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PERSPECTIVES

Rethinking Corticosteroid Therapy in Pediatric Neurology

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Abstract: Corticosteroids have long been a cornerstone in the management of many pediatric neurological disorders. However, their broad immunomodulatory effects raise concerns in the era of precision medicine, where more targeted approaches are increasingly favored, warranting a critical reappraisal of their roles in pediatric neurology. Of particular importance is the possibility that corticosteroids may potentiate components of the innate immune responses and thereby exacerbate neuroinflammation. Accordingly, disease-specific immunopathogenesis should be considered in therapeutic decision-making. The potential for adverse effects, especially with long-term use, remains a major consideration and underscores the need for careful risk–benefit analysis. Several therapeutic innovations, including selective glucocorticoid receptor modulators (such as vamorolone) and intra-erythrocyte dexamethasone delivery, have shown promising safety profiles in Duchenne muscular dystrophy and ataxia telangiectasia, respectively. Beyond the well-established role of corticosteroids in infantile epileptic spasms syndrome, recent evidence suggests that corticosteroids may benefit a substantial proportion of patients with various forms of epilepsy that are resistant to conventional antiseizure medications. Further research is warranted to define the optimal use of corticosteroids with respect to dosing, formulation, timing, and route of administration in various pediatric neurological disorders.

Keywords: corticosteroids, glucocorticoid receptor modulator, glucocorticoid toxicity, intra-erythrocyte drug delivery, neuroinflammation, vamorolone

Text

Corticosteroids have been a cornerstone in the management of many neuroimmunological disorders. Their broad immunomodulatory properties have justified their widespread use across a range of autoimmune and neuroinflammatory conditions. However, in the era of precision medicine, such relatively nonspecific effects on immune pathways, as well as pleiotropic effects on broader physiological systems, may limit their appeal compared to more targeted therapies. This makes the present moment suitable for a critical reappraisal of the therapeutic role of corticosteroids, as exemplified by a recent opinion piece authored by Chiek Teoh et al.[1] Several important issues are discussed in their paper, including disease-specific immunopathogenesis and safety concerns. In my view, additional aspects merit further discussion, especially in the context of pediatric neurology. Corticosteroids have been used across a broad spectrum of pediatric neurological disorders (Table 1); in some conditions, their efficacy is well established, whereas in others their use remains anecdotal or exploratory. Importantly, potential adverse effects unique to the pediatric population, most notably growth suppression, should be carefully weighed when considering corticosteroid therapy.

Corticosteroids: A Double-Edged Sword

Although corticosteroids are classically regarded as anti-inflammatory and immunosuppressive, they can also potentiate certain elements of innate immunity in the CNS in some contexts. This may not be entirely unexpected, as endogenous corticosteroids have likely evolved to prepare the body, including the immune system, adapt to periods of acute stress. Indeed, endogenous or exogenous corticosteroids can prime microglia for enhanced pro-inflammatory responses upon

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Table 1 Corticosteroid Use in Pediatric Neurological Disorders

Established indications
Epileptic spasms
Acute disseminated encephalomyelitis (ADEM)
Autoimmune encephalitis
Multiple sclerosis
Neuromyelitis optica spectrum disorder
Myasthenic gravis
Chronic inflammatory demyelinating polyneuropathy
Bell's palsy
Bacterial and tuberculous meningitis (as adjunctive therapy)[a]
Emerging or anecdotal use
Epilepsies other than epileptic spasms (DEE/EE-SWAS, LGS, etc).
Infection-triggered encephalopathy syndromes and related conditions[b]
Immune effector cell-associated neurotoxicity syndrome (ICANS)

Notes: [a]excluding neonatal meningitis. [b]Corticosteroids may not be indicated in milder forms of infection-triggered encephalopathy syndromes, such as mild encephalopathy with a reversible splenial lesion.
Abbreviations: DEE/EE-SWAS, developmental and/or epileptic encephalopathy with spike-wave activation in sleep; LGS, Lennox-Gastaut syndrome.

subsequent immune challenges. This microglial priming has been demonstrated in rodent models exposed to acute stress or chronic corticosterone treatment, with potentiation evidenced by elevated cytokine responses and inflammasome activation.[2,3] Notably, blocking glucocorticoid receptor (GR) signaling prevents this priming, supporting the permissive role of corticosteroids in neuroinflammatory sensitization. Given that innate immune processes, such as microglial activation and inflammasome signaling, are implicated in a range of neurological disorders, including multiple sclerosis, N-methyl-D-aspartate (NMDA) receptor encephalitis, Rasmussen encephalitis, and febrile infection-related epilepsy syndrome (FIRES),[4] it is essential to re-examine the neuroimmunological effects of corticosteroid therapy in these conditions, particularly with respect to treatment timing and dosage.

Although the context-dependent pro-inflammatory actions of corticosteroids are primarily observed in innate immune processes,[5,6] this does not imply that corticosteroids are universally beneficial in autoimmune (ie, adaptive immune-mediated) neurological disorders. A classic example is Guillain-Barré syndrome (GBS), which responds poorly to corticosteroid therapy. Animal studies suggested that corticosteroids may interfere with macrophage-mediated clearance of myelin and axonal debris, thereby impairing remyelination and delaying recovery.[7] How this mechanism reconciles with the effectiveness of corticosteroids in chronic inflammatory demyelinating polyneuropathy (CIDP), the chronic counterpart of GBS, remains unresolved. Other causes of corticosteroid resistance, such as GRα phosphorylation, increased GRβ expression, or reduced histone deacetylase (HDAC) 2 expression, may also worth investigation in this context.[8] Regardless, these contrasting clinical experiences highlight the importance of tailoring immunotherapy to disease-specific immunopathogenesis.

Enhancing Safety Through Therapeutic Innovations and Steroid Stewardship

Corticosteroid-associated adverse effects are an important consideration when selecting these agents for therapeutic use in children, even when administered for short courses.[9] Particular concerns include their potential impact on linear growth and the developing brain,[10,11] both of which warrant careful monitoring in the pediatric population. Selective glucocorticoid receptor modulators (SEGRMs) have been proposed as a means of reducing adverse effects through more targeted modulation of GR signaling.[12] To date, however, their application remains largely experimental. An encouraging advance in this direction is vamorolone, a dissociative steroid that preserves GR-mediated transrepression while reducing GR-mediated transactivation. Compared with conventional corticosteroids, vamorolone is associated with a more favorable safety profile and has recently been granted regulatory approval for the treatment of Duchenne muscular dystrophy.[13,14] An alternative strategy for enhancing the safety of corticosteroid therapy is intra-erythrocyte delivery.

Table 2 Intrathecal Corticosteroids for Neuroimmunological Disorders

Neuroimmunological Disorder	Corticosteroid Agent	PMID[a]
Autoimmune encephalitis	Methylprednisolone[b]	25040285 (NMDA receptor encephalitis)[30]
	dexamethasone	39128431 (anti-GAD65 encephalitis)[31]
FIRES	Dexamethasone	33547757[21]
HLH	Hydrocortisone[b]	21828139[32]
	prednisolone[b]	16937360[33]
ICANS	Hydrocortisone	37897136,[34] 32407473[35]
	dexamethasone	39321707[36]
Multiple sclerosis	Triamcinolone	22096630[37]
Neuropsychiatric systemic lupus erythematosus	Dexamethasone[b]	40401342[38]

Notes: [a] The references cited here are not comprehensive and serve only as representative examples. [b] along with methotrexate.
Abbreviations: FIRES, febrile infection-related epilepsy syndrome; GAD65, glutamic acid decarboxylase 65-kilodalton isoform; HLH, hemophagocytic lymphohistiocytosis; ICANS, immune-effector cell-associated neurotoxicity syndrome; NMDA, N-methyl-D-aspartate.

This technology encapsulates corticosteroids, specifically dexamethasone sodium phosphate, inside autologous red blood cells, which are then infused back into patients. The novel mode of delivery could be conceived as a sustained-release or long-acting formulation of corticosteroids.[15] It has entered Phase 3 clinical trials for pediatric patients with ataxia telangiectasia, demonstrating a better safety profile compared with oral corticosteroids.[16,17]

When systemic corticosteroids are used to treat central nervous system disorders, their degree of CNS penetration should be considered. For example, dexamethasone may exhibit greater penetration (as reflected by a higher CSF to plasma ratio) and a longer half-life in the CSF compared with prednisolone.[18] Alternative routes of drug delivery, such as transnasal brain-targeted strategies, merit exploration as potential methods to bypass systemic limitations.[19] Intrathecal corticosteroids have gained increasing attention in recent years for the treatment of various neuroimmunological disorders (Table 2), including those with predominant innate immune involvement (eg, FIRES) and adaptive immune involvement (eg, NMDA receptor encephalitis).[20,21] However, the pharmacokinetics and pharmacodynamics of various intrathecal corticosteroid regimens remain insufficiently characterized and require further investigation.[22] Intrathecal corticosteroid administration might limit systemic exposure and thereby attenuate some adverse effects; nonetheless, systemic effects may still occur and should be carefully monitored.[23] It is essential to evaluate corticosteroid formulations for their suitability for intrathecal administration, as certain excipients and additives may be neurotoxic.[24] Intrathecal administration of methylprednisolone has been associated with meningeal thickening and adhesions in canine models; however, the formulation used was not preservative-free, potentially confounding the results.[25] Notwithstanding these concerns, current data from both rodent models and human studies suggest that intrathecal delivery of corticosteroids is generally safe.[26,27] Regardless of the route of administration, it is advisable to systematically monitor potential adverse effects in clinical practice, for example, through the use of standardized tools such as the Glucocorticoid Toxicity Index (GTI),[28] which is now being increasingly used in clinical trials involving corticosteroids. A pediatric version of the GTI is also available.[29]

The Renaissance of Corticosteroids in Pediatric Epileptology

Although corticosteroids are decades-old agents, their applications in pediatric neurology continue to expand, including in epileptology. Beyond their well-established role in infantile epileptic spasms syndrome, recent empirical evidence suggests that corticosteroids may benefit a substantial proportion of patients with various forms of epilepsy that are resistant to conventional antiseizure medications.[39–42] However, these studies were often retrospective in nature and lacked appropriate control groups. Encouragingly, several prospective studies and randomized controlled trials have demonstrated favorable efficacy and acceptable safety profiles of corticosteroids in Lennox-Gastaut syndrome,[43] developmental and/or epileptic encephalopathy with spike-wave activation in sleep (DEE/EE-SWAS),[44] and other forms of pediatric epileptic encephalopathy.[45] These findings align with the growing recognition regarding the roles of inflammation and blood-brain barrier alterations in epilepsy, processes that may be modulated by corticosteroid therapy.[46] Existing

studies, however, have employed heterogeneous treatment protocols with respect to the choice of corticosteroid, as well as dosing and treatment duration.[47] Further rigorous research is needed to more clearly define the optimal regimen of corticosteroids in the management of refractory epilepsy across diverse etiologies.

Concluding Remarks

The complex immunomodulatory effects of corticosteroids are increasingly being elucidated, encompassing both anti-inflammatory and pro-inflammatory actions that are highly context dependent. These mechanistic insights are clinically relevant, as an improved understanding of disease-specific immunopathogenesis may inform more precise and judicious use of corticosteroids and help explain variability in treatment responses.

With continued advances in therapeutic modalities and an increasingly refined understanding of disease pathophysiology, the clinical applications of corticosteroids continue to evolve and expand. Therefore, corticosteroids are likely to remain a cornerstone of therapy for a range of pediatric neurological disorders. The pleiotropic effects of corticosteroids, particularly their potential impact on the developing brain and skeletal maturation in children and adolescents, must be carefully weighed upon prescription. It should also be borne in mind that their effects are highly context-dependent and vary according to dose, timing, and disease stage.[5] Continued research is needed to elucidate disease-specific immunopathogenesis and to define the optimal use of corticosteroids and related agents in pediatric neurological practice.

Data Sharing Statement

Data sharing is not applicable to this article as no new data were created in this study.

Author Contributions

Wei-Sheng Lin: Conceptualization, investigation, writing – original draft, writing – review & editing. The author gave final approval of the version to be published; has agreed on the journal to which the article has been submitted; and agrees to be accountable for all aspects of the work.

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