Lumbar Nerve Root Occupancy in the Foramen in Achondroplasia

A Morphometric Analysis

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Abstract Lumbar stenosis is common in patients with achondroplasia because of narrowing of the neural canal. However, it is unclear what causes stenosis, narrowing of the central canal or foramina. We performed a morphometric analysis of the lumbar nerve roots and intervertebral foramen in 17 patients (170 nerve roots and foramina) with achondroplasia (eight symptomatic, nine asymptomatic) and compared the data with that from 20 (200 nerve roots and foramina) asymptomatic patients without achondroplasia presenting with low back pain without neurologic symptoms. The measurements were made on left and right parasagittal MRI scans of the lumbar spine. The foramen area and root area were reduced at all levels from L1 to L5 between the patients with achondroplasia (Groups I and II) and the nonachondroplasia group (Group III). The percentage of nerve root occupancy in the foramen between Group I and Group II as compared with the patients without achondroplasia was similar or lower. This implied the lumbar nerve root size in patients with achondroplasia was smaller than that of the normal population and thus there is no effective nerve root compression. Symptoms of lumbar stenosis in achondroplasia may be arising from the

central canal secondary to degenerative disc disease rather than a true foraminal stenosis.

Level of Evidence: Level I, prognostic study. See the Guidelines for Authors for a complete description of levels of evidence.

Introduction

Spinal stenosis is reported in 32.1% of patients with achondroplasia [11]. The symptoms in the adult population reflect compressive radiculopathy resulting directly from stenosis of the spinal canal [9]. The spinal canal in achondroplasia is narrowed by shortened pedicles and decreased interpedicle distance attributable to a defect in the endochondral ossification leading to early fusion of the pedicles. Numerous studies document reduction in the entire spinal canal area, caudal tapering, and stenosis of the foramina and the effective area for the neural elements [9, 14, 16, 17]. Spinal stenosis is not a problem before late adolescence [6, 10], but there may be exceptions [5]. As the patient with achondroplasia ages, the stenosis is aggravated by hypertrophy of the ligamentum flavum and degenerative spondylosis [4, 11].

Foraminal stenosis can occur in a canal with average dimensions or with sagittal stenosis [13]. Wynne-Davies et al. [15] postulated there was an associated deficiency in lumbar nerve root development. The shape and dimensions of the foramen are affected by the constitutional variations or degenerative changes [15]. Degeneration of the disc reportedly influences the foraminal height but has no important effect on the sagittal dimensions, which are strictly related to the sagittal diameter of the foramen and length of the pedicle. Cinotti et al. [3] also suggested, in developmental stenosis of the central canal, a foraminal

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stenosis is likely present. This implies an increased incidence of foraminal stenosis in patients with achondroplasia. However, the senior author (SWS) observed the size of the nerve roots was small in patients with achondroplasia compared with the size in patients without achondroplasia. Degenerative changes observed in the foraminal canal in the patients with achondroplasia did not seem markedly different from those in patients without achondroplasia. This observation led us to question the role of the relatively small size of the nerve root in development of symptoms of lumbar stenosis in achondroplasia.

We postulated symptomatic lumbar stenosis in achondroplasia is primarily a central stenosis secondary to degenerative spondylosis rather than nerve root compression attributable to foraminal stenosis because (1) the areas of the foraminal canal and nerve root in the foramina of patients with achondroplasia are smaller than those of patients without achondroplasia and (2) nerve root occupancy in the foraminal canal of patients with achondroplasia is not more than that of patients without achondroplasia.

Materials and Methods

We prospectively followed 23 randomly selected patients with achondroplasia who presented to our outpatient clinic between June 2006 and January 2007. We excluded six of the 23 patients who had thoracolumbar kyphosis greater than 60° and scoliosis greater than 10° because these deformities would influence the measurements. Thus, of the 17 remaining patients for the study, eight were symptomatic (Group I, consisting of five males and three females with symptoms and signs suggestive of lumbar stenosis) and nine had no symptoms referable to the spine (Group II, consisting of two males and seven females who presented for other limb disorders). We considered patients symptomatic [1, 14] if they had the following complaints consistent with stenosis: (1) radicular pain in one or both lower limbs; (2) tingling and/or numbness in the affected limb; and (3) neurogenic claudication. Patients who did not have at least two of the previously mentioned symptoms were excluded from Group I. For Group II, patients could have none of the above-mentioned symptoms. The average age of the symptomatic group was greater ($p = 0.012$) than that of the asymptomatic group (35.6 years [range, 25–47 years] versus 24.2 years [range, 13–43 years], respectively). The control group (Group III) consisted of 20 patients without achondroplasia (11 males, nine females) with an average age of 35.9 years (range, 18–46 years) who presented with backache without any neurologic symptoms. We excluded five control patients whose MR images showed evidence of disc degeneration or facet arthropathy. We measured 170 nerve roots and foramina in

the achondroplasia groups and 200 nerve roots and foramina in the nonachondroplasia group.

Magnetic resonance imaging of the lumbar spine was performed using a 1.5-T scanner (Sonata; Siemens Medical System, Erlangen, Germany) with pulsed sequence: turbo-spin-echo and TR (repetition time) 3150 and TE (echo time) 100 and acquisition time of 3 minutes. T1- and T2-weighted 2-mm sagittal cuts were taken during the study. The T2-weighted parasagittal images on the right and left sides were analyzed as part of this study.

We (HNM) made measurements as described by Attias et al. [2]. The vertical dimension (height) was measured as the distance between the inferior margin of the upper pedicle and the superior margin of the lower pedicle for the foramen dimensions (Fig. 1). The root vertical dimensions were measured as the distance between the upper and lower borders of the root sleeve (Fig. 2). The sagittal dimension (width) was the largest anteroposterior width measured in the same horizontal plane (Figs. 1, 2). The measurements were performed by an independent spine surgeon (HNM) using a digital software program (Piview StarTM; INFINT Technology Co, Ltd, Seoul, Korea). The area was calculated for the intervertebral foramen and exiting nerve root at all the levels from L1 to L5 according to the formula ($\frac{1}{4} \times \pi \times \text{height} \times \text{width}$), not according to the formula ($\text{height} \times \text{width}$) described by Yukawa et al. [17], because the foraminal canal and nerve root areas are oval. The percentage occupancy of the individual roots at L1 to L5 levels with respect to the area of the foramen was calculated in all three groups. This was calculated as ($\text{root area} / \text{foramen area}$) $\times 100$.

We compared the mean cross-sectional areas of the neural foramen and the nerve root and nerve root occupancy in the foramina among the asymptomatic group, symptomatic group, and control group, using Tukey's honestly significant differences (HSD) post hoc test. The results of statistical analysis also provided the rank of foraminal area, nerve root area, and nerve root occupancy among all groups in decreasing order. All analyses were performed using the SPSS software package (Version 12.0; SPSS Inc, Chicago, IL).

Results

Patients with achondroplasia had smaller ($p < 0.0001$) nerve roots than patients without achondroplasia at all lumbar levels. Similarly, at all lumbar levels the foraminal area was decreased ($p < 0.0001$) in patients with achondroplasia when compared with that in the control group. The cross-sectional area of the intervertebral foramen in the symptomatic group progressively increased from L1 to L4 and again decreased at L5. In the asymptomatic group,

Fig. 1A–B Measurement of the foraminal area in (A) the achondroplasia groups and (B) nonachondroplasia group is illustrated. Measurement of foraminal area was performed by measuring the height from the lower border of the upper pedicle to the upper border of lower pedicle and the width from the posterior border of the vertebral body to the anterior border of the lamina at the level of the maximum width of the foramen. The foraminal area was calculated using the formula ($\frac{1}{4} \times \pi \times \text{height} \times \text{width}$) in square millimeters.

Fig. 2A–B Measurement of the nerve root area in (A) the achondroplasia groups and (B) nonachondroplasia group is illustrated. Measurement of the nerve root area was performed by measuring the height from the upper border of the nerve root to the lower border of the nerve root and the width from the anterior border of the nerve root to the posterior border of the nerve root at the level of the maximum width of the nerve root. The nerve root area was calculated using the formula ($\frac{1}{4} \times \pi \times \text{height} \times \text{width}$) in square millimeters.

the foraminal area increased from L1 to L3 and decreased at L4 and L5. In the control group, similarly, it increased from L1 to L3 and then decreased at L5. A similar pattern was observed on the right side (Table 1). The cross-sectional area of the exiting nerve root showed a progressive increase from L1 to L5 in the symptomatic and the asymptomatic groups. The pattern was similar in the control group (Table 2). The root occupancy varied from 37% at L1 to 51% at L5 in Groups I and II. In Group III, it varied from 47% at L1 to 54% at L5. This pattern was similar on the left and right sides (Table 3). The occupancy of the nerve root in foramina in patients with

achondroplasia (Groups I and II) was similar or lower than that in patients without achondroplasia (Group III) (Table 3). Post hoc analysis showed the size of the nerve root and foraminal diameter in Group III, Group II, and Group I in decreasing order.

Nerve root occupancy in patients with achondroplasia was similar or lower than that in patients without achondroplasia (p values at each lumbar level shown in Table 3). Post hoc analysis showed Group III had similar or higher nerve root occupancy than Group II and a higher occupancy than Group I, whereas Group II had a similar or higher occupancy than Group I.

Table 1. Comparison of the foraminal areas (sq mm) among the three groups

Lumbar Level	Left				Right			
	Group I (n = 8)	Group II (n = 9)	Group III (n = 20)	p Value ^a	Group I (n = 8)	Group II (n = 9)	Group III (n = 20)	p Value ^a
L1	160.51 ± 22.62 ^b	153.26 ± 9.39 ^b	223.44 ± 16.50 ^a	< 0.0001	158.24 ± 23.22 ^b	151.27 ± 8.56 ^b	224.29 ± 16.46 ^a	< 0.0001
L2	178.01 ± 16.12 ^b	170.53 ± 10.41 ^b	253.17 ± 21.62 ^a	< 0.0001	160.94 ± 20.08 ^b	167.05 ± 14.09 ^b	251.51 ± 22.72 ^a	< 0.0001
L3	189.15 ± 21.24 ^b	193.87 ± 14.63 ^b	269.35 ± 18.24 ^a	< 0.0001	186.17 ± 14.59 ^b	189.29 ± 13.14 ^b	269.25 ± 17.45 ^a	< 0.0001
L4	195.08 ± 11.31 ^b	186.27 ± 21.05 ^b	242.61 ± 8.06 ^a	< 0.0001	192.55 ± 13.44 ^b	186.94 ± 21.98 ^b	239.64 ± 8.17 ^a	< 0.0001
L5	184.98 ± 8.51 ^b	186.28 ± 16.38 ^b	236.98 ± 5.78 ^a	< 0.0001	183.68 ± 7.91 ^b	182.57 ± 14.10 ^b	231.80 ± 6.74 ^a	< 0.0001

Values are expressed as mean ± standard deviation; ^a p values are for Tukey's HSD post hoc test; ^{a/b} suggests the order of post hoc analysis in decreasing manner when foraminal areas are compared among Groups I, II, and III, which shows Group III has more foraminal area than Groups I and II whereas the foraminal areas for Groups I and II are similar; the left-sided p value is for the left foramen for a particular level and the right-sided p value is for the right foramen for a particular level.

Table 2. Comparison of the nerve root area (sq mm) among the three groups

Lumbar Level	Left				Right			
	Group I (n = 8)	Group II (n = 9)	Group III (n = 20)	p Value ^a	Group I (n = 8)	Group II (n = 9)	Group III (n = 20)	p Value ^a
L1	60.52 ± 10.12 ^b	58.62 ± 14.30 ^b	104.62 ± 7.90 ^a	< 0.0001	60.52 ± 12.21 ^b	58.66 ± 14.33 ^b	105.55 ± 7.46 ^a	< 0.0001
L2	68.94 ± 8.97 ^b	76.10 ± 10.50 ^b	113.99 ± 11.27 ^a	< 0.0001	64.44 ± 10.67 ^b	76.20 ± 11.11 ^b	112.69 ± 10.86 ^a	< 0.0001
L3	68.92 ± 11.33 ^b	78.18 ± 17.30 ^b	114.01 ± 9.00 ^a	< 0.0001	67.49 ± 13.12 ^b	78.86 ± 16.69 ^b	114.87 ± 9.00 ^a	< 0.0001
L4	68.37 ± 11.17 ^b	86.81 ± 18.84 ^b	119.27 ± 12.01 ^a	< 0.0001	67.59 ± 8.86 ^b	86.19 ± 18.55 ^b	119.39 ± 12.07 ^a	< 0.0001
L5	82.99 ± 11.70 ^b	95.46 ± 17.62 ^b	126.64 ± 12.82 ^a	< 0.0001	81.11 ± 9.97 ^b	94.27 ± 18.61 ^b	126.32 ± 12.44 ^a	< 0.0001

Values are expressed as mean ± standard deviation; ^a p values are for Tukey's HSD post hoc test; ^{a/b} suggests the order of post hoc analysis in decreasing manner when nerve root areas are compared among Groups I, II, and III, which shows Group III has more root area than Groups I and II whereas the root areas for Groups I and II are similar; the left-sided p value is for the left root for a particular level and the right-sided p value is for the right root for a particular level.

Table 3. Comparison of the nerve root occupancy (%) among the three groups

Lumbar level	Left				Right			
	Group I (n = 8)	Group II (n = 9)	Group III (n = 20)	p Value*	Group I (n = 8)	Group II (n = 9)	Group III (n = 20)	p Value*
L1	37.65 ± 3.31 ^b	37.87 ± 7.19 ^b	46.82 ± 0.61 ^a	< 0.0001	37.99 ± 2.65 ^b	38.43 ± 7.34 ^b	47.07 ± 0.66 ^a	< 0.0001
L2	38.61 ± 1.85 ^b	44.46 ± 3.84 ^a	44.98 ± 0.86 ^a	< 0.0001	40.68 ± 3.37 ^b	45.43 ± 3.30 ^a	44.79 ± 0.96 ^a	0.0002
L3	36.27 ± 2.54 ^b	39.94 ± 6.17 ^a	42.30 ± 0.70 ^a	0.0004	35.99 ± 4.41 ^b	41.33 ± 6.18 ^a	42.63 ± 0.92 ^a	0.0005
L4	34.85 ± 3.88 ^b	46.12 ± 5.32 ^a	48.90 ± 3.46 ^a	< 0.0001	34.98 ± 2.51 ^c	45.64 ± 4.71 ^b	49.71 ± 3.45 ^a	< 0.0001
L5	44.93 ± 4.56 ^b	50.86 ± 5.44 ^a	53.34 ± 4.15 ^a	0.0005	44.12 ± 3.74 ^b	51.27 ± 6.71 ^a	54.40 ± 3.98 ^a	< 0.0001

Values are expressed as mean ± standard deviation; *p values are for Tukey's HSD post hoc test; ^{a/b/c} suggests the order of post hoc analysis in decreasing manner when nerve root occupancies are compared among Groups I, II, and III, which shows Group III has similar or higher nerve root occupancy than Group II and higher occupancy than Group I whereas Group II has similar or higher nerve root occupancy than Group I; the left-sided p value is for the left nerve root occupancy in the foramen for a particular level and the right-sided p value is for the right nerve root occupancy in the foramen for a particular level.

Discussion

Although spinal stenosis reportedly occurs with high frequency in patients with achondroplasia [11], we have observed few differences in degeneration of the foraminal canal in patients with achondroplasia compared with patients without achondroplasia. This observation led us to question the role of the relatively small size of the nerve root in development of symptoms of lumbar stenosis in patients with achondroplasia. Nerve root occupancy in the foramen is a measure of nerve root compression; if nerve root occupancy is more, nerve root compression will be more. Therefore, low nerve root occupancy indicates no major nerve root compression. We postulated symptomatic lumbar stenosis in patients with achondroplasia is primarily a central stenosis secondary to degenerative spondylosis rather than nerve root compression attributable to foraminal stenosis because (1) the areas of the foraminal canal and nerve root in the foramina of patients with achondroplasia are smaller than those of patients without achondroplasia and (2) nerve root occupancy in the foraminal canal of patients with achondroplasia is not more than that of patients without achondroplasia.

The major shortcoming of our study was the method of measurement of foraminal or nerve root size. Actually, the foramen and nerve root are oval because of obliquity of the canal, but they have been measured based on rectangular area [2, 17]. However, we measured them as an oval area with the formula $\frac{1}{4} \times \pi \times \text{height} \times \text{width}$. Second, we compared nerve root occupancy of symptomatic patients with achondroplasia with asymptomatic patients without achondroplasia, not symptomatic patients. We believed asymptomatic patients had the lowest nerve root occupancy in foramina; that is, if patients with achondroplasia had lower nerve root occupancy in the foramina than the asymptomatic control patients, they would definitely have lower occupancy than patients in the symptomatic group with achondroplasia. Our findings suggest this hypothesis may be true.

Achondroplasia is a relatively common genetic disorder with an incidence of one per 13,500 live births and is associated with various abnormalities of the musculoskeletal system, including developmental cervical and lumbar canal stenosis resulting from a postnatal defect in the development of the vertebrae [16]. The cause of a developmentally narrow lumbar canal in patients with achondroplasia is described as a growth disturbance and premature fusion of the posterior elements of the spine [7, 14]. The narrow spinal canal puts the spinal cord at risk for compression, which has been reported [10, 12]. The spinal canal, as measured by the interpedicle distance, normally widens as one proceeds distally in the lumbar spine from L1 to L5. However, in patients with achondroplasia, the

spinal canal narrows and the interpedicle distance decreases.

No reports have mentioned the foraminal narrowing; instead, all commented on only the central canal. Lutter et al. [7] reported the decrease in the volume of the lateral recess and intervertebral foramina resulted in a reduced area for the dural sac and exiting spinal nerves. Nerve root size was not commented on in their study, and therefore, whether narrowing of the foramina is responsible for nerve root compression is not clear. Hypertrophy of the ligamentum flavum and degenerative spondylotic changes aggravate the preexistent stenosis. The sagittal imbalance of the spine, seen in 10% of patients with achondroplasia [8], may further accelerate the degenerative process. However, whether this preexisting stenosis is central or foraminal, we cannot know without measuring nerve root diameter. Lutter et al. [7] reported a reduction in the volume of the spinal canal, with the changes more pronounced at the intervertebral disc level [7]. Their data suggested the nerve root canals in asymptomatic patients with achondroplasia are markedly narrowed and smaller in the anteroposterior direction and, in addition, the pedicles are thicker and shorter. Their study suggests narrowing of the foraminal area did not cause nerve root compression and, therefore, foraminal stenosis is not the factor for development of symptoms in such patients. The changes in the spine resulting from degenerative changes reduce the area of the canal further. Wynne-Davies et al. [15] suggested there might be a deficiency in the nerve root, but it was not studied further. Previous studies in subjects without achondroplasia have shown the area of the nerve root is 10% to 30% of the area of the foramen. The small size of the root, which was observed consistently as compared with the patients without achondroplasia, intrigued us in the current study.

We observed lower or similar nerve root occupancy in patients with achondroplasia than in patients without achondroplasia, which confirms our hypothesis that in patients with achondroplasia, lumbar stenosis is not the result of foraminal stenosis. Our findings confirm those in previous studies describing a smaller foraminal canal in patients with achondroplasia than in patients without achondroplasia. In addition, the size of the nerve root was smaller than that of the control group. This suggests the percentage occupancy of the root in the foramen between the patients with achondroplasia and the patients without achondroplasia was similar or lower and there was no effective true foraminal stenosis (Table 3).

The percentage occupancy was similar or lower in the achondroplasia group than in the nonachondroplasia group, suggesting in achondroplasia the nerve root size also was decreased, corresponding to a decrease in the foraminal dimensions. Thus, the reserve space available for the nerve root in the foramen is no different from the space available in

a normal lumbar foraminal canal. The osteoligamentous encroachment of the foramen attributable to the degenerative process affects the decrease in the foraminal dimensions in a similar way in patients without achondroplasia and patients with achondroplasia. The emphasis on the compromise of normal roots in an abnormally narrow canal in patients with achondroplasia was not found based on our observations. This suggests the symptoms most likely originate from the central canal secondary to degenerative changes in a developmentally compromised central canal rather than a true foraminal stenosis resulting from the decreased foramen area and narrowed root canal. We believe this study should be followed by larger studies to establish guidelines for diagnosing, quantifying, and treating lumbar spinal stenosis in patients with achondroplasia.

We found symptomatic lumbar stenosis in patients with achondroplasia is more of a central stenosis than a foraminal stenosis as previously reported. The degenerative disc narrowing and facet hypertrophy with osteophyte formation further reduce the size of an already developmentally compromised intervertebral foramen. The small size of the nerve roots with respect to the decreased area of the intervertebral foramen seen in patients with achondroplasia precludes development of foraminal stenosis in all affected populations. Furthermore, the small size of the roots, in addition to the absence of or scanty epidural fat, makes them vulnerable to iatrogenic injury during surgical decompression.

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