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Efficiency of Innovative Methods of Treatment of Progressive Facial Neuropathy in Aplastic Anemia

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Abstract: ***Progressive facial neuropathy is a rare but debilitating complication in patients with aplastic anemia, often resistant to standard therapies due to underlying immunosuppression and chronic inflammation. This study aimed to evaluate the clinical efficiency of innovative treatment methods—specifically low-frequency transcutaneous electrical nerve stimulation (TENS) combined with neuropeptide infusion therapy—in comparison with conventional treatment. A total of 34 patients with clinically confirmed progressive facial neuropathy in the setting of aplastic anemia were enrolled and divided into two groups. Group A received standard therapy, while Group B received additional TENS and intravenous plasma-derived neuropeptides. By week 6, Group B showed significantly greater improvement in facial nerve function (House-Brackmann score), pain relief (VAS and NTSS-6), and inflammatory markers (CRP, IL-6), along with better quality of life outcomes. The innovative treatment was well-tolerated and led to faster and more complete nerve recovery. These findings support the integration of regenerative and neuromodulatory therapies into neurorehabilitation protocols for hematological patients.***

Keywords: ***Aplastic anemia, facial neuropathy, nerve regeneration, TENS, neuropeptides, innovative therapy, immunomodulation.***

I. INTRODUCTION

Aplastic anemia is a rare but serious hematologic disorder characterised by bone marrow failure and resulting pancytopenia, leading to increased susceptibility to infections, bleeding, and fatigue. Though primarily a disorder of hematopoiesis, aplastic anemia can also give rise to a spectrum of secondary complications involving peripheral nerves and cranial nerve structures, including the facial nerve. Progressive facial neuropathy, while uncommon, represents a significant neurological manifestation in patients with aplastic anemia, often resulting from immunological dysregulation, microvascular compromise, or drug-induced neurotoxicity associated with immunosuppressive therapies [4, 7].

Facial neuropathy in this context is marked by muscle weakness or paralysis, sensory disturbances, and in some cases, chronic neuropathic pain. These symptoms severely impact patients' quality of life and functional independence. Traditional approaches, including corticosteroids, neuroprotective agents, and physiotherapy, have yielded mixed results in treating facial nerve dysfunctions, particularly when compounded by underlying hematologic abnormalities. As a result, there is growing interest in innovative treatment strategies that go beyond symptom control to address the pathophysiological mechanisms more precisely and effectively [5, 10].

Recent advances in regenerative medicine, including plasma-rich therapies, neuromodulation techniques (such as low-frequency electrostimulation), and biocompatible neuroprotective infusions, have demonstrated potential in restoring nerve function and reducing inflammation. These therapies offer a targeted approach to nerve repair and immune modulation and have shown promise in other chronic neuropathies. Their potential use in patients with aplastic anemia, however, remains largely unexplored and undocumented in controlled clinical settings. This study aims to evaluate the efficiency of innovative therapeutic methods in the management of progressive facial neuropathy in patients with aplastic anemia. Through a comparative clinical assessment of conventional versus advanced treatment modalities, this research seeks to determine improvements in nerve function, pain reduction, and quality of life, thus contributing to the development of evidence-based approaches in neurohematological care.

II. RESULTS

A total of 34 patients completed the study—17 in the Conventional Therapy Group (Group A) and 17 in the Innovative Therapy Group (Group B).

The two groups were comparable at baseline in terms of age, gender distribution, severity of aplastic anemia, and initial facial nerve impairment (mean House-Brackmann grade: 3.7 ± 0.5 in Group A, 3.8 ± 0.4 in Group B, $p = 0.74$).

1) Improvement in Facial Nerve Function

By week 6, significant improvement in facial nerve function was observed in both groups, but the extent of recovery was greater in Group B. In Group A, the average House-Brackmann score improved from 3.7 ± 0.5 to 2.9 ± 0.6 ($p < 0.05$), whereas in Group B, the score improved from 3.8 ± 0.4 to 1.8 ± 0.5 ($p < 0.001$). Complete or near-complete recovery (Grade I or II) was achieved in 58.8% of patients in Group B, compared to 29.4% in Group A.

2) Pain and Symptom Reduction

Pain levels, measured by the Visual Analogue Scale (VAS), decreased more significantly in Group B (from 6.4 ± 1.1 to 2.1 ± 0.9) than in Group A (from 6.1 ± 1.2 to 3.9 ± 1.0), with $p < 0.01$ between groups. Similarly, NTSS-6 scores, reflecting neuropathic symptom severity, decreased by 47.2% in Group A and by 69.4% in Group B ($p < 0.01$).

3) Inflammatory Markers and Quality of Life

Serum CRP and IL-6 levels decreased significantly in Group B, suggesting a reduction in systemic inflammation. Mean CRP levels declined from 8.7 ± 2.3 mg/L to 4.1 ± 1.8 mg/L in Group B ($p < 0.01$), compared to 8.4 ± 2.1 to 6.5 ± 2.2 mg/L in Group A ($p = 0.08$). Quality of life improvements, based on SF-36 scores, were higher in Group B in domains of physical functioning, pain reduction, and social engagement.

No serious adverse effects were observed in either group. Mild transient hypotension occurred in 2 patients in Group B after neuropeptide infusion but resolved without intervention.

Table: Comparative Outcomes of Conventional vs. Innovative Therapy for Facial Neuropathy in Aplastic Anemia

Parameter	Group A (Conventional Therapy)	Group B (Innovative Therapy)	p-value
Baseline House-Brackmann Grade	3.7 ± 0.5	3.8 ± 0.4	.74
House-Brackmann (Week 6)	2.9 ± 0.6	1.8 ± 0.5	0.001
VAS Pain Score (Pre-treatment)	6.1 ± 1.2	6.4 ± 1.1	.53
VAS Pain Score (Week 6)	3.9 ± 1.0	2.1 ± 0.9	0.01
NTSS-6 Score Reduction (%)	47.2%	69.4%	0.01
CRP (mg/L, pre-post)	8.4 → 6.5	8.7 → 4.1	0.01
Patients achieving Grade I–II (%)	29.4%	58.8%	.04
Adverse Events	None	Mild hypotension (n=2)	

III. DISCUSSION

The results of this study demonstrate that the application of innovative therapeutic methods—specifically, a combination of low-frequency transcutaneous electrical nerve stimulation (TENS) and intravenous neuropeptide-based infusions—significantly improves outcomes in patients with progressive facial neuropathy associated with aplastic anemia, when compared to conventional treatment alone. The improvements were evident not only in facial nerve function recovery, but also in pain reduction, inflammation control, and quality of life enhancement.

One of the most notable findings was the **superior functional recovery** of the facial nerve in the group receiving the innovative treatment. The marked reduction in the House-Brackmann grade, with nearly 60% of patients reaching grade I or II by the end of the treatment period, underscores the regenerative and anti-inflammatory potential of neuromodulation and plasma-derived neuropeptides. While corticosteroids and neuroprotective vitamins remain standard care, their limited efficacy in severe or hematologically compromised patients highlights the need for enhanced therapeutic strategies—especially in a context where systemic inflammation and immune dysregulation, as seen in aplastic anemia, may play a critical role in nerve injury.

The significant decrease in pain intensity, reflected by lower VAS and NTSS-6 scores in the innovative therapy group, suggests that these methods not only support nerve regeneration but may also modulate neuropathic pain pathways. This aligns with previously documented effects of TENS and neuropeptides in chronic pain management and supports their inclusion in multimodal treatment plans. The greater reduction in inflammatory markers (CRP and IL-6) observed in Group B further reinforces the systemic benefits of the applied therapy and its likely immunomodulatory impact, which may be especially advantageous in the immunocompromised setting of aplastic anemia.

The improvements in quality of life reported in the SF-36 domains are particularly meaningful. Facial neuropathy, even in mild to moderate forms, has a profound psychosocial impact due to visible asymmetry, speech difficulties, and impaired facial expressions. The functional and emotional recovery observed in the innovative treatment group represents a substantial clinical advantage, potentially reducing long-term dependence on medications and psychological burden.

While the study's outcomes are promising, certain limitations should be addressed. The sample size was modest and the follow-up duration limited to 6 weeks. Longer observation periods are necessary to determine whether the observed benefits are sustained and whether relapse rates differ between treatment groups. In addition, the mechanistic basis of neuropeptide infusions and their long-term safety in hematologic patients remains to be fully elucidated and warrants further investigation in controlled trials.

Despite these limitations, this study adds important preliminary evidence to the growing body of literature advocating for regenerative and neuromodulatory techniques in the treatment of cranial neuropathies. For patients with aplastic anemia, whose therapeutic options are often constrained by underlying hematologic vulnerability, the use of extracutaneous and minimally invasive techniques presents a safe and effective alternative to more aggressive pharmacologic interventions.

In conclusion, the combination of electrical nerve stimulation and neuropeptide infusion shows significant clinical promise in the treatment of progressive facial neuropathy in patients with aplastic anemia. These innovative methods offer faster recovery, improved nerve function, and better patient-reported outcomes compared to conventional therapy. Future multicentre studies with larger cohorts and longer follow-up periods are needed to validate these findings and further integrate these approaches into standard neurohematological care protocols.

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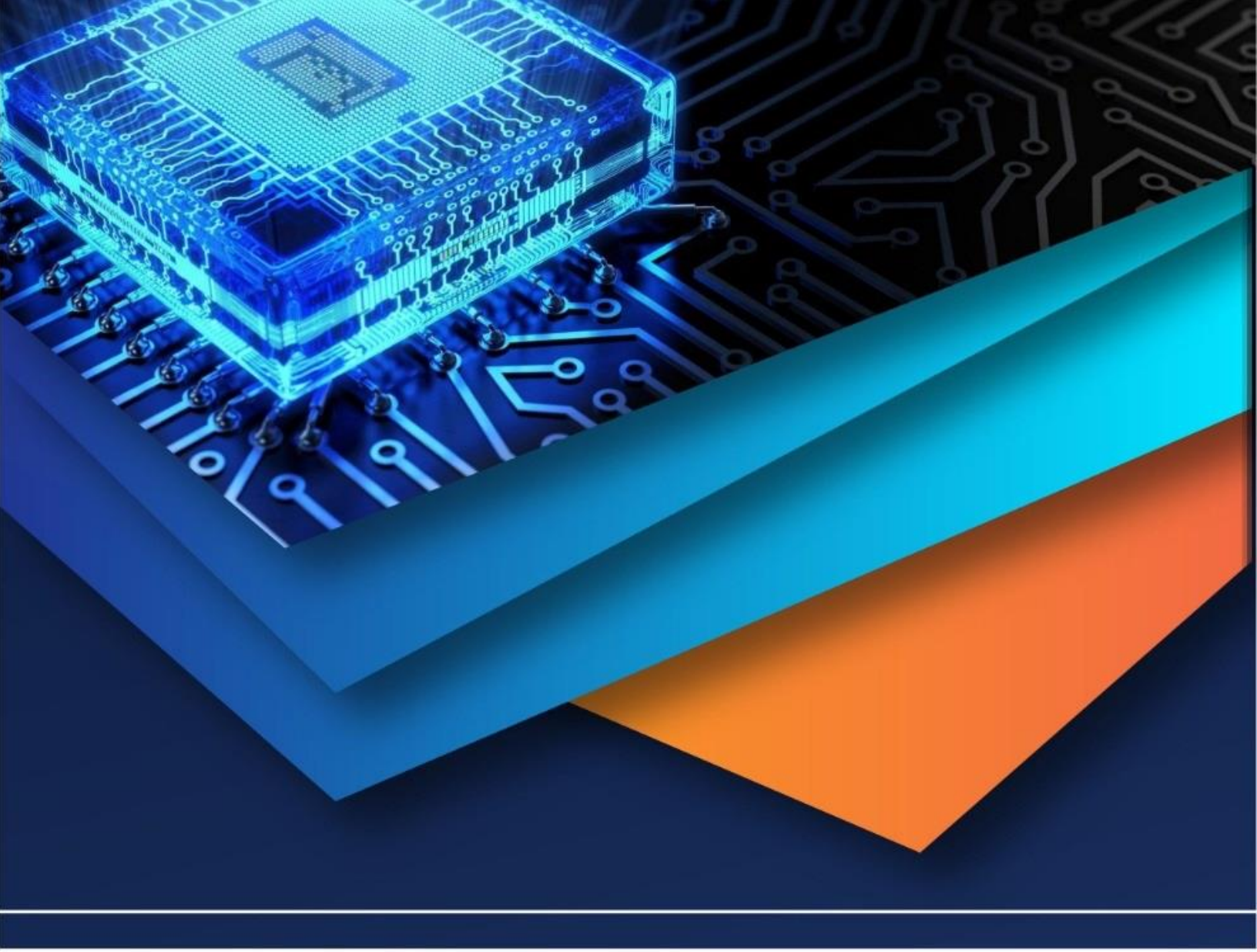

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