Pedia Companion

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Pedia Companion

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Association between Serum 25-Hydroxyvitamin D Levels and Lung Diseases in Preterm Infants

Introduction

Vitamin D is a fat-soluble steroid essential for various biological processes, including calcium and phosphorus absorption, immune regulation, and bone health. It is metabolized in the liver to form 25-hydroxyvitamin D_3 [$25(OH)D_3$], which is further converted in the kidneys to the active form, 1,25-dihydroxyvitamin D_3 [$1,25(OH)D_3$].[1] In addition, vitamin D is crucial for several aspects of lung development, including branching morphogenesis, the proliferation of alveolar type-2 cells, surfactant phospholipid secretion, and overall lung maturation.[2] Adequate vitamin D levels also helps in maintaining bone health and are believed to impact neonatal lung development and disease.[1] Given the importance of vitamin D, this study aims to investigate the vitamin D deficiency status in preterm infants and its correlation with lung diseases in these infants.

Methods

This retrospective cohort study included preterm infants with the gestational age of less than 37 weeks, and a vitamin D test conducted within 24 hours of birth. Data included infant characteristics (gestational age, birth weight, sex, etc.), maternal data (age, pregnancy complications, etc.), and clinical outcomes (use of lung surfactant, respiratory complications). Serum 25(OH)D levels were measured using electrochemiluminescence immunoassay. Vitamin D levels were classified as deficient (<20 ng/mL), insufficient (20-30 ng/mL), or adequate (>30 ng/mL). Logistic regression was used to analyse the relationship between 25(OH)D levels and pulmonary complications.

Results

The study included 237 preterm infants with a mean gestational age of (34.10 ± 1.20) weeks (28-36 weeks). The mean serum 25(OH)D level was (16.97.1±8.06) ng/mL, with 175 cases (73.84%) of infants showing vitamin D deficiency, 39 cases (16.46%) insufficiency, and 23 cases (9.70%) adequacy. Fall was the season with a higher incidence of vitamin D deficiency compared to other seasons. There were no significant differences between the low- and high-vitamin D groups in terms of gestational age, birth weight, sex, delivery mode, and other variables. The use of lung surfactant was significantly higher in the low-vitamin D group. The incidence of respiratory distress syndrome (RDS) and bronchopulmonary dysplasia (BPD) was significantly higher in the low-vitamin D group compared to the high-vitamin D group (Figure

1). Higher serum 25(OH)D levels were associated with a reduced risk of pulmonary complications. A higher 5-minute Apgar score and prenatal hormone use also reduced the risk of complications.

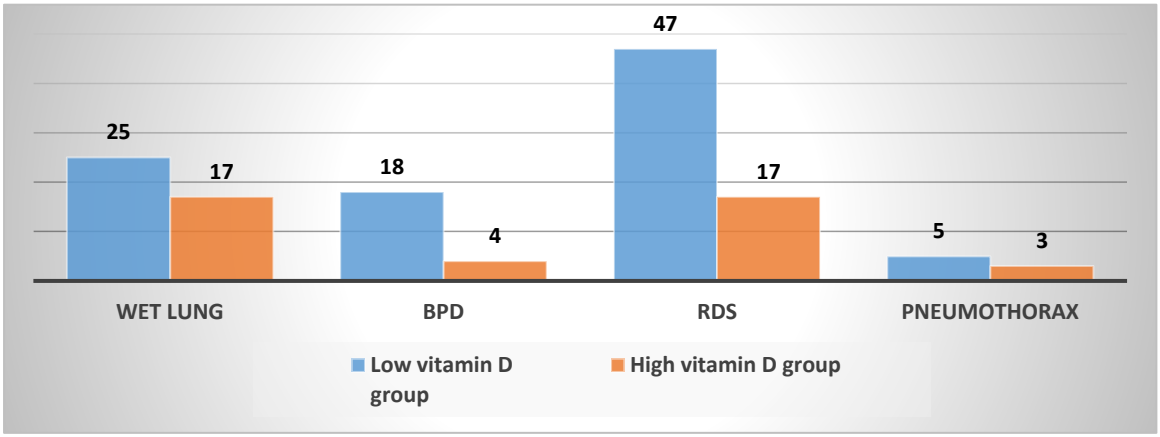

Figure 1. Comparison of pulmonary complications in children between the two groups.

Discussion

Vitamin D deficiency is prevalent among preterm infants and correlates with a higher incidence of lung complications. This study supports existing evidence that vitamin D plays a critical role in lung development and function.[1] Previous study found that low vitamin D levels are associated with a higher incidence of RDS and BPD.[3] Furthermore, Ge et al. demonstrated that vitamin D supplementation reduced the incidence of BPD in preterm infants, reinforcing the protective role of vitamin D against lung diseases.[4] These findings align with the present study results that higher vitamin D levels are associated with reduced pulmonary complications.[1]

Conclusion

Vitamin D deficiency is common in preterm infants and was linked to increased pulmonary complications. Early supplementation of vitamin D could potentially mitigate these risks.

Low vitamin D levels at birth in preterm infants may increase the incidence of respiratory distress syndrome and bronchopulmonary dysplasia.

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Relationship Between Unusual Sleep Patterns and Indoor Environmental Risks in Children with Chronic Cough

Introduction

Chronic cough in children, defined as a cough lasting more than 4 weeks affecting around 5 to 10% of the general population and is a major concern, causing unnecessary medical visits and significant costs.[1] It affects children's sleep, activities, emotions, and overall quality of life. Sleep quality is crucial for children's health, but sleep-disordered breathing (SDB) can disrupt it, leading to various issues. Obstructive sleep apnea syndrome (OSAS), where children snore and may have difficulty breathing during sleep, affects about 25% of those who snore. Indoor environmental factors, such as pollution, mold, pets, and tobacco smoke, also affect sleep quality and respiratory health. However, there was limited research on how these factors impact children with chronic cough.[2] This study aimed to examine the prevalence of abnormal sleep behaviours and the impact of indoor environmental factors on sleep in children with chronic cough.

Methods

This cross-sectional study was conducted on children aged between 3-18 years. Questionnaires were distributed by school teachers to parents or guardians and collected after completion. Children aged 16–18 years completed the questionnaires with their primary carers. Data on sociodemographic factors, allergies, home environmental exposures, and sleep characteristics of the participants were collected using paper-based questionnaires. Logistic regression models were employed to explore associations between abnormal sleep behaviours, chronic cough, and indoor environmental factors, adjusting for confounding variables.

Results

A total of 2,026 valid responses were considered of which 316 responses were from children with chronic cough, and 1,710 were from children without chronic cough, resulting in a survey-weighted prevalence of chronic cough at 15.50%. Children with chronic cough had significantly higher incidences of eczema (45.25% vs. 30.29%), wheezing (24.37% vs. 143%), rhinitis (42.09% vs. 24.32%), food allergy (12.66% vs. 6.90%) and nasosinusitis (8.87% vs. 4.67%) than controls (Figure 1). Family history of sleep disorders (28.16% vs. 19.29%) and adenoid hypertrophy (7.91% vs. 3.92%) was more common in the chronic cough group. The average sleep duration was 8.95 hours for children with chronic cough, and 8.85 hours for controls. Chronic cough was associated with various sleep disturbances, including snoring, restless sleep, bruxism (teeth grinding), and sweating. Mold exposure was identified as a high-

risk factor for chronic cough. In children with chronic cough, recent home decoration was linked to sleepwalking; mold exposure to bruxism; and carpet use to apnea, twitching during sleep, and morning headaches. In the non-chronic cough group, pet ownership was associated with several sleep-related issues.

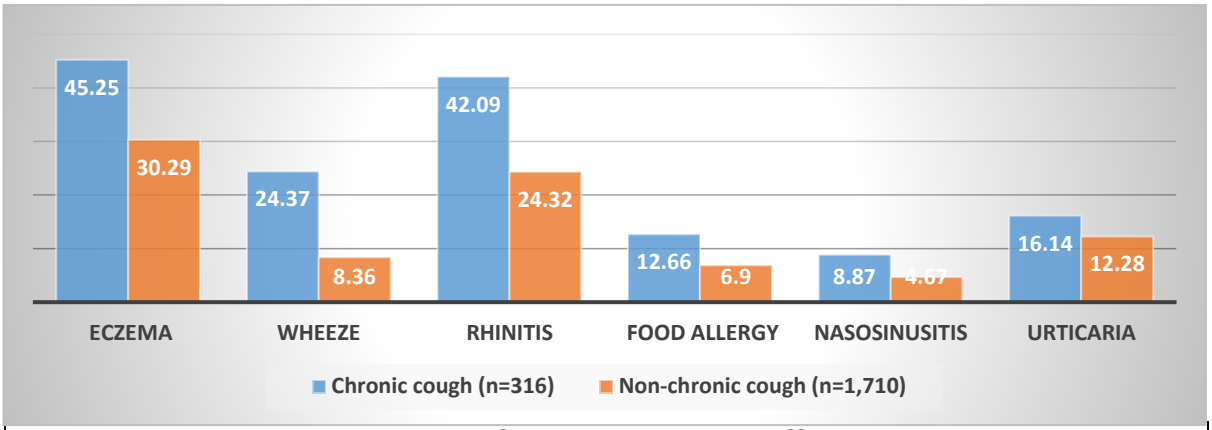

Figure 1. Percentage of participants with different conditions

Discussion

This study highlighted that children with chronic cough were more likely to experience various comorbid conditions and sleep disturbances. These associations align with the notion that chronic cough often accompanies other allergic and respiratory conditions, which are driven by shared underlying mechanisms such as airway inflammation and hyper-responsiveness. A significant aspect of the study is its identification of indoor environmental factors that might contribute to chronic cough and associated sleep disturbances. Specifically, mold exposure, recent home decoration, and carpet use were linked to increased risk of abnormal sleep behaviours in children with chronic cough. The study also reinforces the importance of considering sleep disturbances in the management of chronic cough, as symptoms like snoring, restless sleep, bruxism, and sweating were found to be significantly associated with chronic cough.[2] Additionally, Tiesler et al. found that indoor environmental factors, such as mold and moisture could negatively impact respiratory health and sleep quality, which was consistent with the findings of the present study.[3]

Conclusion

Children with chronic cough were more likely to have abnormal sleep behaviours compared to those without a chronic cough. Factors like recent household decoration, exposure to mold, and carpet use were all linked to these abnormal sleep patterns in affected children.

Children with chronic cough are more prone to some abnormal sleep behaviours.

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Assessment of Zinc, Copper, Magnesium, Calcium and Vitamin D Serum Levels in Children with Idiopathic Drug-Resistant Epilepsy

Introduction

Epilepsy is a prevalent neurological disorder characterized by recurrent, unprovoked seizures. Epilepsy ranks as the third most significant neurological disorder worldwide in terms of disease burden.[1] It affects approximately 51 million people globally, with 3–4% of children experiencing at least one seizure in their lifetime. Despite a variety of causes such as structural, infectious, immunological, metabolic, or genetic disturbances, the etiology often remains unclear. The disorder can lead to neuronal damage and altered neural networks, resulting in long-term effects. Treatment typically involves first-generation or new antiepileptic drugs. However, drug-resistant epilepsy (DRE) occurs in 20% of patients who do not respond to conventional therapies. Mineral and micronutrient deficiencies have been suggested to impact neurological disorders, potentially influencing drug resistance.[2] This study aimed to compare serum levels of zinc (Zn), copper (Cu), magnesium (Mg), calcium (Ca), and vitamin D (Vit D) between well-controlled and DRE patients to assess their role in DRE occurrence.

Methods

A cross-sectional study was conducted on children aged 1–17 years with epilepsy. DRE was defined as having at least one seizure in the past six months despite using two or more antiepileptic drugs for a year. Well-controlled epilepsy was defined as having no seizures during the treatment period. Data on demographics, seizure type, treatment, and dietary history were collected. The primary outcome was comparison of serum levels of different trace elements between the groups. Serum levels of Zn, Cu, Mg, Ca, and Vit D were measured using atomic absorption spectrophotometry and radioimmunoassay.

Results

The study included 64 children of which 33 with DRE and 31 with well-controlled epilepsy. The DRE group had a significantly earlier onset of disease, and a longer treatment period compared to the well-controlled group. Zinc levels were significantly lower in the DRE group ($p=0.007$), but no significant differences were found in serum levels of Vit D, Ca, Cu, or Mg ($p>0.05$) (Figure 1). The frequency of neurodevelopmental delays was higher in the DRE group (75%) compared to the well-controlled group. No significant correlations were observed between treatment duration and serum levels of the trace elements.

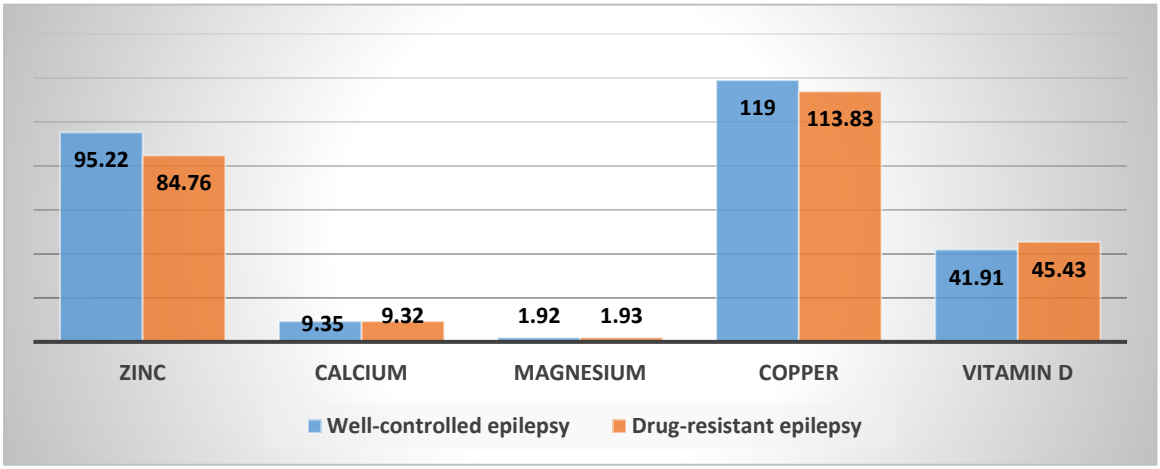

Figure 1. Comparison of serum levels of Zinc, Calcium, Magnesium, copper, and vitamin D between the groups

Discussion

The findings suggest that lower serum zinc levels may contribute to drug-resistant epilepsy, highlighting the potential role of zinc supplementation and monitoring in managing refractory seizures. It was also found that DRE patients showed significantly earlier onset of disease and longer treatment period compared with the well- controlled group. Zinc's modulatory effects on neural excitability, including its role in inhibiting excitatory neurotransmitters and reducing oxidative stress, are consistent with previous research.[2] Prasad et al. reported lower zinc levels in non-responders compared to responders, although differences in other trace elements were not significant.[3] The current study did not find significant differences in serum Cu, Ca, Mg, or Vit D levels between the groups, aligning with some findings but contrasting with others that reported significant differences.[2]

Conclusion

The study concluded that drug-resistant epilepsy patients had an earlier onset of the disease and a higher incidence of neurodevelopmental delays. Lower serum zinc levels were observed in these patients, suggesting that zinc monitoring and supplementation might aid in managing drug-resistant seizures.

Zinc supplementation helps better control of drug-resistant seizures in cases of lower serum levels of Zinc.

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Case Report on Susac Syndrome

Introduction

Susac syndrome is a rare condition marked by inflammation of the small blood vessels specifically in the brain, eyes, and ears.[1] It was first described by Susac et al. in 1979, characterized by a triad of encephalopathy, hearing loss, and branch retinal artery occlusions (BRAO). Due to the variable presentation and overlap with other conditions, Susac syndrome is often underdiagnosed. Encephalopathy typically presents first, with symptoms such as headaches, psychiatric issues, or neurological deficits. Kleffner et al. established diagnostic criteria in 2016 that include cerebral, retinal, and vestibulocochlear manifestations. MRI findings, particularly involving the corpus callosum, are key to diagnosis. The syndrome is rare and even less common in children.[2] This report highlights a case of Susac syndrome in an 11-year-old to underscore its occurrence in pediatric patients and to discuss symptoms, pathophysiology, and diagnostic imaging.

Case Presentation

An 11-year-old male presented with decreased visual acuity in the right eye, severe headaches, and confusion that had developed over the past 3 weeks. Neurological examination revealed

temporal-spatial disorientation, with normal motor and sensory functions. A brain MRI showed multiple nodular signal abnormalities in the central corpus callosum and subcortical white matter above the tentorium (Figure 1). These lesions were hypointense on T1-weighted images, hyperintense on T2-weighted and FLAIR sequences, and exhibited restricted diffusion without contrast enhancement or abnormalities on angiographic sequences. These findings suggested Susac syndrome. The spinal MRI was normal. Fluorescein angiography revealed bilateral retinal artery branch occlusions, and EEG showed diffuse slowing of brain activity. Laboratory tests indicated elevated cerebrospinal fluid protein (170 mg/dL), while systemic vasculitis biomarkers and oligoclonal bands were negative. Audiometry confirmed bilateral sensorineural hearing loss. The patient was diagnosed with Susac syndrome and treated with intravenous immunoglobulins (IVIG) and steroids for 7 days. Follow-up at 1 month showed partial resolution, with complete resolution at 3 months and no new symptoms were found.

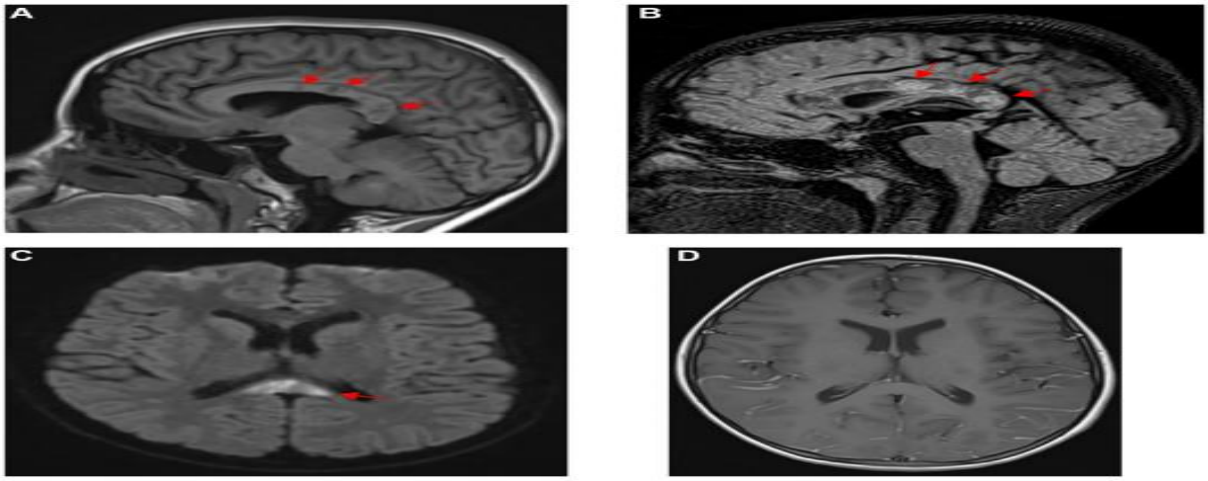

Figure 1. Brain MRI images include sagittal T1 (A), sagittal FLAIR (B), axial diffusion (C), and axial T1 with gadolinium contrast (D). The images reveal small lesions in the corpus callosum (indicated by the red arrow), displaying hypo intensity on T1 (A), hyperintensity on FLAIR (B), restricted diffusion (C), and no contrast enhancement following gadolinium injection (D).

Discussion

Susac syndrome, or SICRET (Small Infarcts of Cochlear, Retinal, and Encephalic Tissues), is a rare microangiopathy likely of dysimmune origin, affecting the brain, retina, and inner ear. This case involves an 11-year-old male who presented with decreased visual acuity, severe headaches, confusion, and temporal-spatial disorientation. MRI revealed characteristic lesions in the corpus callosum, and fluorescein angiography confirmed bilateral retinal artery branch occlusions. Audiometry identified bilateral sensorineural hearing loss, leading to a diagnosis of Susac syndrome.[2] This case aligns with the findings of other studies, which emphasize the importance of MRI in diagnosing Susac syndrome and highlight the challenges of identifying the full clinical triad in younger patients.[3] The patient responded to treatment with intravenous immunoglobulins (IVIG) and steroids, showing partial resolution at one month and complete

resolution at three months. These results were consistent with current treatment guidelines, which recommend early and aggressive immunomodulatory therapy to improve outcomes and prevent severe sequelae.[4]

Conclusion

Susac syndrome is an immune-mediated disease which requires initiating treatment as early as possible to prevent irreversible sequelae. Radiological examinations play an important role in diagnosis.

Magnetic resonance imaging (MRI) is essential for diagnosis and patient monitoring for revealing susac syndrome

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