

星菱承气汤和补阳还五汤对脑缺血大鼠细胞凋亡Fas/FasI和Caspase-3调控的影响

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作者 单位

E-mail

[刘敬霞](#) [宁夏医科大学中医学院,银川 750004](#)

ljx199566@163.com

[李建生](#) [河南中医学院老年医学研究所,郑州 450008](#)

[俞维](#) [宁夏医科大学中医学院,银川 750004](#)

[黑常春](#) [宁夏医科大学中医学院,银川 750004](#)

[刘会贤](#) [宁夏医科大学中医学院,银川 750004](#)

[任非非](#) [宁夏医科大学中医学院,银川 750004](#)

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中文摘要:目的: 观察星菱承气汤和补阳还五汤对脑缺血神经细胞凋亡Fas/FasI和Caspase-3的影响,以明确其抑制神经细胞凋亡的机制。方法: 大鼠随机分为假手术组、模型组、尼莫地平组、星菱承气汤和补阳还五汤组;线栓法制备大鼠中动脉阻塞模型;星菱承气汤($5.0 \text{ g} \cdot \text{kg}^{-1}$)、补阳还五汤($13.0 \text{ g} \cdot \text{kg}^{-1}$)、尼莫地平($10.8 \text{ mg} \cdot \text{kg}^{-1}$)组大鼠分别于造模前4 d灌胃,造模后每日1次;缺血后1,3,7 d取大鼠脑组织,免疫组织化学法检测Fas, FasI, Caspase-3表达。结果: 假手术组大鼠可见少许Fas(16.60 ± 1.36), FasI(19.40 ± 1.72)和Caspase-3(16.35 ± 1.63)表达;大鼠缺血后1,3,7 d的Fas($45.83 \pm 1.44, 36.25 \pm 1.60, 31.37 \pm 2.27$), FasI($44.27 \pm 2.25, 37.68 \pm 2.01, 34.15 \pm 1.55$)和Caspase-3($37.18 \pm 2.78, 29.50 \pm 2.07, 25.26 \pm 3.04$)表达均增强($P < 0.01$);与模型组比较,各用药组Fas, FasI表达减弱,尼莫地平组缺血后1 d,星菱承气汤和补阳还五汤各组Caspase-3表达减弱;各星菱承气汤和补阳还五汤组7 d的Fas, FasI表达、星菱承气汤组3 d的Caspase-3表达均较尼莫地平组减弱;星菱承气汤组缺血后1 d的Fas和FasI, 3 d的Fas和Caspase-3表达均较补阳还五汤组减弱。结论: 脑缺血可引起细胞凋亡Fas/FasI调控的表达上调,星菱承气汤和补阳还五汤可下调Fas/FasI及Caspase-3表达,星菱承气汤抑制Fas和FasI及Caspase-3的作用较补阳还五汤早而显著,以缺血后1 d的调控作用最为明显。

中文关键词: [脑缺血](#) [补阳还五汤](#) [星菱承气汤](#) [细胞凋亡](#) [Fas/FasI](#) [Caspase-3](#)

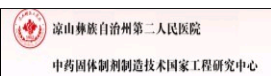
Effects of Xinglou Chengqi Decoction and Buyang Huanwu Decoction on Fas/FasI and Caspase-3 Pathway of Apoptosis in Rats with Cerebral Ischemia

Abstract: Objective: To observe the effects and mechanism of Xinglou Chengqi decoction (XLCQD) and Buyang Huanwu decoction (BYHWD) on Fas/FasI and Caspase-3 of regulating genes of neurons apoptosis caused by cerebral ischemia. Method: Rats were randomly divided into sham, model, Nimodipine, BYHWD and XLCQD groups. Focal cerebral ischemia models were established by middle cerebral artery occlusion with nylon thread. Rats were given with XLCQD ($5.0 \text{ g} \cdot \text{kg}^{-1}$), BYHWD ($13.0 \text{ g} \cdot \text{kg}^{-1}$) and nimodipine ($10.8 \text{ mg} \cdot \text{kg}^{-1}$) by ig before the model preparation, once a day after the operation. At 1, 3, 7 d after operation, neurons apoptosis, expressions of Fas, FasI and Caspase-3 were determined using the method of immunohistochemistry. Result: A few expressions of Fas (16.60 ± 1.36), FasI (19.40 ± 1.72) and Caspase-3 (16.35 ± 1.63) could be observed in rat's brain of sham group. In each model group, expressions of Fas ($45.83 \pm 1.44, 36.25 \pm 1.60, 31.37 \pm 2.27$), FasI ($44.27 \pm 2.25, 37.68 \pm 2.01, 34.15 \pm 1.55$), Caspase-3 ($37.18 \pm 2.78, 29.50 \pm 2.07, 25.26 \pm 3.04$) all increased ($P < 0.01$). Fas and FasI expression in each administrated group all decreased, and the expression of Caspase-3 in Nimodipine 1 d group, each XLCQD and BYHWD group was higher. Fas and FasI expressions in each XLCQD group and 7 d BYHWD group and Caspase-3 in 3 d XLCQD group all decreased than that in Nimodipine groups. The expressions of Fas in 1 d and 3 d, FasI in 1 d, Caspase-3 in 3 d XLCQD groups all decreased than that in BYHWD groups. Conclusion: It was shown that Fas/FasI up-regulation of apoptosis could be caused by cerebral ischemia, and XLCQD and BYHWD could all inhibit the level of Fas, FasI and Caspase-3. The role of XLCQD was more earlier and significant in down-regulating the expressions of Fas, FasI and Caspase-3, especially at 1 d after cerebral ischemia.

keywords: [cerebral ischemia](#) [Buyang Huanwu decoction](#) [Xinglou Chengqi Tang](#) [Fas/FasI](#) [Caspase-3](#)


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