Neuroprotective Enhancement via Peripheral Nerve Microcurrent Stimulation in a Rat Stroke Model

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Objective

To evaluate the neuroprotective impact of peripheral nerve microcurrent stimulation in a rat model of middle cerebral artery occlusion (MCAO).

Materials and Methods

This study involved twenty male Sprague-Dawley rats, divided into four groups (Figure 1): Group-A (Control), which remained untreated; Group-B (Disease), which underwent MCAO without intervention; Group-C (Treatment Post-MCAO), which received microcurrent stimulation post-MCAO for one week; and Group-D (Prevention and Recovery), treated with microcurrent stimulation one week before and after MCAO. Assessments included morphological inspections, motion behavior analytics, histological and immunohistochemical evaluations, and Western blot analysis for protein expression.

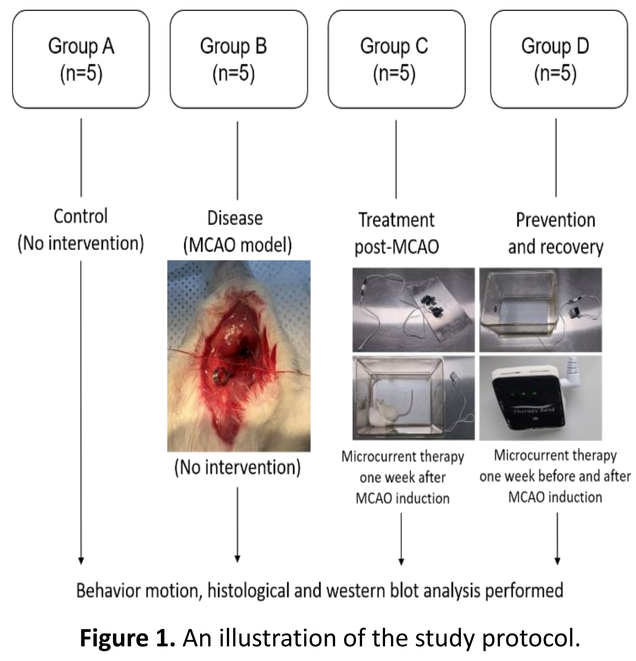

Figure 1. An illustration of the study protocol.

Results

Microcurrent therapy effectively minimized ischemic damage (Figure 2,3). and maintained the structural integrity of CA1 pyramidal neurons in the hippocampus (Figure 4Histological evaluations using H&E staining indicated no infarction in Group-A with pyramidal cell counts at 261.78±3.82. In contrast, infarction percentages and cell counts were 28.55%±1.05%/94.51±6.35 in Group-B, 17.35%±0.83%/184.13±4.66 in Group-C, and significantly improved to 5.40%±0.50%/237.80±3.65 in Group-D (Figure 4, $p < 0.05$,). Movement assessments showed total traveled distances of 1945.24 cm in Group-A, reducing to 767.85 cm in Group-B, and improving in Group-C and Group-D to 1781.77 cm and 2122.22 cm respectively (*$p < 0.05$). Average movement speeds were also recorded: 6.48 cm/s in Group-A, decreased to 2.50 cm/s in Group-B, and increased to 5.43 cm/s in Group-C and 6.82 cm/s in Group-D ($p < 0.05$). Levels of inflammatory markers such as CD68, IL-6, and TNF-α showed significant reduction in treatment groups (Figure 5,6,7, $p < 0.01$,). Protein expression analysis via Western blot demonstrated a decrease in proteins associated with inflammation, oxidative stress, and apoptosis, and an upregulation of angiogenesis markers and modulation of the MAPK signaling pathway in groups undergoing therapy.(Figure 8,9).

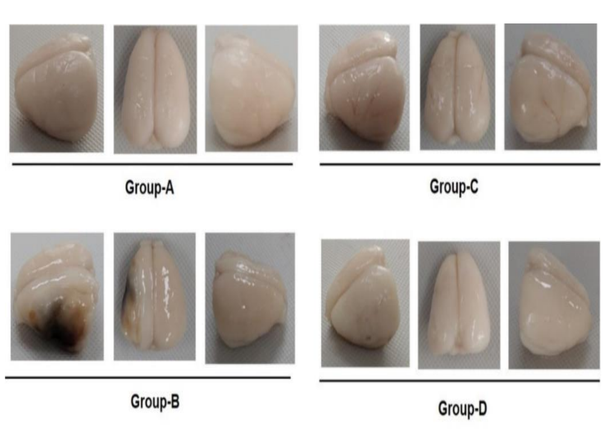

Figure 2. A morphological comparison of brain tissue among experimental groups A–D..

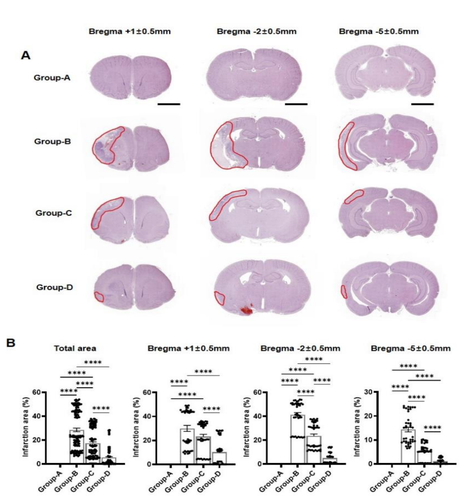

Figure 3. Histological analysis and infarction area quantification n groups A–D.

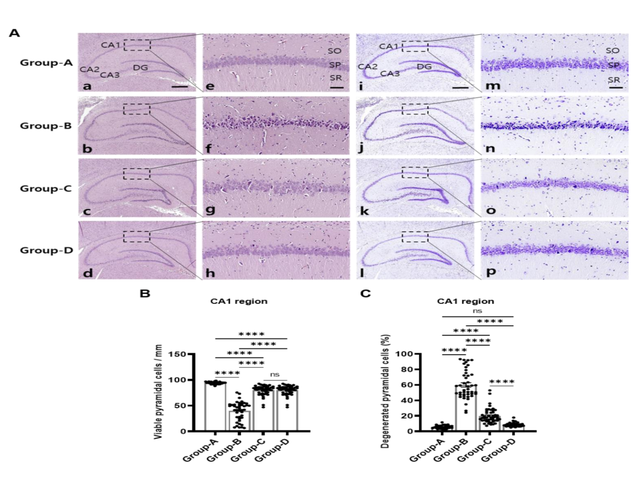

Figure 4. A histological examination and quantitative analysis of the hippocampal regions in groups A–D

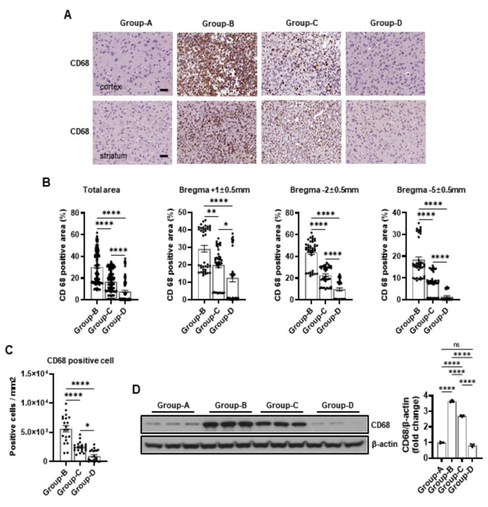

Figure 5. Immunohistochemical and Western blot analyses of CD68 expression in groups A–D.

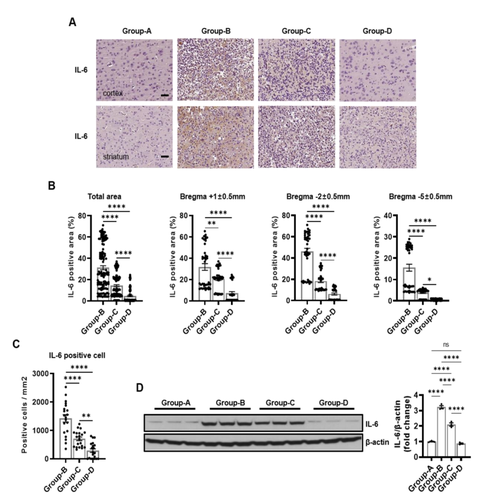

Figure 6. Immunohistochemical and Western blot analyses of IL-6 expression in groups A–D.

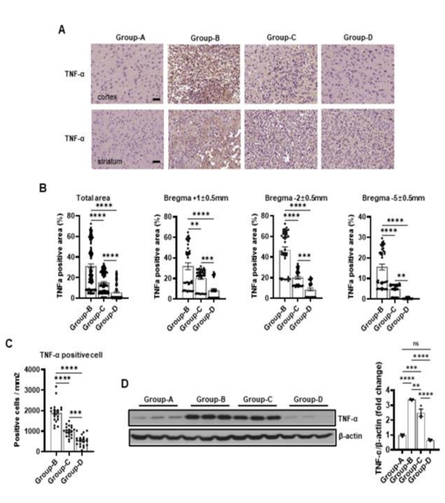

Figure 7. Immunohistochemical and Western blot analyses of TNF-α expression in groups A–D.

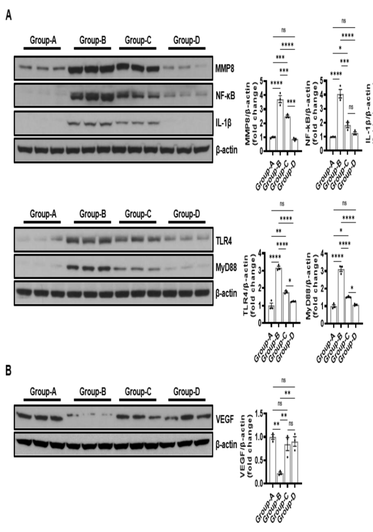

Figure 8. The Western blot results of immune-related proteins (**A**) and angiogenic factors (**B**). (**A**) MMP8, NF-kB, IL-1β, TLR4, and MyD88 in groups A-D (**B**) VEGF.

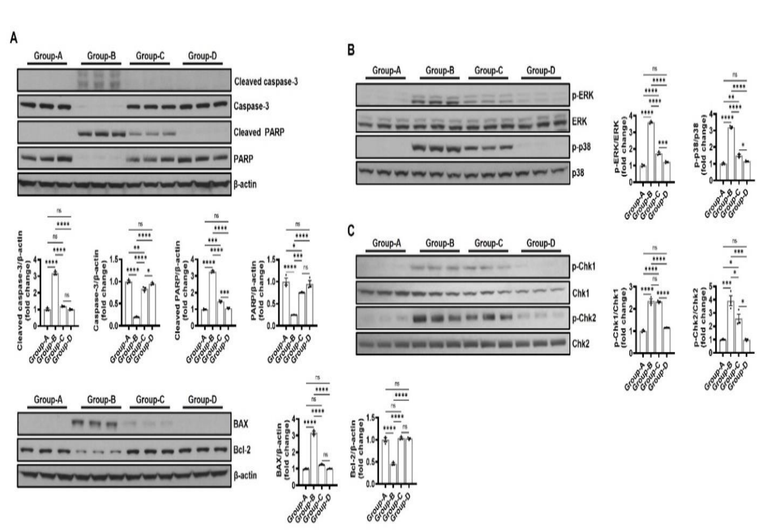

Figure 9. The Western blot results of (**A**) apoptosis-related proteins (caspase-3, cleaved-PARP, BAX, and Bcl-2), (**B**) MAPK proteins (ERK and p38), and (**C**) DNA-damage-related proteins (Chk1 and Chk2 phosphorylation).

Conclusions

Peripheral nerve microcurrent stimulation substantially reduces ischemic damage, lowers inflammation, and improves neurological function, offering promising therapeutic potential for stroke treatment.