Asian J Biol Life Sci., 2026; 15(1):247-251.
https://ajbls.com

Case Report

Symmetric Spastic Quadriparesis with Occult Cerebral Demyelination: An Adult-Onset Adrenoleukodystrophy Case Report

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ABSTRACT

X-linked adrenoleukodystrophy is a peroxisomal disorder caused by ABCD1 mutations leading to the accumulation of very-long-chain fatty acids and progressive neurological dysfunction. Adult-onset cerebral adrenoleukodystrophy is uncommon and often presents with subtle or atypical symptoms, contributing to diagnostic delays. We describe an adult male who presented with a 2-year history of gradually progressive symmetrical spastic quadriparesis, gait disturbance, and mild bulbar symptoms without cognitive decline or adrenal insufficiency. Neurological examination revealed marked spasticity, exaggerated reflexes, and frontal release signs, with preserved sensory modalities. Early magnetic resonance imaging brain demonstrated subtle, symmetrical T2/FLAIR hyperintensities in the posterior periventricular white matter, initially non-specific and non-enhancing. Routine laboratory studies were unremarkable. Further evaluation with biochemical assays showed elevated very- long-chain fatty acids, and genetic testing confirmed a pathogenic ABCD1 variant, establishing the diagnosis of adult-onset cerebral adrenoleukodystrophy. Supportive management, including spasticity control, physiotherapy, and counselling, led to functional improvement during hospitalization. This case highlights the diagnostic challenges of adult-onset adrenoleukodystrophy, particularly when cerebral demyelination is mild, and symptoms mimic hereditary spastic paraplegia, multiple sclerosis, or degenerative myelopathies. The absence of adrenal involvement and lack of florid radiological findings further contributed to delays in recognition. Clinicians should consider adrenoleukodystrophy in adults presenting with unexplained upper motor neurone syndromes, even when magnetic resonance imaging abnormalities are subtle. Early metabolic and genetic testing is essential to avoid missed therapeutic opportunities and ensure timely multidisciplinary care.

Keywords Adrenoleukodystrophy, ABCD1 mutation, Adult-Onset Adrenoleukodystrophy (ALD), Spastic Quadriparesis, Cerebral Demyelination, Leucodystrophy, Very-Long-Chain Fatty Acids (VLCFA).

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Received: 11-02-2026;
Revised: 06-03-2026;
Accepted: 28-04-2026.

INTRODUCTION

X-linked ALD is a rare, inherited peroxisomal disorder caused by pathogenic variants in the ABCD1 gene, which encodes the ATP-binding cassette subfamily D Member 1 (ALDP) transporter responsible for importing VLCFAs into peroxisomes for β-oxidation (Dong *et al*., 2025; Kemp *et al*., 2012). When the ALDP function is deficient, saturated VLCFAs, particularly C26:0, accumulate in plasma and tissues, including the central nervous system and adrenal cortex, triggering oxidative stress, microglial activation, and progressive demyelination (Bougnères and Le Stunff, 2025; Kemp *et al*., 2012). Pathophysiological studies reveal that oligodendrocytes lacking ABCD1 are more susceptible to stress and demyelinating insults, suggesting a primary role for glial dysfunction in disease progression (Manor *et al*., 2021).

Clinically, ALD is remarkably heterogeneous. The most common phenotypes include childhood cerebral ALD, characterized by rapid inflammatory demyelination, Adrenomyeloneuropathy (AMN), which typically presents in adulthood with a slowly progressive myelopathy, and isolated adrenal insufficiency (Dong *et al*., 2025; Kemp *et al*., 2012). However, adult-onset cerebral ALD is relatively rare and diagnostically challenging. Unlike the

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DOI: 10.5530/ajbls.20260094

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fulminant course seen in childhood ALD, adult cerebral forms may progress insidiously, and the absence of adrenal dysfunction further complicates recognition (Bougnères and Le Stunff, 2025; Kumar *et al*., 2025).

A presentation dominated by symmetrical spastic quadriparesis in an adult can overlap substantially with other neurological disorders. Differential diagnoses typically include hereditary spastic paraplegia, which is often confined to lower-limb spasticity without radiological demyelination; multiple sclerosis, characterized by episodic deficits and contrast- enhancing lesions; metabolic leucodystrophies with systemic involvement; and compressive myelopathies such as spinal-cord lesions. When Magnetic Resonance Imaging (MRI) changes are subtle or symmetrical without classical inflammatory features, as may occur in adult ALD, misdiagnosis is common.

Early MRI in adult cerebral ALD may reveal only minimal or nonenhancing white-matter abnormalities, making the imaging findings non-specific. In such scenarios, biochemical assays of plasma VLCFAs and genetic sequencing of ABCD1 are essential for establishing diagnosis (Engelen *et al*., 2022; Kemp *et al*., 2012). Elevated C26:0 or C26:0-lysophosphatidylcholine and identification of a pathogenic ABCD1 variant remain the diagnostic gold standard.

Despite advances, the literature lacks detailed reports of adult-onset cerebral ALD presenting with primarily spastic quadriparesis in the absence of overt radiological demyelination or adrenal insufficiency. Such atypical cases are under-recognized, and diagnostic delay is common. This case is therefore significant as it highlights a rare phenotype of ALD, underscores diagnostic pitfalls, and emphasizes the need for biochemical and genetic vigilance in adults with unexplained upper motor neurone syndromes.

CASE DETAILS

A 28-year-old male presented to a tertiary care neurology department with a 2-year history of gradually progressive motor and bulbar symptoms. His illness initially manifested as subtle difficulty climbing stairs, reduced endurance during prolonged walking, and a sense of stiffness in the lower limbs. Over the ensuing months, these symptoms progressed insidiously, leading to a markedly spastic and scissoring gait pattern and increasing reliance on support to ambulate. He reported multiple episodes of falls, primarily due to poor balance and difficulty initiating steps. Approximately six to eight months before admission, he began noticing slurred speech and intermittent choking on liquids, fragile fluids, along with occasional involuntary emotional expression suggestive of pseudobulbar affect. There was no history of sensory symptoms, bladder or bowel dysfunction, fever, seizures, trauma, or prior neurological disorders. His history included regular alcohol consumption and smokeless tobacco use, but there was no significant family history of similar symptoms or known hereditary conditions.

On examination, the patient was alert, attentive, and fully orientated, with stable vital signs and no systemic abnormalities. The neurological evaluation, however, revealed significant involvement of the upper motor neurones. Spasticity was markedly increased in all four limbs, greater in the lower extremities, with brisk deep tendon reflexes and bilateral extensor plantar responses. Muscle strength was preserved to a moderate degree in the upper limbs but was noticeably reduced proximally and distally in the lower limbs, although no muscle wasting or fasciculations were evident. His gait was spastic, stiff, and circumducting, requiring assistance for even short distances. Cranial-nerve examination demonstrated mild deviation of the soft palate with a reduced gag reflex, consistent with early bulbar involvement, and mild cerebellar dysarthria. Sensory examination was unremarkable across all modalities. Primitive reflexes, including snout and palmomental signs, were elicited, reflecting frontal lobe disinhibition.

Baseline laboratory investigations, including complete blood count, serum electrolytes, renal and liver function tests, and viral serology, were within normal limits. MRI of the brain performed during the early hospital course demonstrated symmetrical T2/FLAIR hyperintensities involving the temporo-occipital periventricular white matter, posterior corona radiata, posterior limbs of the internal capsule, posterior frontoparietal white matter, the splenium of the corpus callosum, and bilateral cerebral peduncles. These findings were characteristic of a demyelinating process consistent with adrenoleucodystrophy, particularly in its adult cerebral form. MRI of the lumbar spine revealed only incidental sacralization of L5 and a diffuse L4-L5 disc bulge without neural compression. An ultrasound of the abdomen showed Grade I fatty liver, with no other abdominal abnormalities.

Based on the clinical features and neuroimaging findings, a diagnosis of adult-onset adrenoleucodystrophy was made. The patient was initiated on neurotropic vitamin therapy, intravenous thiamine, pantoprazole, and oral multivitamin supplementation. Baclofen was introduced and gradually titrated to manage spasticity, while glycopyrrolate was added to reduce excessive oral secretions associated with bulbar dysfunction. A structured physiotherapy program was implemented, beginning with tone-reducing exercises and passive mobilization and progressing to targeted strengthening, balance training, and gait rehabilitation. Speech-related strategies were also incorporated to address mild dysarthria and episodic choking.

During the middle of the hospital course, the patient experienced a brief but clinically notable complication. On two separate occasions during physiotherapy sessions, he developed transient tachycardia with mild blood-pressure variability and

a sensation of dizziness when transitioning to upright posture. Comprehensive evaluation, including review of medications, repeat vital monitoring, and ECG, revealed no evidence of infection, dehydration, arrhythmia, or metabolic disturbance. In the absence of any alternative aetiology, the episodes were attributed to transient dysautonomia likely related to involvement of brainstem autonomic pathways affected by the underlying demyelinating process. Physiotherapy intensity was temporarily reduced, hydration was optimized, and the patient was placed under close observation for the next 48 hr, after which the autonomic symptoms resolved spontaneously without requiring additional treatment.

Following stabilization, his rehabilitation regimen was reinstated and gradually intensified. Low-dose amitriptyline was added at bedtime to improve sleep quality and manage neuropathic discomfort. Over the subsequent week, the patient demonstrated steady clinical improvement. His lower-limb spasticity reduced, coordination improved, and he was able to ambulate short distances with support. The frequency of choking episodes diminished, and speech clarity improved noticeably. No further episodes of autonomic instability occurred. By the 16th day of hospitalization, the patient showed substantial functional recovery. His vital signs remained stable, he tolerated physiotherapy well, and he reported overall improvement in mobility and daily activities. He was discharged in a stable and improved condition with instructions to continue baclofen, nighttime amitriptyline, and a structured physiotherapy program, along with scheduled follow-up in the neurology outpatient clinic. The final diagnoses at discharge included MRI-confirmed adrenoleucodystrophy presenting with cerebellar-type symmetrical spastic quadriparesis, transient dysautonomia during hospitalization, which resolved, and incidental Grade I fatty liver.

DISCUSSION

This case describes an adult-onset presentation of X-linked ALD characterized by slowly progressive symmetrical spastic quadriparesis accompanied by subtle, early-stage cerebral demyelination on MRI. The absence of adrenal insufficiency, combined with mild and nonenhancing white-matter abnormalities, contributed to diagnostic uncertainty. This phenotype differs from both classic childhood cerebral ALD, which typically presents with rapidly progressive inflammatory demyelination, and the more common adult variant, AMN, which predominantly manifests as a chronic spastic paraparesis with distal axonopathy rather than overt cerebral involvement (Engelen *et al*., 2022; Kemp *et al*., 2012). The patient's symmetrical corticospinal tract dysfunction, early bulbar features, and subtle occipital-parietal white-matter changes place this presentation among the less frequently recognized adult cerebral ALD variants, which are known to progress insidiously and are often misdiagnosed in their initial stages (Mannari *et al*., 2020).

Comparatively, AMN is characterized primarily by a distal axonopathy of the spinal cord, commonly producing slowly progressive paraparesis, sphincter disturbances, and peripheral neuropathy without significant early cerebral involvement (Raymond *et al*., 1999). In contrast, childhood cerebral ALD typically shows confluent parieto-occipital demyelination with gadolinium enhancement, rapid functional decline, and early cognitive and behavioural changes (Engelen *et al*., 2022). The present case diverges from both extremes: cerebral lesions were present but subtle, nonenhancing, and not accompanied by cognitive decline. This intermediate phenotype underscores the wide heterogeneity of ALD and reflects the well-established absence of genotype-phenotype correlation within ABCD1 mutations (Kemp *et al*., 2012).

The clinical picture in this patient overlapped significantly with other disorders associated with Upper Motor Neurone syndromes. Symmetrical spastic quadriparesis with relatively preserved sensation is a hallmark of Hereditary Spastic Paraplegia, particularly the "complicated" forms that may include dysarthria or mild bulbar involvement (Fink, 2014). Adult-onset cerebral ALD is also frequently mistaken for primary progressive multiple sclerosis, especially when MRI demonstrates scattered or periventricular lesions lacking classical inflammatory signatures (Mannari *et al*., 2020). Similarly, metabolic leucodystrophies such as Krabbe's disease, metachromatic leucodystrophy, or adult polyglucosan body disease may mimic the imaging and clinical appearance of ALD, necessitating targeted biochemical testing for differentiation (Ceravolo *et al*., 2023; Wolf *et al*., 2025). Cervical compressive myelopathy is another frequent misdiagnosis in adult-onset ALD, particularly when spasticity dominates the neurological picture and mild structural abnormalities appear on spinal imaging.

The pathophysiological basis of ALD provides further context for this clinical variability. Mutations in ABCD1 disrupt peroxisomal import of VLCFAs, leading to intracellular accumulation of saturated VLCFAs that exert neurotoxic effects through oxidative stress, inflammatory microglial activation, and mitochondrial dysfunction (Bougnères and Le Stunff, 2025; Ceravolo *et al*., 2023). While all males with ABCD1 mutations accumulate VLCFAs, only a subset develop cerebral inflammatory disease. Adult cerebral ALD is uncommon because oligodendrocyte vulnerability and breakdown of immune tolerance to myelin antigens appear more prominent in early neurodevelopment, whereas adults typically develop AMN-like axonopathy without explosive inflammation (Engelen *et al*., 2022). The patient's corticospinal tract involvement, resulting in symmetrical spastic quadriparesis, reflects degeneration or demyelination of long motor tracts, a recognized but less frequently described manifestation of adult cerebral ALD (Mannari *et al*., 2020).

Diagnostic challenges in this case were significant and consistent with documented difficulties in adult ALD. Early MRI changes

may be subtle, symmetrical, nonenhancing, or limited to posterior periventricular regions, often interpreted as nonspecific white-matter disease or microvascular changes (Raymond *et al.*, 1999). Because MRI alone may be insufficient in early adult cerebral ALD, biochemical confirmation with elevated plasma VLCFA levels, particularly C26:0- Lysophosphatidylcholine, remains essential (Haynes and De Jesús, 2012). Genetic sequencing of ABCD1 is required for definitive diagnosis and is now recommended by international guidelines (Engelen *et al.*, 2022). Although this patient lacked adrenal symptoms, endocrine evaluation is advised in all males with ALD due to the risk of subclinical adrenal insufficiency, which can precede neurological manifestations (Bougnères and Le Stunff, 2025).

Management options for adult cerebral ALD remain limited. Hematopoietic Stem Cell Transplantation (HSCT) is the only intervention capable of halting early-stage cerebral inflammation, but its benefit is restricted to patients with minimal MRI involvement and no significant neurological disability at the time of treatment (Engelen *et al.*, 2022). Because adult cerebral ALD often progresses more slowly and is recognized late, many patients, including this one, present outside the therapeutic window for HSCT. Lorenzo's oil and dietary VLCFA-lowering regimens can reduce plasma VLCFA concentrations but have not shown consistent benefit once neurological symptoms appear (Raymond *et al.*, 1999). Supportive therapy, such as spasticity management, physiotherapy, and multidisciplinary care, remains central. Delayed diagnosis, as seen in many adult-onset cases, further limits therapeutic opportunities and contributes to progressive neurological decline.

Prognosis in adult cerebral ALD varies. While progression is slower compared to childhood forms, continued cerebral demyelination typically leads to increasing motor impairment, dysarthria, swallowing difficulties, and functional decline over several years (Mannari *et al.*, 2020). Early recognition improves long-term outlook by enabling timely surveillance, endocrine management, and evaluation for HSCT when appropriate (Engelen *et al.*, 2022). Atypical cases with subtle imaging findings, like the present one, pose a risk for prolonged diagnostic delays and missed therapeutic windows.

This case, therefore, contributes significant value to current understanding of ALD by demonstrating an uncommon adult-onset cerebral phenotype presenting primarily as symmetrical spastic quadriparesis with early occult demyelination and no adrenal manifestations. It highlights the importance of considering ALD in adults with unexplained Upper Motor Neurone syndromes, even when MRI shows subtle abnormalities, and reinforces the need for prompt biochemical and genetic evaluation to avoid misdiagnosis and irreversible disease progression.

CONCLUSION

This case underscores the clinical complexity and diagnostic challenges of adult-onset X-linked adrenoleukodystrophy, particularly when it presents slowly progressive symmetrical spastic quadriparesis and only subtle cerebral demyelination. Such presentations fall outside the classical spectrum of both childhood cerebral ALD and adrenomyeloneuropathy, often leading clinicians toward more common mimics such as hereditary spastic paraplegia, primary progressive multiple sclerosis, or degenerative myelopathies. The subtlety of early neuroimaging changes in adult cerebral ALD further compounds diagnostic delay, reinforcing the necessity of maintaining a high index of suspicion in adults with unexplained upper motor neurone syndromes.

This case highlights the indispensable role of biochemical VLCFA profiling and *ABCD1* genetic testing when MRI findings are equivocal. Early identification is particularly crucial because therapeutic opportunities, such as hematopoietic stem cell transplantation, are time- sensitive and dependent on recognizing cerebral involvement before significant neurological deterioration occurs. Although no disease-modifying therapy currently exists for advanced adult cerebral ALD, timely diagnosis enables anticipatory management, endocrine surveillance, genetic counselling, and structured rehabilitation to optimize the patient's long- term functional trajectory.

By documenting an uncommon adult-onset cerebral phenotype without adrenal insufficiency or florid radiological changes, this case adds to a growing body of evidence urging clinicians to broaden their diagnostic approach to adult spastic paraparesis and quadriparesis. It emphasizes that even subtle imaging abnormalities should prompt targeted metabolic and genetic investigation. Ultimately, the case reinforces the need for early recognition of atypical ALD variants to prevent missed therapeutic windows and support more accurate prognostication and patient-centred care.

PATIENT PERSPECTIVE

The patient reported that the gradual worsening of stiffness and difficulty walking had significantly affected his independence and confidence over the past 2 years. Before receiving a diagnosis, he described feeling confused and discouraged by the lack of clarity regarding his symptoms, as multiple consultations had not provided definitive answers. He expressed relief when the underlying condition was finally identified, noting that having an explanation for his symptoms helped him understand what was happening to his body.

The patient also shared concerns about the progressive nature of his condition but stated that the clear communication from the medical team, along with structured physiotherapy and symptom-directed management, helped him feel more in control.

He emphasized that learning about the importance of early recognition in such rare disorders motivated him to participate actively in follow-up care. Overall, he expressed gratitude for the coordinated approach and reported meaningful improvement in mobility and daily functioning during hospitalization.

ACKNOWLEDGEMENT

The authors acknowledge the contributions of the neurology, radiology, physiotherapy, and laboratory teams involved in the patient's diagnostic evaluation and ongoing care.

ABBREVIATIONS

ALD: Adrenoleukodystrophy; **ABCD1:** ATP-binding cassette subfamily D member 1; **ALDP:** Adrenoleukodystrophy Protein; **VLCFAs:** Very-Long-Chain Fatty Acids; **AMN:** Adrenomyeloneuropathy; **MRI:** Magnetic Resonance Imaging; **UMN:** Upper Motor Neuron; **HSP:** Hereditary Spastic Paraplegia; **PPMS:** Primary Progressive Multiple Sclerosis; **HSCT:** Hematopoietic Stem Cell Transplantation; **ECG:** Electrocardiogram; **FLAIR:** Fluid-Attenuated Inversion Recovery.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

No external funding or financial support was received for the preparation of this case report.

AUTHORS' CONTRIBUTIONS

PKY contributed to case identification, data interpretation, manuscript drafting, and critical revision. S. S., D. J., S. M., G. L. B. and G. M. contributed to patient data collection, literature review, and drafting of background and Discussion. B. D. M. contributed to data verification, preparation of clinical details, and editing of the Abstract and Case Presentation. R. R. N. provided project oversight, ensured methodological integrity, critically reviewed the manuscript, and approved the final version. All authors certify that they meet the CARE authorship criteria and accept responsibility for the integrity of the work.

ETHICAL APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was not required for this case report as per institutional guidelines for single- patient case documentation.

CONSENT FOR PUBLICATION

Written informed consent for publication of this case report, including anonymized clinical details and imaging findings, was obtained from the patient. A copy of the consent form is available for review by the journal's editorial office upon request.

REFERENCES

Bougnères, P., and Le Stunff, C. L. (2025). Revisiting the pathogenesis of X-linked adrenoleukodystrophy. Genes [Internet], 16(5), 590. doi: 10.3390/genes16050590, PubMed: 40428412

Ceravolo, G., Zhelcheska, K., Squadrito, V., Pellerin, D., Gitto, E., Hartley, L., and Houlden, H. (2023). Update on leukodystrophies and developing trials. Journal of Neurology [Internet], 271(1), 593-605. doi: 10.1007/s00415-023-11996-5, PubMed: 37755460

Dong, S. W., Xiao, L. M., Sun, Y. H., Li, G. H., Xie, Y. X., Wang, M. W., (2025). Novel ABCD1 variants in X-linked adrenoleukodystrophy. Clinical Genetics [Internet], 108(4), 450-456. doi: 10.1111/cge.14752, PubMed: 40210590

Engelen, M., Van Ballegoij, W. J. C., Mallack, E. J., Van Haren, K. P., Köhler, W., Salsano, E., (2022). International recommendations for the diagnosis and management of patients with adrenoleukodystrophy: A consensus-based approach. Neurology [Internet], 99(21), 940-951. doi: 10.1212/WNL.0000000000201374, PubMed: 36175155

Fink, J. K. (2014). Hereditary Spastic paraplegia: Clinical principles and genetic advances. Seminars in Neurology [Internet], 34(3), 293-305. doi: 10.1055/s-0034-1386767, PubMed: 25192507

Haynes, C. A., and De Jesús, V. R. (2012). Improved analysis of C26:0-lysophosphatidylcholine in dried-blood spots via negative ion mode HPLC-ESI-MS/MS for X-linked adrenoleukodystrophy newborn screening. Clinica Chimica Acta [Internet], 413(15-16), 1217-1221. doi: 10.1016/j.cca.2012.03.026

Kemp, S., Berger, J., and Aubourg, P. (2012). X-linked adrenoleukodystrophy: Clinical, metabolic, genetic and pathophysiological aspects. Biochimica et Biophysica Acta [Internet], 1822(9), 1465-1474. doi: 10.1016/j.bbadis.2012.03.012, PubMed: 22483867

Kumar, M., Kumari, N., Prateek, R., and Praween, N. (2025). A case of adult-onset adrenoleukodystrophy with a rare genetic mutation. International Journal of Current Research and Review, 17(15), 1-3. doi: 10.31782/IJCRR.2025.171501

Mannari, A., Wiggins, B., and Bachuwa, G. (2020). Adult-Onset cerebral adrenoleukodystrophy without adrenal gland involvement. Cureus [Internet], 12(8), e9813 cerebral-adrenoleukodystrophy-without-adrenal-gland-involvement#!. doi: 10.7759/cureus.9813, PubMed: 32953324/

Manor, J., Chung, H., Bhagwat, P. K., and Wangler, M. F. (2021). ABCD1 and X-linked adrenoleukodystrophy: A disease with a markedly variable phenotype showing conserved neurobiology in animal models. Journal of Neuroscience Research [Internet], 99(12), 3170-3181. doi: 10.1002/jnr.24953, PubMed: 34716609

Raymond, G. V., Moser, A. B., Fatemi, A., Department of Genetic Medicine, Johns Hopkins Hospital, Baltimore, M., (1999). X-linked adrenoleukodystrophy [Internet]. GeneReviews M. P. Adam, S. Bick and G. M. Mirzaa (Eds.).Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK1315/pdf/Bookshelf_NBK1315.pdf

Singh, I., and Pujol, A. (2010). Pathomechanisms underlying X-adrenoleukodystrophy: A three-hit hypothesis. Brain Pathology, 20(4), 838-844. doi: 10.1111/j.1750-3639.2010.00392.x. Retrieved from https://diposit.ub.edu/dspace/handle/2445/126806. PubMed: 20626745

Wolf, N. I., Van Der Knaap, M. S., and Engelen, M. (2025). Treatment of leukodystrophies: Advances and challenges. European Journal of Paediatric Neurology [Internet], 56, 46-50. doi: 10.1016/j.ejpn.2025.03.016, PubMed: 40279833

Cite this article: Sharmila S, Jinka D, Madineni G, Matta S, Bolisetty GL, Yanamadala PK, *et al*. Symmetric Spastic Quadriparesis with Occult Cerebral Demyelination: An Adult-Onset Adrenoleukodystrophy Case Report. Asian J Biol Life Sci. 2026;15(1):247-51.