Cerebroprotective Mechanism of Qizhi Formula in Ameliorating Cerebral Ischemia-reperfusion Injury

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Abstract [**Objectives**] To investigate the cerebroprotective mechanisms of Qizhi Formula against cerebral ischemia-reperfusion injury (CIRI) through *in vitro* cellular experiments. [**Methods**] The injury model of BV2 microglia induced by oxygen-glucose deprivation/reoxygenation (OGD/R) was established. The reliability of the model was validated through cell counting kit-8 (CCK-8) and lactate dehydrogenase (LDH) release assays. Successfully modeled BV2 cells were subsequently allocated into six groups: blank, model, nimodipine, Astragali Radix, Hirudo, and Qizhi Formula groups, with corresponding drug-containing sera prepared for each. Enzyme-linked immunosorbent assay (ELISA) was employed to quantify the levels of pro-inflammatory cytokines (TNF-α, IL-1β) and anti-inflammatory cytokines (IL-10, TGF-β) in the cell culture supernatants. Real-time quantitative polymerase chain reaction (RT-qPCR) was conducted to assess the mRNA expression of M1 microglial markers (*CD86*, *iNOS*) and M2 microglial markers (*CD206*, *Arg1*) across the different groups. [**Results**] ELISA results showed that, compared to the blank control group, the model group exhibited a significant increase in pro-inflammatory factor levels alongside a decrease in anti-inflammatory factors. In comparison to the model group, treatment with drug-containing serum from all drug intervention groups significantly downregulated the expression of TNF-α and IL-1β, while upregulating IL-10 and TGF-β levels in OGD/R-injured BV2 cells ($P<0.01$). Furthermore, Qizhi Formula produced superior effects relative to single treatments with Astragali Radix or Hirudo ($P<0.05$). RT-qPCR analysis revealed that OGD/R stimulation significantly increased the mRNA expression of M1 markers *CD86* and *iNOS* in BV2 cells. Treatment with nimodipine and Qizhi Formula markedly inhibited the upregulation of these M1 markers ($P<0.01$) while concurrently enhancing the mRNA expression of M2 markers *CD206* and *Arg1* ($P<0.01$). [**Conclusions**] Qizhi Formula provides cerebroprotection against CIRI by regulating the M1/M2 polarization balance of BV2 microglia and attenuating excessive neuroinflammatory responses, thereby offering pharmacological evidence supporting the use of this formula.

Key words Cerebral ischemia-reperfusion injury (CIRI), Qizhi Formula, Microglial polarization, Wnt/β-catenin signaling pathway, Neuroinflammation

1 Introduction

Ischemic stroke mainly affects middle-aged and elderly individuals, with its high mortality and disabling effects being especially pronounced. As the population ages, the incidence of ischemic stroke continues to rise, accompanied by a notable increase in cases among younger individuals. The condition is characterized by an acute onset, and standard clinical interventions primarily consist of thrombolysis and surgical thrombectomy. However, patients frequently experience cerebral ischemia-reperfusion injury (CIRI) during the restoration of transient blood perfusion. This injury can result in substantial neuronal damage and may lead to "ineffective reperfusion"[1]. CIRI involves complex pathophysiological mechanisms, with microglia-mediated neuroinflammatory responses playing a central role[2-3]. Microglia, the resident immune cells of the central nervous system, become activated in response to ischemic and hypoxic stimuli and can polarize into either the pro-inflammatory M1 type or the anti-inflammatory M2 type. The equilibrium between these two types directly influences neuronal survival[4].

Within the theoretical framework of traditional Chinese medicine (TCM), CIRI is classified under the category of "stroke", with its primary pathogenesis identified as "qi deficiency and blood stasis". Consequently, the principal clinical treatment approach involves tonifying qi and promoting blood circulation, consistent with the TCM principle of addressing the root cause of disease. The application of this qi-tonifying and blood-activating method has been shown to enhance blood supply following cerebral ischemia-reperfusion by mitigating inflammatory responses and oxidative stress, thereby providing significant neuroprotection[5]. Clinical studies have demonstrated that methods aimed at tonifying qi and promoting blood circulation can significantly facilitate the recovery of neurological function, enhance cognitive performance, and effectively improve the functional abilities of patients with CIRI[6-7]. Qizhi Formula, grounded in TCM principles of tonifying qi and promoting blood circulation, is prepared at a 10∶1 ratio of Astragali Radix to Hirudo. This formulation has shown notable clinical efficacy in the treatment of ischemic stroke. Nevertheless, the underlying mechanisms through which it exerts therapeutic effects on CIRI remain to be clarified[8-11].

Therefore, investigating the regulatory effects of drugs on microglial polarization at the cellular level is a critical step in elucidating their mechanisms of action against CIRI. In this study, the BV2 microglial cell injury model induced by oxygen-glucose deprivation/reoxygenation (OGD/R) was employed to assess the

Received: June 6, 2026 Accepted: August 10, 2026

Supported by Inner Mongolia Natural Science Foundation (2024MS08033); National Natural Science Foundation of China (82060908); Joint Research Fund of the Affiliated Hospital of Inner Mongolia Medical University (2023NYFY LHZD010); Special Funds for the Inner Mongolia Autonomous Region Clinical Medical Research Center for Neurological Diseases; Key Clinical Specialties for High-Quality Development in the Capital Region (Neurological Diseases).

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effects of Qizhi Formula and drug-containing sera derived from Astragali Radix and Hirudo. Levels of inflammatory factors and the expression of microglial polarization markers in the cell supernatant were measured to determine the regulatory influence of Qizhi Formula on microglial polarization and to compare its efficacy with that of the individual drugs.

2 Materials and methods

2.1 Experimental animals and cells Fifty healthy male SD rats, aged 8 weeks and weighing between 250 and 280 g, were obtained from Beijing Vital River Laboratory Animal Technology Co., Ltd. (license No.: SCXK [Jing] 2021-0006). All animal experiments and procedures were conducted in strict accordance with the ethical guidelines approved by the Animal Ethics Committee of Inner Mongolia Medical University (Approval No.: YKD202402179). The BV2 microglial cell line utilized in this study was procured from Wuhan Servicebio Technology Co., Ltd. (catalog No.: STCC20009P).

2.2 Methods

2.2.1 Culture of BV2 microglia. BV2 microglia were cultured in a humidified incubator at 37 ℃ with 5% CO_2. Upon reaching 80% – 90% confluence, the cells were digested using trypsin-EDTA solution and subsequently inoculated and subcultured at a ratio of 1∶3[12].

2.2.2 Preparation of drug-containing serum. Fifty SD rats were randomly assigned to 5 groups of 10 animals each using a random number table. The groups were designated as follows: the blank control group (NC), the nimodipine treatment group (NMP), the Astragali Radix treatment group (A), the Hirudo treatment group (H), and the Qizhi Formula treatment group (QZ).

The dosage administered was determined based on the conversion formula of human and animal body surface area in *Pharmacological Experiment Methodology*. Traditional Chinese medicine was administered via gavage at five times the clinical equivalent dose for adults[13], with the following group dosages: Group A, 13.5 g/kg/d; Group H, 1.35 g/kg/d; Group QZ, 14.85 g/kg/d; and Group NMP, 20.16 mg/kg/d. Group NC received an equivalent volume of normal saline. All treatments were administered by gavage twice daily (at 9:00 and 17:00) for 7 consecutive days[14]. The preparation of the TCM decoction involved soaking the herbs in 8 volumes of distilled water for 30 min. The mixture was then brought to a boil over high heat, followed by simmering over low heat for 30 min. The decoction was filtered, after which 6 volumes of water were added, and the mixture was simmered for an additional 20 min. The two decoctions were combined, concentrated at 60 ℃, and stored at 4 ℃ for future use. The preparation method for the decoctions of the individual herbs Astragali Radix and Hirudo was identical to the procedure described above.

One hour following the final intragastric administration, blood samples were obtained from rats via the abdominal aorta. The collected blood was centrifuged at 3 500 rpm for 15 min at 4 ℃ to separate the serum. The serum was then sterilized using a 0.22 μm microporous filter membrane and stored at −80 ℃ for future analysis.

2.2.3 Establishment of OGD/R model. BV2 cells in the logarithmic growth phase were harvested, digested, and adjusted to the desired cell density before being seeded into 96-well plates. The cells were incubated under standard conditions for 24 h to allow for adhesion. The original medium was discarded, and the cells were washed twice with phosphate-buffered saline (PBS). The medium was then replaced with glucose-free Dulbecco's Modified Eagle Medium (DMEM), and the cells were incubated at 37 ℃ in an atmosphere containing 95% N_2 and 5% CO_2 for 6 h to establish an oxygen-glucose deprivation (OGD) model[15]. Following this, the medium was replaced with normal complete medium, and the cells were cultured under normoxic conditions for an additional 24 h to generate the OGD/R model. The control group was maintained in normal medium under normoxic conditions throughout the entire experimental period.

2.2.4 Verification of OGD/R model. After modeling, BV2 cells were inoculated in equal quantities into 96-well plates. The absorbance at a wavelength of 450 nm was measured using the CCK-8 assay to assess the viability of BV2 cells and to confirm the successful establishment of the model. The cell survival rate was calculated using the following formula:

Survival rate (%) = [(*OD* value of the model group − *OD* value of the blank group)/(*OD* value of the control group − *OD* value of the blank group)] × 100%.

To assess the effect of model preparation, the following procedures were conducted: BV2 cells in the logarithmic growth phase were selected, and their density was adjusted to 5×10^4 cells/mL. The cells were then seeded into 96-well plates at a volume of 100 μL per well and cultured under standard conditions for 24 h. Subsequently, the OGD/R model was established following the previously described method. All procedures were performed in strict accordance with the instructions provided with the lactate dehydrogenase (LDH) detection kit. The experimental setup included sample wells, sample control wells, maximum enzyme activity control wells, and background blank control wells. Upon completion of the treatment, the cell supernatant was transferred to a new 96-well plate, and the LDH detection working solution was added. The plate was incubated at room temperature in the dark for 30 min. Finally, the *OD* at 490 nm was measured using an enzyme-linked immunosorbent assay (ELISA) reader. LDH cytotoxicity was calculated using the following formula:

LDH cytotoxicity (%) = [(Absorbance of treated sample well − Absorbance of sample control well)/(Absorbance of maximum enzyme activity control well − Absorbance of sample control well)] × 100%.

2.2.5 Cell grouping and medication. Successfully modeled BV2 cells were categorized into six groups: the blank control group (normal culture with 10% blank serum), the model group (OGD/R with 10% blank serum), the nimodipine treatment

group (OGD/R with 10% serum containing nimodipine), the Astragali Radix treatment group (OGD/R with 10% serum containing Astragali Radix), the Hirudo treatment group (OGD/R with 10% serum containing Hirudo), and the Qizhi Formula treatment group (OGD/R with 10% serum containing Qizhi Formula).

Following 6 h of OGD treatment, the medium in each group was replaced with complete medium containing drug-containing serum, and the cells were subjected to reoxygenation for an additional 24 h. Both the cells and supernatant were collected for further analysis[16].

2.2.6 Detection of inflammatory factors by ELISA assay. The concentrations of TNF-α, IL-1β, TGF-β, and IL-10 in the cell culture supernatants were quantified using ELISA. Gradient dilutions of the standards were prepared, and standard wells, blank wells, and sample wells were established accordingly. The diluted standards and cell supernatants were added to their respective wells. Following plate sealing, incubation was conducted at room temperature. After incubation, the plate was washed to remove unbound substances, and enzyme-labeled antibodies were added for further incubation. Subsequent to a second wash, a chromogenic substrate was introduced and incubated in the dark until a distinct blue color gradient developed. The enzymatic reaction was terminated by adding stop solution. Absorbance values at 450 nm were measured using an ELISA reader. Concentrations of the inflammatory cytokines in each sample were calculated based on the standard curve.

2.2.7 mRNA expression of M1/M2 type markers in BV2 cells by RT-qPCR assay. Following successful modeling, BV2 cells were treated according to the designated grouping protocol and incubated at 37 ℃ with 5% CO_2 for 24 h. Afterwards, the supernatant was removed, and the cells were washed with PBS. Total RNA was extracted from each group of BV2 cells using an RNA extraction kit (DP419, Tiangen Biotech Co., Ltd., China; batch No.: 451330) to assess RNA concentration and purity. Reverse transcription was conducted using a rapid reverse transcription kit (TOYOBO, Japan; batch No.: 741100), and amplification was performed using cDNA as the template. The real-time fluorescence quantitative PCR was carried out under the following conditions: 95 ℃ for 5 sec, 60 ℃ for 10 sec, and 72 ℃ for 15 sec, repeated for 40 cycles. Amplicon specificity was confirmed via melting curve analysis. Primer sequences are presented in Table 1. *GAPDH* served as the reference gene, and relative mRNA expression levels of the target gene were calculated using the $2^{-\Delta\Delta CT}$ method. Each sample was analyzed in triplicate, and results are reported as mean values.

Table 1 Primer sequences

Gene	Forward primer (5′-3′)	Reverse primer (5′-3′)
Arg1	GGCAGAAGTAGAAGAAGAGGA	GTGAGCATCCACCCAGATGA
CD86	TCAAGACATGTGTAACCTGCAC	CAGAACCGACTTTTTCCGGTC
CD206	GAGGACTGCGTGGTGATGAA	CATGCCGTTTCCAGCCTTTC
iNOS	GAAAGCGGTGTTCTTTGCTTCT	CTTATACTGTTCCATGCAGACAACCT
GAPDH	CTCTCTGCTCCTCCCTGTTC	TCACACCGACCTTCACCATC

2.2.8 Statistical analysis. Data analysis was performed using SPSS 26.0. Measurement data are presented as mean ± standard deviation ($\bar{x} \pm s$). One-way analysis of variance (ANOVA) was employed to compare differences among multiple groups. When variances were homogeneous, the *LSD* test was applied for multiple comparisons; otherwise, Dunnett's T3 test was utilized for heterogeneous variances. A *P*-value less than 0.05 was considered statistically significant. Graphs were generated using GraphPad Prism 10.5.0.

3 Results and analysis

3.1 Verification of OGD/R model The CCK-8 assay showed that the survival rate of BV2 cells in the OGD/R group was 41.46% (40.34% – 42.59%). The LDH release rate was 44.78% (43.07% – 45.96%), as illustrated in Fig. 1. These findings indicate that OGD/R treatment significantly decreased cell viability and increased cytotoxicity, confirming the successful establishment of the model.

3.2 Effects of Qizhi Formula on the expression of inflammatory factors in BV2 cells induced by OGD/R The ELISA assay results (Fig. 2) demonstrated that, in comparison to the blank control group, the model group exhibited significantly elevated levels of TNF-α and IL-1β ($P<0.01$), alongside significantly reduced levels of IL-10 and TGF-β ($P<0.01$), indicating that OGD/R induced a pronounced inflammatory response. Relative to the model group, all treatment groups significantly decreased the concentrations of TNF-α and IL-1β and increased the levels of IL-10 and TGF-β ($P<0.05$ or $P<0.01$). Notably, the regulatory effect observed in the Qizhi Formula group was significantly superior to that of the Astragali Radix and Hirudo treatment groups ($P<0.05$), suggesting a synergistic interaction between Astragali Radix and Hirudo.

3.3 Effects of Qizhi Formula on the mRNA expression of microglial polarization markers in BV2 cells The RT-qPCR results (Fig. 3) indicated that, compared to the blank group, the mRNA expression levels of M1 type markers *CD86* and *iNOS* were significantly elevated in the OGD/R group ($P<0.001$), whereas no significant changes were observed in the M2 type markers *CD206* and *Arg1*. These findings indicate that OGD/R predominantly induces M1 type polarization. Relative to the OGD/R group, the NMP and QZ groups exhibited a significant reduction in *CD86* and *iNOS* expression levels ($P<0.01$), alongside a significant increase in *CD206* and *Arg1* expression ($P<0.01$). Notably, the modulatory effects of the Astragali Radix and Hirudo treatment groups on polarization markers were less pronounced than those observed in the Qizhi Formula treatment group.

4 Discussion

Neuroinflammation is a central link of secondary injury following CIRI, with the imbalance of microglial M1/M2 polarization serving as a critical mechanism that exacerbates neuroinflammation and subsequent brain tissue damage. In response to ischemic and

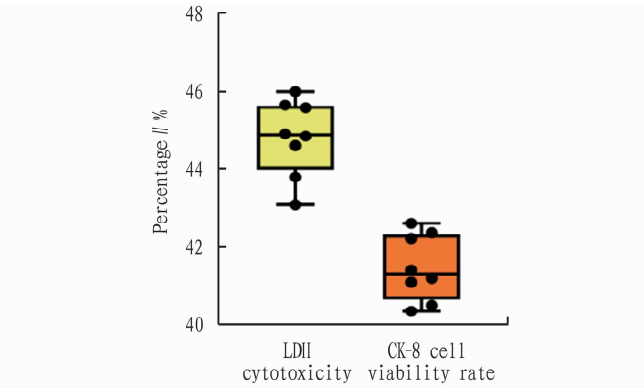
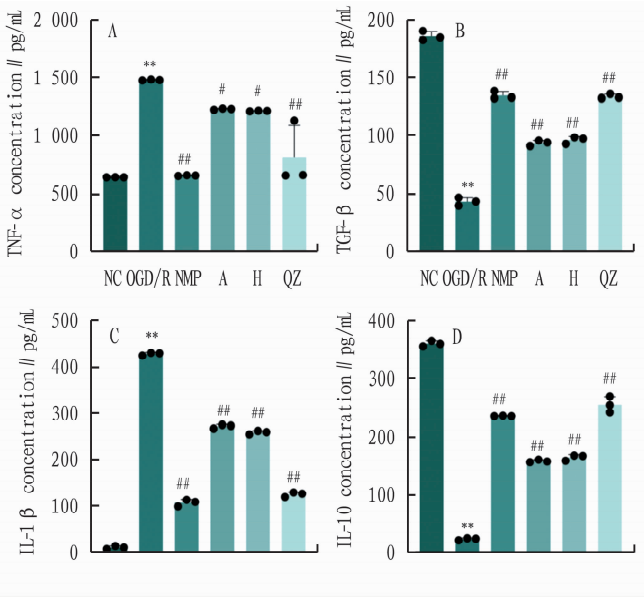
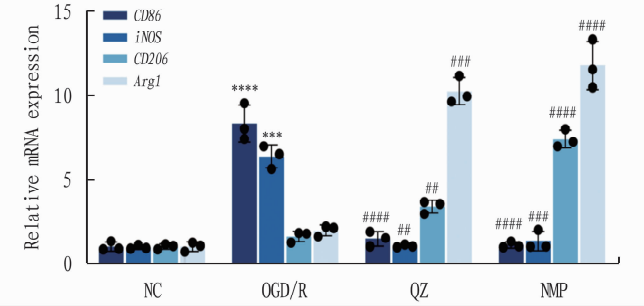


Fig. 1 Verification of OGD/R model (CCK-8 and LDH detection results)



NOTE NC. Blank control group; OGD/R. Model group; NMP. Nimodipine treatment group; A. Astragali Radix treatment group; H. Hirudo treatment group; QZ. Qizhi Formula treatment group.

Fig. 2 ELISA assay results of inflammatory factors in BV2 cells across various groups



NOTE NC. Blank control group; OGD/R. Model group; QZ. Qizhi Formula treatment group. NMP. Nimodipine treatment group. ## indicates $P < 0.01$, *** or ### indicates $P < 0.001$, and **** or #### indicates $P < 0.0001$.

Fig. 3 mRNA expression levels of M1/M2 polarization markers in BV2 cells across various groups

hypoxic stimuli, microglia mainly polarize towards the pro-inflammatory M1 type, releasing large quantities of pro-inflammatory cytokines such as TNF-α and IL-1β, thereby initiating inflammatory cascades that intensify neuronal injury. In contrast, M2 type microglia secrete anti-inflammatory mediators, including IL-10 and TGF-β, which mitigate inflammatory progression and facilitate tissue repair. Consequently, modulating microglial polarization to favor the M2 type and restoring the dynamic equilibrium between M1 and M2 types have become important therapeutic strategies for ameliorating inflammatory damage in CIRI.

In this study, the BV2 microglia OGD/R model was employed to simulate the *in vitro* pathological processes of CIRI. For the first time, the regulatory effects of Qizhi Formula on microglial polarization following ischemia-reperfusion were systematically investigated. The experimental results demonstrated that serum containing Qizhi Formula significantly inhibited OGD/R-induced M1 type polarization of BV2 cells, as evidenced by the downregulation of *CD86* and *iNOS* expression. Concurrently, it promoted M2 type polarization, indicated by the upregulation of *CD206* and *Arg1* expression, thereby effectively correcting the microglial polarization imbalance induced by ischemia-reperfusion. This regulatory effect was further corroborated at the level of inflammatory mediators: treatment with Qizhi Formula markedly reduced the secretion of pro-inflammatory cytokines TNF-α and IL-1β, while increasing the levels of anti-inflammatory cytokines IL-10 and TGF-β. These findings suggest that Qizhi Formula effectively attenuates neuroinflammatory responses associated with CIRI.

It is noteworthy that Qizhi Formula group exhibited significantly superior regulatory effects on inflammatory factors and polarization markers compared to the monotherapy groups of Astragali Radix and Hirudo. This finding clearly reflects the synergistic benefits of combining Astragali Radix and Hirudo. The results not only validate the scientific basis of the TCM theory of "tonifying qi and promoting blood circulation" compatibility but also highlight the advantages of "multi-component, multi-target" coordinated regulation within the complex pathological networks addressed by TCM compound prescriptions.

From the perspective of TCM theory, the primary pathogenesis of CIRI is characterized by "qi deficiency and blood stasis". Astragali Radix is recognized as "a principal herb for tonifying qi", particularly enhancing the qi of the spleen and lungs to promote blood circulation. Hirudo is a potent herb known for its ability to "resolve and remove blood stasis", effectively unblocking meridians and collaterals while dispelling blood stasis. The combination of these two herbs, one tonifying and the other activating, serves to tonify qi, facilitate blood circulation, and remove blood stasis to restore meridian patency, thereby aligning precisely with the therapeutic principle of "tonifying qi and promoting blood circulation". This study offers contemporary pharmacological evidence supporting this classical herbal combination, particularly through mechanisms involving the regulation of microglial polarization and the inhibition of neuroinflammation.

In summary, this study confirmed that Qizhi Formula exerts a cerebroprotective effect against CIRI by modulating the M1/M2 polarization balance of BV2 microglia and attenuating excessive neuroinflammatory responses. The synergistic interaction between Astragali Radix and Hirudo is significant, reflecting the scientific basis of the therapeutic approach aimed at tonifying qi and promoting blood circulation. Future research will focus on elucidating the upstream signaling pathways through which Qizhi Formula regulates microglial polarization and will include *in vivo* experiments to validate these findings, thereby providing more comprehensive experimental evidence to support its clinical application.

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