Cell. Mol. Biol. (ISSN: 1165-158X)

Journal Homepage: www.cellmolbiol.org

Cellular and Molecular Biology

Original Article

Effect of whole-process high-quality nursing on the quality of life of acute pancreatitis patients and the mechanism of miR-126-5p/HOXC8 axis promoting the progression of acute pancreatitis

Zhao Shen[1,*], Qingmei Wang[2], Ru Jiao[1], Yehong Liu[1], Tingting Meng[1], Yunhui Fei[1], Caixia Liu[1]

[1] Department of Gastroenterology, The Affiliated Huaian No.1 People's Hospital of Nanjing Medical University, Huaian, Jiangsu 223300, China

[2] Department of Nursing, The Affiliated Huaian No.1 People's Hospital of Nanjing Medical University, Huaian, Jiangsu 223300, China

Article Info

Article history:

Received: March 25, 2024
Accepted: May 03, 2024
Published: August 31, 2024

Use your device to scan and read the article online

Abstract

The current research was designed to investigate the impact of whole-process high-quality nursing on acute pancreatitis (AP) patients' quality of life as well as the mechanism of miR-126-5p/HOXC8 axis promoting AP progression. One hundred AP patients admitted to our hospital were chosen and separated into control group (CG, n=50) and study group (SG, n=50). The CG took the routine nursing, while the SG adopted the whole-process high-quality nursing. Besides, cerulein (CE) was treated in AR42J cells to establish an experimental model of AP. The proliferation, apoptosis along with inflammation of CE-treated AR42J cells were assessed. The outcomes manifested that in contrast to the CG, the recovery time of bowel sound, the improvement time of abdominal distension, the improvement time of abdominal pain, the exhaust time and the defecation time in the SG presented shorter, the anxiety and depression scores in the SG presented lower, the WHOQOL-100 scores of patients in the SG presented higher in the fields of physiology, psychology, environment and social relations, the total incidence of complications of the SG presented lower, and the total nursing satisfaction of the SG was better. Besides, miR-126-5p presented upregulation in CE-stimulated AR42J cells, and miR-126-5p inhibition increased the proliferation along with repressed the apoptosis and inflammation in CE-stimulated AR42J cells. Moreover, HOXC8 could be the target mRNA of miR-126-5p, and HOXC8 elevation promoted the proliferation along with repressed the apoptosis and inflammation in CE-stimulated AR42J cells. In addition, rescue assays further validated that HOXC8 silence offset the protective impact of miR-126-5p repression on AP cell damage. In conclusion, our study indicated that whole-process high-quality nursing could promote the quality of life of AP patients, and revealed that miR-126-5p inhibition relieved CE-stimulated AR42J cells injury caused by AP via targeting HOXC8. Our study might offer novel insights for AP treatment and nursing.

Keywords: Acute pancreatitis, Apoptosis, HOXC8, Inflammatory response, MiR-126-5p, Whole-process high-quality nursing.

1. Introduction

Acute pancreatitis (AP) is a disease caused by acute inflammation of the pancreas [1]. The pancreas is an organ located in the abdominal cavity whose main function is to secrete digestive enzymes and hormones such as insulin [2]. AP is usually caused by the activation of digestive enzymes inside the pancreas, leading to inflammatory response and tissue damage [3]. The disease is particularly harmful to patients, who often experience pain and discomfort, affecting quality of life. Severe cases can lead to abdominal complications, respiratory problems, and multiple organ dysfunction syndrome [4]. In addition, the pancreas plays a key role in digestion, and pancreatitis can trigger digestive problems such as malabsorption and malnutrition [5]. For patients with AP, in addition to actively implementing symptomatic treatment, it is also necessary to cooperate with scientific nursing intervention to ensure the treatment effect and promote the prognosis of patients [6].

The whole-process high-quality nursing model is a new nursing model that upholds the principle of patient-centered development, which can provide high-quality nursing services in combination with the actual condition and physical along with mental state of patients, so as to alleviate the symptoms of patients as soon as possible and promote their prognosis and rehabilitation [7]. However, the effect of whole-process high-quality nursing on AP patients' quality of life remains unclear.

As reported previously, the severity of AP depends on the degree of pancreatic acinar cell death and inflammatory damage [8]. By regulating inflammatory/anti-inflammatory cytokines, the immune function of the body can be improved, which is conducive to the control of inflammation and improve the prognosis of patients [9]. It has been

* Corresponding author.
E-mail address: 15861789937@163.com (Zhao Shen).
Doi: http://dx.doi.org/10.14715/cmb/2024.70.8.25

found that pancreatic acinar cell apoptosis is increased significantly in AP pathological tissues, and inhibition of pancreatic acinar cell apoptosis can significantly reduce the pathological damage caused by AP [10].

MicroRNAs (miRNAs) belong to small non-coding RNA that primarily pair with the 3'-untranslated region (3'UTR) of target mRNA, and post-transcriptionally degrade or repress mRNA expression [11]. The discovery of miRNA opens up a whole new way to diagnose and treat AP [12]. It has been documented that miR-325-3p alleviates AP via targeting RIPK3 [13]. MiR-146a-5p inhibition enhances AP progression through the TLR9/TRAF6/NLRP3 signaling pathway [14]. Previous literature has unveiled that miR-126-5p presents upregulation in AP patients [15], but its potential along with the mechanism in AP is unknown.

Our research was designed to investigate the impact of whole-process high-quality nursing on AP patients' quality of life along with disclosing the potential and mechanism of miR-126-5p in AP.

2. Materials and methods

2.1. Clinical data

One hundred AP patients admitted to our hospital were chosen to be the research objects, followed by dividing into control group (CG, n=50) and study group (SG, n=50) by random number table method. The CG contained 29 males together with 21 females. The average age was (42.35±3.56) years, ranging 26-70 years. The duration of onset ranged 8-33 hours, with an average duration of (16.52±2.34) hours. The SG contained 30 males together with 20 females. The average age was (42.38±3.62) years, ranging 26-72 years. The duration of onset ranged 8-35 hours, with an average duration of (16.54±2.35) hours. No difference was discovered in general data between 2 groups (P>0.05). Inclusion criteria: (1) AP was confirmed by CT or MRI. (2) Normal consciousness; (3) Patients together with their families gave informed consent to this study and voluntarily signed informed consent. Exclusion criteria: (1) Patients had a history of mental illness; (2) Patients had infectious diseases; (3) Patients had severe complications.

2.2. Nursing methods

The CG took the routine nursing mode, nurses did the basic nursing work and gave the patients symptomatic nursing according to the doctor's advice.

The SG adopted the whole-process high-quality nursing mode, with specific contents as follows: (1) Early admission guidance. After the patient was admitted to the hospital, nurses introduced the actual situation of the hospital and the basic information of the attending physician and the responsible nurse in time to reduce the strangeness of the patient. At the same time, propaganda brochures and videos were used to explain in detail the knowledge related to the disease, the importance of treatment, the precautions in treatment and possible complications. During the explanation, nurses paid attention to the use of easy-to-understand language, avoiding preaching and other ways to ensure that each patient could fully understand and master the relevant knowledge of the disease and treatment.

(2) High-quality psychological guidance. During the treatment of patients, nurses actively communicated with patients many times to understand the psychological and emotional changes of patients at various stages, and gave individual psychological counseling combined with the basic information of patients, personality characteristics, etc., to help patients vent various bad emotions in their hearts and maintain a good attitude during the treatment.

(3) Guidance for the safety of medication. The nurse strictly followed the doctor's advice to administer the drug to the patient. For patients who needed drug sensitivity test before medication, the drug was confirmed by two nurses before medication. At the same time, nurses explained the necessity of drug treatment, usage and dosage, and possible adverse reactions to patients, and urged them to report abnormalities in time. In addition, patients' vital signs were closely monitored during medication, appropriate infusion speed was adopted according to drug differences, and any abnormalities were reported to the doctor in time and treated with cooperation.

(4) High-quality dietary guidance. For patients with acute attacks, water was absolutely forbidden as soon as possible, and intravenous channels were established in time to give nutritional support. For severe patients, nurses did a good job of long-term fasting and did a good job of supporting treatment such as jejunal nutrition tube. Convalescent patients, they needed to gradually change from oil-free light liquid diet to vegan semi-liquid diet, mainly with small and frequent meals, more intake of protamine, restriction of fat consumption, strict prohibition of alcohol and overeating, such as abdominal pain during eating, need to change back to vegan diet.

(5) Strengthening catheter management. Nurses strengthened the observation of each indwelling pipe in patients to avoid bending, blocking, falling off and other phenomena, and recorded the amount, nature, and color of each drainage fluid in patients in detail to avoid serious complications caused by abnormal circumstances.

(6) High-quality discharge guidance. According to the differences in patients' self-care ability, individualized out-of-hospital extended care was provided. Through WeChat, QQ, telephone, home visits and other means, patients had a comprehensive understanding of their psychological state, rehabilitation process, lifestyle, and eating habits after discharge, and all kinds of problems were answered and dealt with in a timely manner to ensure that patients recovered thoroughly after discharge.

2.3. Observation indicators

1) The improvement time of various symptoms in 2 groups was compared, including the recovery time of bowel sound, the improvement time of abdominal distension, the improvement time of abdominal pain, the exhaust time and the defecation time.

2) The anxiety and depression were evaluated by SAS scale and SDS scale respectively. The former was graded on 20 items according to 1-4 points. The latter items 20, 1 to 4 points, high scores indicated that anxiety and depression were more serious.

3) The quality of life after nursing was compared between 2 groups, and the World Health Organization Quality of Life Scale (WHOQOL-100) was used to score the quality of life after nursing. The higher the score, the better the quality of life was.

4) The incidence of complications was compared between 2 groups, including lung infection, lung injury, pancreatic abscess and pressure sore.

5) A simple self-made questionnaire was adopted to evaluate nursing satisfaction, including nursing attitude, operation skills, education effect, etc., with a total of 100 points. The scoring range <70, 70-90, >90 corresponds to dissatisfaction, general satisfaction, satisfaction, and total satisfaction = (satisfaction + general satisfaction) cases/total number of cases.

2.4. Cell culture and treatment

Procell (Wuhan, China) offered rat pancreatic acinar cell line AR42J, which was cultured in Ham's F-12K medium which contained 20% fetal bovine serum plus 1% penicillin and streptomycin at 37 ℃ and 5% CO_2. After 1 week, the cells grew to about 80% of the fusion, digested with trypsin for about 1 min, and passed at the ratio of 1:2 ~ 1:4. AR42J cells were stimulated with 10 nmol/L cerulein (CE) to induce AP cell model.

2.5. Cell transfection

MiR-126-5p mimics/inhibitor or NC mimics/inhibitor was acquired from Ribobio (Guangzhou, China), and shRNAs against HOXC8 or sh-RNA as well as pcDNA3.1-HOXC8 or pcDNA3.1 was acquired from GenePharma (Shanghai, China). Subsequently, these plasmids were transfected into AR42J cells with Lipofectamine 2000 (Invitrogen, USA).

2.6. RT-qPCR

Total RNA could be extracted from cells by TRIzol reagent (Invitrogen, USA), followed by reverse transcription into cDNA using PrimeScript RT Reagent Kit (Takara, Japan). Real-time PCR was implemented with SYBR Premix Ex Taq II (Takara, Japan). GAPDH and U6 could be adopted to be internal controls for calculating mRNA and miRNA based on the $2^{-\Delta\Delta CT}$ method.

2.7. Western blot

Total protein could be isolated from cells with RIPA buffer (Invitrogen, USA) containing 1% protease inhibitor. After measuring the concentration, the protein samples could be separated by 12% SDS-PAGE and then transferred onto PVDF membranes. The membranes were sealed with 5% skimmed milk powder for 1 h, followed by cultivating with primary antibodies including HOXC8 and GAPDH at 4°C overnight. After washing, the membranes were cultivated with secondary antibodies for 1 h at room temperature. At last, ECL was added to develop the signals.

2.8. CCK-8

AR42J cells (5×10^3 cells/well) were placed on 96-well plates for cultivation at 37°C for 24 h, 48 h, and 72 h. The cells were then added with 10 μL CCK-8 reagent at 37°C for 2 h. The optical density (OD) values were measured at 450 nm with a microplate reader.

2.9. Flow cytometry

The treated AR42J cells were digested with trypsin, and washed, followed by suspending in the binding buffer (5×10^5 cells /mL). Subsequently, the cells (200 μL cell suspension) received treatment with 10 μL Annexin V-FITC as well as 10 μL PI for 30 min away from light. The apoptosis rate could be analyzed by flow cytometry.

2.10. ELISA

The treated AR42J cell supernatant was harvested by centrifugation (500 × g, 4°C, 10 min). IL-1β and TNF-α levels were analyzed with an ELISA kit, and OD values for each well were measured at 450 nm on an enzyme-labeler according to kit instructions.

2.11. Luciferase reporter assay

The binding sites between miR-126-5p and HOXC8 were predicted by starBase (http://starbase.sysu.edu.cn/). Accordingly, wild-type HOXC8 (HOXC8 3'UTR-Wt) or mutant HOXC8 (HOXC8 3'UTR-Mut) plasmid was designed. AR42J cells were planted on a 24-well culture plate and grew to a fusion rate of 80%. Then, AR42J cells were co-transfected with HOXC8 3'UTR-Wt or HOXC8 3'UTR-Mut plasmid as well as miR-126-5p mimics or its negative control NC mimics using Lipofectamine 2000 reagent. Firefly luciferase activity and sea kidney luciferase activity were measured 48 h after transfection. Renal luciferase activity was adopted as an internal control. Luciferase activity = firefly luciferase activity/sea kidney luciferase activity.

2.12. Statistical analysis

SPSS 24.0 statistical software was adopted for data analysis. Measurement data were expressed as (x±s), and t-test was adopted for comparison between 2 groups. One-way analysis of variance was adopted for comparison among multiple groups, and SNK-q test was adopted for further pairwise comparison. Count data were expressed as (n, %), and χ^2 test was adopted for comparison. $P<0.05$ meant statistical significance.

3. Results

3.1. Effect of whole-process high-quality nursing AP patients' quality of life

In contrast to the CG, the recovery time of bowel

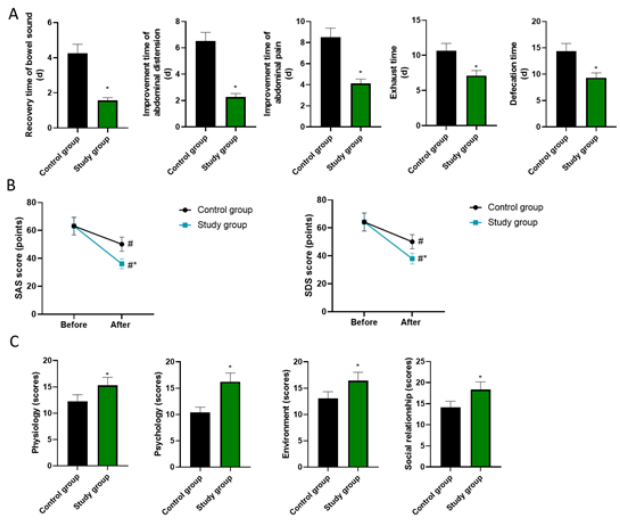

Fig. 1. Effect of whole-process high-quality nursing on the quality of life of patients with AP. (A) Comparison of the recovery time of bowel sound, the improvement time of abdominal distension, the improvement time of abdominal pain, the exhaust time and the defecation time between 2 groups. (B) Comparison of SAS and SDS scores between 2 groups. (C) Comparison of WHOQOL-100 scores in each dimension between 2 groups. Compared with before nursing, #P<0.05, compared with control group, *P<0.05.

sound, the improvement time of abdominal distension, the improvement time of abdominal pain, the exhaust time and the defecation time in the SG presented shorter (P<0.05, Fig. 1A). No difference was seen in anxiety and depression scores between 2 groups prior to nursing (P>0.05). After nursing, the anxiety and depression scores declined in 2 groups, and those in the SG presented lower when comparing with the CG (P<0.05, Fig. 1B). After nursing, the WHOQOL-100 scores of patients in the SG presented higher when comparing with the CG in the fields of physiology, psychology, environment and social relations (P<0.05, Fig. 1C). The total incidence of complications of the SG presented lower when comparing with the CG (P<0.05, Table 1). The total nursing satisfaction of the SG was better than that of the CG (P<0.05, Table 2).

3.2. MiR-126-5p inhibition promotes the proliferation along with represses the apoptosis and inflammation in CE-stimulated AR42J cells

It was revealed in RT-qPCR that miR-126-5p presented high expression in AR42J cells after CE treatment (Fig. 2A). To assess the function of miR-126-5p in CE-stimulated AR42J cells, miR-126-5p inhibitor was transfected into AR42J cells to silence miR-126-5p expression. As presented in Figure 2B, miR-126-5p inhibitor markedly weakened miR-126-5p expression in CE-induced AR42J cells (Fig. 2B). Afterwards, the impacts of miR-126-5p on CE-stimulated AR42J cells viability, apoptosis, along with inflammatory reaction were assessed. CCK-8 assay revealed that AR42J cells viability was weakened after CE treatment, whereas after co-transfection with miR-126-5p inhibitor, the AR42J cells viability could be promoted (Fig. 2C). Moreover, the flow cytometry assay showed that AR42J cells apoptosis was elevated after CE treatment, whereas after miR-126-5p silence, AR42J cells apoptosis was reduced (Fig. 2D). In addition, IL-1β and TNF-α levels could be elevated in CE-induced AR42J cells, but their levels were lessened after silencing miR-126-5p (Fig. 2E).

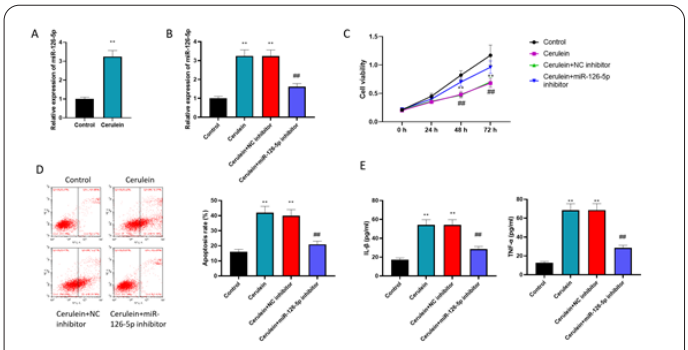

Fig. 2. MiR-126-5p inhibition promotes the proliferation along with represses the apoptosis and inflammatory response in AR42J cells treated with CE. (A) RT-qPCR examined miR-126-5p expression in AR42J cells treated with CE. (B) RT-qPCR examined the transfection efficiency of miR-126-5p inhibitor in AR42J cells treated with CE. (C) CCK-8 examined the viability in AR42J cells treated with CE after miR-126-5p inhibition. (D) Flow cytometry analysis assessed the apoptosis in AR42J cells treated with CE after miR-126-5p inhibition. (E) ELISA examined the levels of IL-1β and TNF-α in AR42J cells treated with CE after miR-126-5p inhibition. **P<0.01, compared with control, $^{\#\#}$P<0.01, compared with CE.

3.3. HOXC8 is the target mRNA of miR-126-5p

Using starBase website, we predicted that HOXC8 was the target mRNA of miR-126-5p, and the binding sites between HOXC8 3'UTR and miR-126-5p were displayed in Fig. 3A. We then overexpressed miR-126-5p expression in CE-stimulated AR42J cells, and found that after trans-

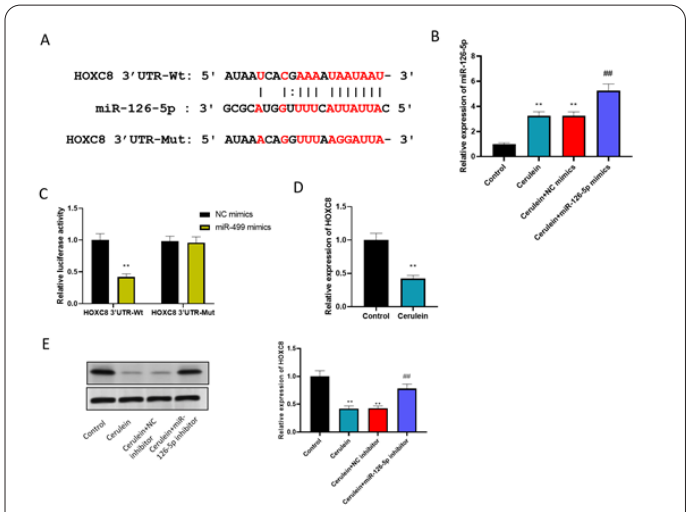

Fig. 3. HOXC8 is the target gene of miR-126-5p. (A) Binding sites between HOXC8 3'UTR and miR-126-5p. (B) RT-qPCR examined the transfection efficiency of miR-126-5p mimics in AR42J cells treated with CE. (C) Luciferase reporter assay assessed the impacts of miR-126-5p mimics on the luciferase activity of HOXC8 3'UTR-Wt/Mut in AR42J cells treated with CE. (D) RT-qPCR examined HOXC8 expression in AR42J cells treated with CE. (E) RT-qPCR and western blot examined HOXC8 expression in AR42J cells treated with CE after miR-126-5p inhibition. **P<0.01, compared with control, $^{\#\#}$P<0.01, compared with cerulein.

Table 1. Incidence of complications in 2 groups.

Groups	N	Lung infection	Lung injury	Pancreatic abscess	Pressure sore	Total incidence rate
Control group	50	3	1	2	2	9 (18.00%)
Study group	50	1	0	0	0	1 (2.00%)
χ^2						7.111
P						0.007

Table 2. Nursing satisfaction in 2 groups.

Groups	N	Satisfaction	General satisfaction	Dissatisfaction	Total satisfaction rate
Control group	50	24	16	10	40 (80.00%)
Study group	50	30	18	2	48 (96.00%)
χ^2					6.061
P					0.013

fection of miR-126-5p mimics, miR-126-5p expression was obviously elevated in CE-induced AR42J cells (Fig. 3B). Subsequently, luciferase reporter assay results manifested that miR-126-5p elevation apparently lessened the luciferase activity of HOXC8 3'UTR-Wt, but barely impacted HOXC8 3'UTR-Mut activity (Fig. 3C). Moreover, it was observed that HOXC8 was low-expressed in CE-stimulated AR42J cells (Fig. 3D). More importantly, after miR-126-5p inhibition, HOXC8 mRNA and protein levels could be elevated in CE-stimulated AR42J cells (Fig. 3E).

3.4. HOXC8 elevation promotes the proliferation along with represses the apoptosis and inflammation in CE-stimulated AR42J cells

We further elevated HOXC8 expression in CE-stimulated AR42J cells to certify the impacts of HOXC8 on AR42J cells proliferation, apoptosis along with inflammatory response after CE treatment. RT-qPCR validated the successful transfection of pcDNA3.1-HOXC8 (Fig. 4A). As revealed in Fig. 4B-4D, elevated expression of HOXC8 definitely increased the proliferation, reduced the apoptosis as well as IL-1β and TNF-α levels in CE-induced AR42J cells.

3.5. HOXC8 silence reverses the protective potential of miR-126-5p repression on cell injury caused by AP

Furthermore, we silenced HOXC8 expression in CE-stimulated AR42J cells, and then carried out rescue assays to prove the interaction between miR-126-5p and HOXC8 in AP. Firstly, RT-qPCR validated the successful transfection of shRNAs targeting HOXC8. Since that sh-HOXC8#1 had better inhibiting effect on HOXC8 expression in AR42J cells treated with CE, sh-HOXC8#1 was selected for follow-up rescue experiments (Fig. 5A). It was discovered that HOXC8 silence reversed the elevated proliferation, suppressed apoptosis along with IL-1β and TNF-α levels in CE-induced AR42J cells (Fig. 5B-5D).

4. Discussion

AP belongs to a disease caused by acute inflammation of the pancreas. The main symptoms include severe pain in the upper abdomen, nausea, vomiting, as well as fever [16]. The most common causes belong to cholelithiasis along with alcohol abuse, while other causes contain hyperlipidemia, infection, and pancreatic duct stones [17]. The disease can bring many harms, the first is severe upper abdominal pain, which not only influences the patient's quality of life but also can result in loss of appetite, nausea, vomiting and other digestive symptoms [18]. AP may also cause pancreatic necrosis and damage pancreatic tissue, which in turn affects the production of digestive enzymes and has a long-term impact on the normal digestion and absorption of food [19]. In extreme cases, AP may lead to multiple organ dysfunction syndrome, endangering patients' lives [20]. Therefore, for patients with AP, active treatment and effective nursing are essential to reduce symptoms, prevent complications, and improve the recovery rate of patients [21].

The whole-process high-quality nursing model belongs to a modern and new nursing model, and the patient-centered nursing service concept runs through the nursing work all the time, so as to provide patients with all-round, multi-level, continuous and dynamic, systematic nursing, which can conform to the clinical requirements of patients in physiological and psychological aspects [22].

In our study, the results revealed that in contrast to the CG, the recovery time of bowel sound, the improvement time of abdominal distension, the improvement time of abdominal pain, the exhaust time and the defecation time in the SG presented shorter, the anxiety and depression scores in the SG presented lower, the WHOQOL-100 scores of patients in the SG presented higher in the fields of physiology, psychology, environment and social relations, the total incidence of complications of the SG presented lower, and the total nursing satisfaction of the SG was better. All these findings suggested that the whole-process high-quality nursing could promote the recovery of intestinal function, relieve negative emotions, promote the quality of life, decrease the occurrence of complications along with elevate the nursing satisfaction of AP patients, which was in line with previous studies [23, 24].

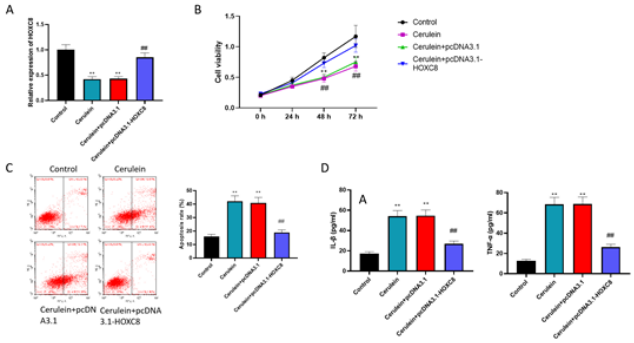

Fig. 4. HOXC8 elevation promotes the proliferation along with represses the apoptosis and inflammatory response in AR42J cells treated with CE. (A) RT-qPCR examined the transfection efficiency of pcDNA31-HOXC8 in AR42J cells treated with CE. (B) CCK-8 examined the viability in AR42J cells treated with CE after HOXC8 elevation. (D) Flow cytometry analysis assessed the apoptosis in AR42J cells treated with CE after HOXC8 elevation. (E) ELISA examined the levels of IL-1β and TNF-α in AR42J cells treated with CE after HOXC8 elevation. **P<0.01, compared with control, $^{\#\#}$P<0.01, compared with cerulein.

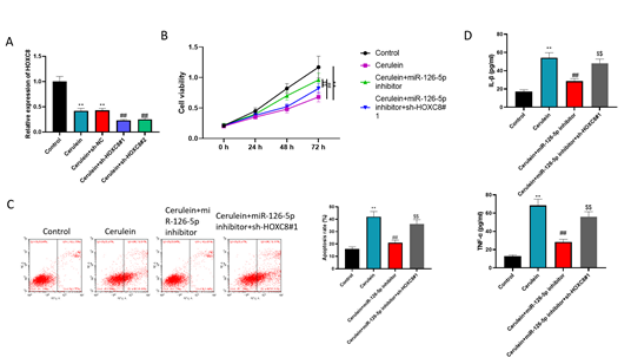

Fig. 5. HOXC8 silence reverses the protective effect of miR-126-5p inhibition on cell injury caused by AP. (A) RT-qPCR examined the transfection efficiency of sh-HOXC8#1/#2 in AR42J cells treated with CE. (B) CCK-8 examined the viability in AR42J cells treated with CE in different groups. (D) Flow cytometry analysis assessed the apoptosis in AR42J cells treated with CE in different groups. (E) ELISA examined the levels of IL-1β and TNF-α in AR42J cells treated with CE in different groups. **P<0.01, compared with control, $^{\#\#}$P<0.01, compared with cerulein, $^{\$\$}$P<0.01, compared with cerulein+miR-126-5p inhibitor

Studies have affirmed that miRNAs have crucial functions in multiple diseases, AP included [25]. In this research, we assessed the potential along with mechanism of miR-126-5p in AP, and the outcomes manifested that miR-126-5p targeted HOXC8 to promote inflammation and cell apoptosis in AP cell model.

As reported previously, CE-stimulated AP belongs to one of the best characteristics along with extensive use of several experimental models [26]. Similarly, we treated AR42J cells with CE to build AP model in vitro. It was discovered that CE inhibited AR42J cell proliferation, promoted AR42J cell apoptosis, along with enhanced AR42J cell inflammation.

Recent literatures have uncovered that miR-126-5p is abnormally expressed in many diseases. Such as, Ren et al. have pointed out that miR-126-5p affects H9c2 cell viability along with apoptosis under hypoxia [27]. Besides, Chen et al. have discovered that miR-126-5p expression is promoted in severe AP patients [28], but its role in AP remains unclear. Here, miR-126-5p presented high expression in CE-stimulated AR42J cells, and miR-126-5p suppression markedly promoted the proliferation, along with repressed the apoptosis and inflammation in CE-induced AR42J cells. In accordance with our outcomes, Liao et al. have proposed that miR-126-5p is upregulated in hypoxia-treated umbilical vein endothelial cells (HUVECs), and miR-126-5p knockdown suppressed hypoxia-induced cell apoptosis and inflammatory response in HUVECs [29].

The HOXC8 is a member of the homeobox class I family [30], and it has been reported that HOXC8 expression is decreased in pancreatic ductal adenocarcinoma, and HOXC8 inhibits pancreatic ductal adenocarcinoma progression and metastasis [31]. Nevertheless, its expression and potential in AP presents unclear. In the present research, it was confirmed that HOXC8 belonged to the target mRNA of miR-126-5p, and HOXC8 presented downregulation in CE-stimulated AR42J cells. Subsequent gain-of-function assays indicated HOXC8 elevation promoted the proliferation along with repressed apoptosis and inflammation in CE-stimulated AR42J cells. Moreover, our study performed rescue assays to validate that HOXC8 silence reverses the protective potential of miR-126-5p downregulation on cell injury caused by AP.

5. Conclusion

In conclusion, our study indicated that whole-process high-quality nursing could promote the quality of life of AP patients, and revealed that miR-126-5p repression relieved CE-induced AR42J cells injury caused by AP via targeting HOXC8. Our results might offer novel insights for AP treatment and nursing.

Conflict of interests

The authors declare no competing interests.

Consent for publications

The author read and approved the final manuscript for publication.

Ethics approval and consent to participate

We have received approval from the Ethics Committee of The Affiliated Huaian No.1 People's Hospital of Nanjing Medical University.

Informed consent

We have received informed consent from the Ethics Committee of The Affiliated Huaian No.1 People's Hospital of Nanjing Medical University.

Availability of data and material

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Authors' contributions

SZ contributed to the study conception and design. Clinical data collection and analysis were performed by WQ, JR and LY. Basic experimental operation, data collection and analysis were performed by MT, FY and LC. The first draft of the manuscript was written by SZ and all authors commented on previous versions of the manuscript.

Funding

Not applicable.

Acknowledgements

Not applicable.

References

1. Mederos MA, Reber HA, Girgis MD (2021) Acute Pancreatitis: A Review. Jama 325 (4): 382-390. doi: 10.1001/jama.2020.20317
2. Lankisch PG, Apte M, Banks PA (2015) Acute pancreatitis. Lancet 386 (9988): 85-96. doi: 10.1016/s0140-6736(14)60649-8
3. Greenberg JA, Hsu J, Bawazeer M, Marshall J, Friedrich JO, Nathens A, Coburn N, May GR, Pearsall E, McLeod RS (2016) Clinical practice guideline: management of acute pancreatitis. Can J Surg 59 (2): 128-140. doi: 10.1503/cjs.015015
4. Wang GJ, Gao CF, Wei D, Wang C, Ding SQ (2009) Acute pancreatitis: etiology and common pathogenesis. World J Gastroenterol 15 (12): 1427-1430. doi: 10.3748/wjg.15.1427
5. Arvanitakis M, Ockenga J, Bezmarevic M, Gianotti L, Krznarić Ž, Lobo DN, Löser C, Madl C, Meier R, Phillips M, Rasmussen HH, Van Hooft JE, Bischoff SC (2020) ESPEN guideline on clinical nutrition in acute and chronic pancreatitis. Clin Nutr 39 (3): 612-631. doi: 10.1016/j.clnu.2020.01.004
6. Johnstone C (2018) Pathophysiology and nursing management of acute pancreatitis. Nurs Stand. doi: 10.7748/ns.2018.e11179
7. Mei L, Xu Y, Shi Q, Wu C (2022) Application Effect and Prognosis of High-Quality Nursing in the Whole Process of Nursing in Lung Cancer Surgery. Evid Based Complement Alternat Med 2022: 9491559. doi: 10.1155/2022/9491559
8. Ma N, Yuan C, Shi J, Zhu Q, Liu Y, Ma X, Li B, Gong W, Xue J, Lu G, Li W, Li J (2022) Interleukin-37 protects against acinar cell pyroptosis in acute pancreatitis. JCI Insight 7 (21). doi: 10.1172/jci.insight.161244
9. Tan JH, Cao RC, Zhou L, Zhou ZT, Chen HJ, Xu J, Chen XM, Jin YC, Lin JY, Zeng JL, Li SJ, Luo M, Hu GD, Yang XB, Jin J, Zhang GW (2020) ATF6 aggravates acinar cell apoptosis and injury by regulating p53/AIFM2 transcription in Severe Acute Pancreatitis. Theranostics 10 (18): 8298-8314. doi: 10.7150/thno.46934
10. Bhatia M (2004) Apoptosis of pancreatic acinar cells in acute pancreatitis: is it good or bad? J Cell Mol Med 8 (3): 402-409. doi: 10.1111/j.1582-4934.2004.tb00330.x
11. Hill M, Tran N (2021) miRNA interplay: mechanisms and consequences in cancer. Dis Model Mech 14 (4). doi: 10.1242/dmm.047662
12. Yang Y, Huang Q, Luo C, Wen Y, Liu R, Sun H, Tang L (2020) MicroRNAs in acute pancreatitis: From pathogenesis to novel

diagnosis and therapy. J Cell Physiol 235 (3): 1948-1961. doi: 10.1002/jcp.29212
13. Jia A, Yang ZW, Shi JY, Liu JM, Zhang K, Cui YF (2022) MiR-325-3p Alleviates Acute Pancreatitis via Targeting RIPK3. Dig Dis Sci 67 (9): 4471-4483. doi: 10.1007/s10620-021-07322-6
14. Deng D, Su Z, Wei B, Zhou J, Yang H, Liang Z (2022) Downregulation of miR-146a-5p Promotes Acute Pancreatitis through Activating the TLR9/NLRP3 Signaling Pathway by Targeting TRAF6 In Vitro Rat Model. Comput Math Methods Med 2022: 1747470. doi: 10.1155/2022/1747470
15. Kuśnierz-Cabala B, Nowak E, Sporek M, Kowalik A, Kuźniewski M, Enguita FJ, Stępień E (2015) Serum levels of unique miR-551-5p and endothelial-specific miR-126a-5p allow discrimination of patients in the early phase of acute pancreatitis. Pancreatology 15 (4): 344-351. doi: 10.1016/j.pan.2015.05.475
16. Lee PJ, Papachristou GI (2019) New insights into acute pancreatitis. Nat Rev Gastroenterol Hepatol 16 (8): 479-496. doi: 10.1038/s41575-019-0158-2
17. Gardner TB (2021) Acute Pancreatitis. Ann Intern Med 174 (2): Itc17-itc32. doi: 10.7326/aitc202102160
18. van Dijk SM, Hallensleben NDL, van Santvoort HC, Fockens P, van Goor H, Bruno MJ, Besselink MG (2017) Acute pancreatitis: recent advances through randomised trials. Gut 66 (11): 2024-2032. doi: 10.1136/gutjnl-2016-313595
19. Mandalia A, Wamsteker EJ, DiMagno MJ (2018) Recent advances in understanding and managing acute pancreatitis. F1000Res 7. doi: 10.12688/f1000research.14244.2
20. Zerem E (2014) Treatment of severe acute pancreatitis and its complications. World J Gastroenterol 20 (38): 13879-13892. doi: 10.3748/wjg.v20.i38.13879
21. Krenzer ME (2016) Understanding acute pancreatitis. Nursing 46 (8): 34-40. doi: 10.1097/01.Nurse.0000484959.78110.98
22. Ruppar TM (2021) High-Quality Reviews Advance Cardiovascular Nursing Science. J Cardiovasc Nurs 36 (1): 2-3. doi: 10.1097/jcn.0000000000000775
23. Wang M, Sun Y, Zhang M, Yu R, Fu J (2022) Effects of high-quality nursing care on quality of life, survival, and recurrence in patients with advanced nonsmall cell lung cancer. Medicine (Baltimore) 101 (37): e30569. doi: 10.1097/md.0000000000030569
24. Fu SL, Sun CH, Shang XX, Liu XS (2019) High quality nursing of children with pneumonia complicated with heart failure. J Biol Regul Homeost Agents 33 (3): 905-910.
25. Xu Y, Sun Y, Yin R, Dong T, Song K, Fang Y, Liu G, Shen B, Li H (2022) Differential expression of plasma exosomal microRNA in severe acute pancreatitis. Frontiers in pharmacology 13: 980930. doi: 10.3389/fphar.2022.980930
26. Jancsó Z, Sahin-Tóth M (2022) Chronic progression of cerulein-induced acute pancreatitis in trypsinogen mutant mice. Pancreatology : official journal of the International Association of Pancreatology (IAP) ... [et al.] 22 (2): 248-257. doi: 10.1016/j.pan.2022.01.007
27. Ren Y, Bao R, Guo Z, Kai J, Cai CG, Li Z (2021) miR-126-5p regulates H9c2 cell proliferation and apoptosis under hypoxic conditions by targeting IL-17A. Experimental and therapeutic medicine 21 (1): 67. doi: 10.3892/etm.2020.9499
28. Chen YJ, Lin TL, Cai Z, Yan CH, Gou SR, Zhuang YD (2021) Assessment of acute pancreatitis severity via determination of serum levels of hsa-miR-126-5p and IL-6. Experimental and therapeutic medicine 21 (1): 26. doi: 10.3892/etm.2020.9458
29. Liao Y, Zou Y, Zhang H (2022) MicroRNA-126-5p Facilitates Hypoxia-Induced Vascular Endothelial Cell Injury via HIPK2. Annals of clinical and laboratory science 52 (6): 918-926.
30. Tsai CY, Liao JB, Lee YC, Yang YF (2023) HOXC8 mediates osteopontin expression in gastric cancer cells. Journal of Cancer 14 (13): 2552-2561. doi: 10.7150/jca.84460
31. Adwan H, Zhivkova-Galunska M, Georges R, Eyol E, Kleeff J, Giese NA, Friess H, Bergmann F, Berger MR (2011) Expression of HOXC8 is inversely related to the progression and metastasis of pancreatic ductal adenocarcinoma. British journal of cancer 105 (2): 288-295. doi: 10.1038/bjc.2011.217