Experimental and Clinical Studies on Inhibitory Effect of Ganoderma lucidum on Platelet Aggregation

TAO Jun (~ $), FENG Ke-yan (~g~)  
Department of Internal Medicine, Tongji Hospital, Tongji Medical University, Wuhan

Summary: In this study we observed the inhibitory effect of Chinese herbal medicine Ganoderma lucidum (GL) on platelet aggregation in 15 healthy volunteers and 33 patients with atherosclerotic diseases. The results showed that the first and the second phase of aggregation of platelets of the healthy volunteers were obviously inhibited \( (P<0.01) \) when watery soluble extract of GL of different concentrations was added to the platelets in vitro, i.e., the reaction speed of platelet aggregation was slowed down. The inhibitory effect was related to dosage. Platelet aggregation induced by ADP in final concentration of 2 \( \mu \text{mol/L} \) and 3 \( \mu \text{mol/L} \) was obviously inhibited, after the patients had taken GL 1 g 3 times a day for 2 weeks, the maximum platelet aggregation inhibition rates were then 31.49 \% \( (P<0.01) \) and 17.7 \% \( (P<0.01) \) respectively. Length and weights (wet and dry) of the extracorporeal thrombi were reduced from 30.05±4.38 mm, 103.95±9.33 mg and 44.89±4.79 mg to 20.45±2.33 mm \( (P<0.05) \), 85.27±8.77 mg \( (P<0.01) \) and 35.1±4.5 mg \( (P<0.01) \) respectively after oral administration of GL. The results of our experiments suggested that the Chinese herbal medicine GL may be an effective inhibitory agent of platelet aggregation. However, its mechanism and active principles remain to be further investigated.

Key words: Ganoderma lucidum, platelet aggregation inhibitory agent

Ganoderma lucidum (GL) is a Chinese traditional herbal medicine. It has been used clinically in our country for treating various diseases for about two thousand years. According to clinical observations and pharmacologic experiments, GL can improve the microcirculation of the organs, increase the amount of blood current, and increase the cAMP concentration in plasma and heart muscle cells\(^{[11]}\). However, its effect of inhibiting platelet aggregation has not been reported in the literature so far. Therefore, we made observations on the effect of GL on platelet aggregation in healthy volunteers and atherosclerotic patients.

SUBJECTS AND METHODS

Subjects

15 healthy volunteers (2 women, 13 men), averaging 38 years in age (25—60), were studied. They did not take any agents which interfere with platelet aggregation one week before the experiment.

33 patients (5 women, 28 men), averaging 64 years in age (50—79), including 22 with old myocardial infarction, 9 with the sequela of cerebral thrombosis and 2 with diabetes mellitus were investigated. They were not given any agents which interfere with platelet aggregation one week before the experiment.

Methods of determination of platelet aggregation

Tablets and watery soluble extract of GL were produced by Tongji Hospital Pharmaceutical Factory (each tablet containing 0.2 g of crude GL; each ml
of watery soluble extract containing 0.4 g of crude GL, pH 7.4), ADP was provided by Merck Co (Germany), the aggregometer type SPX-3, PPP was made by Shanghai Kodar Medical Instrument Factory.

4.5 ml of venous blood drawn from the antecubital vein were mixed with 0.5 ml of 3.8 % trisodium citrate. Platelet-rich plasma (PRP) was separated by centrifugation (10 min at 800 rpm), and platelet-poor plasma (PPP) was obtained by centrifugation (20 min at 3000 rpm). Light transmission was set at 0 % with PRP and 100 % with PPP.

**Extracorporeal test of volunteers**

200 μl PRP plus 10 μl watery soluble extract of GL or normal saline (NS) was incubated at 37°C for 5 min and then 10 μl aggregating agent ADP was added. Platelet aggregation was recorded for 7 min, and then the effect of different dosages of GL on ADP-induced platelet aggregation was observed.

**Intracorporeal test of patients**

The platelet aggregation was determined before and after the patients had taken 1 g GL 3 times daily for two weeks, and then the difference of platelet aggregation before and after medication was compared. Extracorporeal thrombi of 20 patients were determined at the same time.

**Statistics**

The results were presented as x ± sX. Student's t-test was adopted to compare the relative effects of GL on platelet aggregation.

**RESULTS**

1. **The effect of GL on platelet aggregation of healthy volunteers induced by ADP in in-vitro test**

   Venous blood was drawn from antecubital vein to separate PTP and PPP. We used ADP 3 μmol to induce platelet aggregation and observed the effect of three different concentrations of GL (0.25 mg/ml, 0.5 mg/ml and 1.0 mg/ml) on platelet aggregation. The results showed that the 1st-min, 5th-min and maximum rates of platelet aggregation were inhibited in varying degrees. The maximum aggregation inhibition rates were 27.65 %, 51.4 % and 81.92 %, respectively, which displayed very significant difference as compared to the NS group (table 1). The inhibitory effect of GL on platelet aggregation was related to the dosage (fig. 1).

2. **The effect of GL on platelet aggregation and extracorporeal thrombus of patients**

   After two weeks of taking GL tablets, in 33 patients the 1st-min, 5th-min and maximum platelet aggregation induced by ADP (2 μmol and 3 μmol)

![Fig.1](image1)

**Fig.1.** Representative tracing from one healthy volunteer showing the effect of GL on the magnitude and rate of aggregation.

![Fig.2](image2)

**Fig.2.** Effect of GL on the extracorporeal thrombus formation in patients with atherosclerosis. *P<0.05; **P<0.001 as compared with before GL.