21.1	±	6.0	21.6	±	6.8	0.93						
Ankle & Foot																			
Max dorsiflexion in stance (°)	18.1	±	5.7	17.6	±	7.4	17.8	±	6.9	20.5	±	8.6	0.24						
Max plantar flexion in stance (°)	− 8.4	±	8.3	− 8.6	±	8.0	− 7.5	±	7.2	− 6.4	±	6.4	0.53						
Max dorsiflexion in swing (°)	8.9	±	6.0	10.1	±	6.3	8.9	±	5.2	9.4	±	6.8	0.45						

Table 1 (continued)

	Mean ± SD												*p*-value	Controls vs. ASD-Frontal	Controls vs. ASD-HyperTK	Controls 3 vs. ASD-Sagittal	ASD-Frontal vs. ASD-HyperTK	ASD-Frontal vs. ASD-Sagittal	ASD-HyperTK vs. ASD-Sagittal
	Controls			ASD-Frontal			ASD-HyperTK			ASD-Sagittal									
ROM dorsiflexion/plantar flexion (°)	30.1	±	6.9	31.9	±	8.2	27.9	±	7.2	30.5	±	8.9	0.07						
Mean dorsiflexion/plantar flexion (°)	6.0	±	5.1	5.3	±	5.6	6.2	±	5.0	6.6	±	4.6	0.71						
Mean foot progression in stance (°)	− 10.5	±	6.1	− 7.2	±	5.9	− 10.0	±	6.7	− 13.7	±	9.8	**0.001**					*	
ROM foot progression in stance (°)	10.1	±	3.9	10.9	±	3.7	9.5	±	4.6	11.0	±	5.4	0.42						

Statistical significant differences are indicted in bold and *

SVA, PT, PI-LL, VAS and ODI (r = -0.58 to r = − 0.39, all $p < 0.05$, Fig. 5).

Discussion

Spinal deformity is a major cause of gait alterations, consequently affecting patients' quality of life. Previous studies have shown that ASD patients had walking kinematic alterations that were associated with a deteriorated quality of life [17, 29]. This study evaluated gait alterations in ASD depending on their type of spinal deformity.

On free standing radiographs, ASD-Sagittal population presented with moderate-to-severe alteration in sagittal parameters such as SVA, PT, GT and LDI. A decreased lumbar lordosis was noticed in ASD-Sagittal patients, inducing a forward shift of the trunk (increased SVA). As a result, they tended to increase their pelvic retroversion (increased PT), as a compensation for the forward bending of the trunk, and thus adjust their center of gravity [30]. Some patients, who exhausted their pelvic retroversion reserve, had to recruit their knees (increased KF), as an additional compensatory mechanism. Although almost all ASD subjects presented a lumbar apex comparable to controls, ASD-Sagittal showed an altered lumbar lordosis distribution with a decreased LDI indicating that eventual surgical correction should focus on the inferior arc of the lumbar lordosis in these patients [19, 31]. The increased GAP score in some patients in the ASD-Sagittal group suggests that they are at greater risk of postoperative mechanical complications as shown in previous studies [21].

On the kinematic level, sagittal spinal malalignment seems to be the main driver of gait alterations in adult spinal deformity. ASD subjects with alteration in sagittal parameters such as SVA, PT and/or PI-LL mismatch had the most altered gait and quality of life compared to other ASD subgroups and controls.

In fact, the ASD-Sagittal patients in this study were the most affected on the physical level, showing lower PCS and greater ODI when compared to controls (Fig. 4).

Also, ASD-Sagittal group had the most affected GDI that makes them more prone to falls [32]. They had limited flexibility in the pelvis, hips and knees. ASD-Sagittal patients walked with a flexed attitude in the thorax, hips and knees, along with an increased extension of the head most probably to preserve a horizontal gaze. They had no significant difference in dynamic pelvic tilt during gait when compared to control. This explains the lack of knee extension in stance probably participating in the chain of compensation, readjusting the center of gravity above the feet to prevent falling ahead during walking, as formerly observed in previous studies [33, 34]. Moreover, the decreased ROM of the hips and knees in ASD-Sagittal group ensures greater stability

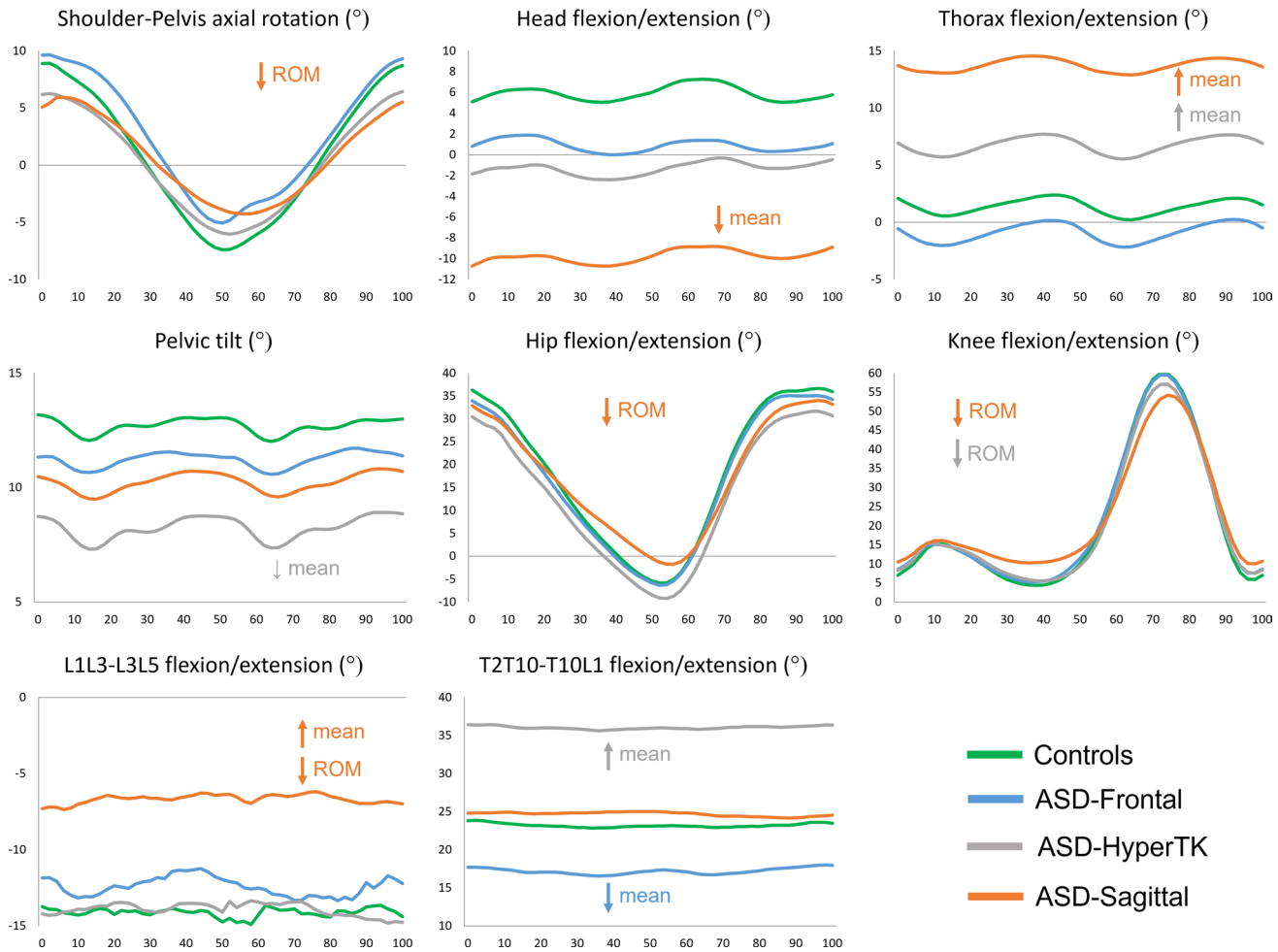

Fig. 4 Average curves of gait kinematics for each subgroup: Controls, ASD-Frontal, ASD-HyperTK and ASD-Sagittal: mean and ROM (range of motion) comparisons

Table 2 Correlations between gait kinematic parameters and spino-pelvic and postural parameters as well as HRQoL outcomes. Only significant correlations were reported ($p < 0.05$)

Pearson's r *correlation coefficient*	SVA	PT	GT	T1T12	L1S1	PI-LL	L4S1	PCS	VAS	ODI
Gait Deviation Index	−0.39	−0.33			0.26	−0.32	0.262	0.34		−0.25
Hip flexion/extension ROM	−0.53				0.23	−0.29	0.286	0.37	−0.37	−0.43
Knee flexion/extension ROM	−0.42	−0.29			0.3	−0.29		0.42	−0.38	−0.49
Walking Speed	−0.53	−0.43	−0.272		0.43	−0.46		0.53	−0.39	−0.5
Step Length	−0.58	−0.44	−0.335		0.41	−0.46	0.260	0.55	−0.44	−0.56
Mean Thorax flexion/extension	0.65	0.5	0.396		−0.38	0.42		−0.3	0.28	0.45
Mean T2T10−T10L1 flexion/extension				0.65		−0.27				
Mean L1L3-L3L5 flexion/extension	0.37	0.38			−0.41	0.46			0.27	0.32

and enables better motion control. Moreover, ASD-Sagittal patients showed decreased ROM in spinal segments and shoulder-pelvis axial rotation due to spine rigidity, especially in the lumbar segments as described in previous studies [35]. They tended to walk slower, with shorter steps, delayed foot off and longer double support time in order to maintain stability during walking.

ASD-HyperTK population suffered from increased TK but had no alteration in the other sagittal parameters such as SVA, PT and PI-LL mismatch. The increased thorax flexion

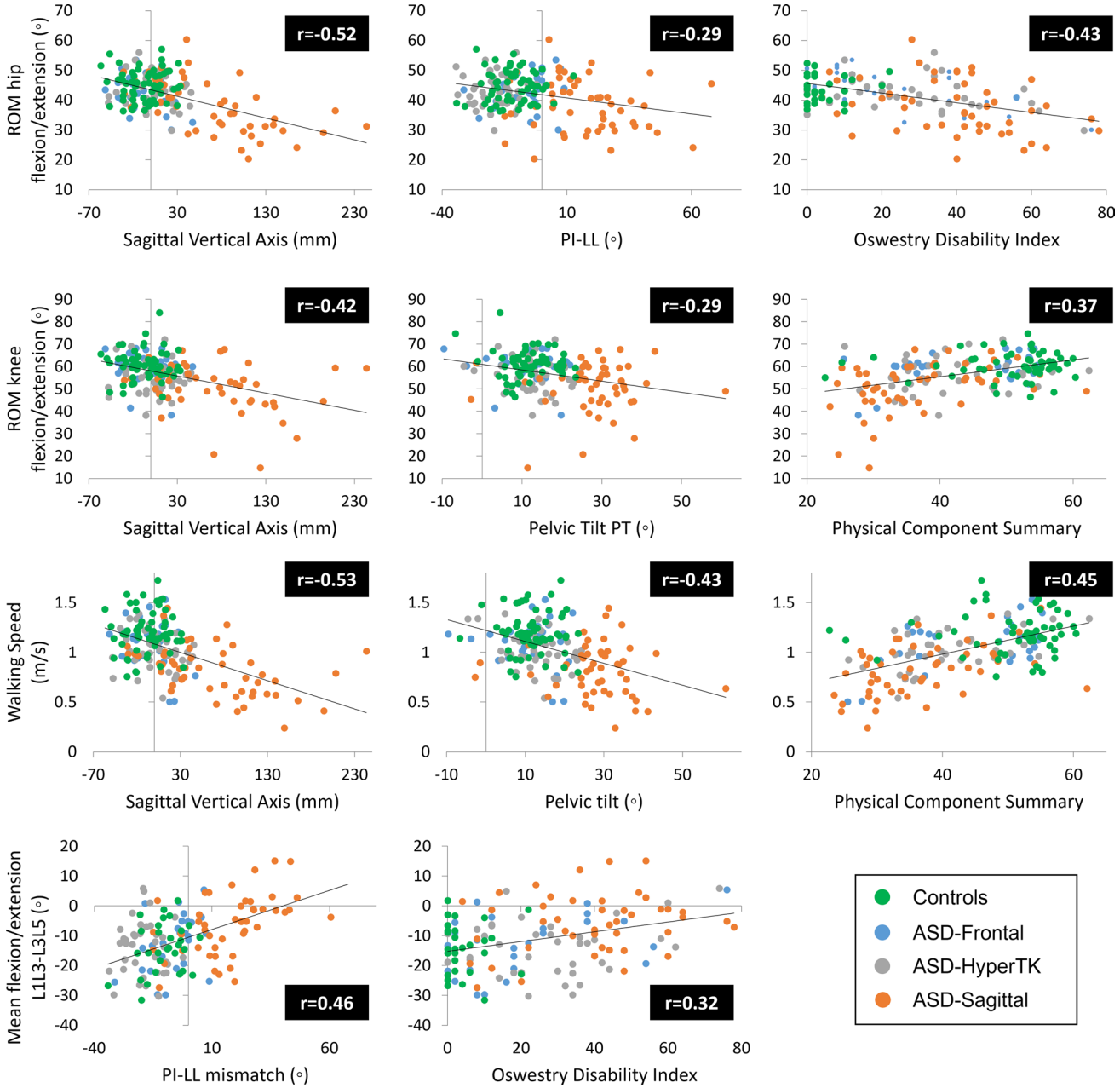

Fig. 5 Correlation between altered kinematic parameters and both radiographic and health-related quality of life (HRQOL) scores

was compensated by an increased pelvic retroversion during gait avoiding by this falling ahead, with no need to flex their knees, unlike ASD-Sagittal group. ASD-HyperTK patients had some altered gait kinematics and quality of life scores but to a lesser extent than ASD-Sagittal patients. In consequence, hyperkyphosis, in presence of conserved lumbar flexibility, does affect motion during gait but to a minor degree than patients with sagittal imbalance.

ASD-Frontal population had almost similar gait patterns compared to controls. As a result, it seems that an isolated frontal scoliosis fails to affect sagittal balance and therefore motion analysis.

The gait alterations of ASD in this study were related to the skeletal radiographic abnormalities and the deteriorated quality of life scores. In fact, static deformities such as SVA, PT and PI-LL mismatch correlated to the following gait alterations: decreased ROM of pelvic obliquity, hip and knee flexion/extension, slower walking speed, shorter steps and lower GDI.

In conclusion, this study showed that sagittal malalignment is the main driver of gait alterations in ASD patients.

ASD patients with sagittal malalignment walked at a slower pace with smaller steps and longer support time. They also exhibited lesser hip and knee extension in stance, with limited mobility in the hips and knees, as a compensation mechanism to the forward trunk tilt. These dynamic changes make them more prone to falls and were correlated to the altered radiographic sagittal parameters and deteriorated quality of life scores. ASD with only hyperkyphosis showed similar changes but to a lesser extent. These changes were not found in subjects with only frontal malalignment.

These results underline the importance of differentiating ASD patients according to the type of radiological deformity since it has different impact on their functionality and therefore their quality of life. Moreover, this study showed that 3D gait analysis is an objective tool to evaluate functionality in ASD patients depending on their type of spinal deformity. Further studies should assess whether surgical correction or physical therapy could reverse these mechanisms, in order to improve gait kinematics and, consequently, the quality of life in these patients.

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Declarations

Conflict of interest None.

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