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TUG1 suppresses neural repair subsequent to cerebral hemorrhage *via* the miR-381-3p/BDNF axis

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Abstract

Intracerebral hemorrhage (ICH) has a high rate of death and disability. LncRNA-TUG1 is essential for the pathological changes secondary to ICH. The purpose of this work was to investigate the possible mechanism by which TUG1 inhibits neural repair subsequent to ICH through adjusting miR-381-3p/brain-derived neurotrophic factor (BDNF). After the ICH model was created, miR-381-3p agomir and pcDNA-TUG1 were injected. The neural function of rats was estimated using the modified neurological severity score. To quantify the expression of genes and proteins, western blotting, immunohistochemistry, and qRT-PCR were used. To confirm the interaction between TUG1 and miR-381-3p and between miR-381-3p and BDNF mRNA, a luciferase reporter assay was employed. In rats treated with miR-381-3p agomir, a trend of improvement in neurological dysfunction was observed, while the pcDNA-TUG1-treated ones showed deterioration. Furthermore, miR-381-3p agomir increased, while pcDNA-TUG1 reduced the expression level of BDNF in ICH rats. TUG1 and BDNF mRNA were validated to attach directly to miR-381-3p. Overexpressing TUG1 inhibited the level of BDNF by sponging miR-381-3p and antagonized its protective effect on neural repair in ICH rats. Our study suggests that TUG1 can sponge miR-381-3p to downregulate BDNF expression and inhibit neural repair following ICH, demonstrating a potential signaling pathway that is conducive to a better understanding of the pathological mechanisms of ICH.

Key words: *intracerebral hemorrhage, neural repair, miR-381-3p, taurine-upregulated gene 1, brain-derived neurotrophic factor.*

Introduction

The mortality and disability rates of intracerebral hemorrhage (ICH) rank first among cerebrovascular diseases [13]. Neurocyte injury is an important factor contributing to ICH patients' disability and fatality [26]. Although a series of studies have conducted in-depth explorations of ICH, the effective treatment methods for ICH are still relatively limited. Therefore, exploring the mechanism of nerve injury and repair after ICH is of great importance in seeking new treatment strategies for this disease.

Numerous prior studies have confirmed the involvement of brain-derived neurotrophic factor (BDNF) in the process of neuroprotection and neural repair following brain injury [8,18,22]. Studies have demonstrated that endogenous BDNF facilitates neurogenesis after ICH, not only in rat models of ICH but also in ICH patients [15]. Additionally, BDNF mRNA in the cortex has been confirmed to be associated with motor deficits following ICH [9].

According to reports, miRNAs are involved in various important physiological and pathological processes [17,19]. Researchers found that in cerebral ischemia-reperfusion injury, miR-381-3p has a neuroprotective function [7]. Besides that, overexpression of microRNA-381-3p can improve the microglial inflammation reaction to hypoxia/ischemia [4]. On the other

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hand, the function of miR-381-3p in ICH is not well documented.

It has been demonstrated that long non-coding RNAs (lncRNAs) are involved in a variety of biological processes. One of these biological mechanisms of lncRNAs is mediation of expression of target mRNAs *via* competitive binding to miRNAs [20]. LncRNA-TUG1 can be used as a new molecular biomarker of diabetes with stroke, and may be a therapeutic target for such patients [2]. Additionally, TUG1 is essential for the pathological changes secondary to ICH [14]. However, the regulatory mechanism of TUG1 in the process of nerve injury and repair following ICH remains to be illustrated.

Using bioinformatics techniques, we predicted potential regulatory feedback loops between miR-381-3p and TUG1/BDNF mRNA. This investigation revealed that TUG1 exacerbated neuronal damage after ICH *via* regulating BDNF expression by downregulating miR-381-3p.

Material and methods

Animals

All procedures with laboratory animals were approved by the Animal Ethics Committee of Renmin Hospital of Wuhan University, and we conducted the animal procedures following the recommendations in the Guide for the Care and Use of Laboratory Animals of the National Institutes of Health. We obtained rats (Sprague Dawley, male, 230 to 270 g) from Shulaibao Biotechnology (Wuhan, China).

Model animals for ICH

The ICH rat model was established according to a published protocol [14]. Briefly, 2% pentobarbital sodium (30 mg/kg) was injected into the peritoneum of rats, and then all anesthetized rats were anchored on a stereotactic platform. After that, we performed a scalp incision for the cranial bone window. A hole was drilled 0.6 mm in front of the bregma and 3.5 mm on the right side. Then we injected autologous blood from the femoral artery (50 µl, injection speed 10 µl/minute) using a needle microinjector that was placed 5.5 mm below the skull. In the control group, rats underwent the same procedures except for injecting autologous blood. All experiments were conducted at a temperature of 37 ±0.5°C.

Construction of plasmids and RNA oligonucleotides

Shanghai Genechem (Shanghai, China) generated the plasmid vectors for pcDNA-TUG1 and pcDNA-NC (the negative control). RiboBio (Guangzhou, China) performed design and synthesis for miR-381-3p agomir and agomir-NC, as well as miR-381-3p mimic and mimic-NC.

Grouping and treatment of animals

The following groups of surviving rats (n = 6/group) were formed: sham, ICH, ICH + pcDNA-TUG1, ICH + pcDNA-NC, ICH + miR-381-3p agomir, ICH + agomir NC, ICH + miR-381-3p agomir + pcDNA-TUG1, as well as ICH + miR-381-3p agomir + pcDNA-NC. Injection of pcDNA-TUG1 or pcDNA-NC into rats was conducted as follows: the day prior to ICH modeling, rats were anesthetized and position fixation was performed. Then, 5 µl of plasmid vector was injected into the right lateral ventricle (0.45 cm below the surface of the skull, 0.15 cm lateral, and 0.08 cm posterior to the bregma). Tail vein administration of miR-381-3p agomir or agomir NC was performed as previously reported [16].

Neurological function evaluation

Three days after ICH, the Modified Neurological Severity Score (mNSS) was administered to access the neurological performance. A pair of researchers blind-tested rats. The mNSS ranged from 0 to 18, with higher scores indicating profound neurological deficits. Rats were then sacrificed for tissues, which were utilized for detailed examination.

Cell culture

The Experimental Teaching Center of Basic Medical Sciences of Wuhan University supplied the HEK293T cell line. Cell culture was performed in a medium (5% CO_2, 37°C).

Immunohistochemistry (IHC)

Paraformaldehyde (PFA, 4%) was used for fixation of the rat brains, paraffin embedding was performed, and then 4 µm sections were produced. After deparaffinization and rehydration, sections were treated with 3% H_2O_2 for 20 min. Microwave heating was used to retrieve antigen on sections in citrate reagent (10 mM, 30 min). Following PBS washes 3 times, goat serum was used for blocking sections. Primary antibody incubation was performed overnight, followed by incubation with the secondary antibody (biotin-conjugated goat-anti-rabbit IgG). After rinsing sections, peroxidase substrate solution was used until the desired staining intensity was reached. BX51 upright microscopy (Olympus; Tokyo, Japan) was used for imaging of immune staining. Light or dark brown denotes BDNF-positivity detected in the membrane or cytoplasmic location.

qRT-PCR

RNA extraction from rat brain tissue was performed using TRIzol reagent (Invitrogen, Carlsbad, USA) following the company-recommended protocol. The gene expression was evaluated by qRT-PCR. To assess the target gene expression levels in the samples, the $2^{-\Delta\Delta Ct}$ protocol was used. GAPDH was used as an internal reference for TUG1 and BDNF mRNA, whereas U6 RNA was used as an internal reference for miR-381-3p. The primer sequences are shown in Table I.

Table I. Primer sequences amplified

Gene		Primer sequences (5′-3′)
TUG1	Forward	ACCCTGTGGAGTACCCAGGAC
	Reverse	GGGTTGTTCTCTAGAGTTGCTGG
miR-381-3p	Forward	CCAGCATAACAGTCTACAGCCA
	Reverse	TATGGTTGTTCACGACTCCTTCAC
BDNF	Forward	ATGGGTTACACGAAGGA
	Reverse	GCCCGAACATACGATT
U6	Forward	CCTGCTTCGGCAGCACAT
	Reverse	AACGCTTCACGAATTTGCGT
GAPDH	Forward	CGCTAACATCAAATGGGGTG
	Reverse	TTGCTGACAATCTTGAGGGAG

Western blotting analysis

SDS-PAGE was used for separation of protein, and protein blotting was performed on PVDF membrane, followed by skim milk (5%) for blocking. Primary antibodies, including anti-GAPDH for reference, were used for overnight incubation of membranes at 4°C. After rinsing three times with TBST solution, secondary antibodies were used to detect primary antibodies. The LI-COR Odyssey Infrared Imaging System (LI-COR Biosciences; Lincoln, NE, USA) was used for imaging of western blotting.

Bioinformatics prediction

Using StarBase (http://starbase.sysu.edu.cn/), the miR-381-3p binding locations in TUG1 as well as BDNF mRNA were predicted.

Luciferase reporter assay

Using Lipofectamine 2000 (Invitrogen, USA) and the company-recommended methodology, transient co-transfection was performed for HEK293T cells with pmirGLO-TUG1-MUT (mutant type)/pmirGLO-TUG1-WT (wild type) and miR-381-3p mimic or mimic-NC. The luciferase test was conducted using the Dual-Luciferase Reporter (DLR) Test System (Promega, USA).

Statistical analysis

SPSS Statistics version 20.0 (IBM, USA) was used to assess the statistical data. Mean ±SD was used for all expression data. To evaluate differences between groups, one-way ANOVA was employed, followed by the Bonferroni post hoc test. A p-value of ≤ 0.05 was considered statistically significant.

Results

MiR-381-3p protects against ICH-induced neurologic deficits

A significant increase in miR-381-3p expression in ICH rats' neural tissue was observed after injecting miR-381-3p agomir (Fig. 1A). By day 3 following treatment, the ICH group of rats and the ICH + miR-381-3p agomir group of rats showed significantly different levels of mNSS (Fig. 1B). The upregulating impact of miR-381-3p on BDNF mRNA was evaluated using qRT-PCR (Fig. 1C). IHC tests showed that in the cerebral tissue of ICH rats, overexpression of miR-381-3p drastically lowered the levels of BDNF protein (Fig. 1D).

TUG1 suppresses neural repair following ICH injury

When rats were administered with pcDNA-TUG1 instead of pcDNA-NC, TUG1 expression was significantly higher (Fig. 2A). On day three after the operation, the ICH rats in the pcDNA-TUG1 group showed significant dysfunction of neurological activity in comparison to the ICH rats in the pcDNA-NC group (Fig. 2B). In the present study, overexpression of TUG1 was found to suppress BDNF mRNA expression significantly (Fig. 2C). IHC demonstrated that overexpression of TUG1 dramatically diminished the BDNF protein expression in the rats' cerebral tissue following ICH (Fig. 2D).

TUG1 targets miR-381-3p as a negative regulator

To identify whether miR-381-3p serves as a bona fide site for TUG1, StarBase analysis (http://starbase.sysu.edu.cn/) was performed and demonstrated that TUG1 was found to have a binding site for miR-381-3p (Fig. 3A). Co-transfection of miR-381-3p mimic drastically inhibited activity of luciferase in TUG1-WT but not in TUG1-MUT-transfected HEK293T cells, as seen in Figure 3B. qRT-PCR was performed to find out whether TUG1 affects miR-381-3p expression, and the results showed a significant decrease in miR-381-3p following transfection of pcDNA-TUG1 compared with pcDNA-NC (Fig. 3C).

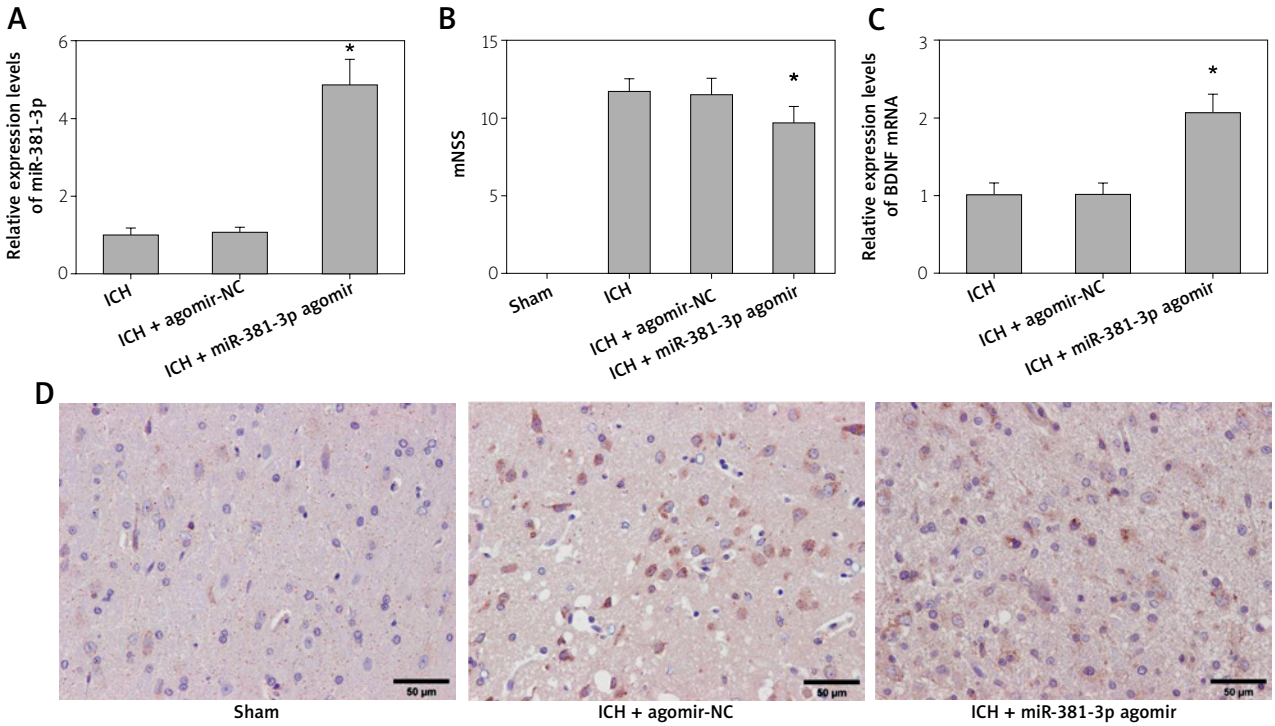

Fig. 1. Overexpression of miR-381-3p promotes neurological function recovery and enhances BDNF expression. **A**) Modulation of miR-381-3p levels by miR-381-3p agomir in ICH rats. **B**) The effect of miR-381-3p on neural function in ICH rats. **C**) Relative BDNF mRNA levels in ICH rats treated with miR-381-3p agomir or agomir-NC. **D**) Expression of BDNF was detected by IHC. Magnification of the microphotograph, 400×. Scale bars, 50 µm. *$p < 0.05$, vs. agomir-NC.

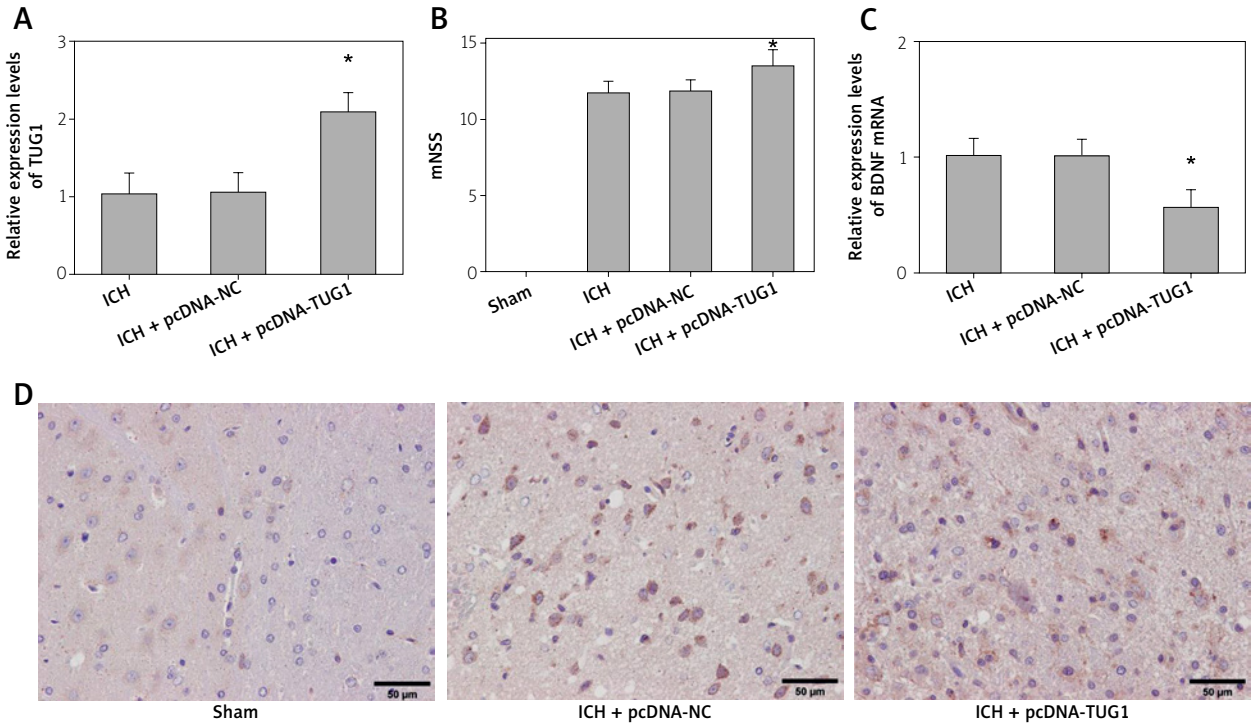

Fig. 2. Overexpression of TUG1 aggravates neurological deficits and exacerbates ICH-injury. **A**) TUG1 was upregulated by pcDNA-TUG1. **B**) The effect of overexpression of TUG1 on neural function in ICH rats. **C**) Relative BDNF mRNA levels in ICH rats treated with pcDNA-TUG1. **D**) Expression of BDNF in different groups was detected by IHC. Magnification of the microphotograph, 400×. Scale bars, 50 µm. *$p < 0.05$, vs. pcDNA-NC.

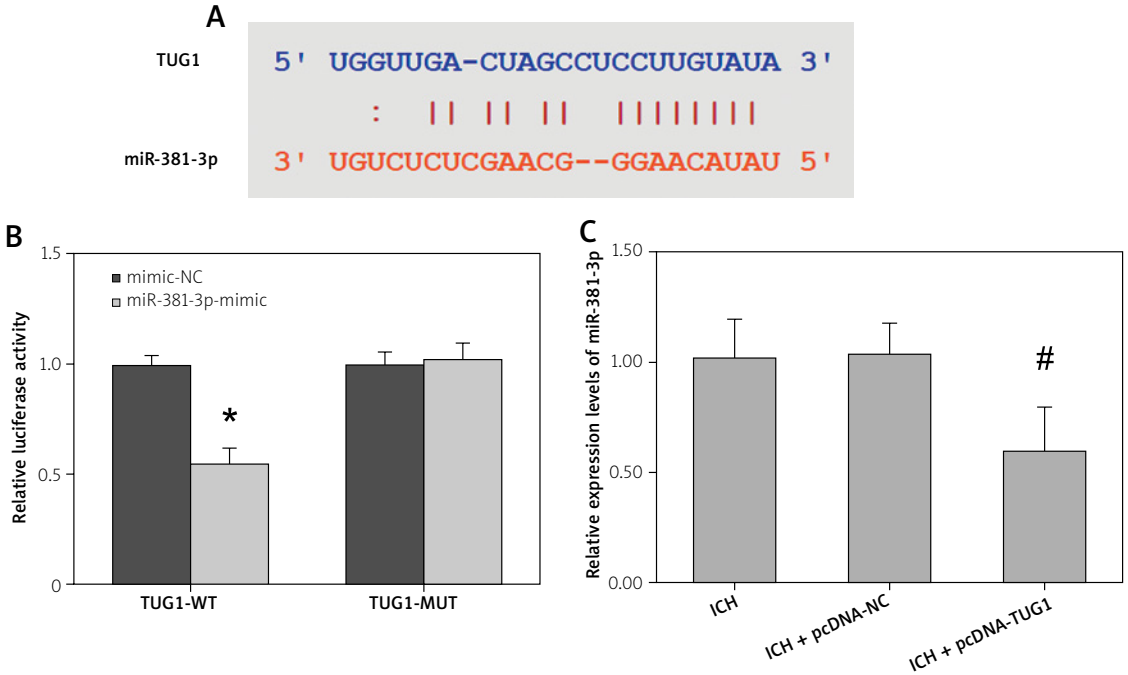

Fig. 3. TUG1 binds to miR-381-3p and regulates miR-381-3p expression. **A**) The binding site of TUG1 on mi-R381-3p was predicted by bioinformatics software. **B**) Luciferase reporter assay of miR-381-3p and TUG1. **C**) miR-381-3p expression in ICH rats treated with pcDNA-NC or pcDNA-TUG1 was detected by qRT-PCR analysis. *$p < 0.05$, vs. mimic-NC, #$p < 0.05$, vs. pcDNA-NC.

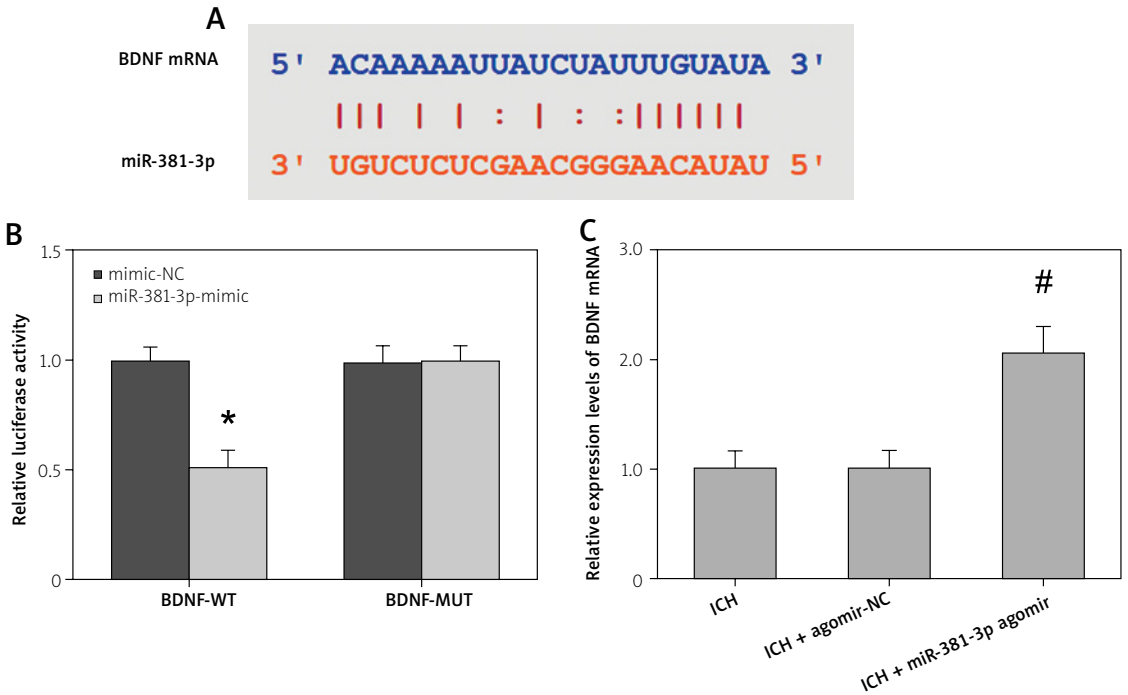

Fig. 4. MiR-381-3p binds to BDNF mRNA and regulates its expression. **A**) The binding site of BDNF mRNA on miR-381-3p was predicted by bioinformatics software. **B**) Luciferase reporter assay of miR-381-3p and BDNF mRNA. **C**) Expression of BDNF mRNA in ICH rats treated with miR-381-3p agomir or agomir-NC was detected by qRT-PCR analysis. *$p < 0.05$, vs. mimic-NC, #$p < 0.05$, vs. ICH + agomir-NC.

MiR-381-3p directly targets and regulates BDNF mRNA

It was anticipated *via* StarBase analysis that miR-381-3p directly targets BDNF mRNA (Fig. 4A). A luciferase reporter experiment was then carried out. Figure 4B demonstrates that luciferase activity was considerably suppressed after cotransfection with miR-381-3p agomir in WT-BDNF but not MUT-BDNF-transfected HEK-293T cells. Furthermore, qRT-PCR verified miR-381-3p's capacity to control BDNF mRNA expression. BDNF mRNA levels were significantly different between the miR-381-3p agomir and control cohorts (Fig. 4C).

TUG1 exacerbates neurological dysfunction and inhibits BDNF expression following ICH possibly by sponging miR-381-3p

We hypothesized that TUG1 affects neurological dysfunction following ICH by controlling the miR-381-3p/BDNF axis. TUG1 overexpression induced attenuation of the miR-381-3p effect on the functional improvement of neurological activity in ICH rats (Fig. 5A). Additionally, irrespective of the model rats' transfection status with miR-381-3p agomir, qRT-PCR analysis demonstrated that excessive expression of TUG1 may reduce the miR-381-3p expression (Fig. 5B). What is more, the miR-381-3p's effect on the upregulation of BDNF mRNA expression in the cerebral tissue of ICH rats was partially reversed by TUG1 overexpression (Fig. 5C). Moreover, IHC data demonstrated that TUG1 overexpression inhibited miR-381-3p's ability to upregulate BDNF protein expression in part (Fig. 5D). Western blotting also demonstrated that TUG1 overexpression attenuated miR-381-3p's ability to promote BDNF protein expression (Fig. 5E).

Discussion

LncRNAs play a vital role in the pathological changes secondary to ICH and may function as the disease's

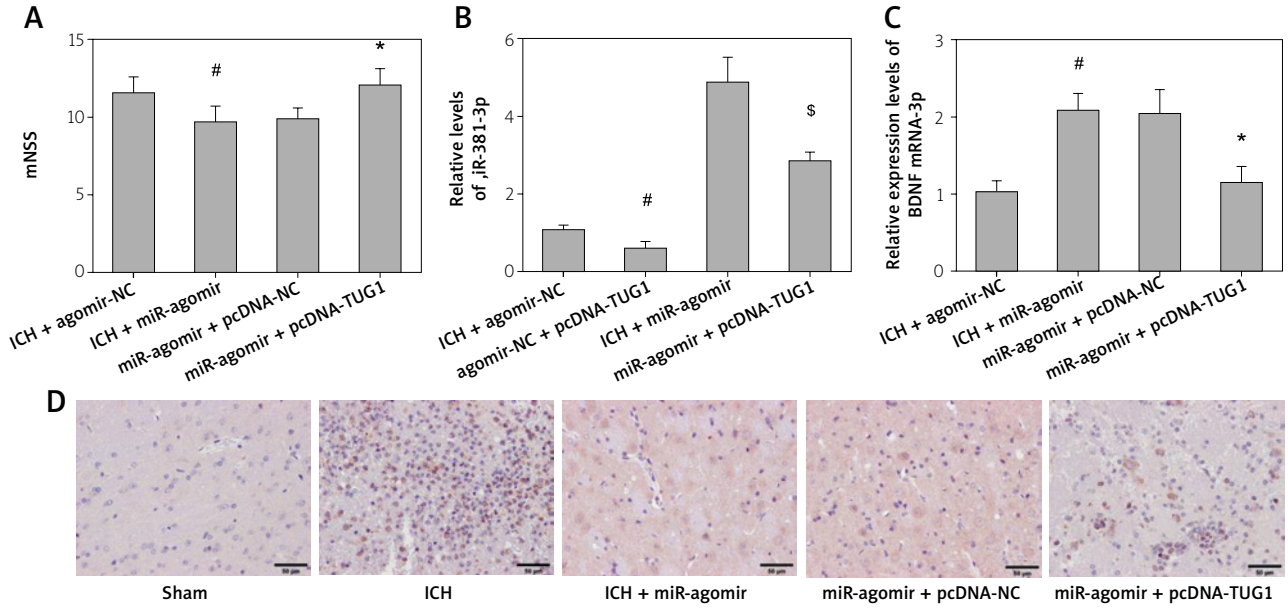

Fig. 5. TUG1 antagonizes the promoting effect of miR-381-3p on BDNF to exacerbate cerebral injury in ICH rats. **A**) Neurological function of ICH rats treated with agomir-NC, miR-381-3p agomir, miR-381-3p agomir + pcDNA-NC, or miR-381-3p agomir + pcDNA-TUG1. **B**) Relative miR-381-3p levels in ICH rats treated with agomir-NC, agomir-NC + pcDNA-TUG1, miR-381-3p agomir, or miR-381-3p agomir + pcDNA-TUG1. **C**) Relative BDNF mRNA levels in ICH rats treated with agomir-NC, miR-381-3p agomir, miR-381-3p agomir + pcDNA-NC, or miR-381-3p agomir + pcDNA-TUG1. **D**) Images from IHC photographs of BDNF in different groups. Magnification of the microphotograph, 400×. Scale bars, 50 µm. **E**) Western blotting analysis of BDNF expression in different groups. $^{\#}p < 0.05$, vs. ICH + agomir-NC, $^{*}p < 0.05$, vs. miR-381-3p agomir + pcDNA-NC, $^{\$}p < 0.05$, vs. ICH + miR-381-3p agomir.

treatment target [23]. It has been established that the lncRNA TUG1 plays a crucial role in ischemic brain injury [3]. In our previous research, TUG1 was confirmed to be involved in the pathophysiological process of vascular regeneration subsequent to ICH [14], and TUG1 downregulation may significantly suppress the inflammatory response after nerve injury [10]. Even so, little is known about TUG1's function and mechanism in neural healing following ICH. We investigated the potential mechanism of TUG1 inhibition of neural repair after ICH. By downregulating BDNF, TUG1 considerably exacerbated ICH-induced cerebral injury, according to this study. Nevertheless, miR-381-3p in ICH rats showed the opposite effects. Additionally, miR-381-3p and TUG1 share direct intermolecular locations for binding, as does miR-381-3p with BDNF mRNA. TUG1 can directly bind to miR-381-3p and then downregulate miR-381-3p to decrease the production of BDNF, so impeding nerve healing along with exacerbating cerebral injury in a rat model of ICH.

MiR-381-3p has a markedly varied expression in patients with HIV-related neurocognitive impairment [21]. It was found that miR-381-3p contributed to the post-ischemic stroke anti-inflammatory response [7]. In addition, miR-381-3p has been proven to have a significant function in regulating the course of development of glioma, and its overexpression can inhibit the biological function of glioma [5]. According to the results of these studies, miR-381-3p has a role in the pathophysiological mechanisms behind a variety of disorders affecting the central nervous system. Nevertheless, the role of miR-381-3p in ICH remains unknown. In this study, we found that, in contrast to TUG1, miR-381-3p dramatically corrected neurological impairments in rats resulting from ICH. The interaction between lncRNAs and microRNAs is one of the classic mechanisms by which lncRNAs exert their biological effects [12]. Therefore, we investigated whether there is an association between miR-381-3p and TUG1. The direct binding of miR-381-3p to TUG1 had been demonstrated by luciferase reporter assays and bioinformatic predictions. Further studies revealed that TUG1 could effectively downregulate miR-381-3p expression, abolishing the curative properties of miR-381-3p against cerebral damage brought on by ICH.

BDNF, a protein with neurotrophic effects, is widely reported to enhance neuroplasticity and is beneficial to stroke recovery [9]. In vivo studies confirmed that BDNF participates in improving neurological function in mice and rats after ICH [1,11]. The expression level and biological effects of BDNF are regulated by miRNAs; however, different miRNAs have opposite regulatory effects on BDNF [6]. Previous studies have verified that miR-195-3p can positively regulate BDNF expression by selectively attacking the 3' UTR region of BDNF mRNA [24], while miR-584-5p directly binds to BDNF mRNA and downregulates its expression [25]. As of now, there is no report on whether miR-381-3p forms a direct attachment to BDNF mRNA to control its expression. Our research indicates that miR-381-3p mitigates ICH-induced cerebral damage by upregulating BDNF, as evidenced by the considerable increases in BDNF mRNA and protein expression we observed in the cerebral region of ICH rats. Additionally, we discovered that miR-381-3p could be attached to BDNF mRNA directly, and that in ICH rats, overexpressing miR-381-3p raised the expression of BDNF expression.

However, our study has some limitations. First, clinical samples are required to validate our findings from the rat model. Second, more research using human tissue samples is necessary to fully understand the function and mechanism of TUG1 in nerve injury and repair after ICH.

Conclusions

To summarize, our research revealed that TUG1 exacerbated cerebral injury following ICH *via* blocking BDNF expression through sponging miR-381-3p, demonstrating a potential lncRNA/miRNA/mRNA regulatory pathway, which may lead to deeper understanding of the pathological processes behind ICH.

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Disclosures

The study was approved by the Bioethics Committee of the Renmin Hospital of Wuhan University (Approval No. WDRM 2023-K017).

The authors report no conflict of interest.

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