Lumbar Degenerative Disc Disease and Tibiotalar Joint Arthritis: A 710-Specimen Postmortem Study

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Osteoarthritis is the most common joint disorder, resulting in significant morbidity and disability. The worldwide prevalence of osteoarthritis was estimated at more than 151 million people, according to data published in 2004.¹ In the United States, almost 27 million adults age 25 years and older suffer from clinically apparent disease.² The spine is one of the most commonly affected joints of arthritis, and idiopathic low back pain is the most frequent complaint in the adult population.³ In adults with low back pain, evidence of lumbar intervertebral disc degeneration is often found on radiography.⁴ In 1 study, evidence of disc degeneration was found in 90% of adults age 50 to 59 years.⁵

Degenerative spinal disease most commonly affects the lumbar spine due to its high degree of mobility and weight-loading.⁶,⁷ Clinical⁸ and experimental studies⁹ have suggested that the degenerative changes in the lumbar spine begin in the intervertebral discs. Degenerative disc disease (DDD) results from a continuum of dehydration, degradation, and remodeling of the intervertebral discs and neighboring vertebrae to accommodate the changes in physical loading.¹¹-¹³ This results in disc-space narrowing, disc bulging and herniation, vertebral rim osteophyte formation, and endplate sclerosis.⁷,¹⁴ Symptomatic neural compression may occur, often manifested by localized lower back and extremity pain, as well as sensory loss and weakness of the lower extremities.¹⁵-¹⁷ Changes in posture and gait may result because of altered sensation, and the consequent abnormal force transmission may predispose joints to accelerated wear and arthrosis.¹⁵,¹⁸

Numerous studies have delineated the association between lumbar spinal disorders and lower extremity arthrosis. Of note, research has demonstrated that hip and/or knee pathology and gait alteration may promote low back pain and lumbar disc degeneration.¹⁹-²¹ Although spinal abnormalities, such as scoliosis, may predispose an individual to accelerated hip degeneration,²⁰ no studies have investigated the relationship between lumbar DDD and ankle osteoarthritis.

Ankle arthritis differs from hip and knee arthritis demographically, occurring approximately 9 times less frequently.²¹ The ankle joint is subjected to more weight-bearing force per square centimeter and is more commonly injured than any other joint in the body.²¹ Trauma and/or abnormal ankle mechanics are the most common causes of degenerative ankle arthritis.²² Other potential causes include inflammatory arthropathies, neuropathic arthropathy, infection, and tumor. The purpose of this study was to determine if a relationship exists
between ankle arthrosis and lumbar disc degeneration, and to delineate if one may promote the onset or progression of the other.

**Materials and Methods**

We randomly chose 710 cadaveric specimens from the Hamann-Todd Osteological Collection in Cleveland, Ohio. The Hamann-Todd Collection contains skeletal remains from more than 3000 individuals who died in Cleveland, Ohio between 1893 and 1938. The cohort for this study included 583 male and 127 female cadavers, ranging in age from 17 to 105 years at the time of death. Table 1 shows the breakdown of these specimens according to age group; of the 710 specimens, 306 were of African American ancestry, and 404 were Caucasian.

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Lumbar DDD was graded at each lumbar spinal level by a single examiner using the Eubanks modification of the Kettler and Wilke classification of vertebral endplate osteophytosis:

Grade 0: normal vertebral endplates;

Grade 1: mild arthrosis, with evidence of osteophytic reaction involving up to 50% of the vertebral endplates;

Grade 2: moderate arthrosis, with evidence of osteophytic reaction involving 50% to 100% of the vertebral endplates;

Grade 3: severe arthrosis, with evidence of osteophytic reaction involving 100% of the vertebral endplates. Osteophytes are hypertrophic and bridging the joint space (Figure 1);

Grade 4: complete ankylosis.
Tibiotalar joint osteoarthritis was evaluated by a single examiner using a modification of the Kellgren-Lawrence classification\(^4\) for knee osteoarthritis:

Grade 0: no discernable wear/osteophytes;

Grade 1: 1-mm osteophyte(s) and/or <25% surface wear;

Grade 2: 1- to 2-mm osteophyte(s) and/or 25% to 50% joint surface;

Grade 3: 2- to 3-mm osteophyte(s) and/or >50% joint surface (Figure 2);

Grade 4: multiple large osteophytes and/or definite bony end deformity.

Statistical analysis was performed on the compiled data using Stata software (StataCorp, College Station, Texas). Linear and logistic regression analyses correcting for confounding factors of age, sex, race, and height were performed using a standard \(P\)-value cutoff \((P < .05)\) and 95% confidence interval to determine statistical
significance.

Results

Patients were considered to have osteoarthritis of the tibiotalar joint if either of the extremities measured grade 1 or higher. Of the 710 specimens selected, 14 specimens did not have adequate bone available for bilateral tibiotalar joint measurement, either from extensive bone degradation or amputation. Of the remaining 696 specimens, 586 had some degree of tibiotalar osteoarthritis present (Table 2). Regression analysis showed a significant positive association between right- and left-ankle osteoarthritis (coefficient: 0.491, \( P < .01 \)). Tibiotalar joint arthritis was classified as severe if either extremity had arthrosis of grade 3 or higher. Of the 586 specimens that had tibiotalar joint arthritis, only 16% (97 specimens) had severe tibiotalar joint arthritis.

Data regarding lumbar disc degeneration were available for 516 of the 710 specimens selected, 443 of which showed some disc degeneration. Disc degeneration was most prevalent and significant at the L4-L5 and L3-L4 intervertebral levels (Figures 3, 4). Of these 516 specimens, 30 had degeneration at 1 level, 47 specimens had degeneration at 2 levels, 29 specimens had degeneration at 3 levels, 52 had degeneration at 4 levels, and 285 specimens had degeneration at all 5 lumbar levels. The majority of specimens were found to have some degree of degeneration at all 5 lumbar spinal levels (Figure 5). Severe lumbar DDD was defined as grade 3 or higher osteoarthritis present in at least 1 of the 5 lumbar levels. Of the 516 specimens that showed some degree of disc degeneration, 152 were classified as severe. When stratified by number of spinal levels, only 30% of specimens were found to have evidence of severe arthrosis, the majority of which was located at only 1 lumbar segment (Figure 6).
Figure 3. Percentage of specimens with arthrosis at each intervertebral level.

Figure 4. Mean grade of lumbar disc degeneration by intervertebral level.

Figure 5. Percentage of specimens with lumbar disc degeneration, based on number of lumbar levels involved.

Figure 6. Percentage of specimens with severe lumbar disc degeneration, based on number of lumbar levels involved.
Linear regression analysis of the data showed a statistically significant positive association between lumbar disc degeneration and tibiotalar osteoarthritis (coefficient: 0.844, \( P < .01 \)), even when correcting for confounding factors, such as age, sex, and race (coefficient: 0.331, \( P < .01 \)).

Additional analysis of the data demonstrated that tibiotalar joint arthritis remained significantly associated with lumbar DDD across each lumbar level: L1-L2 (coefficient: 0.269, \( P < .01 \)), L2-L3 (coefficient: 0.283, \( P < .01 \)), L3-L4 (coefficient: 0.299, \( P < .01 \)), L4-L5 (coefficient: 0.240, \( P < .02 \)), L5-S1 (coefficient: 0.167, \( P < .05 \)).

The presence of 3 or more levels of lumbar DDD significantly increased the possibility of developing severe tibiotalar joint arthritis. Lumbar DDD that encompassed 3 levels showed the highest odds for development of severe tibiotalar joint arthritis with an odds ratio (OR) of 20.542 (Table 3).

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<table>
<thead>
<tr>
<th>No. of Lumbar Levels</th>
<th>Odds Ratio</th>
<th>( P )</th>
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<tbody>
<tr>
<td>2</td>
<td>0.40</td>
<td>.104</td>
</tr>
<tr>
<td>3</td>
<td>20.542</td>
<td>.007</td>
</tr>
<tr>
<td>4</td>
<td>11.628</td>
<td>.026</td>
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<tr>
<td>5</td>
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When subjects were compared by decade, the mean grade of tibiotalar joint arthritis was significantly higher than lumbar DDD in specimens who died in their 20s and 30s. This difference was insignificant in the fourth decade, and thereafter the mean value of lumbar DDD surpassed that of tibiotalar joint arthritis (Figure 7).

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In contrast, severe lumbar DDD was more prevalent than severe tibiotalar joint arthritis in individuals age 20 years or older (Figure 8). There were no specimens under age 20 years with severe lumbar DDD or severe tibiotalar joint arthritis.
Logistic regression showed that individuals with severe lumbar disc degeneration had significantly higher odds of developing severe ankle arthritis (OR: 1.93, \( P < .05 \)). Similarly, individuals with severe tibiotalar joint arthritis were just as likely to develop severe lumbar DDD with an OR of 1.97 (\( P < .05 \)).

**Discussion**

Multiple joint involvement in osteoarthritis is well established with a wide range of evidence linking lower extremity joint pathology and lumbar spinal disease. In 1983, Offierski and MacNab\(^{20}\) were the first to describe hip-spine syndrome. In the next year, a study by Sponseller and colleagues\(^{25}\) of pediatric patients after hip arthrodesis further substantiated the association between spine and extremity disease, and demonstrated a continued cause and effect relationship after surgery.

Lumbar spinal degeneration has also been correlated with knee osteoarthritis. Tsuji and colleagues\(^{26}\) reported that degenerative changes in spinal alignment result in increased thigh muscle tension and knee flexion. Furthermore, in their radiographic analysis of 682 individuals, Horvath and colleagues\(^{27}\) also showed that individuals with spinal degeneration had a higher prevalence of knee and hip osteoarthritis.

One might hypothesize from this evidence that lumbar spinal degeneration and ankle arthritis would also be interrelated, given their interconnected role in lower extremity force transmission. Surprisingly, the literature correlating lumbar degeneration and lower extremity osteoarthritis has overlooked this association and has focused solely on the hip and knee. To our knowledge, this study is the first to identify a statistically significant association between tibiotalar joint osteoarthritis and lumbar disc degeneration.

The literature supported analysis of our data. Miller and colleagues\(^{28}\) evaluated disc degeneration in 600 autopsy specimens using the Nachemson\(^{29}\) grading system. This system categorizes disc degeneration into 4 grades based on macroscopic appearance. Miller and colleagues\(^{28}\) reported evidence of degenerative changes as early as the second decade of life, primarily involving the L3-L4 and L4-L5 levels. Of note, the Nachemson\(^{29}\) classification system includes only evidence of marginal osteophytes in grade 4 disease, which was not identified by Miller and colleagues\(^{28}\) until the fourth decade. These results were similar to those in our study, in which the L3-L4 and L4-L5 intervertebral levels were most commonly affected. However, in our study, significant degenerative changes were found in the third decade of life.
In addition, the percentage of specimens with severe disc degeneration increased with each decade (Figure 8). A substantial amount of histologic evidence demonstrates the progression of disc degeneration with age. With increased age, there is a gradual decrease in the osmotic swelling of intervertebral discs and a 2-fold decrease in disc hydration between adolescence and the eighth decade. Furthermore, the nucleus pulposus undergoes progressive fibrosis with a 5-fold decrease in the fixed-charge density of nucleus glycosaminoglycans, and a 2-fold increase in intervertebral disc creep while under compression after age 30 years.

While analyzing our findings, we had difficulty in determining which pathologic condition debuts and, subsequently, affects the other. According to our results, the mean grade of tibiotalar joint arthritis was higher than that of DDD in specimens through the third and fourth decades of life (Figure 7). After the age of 50 years, the mean grade of DDD surpasses that of tibiotalar arthritis. This may be initially interpreted that development of tibiotalar joint arthritis precedes lumbar disc degeneration. Ankle osteoarthritis is relatively rare, and given that the vast majority of ankle osteoarthritis is secondary to trauma, we would expect to see a higher incidence of ankle osteoarthritis in a younger, more active cohort. In addition, given our finding that ankle arthritis is related to lumbar disc degeneration, one could speculate that tibiotalar arthritis at a young age predisposes an individual to developing lumbar degeneration later in life.

However, this conclusion is inherently flawed; closer examination of the data revealed that the mean grade of tibiotalar arthritis and DDD in the third and fourth decades is relatively low, between grade 0 and grade 1 (Figure 7). Therefore, it is difficult to arrive at a conclusion when comparing such small values. Second, we must remember that we are comparing an average value of disc degeneration across all lumbar levels. When a specimen has only 1 disc that is severely degenerated, this value is averaged across all 5 lumbar levels and, thus, the overall mean grade of arthrosis is significantly diminished.

In fact, data from previous studies concur with the second argument. Upper-level lumbar disc degeneration is relatively rare and the vast majority of patients with disc degeneration present with significant disease in only 1 or 2 discs. Analysis of the specimens in this study revealed bony evidence of disc degeneration present at all 5 lumbar levels in over half of the specimens examined (57%). However, the majority of specimens in this cohort exhibit only low-grade degeneration. When specimens were analyzed for severe arthrosis (grade 3 and higher), nearly half of the specimens were found to have severe disease involving only 1 intervertebral disc (Figure 6). Data from Miller and colleagues and the present study show that the upper lumbar levels were relatively spared; the L3-L4 and L4-L5 lumbar levels showed the highest prevalence and severity of degenerative change.

To address this issue, we evaluated the percentage of specimens per decade with severe arthrosis (grade 3 and higher) of at least 1 lumbar intervertebral disc and 1 tibiotalar joint. Severe lumbar disc degeneration was found to be more prevalent than severe ankle arthritis in individuals age 20 years or older (Figure 8). Therefore, we postulate that significant degenerative changes in the lumbar spine precede the development of severe ankle arthritis.

One can further speculate that sequelae from lumbar disc degeneration may lead to the development of tibiotalar arthritis, given our finding that severe lumbar degeneration predisposes an individual to the development of ankle arthritis. Because significant lumbar disc degeneration has long been known to result in both spinal nerve and cord compression, we hypothesize that this resultant neurocompression promotes altered gait and translation of atypical forces to the ankle and foot, thus predisposing to the onset and/or progression of osteoarthritis. In support of this hypothesis, Morag and colleagues demonstrated that neurologic compression produced an altered posture and gait because of lost motor function and afferent proprioceptive sensation. This form of neurologic compromise may exert atypical forces upon the foot and ankle, predisposing the joint to accelerated wear and primary arthrosis.
In addition, DDD involving 3 or more lumbar intervertebral levels was found to significantly increase the likelihood of the subject having severe tibiotalar joint arthritis. Provided that lumbar disc degeneration typically involves significant degeneration at 1 level, we assume that significant arthrosis at 3 or more levels correlates to an overall more severe DDD with a higher corresponding likelihood of neural compression. However, compression of peripheral lower extremity nerves has been shown to result in neuropathic arthropathy akin to the diabetic Charcot foot. This could be a possible mechanism of accelerated ankle arthritis, but this study did not examine soft-tissue disease nor take into account other medical comorbidities of each specimen, including genetic predispositions towards osteoarthritis.

It should be noted that the aforementioned causative relationship between lumbar disc degeneration and tibiotalar arthritis is speculative and cannot be demonstrated definitively by this investigation. We acknowledge limitations of this study and the need for further research of the possible causative mechanism(s) of accelerated ankle arthrosis secondary to lumbar spinal disease. Ideally, the questions posed by our report would be answered via a large prospective cohort study that utilized both serial imaging and autopsy analysis. Unfortunately, this form of study is logistically and financially difficult to perform.

This was a retrospective cadaveric study in which determination of arthrosis severity was based solely on bony evidence. Therefore, the role of soft-tissue disease in the pathogenesis of arthrosis of the lumbar spine and tibiotalar joint could not be assessed, nor could definitive associations to clinically symptomatic disease. We made the assumption that progression of bone degeneration in both the lumbar spine and tibiotalar joint corresponded equally to the associated soft-tissue changes. Given this assumption, we cannot definitively conclude that degeneration of the lumbar spine precedes that of the ankle, because the absence of magnetic resonance imaging or fresh autopsy specimens in our study misses the early degenerative changes in the discs that precede the bony alteration measured in our study. Furthermore, readers should note that since this study compared only bone morphology, no emphasis was placed on clinical manifestation of lumbar disc degeneration or tibiotalar joint arthritis. As mentioned earlier, radiologic evidence of disc degeneration was found in 90% of adults age 50 to 59 years, according to a study by Hult; however, it is important to note that not all individuals studied were symptomatic clinically. Unfortunately, medical records were not available for the bony specimens, and clinical correlations could not be assessed during this investigation.

Furthermore, no special attention was given to other pathologic conditions observed during specimen measurement. The presence of diseases, such as osteoporosis, spondylolysis, or previous traumatic injury, may have had implications in the resultant joint degeneration. Finally, the evaluation of arthrosis was performed subjectively without measuring reliability. However, the present analysis includes a large sample, each joint type was reviewed by a single examiner, and used a classification system that was modeled on a validated grading system. Ideally, multiple individuals should have been used for each type of measurement, with subsequent analysis of intraobserver and interobserver reliability.

**Conclusion**

Based on our study of a large population of adult skeletal specimens, we ascertained that lumbar intervertebral disc degeneration and tibiotalar osteoarthritis are associated. The prevalence of severe lumbar disc degeneration was higher than that of tibiotalar joint arthritis in individuals age 20 years or older. This may suggest that gait changes from disc degeneration or neural compression in the lumbar spine may play a role in the development of ankle osteoarthritis. Additionally, subjects with severe disc degeneration were twice as likely to develop
significant tibiotalar osteoarthritis. This must be considered in the differential when treating patients with degenerative changes of the lumbar spine and leg pain.

Key Info

Figures/Tables

References

References


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