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Neuroprotective potential of glibenclamide is mediated by antioxidant and anti-apoptotic pathways in intracerebral hemorrhage

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Tables and Figures: Figures 1-9;

Highlights

- Glibenclamide has neuroprotective effects in ICH
- Glibenclamide can scavenge free radicals and inhibit cell apoptosis
- Glibenclamide can downregulate pro-apoptotic activated-caspase-3 and Bax, and upregulate anti-apoptotic Bcl-2

Abstract

The sulfonylurea receptor 1 (SUR1)-regulated $NC_{ca\text{-}ATP}$ channels were progressively upregulated and demonstrated unchecked opening in central nervous system (CNS) injury, which induced cerebral damage. Glibenclamide (GLI) can block $NC_{ca\text{-}ATP}$ channels and consequently exert protective effects. Recent studies have found that GLI has antioxidative effects. In this study, we primarily explored the antioxidative effects of GLI in a rat model of intracerebral hemorrhage (ICH). We found that GLI could scavenge free radicals, reduce activated-caspase-3 expression, increase the Bcl-2/Bax ratio, inhibit apoptosis, and improve functional neurological outcomes in a rat model of ICH.

Keywords

Intracerebral hemorrhage; glibenclamide; oxidative stress; apoptosis

1. Introduction

Intracerebral hemorrhage (ICH) accounts for approximately 10-15% of all stroke cases.