Disk Degeneration in Lumbar Spine Precedes Osteoarthritic Changes in Hip

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Abstract

It is not clear whether spinal degeneration leads to hip arthritis, or hip arthritis leads to spinal degeneration. We conducted a study to determine which degenerative process precedes the other.

We examined 340 cadaveric human specimens from the Hamann-Todd Osteological Collection (Cleveland, Ohio). Lumbar endplate degeneration was graded on a scale of 0 to 4, and hip degeneration on a scale of 0 to 3. Linear regression was used to analyze the relationship between hip osteoarthritis (OA) and lumbar degenerative disk disease (DDD). Exact tests were used to identify differences in each age group.

Hip OA was significantly associated with endplate degeneration at the L1, L3, and L5 levels (P<.02). Of the specimens younger than 29 years, 35% had evidence of DDD in at least 1 lumbar level, and 17% of hip OA changes. At 70 years, 100% of the specimens had evidence of DDD and 50% of hip OA changes. There was a significant association between lumbar DDD and hip OA changes (P<.05). Early lumbar DDD was twice as common as hip OA changes in the early 20s age range.

These findings suggest that lumbar degeneration precedes hip degeneration and may be a causative factor for hip OA.

Osteoarthritis (OA), the clinical syndrome of joint pain and dysfunction caused by joint degeneration, is the most common joint disease. There are no consistently effective methods for preventing OA or slowing its progression and symptomatic treatments provide limited benefit for many patients. OA disables about 10% of people older than 60 years. It occurs frequently in the hip, spine, and knee but rarely in the ankle, wrist, elbow, and shoulder. The most important universal risk factor is age. OA incidence rises precipitously with age. As a result, the prevalence and burden of the disorder are increasing rapidly. The disease process is aggravated by changes that come with advancing age: shrinkage and loss of cartilage space, and osteophyte formation caused by degeneration.[1-3]

Analysis of the impact of OA on the spine and hip raises several questions: (1) What is the relationship between hip OA and lumbar DDD? (2) Do OA changes in one region precede degenerative changes in the other? (3) How does the incidence of hip OA and spine OA increase progressively with age? The relationship between hip and spine degeneration was first described by Offierski and MacNab.[4] It was hypothesized that degeneration in one area could lead to decompensation in the other; however, which precedes the other is unclear. Answering these questions could lead to effective methods of diagnosing and preventing OA and slowing its progression.

We studied a large number of skeletal specimens to objectively evaluate the degeneration that occurs in the hip and spine and to determine which degenerative process precedes the other.

Materials and Methods

The Hamann-Todd Osteological Collection in Cleveland, Ohio contains more than 3300 treated and dried human specimens representing people who died in Cleveland between 1893 and 1938. We randomly selected 340 of these specimens (298 male, 42 female) for examination. Age ranged from 18 to 105 years.

Figure 1. Lumbar vertebrae and hip specimens from Hamann-Todd Osteological Collection.

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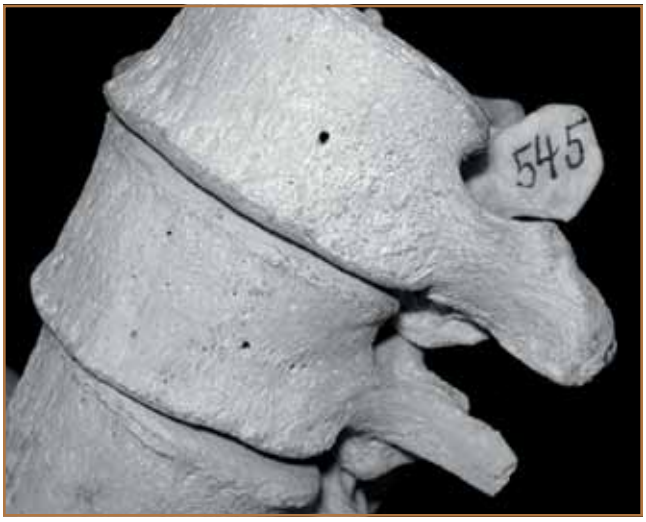

Figure 2. Lumbar vertebrae with grade 0 arthrosis.

Figure 3. Lumbar vertebrae with grade 3 arthrosis.

Figure 4. Hip specimen with grade 1 arthrosis.

Figure 5. Hip specimen with grade 2 arthrosis.

Forty specimens were from blacks and 300 from whites.

Two examiners subjectively measured the lumbar vertebra and the corresponding hip joints of the gross specimens for evidence of endplate arthrosis in the lumbar spine and OA changes in the hip (**Figure 1**). Disk degeneration and arthrosis at all lumbar levels (L1-L5) were measured using the classification of Wilke[5] and Kettler.[6] Degenerative disease was graded on a scale of 0 (no arthrosis) to 4 (complete ankylosis) using previously defined markers.[7-9] The vertebral endplates were graded on a scale of 0 to 4 as well:

0 Normal facet joints or vertebral endplates (**Figure 2**).
1 Mild arthrosis with evidence of osteophytic reaction involving up to 50% of facet joints or vertebral endplates.
2 Moderate arthrosis with evidence of osteophytic reaction involving 50% to 100% of facet joints or vertebral endplates.
3 Severe arthrosis with evidence of osteophytic reaction involving 100% of facet joints or vertebral endplates and hypertrophic osteophytes bridging the joint space (**Figure 3**).
4 Complete ankylosis.

Of the 340 specimens, 334 had bilateral symmetric OA findings and 6 unilateral OA findings; the latter had only 1 femur. An arbitrary but simple and straightforward 0-to-3 scheme with high (>95%) interrater reliability was used to grade all hips for degeneration:

0 No evidence of degenerative changes.
1 Less than 50% osteophyte formation around subcapital ring and mild spurring (**Figure 4**).
2 More than 50% osteophyte formation around subcapital ring with more extensive spurring (**Figure 5**).
3 Global degeneration and multiple osteophyte complexes promoting head deformity.

In addition, the age, sex, and race of each specimen were recorded. Then, an analysis of variance was performed with stepwise multiple linear regression models to test the relationship between hip OA and lumbar endplate degeneration. Fisher

exact tests were used to identify and compare the degenerative changes in hip and spine in each age-decade group. The standard significance cutoff of P<.05 was used.

Results

Three hundred forty specimens were examined. **Table I** shows the full distribution of specimens by age decade, sex, and race.

A stepwise linear regression analysis revealed that in all specimens, hip OA was significantly associated with endplate degeneration at the L1, L3, and L5 levels (P<.02). Fisher exact tests demonstrated significant differences in each age group (P<.01). Of the 19 specimens younger than 30 years, 7 had evidence of degenerative disk disease (DDD) in at least 1 lumbar level, and 3 had evidence of hip OA changes. In all age groups, lumbar DDD was 2 to 5 times more common than hip OA changes. Evidence of DDD in lumbar vertebra was present in all specimens older than 60 years, and evidence of hip OA changes was present in half the specimens older than 60 years. **Table II** summarizes these findings. Overall, the degenerative changes in the lumbar vertebra were 2 to 6 times more common than the degenerative changes in the hips.

Analysis of the relationship between DDD level and hip OA revealed that grade 0 or grade 1 DDD at any lumbar level was associated with hip OA changes in less than 16% of the corresponding specimens, whereas grade 2, 3, or 4 DDD was associated with hip OA changes in more than 30% of the corresponding specimens.

Table I. Age, Sex, and Race of Sampled Specimens

Age Group, y	Sex		Race		Total No. of Specimens
	Female	Male	White	Black	
15-24	2	6	3	5	8
25-34	6	26	23	9	32
35-44	14	71	80	5	85
45-54	12	100	97	15	112
55-64	4	63	65	2	67
65-74	3	24	26	1	27
>75	1	8	6	3	9
Total	42	298	300	40	340

Table II. Hip and Spine Degenerative Changes in Specimens

Age Group, y	No. of Specimens		
	With Hip Osteoarthritis	With Lumbar Vertebrae Degeneration	Total[a]
<29	3	7	19
30-39	7	48	60
40-49	17	98	103
50-59	17	85	87
60-69	20	51	52
>70	7	19	19

[a]Total number of specimens in the age group.

Discussion

The term *hip-spine syndrome* was first used by Offierski and MacNab[4] to describe concurrent hip and spine disease in the older population presenting with variable symptoms that include hip pain, pain over the anterior aspect of the thigh caused by L3-L4 segment instability or L4 root involvement, a limp, groin pain, and a mobility deficit. The authors further classified hip-spine syndrome into simple, secondary, and complex. Given the variable presentation of the disease, diagnosis can be perplexing. In addition, it is not clear how the disease progresses and whether spinal degeneration leads to hip arthritis, or hip arthritis leads to spinal degeneration.

The biomechanical characteristics of the spine and the hip joint are altered by soft-tissue and bony changes, including intervertebral disk degeneration, facet joint arthrosis, and hypertrophy of the ligamentum flavum.[10,11] Studies have suggested that the contribution of the lumbar spine relative to that of the hip is reduced in patients with low back pain, and, as a result, these patients use various strategies to try to compensate for their limited hip and lumbar spine motions, significantly altering lumbar spine–hip joint coordination, particularly in those with a positive straight leg raise sign.[12,13] Patients with a history of low back pain may therefore use more hip motion during early forward bending. Esola and colleagues[14] concluded that the pattern of lumbar spine motion and hip motion during forward bending differs between people with a history of low back pain and people who are healthy. These findings suggest that altered biomechanics in the spine may in fact lead to early degeneration in the hip joints. Our study results showed that early degeneration is more common in the lumbar spine than in the hip joint and that progressively increasing degeneration in the lumbar spine is associated with hip OA.

Saunders and colleagues[15] conducted a clinical and radiographic study of the spine in 150 subjects (75 patients with hip OA, 75 controls) and concluded that the radiographic degenerative changes in the lumbar spine were 2 to 3 times more frequent in the patients with hip OA than in the controls. Sponseller and colleagues[16] evaluated 53 patients younger than 35 years who had hip arthrodesis at least 20 years prior to their study, and reported a high incidence of low back pain. Fitzgerald and Newman[17] studied 43 patients with spondylolisthesis and found that almost 20% had hip OA severe enough to require surgery. Therefore, hip degeneration and spinal abnormalities appear to be related. These findings make it clear that degeneration in the hip can lead to changes in the spine, and degeneration in the spine can lead to changes in the hip. The question, then, is which pathology occurs first in normal people and predisposes them to the other pathology.

Other clinical studies have had variable findings. McNamara and colleagues[18] reported that of 14 patients who underwent joint arthroplasty for lower extremity and spine degenerative disease, 7 (50%) required lumbar decompression for complete

remission of pain. Similar findings reported by Bohl and Steffee[19] suggested that the main pathology was related to the lumbar spine, not the hip joint. In another study, however, Ben-Galim and colleagues[20] evaluated 25 patients (age range, 32-84 years) with hip and lower back pain and concluded that hip OA caused abnormal gait and spinal sagittal alignment and was associated with degenerative lumbar spine changes causing pain.

These clinical studies had sample sizes of 14 to 150, and the patients had preexisting hip or spine diseases, so there was some selection bias. A normal population must be studied to determine the actual incidence and progression of degenerative changes in hips and spines, and whether changes in the spine lead to changes in the hip, or changes in the hip lead to changes in the spine. In the present cross-sectional study, we morphoanatomically compared a much wider array of representative US individuals, from adolescents to very old adults. We examined the incidence of degenerative changes at each lumbar level and in the hips to compare and correlate the changes occurring over age in a normal population. Degenerative changes were 2 to 5 times more common in the lumbar spine than in the hips. Our study results seem to indicate that degenerative changes in the lumbar spine lead to altered biomechanics in as early as the fourth decade causing soft-tissue alterations and gait changes that can be a significant causative factor leading to hip OA.

As this was a retrospective, cadaveric study, symptom severity could not be correlated with degeneration of the lumbar spine or hip. Ideally, a prospective cohort study would follow a large group of patients with serial imaging studies and autopsy analysis after death. Such a study would provide the most satisfactory answers to the present questions. The problem is that such a study would be logistically difficult and financially prohibitive. The present study provides the most reliable answers regarding the degenerative changes in the anatomy of lumbar spine and hip with advancing age in a normal population. This information can be used as reference for further clinical studies. In addition, genetics and other external factors may predispose individuals to lumbar spondylosis and hip OA. In such cases, the altered biomechanics of the axial skeleton may lead to early lumbar degeneration compared with hip OA. Thus, further clinical studies are needed to clearly establish whether it is the altered biomechanics or the lumbar DDD itself that causes hip OA.

Although the grading system used for hip OA is arbitrary and has not been validated, the conclusions of this study rest on the distinction between no OA (grade 0) and some OA (grade 1 or higher), not on the distinction between grades 1 and 2, or between grades 2 and 3, for which this simple grading system showed high (>95%) interrater reliability.

In conclusion, we examined the progression of degeneration in the lumbar spine and in the hip, and the association between the 2 processes. Our cadaveric skeletal analysis showed a significant association between lumbar DDD and hip OA. Lumbar DDD changes preceded hip OA changes, with one-third of the specimens in the 20s age group showing evidence of early lumbar DDD changes but only 17% of those specimens showing evidence of hip OA changes. Lumbar DDD changes were present in all the specimens older than 70 years, but hip OA changes were present in only 50% of those specimens. These findings suggest that lumbar degeneration may precede hip degeneration and that lumbar DDD may lead to the development of hip OA.

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