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Impact of *Helicobacter pylori* Eradication on Serum Valproic Acid Levels in Children with Idiopathic Generalized Epilepsy

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Introduction

Epilepsy, one of the most prevalent neurological disorders among children, is characterized by recurrent, unprovoked seizures stemming from abnormal neuronal activity in the brain. Idiopathic Generalized Epilepsy (IGE) constitutes a major subset of epilepsy in children, often managed effectively with Antiepileptic Drugs (AEDs) such as Valproic Acid (VPA), a broad-spectrum anticonvulsant. VPA has been the cornerstone in treating generalized seizures due to its favorable efficacy profile, yet its therapeutic window is narrow, and serum levels must be tightly regulated to prevent toxicity or loss of seizure control. Meanwhile, Helicobacter pylori (H. pylori), a gram-negative bacterium infecting the gastric mucosa, is frequently acquired in childhood and can lead to chronic gastritis, ulcers, and subtle changes in gastrointestinal function that affect drug absorption. Although primarily linked with gastrointestinal pathology, H. pylori infection has systemic implications, including altered pharmacokinetics of orally administered medications due to modifications in gastric pH, mucosal inflammation, and liver enzyme modulation [1].

Description

Valproic acid is primarily absorbed in the gastrointestinal tract and undergoes hepatic metabolism, with serum concentrations influenced by multiple physiological and pathological factors. H. pylori infection, though often asymptomatic in children, leads to mucosal inflammation and hypochlorhydria, altering gastric pH and potentially impairing the absorption of weakly acidic drugs like VPA. Additionally, chronic infection can impact hepatic enzyme systems via cytokine modulation, which may affect the metabolic clearance of valproic acid. Several clinical observations have suggested that children with poorly controlled epilepsy despite compliance and appropriate dosing may harbor underlying gastrointestinal conditions, including H. pylori infection, that disrupt drug pharmacokinetics. Upon eradication therapy typically involving a combination of antibiotics and Proton Pump Inhibitors (PPIs) gastrointestinal function may normalize, potentially enhancing the absorption and bioavailability of VPA. However, PPIs used during eradication can transiently elevate gastric pH, possibly delaying absorption or causing fluctuations in drug levels. Thus, the impact of H. pylori eradication on valproic acid levels is complex, involving a dynamic interplay between microbial eradication, mucosal healing, pH normalization, and hepatic metabolism [2].

This paper aims to explore the emerging relationship between H. pylori infection, its eradication, and subsequent effects on serum valproic acid levels in children with idiopathic generalized epilepsy, evaluating how gastrointestinal microbial dynamics can influence neurological pharmacotherapy and long-term seizure control. The recognition that H. pylori, a bacterium primarily associated with gastrointestinal disease, can indirectly affect neurological outcomes through pharmacokinetic alterations emphasizes the need for multidisciplinary approaches to epilepsy management, combining gastroenterology, neurology, and pharmacology. As VPA absorption is pH-dependent and relies on normal gastrointestinal physiology, the presence or eradication of H. pylori could significantly influence serum drug concentrations. Additionally, more longitudinal studies are needed to confirm long-term outcomes, assess the safety of repeated eradication therapy, and explore whether similar dynamics apply to other AEDs. Ultimately, by integrating microbial management into epilepsy treatment strategies, clinicians can move closer to precision medicine approaches that account for the full spectrum of individual variability biological, microbial, and environmental in optimizing neurological health and therapeutic efficacy [3].

Moving forward, the incorporation of routine screening for H. pylori in children with unexplained fluctuations in antiepileptic drug levels or persistent seizures despite adherence may become an important adjunct in epilepsy care. Clinical studies have shown that post-eradication, children previously infected with H. pylori exhibit more stable or elevated serum VPA levels, leading to improved seizure control and reduced breakthrough episodes. These findings underscore the importance of considering gastrointestinal infections as a potential confounder in antiepileptic drug management. In pediatric patients, the developing gut microbiome and immature immune system may exacerbate these interactions, making routine screening and management of H. pylori more clinically relevant in refractory epilepsy cases. Furthermore, variability in the CYP450 enzyme system, gut permeability, and enterohepatic circulation in children adds another layer of complexity. It is essential to monitor serum VPA levels before and after eradication therapy, not only to adjust dosing but also to anticipate possible side effects or toxicities due to increased bioavailability [4].

This connection between gut health and central nervous system pharmacology reflects a broader paradigm shift in understanding how the micro biome and infectious agents influence systemic drug efficacy. Recent studies have raised questions about the role of subclinical infections like H. pylori in modulating the bioavailability of critical medications such as VPA, especially in pediatric populations with epilepsy who require stable therapeutic levels. While anti-seizure medications are effective in controlling seizures for the majority of patients, about one-third remain drug-resistant, requiring alternative interventions such as epilepsy surgery, neuromodulation therapies (e.g., VNS or RNS), or dietary treatments like the ketogenic diet. he causes of epilepsy are diverse and span genetic mutations, structural brain abnormalities, infections, autoimmune processes, metabolic disturbances, and unknown origins. Diagnosis typically involves clinical assessment supported by Electroencephalography (EEG), neuroimaging (such as MRI), and sometimes genetic or metabolic testing.Advances in neuroscience and technology particularly in brain imaging, computational modeling, and deep learning are continuously reshaping our understanding of epilepsy, with emerging approaches aiming not only to detect and predict seizures but also to uncover the underlying network dynamics and causal brain mechanisms that drive epileptogenesis [5].

Conclusion

The eradication of Helicobacter pylori in children with idiopathic generalized epilepsy has a significant, though often underappreciated, impact on the pharmacokinetics of valproic acid, one of the most commonly used antiepileptic medications. As this review illustrates, the presence of H. pylori can subtly disrupt the gastrointestinal environment, impair drug absorption, and alter systemic metabolism, thereby affecting therapeutic drug levels and potentially compromising seizure control. Successful eradication restores

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gastric physiology, leading to improved absorption and more consistent serum concentrations of valproic acid, which in turn enhances the clinical response and seizure management in affected children. These findings highlight the intricate and often overlooked relationship between gut microbiota, infection, and central nervous system pharmacotherapy. They underscore the necessity for clinicians to adopt a holistic view of epilepsy treatment considering not just neurological parameters but also gastrointestinal and microbial factors that may modulate drug response.

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Conflict of Interest

There are no conflicts of interest by author.

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