Effects of Smoking on Rat Peptic Ulcer

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Abstract. 40 SD rats weighing 200 ± 10 g were used, 12 of 40 rats were randomly selected as those in sham group, and the remaining 28 were used to establish the acetic acid-induced gastric ulcers to observe effects of smoking on the rat gastric ulcer and ulcer area. The acetic acid-induced gastric ulcer model in rats is reliable. Smoking can delay or the healing of ulcer, which may be related to the increase of gastric acid secretion induced by smoking, the increase in oxygen free radicals that can damage the mucosa of stomach, the decrease of pyloric sphincter tension, the reflux of bile and duodenal fluid to the stomach, and the reduction of prostaglandin E content in mucosa. It is recommended that in the treatment process of patients with gastric ulcer, they should stay away from smoking places, as much as possible avoid the hazards of passive smoking.

Introduction

The number of smokers is large and the number of people suffering from hazards of passive smoking is increasing more and more[1-5]. Peptic ulcer is one of common diseases throughout the world. More attention is always paid to the research on factors to induce or cause peptic ulcer[6-7]. In this study, an acetic acid-induced gastric ulcer tat model was established effects of smoking on the gastric ulcer were observed, including the ulcer area and the gross morphological changes in peptic ulcer, which was designed to provide experimental evidences for the prevention and treatment of peptic ulcer.

Materials

Experimental animals

SD rats, weighing $200 \pm 10g$ and provided by Changchun Yisi Experimental Animal Research Center,

were raised in separate cages, eating and drinking freely and at 20-25 °C for 5 days to acclimate.

Methods

Establishment of animal model

12 of 40 rats were randomly selected as those in sham group, and the remaining 28 were used to establish the rat peptic ulcer model. Before the surgery, the rats were fasted for 24 h. After the rat was anesthetized with 10% chloral hydrate, its abdominal cavity was opened along the epigastric midline line below the processus xiphoideus to expose the stomach; the anterior wall near the pylorus of stomach was touched two times with a piece of filter paper soaked in 100% acetic acid solution (be

noted to avoid the blood vessels), each time lasted for 30s and the touched place was cleaned and dried with a cotton swab; after the abdominal cavity was washed with saline, the incision was sutured. All the rats used for the establishment of the animal model were treated in the same way described above. During the surgery, the aseptic surgical procedure was followed strictly. The surgical process of rats in the sham group was the same as that of rats used for the establishment of the animal model, but the filter paper soaked with acetic acid was replaced by one soaked with saline to touch the stomach wall.

Animal grouping

On the 3rd day after the modeling surgery, dead rats were excluded, and 20 surviving rats were selected and randomly divided into a model group and a smoking group, 10 rats in each group. Rats in the smoking group were placed in a transparent box from 12:00 to 20:00 every day successively for 10 days, in which 10 lighted cigarette were fixed in the transparent box. Rats in the model group and the sham group were placed in a transparent box in the same way at the same time continuously 10 days, but no fixed cigarette in the transparent box.

Detection indicators and methods

Observation on changes in the gross morphology of stomach: On the 10th day, all the rats were fasted for 24 h, and then decapitated; the abdominal cavity was aseptically opened and the stomach was taken; the stomach was opened along the greater curvature of the stomach and ice saline was used to rinse the stomach content; the gastric mucosa was flatten on a flat plate and dried with filter paper; changes in the gross morphology of the front stomach wall mucosa near the pylorus, which had corroded by acetic acid, were observed with a visual inspection.

Observation on ulcer area: On the 10th day, all the rats were fasted for 24 h, and then decapitated; the abdominal cavity was aseptically opened and the stomach was taken; the stomach was opened along the greater curvature of the stomach and ice saline was used to rinse the stomach content; the gastric mucosa was flatten on a flat plate and dried with filter paper; the maximum length diameter and the maximum width diameter of ulcers were measured with a vernier caliper, and then the ulcer area was calculated (the ulcer area = the maximum length diameter ×the maximum width diameter).

Results

Observation on changes in the gross morphology of stomach

The gastric ulcer of rats in the smoking group showed a round or oval sunken and a crater-like shape, its edge was clear, its bottom was covered with a layer of gray inflammatory exudate and sphacelus, the mucosa folds surrounding the ulceration became thicker, and parts of ulcer tissues adhered to the surrounding tissues. Morphological changes in ulcers of rats in the model group were similar to those in the smoking group, but the ulcer area was smaller, and there were less exudate and sphacelus. The gastric mucosa of rats in the sham group showed no abnormalities.

Observation on the ulcer area

The ulcer areas of rats in each group was observed after the the anatomy of the stomch. There was no ulcer in rats in the sham group. Compared with that in the model group, the ulcer area of rats in the smoking group increased significantly, with a statistically significant difference (P < 0.05) as shown in table 1, suggesting that smoking can aggravate the ulcer induced by acetic acid in rats.

_	Table 1 The ulcer areas of rats in each group ($X \pm s$, n=10)	
_	Group	Ulcer areas
	Sham	0
	Model	7.4±1.70
	Smoking	10.3±1.98*

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Note: *Compared with those in the model group, P<0.05.

Discussion

Gastric ulcer is referred to a localized tissue defect disease of mucous layer and submucosa, even muscularis and serosa, soaked in gastric juice containing gastric acid and pepsin, and caused by a variety of factors[6-7]. The establishment of an ideal and stable animal model is the key to a experimental study on animals. The research on the pathogenesis and the treatment of peptic ulcer diseases is also reliable on the animal model of ulcer diseases. There are various animal models of gastric ulcer disease and the acetic acid-induced gastric ulcer model is commonly used currently. In this study, the acetic acid-induced gastric ulcer model in rats was used, since this method was considered to be simple, able to result in a single round ulcer, and controllable and stable in the measurement of the size and depth of ulcer. It is well known that an animal ulcer model with significant ulcers in the local damaged tissue, basically unanimous ulcers in size and depth, and pathological changes of ulcer consistent with the characteristics of human ulcer, is no doubt a good animal ulcer model used for the study on the pathogenesis of ulcer and the evaluation of efficacies of anti-ulcer drugs.

The results of this experiment showed that ulcer areas of rats in the smoking group were significantly larger than those in the model group, indicating that smoking can delay the healing of ulcer. The exact mechanism of smoking effects on the ulcer formation and healing is still not clear, which may be related to the increase of gastric acid secretion induced by smoking ^[4], the increase in oxygen free radicals that can damage the mucosa of stomach, the decrease of pyloric sphincter tension, the reflux of bile and duodenal fluid to the stomach, and the reduction of prostaglandin E content in mucosa and other factors. It is recommended that in the treatment process of patients with gastric ulcer, they should stay away from smoking places, as much as possible avoid the hazards of passive smoking.

Summary

The acetic acid-induced gastric ulcer model in rats is considered to be simple, able to result in a single round ulcer, and controllable and stable in the measurement of the size and depth of ulcer. The results indicate that smoking can delay the healing of ulcer, which may be related to the increase of gastric acid secretion induced by smoking, the increase in oxygen free radicals that can damage the mucosa of stomach, the decrease of pyloric sphincter tension, the reflux of bile and duodenal fluid to the stomach, and the reduction of prostaglandin E content in mucosa. It is recommended that in the treatment process of patients with gastric ulcer, they should stay away from smoking places, as much as possible avoid the hazards of passive smoking.

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