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Demographic and Clinical Characteristics of Patients With Cervical Spine Degeneration Reveal Frequent Cervicolumbar Tandem Spinal Stenosis in Mexico

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ABSTRACT

Background: Limited literature exists regarding the differences in demographics, causes, comorbidities, presentation, and structural changes associated with cervical spine degeneration in patients from distinct geographic regions. The authors aimed to evaluate the demographic and clinical characteristics of patients with cervical spine degeneration admitted to a single center in Mexico.

Methods: This study enrolled patients with degenerative disease of the cervical spine. Clinical data were retrieved from medical records and retrospectively characterized.

Results: A total of 50 patients with cervical spine degeneration were included in the analysis. Of these, 26% were men with a median age of 54 years. Hypertension, depression, anxiety, obesity, and alcohol consumption were presented in about a quarter of the participants. In addition, we observed hypertriglyceridemia and hypercholesterolemia in 72% and 46% of participants, respectively. The median duration of symptoms was 11 months, including radicular arm/neck pain (80%), tingling (80%), reduced muscle strength (48%), and gait disturbances (48%). Forty percent of patients had 2 cervical segments radiologically involved, mainly at C5–C6, with changes such as disc herniation (88%), foraminal stenosis with nerve root compression (67%), reduced spinal canal-to-vertebral body ratio (38%), and ligamentum flavum hypertrophy (24%). Also, 22% of patients showed degenerative cervical myelopathy. Strikingly, 48% of enrolled individuals showed cervicolumbar tandem spinal stenosis, mainly in L4–L5 and L5–S1, who were generally older, had a longer duration of symptoms, and had a higher comorbidity burden, including hyperglycemia, hypertension, and depression.

Conclusions: The demographic and clinical characteristics of degenerative cervical spine disease in Mexico differ with respect to other geographical regions by a younger age of diagnosis, a high frequency of cardiovascular, metabolic, and mental health comorbidities, and an increased prevalence of concomitant lumbar spinal stenosis.

Clinical Relevance: Our findings reveal a considerably high burden of cervicolumbar tandem spinal stenosis as a distinctive feature of Mexican patients with cervical spine degeneration.

Level of Evidence: 1.

Cervical Spine

Keywords: cervical spine, cervical radiculopathy, cervical spondylotic myelopathy, degenerative disc disease, spine surgery, tandem spinal stenosis

INTRODUCTION

Degenerative disease of the cervical spine encompasses a continuum of chronic and progressive structural and physiological changes in the articular components of the cervical vertebrae.^{1,2} These alterations result from the degeneration of the intervertebral discs, named degenerative disc disease (DDD), which begins with radial and circumferential disc fissures, decreased disc height, and collapse, accompanied by local synovitis and disruption of the dynamic homeostasis of the spine. Then, changes in alignment and stability generate stress

on the facet joints, resulting in disc protrusion, disc extrusion, and spondylotic abnormalities of the cervical spine, such as facet hypertrophy, osteophyte formation, ligamentum flavum hypertrophy (LFH), and facet subluxation. The chronic stages of the disease are characterized by ankylosis of the cervical spine and narrowing of the cervical canal, which can cause compression of the spinal cord, a condition named degenerative cervical myelopathy (DCM).^{3–5}

The clinical manifestations of degenerative spondylosis and stenosis of the cervical spine vary greatly according to the stage of the disease. As such, affected

individuals may present with a constellation of complaints that range from asymptomatic radiological changes to neck/arm pain, neck stiffness, sensitive alterations, muscle atrophy and reduced muscle strength in the upper limbs, headaches, and spinal cord dysfunction.⁶⁻¹³ Hence, although the identification of cases is straightforward in some instances, the diagnosis can be very challenging due to different rates of disease progression and accompanying comorbidities that may affect the risk and severity of the manifestations. For instance, although cervical disc degeneration is a naturally age-related phenomenon occurring worldwide, studies have found variable contributions of factors other than age to the risk and presentation of the disease, including genetics, gender, overweight, obesity, atherosclerosis, diabetes, occupation, contact sports, prior surgeries, smoking, and alcohol consumption.^{3,14-16}

Adding complexity, several sociocultural differences may modify the perception of the manifestations in different regions.¹⁷ Also, concomitant degenerative lumbar stenosis, known as cervicolumbar tandem spinal stenosis (CLTSS), has been identified in variable frequencies across populations, contributing to the clinical heterogeneity of the disorder.¹⁸ Importantly, there is a paucity of studies addressing differences in demographics, causes, comorbidities, presentation, and frequency of concomitant lumbar stenosis in patients with cervical spine degeneration from distinct geographic regions. Furthermore, there is an underrepresentation of Latin American populations in large prospective studies evaluating global variations in cervical spine degeneration phenotypes.¹⁷

Therefore, the objective of this study was to describe the demographics and presentation of Mexican patients with degenerative disease of the cervical spine. Our results identify particular characteristics of Latin American patients with cervical spine degeneration, highlighting a high burden of CLTSS. The information provided here may be relevant for spine surgeons in the Americas, where a rise in the prevalence of DCM is predicted to occur due to the regional demographic and epidemiological transition.

MATERIALS AND METHODS

We conducted a retrospective revision of clinical records from prospectively enrolled patients with spine diseases who consecutively attended the outpatient clinic of a private tertiary neurosurgery reference center in northern Mexico City between January 2016 and December 2021. Patients older than 18 years were eligible if they had complained of a combination of

manifestations secondary to cervical spine degeneration, including intractable cervical radiculopathy (neck/arm pain), reduced muscle strength, gait problems, and subjective (paresthesia and numbness) or objective sensory changes in the upper limbs during the first clinical evaluation. Individuals with nondegenerative cervical compression, prior cervical spine surgery, cervical trauma, thoracic spinal stenosis, fractures, tumors, rheumatoid arthritis, ankylosing spondylitis, movement disorders, or incomplete medical records were excluded from the study.

Data were collected and recorded on a special form in Microsoft Excel (MS Excel 365) by 2 trained spinal surgery fellows. The information retrieved corresponded to the findings made in the first clinical interview, physical examination, and radiological and laboratory evaluation before receiving treatment. Retrieved data from participants included age, gender, anthropometrics, comorbidities, history of previous nonspinal surgeries, symptoms, vital signs, routine laboratory test results, and preoperative radiological findings in the magnetic resonance imaging (MRI) of the cervical spine.

Radiological findings of interest were the number of spinal segments radiologically involved and the frequency of the following changes: disc herniation, foraminal stenosis with nerve root compression, myelopathy (intramedullary hyperintense signal on T2-weighted MRI and contrast enhancement on post-gadolinium T1-weighted imaging), spondylolisthesis, and LFH. Also, plain lateral radiographs of the cervical spine were used to identify cervical canal stenosis using a cut-off value ≤ 0.8 in the spinal canal-to-vertebral body ratio.¹⁴ In addition, we evaluated the presence of concomitant radiological degenerative lumbar spinal stenosis. For this purpose, CLTSS was defined as having coexisting symptomatic or asymptomatic degenerative lumbar spine changes in the MRI neuroaxis.¹⁸

STATISTICAL METHODS

Descriptive statistics were used to characterize the study population clinically. Frequencies and proportions were calculated for categorical data. Medians and interquartile ranges were used for continuous variables. Differences between groups were assessed by the Fisher exact test, χ^2 test, or Mann-Whitney *U* test, as appropriate. All analyses were conducted using GraphPad Prism 8 (La Jolla, CA, USA). Specific analysis tests are also mentioned in the tables. Two-tailed *P* values ≤ 0.05 were considered significant.

RESULTS

From a total of 160 patients with spinal disorders identified in our clinical record database, 50 met the inclusion criteria. Their demographic and clinical characteristics are summarized in Table 1. Our cohort included 13 men and 37 women, with a median age of 54 years. Of these, all were of non-Caucasian Hispanic ethnicity. Their median body mass index (BMI) was about 26 kg/m², with up to a quarter of study participants being obese. Other frequent comorbidities in the overall study population included hypertension, depression, anxiety, alcohol consumption, and smoking. In addition, 14% of patients were diabetic, 3 had renal failure, and 1 reported hepatic failure. Notably, up to two-thirds of enrolled individuals had at least 1 prior nonspinal surgical intervention.

The average duration of symptoms was 11 months. The most frequent complaints of cervical spine degeneration were radicular arm/neck pain or stiffness, followed by paresthesia, reduced muscle strength of the upper and/or lower limbs, and gait disturbances. Also, 13 patients had numbness and 8 reported headaches; sensory alterations were objectively identified in only 5 patients.

In general, patients had degenerative alterations in a median of 2 cervical spine segments, with C5–C6 and C4–C5 being the levels most frequently compromised in 92% and 88% of patients, respectively. The main degenerative changes observed in the radiological studies included disc herniations (88%), stenosis in the lateral recess and neural foramen leading to nerve root compression (67%), a significant reduction in the cervical canal anteroposterior diameter (38%), LFH (24%), myelopathy (22%), and spondylolisthesis (4%).

Vital signs and laboratory test results at baseline were within normal ranges in most patients. In this regard, leukocytosis was observed in 16% of participants, neutrophilia in 18%, lymphopenia in 7%, and anemia in 14%. Other common alterations included hyperglycemia (20%), high blood urea nitrogen (BUN) levels (32%), elevated creatinine (11%), hyperuricemia (2%), hyperbilirubinemia (3%), high aspartate aminotransferase (16%), alanine aminotransferase (25%), and lactate dehydrogenase (LDH, 42%) levels, as well as hypercholesterolemia (46%) and hypertriglyceridemia (72%).

Interestingly, almost 50% of patients with degenerative disease of the cervical spine showed concomitant radiological changes in the lumbar region. As shown in Table 2, the median lumbar levels involved were 2, with 46% and 44% of patients showing changes in L4–L5

Table 1. Participant characteristics.

Characteristics	N = 50
Demographic	
Age, y	54.0 (46.5, 68.8)
Male gender	13 (26%)
Hispanic race	50 (100%)
Body mass index, kg/m ²	26.6 (24.2, 30.3)
Comorbidities	
Hypertension	14 (28%)
Depression	14 (28%)
Anxiety	14 (28%)
Obesity	13 (26%)
Alcoholism	13 (26%)
Smoking	11 (22%)
Diabetes	7 (14%)
Renal failure	3 (6.0%)
Hepatic failure	1 (2.0%)
Previous surgery	35 (70%)
Clinical manifestations	
Symptom duration, mo	11.6 (0.0, 120) ^a
Radicular arm/neck pain	39 (80%)
Paresthesia/tingling	35 (70%)
Reduced muscle strength	24 (48%)
Gait changes	24 (48%)
Numbness/hypoesthesia	13 (26%)
Headache	8 (16%)
Objective sensory impairment	5 (10%)
Radiological findings	
Cervical segments, n	2.0 (2.0, 3.0)
1	2 (4.0%)
2	24 (48%)
3 or more	19 (38%)
C5–C6	46 (92%)
C4–C5	44 (88%)
C6–C7	21 (42%)
C3–C4	15 (30%)
C2–C3	2 (4.0%)
C7–T1	1 (2.0%)
Disc herniation	44 (88%)
Foramen stenosis	33 (67%)
Central canal stenosis	19 (38%)
Ligament flavum hypertrophy	12 (24%)
Myelopathy	11 (22%)
Spondylolisthesis	2 (4.0%)
Vital signs	
Temperature, °C	36.3 (36.1, 36.5)
Heart rate, bpm	76.0 (70.0, 88.0)
Respiratory rate, rpm	19.0 (18.0, 20.0)
Mean arterial blood pressure, mmHg	86.7 (82.5, 93.3)
>100 mmHg	6/46 (13%)
Saturation, %	94.0 (92.0, 95.0)
Laboratory parameters	
White blood cells, per μ L	7200.0 (5900.0, 8900.0)
>10,000 μ L	7/44 (16%)
Neutrophils, per μ L	4597.0 (3382.2, 6041.0)
>8000 μ L	7/39 (18%)
Lymphocytes, per μ L	1978.0 (1696.5, 2434.0)
<1000 μ L	3/39 (7.7%)
Hemoglobin, g/dL	14.3 (13.6, 15.6)
Anemia	6/43 (14%)
Hematocrit, %	44.0 (39.8, 46.3)
Platelets, per μ L	262.0 (236.2, 305.0)
Glucose, mg/dL	98.0 (87.0, 108.0)
>126 mg/dL	9/45 (20%)
Blood urea nitrogen, mg/dL	14.2 (12.1, 22.0)
>20 mg/dL	13/41 (32%)
Creatinine, mg/dL	0.7 (0.7, 0.9)
>1.1 mg/dL	5/45 (11%)
Uric acid, mg/dL	5.1 (3.7, 6.1)
>8.5 mg/dL	1/43 (2.3%)
Na, mEq/L	139.0 (138.0, 142.0)
K, mmol/L	4.0 (3.8, 4.4)

Table 1. Continued.

Characteristics	N = 50
Ca, mg/dL	9.3 (9.1, 9.6)
Bilirubin, mg/dL	0.5 (0.3, 0.7)
>1.2 mg/dL	1/38 (2.6%)
Aspartate aminotransferase, U/L	22.0 (19.0, 27.0)
>33 U/L	6/37 (16%)
Alanine aminotransferase, U/L	27.5 (16.8, 35.5)
>36 U/L	9/36 (25%)
Lactate dehydrogenase, IU/L	219.5 (183.2, 415.0)
>280 IU/L	14/34 (41%)
Cholesterol, mg/dL	198.0 (165.0, 208.0)
>200 mg/dL	19/41 (46%)
Triglycerides, mg/dL	181.9 (112.5, 236.0)
>150 mg/dL	29/40 (72%)

Note: Data are displayed as n (%), n/N (%), or median (interquartile range).

^aDuration of symptoms is displayed as median with range. N is the total number of participants with available information.

and L5–S1. Less than 10% of patients had alterations in other segments of the lumbar spine.

Remarkably, patients with CLTSS significantly differed from those without CLTSS by a higher median age (61 years vs 51 years, *P* < 0.05) and higher frequency of hypertension and depression as comorbidities. Also, CLTSS patients showed a longer duration of symptoms from onset (16 months vs 7 months, *P* < 0.05), as illustrated in Table 3. Nonetheless, the profile of clinical manifestations of cervical spine degeneration was comparable between patients with and without CLTSS. Similarly, both groups showed homogeneity in the numbers of levels affected and patterns of radiological changes in the cervical spine, although there was a trend toward a higher frequency of alterations in segments other than C4–C5 and C5–C6 among the CLTSS group (Table 3). Furthermore, central stenosis of the cervical canal was more common in CLTSS than non-CLTSS patients, although the difference did not reach statistical significance. Finally, patients with CLTSS showed a higher frequency of hyperglycemia and elevated BUN levels as compared with those without coexisting cervical and lumbar spine degeneration (Table 4).

Table 2. Lumbar spine changes in patients with degenerative cervical spinal disease.

Characteristics	N = 50
Cervicolumbar tandem spinal stenosis	24 (48%)
Lumbar segments, n	2.0 (1.0, 2.0)
1	3 (12%)
2	14 (58%)
≥3	7 (29%)
L4–L5	23 (46%)
L5–S1	22 (44%)
L3–L4	3 (6.0%)
L1–L2	1 (2.0%)
L2–L3	1 (2.0%)

Note: Data are displayed as n (%) or median (interquartile range).

The rest of the laboratory parameters and vital signs were equivalents between groups.

DISCUSSION

Understanding the variations in demographics, comorbidities, causes, and presentation of degenerative disorders of the cervical spine across different regions and in distinct sociocultural settings is pivotal in improving diagnostic strategies and preventing long-lasting consequences via opportune treatment. Indeed, differences in perception of disease manifestations, impact on quality of life, comorbidity burden, access to medical attention, and availability of diagnostic resources are important to be considered for developing international guidelines applicable to different populations. Unfortunately, large comparative analyses of the clinical features, diagnostic approaches, and therapeutic preferences advocated for DDD and DCM have focused mainly on European, Asian, and North American cohorts.¹⁷ In contrast, limited literature exists about the prevalence and characteristics of degenerative disorders of the cervical spine in Latin American countries.

Importantly, the region of the Americas has experienced a demographic and epidemiological transition toward a higher burden of nontransmissible disorders related to aging and lifestyle changes.¹⁹ As such, an increasing frequency of metabolic comorbidities, postural problems, dietary changes, and sedentarism has been accompanied by a rise in musculoskeletal disorders. In this setting, Mexico is among the regions that have exhibited the most dramatic transitions to chronic degenerative conditions associated with aging and unhealthy behaviors, including overweight, obesity, and diabetes, among others. Despite this, the exact epidemiology and clinical characteristics of DDD and DCM in Mexico and other Latin American countries have been scarcely analyzed.²⁰ Recently, Zárate-Kalfópulos showed that degenerative disease-causing myelopathy is present in about 20% of the patients with cervical spinal cord injury from Mexico City.²¹ However, the knowledge of this condition in the region is obscured by the higher frequency of traumatic spinal cord injury.

In this study, we aimed to provide a clinical characterization of the principal demographic properties, causes, radiological findings, and manifestations of Latin American patients with cervical spine degeneration. Interestingly, our single-center cohort showed features validating previous international efforts to address geographical differences in cervical spinal degenerative disease. For instance, in a multicenter study including data from 2 large international cohorts and involving

Table 3. Comparison of demographic and clinical characteristics of patients with CLTSS.

Characteristics	CLTSS N = 24	Non-CLTSS N = 26	P Value
Demographics			
Age, y	61.0 (52.0, 73.0)	51.5 (44.5, 60.0)	0.022
Male gender	8 (33%)	5 (19%)	0.3
Body mass index, kg/m ²	26.6 (23.8, 31.4)	26.3 (24.2, 29.6)	0.9
Comorbidities			
Obesity	7 (29%)	6 (23%)	0.6
Hypertension	11 (46%)	3 (12%)	0.007
Depression	11 (46%)	3 (12%)	0.007
Anxiety	9 (38%)	5 (19%)	0.2
Alcoholism	6 (25%)	7 (27%)	0.9
Smoking	5 (21%)	6 (23%)	0.8
Diabetes	5 (21%)	2 (7.7%)	0.2
Renal failure	2 (8.3%)	1 (3.8%)	0.6
Hepatic failure	1 (4.2%)	0 (0%)	0.5
Previous surgery	18 (75%)	17 (65%)	0.5
Clinical manifestations			
Symptom duration, mo	16.0 (0.0, 120)	7.1 (0.0, 58.0)	0.023
Radicular/neck pain	18 (78%)	21 (81%)	>0.9
Paresthesia/tingling	17 (71%)	18 (69%)	>0.9
Reduced muscle strength	14 (58%)	10 (38%)	0.2
Gait changes	14 (58%)	10 (38%)	0.2
Numbness/hypoesthesia	8 (33%)	5 (19%)	0.3
Headache	5 (21%)	3 (12%)	0.5
Sensory impairment	4 (17%)	1 (3.8%)	0.2
Radiological findings			
Cervical segments, n	2.5 (2.0, 3.0)	2.0 (2.0, 3.0)	0.4
1	0 (0%)	2 (7.7%)	0.5
2	12 (50%)	12 (46%)	0.8
3	8 (33%)	11 (42%)	0.5
4 or more	4 (17%)	1 (3.8%)	0.2
C2–C3	2 (8.3%)	0 (0%)	0.2
C3–C4	10 (42%)	5 (19%)	0.084
C4–C5	21 (88%)	23 (88%)	>0.9
C5–C6	21 (88%)	25 (96%)	0.3
C6–C7	11 (46%)	10 (38%)	0.6
C7–T1	1 (4.2%)	0 (0%)	0.5
Disc herniation	21 (88%)	23 (88%)	>0.9
Foraminal stenosis	17 (74%)	16 (62%)	0.4
Central canal stenosis	12 (50%)	7 (27%)	0.093
Myelopathy	7 (29%)	4 (15%)	0.2
Ligament flavum hypertrophy	5 (21%)	7 (27%)	0.6
Spondylolisthesis	2 (8.3%)	0 (0%)	0.2

Abbreviation: CLTSS, cervicolumbar tandem spinal stenosis.

Note: Data are displayed as n (%) or median (interquartile range).

*Duration of symptoms is displayed as median with range. N is the total number of participants with available information. Differences between groups were analyzed with the Fisher exact test, χ^2 test, or Mann-Whitney U test, as appropriate.

80 Latin American patients from Venezuela and Brazil, Fehlings and colleagues highlighted important distinctive features of native-American people with DCM, including younger age of presentation, longer duration of symptoms, higher frequency of disc herniation, and LFH compared with patients from other regions.¹⁷ Similarly, we also found that, in Mexico, cervical spine degeneration primarily affects people in their 50s and has disc herniation as the leading cause of cervical stenosis.

Importantly, we identified certain differences in our cohort with respect to other populations, such as a higher frequency of women and a shorter duration of symptoms, which might be caused by selection bias due to the single-center private hospital-based design

of our study. Remarkably, we also provided information about comorbid conditions affecting patients with cervical spine stenosis, including hypertension, depression, anxiety, and obesity, all of which might alter the risk and natural course of the disease.

Depression and anxiety have been found in up to a third of patients with cervical spine spondylosis and may affect the severity of DCM.^{22,23} Hypertension is also frequently observed among individuals with cervical spine degeneration, and some authors propose that degenerative disc disorders could produce nerve and spinal cord compression, sympathetic reflex, and hypertension.²⁴ Obesity and DDD share risk factors for disease development, including postural problems and sedentarism. A high BMI is also important for operative

Table 4. Vital signs and laboratory parameters of patients with CLTSS.

Characteristics	CLTSS N = 24	Non-CLTSS N = 26	P Value
Vital signs			
Temperature, °C	36.3 (36.0, 36.5)	36.3 (36.2, 36.5)	0.7
Heart rate, bpm	78.0 (72.0, 88.0)	75.0 (70.0, 87.0)	0.5
Respiratory rate, rpm	19.0 (18.0, 20.0)	20.0 (18.0, 20.0)	0.5
Mean arterial blood pressure, mmHg	86.7 (83.3, 95.0)	86.7 (80.0, 90.0)	0.3
>100 mmHg	4/23 (17%)	2/23 (8.3%)	0.4
Saturation, %	93.0 (91.0, 95.0)	94.0 (92.0, 95.0)	0.2
Laboratory parameters			
White blood cells, per μ L	7070.0 (5772.5, 8665.0)	7300.0 (6000.0, 8900.0)	0.4
>10,000/ μ L	3/22 (14%)	4/22 (18%)	> 0.9
Neutrophils, per μ L	3834.0 (3401.5, 5790.0)	5184.0 (3438.5, 6113.5)	0.3
>8000/ μ L	4/21 (19%)	3/18 (16%)	> 0.9
Lymphocytes, per μ L	1964.0 (1572.0, 2340.5)	1978.0 (1763.0, 2530.5)	0.3
<1000 / μ L	2/20 (10%)	1/19 (5.3%)	> 0.9
Hemoglobin, g/dL	14.3 (13.6, 16.1)	14.3 (14.0, 15.1)	0.7
Anemia	2/21 (9.5%)	4/22 (17%)	0.7
Hematocrit, %	43.5 (39.0, 46.8)	44.0 (41.4, 46.0)	> 0.9
Platelets, per μ L	256.0 (234.0, 305.0)	262.0 (238.0, 304.0)	> 0.9
Glucose, mg/dL	107.9 (98.0, 134.0)	90.0 (83.8, 102.0)	0.010
>126 mg/dL	7/21 (33%)	2/24 (8.3%)	0.061
Blood urea nitrogen, mg/dL	21.7 (14.2, 24.3)	13.2 (10.8, 15.1)	0.009
>20 mg/dL	9/17 (53%)	4/24 (17%)	0.014
Creatinine, mg/dL	0.8 (0.7, 0.9)	0.7 (0.7, 0.8)	0.2
>1.1 mg/dL	4/21 (19%)	1/24 (4.2%)	0.2
Uric acid, mg/dL	5.7 (4.0, 6.9)	4.8 (3.7, 5.8)	0.3
>8.5 mg/dL	0/19 (0%)	1/24 (4.2%)	> 0.9
Na, mEq/L	138.6 (137.0, 142.0)	141.0 (138.0, 142.0)	0.4
K, mmol/L	4.2 (3.9, 4.4)	4.0 (3.8, 4.1)	0.2
Ca, mg/dL	9.4 (9.2, 9.6)	9.3 (9.1, 9.5)	0.7
Bilirubin, mg/dL	0.3 (0.3, 0.5)	0.5 (0.4, 0.7)	0.025
>1.2 mg/dL	0/14 (0%)	1/24 (4.2%)	> 0.9
Aspartate aminotransferase, U/L	19.0 (18.0, 24.5)	24.0 (20.2, 30.8)	0.12
>33 U/L	1/15 (6.7%)	5/22 (23%)	0.4
Alanine aminotransferase, U/L	22.0 (16.3, 31.0)	32.0 (18.5, 42.0)	0.14
>36 U/L	1/14 (7.1%)	8/22 (36%)	0.062
Lactate hydrogenase, IU/L	219.5 (181.8, 346.8)	234.0 (183.2, 427.8)	0.7
>280 IU/L	4/12 (33%)	10/22 (45%)	0.5
Cholesterol, mg/dL	198.0 (139.5, 240.0)	194.5 (169.2, 207.5)	0.9
>200 mg/dL	9/19 (47%)	10/22 (45%)	> 0.9
Triglycerides, mg/dL	169.4 (108.8, 198.2)	190.0 (160.0, 238.2)	0.2
>150 mg/dL	11/18 (61%)	18/22 (82%)	0.14

Abbreviation: CLTSS, cervico-lumbar tandem spinal stenosis.

Note: Data are displayed as n (%), n/N (%), or median (interquartile range). N is the total number of participants with available information. Differences between groups were analyzed with the Fisher exact test, χ^2 test, or Mann-Whitney U test, as appropriate.

outcomes in patients receiving spine surgery since obesity may interfere with preoperative radiological images, hinder intubation for general anesthesia, complicate positioning for incision, and increase the operative risk associated with metabolic comorbidities.²⁵

In addition, we also found common laboratory test alterations accompanying cervical spine degeneration, highlighting dyslipidemia, high LDH and BUN levels, leukocytosis, and anemia. Dyslipidemia has been associated with DDD in other regions of the spine, and it is believed that high lipid levels promote inflammation and degeneration of intervertebral discs.^{26,27} Meanwhile, elevated LDH and leukocytosis might traduce a chronic inflammatory and injury process of the spine. Finally, increased BUN levels (traducing dehydration and/or renal failure) and anemia are important alterations to

be corrected in patients waiting for elective surgery to improve perioperative outcomes.

Strikingly, the most relevant finding of our study was the high frequency of concomitant lumbar stenosis in our cohort since almost half of our patients presented radiological CLTSS. This entity is increasingly recognized as an important source of morbidity secondary to compressive degeneration of the spine.²⁸ The burden of CLTSS differs between distinct regions, although it has been observed radiologically in 30% to 85% of patients with asymptomatic cervical or lumbar stenosis, causing symptoms in up to a third of the affected individuals.¹⁸ However, the frequency of CLTSS is unknown in native-American Hispanic patients. Of note, CLTSS imposes several challenges on spine surgeons due to its great clinical heterogeneity. Accordingly, this condition is

characterized by a triad consisting of intermittent claudication, progressive gait disorder, and mixed myelopathy and polyradiculopathy manifestations in the upper and lower extremities.²⁹

Furthermore, controversies exist about the most effective surgical treatment for CLTSS, with some groups prioritizing the decompression of the spinal region causing the most severe symptoms and disability first (typically the cervical region, followed by lumbar spine surgery).^{18,30–35} Notably, in some individuals, this surgical pattern reduces the requirement for a second stage, as cervical surgery also improves lumbar symptoms.^{30,31,36,37} Thus, it is important to identify the clinical characteristics and severity of compression of different populations with CLTSS to make more specific and effective interventions. Overall, our findings reflect a high burden of CLTSS among Mexican patients attending spine surgery centers due to degenerative cervical spinal disease.

Our study has limitations that should be considered when interpreting the results. First, we conducted a retrospective single-center investigation. Also, the small sample size requires validation of our results in more extensive prospective studies. This caveat was due to the rigorous inclusion criteria to enroll only patients with degenerative disease of the cervical spine.

CONCLUSION

We described the demographic and clinical characteristics of Latin American patients with degenerative cervical spine disease from Mexico City. Our findings reveal a younger age of diagnosis, a high frequency of comorbidities like hypertension, mood disorders, obesity, and dyslipidemia, and a considerably high burden of CLTSS as distinctive features of our cohort with respect to patients from other geographical regions.

REFERENCES

1. Tsutsumimoto T, Shimogata M, Yui M, Ohta H, Misawa H. The natural history of asymptomatic lumbar canal stenosis in patients undergoing surgery for cervical myelopathy. *J Bone Joint Surg Br.* 2012;94(3):378–384. doi:10.1302/0301-620X.94B3.27867
2. Nurick S. The pathogenesis of the spinal cord disorder associated with cervical spondylosis. *Brain.* 1972;95(1):87–100. doi:10.1093/brain/95.1.87
3. Baron EM, Young WF. Cervical spondylotic myelopathy: a brief review of its pathophysiology, clinical course, and diagnosis. *Neurosurgery.* 2007;60(1 Suppl 1):S35–41. doi:10.1227/01.NEU.0000215383.64386.82
4. Montgomery DM, Brower RS. Cervical spondylotic myelopathy. Clinical syndrome and natural history. *Orthop Clin North Am.* 1992;23(3):487–493.
5. Shedid D, Benzel EC. Cervical spondylosis anatomy: pathophysiology and biomechanics. *Neurosurgery.* 2007;60(1 Suppl 1):S7–13. doi:10.1227/01.NEU.0000215430.86569.C4
6. Okada E, Matsumoto M, Ichihara D, et al. Aging of the cervical spine in healthy volunteers: a 10-year longitudinal magnetic resonance imaging study. *Spine (Phila Pa 1976).* 2009;34(7):706–712. doi:10.1097/BRS.0b013e31819c2003
7. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg Am.* 1990;72(8):1178–1184.
8. Lehto IJ, Tertti MO, Komu ME, Paajanen HE, Tuominen J, Kormano MJ. Age-related MRI changes at 0.1 T in cervical discs in asymptomatic subjects. *Neuroradiology.* 1994;36(1):49–53. doi:10.1007/BF00599196
9. Heller JG. The syndromes of degenerative cervical disease. *Orthop Clin North Am.* 1992;23(3):381–394.
10. Nilsson N. The prevalence of cervicogenic headache in a random population sample of 20–59 year olds. *Spine (Phila Pa 1976).* 1995;20(17):1884–1888. doi:10.1097/00007632-199509000-00008
11. Holmes A, Wang C, Han ZH, Dang GT. The range and nature of flexion-extension motion in the cervical spine. *SPINE (Phila Pa 1976).* 1994;19(22):2505–2510. doi:10.1097/00007632-199411001-00003
12. Voskuhl RR, Hinton RC. Sensory impairment in the hands secondary to spondylotic compression of the cervical spinal cord. *Arch Neurol.* 1990;47(3):309–311. doi:10.1001/archneur.1990.00530030085020
13. Iyer S, Kim HJ. Cervical radiculopathy. *Curr Rev Musculoskelet Med.* 2016;9(3):272–280. doi:10.1007/s12178-016-9349-4
14. Torg JS, Naranja RJ, Pavlov H, Galinat BJ, Warren R, Stine RA. The relationship of developmental narrowing of the cervical spinal canal to reversible and irreversible injury of the cervical spinal cord in football players. *J Bone Joint Surg Am.* 1996;78(9):1308–1314. doi:10.2106/00004623-199609000-00003
15. Gore DR, Carrera GF, Glaeser ST. Smoking and degenerative changes of the cervical spine: a roentgenographic study. *Spine J.* 2006;6(5):557–560. doi:10.1016/j.spinee.2005.12.003
16. Singh A, Tetreault L, Fehlings MG, Fischer DJ, Skelly AC. Risk factors for development of cervical spondylotic myelopathy: results of a systematic review. *Evid Based Spine Care J.* 2012;3(3):35–42. doi:10.1055/s-0032-1327808
17. Fehlings MG, Kopjar B, Ibrahim A, et al. Geographic variations in clinical presentation and outcomes of decompressive surgery in patients with symptomatic degenerative cervical myelopathy: analysis of a prospective, international multicenter cohort study of 757 patients. *Spine J.* 2018;18(4):593–605. doi:10.1016/j.spinee.2017.08.265
18. Bai Q, Wang Y, Zhai J, Wu J, Zhang Y, Zhao Y. Current understanding of tandem spinal stenosis: epidemiology, diagnosis, and surgical strategy. *EFORT Open Rev.* 2022;7(8):587–598. doi:10.1530/EOR-22-0016
19. Alvarez J-A, Aburto JM, Canudas-Romo V. Latin American convergence and divergence towards the mortality profiles of developed countries. *Popul Stud (Camb).* 2020;74(1):75–92. doi:10.1080/00324728.2019.1614651
20. Cahueque Lemus MA, Cobar Bustamante AE, Ortiz Muciño A, Caldera Hernandez G. Clinical outcome of anterior vs posterior approach for cervical spondylotic myelopathy. *J Orthop.* 2016;13(3):123–126. doi:10.1016/j.jor.2016.03.006

21. Zárte-Kalfópulos B, Jiménez-González A, Reyes-Sánchez A, Robles-Ortiz R, Cabrera-Aldana EE, Rosales-Olivarez LM. Demographic and clinical characteristics of patients with spinal cord injury: a single hospital-based study. *Spinal Cord*. 2016;54(11):1016–1019. doi:10.1038/sc.2016.41
22. Lin SY, Sung FC, Lin CL, Chou LW, Hsu CY, Kao CH. Association of depression and cervical spondylosis: a nationwide retrospective propensity score-matched cohort study. *J Clin Med*. 2018;7(11):387;11. doi:10.3390/jcm7110387
23. Stoffman MR, Roberts MS, King JT. Cervical spondylosis, depression, and anxiety: a cohort analysis of 89 patients. *Neurosurgery*. 2005;57(2):307–313. doi:10.1227/01.neu.0000166664.19662.43
24. Peng B, Pang X, Li D, Yang H. Cervical spondylosis and hypertension: a clinical study of 2 cases. *Medicine (Baltimore)*. 2015;94(10):e618. doi:10.1097/MD.0000000000000618
25. Delgado-López PD, Castilla-Díez JM. Impact of obesity in the pathophysiology of degenerative disk disease and in the morbidity and outcome of lumbar spine surgery. *Neurocirugía (Astur: Engl Ed)*. 2018;29(2):93–102. doi:10.1016/j.neucir.2017.06.002
26. Longo UG, Denaro L, Spiezia F, Forriol F, Maffulli N, Denaro V. Symptomatic disc herniation and serum lipid levels. *Eur Spine J*. 2011;20(10):1658–1662. doi:10.1007/s00586-011-1737-2
27. Sasani M, Aydın AL, Aytan N, et al. Effect of a hypercholesterolemia as a starting factor on spinal degeneration in rabbits and role of vitamin E (A-tocopherol). *Surg Neurol Int*. 2016;7:36. doi:10.4103/2152-7806.180092
28. Overley SC, Kim JS, Gogel BA, Merrill RK, Hecht AC. Tandem spinal stenosis: a systematic review. *JBJS Rev*. 2017;5(9):e2. doi:10.2106/JBJS.RVW.17.00007
29. Jannelli G, Baticam NS, Tizi K, Truffert A, Lascano AM, Tessitore E. Symptomatic tandem spinal stenosis: a clinical, diagnostic, and surgical challenge. *Neurosurg Rev*. 2020;43(5):1289–1295. doi:10.1007/s10143-019-01154-9
30. Luo CA, Kaliya-Perumal AK, Lu ML, Chen LH, Chen WJ, Niu CC. Staged surgery for tandem cervical and lumbar spinal stenosis: which should be treated first *Eur Spine J*. 2019;28(1):61–68. doi:10.1007/s00586-018-5795-6
31. Yamada T, Yoshii T, Yamamoto N, et al. Clinical outcomes of cervical spinal surgery for cervical myelopathic patients with coexisting lumbar spinal canal stenosis (tandem spinal stenosis): a retrospective analysis of 297 cases. *Spine (Phila Pa 1976)*. 2018;43(4):E234–E241. doi:10.1097/BRS.0000000000002289
32. Yamada T, Yoshii T, Yamamoto N, Hirai T, Inose H, Okawa A. Surgical outcomes for lumbar spinal canal stenosis with coexisting cervical stenosis (tandem spinal stenosis): a retrospective analysis of 565 cases. *J Orthop Surg Res*. 2018;13(1):60. doi:10.1186/s13018-018-0765-6
33. Cao J, Gao X, Yang Y, et al. Simultaneous or staged operation for Tandem spinal stenosis: surgical strategy and efficacy comparison. *J Orthop Surg Res*. 2021;16(1):214. doi:10.1186/s13018-021-02357-x
34. Aydoğan M, Oztürk C, Mirzanli C, Karatoprak O, Tezer M, Hamzaoglu A. Treatment approach in tandem (concurrent) cervical and lumbar spinal stenosis. *Acta Orthop Belg*. 2007;73(2):234–237.
35. Pennington Z, Alentado VJ, Lubelski D, et al. Quality of life changes after lumbar decompression in patients with tandem spinal stenosis. *Clin Neurol Neurosurg*. 2019;184:105455. doi:10.1016/j.clineuro.2019.105455
36. Felbaum DR, Fayed I, Stewart JJ, Sandhu FA. Relief of lumbar symptoms after cervical decompression in patients with tandem spinal stenosis presenting with primarily lumbar pain. *Cureus*. 2016;8(12):e940. doi:10.7759/cureus.940
37. Inoue T, Ando K, Kobayashi K, et al. Primary cervical decompression surgery may improve lumbar symptoms in patients with tandem spinal stenosis. *Eur Spine J*. 2021;30(4):899–906. doi:10.1007/s00586-020-06693-0

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