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# Complete Recovery Chronic Retro-Odontoid Pseudotumor Et HNP with Laminectomy and Cervical Disc Replacement

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**Abstract:** A rare case found, 54-year-old male patient was experiencing pain in his neck since 20 years ago and had been gradually worsening with weakness of upper and lower extremity. Magnetic resonance images and cervical spine plain photo showed a retro-odontoid pseudotumor and HNP. This lesion behaves as a tumor-like lesion that causes inflammation. HNP cervical related with CX3CL1/CX3CR1 interaction in Rheumatoid Arthritis (RA). The paper aims to report a rare case of a patient with complete recovery chronic retro-odontoid pseudotumor C1 and hernia nucleus pulposus C3-4 C4-5 C6-7. The patient was treated with a two-phase procedure: laminectomy and cervical disc replacement. The patient had paresis and tingling in the upper and lower extremities for the past three months with radiating neck pain for 20 years after the two-phase operation. The patient completely recovered after underwent two phases of procedure and 2-years follow-up evaluation. Chronic retro odontoid pseudotumor C1 et hernia nucleus pulposus C3-4 C4-5 C6-7 can recover completely with laminectomy and cervical disc replacement without symptoms or complaint after two years. HNP cervical can relate to rheumatoid arthritis that causes retro odontoid pseudotumor in this case.

**Keywords:** hernia nucleus pulposus, retro-odontoid pseudotumor, rheumatoid arthritis.

## 椎板切除术和颈椎间盘置换术完全恢复慢性后齿状突假瘤

**摘要:** 一个罕见的案例发现，54 岁的男性患者从 20 年前开始颈部疼痛，并逐渐恶化，上肢和下肢无力。磁共振图像和颈椎平片显示后齿状突假瘤和 HNP。这种病变表现为引起炎症的肿瘤样病变。HNP 颈椎病与类风湿性关节炎（RA）中 CX3CL1/CX3CR1 的相互作用有关。本文旨在报告一例罕见的完全康复慢性齿状突后假瘤 C1 和疝髓核 C3-4 C4-5 C6-7 患者。该患者接受了两阶段手术：椎板切除术和颈椎间盘置换术。患者术后 3 个月出现上下肢麻痹、刺痛，放射性颈痛 20 年。经过两期手术和 2 年随访评估，患者完全康复。慢性齿状突后假瘤 C1 和疝髓核 C3-4 C4-5 C6-7 可以通过椎板切除术和颈椎间盘置换术完全康复，两年后无症状或主诉。在这种情况下，HNP 颈椎病可能与导致后齿状突假瘤的类风湿性关节炎有关。

**关键词：** 髓核疝、牙后突假瘤、类风湿性关节炎。

## 1. Introduction

Cases of retro odontoid pseudotumor (ROP) are very rare. This mass is an uncommon condition in RA patients [1]. In 2004-2015, only three cases were found with rheumatoid arthritis from 38 retro-odontoid pseudotumor individuals [2]. There are no case reports

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that report retro-odontoid pseudotumor et causa rheumatoid arthritis patients with cervical HNP. Several theories mention the relationship between cervical HNP and RA, namely the contribution of the CX3CL1/CX3CR1 interaction [3].

In this report, we would like to describe a case of ROP caused by rheumatoid arthritis with cervical HNP over 20 years and complete recovery after two years of follow-up. The surgical intervention was performed by laminectomy and cervical disc replacement.

## 2. Case Report

A 54-year-old man complained of neck and back of head pain associated with activity disturbance for 20 years. The patient also complained of tingling behind the head, neck, arms, chest, stomach, and legs. The patient experienced a decrease in muscle strength in the upper and lower limbs, so that patients were difficult to walk and using a spoon independently. Patients had difficulty holding a spoon. Weakness in the upper and lower extremities was felt for the last three months before the patient was operated on.

Neurological status of patient show muscle strength based on Medical Research Council Scale Manual Muscle Testing Scale result is upper limb 3/3 and lower limb 3/3. Pathologic reflex Koebner, Babinski, and Clonus are positive. Physiologic reflex ankle reflex and jerk Achilles reflex are increased. Autonomic nerve pathways defecation and clonus were found to be disturbed, but urination was normal. A blood test revealed high CRP, normal full blood count, erythrocyte sedimentation rate, renal function test, liver function test, phosphate level, and alkaline phosphatase level.

Then the patient was conducted by the MRI supporting examination of C1-high stenosis with a mass of retro odontoid tumors. This patient also has Central Type Discus Herniates at C3-4, C4-5, and C5-6. The mass shows hypointense impinging the C1 spinal cord with no contrast enhancement.

Then cervical spine plain photo inspection of dynamic Cervix Plain AP, lateral, flexion, and extension showed no instability and subluxation in C1 and C2 atlantoaxial. In Cervical CT scan showed a retro odontoid tumor with intact bone. The process occurs in soft tissue, but bones are not affected. A diagnosis of retro odontoid pseudotumor with no instability and hernia nucleus pulposus at C3-4, C4-5, and C5-6 regions was made. The diagnosis was established because of patients' symptoms (indicating an inflammatory process), and protrusion of the disc to the spinal cord means herniated disc.

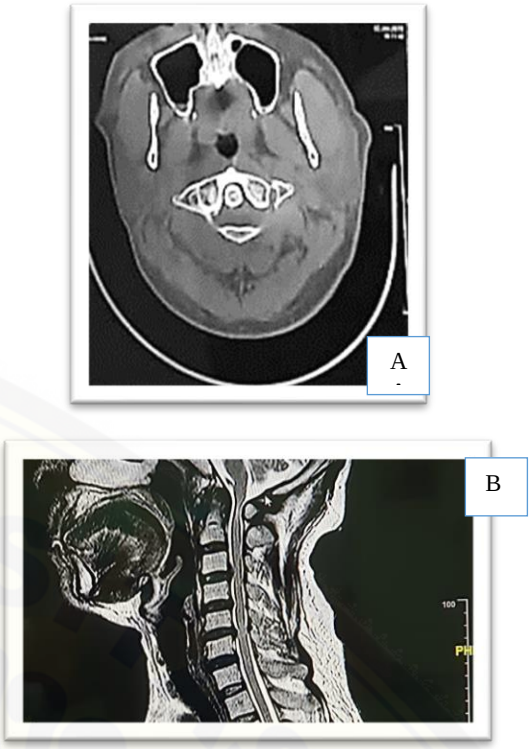


Fig. 1 MRI of the cervical spine in A. Axial plane and B. Sagital plane show mass of retro odontoid tumor

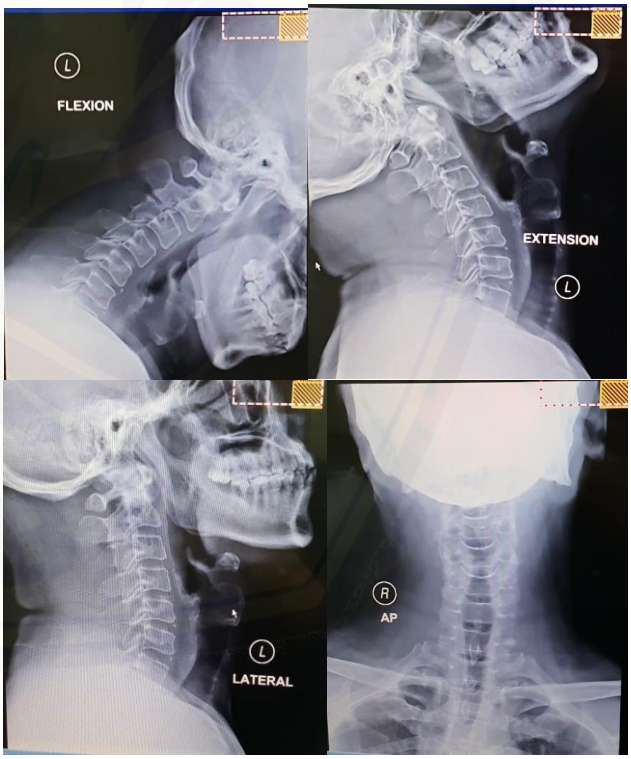


Fig. 2 Cervical spine plain photo inspection of dynamic Cervix Plain AP lateral and dynamic flexion and extension showed no instability and no subluxation in C1 and C2 atlantoaxial

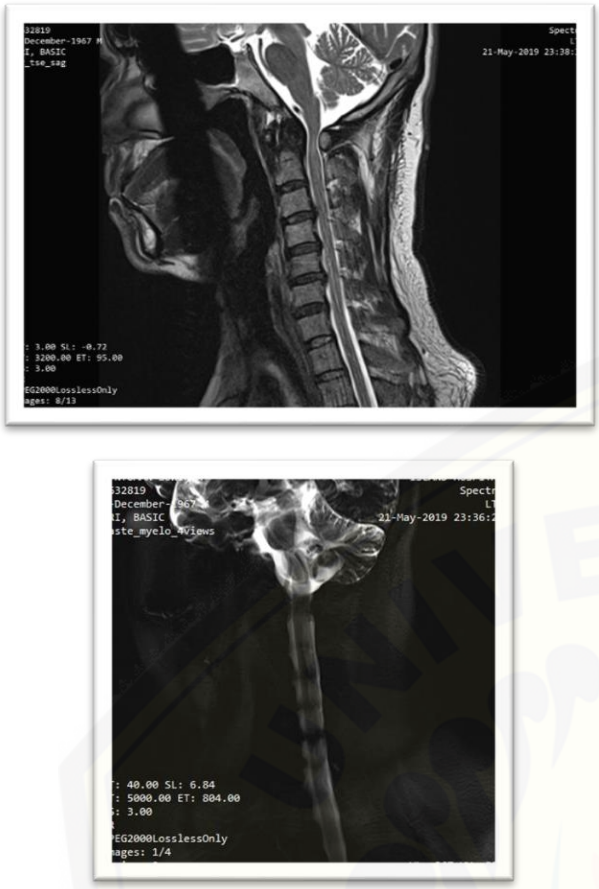

Fig. 3 Cervical CT scan showed a retro-odontoid tumor with intact bone

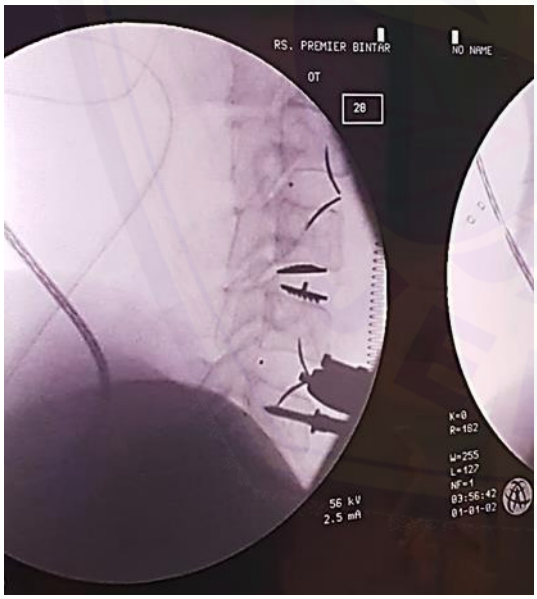


Fig. 4 Operation Cervical Disc Replacement C3-4 C4-5 C6-7

The surgical intervention is carried out in 2 phases. Stage 1 patient in the prone position, C1 laminectomy was performed for spinal cord decompression, the operation did not do stabilization and fusion because the cervical condition C1 C2 was stable.

An incision is made in the neck of the area to be laminectomy—incision in the back over the affected vertebrae. The muscles are separated from the spine. A small instrument is used to remove the appropriate lamina. Posterior C1 arches were removed. Therefore, the spinal cord is not decompressed. The spinal cord is decompressed once the bone and other tissues have been removed and discarded. Each nerve root (when appropriate) is identified and carefully decompressed (this is known as a "rhizolysis").

Furthermore, patients in stage 2 surgery are carried out on the same day and date. The patient is in the spine position. Cervical disc replacement is performed C3-4, C4-5, and C5-6. An incision surgical cut was made on the front of the neck, important structures of the neck were carefully moved to the side until can see the bone vertebrae and cervical disk. Biopsy was done to check histopathology. The cervical disk that was being replaced was removed; the last artificial disk was secured into the empty disk space. The patient used a soft neck collar after surgery to restrict motion.

Histopathological examination confirmed that the mass is rheumatoid arthritis.

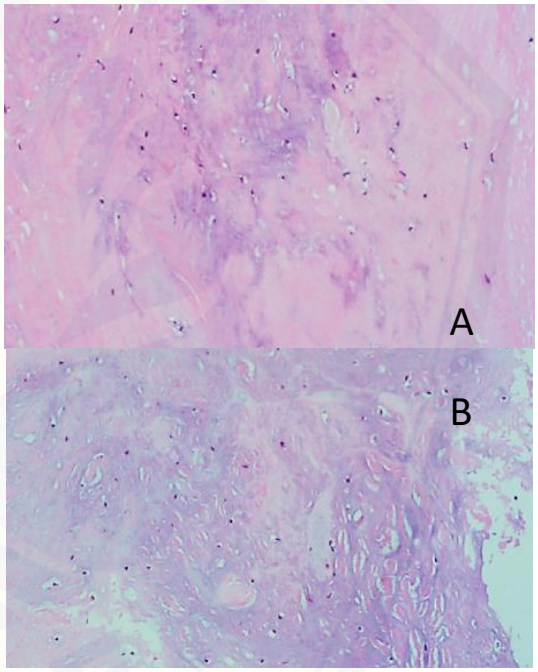


Fig. 5 Histopathological examination (H&E stain) A. Fibroid, collagenous tissue/ degenerated material with myxoid changes; B. fibroid degeneration and cartilaginous metaplasia, inflammatory cell infiltration

The results of the operation were good. The patient removed the stitches on day 10. The patient used a collar brace for six weeks. The motor, sensory, autonomic function returned to normal in the 4th week postoperatively. Two years of follow-up evaluations showed no complaint in motor, sensory and autonomic function.

## 3. Discussion

Retro-odontoid pseudotumor is a rare case that can cause compression of the spinal cord [4]. The etiology that causes this condition is rheumatoid and non-rheumatoid. Cases of retro-odontoid pseudotumor are often found with atlantoaxial instability [5]. A

pseudotumor discovered by Sze in 1986 represents a mass at the craniocervical junction with an atypical expansive lesion presentation. Lesions in this segment in patients with rheumatoid arthritis are described as pannus [6]. These lesions can cause erosion of the subchondral bone and cartilage of the synovial senses due to inflammation [5].

Patients with rheumatoid arthritis have a high risk of developing cervical spine ROP in 40% of cases. Many rheumatoid pseudotumor masses form in the anterior atlantoaxial subluxation [7]. However, patients with RA very rarely develop ROP. The disease is unusual, especially if it is not associated with atlantoaxial instability [8].

The pathophysiology underlying the mechanism of ROP in cervical spine RA is the formation of pannus caused by the release of proteases that can destroy the extracellular matrix in cartilage and trigger the proliferation of fibroblast cells [9]. There is also the genesis of tissue deposition in the retro-odontoid space in patients with ROP, especially in the cruciate ligament band.

This patient had symptoms for 20 years, indicating that the pseudotumor growth was expansive, plus the presence of HNP in the patient. HNP has different pathophysiology related to the degeneration of hydrophilic proteoglycans in the nucleus pulposus of the vertebral disc [10]. The vertebral disc is dehydrated, which reduces the water uptake capacity, causing the shock absorption capacity to decrease [11]. Vertebral load compresses the annulus fibrosis causing extrusion of the nucleus pulposus, causing disc herniation/rupture [12]. This compression of the spinal cord causes myelopathy.

The relationship between the presence of retro-odontoid pseudotumor et causa rheumatoid arthritis with cervical HNP in this patient is the role of the CX3CL1/CX3CR1 interaction that contributes to cervical hernia nucleus pulposus [13]. CXCL1 is a signaling chemokine with a CXCR1 receptor, has a role in the recruitment and adhesion of monocytes/macrophages and T cells in atherosclerotic disease, rheumatological disorders, and even HIV without integrin activation [14]. CXCL1 expression was increased in regional disc tissue endothelial cells in cervical HNP. CD4+ T cells and NK cells initiate inflammation and cytotoxicity of the rheumatoid arthritis synovium [15]. In addition, the interaction of CX3CL1/CX3CR1 plays an important role in inflammatory cell migration into degenerative herniated disc tissue. The most likely hypothesis is that due to fractalkine up-regulation, CD4+ T cells and NK cells migrate toward the inflammatory disc degeneration lesion [16].

This patient underwent Laminectomy and Cervical Disc Replacement surgery, which was described previously. Complete recovery with symptoms lasting for 20 years can be reversed. The theory that explains this is the theory of cell regeneration.

Denervation supersensitivity means loss of innervation leads to increased sensitivity of the postsynaptic membrane. This is due to an increase in receptor synthesis and a consequent increase in the number of receptors on the postsynaptic membrane or an increase in the sensitivity of G-protein-associated receptors to the receptor complex [17].

Synaptic plasticity is a second increase in the size of the postsynaptic potential after the initial stimulus. This occurs due to the accumulation of calcium in the nerve terminals. In the presence of an action potential at the nerve terminal, calcium enters through voltage-gated calcium channels; a transient increase in calcium concentration triggers the release of transmitters [18, 19].

Long-term potentiation of synaptic plasticity. High-frequency activation at some synapses will last a long time, increasing transmission efficiency. The mechanism involves depolarization of the postsynaptic membrane, caused by the release of glutamate at the presynaptic terminal and activation of the AMPA glutamate receptor. The result will increase the sensitivity of the postsynaptic membrane [20].

Collateral branch plasticity. In response, the axon that is not damaged performs collateral branches on the axon that is affected by the lesion. These axons form contacts that replace the axons affected by the lesion. This mechanism is a compensatory nerve so that it can function again [21].

Regeneration of interrupted axons: after the axon is severed, the distal part undergoes degeneration or what is known as Wallerian degeneration, an irreversible process, and the process takes several days in the peripheral nervous system (PNS), lasting several weeks in the central nervous system (CNS). At the same time, the proximal portion undergoes retraction, depending on location, species, and other factors. After that, the axon tries to regenerate and re-establish the connection to the postsynaptic target [22].

## 4. Conclusion

The patient, a 54-year-old man, had complaints of neck pain for 20 years with weakness in all four extremities three months before surgery. The patient had a C1 retro-odontoid pseudotumor and C3-4, C45, C56 HNP. The patient's CRP has increased. Rheumatoid arthritis carries a risk of retro-odontoid pseudotumor in rare cases. HNP in this patient is suspected of having a strong relationship with retro-odontoid pseudotumor, the potential contribution of CX3CR1-expressing CD4 (+) T-cells and NK cells in response to inflammation in the pathogenesis of cervical disc degeneration, which may lead to herniation. These diseases in this patient can cause myelopathy, but the patient can recover completely without any symptoms after two years. The theory explains neural cell regeneration denervation

supersensitivity, synaptic plasticity, long-term potentiation of synaptic plasticity, collateral branch plasticity, and regeneration of disconnected axons.

## References


[1] YU, S.H., CHOI, H.J., CHO, W.H., CHA, S.H., and HAN, I.H. Retro-Odontoid Pseudotumor without Atlantoaxial Subluxation or Rheumatic Arthritis. *Korean Journal of Neurotrauma*, 2016, 12(2): 180-184.

[2] PARK, J.H., LEE, E., LEE, J., KANG, Y., AHN, J.M., YEOM, J.S., and KANG, H.S. Postoperative Regression of Retro-odontoid Pseudotumor after Atlantoaxial Posterior Fixation: 11 Years of Experience in Patients With Atlantoaxial Instability. *SPINE*, 2017, 42(23): 1763–1771.

[3] PENG, Z., CHEN, R., FANG, Z., CHEN, B., WANG, Z., and WANG, X. Increased local expressions of CX3CL1 and CCL2 are related to clinical severity in lumbar disk herniation patients with sciatic pain. *Journal of Pain Research*, 2017, 10: 157-165.

[4] CERTO, F., MAIONE, M., VISOCCHI, M., and BARBAGALLO, G.M. Retro-odontoid Degenerative Pseudotumour Causing Spinal Cord Compression and Myelopathy: Current Evidence on the Role of Posterior C1-C2 Fixation in Treatment. *Acta neurochirurgica. Supplement*, 2019, 125: 259-264.

[5] FIANI, B., HOUSTON, R., SIDDIQI, I., ARSHAD, M., REARDON, T., GILLILAND, B., DAVATI, C., and KONDILIS, A. Retro-Odontoid Pseudotumor Formation in the Context of Various Acquired and Congenital Pathologies of the Craniovertebral Junction and Surgical Techniques. *Neurospine*, 2020, 18: 67-78.

[6] SHI, J., ERMANN, J., WEISSMAN, B.N., SMITH, S.E., and MANDELL, J.C. Thinking beyond pannus: a review of retro-odontoid pseudotumor due to rheumatoid and non-rheumatoid etiologies. *Skeletal Radiology*, 2019, 1-13.

[7] GARWIN, S.R. Rheumatoid Arthritis of the Cervical Spine. *Medscape*, 2019.

[8] LACY, J., BAJAJ, J., and GILLIS, C.C. *Atlantoaxial Instability*. Dalam: StatPearl, 2021.

[9] HOEGERL, C., and BESHAI, R. Cervical Pannus without Rheumatoid Arthritis or Trauma. *Federal practitioner: for the health care professionals of the VA, DoD, and PHS*, 2020, 37(4): 194-197.

[10] SILAGI, E.S., SHAPIRO, I.M., and RISBUD, M.V. Glycosaminoglycan synthesis in the nucleus pulposus: Dysregulation and the pathogenesis of disc degeneration. *Matrix biology: journal of the International Society for Matrix Biology*, 2018, 71-72: 368-379.

[11] FURMAN, M. Cervical Disc Disease. *Medscape*, 2020.

[12] INDRA, R., ILYAS, M., MUIS, M., MURTALA, B., ALFIAN, A., and KAELAN, C. Hubungan Kadar Lipid Darah Dengan Degenerative Disc Disease Berdasarkan Klasifikasi Pfirrmann Menggunakan Magnetic Resonance Imaging Lumbosacral pada Pasien Nyeri Punggung Bawah. *Majalah Kesehatan Pharmamedika*, 2020.

[13] LI, Y., FANG, Z., GU, N., BAI, F., MA, Y., DONG, H., YANG, Q., and XIONG, L. Inhibition of chemokine CX3CL1 in spinal cord mediates the electroacupuncture-induced suppression of inflammatory pain. *Journal of Pain Research*, 2019, 12, 2663 - 2672.

[14] JACOBSEN, D.P., MOEN, A., HAUGEN, F., and GJERSTAD, J. Hyperexcitability in Spinal WDR Neurons following Experimental Disc Herniation Is Associated with Upregulation of Fractalkine and Its Receptor in Nucleus Pulposus and the Dorsal Root Ganglion. *International Journal of Inflammation*, 2016.

[15] PAWELEC, P., ZIEMKA-NALECZ, M., SYPECKA, J., and ZALEWSKA, T. The Impact of the CX3CL1/CX3CR1 Axis in Neurological Disorders. *Cells*, 2020, 9(10): E2277.

[16] SUN, Z., LIU, B., and LUO, Z. The Immune Privilege of the Intervertebral Disc: Implications for Intervertebral Disc Degeneration Treatment. *International Journal of Medical Sciences*, 2020, 17(5): 685 - 692.

[17] BÜTTNER, R., SCHULZ, A., REUTER, M., AKULA, A.K., MINDOS, T., CARLSTEDT, A., RIECKEN, L.B., BAADER, S.L., BAUER, R., and MORRISON, H. Inflammaging impairs peripheral nerve maintenance and regeneration. *Aging Cell*, 2018, 17(6).

[18] JACKMAN, S.L., and REGEHR, W.G. The Mechanisms and Functions of Synaptic Facilitation. *Neuron*, 2017, 94(3): 447-464.

[19] NANOU, E., and CATTERALL, W.A. Calcium Channels, Synaptic Plasticity, and Neuropsychiatric Disease. *Neuron*, 2018, 98(3): 466-481.

[20] SCARNATI, M.S., CLARKE, S.G., PANG, Z.P., and PARADISO, K.G. Presynaptic Calcium Channel Open Probability and Changes in Calcium Influx Throughout the Action Potential Determined Using AP-Waveforms. *Frontiers in Synaptic Neuroscience*, 2020, 12.

[21] DUMOULIN, A., TER-AVETISYAN, G., SCHMIDT, H., and RATHJEN, F. Molecular Analysis of Sensory Axon Branching Unraveled a cGMP-Dependent Signaling Cascade. *International Journal of Molecular Sciences*, 2018, 19(5): 1266.

[22] HUSSAIN, G., WANG, J., RASUL, A., ANWAR, H., QASIM, M., ZAFAR, S., AZIZ, N., RAZZAQ, A., HUSSAIN, R., DE AGUILAR, J.G., and SUN, T. Current Status of Therapeutic Approaches against Peripheral Nerve Injuries: A Detailed Story from Injury to Recovery. *International Journal of Biological Sciences*, 2020, 16(1): 116-134.


**参考文:**


[1] YU, S.H., CHOI, H.J., CHO, W.H., CHA, S.H., 和 HAN, I.H. 無寰樞椎半脫位或風濕性關節炎的後齒狀突假瘤。韓國神經創傷雜誌, 2016, 12(2): 180-184。

[2] PARK, J.H., LEE, E., LEE, J., KANG, Y., AHN, J.M., YEOM, J.S., 和 KANG, H.S. 寰樞椎後路固定術後齒狀突後假瘤的術後消退：11 年寰樞椎不穩患者的經驗。脊柱，2017，42（23）：1763-1771。

[3] PENG, Z., CHEN, R., FANG, Z., CHEN, B., WANG, Z., 和 WANG, X. 薩薩克 1 和 覆銅板 2 局部表達增加與腰椎間盤突出症患者的臨床嚴重程度有關坐骨神經痛。疼痛研究雜誌，2017，10：157-165。

[4] CERTO, F. 、MAIONE, M. 、VISOCCHI, M. 和 BARBAGALLO, G.M. 導致脊髓壓迫和脊髓病的後齒狀突退行性假瘤：關於後路固定在治療中作用的當前證據。神經外科學報。增刊，2019，125：259-264。

[5] FIANI, B., HOUSTON, R., SIDDIQI, I., ARSHAD, M., REARDON, T., GILLILAND, B., DAVATI, C., 和 KONDILIS, A. 後齒狀突假瘤的形成顱椎交界處和手術技術的各種獲得性和先天性病理的背景。神經脊柱, 2020, 18: 67-78。

[6] SHI, J., ERMANN, J., WEISSMAN, B.N., SMITH, S.E., 和 MANDELL, J.C. 超越血管翳的思考：對類風濕和非類風濕病因引起的齒狀突後假瘤的回顧。骨骼放射學，2019，1-13。
[7] GARWIN, S.R. 頸椎類風濕性關節炎。醫學景觀，2019 年。
[8] LACY, J., BAJAJ, J., 和 GILLIS, C.C. 寰樞椎不穩。達拉姆：統計珍珠，2021 年。
[9] HOEGERL, C., 和 BESHAI, R. 無類風濕性關節炎或創傷的宮頸翳。聯邦從業者：對於弗吉尼亚州、国防部和小灵通的醫療保健專業人員，2020, 37(4): 194-197。
[10] SILAGI, E.S., SHAPIRO, I.M., 和 RISBUD, M.V. 髓核中的糖胺聚醣合成：調節異常和椎間盤退變的發病機制。矩陣生物學：國際矩陣生物學學會會刊, 2018, 71-72: 368-379.
[11] FURMAN, M. 頸椎間盤疾病。醫學景觀，2020。
[12] INDRA, R., ILYAS, M., MUIS, M., MURTALA, B., ALFIAN, A., 和 KAELAN, C. 血脂水平與退行性椎間盤疾病 基於 菲爾曼分類使用磁共振成像腰骶部腰痛患者。醫藥健康雜誌，2020 年。
[13] LI, Y., FANG, Z., GU, N., BAI, F., MA, Y., DONG, H., YANG, Q., 和 XIONG, L. 抑制脊髓中趨化因子 薩薩克 1 介導電針誘導的炎症性疼痛抑制。疼痛研究雜誌，2019, 12, 2663 - 2672。
[14] JACOBSEN, D.P., MOEN, A., HAUGEN, F., 和 GJERSTAD, J. 實驗性椎間盤突出症後脊髓 寬動態 神經元的過度興奮與髓核和背根神經節中 精炼机及其受體的上調有關。國際炎症雜誌，2016 年。
[15] PAWELEC, P.、ZIEMKA-NALECZ, M.、SYPECKA, J. 和 ZALEWSKA, T. 薩薩克 1/客戶經理 1 軸對神經系統疾病的影響。細胞，2020 年，9(10)：E2277。
[16] SUN, Z., LIU, B., 和 LUO, Z. 椎間盤的免疫特權：椎間盤退變治療的意義。國際醫學科學雜誌, 2020, 17(5): 685 - 692。
[17] BÜTTNER, R., SCHULZ, A., REUTER, M., AKULA, AK, MINDOS, T., CARLSTEDT, A., RIECKEN, LB, BAADER, SL, BAUER, R., 和 MORRISON, H. 炎症會損害周圍神經的維持和再生。老化細胞, 2018, 17(6)。
[18] JACKMAN, S.L., 和 REGEHR, W.G. 突觸促進的機制和功能。神經元, 2017, 94(3): 447-464。
[19] NANOU, E., 和 CATTERALL, W.A. 鈣通道、突觸可塑性和神經精神疾病。神經元, 2018, 98(3): 466-481。
[20] SCARNTI, M.S., CLARKE, S.G., PANG, Z.P., 和 PARADISO, K.G. 突觸前鈣通道開放概率和鈣流入的變化在整個動作電位中使用美联社波形確定。突觸神經科學前沿，2020 年，12。
[21] DUMOULIN, A., TER-AVETISYAN, G., SCHMIDT, H., 和 RATHJEN, F. 感覺軸突分支的分子分析揭示了 生产质量管理规范 依賴的信號級聯。國際分子科學雜誌, 2018, 19(5): 1266.
[22] HUSSAIN, G., WANG, J., RASUL, A., ANWAR, H., QASIM, M., ZAFAR, S., AZIZ, N., RAZZAQ, A., HUSSAIN, R., DE AGUILAR, J.G., 和 SUN, T. 外周神經損傷治療方法的現狀：從損傷到恢復的詳細故事。國際生物科學雜誌, 2020, 16(1): 116-134。