e-ISSN: 2807-7970
doi: 10.20473/aksona.v5i2.53453

AKSONA

Volume 5 Number 2, July 2025

# Clinical Improvement of Chronic Spinal Cord Injury and Immune Thrombocytopenia (ITP) with Corticosteroids Administration: A Case Report

**Athalia Anastasia Talaway[1], Abdulloh Machin[1], Dedy Kurniawan[1]**
[1] Department of Neurology, Faculty of Medicine, Universitas Airlangga; Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

**Article info**

***Article History:***
Received Jan 2, 2024
Revised Oct 23, 2024
Accepted Nov 20, 2024
Published

***Keywords:***
Immune thrombocytopenia
Spinal cord injury
Steroids
Therapeutic option

**ABSTRACT**

**Introduction:** Spinal cord injury (SCI) is a significant medical condition caused by either traumatic events or pathological diseases, which leads to neurological deficits and various levels of motor, sensory, and autonomic dysfunction. Several treatments, including corticosteroids, have been proposed to reduce secondary neuronal damage, but they are still controversial. Meanwhile, immune thrombocytopenia (ITP) can be treated with corticosteroids. The lack of research necessitates a review of steroids as a therapy for ITP and spinal cord trauma. **Case:** A 76-year-old woman complained of weakness in both legs for 15 days before being admitted to the hospital. The weakness was noticed after experiencing a fall. Additionally, the patient complained about having difficulty with both defecating and urinating. On examination, muscle strength in the lower limbs was graded 4 on both sides according to the Medical Research Council (MRC) scale. The classification of the American Spinal Injury Association (ASIA) score was D, with neurological level injury (NLI) at the 6th thoracic level with immune thrombocytopenia (ITP). Considering the patient's condition, steroids were administered as a treatment option. Fortunately, the patient showed clinical improvements, with the ASIA score improving from D to E, suggesting a positive response to steroids and potential for neurological recovery. **Conclusion:** Steroids may be regarded as a possible treatment alternative for individuals suffering from spinal cord injuries and immune thrombocytopenic purpura (ITP).

***Corresponding Author***
Abdulloh Machin
Department of Neurology, Faculty of Medicine, Universitas Airlangga; Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
email: dr.machin95@gmail.com

Available at https://e-journal.unair.ac.id/index.php/aksona

This work is licensed under a Creative Commons Attribution-ShareAlike 4.0 International License

## INTRODUCTION

Spinal cord injury is a severe medical condition that leads to long-term disability and morbidity.[1] More than half of injury survivors are unable to return to their normal lives.[2] Trauma can result from direct or indirect injury to the spinal cord, such as nerve tissue and blood vessels tearss or bone structure damage. Indirect causes may include hemorrhage, edema, or cavitation.[3]

The American Spinal Injury Association (ASIA) classifies spinal cord injury into five categories based on motor and sensory function:[3]

A = Complete
No motor or sensory function in the entire dermatome segment from the point of the lesion to S4-S5.

B = Incomplete
Impaired motor function below the lesion (including the S4-S5 segment), but the sensory function is still good.

C = Incomplete
Sensory function is still good, the motor function below the lesion is still functional and the majority of the muscles below the lesion have a motor strength of less than 3.

D = Incomplete
Sensory function is still good, and the motor function below the lesion and the majority of muscles have motor strength ≥ 3.

E = Normal
Normal motor and sensory function.

The use of corticosteroids in the treatment of spinal cord injury remains controversial. The National Acute Spinal Cord Injury Studies (NASCIS-II) and NASCIS-III suggest that methylprednisolone may be administered promptly to adult patients with acute, non-penetrating spinal cord injury. However, for injuries occurring beyond eight hours, NASCIS does not recommend the use of steroids.[4]

The central nervous system's inability to recover from spinal cord injury (SCI) creates a critical clinical issue. For years, researchers and clinicians have been exploring many potential solutions based on the pathophysiology of SCI to improve nerve function. Their research has resulted in ongoing efforts to develop pharmacological treatments that reduce neuronal damage and enhance recovery.[5] In addition, both pharmacological and non-pharmacological therapies have shown benefits in restoring motor functions and reducing neurological damage.[6]

Immune Thrombocytopenia (ITP) is an acquired autoimmune disorder characterized by a low platelet count due to both the destruction of platelets and decreased platelet production. The incidence of ITP is estimated to be between 2 and 5 cases per 100,000 people in the general population. Corticosteroids are commonly used to treat ITP.[7] They work by suppressing the immune response, and can lead to an increase in platelet counts.

The intersection of these two conditions can complicate clinical management. Immune dysfunction associated with ITP can hinder recovery and rehabilitation efforts following spinal injury. Corticosteroids are commonly used to a treat ITP, but they are not recommended for spinal cord injury lasting over 8 hours. Therefore, this review examines the use of corticosteroids as a therapy for both ITP and spinal cord injury, gien the limited existing research, and also to explore the clinical improvement observed in a patient with chronic SCI and ITP following corticosteroid administration.

## CASE

A 76-year-old woman complained of weakness in both legs after slipping at home. The symptoms had begun 21 days before admission, initially as tingling sensations spreading from the buttocks to the right leg. Subsequently, both legs were weak 15 days before admission, making it difficult to walk. Four days before to admission, she also complained of difficulty in defecating and urinating. Initially, the family started home treatment with infusions due to suspected potassium deficiency, but as the condition did not improve, the patient was brought to Dr. Soetomo General Academic Hospital. Weakness in the hands was reported, while shortness of breath, slurred speech, and facial drooping were denied. She also reported experiencing fever for 3 days prior to admission. The patient denied any previous history of diarrhea, cough, cold, hypertension, diabetes mellitus, stroke, tuberculosis, and tumor.

During the general examination, the patient's blood pressure was 130/90 mmHg, and her body temperature was 37.30 °C with suprapubic distension. Other findings from the general examinations were within normal limits. On neurological examination, superior limb motor strength was 55555/55555, while inferior limb motor strength was 44444/44444; the sensory examination was inconsistent; superior limb proprioception was normal, while inferior limb proprioception was abnormal. Bicep and tricep reflexes were +2/+2, and the physiological reflexes of the Patellar and Achilles were +1/+1. The patient had urinary retention. Her ASIA score was determined to be D, indicating a thoracic neurological level injury (NLI) at the 6th level. Further examination revealed

hypohidrosis (reduced perspiration) extending to the thoracic six myelum segments.

A thoracolumbar X-ray, revealed thoracolumbar spondylosis. A thoracolumbar MRI with contrast showed suspected chronic bleeding as high as VTh 4–7 accompanied by cord edema as high as VTh 2–5, degenerative spine disease with a bulging disc, facet joint arthropathy causing mild foraminal stenosis as high as VL 2-4, lumbar *dextroscoliosis*, and spondylosis (Figure 1). Laboratory results were as follows: hemoglobin 10 g/dL, platelets 26x10$^3$/µl, potassium 3.1mmol/L, albumin 2.67 g/dl, Erythrocyte Sedimentation Rate 82 mm/h, C-Reactive Protein 7 mg/dL, Antinuclear Antibody test 20.54 AU/ml, C3 110 mg/dL, C4 27.9mg/dL, HIV non-reactive. Peripheral blood smear findings includednormochromic normocytic anemia, leukocytosis with atypical lymphocytes (+), and thrombocytopenia.

The patient was then referred to the internal medicine and medical rehabilitation departments, where ITP was diagnosed. A thoracolumbosacral corset othosis and methylprednisolone injection (3 x 125 mg) were prescribed to the patient.

After a week of treatment at Dr. Soetomo General Academic Hospital, a follow-up neurological examination showed that the superior limb motor strength was 55555/55555, inferior limb motor strength was 4+4+4+4+4+4+, sensory and proprioceptive functions were within normal limits in both upper and lower limbs. The bicep and tricep reflexes were +2/+2, while the physiological reflexes of the Patellar and Achilles were +1/+1. Urinary retention persisted. Laboratory examination: HB 10.2g/dl, platelets 36 x10$^3$/µl, potassium 3.8mmol/l, and albumin 2.9g/dL. Consequently, we reduced the methylprednisolone therapy to 3 x 62.5 mg intravenously.

The patient was treated for 13 days at Dr. Soetomo General Academic Hospital. On the 13th day, neurological examination revealed motor strengths of 55555 and 55555 in both upper and lower limbs. Sensory and proprioceptive functions were within normal limits. Patellar and Achilles reflexes were +2/+2. There was no urinary retention or difficulty in defecating. Her ASIA score had improved to E. The laboratory results indicated an HB level of 10.3 g/dL, platelets 46 x10$^3$/µl, potassium 3.9mmol/L, and albumin 3.28 g/dL. The patient was discharged with methylprednisolone 3 x 16 mg.

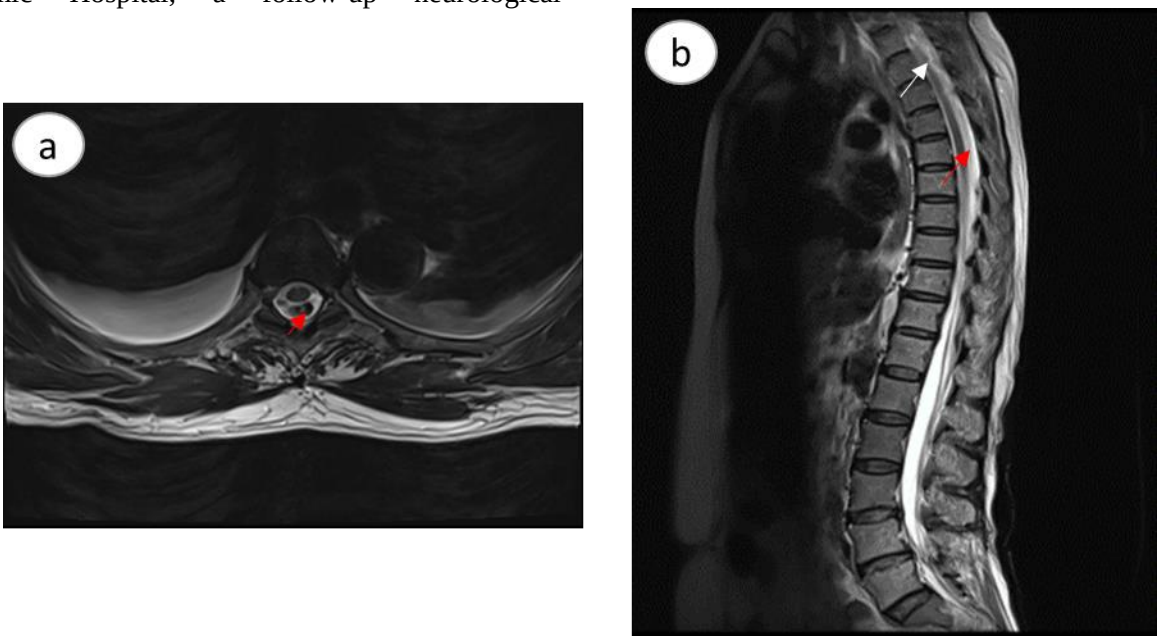


Figure 1. Axial T2 image (a) and sagittal T2 image (b) of the thoracal spine demonstrating cord oedema (white arrow) with chronic haemorrhage (red arrow)

Spinal cord injury (SCI) is frequently caused by a sudden traumatic blow to the spine, resulting in vertebral fractures or dislocations.[8] Spinal cord injuries can be classified as primary or secondary injuries.[9] The primary injury refers to the immediate mechanical damage to axons, blood vessels, and cell membranes that occurs due to direct trauma to the spinal cord, such as vertebral compression or fractures.[10] Following the primary injury, a secondary injury process begins, leading to the expansion of the damage and exacerbating neurological deficits.[11] Secondary injury can be further classified into acute (within 48 hours), subacute (2 to 14 days), intermediate (14 days to 6 months), and chronic (more than 6 months) (Figure 2).[10] The secondary phase of spinal cord injury can last up to weeks or even

months.[12]

In this case, the patient had intermediate-phase spinal cord injury and ITP-induced thrombocytopenia. The intermediate phase is part of the secondary injury process, which usually occurs more than 14 days after the primary trauma. During this phase, astrocytes around the lesion proliferate and form a tight network to isolate the lesion's core and prevent further expansion. However, these closely connected astrocytes create a physical barrier known as glial scarring (gliosis). Additionally, astrocytes also secrete chondroitin sulfate proteoglycan (CSPG), which inhibits axon regeneration., Corticosteroids were administered for two weeks to treat the condition, leading to clinical improvement in motor strength (from 4 to 5) as well as relief from autonomic and sensory disturbances. Corticosteroids have been shown to have neuroprotective effects in spinal cord injury, including improved vascular perfusion, inhibition of calcium influx and accumulation, modulation of inflammatory cell activity, inhibition of lipid peroxidation, inflammatory cytokines, and reduction of edema, all of which occur in acute spinal cord injury.

## DISCUSSION

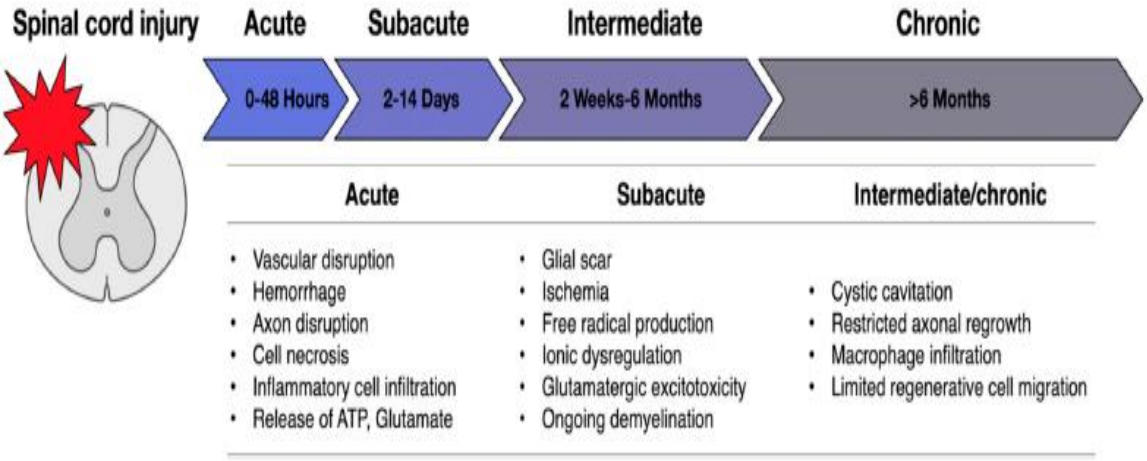


Figure 2. Pathophysiological events during acute, subacute, and chronic phases[11]

Administering methylprednisolone more than 8 hours post-injury is not advised by NASCIS, as it may lead to worse outcome than placebo and raise the chances of serious problems like pneumonia, sepsis, gastrointestinal bleeding, acute respiratory failure, and death.[4]

In NASCIS 1, the effect of methylprednisolone (MP) was evaluated using a 1000 mg loading dose, followed by 250 mg every 6 hours for 10 days, as opposed another group receiving a 100 mg loading dose, followed by 25 mg every 6 hours.[13] MP is a glucocorticoid that has been proposed by basic science studies to provide an anti-inflammatory effect that might decrease the severity of secondary spinal cord injury.[14] In addition, NASCIS 2 evaluated the effect of methylprednisolone (MP) using 30 mg/kg bolus followed by 5.4 mg/kg/h for 23 hours in patients with acute SCI (< 12 hours). The results showed no significant differences between the groups, although there was a significant improvement in motor function when the patient was treated within 8 hours. Subsequently, NASCIS 3 suggested that starting MP treatment between 3 and 8 hours after injury and continuing for 48 hours, was associated with better neurologic outcomes.[13] The best techniques for preventing pressure ulcers after thoracic and lumbar trauma is repositioning a supine individual every 2 hours.[15]

In addition to spinal cord injuries, patients with a platelet count below 30 × $10^9$/L, whether asymptomatic or experiencing mild to significant bleeding, may require careful management. Severe thrombocytopenia often needs a more aggressive treatment strategy, which may involve a combination of high-dose steroids and other rescue therapies.[16] According to studies, giving short-term intravenous pulse methylprednisolone for acute ITP in two separated doses rather than a single dose can affect the rate and extent of platelet count recovery.[17] Clinically, healthy people without infection or inflammation have a low risk of bleeding. However, ITP patients are more likely to experience bleeding in the skin, mucosal membranes, gastrointestinal tract, or other organs.[18] ITP is a condition where the body has a low number of platelets because of immune-mediated destruction of platelets or megakaryocytes. Platelet aggregation may also cause low platelet counts, and the condition is often marked by unusual patterns of thrombosis and

bleeding. Corticosteroids play a key role in managing ITP by suppressing the production of antibody, modulating the reactivity of T and B cells, increasing platelet count and affecting platelet production, and maintaining vascular endothelial integrity to reduce bleeding episodes.[19]

In this case, spinal cord injury may have triggered inflammation and damage to blood vessel, resulting in thrombocytopenia caused by ITP. In this situation, steroids are administered to treat ITP by increasing platelet count and maintaining vascular endothelial integrity, which may indirectly aid clinical improvement in spinal cord injury. Strength training, cardiovascular-focused exercise, respiratory conditioning, transfer or mobility training, and stretching to prevent muscle contractures are essential elements of physical rehabilitation.[20]

The limitation of this study is based on one individual, which may not be generalizable to other patients with similar conditions, so still need more research, but the advantage from this case report is increase awareness among healthcare professional to find out the relationship between SCI and ITP for developing better management strategies.

## CONCLUSION

Therefore, in patients with spinal cord injury and ITP, steroids may be considered a potential therapeutic option, not as a main therapy of spinal cord injury, but as an ITP therapy that indirectly improves the clinical course of the spinal cord. However, it is most important to note that recovery happens through aggressive rehabilitation.

### Acknowledgement
The author would like to thank colleagues from the Department of Neurology, Faculty of Medicine, Universitas Airlangga, and Dr. Soetomo General Academic Hospital for supporting this manuscript

### Conflict of Interest
No conflict of interest was declared by the authors.

### Funding
The authors declared that this study has received no financial support.

### Author Contributions
AT contributed to the data collection and wrote the manuscript. AM and DK serve as senior authors, manuscript conceptor, and manuscript reviewer.

## REFERENCES

1. Margetis K, Das JM, Emmady PD. Spinal cord injuries. Treasure Island (FL): StatPearls Publishing; 2025. [Web page]
2. Yılmaz T, Turan Y, Keleş A. Pathophysiology of the spinal cord injury. *J Clin Exp Investig*. 2014; 5(1):131–6. doi: 10.5799/ahins.01.2014.01.0378
3. Machin A, Kurniawan D. Trauma medula spinalis. In: Hidayati AN, Akbar MIA, Rosyid AN, editors. Gawat Darurat Medis dan Bedah. Surabaya: Airlangga University Press; 2018. p. 329–52. [Book]
4. Grant RA, Quon JL, Abbed KM. Management of acute traumatic spinal cord injury. *Curr Treat Options Neurol*. 2015; 17(2):6. doi: 10.1007/s11940-014-0334-1
5. Neunert C, Terrell DR, Arnold DM, Buchanan G, Cines DB, Cooper N, et al. American society of hematology 2019 guidelines for immune thrombocytopenia. *Blood Adv*. 2019; 3(23):3829–66. doi: 10.1182/bloodadvances.2019000966
6. Anjum A, Yazid MD, Fauzi Daud M, Idris J, Ng AMH, Selvi Naicker A, et al. Spinal cord injury: Pathophysiology, multimolecular interactions, and underlying recovery mechanisms. *Int J Mol Sci*. 2020; 21(20):7533. doi: 10.3390/ijms21207533
7. Zhang Y, Al Mamun A, Yuan Y, Lu Q, Xiong J, Yang S, et al. Acute spinal cord injury: Pathophysiology and pharmacological intervention (review). *Mol Med Rep*. 2021; 23(6):417. doi: 10.3892/mmr.2021.12056
8. Hu HZ, Granger N, Jeffery ND. Pathophysiology, clinical importance, and management of neurogenic lower urinary tract dysfunction caused by suprasacral spinal cord injury. *J Vet Intern Med*. 2016; 30(5):1575–88. doi: 10.1111/jvim.14557
9. Lee BJ, Jeong JH. Review: Steroid use in patients with acute spinal cord injury and guideline update. *Korean J Neurotrauma*. 2022; 18(1):22–30. doi: 10.13004/kjnt.2022.18.e21
10. Jeong HJ, Yun Y, Lee S-J, Ha Y, Gwak S-J. Biomaterials and strategies for repairing spinal cord lesions. *Neurochem Int*. 2021; 144:104973. doi: 10.1016/j.neuint.2021.104973
11. Alizadeh A, Dyck SM, Karimi-Abdolrezaee S. Traumatic spinal cord injury: An overview of pathophysiology, models and acute injury mechanisms. *Front Neurol*. 2019; 10. doi: 10.3389/fneur.2019.00282
12. Quadri SA, Farooqui M, Ikram A, Zafar A, Khan MA, Suriya SS, et al. Recent update on basic mechanisms of spinal cord injury. *Neurosurg Rev*. 2020; 43(2):425–41. doi: 10.1007/s10143-018-1008-3
13. Donovan J, Kirshblum S. Clinical trials in traumatic spinal cord injury. *Neurotherapeutics*. 2018; 15(3):654–68. doi: 10.1007/s13311-018-0632-5
14. Rogers WK, Todd M. Acute spinal cord injury. Best Pract Res *Clin Anaesthesiol*. 2016; 30(1):27–39. doi: 10.1016/j.bpa.2015.11.003
15. Thomas AX, Riviello JJ, Davila-Williams D, Thomas SP, Erklauer JC, Bauer DF, et al. Pharmacologic and acute management of spinal cord injury in adults and children. *Curr Treat Options Neurol*. 2022; 24(7):285–304. doi: 10.1007/s11940-022-00720-9
16. DeSouza S, Angelini D. Updated guidelines for immune thrombocytopenic purpura: Expanded management options. *Cleve Clin J Med*. 2021; 88(12):664–8. doi: 10.3949/ccjm.88a.20201
17. Bozkurt Turhan A, Canan Özdemir Z, Bör Ö. Use of single- or two-dose pulse methylprednisolone in the treatment of acute immune thrombocytopenic purpura. *The Med Bull Sisli Hosp*. 2017; 52(4):279–84. doi: 10.5350/SEMB.20171130112516
18. Onisâi M, Vlădăreanu A-M, Spînu A, Găman M, Bumbea H. Idiopathic thrombocytopenic purpura (ITP) – new era for an old disease. *Rom J Intern Med*. 2019; 57(4):273–83. doi: 10.2478/rjim-2019-0014
19. Thachil J. Alternate considerations for current concepts in ITP. *Hematology*. 2014; 19(3):163–8. doi: 10.1179/16078454137.0000000104
20. Ahuja CS, Wilson JR, Nori S, Kotter MRN, Druschel C, Curt A, et al. Traumatic spinal cord injury. *Nat Rev Dis Prim*. 2017; 3(1):17018. doi: 10.1038/nrdp.2017.18