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Research Report

The effects of electroacupuncture treatment on the postoperative cognitive function in aged rats with acute myocardial ischemia–reperfusion

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ABSTRACT

Many literatures have proven that postoperative cognitive dysfunction (POCD) was very common in old patients after the injury of acute myocardial ischemia–reperfusion (AMIR) clinically such as the off-pump coronary artery bypass surgery (OPCAB) without definite mechanism; however, reports on the animal experiments were rarely seen. We hypothesized that AMIR could contribute to cognitive dysfunction, and this severe injury might be impeded by EA via hindering neuroinflammation and oxidative stress response as well as modulating the balance of the autonomic nervous system. The aged male Sprague Dawley rats were randomly assigned into three experimental groups: sham (sham operation), AMIR, and EA (electroacupunture treatment, acupoints GV20 and ST36+AMIR) groups. The survival rate, heart rate variability analysis, examination of pathology within the hippocampal CA1, oxidative stress, systemic inflammation and the behavior testing were evaluated by their corresponding methods. The results showed that the rats subjected to AMIR had lower survival rates, higher malondialdehyde (MDA), decreased superoxide dismutase (SOD) activity, more microglial activation, and presented evidence of severe brain injury and cognitive dysfunction on the 1st, 3rd, 7th days after reperfusion compared to sham-operated controls. Most important of all, the above damages induced by the AMIR were significantly improved by the EA treatment. Our findings indicated that EA treatment could be a neuroprotective therapy for the cognitive dysfunction induced by the AMIR event, which might be attributablefor balancing the autonomic nervous system, inhibiting the neuronic apoptosis, hindering microglial activation, attenuating oxidative stress and restraining the central and peripheral inflammation reactions.

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1. Introduction

Acute myocardial ischemia–reperfusion (AMIR) is a frequent phenomenon in the procedures such as acute myocardial infarction, collapse of heart stent and cardiac surgery, especially the coronary artery bypass surgery, which is associated with a predictable incidence of injury to the vital organs. Many literatures have proven that postoperative cognitive dysfunction (POCD) was very common in elderly patients subjected to cardiac surgery with or without cardiopulmonary bypass (Knipp et al., 2008; Sendelbach et al., 2006). However, the actual mechanism underlying this phenomenon has not been clarified till now.

POCD is a thorny complication with increased morbidity and mortality after major surgery which frequently occurs in aged patients (Simone and Michael, 2008). In spite of undefined etiology, several risk factors have been investigated including old age, poor levels of education (Stern, 2002), genetic polymorphism (Yingmin, 2012), surgery injury (Rasmussen and Siersma, 2004), anesthetic drugs, previous cerebrovascular diseases and so on. Although cardiopulmonary bypass (CPB) has been a controversial factor contributed to the POCD in cardiac surgery, recent randomized controlled trials found no significant difference in postoperative cognitive dysfunction in patients undergoing conventional coronary artery bypass grafting (CABG) with CPB and those undergoing CABG without CPB (Bruggemans, 2013; Marasco et al., 2008). Increasing studies demonstrated that the main internal pathogenesis of POCD was neuroinflammation (Monk et al., 2008; Huang et al., 2008), but the predisposing factors of the neuroinflammation and how it worked in the formation of POCD should need to be further investigated. Neurocognitive decline in the early postoperative period after off-pump coronary artery bypass surgery (OPCAB) was a commonly occurring phenomenon and the elderly subjected to OPCAB were suffered more from the POCD with several possible reasons such as degenerative changes of all systems especially the brain and more previous diseases (Baba et al., 2007). However, the internal mechanism of the POCD triggered by AMIR and the pathological changes in the brain after the OPCAB have not yet been discovered thoroughly.

Depending on distinctly special acupoints, electroacupuncture (EA) treatment has been confirmed and used in many diseases such as myocardial ischemia (Zhou et al., 2005), the severe pain induced by cancer (Chen et al., 2013), epilepsy (Yi et al., 2013), depression (Ji et al., 2013), diabetes mellitus (Lin et al., 2013) and cerebral related diseases (Tan et al., 2013), which has received significant effects. Furthermore, increasing evidence has shown that EA can attenuate cognitive deficits by means of maintaining sympathetic–parasympathetic balance, inhibiting apoptosis (Shufang, 2010) and neuroinflammation (Jun, 2007) as well as regulating cell proliferation (Liu et al., 2011) in the hippocampus. But a few conflict reports appeared that EA made no sense in POCD (Yang et al., 2009). Hence, more researches are needed to clarify whether EA can ameliorate POCD and, if so, what mechanism could be underlying this neuroprotective value.

Based on the existing clinical literature, we hypothesized that AMIR could contribute to cognitive dysfunction, and this severe injury might be impeded by EA via hindering neuroinflammation and oxidative stress response as well as modulating the balance of autonomic nervous system. The consequence of our study should provide a better understanding of long-term detriments associated with AMIR and the neuroprotective mechanism of EA treatment.

2. Results

2.1. Physiological data

Body weight and rectal temperature did not have any significant difference among all the groups. The MAP of all rats was maintained average 92–96 mmHg, and the HR was average 376–392 bpm. No difference was discovered among all the groups.

2.2. Survival rates

The 7 days-survival rate of the rats subjected to AMIR (40%, 8/20 rats) was significantly different from the sham group (100%, 20/20 rats) ($P<0.05$), while the EA group (75%, 15/20 rats) was also higher than the AMIR group (40%, 8/20 rats) ($P<0.05$). However, there was no striking difference between the EA group and the sham group (Fig. 1).

2.3. Excitability of the sympathetic nerve

The ratio of LF:HF was elevated significantly when the rats were subjected to ischemia in the EA and AMIR groups, while no change in the sham group. HRV in the AMIR group was higher significantly than the other two groups at the moment prior to being sacrificed on the 1st and 3rd days ($P<0.05$) (Table 1), which represented that the excitability of the

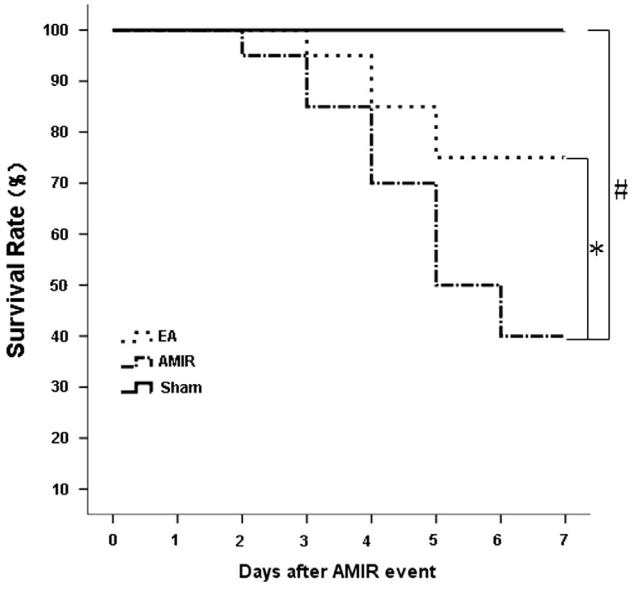

Fig. 1 – Survival rates among the three groups up to the 7th day after the AMIR event. Data are expressed in percentages. Each data-point represents the group mean ($n=20$; $^{\#}P<0.05$ vs. sham; $^{*}P<0.05$ vs. EA).