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Research Progress of Traditional Chinese Medicine in the Prevention and Treatment of Myocardial Ischemia-reperfusion Injury by Regulating BNIP3 Pathway

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Abstract: ***Myocardial ischemia-reperfusion injury (MIRI) is a major challenge in the treatment of cardiovascular diseases. The pathogenesis of MIRI is complex and involves a variety of signaling pathways and molecular mechanisms. In recent years, BNIP3 (Bcl-2/adenovirus E1B 19kDa protein-interacting protein 3), as a key molecule regulating mitochondrial function and apoptosis, has attracted more and more attention in MIRI. This article discusses the regulation of BNIP3 pathway, inhibition of over-activation of HIF-1α/BNIP3 signalaxis, fine regulation of mitophagy flow, inhibition of BNIP3 mediated mitochondrial pathway apoptosis, and anti-oxidation and anti-inflammatory effects, so as to aleviate MIRI.***

Keywords: Myocardial ischemia-reperfusion injury, BNIP3, Mitophagy, Apoptosis, Oxidative stress, Mechanisms.

1. Introduction

Myocardial ischemia-reperfusion injury (MIRI) is a common complication of cardiovascular diseases, with a complex pathogenesis. The core mechanisms involve oxidative stress burst, calcium overload, inflammatory response, and mitochondrial dysfunction [1]. As a member of Bc1-2 family, BNIP3 is a pro-apoptotic protein, which plays an important role in the process of myocardial ischemia-reperfusion injury. Studies have shown that overexpression of BNIP3 can induce mitophagy, which in turn promotes the apoptosis of cardiomyocytes [2]. From the perspective of traditional Chinese medicine theory, MIRI can be classified as “re-injury” after “chest impediment (Xiong Bi)”. The key to the pathogenesis is that qi deficiency and blood stasis run through the whole process, and the sudden deficiency of “vital qi” and hyperactivity of “blood stasis and toxin” coexist after reperfusion. Traditional Chinese medicine (TCM) shows unique advantages in overall regulation and multi-target intervention through the treatment of “invigorating qi and activating blood circulation, detoxifying collaterals” and other methods. In recent years, more and more studies have focused on the regulation of BNIP3 pathway by traditional Chinese medicine, in order to find a new strategy for the prevention and treatment of myocardial ischemia-reperfusion injury. Studies have found that traditional Chinese medicine can affect the expression and function of BNIP3 through a variety of ways, thereby reducing the injury in the process of myocardial ischemia-reperfusion [3]. On the one hand, TCM can regulate BNIP3-mediated mitophagy to avoid cardiomyocyte death caused by excessive autophagy [4]. On the other hand, TCM can also inhibit BNIP3-mediated apoptosis and protect cardiomyocytes from injury. In addition, the antioxidant and anti-inflammatory effects of TCM are also closely related to its regulation of BNIP3 pathway, which together constitute multiple mechanisms of TCM in the prevention and treatment of myocardial ischemia-reperfusion injury.

2. Overview of BNIP3

BNIP3, whose full name is Bcl-2/adenovirus E1B 19kDa interacting protein 3, is a protein located on the outer membrane of mitochondria. It plays a crucial role in regulating apoptosis and mitochondrial autophagy. As a downstream target gene of hypoxia-inducible factor-1α (HIF-1a), BNIP3 is strongly induced under ischemic and hypoxic environment [5]. At moderate levels, the removal of damaged mitochondria is a protective mechanism. However, under the acute stress of MIRI, it often leads to excessive or uncontrolled mitophagy, but instead promotes cell death and induces mitophagy. Apoptosis that initiates the mitochondrial pathway will cause mitochondrial membrane permeabilization (MOMP), release cytochrome C, and lead to caspase cascade activation. Damaged mitochondria itself is a source of ROS, which promotes the generation of ROS and forms a vicious cycle.

3. Role of BNIP3 in MIRI

3.1 Suppress the Overactivation of the HIF-1α/BNIP3 Signaling Axis

During myocardial ischemia-reperfusion, the excessive activation of HIF-1α induces the overexpression of BNIP3, subsequently triggering a series of adverse reactions [6]. Traditional Chinese medicine, through its unique pharmacological effects, can inhibit the excessive activation of HIF-1α, thereby downregulating the expression level of BNIP3 [7]. This regulatory mechanism helps mitigate the excessive occurrence of mitophagy, preventing myocardial cells from being damaged due to excessive autophagy. Meanwhile, by inhibiting BNIP3-mediated mitochondrial pathway apoptosis. Traditional Chinese medicine can also effectively reduce the number of apoptotic cardiomyocytes, further protecting myocardial tissue from reperfusion injury. Research shows [7], Certain monomers or compounds of traditional Chinese medicine exhibit significant inhibitory

effects on the HIF-1α/BNIP3 signaling axis, providing a scientific basis for the prevention and treatment of myocardial ischemia-reperfusion injury with traditional Chinese medicine.

3.2 Precise Regulation of Mitochondrial Autophagy Flux

Mitochondrial autophagy is a complex physiological process involving multiple steps such as mitochondrial recognition, encapsulation, separation, and degradation [8]. Traditional Chinese medicine can achieve fine regulation of mitochondrial autophagy flux by regulating BNIP3 and related signaling pathways. Moderate mitophagy helps to eliminate damaged mitochondria and maintain cellular homeostasis. Excessive mitophagy may lead to cardiomyocyte death. Traditional Chinese medicine can balance the level of mitophagy, effectively eliminating harmful mitochondrial fragments while preventing damage caused by excessive autophagy. In addition, traditional Chinese medicine can also regulate other signaling pathways related to mitophagy, such as the Pink1/Parkin pathway. Further enhance the regulatory ability over mitochondrial autophagy flux [9]. This multi-target regulatory approach endows traditional Chinese medicine with unique advantages in preventing and treating myocardial ischemia-reperfusion injury.

3.3 Inhibiting BNIP3-mediated Mitochondrial Pathway Apoptosis

During myocardial ischemia-reperfusion injury, the overexpression of BNIP3 is one of the key factors triggering mitochondrial pathway apoptosis. Traditional Chinese medicine, through its pharmacological effects of multi-component and multi-target, can effectively inhibit the pro-apoptotic activity of BNIP3, thereby reducing the degree of cardiomyocyte apoptosis. This regulatory mechanism is mainly realized through the following approaches: Firstly, traditional Chinese medicine can downregulate BNIP3 expression, diminish its binding to the outer mitochondrial membrane, and consequently inhibit mitochondrial membrane permeabilization and the release of cytochrome C. Secondly, traditional Chinese medicine can also modulate signaling pathways associated with BNIP3, such as the expression of other Bcl-2 family members, thereby enhancing cardiomyocytes' resistance to apoptotic signals. In addition, some active ingredients in traditional Chinese medicine (TCM) exhibit direct antioxidant and anti-inflammatory effects, which can alleviate oxidative stress and inflammatory responses during myocardial ischemia-reperfusion, further protecting myocardial cells from the threat of apoptosis. Through these comprehensive regulatory mechanisms, TCM demonstrates broad application prospects in preventing and treating myocardial ischemia-reperfusion injury.

4. Regulating BNIP3 Pathway with Traditional Chinese Medicine for the Treatment of Myocardial Ischemia-reperfusion Injury (MIRI)

4.1 Monomer of Chinese Materia Medica

4.1.1 Phenolic compounds

Phenolic compounds are a class of compounds with a phenolic hydroxyl structure, which are widely present in various traditional Chinese medicines. Research has shown that phenolic compounds play a significant role in regulating the BNIP3 pathway. They can reduce the induced expression of BNIP3 by inhibiting the activity of HIF-1α, thereby alleviating mitochondrial autophagy and apoptosis during myocardial ischemia-reperfusion. In addition, phenolic compounds exhibit potent antioxidant properties, capable of scavenging free radicals and mitigating the damage inflicted by oxidative stress on myocardial cells.

Curcumin, as a typical phenolic compound, is widely present in traditional Chinese medicines such as turmeric. Research has shown that curcumin exhibits significant antioxidant and anti-inflammatory effects, capable of alleviating oxidative stress and inflammatory responses during myocardial ischemia-reperfusion [10]. More significantly, curcumin can effectively suppress the occurrence of mitochondrial autophagy and apoptosis by inhibiting HIF-1αactivity and downregulating BNIP3 expression levels [10]. This discovery provides a scientific basis for the application of curcumin in the prevention and treatment of myocardial ischemia-reperfusion injury. In addition to curcumin, other phenolic compounds such as quercetin and resveratrol have also been proven to regulate the BNIP3 pathway [11] [12].

4.1.2 Saponins

Saponins are a class of compounds with glycosidic bond structures, primarily found in traditional Chinese medicines such as ginseng and astragalus. Research has shown that saponins also exhibit a significant role in regulating the BNIP3 pathway [6]. They can act on signaling molecules upstream of BNIP3, such as by inhibiting the activity of HIF-1α to reduce the induced expression of BNIP3, thereby inhibiting mitochondrial autophagy and apoptosis. In addition, saponin compounds also have the effect of enhancing the antioxidant capacity of cardiomyocytes, which can alleviate oxidative stress damage during myocardial ischemia-reperfusion.

Ginsenoside Rg1, Rb1, and panaxsaponin, as one of the main active ingredients in Panax ginseng and Panax quinquefolium, have been proven to protect myocardial cells from ischemia-reperfusion injury. One of its mechanisms of action is to inhibit the abnormal activation of BNIP3 in MIRI by regulating upstream signals (such as HIF-1α, AMPK/PI3K-Akt), thereby suppressing mitochondrial autophagy and apoptosis, while enhancing the antioxidant capacity of cardiomyocytes [13]. Astragaloside, as the main active component of astragalus, also has the effect of regulating the BNIP3 pathway and can alleviate myocardial ischemia-reperfusion injury [14]. Panax notoginseng saponins are the main active ingredients extracted from Panax notoginseng. Relevant research has shown that panax notoginseng saponins can significantly inhibit the activity of HIF-1α, reduce the induced expression of BNIP3, and thereby inhibit mitochondrial autophagy and cell apoptosis [15]. Leveraging this mechanism of action, panax notoginseng saponins have emerged as a significant potential drug for the prevention and treatment of myocardial ischemia-reperfusion injury [16]. Furthermore, panax notoginseng saponins exhibit various pharmacological effects, including improving

microcirculation and inhibiting platelet aggregation [17]. Ophiopogon japonicus saponin, the main active ingredient extracted from Ophiopogon japonicus, has been shown to exhibit significant effects in regulating the BNIP3 pathway. It can effectively alleviate mitochondrial autophagy and apoptosis during myocardial ischemia-reperfusion by inhibiting the activity of HIF-1α and downregulating the expression level of BNIP3. Furthermore, Ophiopogon japonicus saponin possesses strong antioxidant properties, capable of scavenging free radicals and reducing the damage to myocardial cells caused by oxidative stress.

4.1.3 Flavonoids

Flavonoids are a class of compounds with a flavonoid skeleton, widely distributed in various traditional Chinese medicines such as Ginkgo biloba leaves, Scutellaria baicalensis, and Pueraria lobata. Studies have shown that flavonoids exhibit significant effects in regulating the BNIP3 pathway and preventing myocardial ischemia-reperfusion injury. They can affect the expression and function of BNIP3 through various mechanisms, thereby exerting a protective effect. On the one hand, flavonoids can inhibit the activity of HIF-1α, reduce the induced expression of BNIP3, and subsequently suppress mitochondrial autophagy and apoptosis. This effect helps maintain the normal function of mitochondria and reduces cardiomyocyte death. On the other hand, flavonoids also possess potent antioxidant properties, capable of scavenging free radicals and mitigating the damage to cardiomyocytes caused by oxidative stress. Furthermore, flavonoids can regulate inflammatory responses, alleviate the inflammatory response during myocardial ischemia- reperfusion, and further protect myocardial tissue from damage.

Baicalein, as a typical flavonoid compound, has been proven to exhibit significant antioxidant, anti-inflammatory, and anti-apoptotic effects [18]. It can alleviate mitochondrial autophagy and cell apoptosis by inhibiting the HIF-1α/BNIP3 signaling axis, while exerting antioxidant and anti-inflammatory effects, thereby effectively preventing and treating myocardial ischemia-reperfusion injury [18]. Studies have shown that flavonoids, as an important component of traditional Chinese medicine, have broad application prospects in regulating the BNIP3 pathway and preventing myocardial ischemia-reperfusion injury.

4.1.4 Quinones

Quinones are a class of compounds containing a quinone structure, which are found in various traditional Chinese medicines. Recent studies have shown that quinones also play a significant role in regulating the BNIP3 pathway and preventing myocardial ischemia-reperfusion injury [19]. They can exert myocardial protective effects by influencing the expression and function of BNIP3 through specific mechanisms. On the one hand, quinones can act on upstream signaling molecules of BNIP3, such as inhibiting the activity of HIF-1α, thereby reducing the induced expression of BNIP3. This effect helps to suppress the excessive occurrence of mitochondrial autophagy and the triggering of apoptosis, thus maintaining the normal physiological function of cardiomyocytes. On the other hand, quinones also possess antioxidant properties, capable of scavenging free radicals and alleviating the damage to cardiomyocytes caused by oxidative stress, further protecting myocardial tissue from reperfusion injury.

Emodin, as a typical quinone compound, has been proven to exhibit significant antioxidant and anti-apoptotic effects [20]. It can mitigate the occurrence of mitochondrial autophagy and apoptosis by regulating the BNIP3 pathway, while exerting antioxidant effects, thereby effectively preventing and treating myocardial ischemia-reperfusion injury. Tanshinone IIA and salvianolic acid B are the main active ingredients in Salvia miltiorrhiza, and they exhibit unique effects in preventing and treating myocardial ischemia-reperfusion injury [21]. As a quinone compound, tanshinone IIA possesses remarkable antioxidant properties. It can scavenge free radicals and mitigate oxidative damage to mitochondria during myocardial ischemia-reperfusion, thereby protecting the structure and function of mitochondria. By reducing oxidative stress on mitochondria, tanshinone IIA can indirectly stabilize the function of BNIP3, preventing its excessive activation or inactivation due to oxidative damage. This effect helps maintain the normal balance between mitochondrial autophagy and apoptosis, reducing cardiomyocyte death. Salvianolic acid B also possesses potent antioxidant effects, which can protect cardiomyocytes from damage by scavenging free radicals and reducing oxidative stress. Salvianolic acid B can also regulate inflammatory responses and alleviate the inflammatory response during myocardial ischemia-reperfusion, further protecting myocardial tissue. Studies have shown that the combined application of tanshinone IIA and salvianolic acid B can exert a synergistic effect, more effectively reducing myocardial ischemia-reperfusion injury [21].

4.2 Traditional Chinese Medicine Compound Prescriptions and Preparations

Studies have shown that Buyang Huanwu Decoction can alleviate mitochondrial autophagy and apoptosis by inhibiting the activity of HIF-1α and downregulating the expression level of BNIP3, thereby improving myocardial microcirculation, reducing the damage to myocardial cells caused by ischemia and hypoxia, and further promoting the repair and regeneration of myocardial cells [22] [23]. In addition, Buyang Huanwu Decoction can also regulate inflammatory responses and alleviate the inflammatory response during myocardial ischemia-reperfusion, providing a favorable environment for the repair of myocardial cells. Research has shown that the active components in Xuefu Zhuyu Decoction can act on upstream signaling molecules of BNIP3, such as HIF-1α, by inhibiting their activity to reduce the induced expression of BNIP3, thereby maintaining the normal function of mitochondria and reducing cardiomyocyte death [24]. Xuefu Zhuyu Decoction also exhibits potent antioxidant and anti-inflammatory properties, capable of eliminating free radicals and mitigating the damage to myocardial cells caused by oxidative stress and inflammatory reactions, thereby further shielding myocardial tissue from reperfusion injury. Similarly, the active ingredients in Linggui Zhugan Decoction, Zhenwu Decoction, and Huanglian Jiedu Decoction reduce the induced expression of BNIP3 by inhibiting its activity, thus preserving the normal function of mitochondria and protecting myocardial cells from damage.

As traditional Chinese medicine preparations, Shenfu Injection [25], Qishen Yiqi Dropping Pill [26], Liqi Huoxue Dropping Pill [27], Compound Danshen Dropping Pill [28], Shengmai Yin [29], and Xintong Granules [30] have also demonstrated significant effects in treating myocardial ischemia-reperfusion injury. Its main components include ginsenosides and ophiopogonin, which can effectively inhibit mitochondrial autophagy and apoptosis by suppressing the activity of HIF-1α and downregulating the expression level of BNIP3. Qishen Yiqi Dropping Pill not only protects mitochondria from damage but also enhances the antioxidant capacity of myocardial cells, thereby reducing the damage caused by oxidative stress to myocardial cells [31]. Qishen Yiqi Dripping Pills can also regulate inflammatory reactions, alleviate inflammatory reactions during myocardial ischemia-reperfusion, and provide a good repair environment for myocardial cells [26]. Clinical studies have shown that Qishen Yiqi Dripping Pills can significantly improve cardiac function indicators in patients with myocardial ischemia-reperfusion injury, reduce the release of myocardial enzymes, lower the apoptosis rate of myocardial cells, and effectively alleviate myocardial ischemia-reperfusion injury.

In summary, traditional Chinese medicine (TCM) demonstrates unique advantages in treating myocardial ischemia-reperfusion injury (MIRI) by regulating the BNIP3 pathway and its related signaling molecules. Various forms of TCM, including single Chinese herbal medicines and their active ingredients, compound prescriptions, and preparations, can effectively alleviate MIRI through multiple mechanisms, such as inhibiting the excessive activation of the HIF-1α/BNIP3 signaling axis, finely regulating mitochondrial autophagy flux, suppressing BNIP3-mediated mitochondrial pathway apoptosis, and exerting antioxidant and anti-inflammatory effects.

5. Summary and Outlook

MIRI, as a cardiovascular disease that plagues humanity, currently lacks an ideal treatment plan due to its complex etiology and pathogenesis. Traditional Chinese medicine has demonstrated significant potential in treating MIRI, particularly through the Bcl-2/adenovirus E1B 19kD-interacting protein 3 (BNIP3) signaling pathway, which exerts multifaceted protective effects. This article first provides an overview of BNIP3 and its mechanism of action in myocardial ischemia-reperfusion injury (MIRI), elucidating the significant importance of regulating BNIP3 in the treatment of MIRI. It then summarizes the research progress on the prevention and treatment of MIRI through the regulation of BNIP3 by Chinese herbal monomers, active ingredients, compounds, and preparations. Traditional Chinese medicine (TCM), as an important component of traditional medicine, contributes to the treatment of myocardial ischemia-reperfusion injury (MIRI) with its holistic concept and syndrome differentiation thinking. Moreover, related traditional Chinese medicines and compound prescriptions have advantages such as multi-level, multi-target, and low side effects, which have gradually attracted widespread attention. In recent years, research has confirmed that TCM plays a significant role in the treatment of MIRI through BNIP3. Its mechanism of action involves regulating inflammatory response, oxidative stress, energy metabolism, apoptosis, and autophagy, providing theoretical support for the application of TCM in the treatment of MIRI.

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