PREVALENCE OF DEVELOPMENTAL LUMBAR SPINAL CANAL STENOSES AMONG ADULT POPULATION IN THE COASTAL REGION OF KENYA

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ABSTRACT

Background: There is considerable variability in the size of the adult lumbar spinal canal between and within populations.

Objective: This study purposed to determine the prevalence of Developmental Lumbar Spinal Canal Stenosis (DLSS) in an impoverished population. Assuming DLSS is part of generalized stunting and therefore, influenced by nutrition, a higher prevalence rate of DLSS was expected in the region. This would explain the severity of symptoms encountered in association with chronic low back pain and radiculopathy.

Design: Observational cross-sectional survey.

Methods: The study was carried out between October 2017 and January 2018. One thousand one hundred and ninety-eight people were recruited and 436 participants were sampled for the study. Basic anthropometric measurements were done. History of hunger and food shortage was taken. Observations were made for presence or absence of enamel hypoplasia and spina bifida. Using axial sections of MRI and CT scans, canal dimensions were measured in each of the 5 lumbar vertebras (L1-L5).

Results: Males constituted 50.3% of the sample population, rest were female. The mean age was 45 years. The mean canal depth was 13.8 ±2.5 mm, width 17.8±3.6 mm, and the mean cross-sectional area was 200 ±70 mm^2. Developmental lumbar spinal canal stenosis was diagnosed when the CSA was less than -2SD. The prevalence rate of DLSS was 19%.

Conclusion: The prevalence of DLSS in the African population living in the Coastal regions of Kenya was found to be 19% and highly related to stunting.

Key words: Prevalence, Poverty**,** malnutrition**,** Developmental, Stenosis

INTRODUCTION

Lumbar spinal canal stenosis is narrowness of the vertebral canal both in its depth and width. Spinal stenosis is defined statistically as a spinal canal less than -2SD of the unit measurement below mean for the population. Developmental spinal stenosis is characterized by pre-existing narrowed spinal canals. This is a developmental aberration that occurs in early life. The depth of the canal (AP diameter) is fully complete by 5 years of age without a chance for catch up (1). It is therefore, vulnerable to early life stresses such as malnutrition which may also present as low birth weight and prematurity. Apart from food shortage due to poverty, other contributors to prenatal and neonatal malnutrition include placenta disorders and maternal factors such as chronic illness, age, parity and drugs and chemicals. Therefore, all these factors may potentially influence spinal canal development, especially the depth (2). On the other hand the width of the spinal canal diameter (transverse diameter) continue to grow until around 17 years (3), giving it room for catch up . Therefore, in a narrow canal, the depth is expected to be relatively more affected.

Developmental spinal stenosis as a diagnosis is not new but has long historical background. In 1954,

Henk Verbiest was the first person in the literature to use the term "developmental narrowing" of the lumbar spinal canal while reporting a series of 28 patients he had managed with radicular syndrome. This was in contradistinction to the previously recorded congenital stenosis which tends to be syndromic (4). Epstein *et al* (5), in 1962 discussed the shape and size of the spinal canal in relation to its narrowness and nerve root compression (5). In 1980, Getty *et al*, in a review of 31 patients from Norfolk and Norwich hospitals encountered 3 cases of what he referred to as "idiopathic developmental spinal stenosis". Postacchini *et al* (6), in 1993 discussed multiple laminotomy as the ideal treatment for developmental stenosis. Cheung *et al* (7), while discussing the paradoxical relationship between the ligamentum flavum hypertrophy and spinal stenosis uses the term developmental lumbar spinal stenosis. Cheung *et al* (8), in a radiological survey of 56 subjects for the best screening method for spinal stenosis again uses the term developmental lumbar spinal stenosis.

In developmental stenosis the pedicles are commonly shortened indicating vertebral hypoplasia, which occurs in the second trimester of pregnancy (9,10). According to Alvarez *et al* (11), the average lumbar spine canal depth (AP or mid-sagittal diameter) is 16-20mm; mean 18mm; while the width (transverse or interpedicular diameter) should be within 22-28mm, mean 25mm. That gives a cross-sectional area of between 276-440mm^3 mean 350mm^3. These measurements are from populations in the developing world that work as for general guideline. Lumbar canal stenosis may cause constrictive neuropathy involving the cauda equina and nerve roots. Patients with lumbar spinal stenosis complain of radiculopathy. Individuals with developmental lumbar canal stenosis become symptomatic early in adult life (30- 50 years), when mild degenerative changes that would otherwise be innocuous are sufficient to cause nervous compression. The condition becomes severer as the canal narrows due to facet joint hypertrophy, syndesmophytes, disc herniation and hypertrophy of the ligamentum flavum as degeneration advances. Kelsey *et al*. (12) in their study on lumbar spine degeneration reported degenerative changes in 95% of their cases.

Lumbar spinal canal stenosis has been shown to be an important contributor to the chronic low back pain syndromes by many authors (5, 13-15). Porter and Ward (16) found a highly significant difference in the canal size of symptomatic individuals when compared to asymptomatic volunteers. On the average, patients requiring surgical intervention have a smaller spinal canal area at the pedicular level compared to asymptomatic individuals. Therefore, the canal size is a crucial determinant of neurological outcome in spine degenerative pathology.

The radiological modality of choice for evaluation of spinal canal stenosis and its pathology is MRI (17). There are many advantages of MRI when compared to other modalities (plain X-rays or ultrasonography) which include: non-invasiveness, non-radiation, high sensitivity and high soft tissue contrast which clearly depicts nervous tissue, ligaments and other paraspinal soft tissues (17,18). The MRI scans allow accurate measurement of the canal dimensions. Similarly, canal measurement in the axial CT scan view is as accurate as the canal borders are well exposed. To determine developmental spinal canal stenosis, measurements are done at the interpedicular level where the canal is spared the degenerative process, that would affect particularly the disc level (19).

The purpose of this study was primarily to determine the prevalence of Developmental Lumbar Spinal Canal Stenosis (DLSS) among adult population in the coastal region of Kenya and secondarily highlight the contribution of this condition in the aetiology of low back pain with or without radiculopathy.

MATERIALS AND METHODS

The hypothesis in this study was that DLSS is part of generalized dystrophic growth and is as a result of early life malnutrition. The study was a cross sectional survey which was conducted between 2nd October 2017 to 13th January 2018 in seven radiological centres. All study participants were voluntary adults who agreed and consented to participate in the study. They all filled a semi structured questionnaire which enquired on their bio data, family history; childhood experiences (particularly sicknesses and lack of food). Required

onsite observations and measurements were done and entered on the same individualized questionnaires. All data was collected simultaneously on the spot.

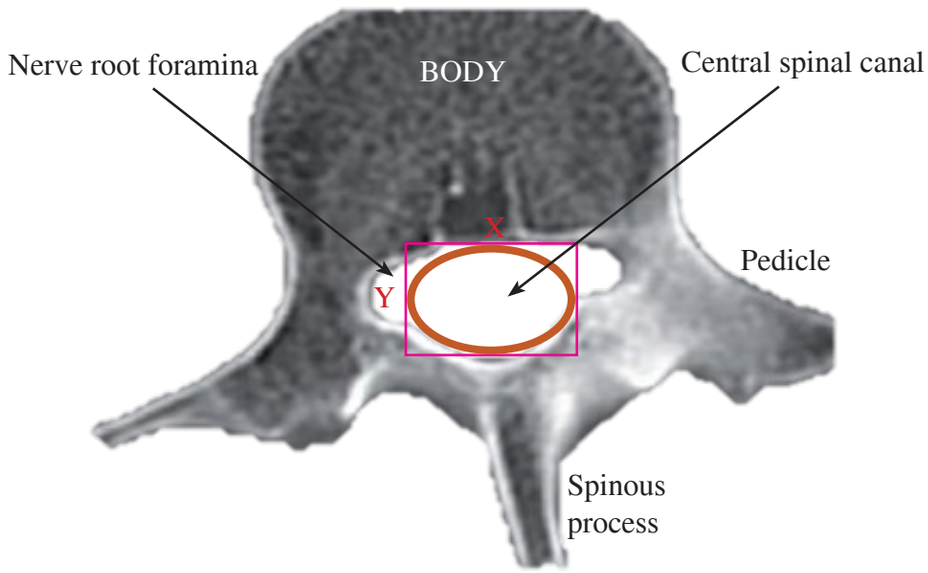
All the participants were adult black Africans who were born and spent their first 5 years of life in the coast region. Black Africans, living in ancestral locations were hoped to share similar genetic material to a large extent. Those not included in the study were people of non-African or of mixed race, those with musculoskeletal disorders such as hip dysplasia or scoliosis, those that were syndromic (dwarfism and achondroplasia), and those with prior spine surgery, spine fractures, infection or tumours. Dysraphism was not excluded but tallied as part of the study.

The bio data of the participant taken included age, gender, marital status, occupation, and level of education. The following measurements were done and recorded in the same questionnaire: height, weight, and head circumference and chest width (measured as in interclavicular distance). Body Mass Index (BMI) was calculated from height and weight.

Lumbar spinal canal dimensions were prospectively measured on ambulatory patients undergoing CT or MRI scans. The scans were done on a Siemens sixteen-slice multi-detector CT scanner or a GE 1.5T MRI scanner (General Electric, USA). Measurements of the spinal canal were done on the axial scans of each lumbar vertebra from LV1 to LV5 at the interpedicular level. The dimensions of the spinal canal are marked anteriorly by the posterior edge of the vertebral body, posteriorly by the anterior edge of the spinous process and laterally by the medial edges of the facets or pedicles (Figure 1).

Figure 1

Illustrates the method of measuring the spinal canal parameters: X and Y lines representing the radii of the canal in orthogonal views. The transverse diameter (X) is longer than the AP diameter (Y). The calculation of the cross-sectional area was calculated using the mathematical formula for calculating the area of an oblong as follows: $A = \pi xy$



Measurements were done using those landmarks. All the measurements were taken, for each segment of the central lumbar canal at the pedicular level. These measurements corresponded to the anteroposterior diameter (APD) to determine the depth of the canal, the Transverse Diameter (TRD) to determine the width of the canal and the Cross-Sectional Area (CSA) which was calculated from the two orthogonal diameters. The measurements of the spinal canal were done on tracings of the central spinal canal in the axial scans each lumbar vertebra at the pedicular level. A rectangle or square was drawn on the edges of the canal tracing (Figure 1), which allowed accurate and consistent measurement of the dimensions, bearing the magnification.

Data analysis: All data was entered into standardized forms on worksheets prepared for raw data entry and subsequently analysed. The data was analysed using worksheets, IBM SPSS version 20.0 (SPSS Inc., Chicago, Illinois, US). Descriptive statistics on IBM

SPSS were used to analyse for frequencies and for central tendency (mean, median and mode) and for dispersion (standard error, standard deviation, range and sample variance). In comparing of proportion chi-square statistics in cross-tabulation was used. When comparing means, for two groups, independent-samples t-test or simply means in SPSS was used; and when comparing means with many levels, One-Way ANOVA in SPSS was used. Odds ratio was used to test for any associations. A P value <0.05 was considered statistically significant.

RESULTS

The study population consisted of all individuals whose spinal canal parameters could be measured in a CT or MRI scan. A total of 436 individuals were sampled out of 1198 individuals. The 436 participants had CT or MRI scans of the lumbosacral spine and were drawn from the indigenous black population of Coastal Kenya (a matched 436 were used to in the larger study to control for the determinant variable, while the remaining 326 were sampled out).

Demographics of the study population: In this sample, various physical characteristics of the participants which represented the individual phenotype were obtained. This included age, gender, height, weight, Body Mass Index (BMI), head circumference, and chest width. Males constituted 50.3% of the sample population, rest were female. The mean age was 43.2 years.

The mean sample height was 169.2± 8.1cm. One hundred eighteen, 118 (27%) were -2SD or less and were considered stunted. The mean sample weight was 74.2 ± 12.3Kg. Twenty-six (6%) were -2SD or less and were considered underweight, while 37 (8.5%) were ≥+2SD and considered obese. The Body Mass Index (BMI) calculated as a ratio of weight in kilograms and the square of height in meters was determined. Normal BMI is 18.5 to 24.9; BMI less than 18.5 is considered underweight, 25 to 29.9 overweight and 30 or more obese. Using this criterion, only 6 participants (1.4%) were underweight while 73 (16.7%) were obese. The mean sample BMI was 26.0 ±4.7. The participants had their head circumferences taken to the nearest 0.1cm. The normal range for adult head circumference is 52cm to 60cm with a mean of 56cm. The mean sample head circumference was 57.2 ± 4.9cm, with 10 (2%) below 52cm and 10(2%) above 60cm.

The chest width was determined by measuring the interclavicular distance in cm, from the tip of lateral end of one clavicle to the tip of the lateral end of the other. This distance is not affected by gain or loss of weight. The mean sample chest breadth was 32.6 ± 2.7cm (range 26 to 45cm). Ten participants, 10 (2.3%) were -2SD or less and were considered thin, while 14 (3.2%) were ≥+2SD and considered stout.

Canal measurements: The sample means were as follows: depth 13.8 ±2.5mm, width 17.8±3.6mm, and cross-sectional area of 200.4 ±70mm^2, Table 1.

Table 1
Canal dimensions

Dimensions	Means	-2SD Cut off	Tally (N = 436)	Prevalence
APD (Depth)	13.8 ± 2.5mm	9mm	83	19%
TRD (Width)	17.8 ± 3.6mm	10.8mm	13	3%
CSA	200.4 ±70mm^2	60mm²	70	16%

Table 1 shows the means of various canal dimensions and the prevalence of developmental lumbar spinal stenosis in the sample population. The dimensions are anteroposterior diameter (APD), Transverse diameter (TRD) and cross-sectional area (CSA).

The prevalence of DLSS in the study sample using APD was 19%. The means of anteroposterior diameters show the canal is largest at LV1 and consistently narrows down to LV4 but deepens again at LV5, (Table 2).

Table 2

The means of anteroposterior diameter from first lumbar vertebra (LV1) to fifth lumbar vertebra (LV5)

N = 436	Mean	Std. Deviation	Std. Error Mean	t	df	Sig. (2-tailed)
LV1	14.2	2.7	.1312	108.043	435	<0.001
LV2	14.1	2.9	.1374	102.717	435	<0.001
LV3	13.7	2.8	.1339	102.456	435	<0.001
LV4	13.5	3.0	.1436	94.205	435	<0.001
LV5	13.8	3.2	.1534	90.142	435	<0.001

Mean 14±2.5mm

Table 2 summarizes the depth or anteroposterior diameter at various levels of the lumbar spine and the statistical significance between them.

Similarly, the means of transverse diameters show the canal is largest at LV1 and consistently narrows down to LV5, (Table 3).

Table 3

The means of transverse diameter from first lumbar vertebra (LV1) to fifth lumbar vertebra (LV5)

N = 436	Mean	Std. Deviation	Std. Error Mean	t	df	Sig. (2-tailed)
LV1	18.7	3.5	.1674	105.786	435	<0.001
LV2	18.2	3.0	.1909	95.119	435	<0.001
LV	18.1	3.9	.1870	96.558	435	<0.001
LV4	17.8	4.2	.2016	88.367	435	<0.001
LV5	17.5	5.1	.2453	71.317	435	<0.001

Mean 18.1±3.6 mm

Table 3 summarizes the transverse diameter at various levels of the lumbar spine and the statistical significance between them.

The means of cross-sectional area confirm narrowing canal from LV1 to LV4/5 (Table 4).

Table 4

The means of cross-sectional area from first lumbar vertebra (LV1) to fifth lumbar vertebra (LV5)

N = 436	Mean	Std. Deviation	Std. Error Mean	t	df	Sig. (2-tailed)
LV1	206.5	71.2	3.4102	59.367	435	<.001
LV2	202.5	75.8	3.6290	56.890	435	<.001
LV3	199.2	71.9	3.4428	57.846	435	<.001
LV4	195.0	76.7	3.6755	53.064	435	<.001
LV5	195.8	93.3	4.4682	44.483	435	

Mean 200±70mm^2

Table 4 summarizes the Cross-Sectional Area at various levels of the lumbar spine and the statistical significance between them.

A cut off for diagnosis of DLSS was minus -2 SD from the mean in each parameter. These cut off figures were 9mm, 10.8mm, and 60mm^2 for depth (APD), width (TRD) and sectional area (CSA) respectively. All the canal measurements were transformed to z scores for accurate comparison and for prognostic grouping. These groups are: normal range, those less than -1SD, less than -2SD, and those less than -3SD (Table 5).

Table 5
Range of measurements according to Z-scores

Z-scores	APD mm	TRD mm	CSA mm²
0	13.8 ±2.5	17.8±3.6	200.4±70
-1	11.5	14.4	130
-2	9	10.8	60
-3	6.5	7.2	-10

Table 5 summarizes the distribution of range of measurement according to the degree of dispersion from the mean. The parameters are anteroposterior diameter (APD), transverse diameter (TRD), and cross-sectional area (CSA), measured in millimetres (mm).

Developmental anomalies associated with DLSS: Various developmental anomalies that are strongly associated with some form of malnutrition were investigated for possible association with developmental spinal stenosis. These were stunting, enamel hypoplasia and spina bifida occulta. The prevalence of these variables in the study population was, 27% for stunting, 67% for Spina Bifida Occulta (SBO), and 16% for enamel hypoplasia. Correlation of these variables with DLSS showed only stunting ($r = -0.104$, $p = 0.030$) and enamel hypoplasia ($r = -0.226$, $p = <0.0001$) correlated negatively but weakly with DLSS (Table 6).

Table 6
Correlation of other developmental anomalies with developmental lumbar spinal stenosis measured as the mean cross-section area (MCSA)

Correlations	MCSA	
	Pearson Correlation Moment	sig.
Enamel hypoplasia	-0.226	<.001
Spina bifida occulta	-.039	.412
Stunting	-0.104	.030

Table 6 shows the Pearson correlation moment between the developmental anomalies and developmental lumbar spinal stenosis.

Secondary outcomes; The disease outcomes associated with congenital narrowness of the lumbar spinal canal are chronic low back pain and symptoms of nervous tissue compression (radiculopathy).

Out of the 84 cases of DLSS, 69 (82%) suffered LBP, compared to 53% of those without DLSS. The difference was statistically significant with chi square in cross tabulation returning a P value = <0.001.

Radiculopathy: Sixty-three per cent, of DLSS cases (53/84) suffered radiculopathy, compared to 21% of those without DLSS. The difference was statistically significant with chi square in cross tabulation returning a P value ≤ 0.001.

DISCUSSION

Chronic low back pain particularly associated with radiculopathy is common symptoms of lumbar spinal canal stenosis. Lumbar spinal canal stenosis in an adult has hitherto been considered acquired condition. Degenerative encroachment of the canal by bony and soft tissues (osteophytes, disc, and hypertrophied ligaments) has been implicated. The study on developmental lumbar spinal stenosis proposes an underlying morphological problem that is further complicated by degeneration. The two conditions cannot easily be separated from one another. Previous studies concur, and confirm that the size of the spinal canal has considerable bearing on the likelihood of nerve root compression (9) with increased need for decompression surgery (19). A population with a high prevalence of DLSS will have high incidences of debilitating low back pain.

In this study there was no gender preponderance; male and female ratio was equal. The mean age for the sample was 43.2 years. The cut off in this study for diagnosis of DLSS was -2 SD (APD <9mm, TRD <10.8 mm and CSA<60 mm^2). Ullrich *et al* (20) in a US-based study, suggested a CSA value of <145 mm^2 as a measure of 'developmental stenosis' at L3. Griffith *et al* (21) findings were a mid-vertebral spinal canal CSA of <212 mm^2 in males and <213 mm^2 in females and which to them placed a patient in the 25% quartile of the population. A systematic review done in 2011 identified APD of less than 10mm and CSA of the spinal canal less than 90 mm^2 as cut off values for diagnosis of lumbar spinal canal stenosis (22). A study conducted in 2012 (referred to as Delphi Study) (23) found that there are no standard quantitative criteria for defining anatomic lumbar spinal canal stenosis on imaging but a multispecialty joint committee formed in 2014 revised lumbar spine nomenclature and recommended grading of the spinal canal stenosis as mild, moderate, or severe.

The canal means were 13.8mm for depth, 17.8mm for transverse diameter and 200.4mm^2 for cross sectional area. Alvarez *et al* (11), studying a Caucasian population reported average lumbar spinal canal depth to be 16-20mm with a mean of 18mm, and the width to be 22–28 (mean = 25mm) yielding a cross sectional area of 276-440mm^2 (mean = 350mm^2). Using these values as standard guidelines, the values in this study are 78%,

72%, and 55% for (APD, TRD, and CSA respectively) of the normal; which makes them lie between 25th to 50th quartile. The population under study has narrower canals than reported in other populations. This agrees with the recommendation that geographic, racial and gender differences in developmental size of the spinal canal do exist so that each region, race and gender should have its own reference range (19, 21).

The overall prevalence of DLSS in this population is 19%. The value is much higher than reported in other studies. Schroeder *et al* (24) reported a prevalence of developmental lumbar stenosis of 9.3% in the American population. Kalichman *et al* (25) in the Framingham study concluded that the prevalence of "congenital stenosis" was 7.3%. Both authors studied populations in the developed world.

There was a significant correlation between DLSS, stunting and enamel hypoplasia. The prevalence of stunting was 27% in the study population. When the individuals with DLSS were analysed for stunting, 57% were found to be stunted against 25% of those without DLSS ($p = 0.0413$). DLSS was also found to have a moderate negative linear relationship with stunting ($r = -0.104$, $p = 0.030$). Stunting is defined as failure of linear growth of long bones. DLSS appear to be caused by failure of growth of short bones; failure of elongation of pedicles which remain shortened causing shallow depth of the spinal canal. The reason for this is not clear but may be related to inadequate nutrition. The insult occurs early in the second trimester of pregnancy (9). Most of the growth of the body and neural arches occur in the neurocentral synchondrosis. Jeffrey *et al* (2) reported reduced midsagittal diameter and cross sectional area in low birth weight babies. The conclusion in this study is DLSS is part and parcel of generalized stunting and in the majority of cases is due to inadequate nutrition.

Spina bifida is a developmental defect of the posterior elements of the lumbar spine where the spinous processes and part of the lamina fail to develop. In this study, the prevalence of spina bifida was found to be 67% in the study population. Urrutia *et al* (26) in a study in Chile found the prevalence of spina bifida occulta to be about 41.2%. Spina bifida which is the milder form of neurotube defect is caused by micronutrient deficiency of folic acid and occurs very early in the embryonic life.

Adult teeth may reflect early childhood nutritional experience. The enamel on the permanent teeth ossifies continuously during early childhood and therefore, any interference in that process will produce defects. Most defects occur in the enamel in childhood due to malnutrition and other stresses. These defects never repair and are visible in adult teeth as pits and clefts in sharp teeth. Enamel hypoplasia could therefore be considered a biomarker of early childhood nutritional status.

This study showed prevalence rate of enamel hypoplasia of 16%. However, those individuals with DLSS showed a frequency of 6% compared to those without (20%) P=0.0001. There was a weak negative linear correlation ($r = -0.226$, $p \leq 0.0001$). Both DLSS and EH appear occur at different periods in early life. DLSS occur as a result of prenatal stress (2) while enamel hypoplasia occur in infancy. This may explain the lack of relationship between DLSS and EH as prenatal malnutrition maybe corrected during neonatal period, for example through breastfeeding.

CONCLUSION

The prevalence of DLSS in the study population was 19%. Developmental lumbar spinal stenosis is associated with increased morbidity and disability due to low back pain and radiculopathy. Finally, DLSS will be diagnosed if the APD is less than 9mm, TRD of less than 10.8mm or a CSA of less than 60mm^2 in the population under study and perhaps, in other impoverished populations.

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