THE EFFECT OF A SPINA BIFIDA-DEFECT AT THE LUMBOSACRAL JUNCTION ON SPINOPELVIC ALIGNMENTS

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ABSTRACT

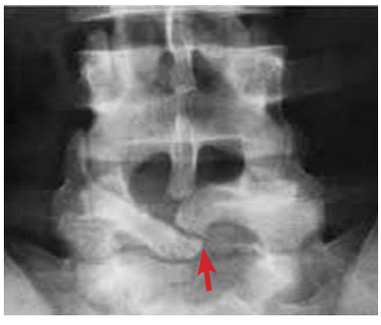
Background: Spina bifida defects, which are mild neurotube defects are commonly found at the lumbosacral junction. The defect which is associated with micronutrient deficiency (folate) is highly prevalent in impoverished populations; and because the defect occurs early in life, it has the potential to influence pelvic growth and in consequence the pelvic incidence.
Objective: The purpose of this study was to determine whether the presence of a Spina Bifida Occulta (SBO) defect at lumbosacral junction (L5 or S1) has any effect on the pelvic incidence.
Design: Prospective randomized case control study.
Methods: This study enrolled 172 consecutive patients with low back pain patients requiring X-rays of the lumbosacral spine. The X-ray films were screened for spina bifida occulta defect at the lumbosacral junction (L5, S1). Group 1 consisted of those patients with spina bifida occulta defect (SBO-defect) while Group 2 consisted of those without the defect. Measurements of the spino-pelvic parameters were done manually on all the films, followed by calculation of pelvic incidence/lumbar lordosis mismatch (PI-LL+ 10), the primary outcome (or deformity). The means of the two groups were subjected to independent t-test and Pearson Correlation Moment for relationships.
Results: A total of 172 patients were enrolled 88 had SBO-defect (Group 1) and 84 were without defect (Group 2). There were significant differences in the pelvic incidence and the PI-LL+10 mismatch between the two groups, with Group 1 showing higher values (P-value < 0.001). Other spino-pelvic parameters except sacral slope showed significant differences.
Conclusion: The SBO-defect which occurs early in foetal life, appear to cause compensatory pelvic growth with increased pelvic incidence. Consequently, the sacral orientation and other pelvic measurements become affected.

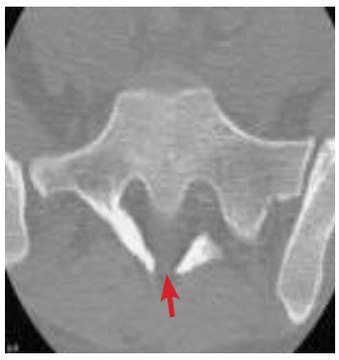
Key words: Spina-bifida-occulta, Spina-bifida-defect, Spinopelvic-parameters, Pelvic incidence, Pelvic tilt, Sacral slope, Lumbar lordosis, Pelvic-incidence-lumbar-lordosis-mismatch

INTRODUCTION

Spina bifida or split spine is a developmental defect of the posterior elements of the lumbar spine. The defect is from incomplete development of the spinous process and parts of the lamina leaving an osseous gap covered by a membrane. The lesion is commonly confined to the sacrum and extends into the lumbar region. Spina bifida is a common neural tube defect which is clearly visible on the anteroposterior radiograph of the lumbosacral spine and or CT scan (Figure 1).

Figure 1
Images of spina bifida as seen on plain radiograph AP view (left) and CT scan (right). Failure of fusion of posterior elements is clearly visible (arrows)





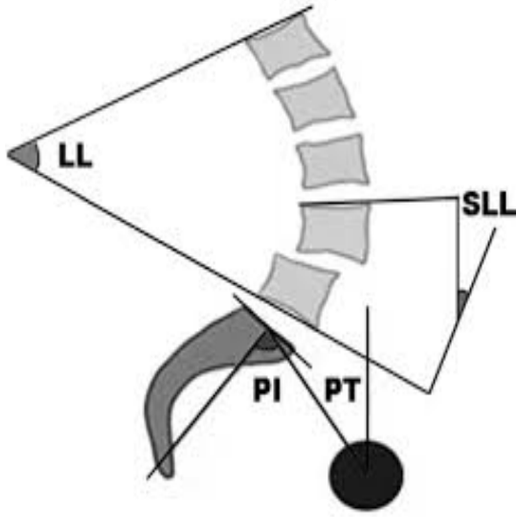
The neurotube defects occur early in embryonic life and multiple studies show lack of folic acid (folate) as the main cause (1-4). Neurotube defects appear in a spectrum of severity; Spina Bifida Occulta (SBO) is the mild form of the anomaly and is compatible with survival to adulthood. On the other hand, children with the severer form of the disease, such as the open neurotube defect (spina bifida cystica) and the cranial varieties have increased morbidity and mortality throughout life (5).

Traditionally it was believed that SBO is asymptomatic, although some previous studies have associated the condition with low back pain (6,7). Small defects may remain asymptomatic but large defects are thought to cause instability at the

lumbosacral junction. Degenerative spondylolisthesis affects mainly the L4/5 lumbar segment, whereas spina bifida defects occur commonly at L5/S1 level. The S1 vertebra is more affected than L5 which is more affected than L4 (8). Since S1 is fixed to the pelvis and its positional orientation is influenced by this relationship, instability occurs when L5 is affected. The L5 lumbar in vertebra is reduced in size and acquires almost a blunt-wedge-shape when viewed in the lateral view and a 'butterfly shape' in the AP view (Figure 2). The deformation of L5 increases the possibility of instability and hence, chronic pain. The segment also suffers more of disc degeneration than other lumbar segments, perhaps from the effect of the shearing forces (9).

Figure 2

Illustration showing the reference line for measuring Pelvic Incidence (PI), Pelvic Tilt (PT), Lumbar Lordosis (LL), and distal or Segmental Lumbar Lordosis (SLL)



While spinopelvic alignments have been described by Duval-Beaupère *et al* (10), particularly the correlation between lumbar lordosis and the pelvic incidence, the relationship between SBO and the pelvic incidence, with consequences that alter the sacral slope and pelvic tilt has not been studied before. The interdependence between the spinal curves and the orientation of the pelvis has been reported in lumbosacral junctions with isthmic spondylolisthesis (11). While isthmic spondylolisthesis causes instability of this transitional zone, the effect of an SBO-defect at this level is unknown. It is postulated in this study that since the defect occurs early, it may affect the growth of the pelvis and its subsequent sacral orientation. This in turn causes compensatory pelvic tilt and reduced lumbar lordosis. However, SBO defect maybe fixed or unstable. An unstable defect will either limit or accentuate spinopelvic movements. As these movements require strong musculature, especially of the abdominal and paraspinal muscles, which tends to deteriorate hence, pain and deformity as the individual ages. The hypothesis is that large defects are a cause of lumbosacral instability and chronic low back pain.

MATERIALS AND METHODS

Study design and participants: This was a prospective, randomised comparative study at the Mombasa Hospital between January and December 2018. All patients that required lumbosacral spine X-rays for Low Back Pain (LBP) were recruited for the study. Personal identifiers were removed, and patients were labelled with case numbers. The patients were aged between 18 and 60 years. The inclusion criterion was skeletally mature, ambulant individual with an adequate lumbosacral X-ray (both AP and true lateral views showing both hips). Exclusion criterion were patients with overt presence of instability at the lumbosacral junction and those with previous spine or hip surgery. Also excluded were those patients with films showing vertebral bone pathology such as fractures, tumours, and infections. Participants with gross obesity (BMI >30) and those skeletally immature (<18 years of age) and those with severe degenerative spine (> 61 years) were excluded (fixed deformities). The remaining cases were systematically randomised by removing every third case from a random number 52.

The study was carried out with approval of the Hospital Institutional Review Board. All the individuals included willingly consented in writing for participation and for the use of their radiographs in the study.

Procedures: Recruited patients were sequentially given a serial number that became their identity. A semi-structured questionnaire was used to record personal details and all measurement findings. A detailed history of illness, including indication for X-rays was taken. Gender and age were recorded and height and weight taken. Two dedicated radiographers briefed on the requirements of the study were tasked to do the exposures and deliver the films to the author. The films were done on a Siemens Axiom Iconos R200 (German). Measurements of the spino-pelvic parameters were done manually on all the films independently by two examiners, a trained research assistant and the author. The results were reconciled or repeated for large variances. The parameters measured were Pelvic Incidence (PI), Lumbar Lordosis (LL), and Distal Lumbar Lordosis (DLL), Sacral Slope (SS) and Pelvic Tilt (PT)). The pelvic incidence/lumbar lordosis mismatch (PI-LL+ 10) was calculated. Lumbar lordosis was measured from the superior endplate of L1 to superior endplate of S1 and distal lumbar lordosis was measured from the superior endplate of L4 to superior endplate of S1. Finally,

the films were screened for spina bifida occulta defect (SBO-defect) at the lumbosacral junction (L4, L5, and S1). Those with SBO-defect at L5 or S1 were grouped together (Group 1) and those without as Group 2. For the regression model, the study used the pelvic incidence as the predictor variable as it represented the final effect of SBO-defect. The PI-LL+10 mismatch representing the consequent deformity was used as the outcome variable. Linear Regression was used to predict the value of PI-LL+10 mismatch on the value of the predictor variable pelvic incidence.

Outcome measures: The presence of SBO defect was the predictor or independent variable. The (PI-LL+ 10) representing the observed deformity was the primary outcome, while the pelvic incidence, lumbar lordosis, sacral slope, pelvic tilt were the secondary outcome variables. The analytical groups were Group I (patients with radiological SBO-defect) and Group 2 (patients without SBO defect).

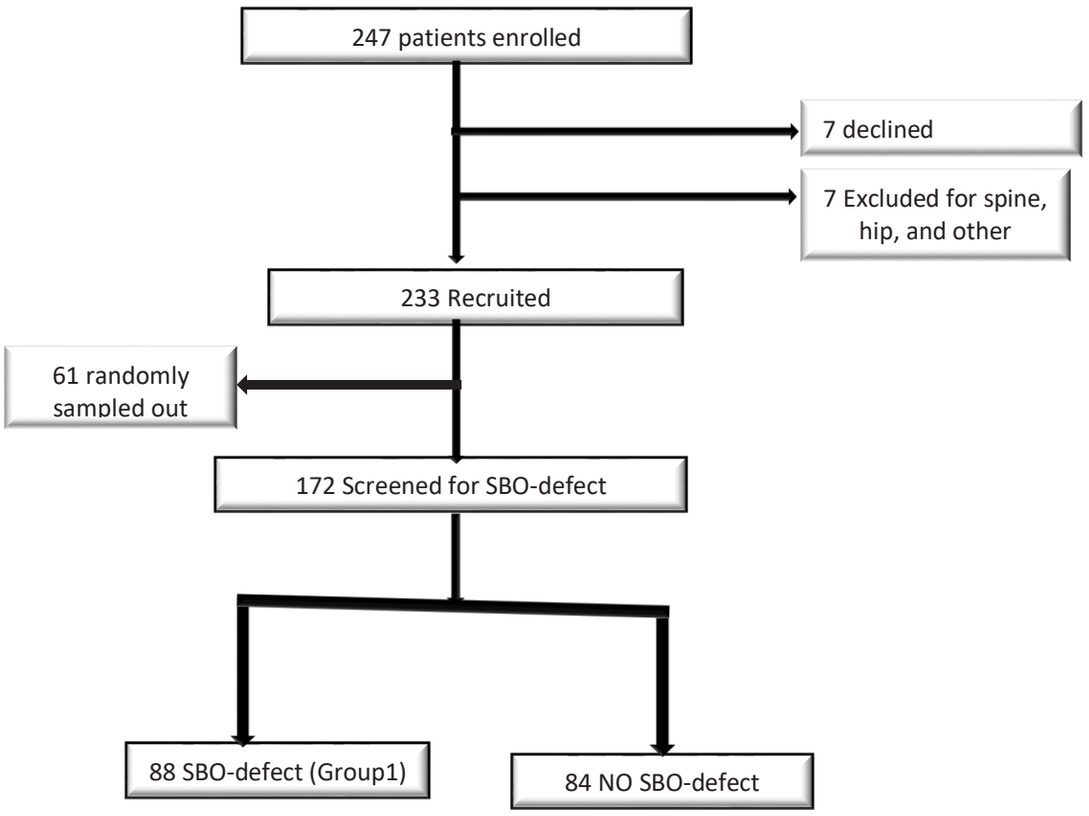
Statistical analysis: The study used a sample size calculator to calculate the minimum sample size for unknown population with a standard normal deviation set at 95% confidence level (1.96), probability of picking a positive response set at 50% (0.5) and with a confidence interval of 5% (0.05). The Sample size calculation was done for 90% power, confidence level of 95%, significance level of 0.05, and assumed odds ratio of 2% and exposure in controls of 40% (0.4). A sample size calculator showed a sample size of 80 in each group was adequate. Two hundred and forty seven participants were enrolled to compensate for possible attrition. Data were analysed using SPSS 20.0 (SPSS, Inc., Chicago, IL). The means were compared between the groups and significance tested with independent t-test or non-parametric tests. Pearson correlation moment was performed to establish relationships between the parameters. Linear regression was applied to model the relationship between PI (as the predictor variable) and PI-LL mismatch as the outcome variable. Data were presented as the mean value ± standard deviation with statistical significance set at $P < 0.05$.

RESULTS

From January 2018 to December 2018, a total of 247 patients met the inclusion criteria. Seven patients declined to participate, 240 were screened for the presence of S BO-defect. Another seven were excluded for spine or hip defects and 61 were randomly sampled out. One hundred and seventy-two were enrolled for the study, 88 in Group 1 and 84 in Group 2 (Figure 3).

Figure 3

Sample profile showing exclusions, sampling, and grouping. Group1 indicates those with SBO-defect and Group 2, those without



There were 80 males and 92 females with a mean age of 43.9 years. There were 41 (46.6%) males in Group 1 and 39 (46.4%) in Group 2, this difference in gender distribution was not statically significant (P = 0.168). The average age was 40.0 years in Group 1 and 44.9 years in Group 2 (P = 0.023. The demographic measurements and the means are shown in Table 1.

Table 1

Gender and age distribution within the groups

Group	Male	Female	Total	Mean age (years)	T-test
Group 1	41	47	88	40.0±10.1	
Group 2	39	45	84	44.9±13.1	P=0.023
Total (sample)	80	92	172	43.9±11.3	

Chi square P = 0.168

There were no statistically significant differences in height, weight, and BMI measurements between the two groups (P >0.05). This were then carried out, showing the statistically significant differences between the two groups in all spinopelvic measurements (PI, LL, PT and DLL), P<0.001) except for Sacral Slope (P< 0.205) (Table 2).

Table 2

Summary of means of various measurements in the sample and the two groups

	Means			T-test
	Sample	Group 1	Group 2	P-value
Height (Metres)	1.65±0.1	1.65±0.1	1.67±0.1	0.150
Weight (Kg)	78.5±14.1	78.8±13.9	78.3±14.0	0.555
BMI	28.7±5.2	29.2±5.4	28.2±5.1	0.768
Lumbar Lordosis (L1-S1)	35.9±11.2	29.6 ± 11.3	43.0 ± 6.2	<0.001
Distal Lumbar Lordosis (L4-L5)	16.5±6.2	16.6 ± 7.5°	15.0 ± 3.5°	0.016
Sacral Slope	37.2±9.1	34.8 ± 11.0	38.4 ± 4.1	0.205
Pelvic Tilt	19.7±11.7	23.4 ± 10.3	11.0 ± 8.0	<0.001
Pelvic Incidence	57.3±10.7	58.4 ± 10.1	50.3 ± 5.4	<0.001
PI-LL	11.5±14.7	19.4±10.7	-2.7±7.0	<0.001

Correlation: Pearson correlation moment was done to explore the relationship between PI-LL+10 and the measured spinopelvic parameters. PI-LL+10 mismatch has a significant positive linear relationship with pelvic incidence and pelvic tilt (r = 0.7), a significant negative linear relationship with lumbar lordosis (r = -0.6) and no relationship with sacral slope (r = -0.1). Distal lumbar lordosis has a significant but weak negative relationship with PI-LL mismatch (r = -0.2) (Table 3).

Table 3

Correlations between "PI-LL + 10 Mismatch" and spinopelvic measurements in patients with and without an SBO-defect (N = 172)

Pelvic incidence	.653	.000	Significant strong positive linear relationship
Pelvic tilt	.661	.000	Significant strong positive linear relationship
Sacral slope	-.118	.118	No relationship
Lumbar lordosis	-.640	.000	Significant moderate negative linear relationship
Distal lordosis	-.150	.047	Significant but poor negative linear relationship

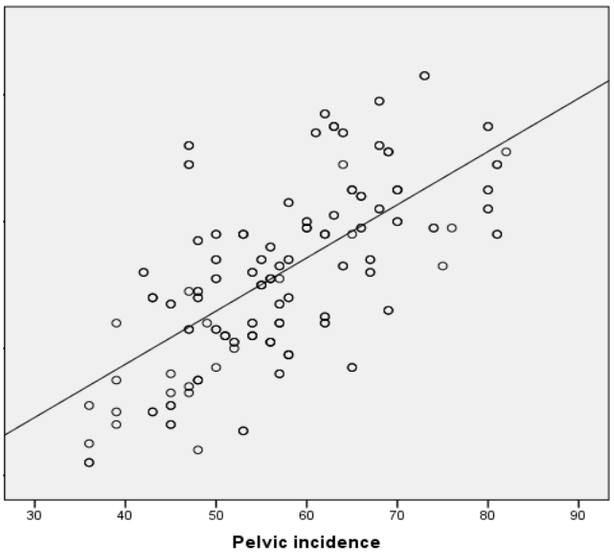
Regression: There was simple correlation of 0.653 (0.7) which indicates a moderate degree of correlation with R^2 value of 0.426 which indicates that 43% of the total variation in the dependent variable (PI-LL+10 mismatch) can be explained by the independent variable (Pelvic Incidence), P < 0.001 (Table 4 and Figure 4).

Table 4

Model summary and ANOVA for regression

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
	.653	.426	.423	10.480
ANOVA				
		Sum of squares	df	Mean square
	Regression	14206.894	1	14206.894
	Residual	19109.464	171	109.825
	Total	33316.358	172	

Figure 4
Shows the linear regression graph with R2 of 0.426 and a linear positive relationship



DISCUSSION

The prevalence SBO in this index population was found to be 67% (12), which is very high. This is explained by frequent draughts that causes shortage of green leafy vegetables which is the main source of folate. In comparison, Urrutia *et al* (13) in a radiological study of 228 patients in Chile, found the prevalence of SBO to be about 41.2%.

Spinopelvic malalignment as a consequence of developmental and degenerative anomalies at the lumbosacral junction is an expected occurrence. The sacrum is fixed to the ilium which acts as a pylon or anchor. The shape, size and orientation of the sacrum determines the overall alignment of the lumbar spine. Functionally, the sacrum is part of the pelvis and, therefore, deformation of the sacrum will affect the pelvis, in this case by way of thickening of the pelvis and increasing the pelvic incidence. The pelvic growth then results in malposition. When confronted with malposition the primary compensatory mechanism is through the soft tissues (muscles and ligaments). This compensation is often adequate but with age-related degeneration, the muscles lose strength and tone leading to failure of compensation. This muscular contraction referred to as muscle spasm may cause increased stress and strain which may be symptomatic.

The lumbosacral joint is capable of three-dimensional movements with a range of motion estimated to be 4-6º for flexion and extension, 3º of axial torque (one side), and 5º of lateral bending (one side) (14). Faulty mechanics will either accentuate or inhibit these movements, which alters the spinopelvic alignment. This study finds the presence of a spinal bifida defect to be associated with an increased pelvic incidence and reduced lumbar lordosis, with the final compensatory position being pelvic tilt. The probable mechanism is that the sacral inclination leads to pelvic shifting (retropulsion) to contain the defect leading to pelvic tilt as a final position. The result is increased pelvic tilt and reduced lumbar lordosis. This position is precariously maintained by spinal and paraspinal muscle contraction in order to maintain posture.

The SBO-defect causes increased lordosis in the distal lumbar vertebra, due to the reduced size of the posterior aspect of L5 and in a few cases L4 vertebral bodies (wedge effect). This study has confirmed that individuals with an SBO-defect have increased intrinsic distal lordosis ($16.6 \pm 7.5°$ vs. $15.0 \pm 3.5°$ for non-SBO-defect, $P<0.001$), although this lordosis is not adequate to alter the overall lumbar lordosis and a mismatch is maintained.

Being a transition segment, L5–S1 is fundamentally expected to experience axial and shearing forces, and therefore, to allow more axial transmission and less shearing, the angle of lordosis must relate to the sacral inclination, which in turn is influenced by the position of the pelvis (pelvic tilt). From a mechanical point of view, increased lumbar lordosis should cause greater shear forces and less axial forces. Conversely, decreased lumbar lordosis reduces shear forces and increases axial transmission (15,16). In this study, individuals with an SBO-defect have decreased overall lumbar lordosis ($29.6 \pm 11.3º$) compared to those without defect $43.0 \pm 6.2º$, $P <0.001$. The reduced overall lordosis can be explained by decreased sacral slope and muscular activity. As $16.6 \pm 7.5°$ vs. $15.0 \pm 3.5°$ for non-SBO-defect, $P<0.001$ected, the lumbar lordosis maintained a positive linear relationship with sacral slope ($r = 0.6$), and a negative linear relationship with pelvic tilt ($r = -0.4$), both being statistically significant. Individuals with a SBO-defect have decreased overall lumbar lordosis and may easily present with a flat back and pain.

The pelvic incidence is a morphological measurement that varies from one individual to another, being specific to each person. Pelvic incidence is therefore, a reliable value for inter-individual variations of spinal alignment measurements (17). Pelvic incidence measures the depth of the pelvis as acquired during individual development, and which stabilises after skeletal maturity; which means that certain factors can influence the size of the pelvis and consequently, that of pelvic incidence. Pelvic incidence has influence over positional parameters in

order to ensure trunk posture. A low value for PI means the range of adaptation for the pelvis is limited, in the event of malalignment. However, higher than normal values for PI have been associated with progressive spondylolysis, but were also found to have very tilted sacral slopes (18,19). Rothenfluh *et al*, (20) suggested that patients with adult spinal deformity should be assessed for pelvic incidence minus lumbar lordosis mismatch (PI-LL) as a primary indication for surgery in the symptomatic patient. This parameter eliminates individual differences in PI as the mismatch should not be more than 10 degrees. In this study individuals with SBO-defect had larger pelvic incidences than those without defect, a mean PI of 58.4 ± 10.1º compared to 50.3 ± 5.4º, $P < 0.001$. This suggests that the SBO-defect which occurs very early in life causes extra stimulation for the pelvis to grow in order to stabilise the junction. The pelvic incidence maintained a positive linear relationship with sacral slope ($r = 0.5$), lumbar lordosis ($r = 0.4$), and with pelvic tilt ($r = 0.4$), all statistically significant with $P < 0.001$.

Apart from increased PI in those individuals with SBO-defect, the PI-LL+10 mismatch was even more pronounced in those with defect (PI-LL 19.4 ± 10.7º in Group1 against -2.7 ± 7.0º in Group 2), which clearly demonstrate the deforming effect of the defect and failure of compensatory mechanisms. This was confirmed by linear regression which showed that 43% of the total variation in the PI-LL+10 mismatch can be explained by the increased pelvic incidence in those patients with SBO-defect.

Other findings in the study was increased pelvic tilt in individuals with SBO-defect, a value of 23.4º ± 10.3º in those with defect against 11.0 ± 8.0º those without, $P < 0.0001$. The pelvic tilt is a positional parameter that reflects attempts at compensation to spinal deformity. High values of PT express compensatory pelvic retroversion caused by sagittal spinal malalignment (18). The sacral slope maintained a moderate negative linear relationship with pelvic tilt ($r = -0.5$), confirming this relationship. It can be concluded that increased pelvic tilt attempts to stabilize the junction and prevent lumbosacral shear and development of instability.

The sacral slope is the angle of inclination between the horizontal and the sacral plate. The sacral inclination is a critical spinal parameter as increased sacral slope angle has been associated with progression of instability and spondylolisthesis; the higher the sacral slope, the greater the likelihood of spondylolisthesis (16,17). In addition, increased sacral slope creates greater lumbosacral lordosis, which in turn creates higher stress across the par's region (15). In this study this relationship is seen where lumbar lordosis balances with sacral slope angle (Group 1 LL = 29.6 ± 11.3º vs. SS = 34.8 ± 11.0º and in Group 2 LL = 43.0 ± 6.2º vs. SS = 38.4 ± 4.1º, $P = 0.002$). The sacral slope maintained a negative linear relationship with pelvic tilt ($r = -0.5$), since the sacrum is fixed to the pelvis. Abnormal sacral slope will increase lumbosacral shear and progression of instability. In conclusion, individuals with a SBO-defect have decreased sacral slope caused by compensatory pelvic retropulsion. External validation of this study is required. The frequency of instability as a result of these defects also require further studies as the presence of SBO would point to possible instability and alter approach to management.

CONCLUSIONS

The SBO-defect which occurs early in foetal life, appear to cause compensatory pelvic growth with increased pelvic incidence. The pelvis then takes the role of adjusting for sagittal alignment of the lumbar spine by increasing pelvic tilt, reducing sacral slope and lumbar lordosis. This creates a PI-LL+10 mismatch, which can be used as a measure of deformity.

ACKNOWLEDGEMENTS

I wish to acknowledge the dedication of The Mombasa Hospital Radiographers and my study assistant Mr William Shongolo. They made the work easier to do.

Declaration of conflicting interests: There was no conflict of interest of any form.

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