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# Lumbar disc herniation in systemic lupus erythematosus: A case report

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## ABSTRACT

The most frequent neurological manifestation in SLE (Systemic Lupus Erythematosus) is cognitive disorder, so lumbar disc herniation in SLE is uncommon. Disc herniation is a degenerative disc disorder (DDD) that is often correlated with aging. However it can be caused by an inflammatory reaction. TNF-α (Tumor Necrosis Factor-alpha) is an essential pro-inflammatory cytokine in DDD-related SLE. We present the case of a 26-year-old female with SLE and disc herniation L5-S1.

**Keywords:** disc herniation, systemic lupus erythematosus, tumor necrosis factor-alpha

## INTRODUCTION

Bilateral lower limb weakness in SLE is a rare neurological manifestation because the most common finding in the present study is cognitive disorders, including delirium, dementia, and mild cognitive impairment (57.89%) [1]. Some theories of its pathogenesis are BBB (Blood-Brain Barrier) disruption, complement activation, cytokine effects, autoantibody production and immune complex (IC) deposition that cause injury of the cerebral vessels and impairment in neuronal function [2,3].

The weakness can be related to CNS (Central Nervous System) and PNS (Peripheral Nervous System) involvement, and we reported a disc herniation L5-S1. Disc herniation is a degenerative process of intervertebral discs and is generally found in average 40-year-old patients [4]. This is a case report of a 26-year-old female with lumbar disc herniation and SLE.

## CASE REPORT

A 26-year-old female with SLE visited the rheumatology clinic due to bilateral lower limb weakness one week ago. She also had nausea and felt her feet hurt. There was neither urinary nor fecal incontinence. In these three months, she was on SLE therapy (Methylprednisolone 16 mg/12 hours, Hydroxychloroquine 200 mg/day, Methotrexate (MTX) 7.5 mg/week, Folic acid 5 mg/week (a day after taking MTX)). Her ANA, Anti-RNP, and Anti-Smith antibodies were positive.

Upon arrival at our rheumatology clinic, the blood pressure, respiratory rate, heart rate, and tympanic membrane temperature were normal. The patient denied having trauma before the weakness and pain. The patient was oriented well, had a good mental health status, and was well nourished. Neurological examination showed decreased muscle power of the bilateral lower limbs and scored 3/3 (MRC scale). The deep tendon reflex (knees reflex, ankle reflex) and sensibility of the bilateral lower limb were diminished. There was also no Babinski sign elicited. Blood tests were normal, including complete cell count, blood sugar, and a biochemical panel. Regardless, the NLR (Neutrophil-to-Lymphocyte Ratio) was high (3.37). The MRI spine showed disc herniation L5-S1 without any sign of myelitis (Figure 1).

Based on her MRI spine results, she was referred to a neurologist and was given an oral 100 mg of Gabapentin and an injection of 500 mcg/ml of Mecobalamin for 12 hours. Oral 30 mg of Lansoprazole was given for her nausea, and she continued her SLE therapy with the same regimens as before. Her final diagnosis was lumbar disc herniation and SLE. Finally, her pain improved, and she was discharged and went to the neurologist for further treatment.

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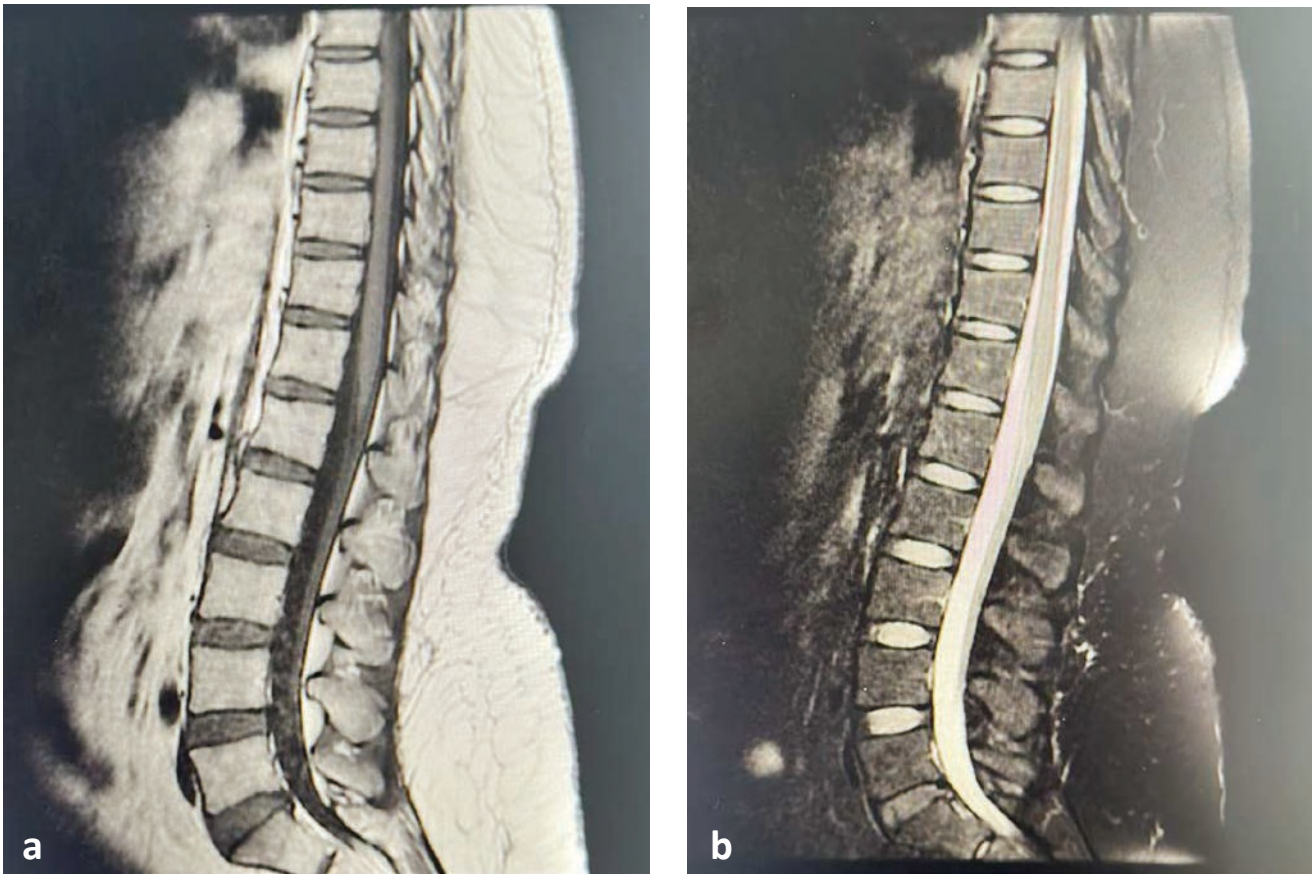


**FIGURE 1.** MRI of the spine indicated there was a disc herniation L5-S1
a) Sagittal, T1, b) Sagittal, T2 fat saturated

## DISCUSSION

We present the case of a 26-year-old female with lumbar disc herniation in SLE. She was diagnosed with SLE by 3 months ago with ANA, Anti-RNP, and Anti-Smith antibodies were positive. There are still a few studies, in addition to spinal cord and the spine involvement commonly found is myelitis. Furthermore, it only occurs in 1-2% of cases within the first five years after disease onset. It is associated with high titers of ANA, anti-double-stranded DNA antibodies, and hypocomplementemia, leading to arterial thrombosis [5,6]. Its symptoms are loss of strength in the lower limbs, swelling of the affected area, fever, acute urinary retention, and abdominal or lumbar pain [7].

Lumbar disc herniation is a part of DDD that is not only because of aging but also an inflammatory reaction [8]. It has been characterized by the infiltration of CD68 macrophages, T cells (CD4, CD8), and neutrophils in blood vessels and nociceptive nerve fibers [9]. They release cytokines, like TNF-α, IL-1 α/β, IL-6, IL-17, IL-8, IL-2, IL-4, IL-10, and IFN-γ [10]. TNF-α is the crucial cytokine that impairs the morphology, cortical contractility, hydraulic permeability, and stiffness of the nucleus pulposus cells [11]. Other than that, TNF-α also promotes ECM (Extracellular Matrix) degradation, apoptosis of intravertebral disc cells (nucleus pulposus, annulus fibrosus), and amplifies the inflammatory responses [12].

A high serum level of TNF-α is found in SLE patients with activity disease and has been linked to the severity of the disease, even though the TNF-α involvement in the pathogenesis is unclear [13]. In our case, high NLR is also related to activity disease of SLE and IC-mediated inflammation [14]. Moreover, ICs (Immune Complexes) in SLE can significantly induce the release of TNF-α and are responsible for elevated levels of TNF-α in the serum [15]. TNF-α has been implicated in the pathophysiological processes of both DDD and SLE. As a consequence, patient with SLE is more susceptible to having DDD, especially disc herniation.

## CONCLUSION

SLE is an autoimmune disease that has many manifestations. Furthermore, it can be associated with DDD, and the incidence is rare. Inflammatory reaction, especially TNF-α, is the primary pathophysiology to which they are related. As a result, SLE patient with activity disease is prone to have DDD, including disc herniation.

*Ethics approval and consent to participate:*
The patient provided verbal agreement for publication of the data.
No data of the patients that can be identified.

## REFERENCES


1. Joseph FG, Scolding NJ. Neurolupus. *Pract Neurol.* 2010 Feb;10(1):4-15. doi: 10.1136/jnnp.2009.200071. PMID: 20130291.
2. Kakati S, Barman B, Ahmed SU, Hussain M. Neurological Manifestations in Systemic Lupus Erythematosus: A Single Centre Study from North East India. *J Clin Diagn Res.* 2017 Jan;11(1):OC05-OC09. doi: 10.7860/JCDR/2017/23773.9280. Epub 2017 Jan 1. PMID: 28273990; PMCID: PMC5324435.
3. Hanly JG. Diagnosis and management of neuropsychiatric SLE. *Nat Rev Rheumatol.* 2014 Jun;10(6):338-47. doi: 10.1038/nrrheum.2014.15. Epub 2014 Feb 11. PMID: 24514913.
4. Schroeder GD, Guyre CA, Vaccaro AR. The epidemiology and pathophysiology of lumbar disc herniations. Seminars in Spine Surgery. 2016;28(1):2-7. doi: 10.1053/j.semss.2015.08.003.
5. Beh SC, Greenberg BM, Frohman T, Frohman EM. Transverse myelitis. *Neurol Clin.* 2013 Feb;31(1):79-138. doi: 10.1016/j.ncl.2012.09.008. PMID: 23186897; PMCID: PMC7132741.
6. Perez Giraldo GS, Ortiz Garcia JG. Immune-Mediated Disorders Affecting the Spinal Cord and the Spine. *Curr Neurol Neurosci Rep.* 2021 Jan 3;21(1):3. doi: 10.1007/s11910-020-01088-6. PMID: 33392785; PMCID: PMC7779105.
7. Menor Almagro R, Ruiz Tudela Mdel M, Girón Úbeda J, Cardiel Rios MH, Pérez Venegas JJ, García Guijo C. Myelitis transverse in Sjögren's syndrome and systemic lupus erythematosus: presentation of 3 cases. *Reumatol Clin.* 2015 Jan-Feb;11(1):41-4. English, Spanish. doi: 10.1016/j.reuma.2014.03.008. Epub 2014 Jun 7. PMID: 24913964.
8. Wang L, He T, Liu J, Tai J, Wang B, Zhang L, et al. Revealing the Immune Infiltration Landscape and Identifying Diagnostic Biomarkers for Lumbar Disc Herniation. *Front Immunol*. 2021 May 27;12:666355. doi: 10.3389/fimmu.2021.666355. PMID: 34122424; PMCID: PMC8190407.
9. Nakazawa KR, Walter BA, Laudier DM, Krishnamoorthy D, Mosley GE, Spiller KL, et al. Accumulation and localization of macrophage phenotypes with human intervertebral disc degeneration. *Spine J.* 2018 Feb;18(2):343-356. doi: 10.1016/j.spinee.2017.09.018. Epub 2017 Oct 12. PMID: 29031872; PMCID: PMC5815908.
10. Risbud MV, Shapiro IM. Role of cytokines in intervertebral disc degeneration: pain and disc content. *Nat Rev Rheumatol*. 2014 Jan;10(1):44-56. doi: 10.1038/nrrheum.2013.160. Epub 2013 Oct 29. PMID: 24166242; PMCID: PMC4151534.
11. McHugh J. Linking cell mechanobiology and inflammation in IVD degeneration. *Nat Rev Rheumatol.* 2020 Nov;16(11):604. doi: 10.1038/s41584-020-00510-0. PMID: 32963326.
12. Wang C, Yu X, Yan Y, Yang W, Zhang S, Xiang Y, et al. Tumor necrosis factor-α: a key contributor to intervertebral disc degeneration. *Acta Biochim Biophys Sin* (Shanghai). 2017 Jan;49(1):1-13. doi: 10.1093/abbs/gmw112. Epub 2016 Nov 17. PMID: 27864283.
13. Ghorbaninezhad F, Leone P, Alemohammad H, Najafzadeh B, Nourbakhsh NS, Prete M, et al. Tumor necrosis factor-α in systemic lupus erythematosus: Structure, function and therapeutic implications (Review). *Int J Mol Med.* 2022 Apr;49(4):43. doi: 10.3892/ijmm.2022.5098. Epub 2022 Feb 9. PMID: 35137914.
14. Han BK, Wysham KD, Cain KC, Tyden H, Bengtsson AA, Lood C. Neutrophil and lymphocyte counts are associated with different immunopathological mechanisms in systemic lupus erythematosus. *Lupus Sci Med.* 2020 May;7(1):e000382. doi: 10.1136/lupus-2020-000382. PMID: 32444416; PMCID: PMC7247402.
15. Gao XJ, Qu YY, Liu XW, Zhu M, Ma CY, Jiao YL, et al. Immune complexes induce TNF-α and BAFF production from U937 cells by HMGB1 and RAGE. *Eur Rev Med Pharmacol Sci.* 2017 Apr;21(8):1810-1819. PMID: 28485798.


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