

Surgical Correction of Severe Lumbar Spine Instability and Sagittal Deformity in a Patient with Rheumatoid Arthritis Without the Utilization of an Osteotomy

Jang Won Yoon¹ and Sassan Keshavarzi^{2*}

¹Department of Neurosurgery, Mayo Clinic, Jacksonville, FL 32224, USA

²California Brain and Spine Institute, Bakersfield, CA 93301, USA

Abstract

Purpose: Complications from RA can involve the thoracolumbar spine and cause severe spine instability and sagittal deformity. We present a surgical strategy to restore sagittal balance in a patient with chronic rheumatoid arthritis.

Methods: A retrospective chart review was performed.

Results: We report the case of a 53-year-old woman with RA and severe back pain. She had dynamic instability of her lumbar spine with spondylololthesis at L3-L4 and L4-L5 as well as hypermobility at the C1-2 level without rheumatoid pannus. On 36-inch standing films, she had a sagittal vertical axis (SVA) of 219 mm. She underwent anterior and posterior instrumented fusion to restore her sagittal imbalance, which resulted in excellent relief of her back pain and marked improvement in ambulation and function.

Conclusion: Patients with RA can develop severe sagittal deformity that could be the source of back pain. In patients with advanced RA, chronic inflammation could lead to ligament laxity that can result in spinal deformity under axial load. This deformity is amenable to reconstruction without the utilization of an osteotomy, which can produce excellent pain relief and restoration of function.

Keywords: Rheumatoid arthritis; Rheumatoid factor; Methotrexate; Ant-CCP; Spine surgery; Deformity; Sagittal imbalance; Spine instrumentation; Osteotomy

Introduction

Rheumatoid arthritis (RA) is a debilitating autoimmune disorder affecting 0.5% to 1.5% of the population in the USA [1]. Patients commonly present with involvement of joints in their hands, wrists, and ankles, but in advanced forms, it can lead to the destruction of major joints, including the knee, hip, shoulder, and elbow [2]. It is estimated that 43% to 88% of RA patients suffer from cervical spine involvement: atlantoaxial subluxation, atlantoaxial impaction, and subaxial subluxation have been described in the literature [3]. Less attention has been directed at lumbar ligament laxity and facet destruction. We present a patient with advanced RA with severe cervical and lumbar hypermobility. The lumbar ligament laxity and facet destruction resulted in severe sagittal deformity. The surgical strategy had acceptable results with reduction, stabilization, resolution of back pain and restoration of function.

Case Report

A 53-year-old female with advanced RA (cyclic citrullinated peptide antibody titer >250) and chronic back pain presented with eight years of debilitating lower back pain with radiation into the left lower extremity. She was closely followed by her rheumatologist and treated with 12.5mg methotrexate weekly and 2mg prednisone daily. From 2004 to 2012, there was progression of her L4-L5 grade 1 anterolisthesis to a grade 3 anterolisthesis as well as development of a grade 2 anterolisthesis at L3-L4 (Figures 1A and 1B). Computed tomography (CT) of her lumbar spine demonstrated a complete reduction of her standing olisthesis into normal alignment in the supine position (Figure 1C). Consistent with the extent of her dynamic instability, there was severe widening of bilateral facet at L3-L4 and L4-L5 (Figure 2). She had a positive sagittal vertical axis (SVA) of 219 mm on a 36-inch standing radiograph (Figure 3A).

Senior author (S.K.) offered the patient anterior lumbar interbody fusion (ALIF) from L3 to S1 and T10 to pelvis posterior instrumented

fusion. After the three-level ALIF, a standing film demonstrated a reduction of her SVA from 219 mm to 48 mm (Figure 3B). On 10-month follow-up 36-inch standing radiograph, her sagittal balance was completely restored and maintained (Figures 3C and Figure 4). At her 15-month follow-up, she was reluctant to obtain further imaging, but continued to ambulate with clinical maintenance of her sagittal correction with excellent pain relief (Figure 5). Unfortunately, she was diagnosed with stage II anal cancer and had to undergo chemotherapy and radiation. She was recently sent to hospice.

Discussion

Studies have found that 40% of patients with RA suffer from chronic low back pain, 19% from leg pain, 14% from leg numbness, and 12% from intermittent claudication [3]. Scoliosis and olisthesis were found in 28% and 23% of these patients, respectively. Despite the high prevalence of chronic lumbar pathology in RA patients, their lumbar spine instability and sagittal imbalance has not been extensively discussed in the literature.

It has been theorized that discovertebral destruction in RA is a consequence of spinal instability caused by apophyseal arthritis and ligament laxity [4,5]. Resnick postulated that apophyseal joint instability and vertebral body osteoporosis contribute to the formation of subchondral nodes. Over time, continued inflammation can lead

*Corresponding author: Sassan Keshavarzi, M.D., California Brain and Spine Institute, 2701 Chester Ave #102, Bakersfield, CA 93301, Tel: 844-637-8363; Fax: 844-637-8332; E-mail: skeshavarzi@cabrainandspine.com

Received March 16, 2017; Accepted March 27, 2017; Published March 29, 2017

Citation: Yoon JW, Keshavarzi S (2017) Surgical Correction of Severe Lumbar Spine Instability and Sagittal Deformity in a Patient with Rheumatoid Arthritis Without the Utilization of an Osteotomy. J Spine 6: 363. doi: [10.4172/2165-7939.1000363](https://doi.org/10.4172/2165-7939.1000363)

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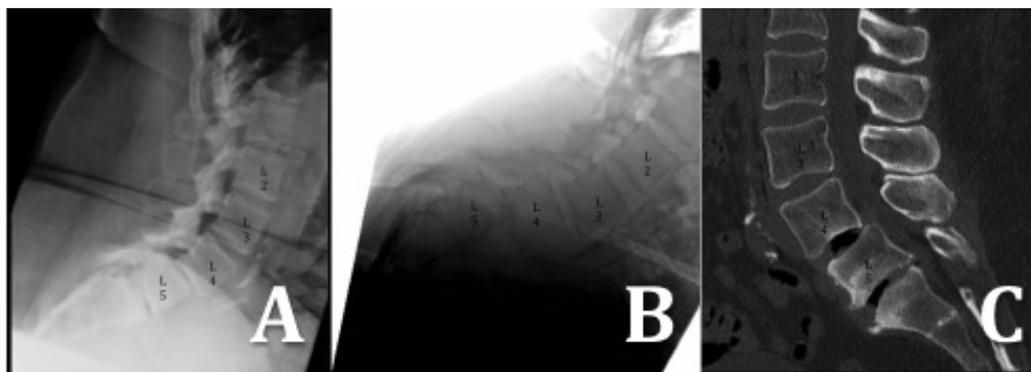


Figure 1: A) Standing x-ray of the lumbar spine demonstrates grade 1 anterolisthesis of L4 on L5 in 2005. B) In 2013, spondylolisthesis progressed to grade 3 anterolisthesis L4 on L5 and grade 2 anterolisthesis of L3 on L4. C) CT in 2013 demonstrates a near complete reduction of her olisthesis at L3/4 and L4/5 when the patient is lying supine.

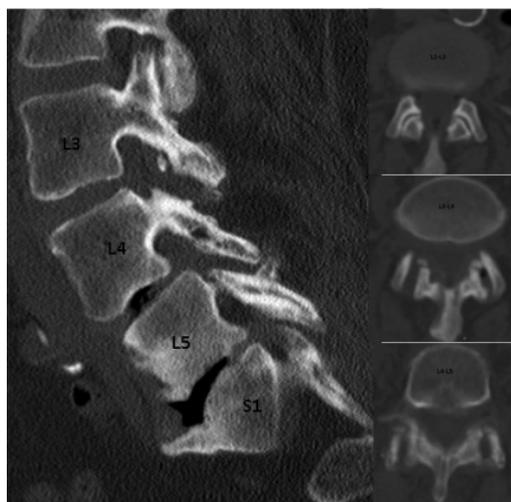


Figure 2: CT scan of her lumbar spine demonstrates intact C2-3 facets, however, there was severe widening of bilateral facet at L3-L4 and L4-L5.

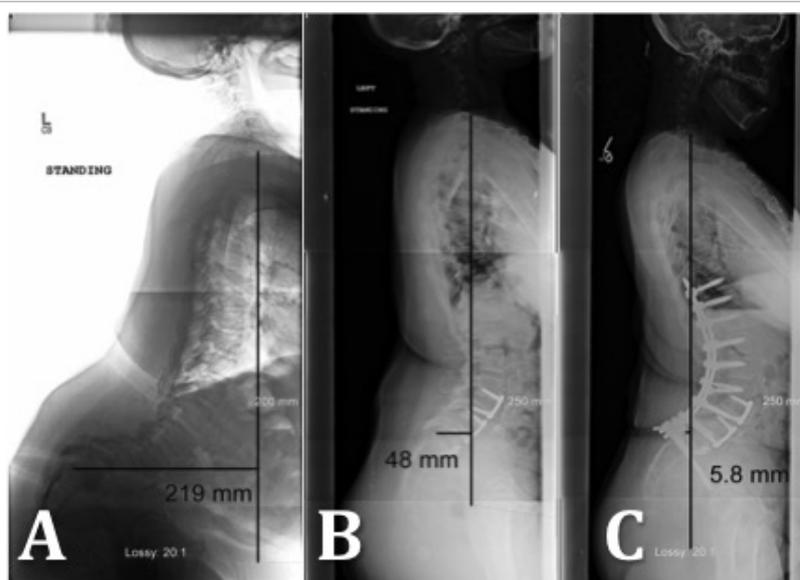


Figure 3: A) Preoperative standing x-ray of the spine demonstrates sagittal vertical axis (SVA) of 219 mm. B) After a three-level ALIF at L3/4, L4/5 and L5/S1, SVA decreased from 219 mm to 48 mm. C) 36-inch standing radiograph taken at 10-month follow-up. She underwent stage two posterior instrumented fusion from T10 to the pelvis with a near complete resolution of her sagittal deformity and a SVA reduced to 5.8 mm.



Figure 4: Post-operatively the patient's deformity is completely restored and SVA is now 5.8 mm. The patient is now standing upright and reports a complete relief of her back pain.

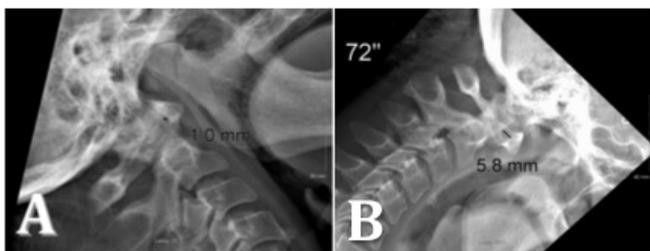


Figure 5: (A and B) Flexion-extension film of her cervical spine demonstrates atlantoaxial hypermobility. The atlanto-dental index increased from 1.0 mm to 5.8 mm on flexion/extension x-ray.

to disc space loss, vertebral lesions, and reactive bone sclerosis [4]. Kawaguchi hypothesizes that facet joints and endplates may be the targets in RA given the fact that: 1) the structure of the facet joints in the spine is the same as the peripheral joints and 2) the endplates are predominantly composed of type II hyaline collagen, which has a similar biochemical structure to synovial joints. T cells (predominantly Th1 cells), B cells, plasma cells, natural killer cells, dendritic cells, and mast cells become activated by cytokines and invade synovial joints [6]. The autoantibodies lead to further activation of the complement system and neutrophils. Synovial fibroblasts, macrophages, and T cells secrete interleukin-1, interleukin-17 and tumor necrosis factor. Matrix metalloproteinases and collagenases become activated downstream and lead to osteoclast activation and ultimately destruction of adjacent cartilage, tendons, and bone [7,8]. The ongoing inflammatory response leads to ligamentous laxity and facet involvement that in advanced RA may lead to gross spinal instability [9]. This was well demonstrated in our patient both in the lumbar (Figure 1) and cervical spine (Figure 5).

Positive sagittal imbalance has been established as a clinically

relevant parameter in the treatment algorithm for back pain and functional outcome [10-14]. Reduction of theolisthesis, correction of the sagittal deformity and stabilization of the lumbar spine were the goal of surgery. Our surgical strategy was to take advantage of the complete reduction of the lumbar spine in the supine position and to fix that reduction with multi-level ALIF. This allowed us to get significant SVA correction without the employment of an osteotomy. Her spine remained flexible secondary to ligamentous laxity and facet destruction from advanced rheumatoid arthritis as evidenced by complete reduction of her olisthesis in supine position. In rigid fixed spinal deformity, osteotomy may be required to restore lumbar lordosis and SVA. Approximately 30 degree of correction is expected with pedicle subtraction osteotomy (PSO) at one level [15]. Although, 3-column osteotomy is a powerful tool, it is associated with significant surgical complications. Smith et al. reported 78% surgical complications that underwent 3-column osteotomy in 82 patients with rigid fixed deformity [16]. Therefore, in patients with advanced rheumatoid arthritis who maintains flexibility, the surgical correction of sagittal deformity may be achieved without adding osteotomy. Posterior fusion from T10 to the pelvis was performed to support the anterior construct and maintain her correction. Studies have shown RA patients on methotrexate are at increased risk of pseudoarthrosis [17]. Although there are many strategies to the extent of posterior fusion, we opted to fuse from T10 to pelvis to increase the chance of fusion and to maintain the correction of sagittal imbalance. This case highlights the importance of obtaining global images and dynamic studies of the spine to make the correct diagnosis. This is especially true for patients with systemic illnesses such as RA that can lead to advanced degeneration of the spine in multiple locations.

Patients with advanced RA can develop lower back pain secondary to gross lumbar spine instability. There are many surgical strategies that can be employed to achieve restoration of SVA imbalance and spine stabilization in patients with advanced RA. We present one of many solutions that can be utilized by spine surgeons. Correction of sagittal balance with lumbar stabilization can lead to relief of back pain and the restoration of function.

Acknowledgement

The authors would like to thank Victoria L. Jackson, MLIS, ELS (Academic and Research Support, Mayo Clinic, Jacksonville, FL) for her editorial assistance in the preparation of this manuscript.

Competing Interests and Funding

No conflict of interest is reported.

No funding was received.

References

1. Lawrence RC, Helmick CG, Arnett FC, Deyo RA, Felson DT, et al. (1998) Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. *Arthritis Rheum* 41: 778-799.
2. Mesfin A, Dafrawy MHE, Jain A, Hassanzadeh H, Kostuik JP, et al. (2015) Surgical outcomes of long spinal fusions for scoliosis in adult patients with rheumatoid arthritis. *J Neurosurg Spine* 22: 367-373.
3. Kawaguchi Y, Matsuno H, Kanamori M, Ishihara H, Ohmori K, et al. (2003) Radiologic findings of the lumbar spine in patients with rheumatoid arthritis, and a review of pathologic mechanisms. *J Spinal Disord Tech* 16: 38-43.
4. Resnick D (1978) Thoracolumbar spine abnormalities in rheumatoid arthritis. *Ann Rheum Dis* 37: 389-391.
5. Martel W (1977) Pathogenesis of cervical discovertebral destruction in rheumatoid arthritis. *Arthritis Rheum* 20: 1217-1225.
6. Firestein GS (2005) Immunologic mechanisms in the pathogenesis of rheumatoid arthritis. *J Clin Rheumatol* 11: S39-44.

7. Muller-Ladner U, Pap T, Gay RE, Neidhart M, Gay S (2006) Mechanisms of disease: The molecular and cellular basis of joint destruction in rheumatoid arthritis. *Nat Clin Pract Rheumatol* 1: 102-110.
8. Ainola MM, Mandelin JA, Liljestrom MP, Li TF, Hukkanen MV, et al. (2005) Pannus invasion and cartilage degradation in rheumatoid arthritis: involvement of MMP-3 and interleukin-1beta. *Clin Exp Rheumatol* 23: 644-650.
9. Mallory GW, Halasz SR, Clarke MJ (2014) Advances in the treatment of cervical rheumatoid: Less surgery and less morbidity. *World J Orthop* 5: 292-303.
10. Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, et al. (2005) The impact of positive sagittal balance in adult spinal deformity. *Spine* 30: 2024-2029.
11. Akbar M, Wiedenhofer B (2011) Sagittal deformity. Basic principles of surgical strategies. *Der Orthopade* 40: 661-671.
12. Blondel B, Schwab F, Ungar B, Smith J, Bridwell K, et al. (2012) Impact of magnitude and percentage of global sagittal plane correction on health-related quality of life at 2-years follow-up. *Neurosurgery* 71: 341-348.
13. Schwab F, Patel A, Ungar B, Farcy JP, Lafage V (2010) Adult spinal deformity-postoperative standing imbalance: how much can you tolerate? An overview of key parameters in assessing alignment and planning corrective surgery. *Spine* 35: 2224-2231.
14. Djurasovic M, Glassman SD (2007) Correlation of radiographic and clinical findings in spinal deformities. *Neurosurg Clin N Am* 18: 223-227.
15. Popa I, Oprea M, Andrei D, Mercedesz P, Mardare M, et al. (2016) Utility of the pedicle subtraction osteotomy for the correction of sagittal spine imbalance. *Int Orthop* 40: 1219-1225.
16. Smith JS, Shaffrey CI, Klineberg E, Lafage V, Schwab F, et al. (2017) Complication rates associated with 3-column osteotomy in 82 adult spinal deformity patients: retrospective review of a prospectively collected multicenter consecutive series with 2-year follow-up. *J Neurosurg Spine* 17: 1-14.
17. Gerster JC, Bossy R, Dudler J (1999) Bone non-union after osteotomy in patients treated with methotrexate. *J Rheumatol* 26: 2695-2697.