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Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS): A Comprehensive Review of Clinical Manifestations, Diagnostic Strategies, and Therapeutic Insight

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ABSTRACT: Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) is a rare neurodegenerative disorder characterized by a triad of cerebellar ataxia, sensory neuropathy, and bilateral vestibular areflexia. The recent discovery of biallelicintronicpentanucleotide repeat expansions in the RFC1 gene has enhanced understanding of its genetic basis. No curative pharmacological treatments exist; management focuses on symptomatic relief and supportive multidisciplinary care. This review summarizes clinical features, diagnostic strategies, and emerging therapeutic options. We also present a case of a 6-year-old male child who exhibited vomiting and tonic posturing. MRI revealed bilateral symmetrical white matter abnormalities and elevated neurochemical markers indicative of neurodegeneration. This case highlights the importance of considering CANVAS in pediatric patients with unexplained neurological symptoms. Emphasizing current management and the urgent need for pharmaceutical research, this review aims to enhance diagnosis and promote the development of disease-modifying treatments.

KEYWORDS: Cerebellar Ataxia, Sensory Neuropathy, Vestibular Dysfunction, Neurodegenerative Disorder

I. INTRODUCTION:

Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) is an uncommon, progressively debilitating neurodegenerative disorder characterized by the simultaneous involvement of three crucial neurological domains: cerebellar dysfunction, sensory neuropathy, and bilateral vestibular failure.[1] First systematically described in recent years, CANVAS represents a unique clinical syndrome distinguished by the triad of cerebellar ataxia leading to gait and limb coordination deficits, sensory neuropathy predominantly involving the dorsal root ganglia resulting in profound sensory ataxia, and bilateral vestibular areflexia that manifests clinically with oscillopsia and balance impairment.[2] Traditionally considered a disorder of middle-aged and elderly adults, emerging evidence now identifies paediatric and younger adult cases, broadening the demographic spectrum and challenging clinicians to recognize the disease earlier. The rarity and variable manifestations of CANVAS, coupled with overlapping features shared by other hereditary, autoimmune, or degenerative ataxias and neuropathies, often result in delayed or missed diagnosis.[3] Additionally, the underlying molecular pathogenesis, largely attributed to balletic repeat expansions in the RFC1 gene, implicates a novel genetic mechanism distinct from classical neurodegenerative disorders. Radiological investigations, especially advanced MRI and MR spectroscopy, demonstrate characteristic patterns of cerebellar and brainstem atrophy alongside neurochemical alterations, contributing to diagnostic clarity. Clinically, patients gradually develop progressive imbalance, chronic cough, neuropathic symptoms, and vestibular-related visual disturbances, which together severely affect quality of life.[4] Despite no curative treatment presently available, understanding the natural history and multidisciplinary management strategies is vital to optimizing patient outcomes. This comprehensive review aims to delve into the epidemiology, pathophysiological mechanisms, clinical spectrum, diagnostic approach, and current management paradigms of CANVAS, exemplified by a rare pediatric case to highlight the importance of vigilance across all age groups.

EPIDEMIOLOGY: Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) is a rare neurodegenerative disorder with an incompletely understood epidemiological profile, largely due to its recent recognition and diagnostic challenges. According to Cortese et al.

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(2021), the true prevalence of CANVAS is likely underestimated because the syndrome presents with subtle and overlapping neurological symptoms that can be misdiagnosed as other ataxias or neuropathies Typically, CANVAS manifests in late adulthood, most commonly in the sixth and seventh decades of life, making it a disease predominantly of middle-aged and elderly populations.[5] However, recent case series, including familial reports by Gökçay et al. (2024), indicate that the age spectrum is broader than initially appreciated, with emerging paediatric cases increasingly reported. This suggests possible underrecognition of CANVAS in younger patients, who might present with atypical features that delay diagnosis.[6]The gender distribution appears roughly equal, although some adult cohorts report a slight male predominance.[7] In contrast to many neurodegenerative disorders that exhibit a female predominance, CANVAS does not appear to be markedly gender-biased (Cortese et al., 2021) Family clustering is uncommon but has been documented, as noted by Gökçay et al. (2024) in their report of a Turkish family with five affected siblings, supporting a genetic contribution, particularly related to biallelicintronic expansions in the RFC1 gene. This familial aggregation highlights the importance of genetic counseling and consideration of inheritance patterns in affected families.[8]

ETIOLOGY: Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) is primarily a neurodegenerative disorder with a strong genetic basis.[9] The leading etiological factor identified is a biallelicintronicpenta-nucleotide repeat expansion (AAGGG)n in the *Replication Factor C Subunit 1 (RFC1) gene.[10] This discovery, reported by Cortese et al. (2019), has transformed understanding of CANVAS as a genetically mediated disease caused by non-coding repeat expansions that disrupt normal gene function, although the exact pathogenic mechanism remains under investigation. Most cases of CANVAS appear sporadic with no clear family history; however, familial clustering has been documented. Gökçay et al. (2024) reported a family with multiple affected siblings, reinforcing a hereditary component in some cases linked with RFC1 repeat expansions. This suggests autosomal recessive inheritance in many affected families, although exceptions and incomplete penetrance may occur.[11]

- **Beyond genetic causes, other etiologies have been considered and excluded in typical CANVAS diagnosis, including:**

1. **Autoimmune and paraneoplastic neuropathies:** Autoimmune processes causing sensory neuronopathy may mimic CANVAS, necessitating exclusion via serologic and paraneoplastic panels. Hirano et al. (2023) explored immune-mediated neuropathies with RFC1 mutation backgrounds, suggesting potential overlaps or triggering autoimmune responses.[12]
2. **Other neurodegenerative ataxias:** Differential diagnosis includes late-onset Friedreich's ataxia, spinocerebellar ataxias, and sensory neuronopathies of various origins, but these usually show distinct genetic and clinical profiles.
3. **Vascular and metabolic causes:** These are generally ruled out through clinical evaluation, imaging, and laboratory testing.[13]

PATHOPHYSIOLOGY:
Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) is characterized by a distinctive neurodegenerative process that selectively involves three key neural structures: the cerebellum, the dorsal root ganglia (sensory ganglia), and the vestibular system.

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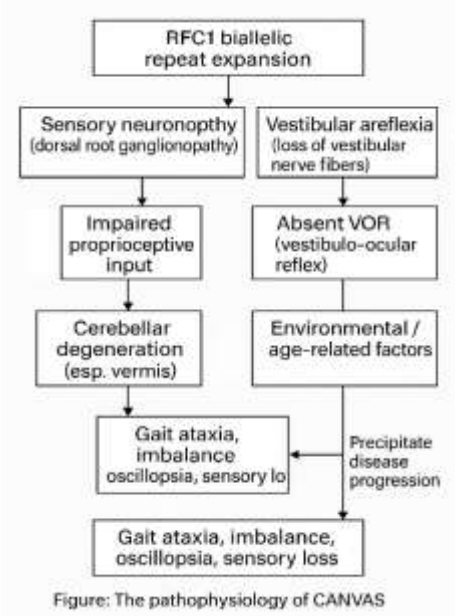

Figure: The pathophysiology of CANVAS

Cerebellar Degeneration: The hallmark of CANVAS is progressive loss of cerebellar Purkinje cells and their dendritic arborisation, primarily affecting the vermin and adjacent cerebellar cortex. This degeneration leads to impaired motor coordination, gait instability, and limb ataxia. Histopathological studies report marked neuronal loss and gliosis within cerebellar regions, which correlates clinically with ataxia and dysarthria.

Sensory Neuronopathy (Ganglionopathy): CANVAS involves significant loss of large dorsal root ganglion neurons, resulting in a sensory neuronopathy rather than a distal axonal neuropathy. This preferential involvement of the sensory ganglia results in profound deficits in proprioception and the vibratory sense, manifesting as sensory ataxia. Damage to these ganglia impairs afferent input to the spinal cord and cerebellum, thereby exacerbating balance and coordination issues.

Bilateral Vestibular Areflexia: Degeneration extends to the vestibular nerve fibers and Scarpa's ganglia, causing bilateral vestibular hypofunction. Clinically, this produces oscillopsia, impaired vestibulo-ocular reflexes, and imbalance, particularly noticeable during head movements or in low-visibility conditions. The bilateral nature of vestibular loss differentiates CANVAS from other vestibular disorders that are typically unilateral.

Neuroimaging Correlates: Magnetic Resonance Imaging (MRI) frequently reveals cerebellar atrophy, particularly of the vermis. Additionally, there are characteristic bilateral symmetrical hyperintense signals in deep and subcortical white matter seen on T2-weighted and FLAIR sequences, involving structures such as the globuspallidus, thalami, and brainstem. These signal changes suggest a widespread neurodegenerative process affecting multiple CNS sites. MR spectroscopy often demonstrates elevated N-acetyl aspartate (NAA) and an increased NAA/Creatine ratio, reflecting neuronal loss and gliosis.

Molecular Pathogenesis: The underlying cause is linked to a biallelicintronic expansion of the AAGGG pentanucleotide repeat in the RFC1 gene, which codes for Replication Factor C subunit 1, a critical protein for DNA replication and repair. The repeat expansion likely leads to aberrant RNA or protein products that progressively impair neuronal survival. However, the exact pathological mechanisms, including whether the mutation causes a toxic gain-of-function or loss-of-function effect, remain to be fully elucidated.

Clinical Consequences of Pathophysiology: The combined degeneration of cerebellar, sensory, and vestibular systems results in a compounded effect on balance and motor coordination, leading to the characteristic clinical triad. Loss of proprioceptive sensory input from ganglionopathy disrupts the sensory feedback loop essential for motor control, while cerebellar involvement impairs motor planning and coordination. Vestibular loss further impairs postural stability and visual fixation, leading to recurrent falls and visual symptoms like oscillopsia.[14]

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MANAGEMENT AND THERAPEUTIC STRATEGIES IN CANVAS

Currently, there is no curative treatment for Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS). Management focuses primarily on symptomatic relief, supportive care, and improving patients' quality of life through multidisciplinary interventions. Given the progressive neurodegenerative nature of CANVAS, timely diagnosis and comprehensive management are crucial.

PHARMACOLOGICAL MANAGEMENT

- ✓ Symptomatic Pharmacotherapy: While no disease-modifying drugs exist, several symptomatic treatments may help alleviate specific manifestations:
- ✓ Ataxia and gait disturbances: Pharmacological agents such as riluzole or idebenone have been explored in other ataxias, but data in CANVAS are limited. Off-label use may be considered on a case-by-case basis.
- ✓ Neuropathic pain: Medications including gabapentin, pregabalin, and duloxetine can be employed to manage neuropathic symptoms.
- ✓ Vestibular symptoms: Vestibular suppressants like betahistine may provide symptomatic relief in some cases but are used cautiously to avoid impairing compensatory mechanisms.
- ✓ Supportive Pharmacotherapy: Addressing associated symptoms such as spasticity, muscle cramps, or autonomic dysfunction may improve patient comfort. Emerging Therapies and Research: Understanding of the RFC1 gene intronicpentanucleotide repeat expansion as the core pathogenic mechanism opens potential avenues for targeted therapies. Ongoing investigations into gene therapy, RNA-based interventions, or small molecules modulating related pathways may, in the future, translate into disease-modifying options.

Non-Pharmacological and Multidisciplinary Management

- ✓ Physical and Occupational Therapy: Customized rehabilitation programs focusing on balance, coordination, and strength training are essential for maintaining mobility and delaying disability progression.
- ✓ Vestibular Rehabilitation Therapy (VRT): Tailored VRT aids in compensation for vestibular hypofunction, improving stability and reducing fall risk.
- ✓ Speech and Swallowing Therapy: To address dysarthria and dysphagia when present.
- ✓ Nutritional Support and Assistive Devices: May be necessary to prevent complications and improve independence.
- ✓ Psychosocial Support: Addressing mental health and caregiver burden is integral, with input from psychology and social work professionals.

Importance of Multidisciplinary Care Teams Including Pharmacists: Involvement of clinical pharmacists is vital to optimize medication regimens, minimize polypharmacy risks, and ensure appropriate symptomatic pharmacotherapy. Pharmacists can also educate patients on medication adherence and potential side effects.

II. CASE PRESENTATION:

A 6-year-old male child was admitted to the Government General Hospital, Kadapa, presenting with emesis and abnormal motor activity. The abnormal movements occurred after emesis and were characterized by tonic posturing accompanied by ocular deviation, persisting for approximately 5 minutes. There was a reported history of one episode of emesis preceding the abnormal movements. The child's medical history included febrile seizures, for which he had been intermittently receiving Syrup Clobium as prophylaxis. The child's weight upon admission was 10.5 kg. Clinical examination revealed an alert child with notable neurological findings. The pupils were dilated but responsive to light stimuli, indicating an intact pupillary reflex. A positive nystagmus sign was observed, suggesting vestibular involvement. The child exhibited abnormal motor activity, including tonic posturing and ocular deviation. No other significant findings were noted on systemic examination.

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Magnetic resonance imaging (MRI) of the brain demonstrated bilateral symmetrical diffuse signal abnormalities in the deep and subcortical white matter, with involvement of the globuspallidus, thalami, and brainstem, consistent with findings in CANVAS syndrome. Additionally, elevated levels of N-acetyl aspartate (NAA) and an increased NAA/Creatine ratio were noted, indicative of neurodegenerative changes commonly observed in this syndrome.

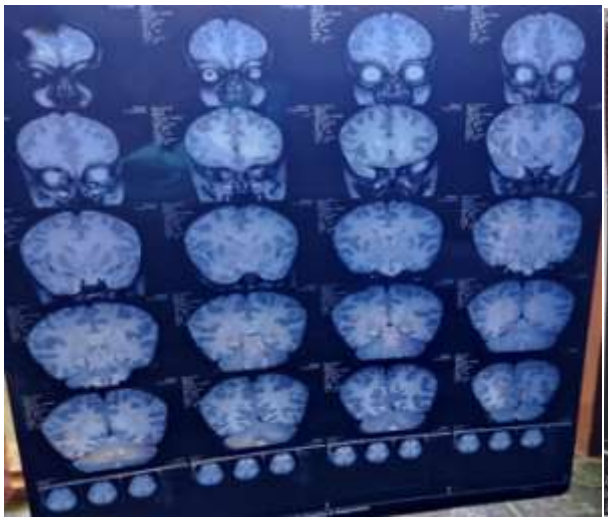

Fig 01: bilateral symmetrical diffuse signal abnormalities in the deep and subcortical white matter, including the globuspallidus,

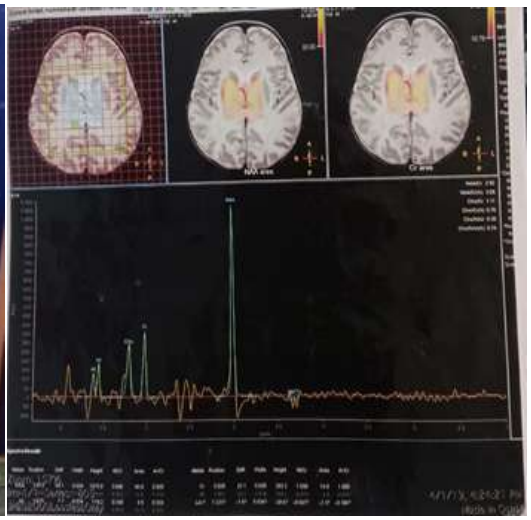

Fig 02: Altered metabolic peaks, possibly indicating neuronal degeneration, and gliosis changes seen.

Based on the clinical presentation, MRI findings, and metabolic studies, a diagnosis of Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) was established. On Day 1 of admission, the child was initiated on intravenous fluid therapy (DNS at 40ml/hr) for hydration. InjLevipil 200mg IV BD (2ml + 8ml in NS) was administered for seizure prophylaxis, with doses given at 12 AM and 12 PM. InjOndansetron 4mg IV BD (0.7cc) was administered for antiemetic purposes. On Day 2, the child received Inj Calcium Gluconate 10ml in NS at 20ml/hr at 9 AM and 9 PM to address potential calcium imbalances. Upon discharge, the child was prescribed Syrup Levipil 5ml BD for continued seizure prophylaxis, Syrup Calcin 5ml OD for calcium supplementation, and Syrup Multivitamin 5ml OD for nutritional support. The family was advised to adhere to regular follow-up appointments to monitor the child's neurological progress.

III. DISCUSSION:

A comparative discussion of CANVAS (Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome) based on recent authoritative literature reveals strong consensus alongside emerging nuanced insights.[15] Authors such as

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Sullivan et al. (2021) and Cortese et al. (2020) agree that CANVAS typically presents in middle to late adulthood (50-70 years) with an equal sex distribution and is characterized by the classical triad of progressive cerebellar ataxia, sensory neuropathy or neuronopathy, and bilateral vestibular areflexia.[16] They emphasize that sensory neuronopathy is ubiquitous in genetically confirmed cases, while some patients manifest the full triad only after several years, reflecting phenotypic variability.[17] Common additional symptoms include chronic spasmodic cough often predating neurological symptoms by decades,autonomic dysfunction, and dysphagia. The triad's components independently contribute to imbalance and disability, with combined degeneration severely impairing vestibule ocular reflexes.[18] The genetic cause is unanimously identified as biallelicintronic AAGGG pentanucleotide repeat expansions in the RFC1 gene (chromosome 4p14), inherited autosomal, resulting in neurodegeneration of cerebellar, sensory, and vestibular systems. Studies like Singh et al. (2024) contribute mechanistic insights into disrupted DNA replication and RNA processing as pathogenic pathways.[19] Diagnosis incorporates clinical assessment of the triad, neurophysiological confirmation of severe axonal sensory neuropathy and vestibular impairment, MRI evidence of cerebellar (notably vermian) atrophy, and genetic confirmation via flanking and repeat-primed PCR or Southern blot. Cortese and colleagues highlight that genetic testing is crucial even when the triad is incomplete, as isolated sensory neuropathy or vestibular deficits may precede full symptomatology.[20] Emerging long-read sequencing technologies promise improved detection accuracy.[21] Management is uniformly described as supportive, focusing on physical and vestibular rehabilitation, assistive devices, and symptom control, including neuropathic pain and chronic cough. Despite slow progression over 10-15 years, leading to significant disability and frequent need for walking aids, CANVAS is generally not directly life-threatening, though complications such as aspiration pneumonia may occur.[22]In summary, across multiple key articles from 2020 to 2025, authors converge on CANVAS's hallmark clinical triad, genetic etiology, diagnostic strategies, and the current lack of disease-modifying treatment, while expanding the recognized symptom spectrum and emphasizing early genetic testing to address phenotypic variability and ensure accurate diagnosis. This comparative perspective integrates findings from Sullivan et al. (2021), Cortese et al. (2020, 2022), Yun et al. (2024), and Singh et al. (2024), providing a comprehensive, current understanding of CANVAS's clinical and genetic profile, diagnostics, and care.

IV. CONCLUSION:

CANVAS is a progressive neurodegenerative disorder lacking disease-modifying therapies, with current management focusing on symptomatic and supportive pharmacological treatment. The RFC1 gene discovery offers avenues for future targeted therapy. Multidisciplinary care remains essential to optimize outcomes. Our case of a 6-year-old male child with early neurological signs underscores the need to consider CANVAS in pediatric patients. Intensified pharmaceutical research and early diagnosis are crucial to improving care and developing effective therapies.

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CONFLICT OF INTEREST: The authors declare that there is no conflict of interest

ABBREVIATIONS :CANVAS: Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome, RFC1: replication factor C Subunit, NAA: N-acetyl aspartate, NCS: Nerve conduction studies, MRI: Magnetic resonance imaging

PATIENT CONSENT The patient referenced in this case report has provided consent for publication, acknowledging the report's nature and understanding that their identity will be kept confidential. The patient is satisfied with the medication they received.

V. SUMMARY

An uncommon neurological disease called Cerebellar Ataxia with Neuropathy and Vestibular Areflexia Syndrome (CANVAS) is known to mostly affect adults, while there is growing evidence that it can also manifest in children. The 6-year-old boy in this case report was admitted with vomiting and atypical motions, such as ocular deviation and tonic posture. The child was taking syrup Clobium as a preventative measure on an occasional basis because of a history of febrile

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seizures. A clinical examination showed dilated pupils that were responsive to light and nystagmus. Bilateral symmetrical anomalies in the brainstem, thalami, globuspallidus, and cerebral white matter were shown on MRI results, along with higher levels of N-acetyl aspartate (NAA), which suggested a neurodegenerative process. After receiving intravenous fluids, antiemetics, supportive care, seizure prophylaxis (Levipil), and outpatient monitoring, the kid was treated. Traditional recognition of CANVAS as an adult-onset illness suggests that it may be underdiagnosed because of its fluctuating appearance in pediatric populations. This instance emphasizes the necessity for more research into the early-onset signs and genetic foundations of CANVAS, as well as the significance of considering it in children who exhibit unexplained neurological symptoms.

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