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Chronic low back pain – its association with lumbar spinal canal diameter – a cross-sectional hospital-based study in a tertiary care hospital in North Bengal

Mahadeb Mandal[1], Durga Prasad Chakraborty[2], Aniruddha Chattopadhyay[3], Pulakesh Sarmah[4], Nirmal Kumar Bera[5]

[1]Junior Resident, [5]Professor and Head, Department of Psychiatry, [2]Associate Professor and Head, Department of Neuromedicine, North Bengal Medical College, Darjeeling, [3]Assistant Professor, Department of Neurosurgery, Institute of Post-Graduate Medical Education and Research and Bangur Institute of Neurosciences, Kolkata, West Bengal, [4]Senior Resident, Department of Psychiatry, Lokopriya Gopinath Bordoloi Regional Institute of Mental Health, Tezpur, Assam, India

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ABSTRACT

Background: Low back pain (LBP) is a very common cause of disability in working age adults, lumbar canal stenosis (LCS) being one of the chief anatomical correlates. However, number of studies seeking role of LCS in the origin of LBP are not many, particularly in India. **Aims and Objectives:** The aims of this study were to investigate LBP and its relation to lumbar canal diameter (LCD), also to evaluate the impact of psychosomatic factors on LBP. **Materials and Methods:** Eighty symptomatic (LBP) and 41 asymptomatic (without LBP) subjects were chosen and LCD for three lowest lumbar segments measured by 1.5 Tesla magnetic resonance imaging for both symptomatic and asymptomatic subjects was ascertained. Canal diameters of both groups were compared by Pearson's Chi-square test. Pain intensity of patients was assessed using visual analog scale (VAS)-pain score. Using tests of normality and non-parametric test Spearman's rank coefficient, correlation between VAS score (pain intensity) and lowest canal diameter of the cases was evaluated. To evaluate the role of psychosomatic factors in LBP, the number of subjects (LBP) with somatic symptoms disorder (SSD) score ≥8 was ascertained. **Results:** The results were as follows: (1) Significant association between presence of LCS (diameter <10 mm) and LBP (P=0.015). (2) No significant correlation between intensity of LBP (VAS score) and LCD. (3) 13.75% of LBP patients had SSD (Male 8% and Female 23.33%). **Conclusion:** LCS may be an important factor in the origin of LBP. More studies are needed in this regard and also seeking correlation between LBP and other anatomical factors. Psychosocial factors may play important role in the origin and maintenance of LBP.

Key words: Low back pain; Lumbar canal diameter; Lumbar canal stenosis; Somatic symptom disorder; Visual analog scale pain score

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INTRODUCTION

Degenerative spinal disease resulting in low back pain (LBP) is one of the most common causes of disability in working age adults. Lumbar spine is the most common location followed by cervical spine.[1] Lumbar canal stenosis (LCS), first described in 1954, is a chief anatomical correlate of LBP.[2] However, studies seeking the role of LCS in the origin of LBP are not many in number,[3] particularly in India. Natural history of LCS in any individual is also unpredictable.[4]

LBP originating from degenerative lumbar spine is very common[5] and magnetic resonance imaging (MRI) has

Address for Correspondence:
Dr. Mahadeb Mandal, Junior Resident, Department of Psychiatry, North Bengal Medical College, Sushrutanagar, Darjeeling - 734 012, West Bengal, India. **Mobile:** +91-8981145616. **E-mail:** mahadeb007@gmail.com

become the investigation of choice in the evaluation of LBP and LS degenerative disease.[6] LCS may be primary (due to disorder of development)[7] or secondary (acquired) stenosis – most commonly from degenerative changes or consequent to infection, trauma, etc. Predominant sites of degenerative LS disease are lower three levels of lumbar spine,[8] the focus of our present study.

The natural history of LCS is also still obscure with studies reporting half of the patients remaining stable and a quarter each improve or worsen.[9] In the origin of LBP, apart from anatomic factors such as degenerative LCS, intervertebral disk degeneration (DD), and facet arthropathy all have been stated important, but studies contradict somewhat on their relative importance.[10] Apart from the known anatomic factors related to LBP, one interrelated factor is the psychosocial one as evident in some study.[11]

Aims and objectives

The aims of our present study were to explore the sociodemographic profile of LBP patients, to explore the association between LCS and presence of LBP, to explore the quantitative relation between the lumbar canal diameter (LCD) and intensity of the LBP, and to have a measure of psychosomatic abnormality in our study population.

MATERIALS AND METHODS

A hospital-based analytical cross-sectional study was conducted in North Bengal Medical College and Hospital (NMBCH) from July 2021 to December, 2021. It involved 80 patients with chronic LBP (duration more than 12 weeks) and 41 comparison subjects without LBP who presented with other symptoms to the Neuromedicine Outpatient Department, North Bengal Medical College and Hospital (NBMCH). Patients with chronic LBP, 20 years and above, and willing to give informed consent were included in the study. Patients with history of operation, infection (e.g., TB), trauma, malignancy, or non-infective inflammatory disease (e.g., Ankylosing Spondylosis) in the lumbar region were excluded from the study.

Ethical approval to conduct the study was taken from the Institutional Ethics Committee. Informed consent was taken from all participants. The clinicodemographic data of patients and comparison subjects were recorded using a pro forma (structured questionnaire). Visual Analog Scale (VAS) pain scale was used to assess the intensity of LBP for each patient. A simple form of VAS, understandable to patients, was chosen, with a horizontal straight line of 100 mm length, divided into ten segments, each 10 mm long. Extreme limits – 0 measured no pain and 10th segment (100 mm) means worst pain.[12] Each patient was asked to give score for the most intense pain experienced.

1.5 Tesla MRI of LS spine was done for each of the 80 patients and 41 comparison subjects with particular emphasis to the anteroposterior (AP) diameter of lumbar canal of lower three segments. The least canal diameter of these three was chosen. The cut-off diameter (AP) for LCS taken was 10 mm (i.e., <10 mm considered as LCS) (Youmans Neurological surgery, 6th ed, 2011, Vol. 3).

Within the patient group, number of patients having somatic symptom scale-8 (SSS-8) scores were ascertained. SSS-8 is a brief measure for assessing the somatic symptoms burden which includes eight items, each scored 0–4. Total score being 32, 8, or above is positive for somatic symptoms disorder (SSD). Convenience sampling was used.

Statistical analysis

Descriptive statistics were performed for the calculation of absolute frequencies, percentages and measures of central tendency. Normality was checked using Kolmogorov–Smirnov and Shapiro–wilk tests. Pearson's Chi-square test was used to see the association between the two groups (patient and comparison subjects) regarding canal stenosis and P-value was determined. Non-parametric test Spearman's rho was used to assess the level of association between non-normally distributed VAS score and Lowest Canal Diameter. These data analysis was performed using the SPSS (Statistical Package for the Social Sciences) version 25 and $P<0.05$ was considered statistically significant.

RESULTS

Figure 1 shows the flow chart of the study. There are 80 cases of chronic LBP and 41 comparison subjects. Out of 80 cases, 50 (62.5%) are male, while the rest 30 (37.5%) are female. Similarly in the comparison subjects, 29 (70.7%) are male, while the rest 12 (29.3%) are female. The age distribution of cases ranges from 21 years to 75 years with

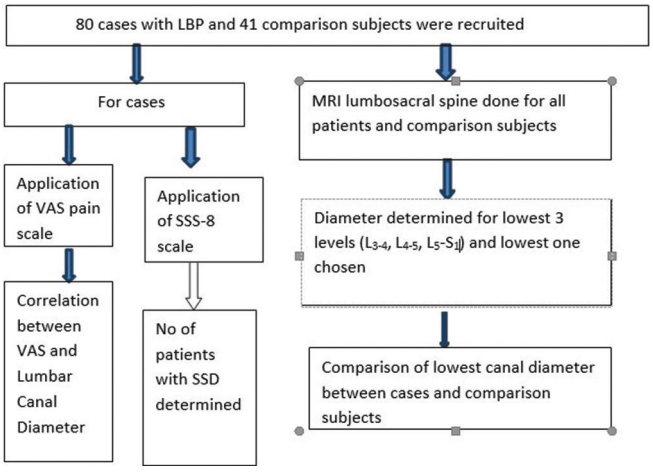

Figure 1: Flow diagram of the study

the median age of 42 years. The ages of the comparison subjects range from 16 years to 63 years with the median age of 40 years. Among the cases, the duration of LBP ranges from 90 days to 2920 days (8 years). The mean duration of chronic LBP is 281 days (Table 1).

LCD was measured with MRI and the least of the lower three levels of lumbosacral spine was considered. Among the cases, stenosis (i.e., lowest diameter <10 mm) was present in 34 (42.5%), while it was absent in 46 (57.5%). Among the comparison subjects, stenosis was present in 8 (19.5%), while it was absent in 33 (80.5%). Pearson's Chi-square test was applied and P=0.015, which implies significant difference between cases and comparison subjects as to the presence of stenosis (Table 2).

Pain intensity among the cases was measured using the VAS pain scale, the scores being distributed non-normally. Spearman's rho Correlation Coefficient was used to see the presence of correlation, if any, of VAS scores with the LCD. P=0.458 shows that no such correlation exists between pain intensity and canal diameter (Table 3).

Table 1: Characterization of cases with LBP (n=80) and comparison subjects (n=41)

Variables	n (%)
Cases	
Male	50 (62.5)
Female	30 (37.5)
Comparison subjects	
Male	29 (70.7)
Female	12 (29.3)
Age (Median) in years	
Cases	42 (21–75)
Comparison subjects	40 (16–63)
Mean Duration of LBP of patients in days	281 (90–2920)

LBP: Low back pain

Table 2: Association between low back pain and presence of canal stenosis

Stenosis	Cases	Comparison group	Pearson's Chi-square	P-value
Present	34	8		
Absent	46	33	6.321	0.015
Total	80	41		

Table 3: Correlation between VAS scores and Lowest Canal Diameter

Variables	N	1	2	P-value*
VAS Scores	80	-	-0.084	
Lowest canal diameter	80	-0.084	-	0.458

*Spearman's rho, Correlation is not significant at the 0.01 level (two-tailed), VAS: Visual analog scale

Out of 80 cases (M=50 and F=30), 11 cases (M=4 and F=7) fulfilled the criteria of SSD assessed by SSS-8. Thus, 13.75% of cases had SSD. Furthermore, out of 50 male patients 4 (8.0%) and out of 30 female patients 7 (23.33%) had SSD.

DISCUSSION

LBP remains among the greatest concerns for public health system as shown in a European study.[13] A number of MRI identified lesions, for example, canal stenosis, facet arthropathy, and disk protrusion/degeneration are associated with LBP.

LBP, particularly chronic LBP, is a mixed pain syndrome comprising both neuropathic and nociceptive elements arising from both canal and facet joint structures.[14] Contrary to the popular belief neuropathic pain is not restricted to typical radiculopathy, as shown by Attal et al.[15] Our present study focused on the relation between LCS and intensity of LBP, assessed by VAS pain score. The presence and severity of central LCS could be assessed with Schizas qualitative morphological classification based on the CSF/rootlet ratio in axial T_2-weighted MRI images at intervertebral disk level.[16] Correlating symptoms and physical examination findings were an important task, particularly when invasive intervention was considered. This was made more challenging in absence of a universally accepted radiographic definitions for the diagnosis of central, lateral, or foraminal stenosis. Most studies relied on criteria published by Verbiest et al., where he defined absolute stenosis as a diameter of <10 mm.[17] Although this method was criticized for ignoring the trefoil shape of lumbar spinal canal and intrusion of ligamentum flavum and disk material in degenerative stenosis,[18] in absence of a better definition, <10 mm is accepted as the current definition of LCS.

For pain assessment, we used VAS pain scale, defined by Gould et al., as a measurement instrument that tries to measure a characteristic or attitude believed to range across a continuum of values that can't be directly measured. VAS, numeric rating scale, and pain severity subscale of brief pain inventory, the three pain measuring tools are shown not to have superior measurement properties among themselves.[19] We used the simplest form of VAS, a straight horizontal line 100 mm long, the ends defined as extreme limits of the parameters to be measured (no pain to pain as bad as can be).

In our study, percentage of male and female subjects was 62.5% and 37.5%, respectively, the ratio being 1.6:1. This correlated well with the Indian study.[3] Regarding age

distribution of cases the range was 21–75 years (median age 42 years), whereas the Indian study[3] showed maximum number of cases (34%) were in the 4th decade.

In our study, the range of the duration of the LBP was 90–2920 days, the mean being 281 days. In a Czech study, the mean duration was much more, mean 66.1 months (range 3–360 months). However, this might have reflected the difference in study designs in two studies.

Our study showed a significant association between presence of LBP and presence of LCS. Pearson's Chi-square test was applied to ascertain any significant difference between cases and asymptomatic subjects as to the presence of stenosis that showed P=0.015 which was significant. This contradicted with the finding of the Indian study[3], where they found insignificant relation (P=0.7927) between axial back pain and canal stenosis. This was the finding also of other study by Siddique et al.[20] In real life scenario, apart from canal stenosis, many other lesions such as Modic change (MC) are associated with LBP.[21] A large-scale population-based cohort study concluded that MC was associated with presence and severity of LBP.[22] We opine that more studies are needed, which will compare symptomatic (LBP) versus asymptomatic groups of subjects on the basis of single pathologies (e.g., DD and canal stenosis) separately as well as multiple pathologies with multivariate analysis. Still, our study clearly indicated canal stenosis as an associate of LBP which might be used as a guide in future studies or clinical intervention.

We performed statistical tests of normality (within the patient group with LBP) in the duration of LBP, VAS score, and lowest canal diameter. No significant correlation of VAS pain score with lowest canal diameter was found. These findings correlated with other studies conducted by Kuittinen et al.,[23] and Geisser et al.[24] Absence of this correlation could be explained in view of the fact that canal stenosis was not the single key element in the pathogenesis of LBP.

Our study also focused on the presence of SSD in association with LBP. Out of 80 cases in total, 11 cases (13.75%) fulfilled the criteria of SSS-8. Of 50 male subjects, 4 (8.0%) and, of 30 female subjects, 7 (23.33%) had SSD. This may direct one's attention to the facts that (1) psychosocial factors may play important roles in the origin and maintenance of LBP and (2) gender may be a differential factor in the interplay of SSD and LBP. This finding was in keeping with other studies like that conducted by Bener et al.[25] Although our data were not statistically analyzed, this calls for a larger study focusing on the relation between LBP and psychosomatic factors.

Limitations of the study

The sample size was small and the observation time was short. The patient selection criteria for the above study are arbitrary. Furthermore, it may not reflect the general population since the study samples were selected from the patients seeking treatment in hospital.

CONCLUSION

1. There was statistically significant association between presence of LCS and LBP
2. No significant correlation was found between intensity of LBP (VAS pain score) and LCD
3. 13.75% patients in our study with LBP had somatic symptom disorder (SSD) (male – 8.0% and female – 23.33%). Thus, psychosomatic factors might have played important roles in the origin and maintenance of LBP.

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REFERENCES

1. Guglielmi G, De Serio A, Leone A, Agrosì L and Cammisa M. Imaging in degenerative disease of the lumbar spine. Rays. 2000;25(1);19-33.
2. Verbiest H. A radicular syndrome from developmental narrowing of the lumbar vertebral canal. J Bone Joint Surg Br. 1954;36(2):230-237.
   https://doi.org/10.1302/0301-620x.36b2.230
3. De C, Ray MK, Chatterjee B, Duttaroy S, Ghosh PK and De C. Correlation between clinical and imaging finding of symptomatic degenerative lumbar spine disease. Saudi J Sports Med. 2018;18(2):79.
   https://doi.org/10.4103/sjsm.sjsm_24_17
4. Herno A. Spinal stenosis without deformity: Nonoperative treatment. In: The Lumbar Spine. Philadelphia, PA: Lippincott Williams & Wilkins; 2004. p. 490-494.
5. Hyodo H, Sato T, Sasaki H and Tanaka Y. Discogenic pain in acute nonspecific low-back pain. Eur Spine J. 2005;14(6):573-577.
   https://doi.org/10.1007/s00586-004-0844-8
6. Jarvik JG, Hollingworth W, Martin B, Emerson SS, Gray DT, Overman S, et al. Rapid magnetic resonance imaging vs

radiographs for patients with low back pain: A Randomized Controlled Trial. JAMA. 2003;289(21):2810-2818.
https://doi.org/10.1001/jama.289.21.2810

7. Ciricillo SF and Weinstein PR. Lumbar canal stenosis. Western J Med. 1993;158(2):171.
8. Amundsen T, Weber H, Lilleås F, Nordal HJ, Abdelnoor M and Magnaes B. Lumbar canal stenosis. Clinical and radiologic features. Spine. 1995;20(10):1178-1186.
https://doi.org/10.1097/00007632-199505150-00013
9. Johnsson KE, Rosen I and Uden A. The natural course of lumbar canal stenosis. Clin Orthop Related Res. 1992;279:82-86.
10. Borenstein DG, O'Mara JW, Boden SD, Lauerman WC, Jacobson A, Platenberg C, et al. The value of magnetic resonance imaging of the lumbar spine to predict low-back pain in asymptomatic subjects: A seven-year follow-up study. JBJS. 2001;83(9):1306-1311.
https://doi.org/10.2106/00004623-200109000-00002
11. Boos N, Semmer N, Elfering A, Schade V, Gal I, Zanetti M, et al. Natural history of individuals with asymptomatic disc abnormalities in magnetic resonance imaging: Predictors of low back pain-related medical consultation and work incapacity. Spine. 2000;25(12):1484-1492.
https://doi.org/10.1097/00007632-200006150-00006
12. Streiner DL, Norman GR and Cairney J. Health Measurement Scales: A Practical Guide to their Development and Use. United States: Oxford University Press; 2015.
https://doi.org/10.1002/wmh3.140
13. Airaksinen O, Brox JI, Cedraschi C, Hildebrandt J, Klaber-Moffett J, Kovacs F, et al. European guidelines for the management of chronic nonspecific low back pain. Eur Spine J. 2006;15(2):s192.
https://doi.org/10.1007/s00586-006-1072-1
14. Suri P, Hunter DJ, Rainville J, Guermazi A and Katz JN. Presence and extent of severe facet joint osteoarthritis are associated with back pain in older adults. Osteoarthritis Cartilage. 2013;21(9):1199-1206.
https://doi.org/10.1016/j.joca.2013.05.013
15. Attal N, Perrot S, Fermanian J and Bouhassira D. The neuropathic components of chronic low back pain: A prospective multicenter study using the DN4 Questionnaire. J Pain. 2011;12(10):1080-1087.
https://doi.org/10.1016/j.jpain.2011.05.006
16. Schizas C, Theumann N, Burn A, Tansey R, Wardlaw D, Smith FW, et al. Qualitative grading of severity of lumbar canal stenosis based on the morphology of the dural sac on magnetic resonance images. Spine 2010;35(21):1919-24.
https://doi.org/10.1097/brs.0b013e3181d359bd
17. Verbiest HE. Pathomorphologic aspects of developmental lumbar stenosis. Orthop Clin North Am. 1975;6(1):177-196.
https://doi.org/10.1016/s0030-5898(20)31210-4
18. Genevay S and Atlas SJ. Lumbar canal stenosis. Best Pract Res Clin Rheumatol. 2010;24(2):253-265.
19. Gould D and Crichton N. Information point: Visual analogue scale (VAS). J Clin Nurs. 2001;10(5):697-706.
20. Siddiqui AH, Rafique MZ, Ahmed MN and Usman MU. Role of magnetic resonance imaging finding in lumbar spondylosis. J Coll Psysicians Surg Pak. 2005;15:296-299.
21. Määttä JH, Wadge S, MacGregor A, Karppinen J and Williams FM. ISSLS prize winner: Vertebral endplate (Modic) change is an independent risk factor for episodes of severe and disabling low back pain. Spine. 2015;40(15):1187-1193.
https://doi.org/10.1097/brs.0000000000000937
22. Mok FP, Samartzis D, Karppinen J, Fong DY, Luk KD and Cheung KM. Modic changes of the lumbar spine: Prevalence, risk factors, and association with disc degeneration and low back pain in a large-scale population-based cohort. Spine J. 2016;16(1):32-41.
https://doi.org/10.1016/j.spinee.2015.09.060
23. Kuittinen P, Sipola P, Saari T, Aalto TJ, Sinikallio S, Savolainen S, et al. Visually assessed severity of lumbar spinal canal stenosis is paradoxically associated with leg pain and objective walking ability. BMC Musculoskelet Disord. 2014;15(1):1-8.
https://doi.org/10.1186/1471-2474-15-348
24. Geisser ME, Haig AJ, Tong HC, Yamakawa KS, Quint DJ, Hoff JT, et al. Spinal canal size and clinical symptoms among persons diagnosed with lumbar canal stenosis. Clin J Pain. 2007;23(9):780-785.
https://doi.org/10.1097/ajp.0b013e31815349bf
25. Bener A, Verjee M, Dafeeah EE, Falah O, Al-Juhaishi T, Schlogl J, et al. Psychological factors: Anxiety, depression, and somatization symptoms in low back pain patients. J Pain Res. 2013;6:95.
https://doi.org/10.2147/jpr.s40740

Authors Contribution:
MM- Literature review and manuscript preparation, data collection; **DPC-** Concept and design of the study, preparation of the first draft, data collection and data management; **AC-** Revision of the manuscript; **PS-** Statistical analysis and interpretation; **NKB-** Overall supervision, guidance about the psychiatric parts of the study, finalizing the manuscript

Work attributed to:
North Bengal Medical College, Sushrutanagar, Darjeeling - 734 012, West Bengal, India

Orcid ID:
Dr. Mahadeb Mandal - https://orcid.org/0000-0002-3301-0032
Dr. Durga Prasad Chakraborty - https://orcid.org/0000-0003-1356-7877
Dr. Aniruddha Chattopadhyay - https://orcid.org/0000-0002-4000-7181
Dr. Pulakesh Sarmah - https://orcid.org/0000-0002-3449-0517
Dr. Nirmal Kumar Bera - https://orcid.org/0000-0002-2411-7137

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