treatment and after treatment 28 days. According to Cerebral infarction volume formula from Pullitono(Infarct volume cm3 = length×width×CT slice number×thickness÷2), the mean value was determined by two neurologists. Adopt BartheI index to evaluate the changes in activities of daily living about 102 cases before treatment and after treatment for 28 days.

Results

1.Two groups of patients after treatment for 28 days, compared with their serum PAPP-A levels, the difference was statistically significant, the acupuncture group was better than the control group(P＜0.05).

2.Two groups of patients Cranial CT scan after treatment for 28 days, infarct volume decreased significantly, and the acupuncture group was better than the control group (P < 0.05).

3.Two groups of patients after treatment for 28 days, BartheI index of daily life was significantly higher than before treatment, and the acupuncture group was better than the control group (P < 0.01).

Conclusion

Acupuncture can significantly reduce the serum PAPP-A level in patients with carotid atherosclerosis cerebral infarction, reduce the cerebral infarction volume, improve the patient's daily life BI index, and it plays the role of prevention and treatment of acute cerebral infarction. The mechanism may be that acupuncture reduces the serum PAPP-A level to decrease the degradation and inflammatory reaction of extracellular matrix in plaque, to promote plaque stabilization, and then reduce the occurrence and development of ischemic stroke.

Key words: acupuncture; cerebral infarction; Carotid atherosclerotic plaque; PAPP-A

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GUANXINNING INJECTION FEMORAL HEAD PERFUSION OF RABBIT FEMORAL HEAD NECROSIS BFGF EXPRESSION

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Abstract: Basic fibroblast growth factor (FGF-b) is currently the strongest known to promote cell growth factor plays an important role in blood vessels and osteoblasts regeneration. The bFGF can stimulate the capillary endothelial cell migration and proliferation,which form the capillary sprouts and promote the formation of new blood vessels,while the release of at least two interstitial JiangMei plasminogen activator and collagenase.From the experiment, one can estimate the pathological process ANFH, the problem is likely to exist bFGF lacking. Due to the presence of ischemic factors, resulting in the relative lack of bFGF; on the contrary, the lack of bFGF does not help improve blood circulation and regeneration of small blood vessels, which reduces the biological activity of bone morphogenetic protein, leading to necrosis of the femoral head gradual, slow new bone repair and affect the bone repair.In this study, the saline group and bFGFmRNA bFGF expression decreased gradually in addition to saline treatment showed avascular invalid, but also shows the influence of ischemic bone repair this problem and the expression of the experimental group was gradually increased during the administration period, suggesting that Guanxinning injection can improve ischemia, thereby contributing to the blood vessel and bone regeneration and repair of bone necrosis.

Keywords: Guanxinning injection femoral head perfusion, INFH, bFGF, experimental study

Necrosis of femoral head necrosis is to reduce the mechanical properties of bone tissue, resulting in an obstacle common and difficult disease. First, the general pathogenesis of vascular rupture, after the emergence of avascular change last bone cells, bone marrow cells, cartilage cells necrosis. The incidence way through the often interrelated, leading to difficult to understand the mechanism. Currently ultimate goal of treatment is to protect the femoral head , the main methods are pith decompression, bone grafting, osteotomy, the surgical treatment has some effect, but still can not achieve satisfactory results. At present, domestic scholars of Chinese medicine treatment of femoral head necrosis conducted extensive research, we have achieved great results. We apply Guanxinning injection femoral head necrosis femoral head perfusion expressing bFGF were studied.

Objective: To investigate the Guanxinning injection femoral head perfusion on bFGF expression.

Materials and methods: 90 New Zealand white rabbits were randomly divided into normal control group, saline group, Guanxinning group, after the successful model of each group were sacrificed at 1,3,6,9,12 weeks after administration of femoral bones PCR, Western blot assay bFGF content.

Results and discussion:

Blank group and saline group, bFGF and bFGFmRNA have little expression, while the saline group with prolonged treatment time, bFGF protein expression gradually weaken and gradually lower than the control group, the control group in the first week of bFGF and both bFGFmRNA a small amount of expression, but there was no blank group and saline group significant difference (P>0.05), the first three weeks when there is significant expression, there are significant differences (P<0.05) compared with the control group, to 6 weeks with a blank and saline group has significant difference (P<0.01).

Guanxinning not only has the role of blood circulation and also has the effect of Bushenzhuanggu by experimental observations. At each time point as the extension of duration of bFGF and bFGF-mRNA expression also gradually enhanced significantly.

Therefore, we speculate that Guanxinning are similar to other traditional Chinese medicine (TCM) for activating blood circulation, also can through the expression of bFGF promotes the regeneration of blood vessels to promote bone repair.

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MECHANISM STUDY ON THE ANTI-INFLAMMATION, ANTIOXIDANT AND ATTENUATING NEURONAL APOPTOSISEFFECT OF DANGGUI-SHAOYAO-SAN.

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Abstract: Danggui-Shaoyao-San (DSS) is a famous Chinese herbal formula, which has long been used for pain treatment and has been demonstrated to possess and anti-depressant effects. In addition, it has been widely used in the treatment of various cognitive diseases. This paper reviews the mechanism of DSS on the anti-inflammation, antioxidant and attenuating neuronal apoptosis effect of DSS for the treatment of Alzheimer Disease (AD).

Keywords:Alzheimer disease; traditional Chinese medicine; anti-inflammation; antioxidant; neuronal apoptosis

Anti-inflammation,Antioxidant

Previous studies have shown that DSS produces antidepressant-like effect in rodents. This study shows that the antidepressant-like activity of DSS is probably mediated by the modulation of central monoamine neurotransmitter systems and the reduction of oxidative stress.[1] Moreover, the renoprotective effects of DSS in STZ-diabetic rats not only were attributable to regulate plasma glucose to attenuate AGEs expression in diabetic glomeruli but also likely reflected its antioxidant activity.[2] DSS significantly reduced the expression of the IL-1beta, IL-6, TNF-alpha mRNA, and the level of the NO depressed the neuron apoptosis in the hippocampus.[3] Treatment with DSS had significant analgesic effects on ETM-induced pain through attenuated the Fos and Iba-1 levels at POD 1, which was accompanied with inhibition of both neuronal and microglial activation.[4] DSS was a useful therapeutic agent for short- and long-term inflammation induced pain, through both anti-inflammatory and suppression of central sensitization mechanisms.[5]

Treatment for cognitive impairment

A downstream pathway for DSS induction of melatonin synthesis in the rat pineal gland acts via cyclic AMP-dependent cascade and transcription mechanism.[6] DSS mediates the modulation of central monoamine neurotransmitter systems and ameliorates dysfunction of the central cholinergic nervous system and scopolamine-induced decrease in ACh levels. DSS improves the function of the dopaminergic, adrenergic, and serotonergic nervous systems. DSS can alleviate cognitive dysfunction of Alzheimer's disease (AD) patients.[7] JD-30 is one of the chief active fractions extracted from DSS by its ability to ameliorate deterioration of cognition, and by blocking and disrupting the aggregation of Abeta so that synaptic