The Development of the Ruminal Ulcer in the Albino Rats Fed with Low Protein Diet associated with Rocking Stress

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ABSTRACT

The pathological changes of the gastric rumen produced by rocking stress in 38 young male albino rats fed on low protein diet for a short period and in 20 young male albino rats fed on low protein diet only as the control rats, were investigated macroscopically and histologically.

The ulcerative lesions of the gastric rumen were observed in 17 out of 38 experimental rats accompanied with hyperplasia and cornification of the ruminal epithelial layer and more or less edematous changes of the tunica propria near the ulcer site.

The authors' finding of the ruminal ulcer in albino rats produced by rocking stress associated with feeding on a low protein diet for a short period were compared with the results of others. The authors discussed other opinions concerning of the gastric ruminal ulcer of the albino rats produced in various ways.

INTRODUCTION

Singer (1931) first produced the lesions in the rumen of albino rats by a diet of bread mixed with sawdust. Thereafter, a number of investigators also produced lesions in the rumen by various methods, and discussed the mechanism of the formation of the ruminal ulcer. Pappenheimer and Larimore (1924) reported that the ruminal ulcer of the rats could be produced by feeding of rachitic diet (diet 84) in the 7 out of 35 rats. They also produced the ruminal ulcers by feeding various other diets. Sharpless (1937, 1940) also produced lesions in the rumen of rats by feeding with low protein diet. Shay et al. (1945) produced ulcers of forestomach in rats by ligature of the pylorus. The rat was kept on a fasting diet for 48 to 72 hours, and then the pylorus was ligated for 16 to 18 hours.

Robert and Nezamis (1958) produced the same ulcers of the forestomach without ligature of pylorus by having the rats on a fasting diet alone for four days. Levrat and Lambert (1959) produced ulceration in the glandular portion by rocking stress and intermittent photic stimulation. However, Lee (1964), Moon (1964), and Park (1967) produced lesions of the forestomach by rocking stress without photic stimulation. They fed the rats on a biologically low protein diet. The present study is designed in order to produce lesions of the forestomach of rats on low protein diet, and to observe the procedure of the ulcer formation in detail.

MATERIALS AND METHODS

38 healthy young male rats weighing about 100 gm. each were used, and were fed on low protein diets for 10 to 15 days and then rocking stress was applied. The low protein diet was composed as follows: 90.0% of powdered rice, 1.0% of Brewers yeast, 4.0% of salt mixture.
(Hubbel-Mendel-Wakemen-Mixture), and 5.0% of Olive oil. The rats were rocked for four hours every day for 7 consecutive days. On the first day the rats were rocked at a frequency of 180 per minute with the rocking distance of 4 cm. The frequency was increased by 20 per minute every day until a frequency of 260 per minute reached and this rats was continued for 3 additional days.

For the control group 20 rats were fed on the same diet without any rocking treatments. 24 hours after the last rocking treatment, all the rats were sacrificed and the stomachs examined macroscopically and histologically.

RESULTS

1. Control group:

In the control group on low protein diet alone, ruminal ulcer was found in only one out of 20 rats: the histopathological changes of the ulcer were similar to those in “rocking” experimental group.

In the rest of the control rats hardly any histopathological changes were observed (Fig. 1).

2. Experimental group:

a) Macroscopically round or elliptic forms of ulcerative were evident and marked by elevated peripheral mucosa. The centers of the lesions were depressed as a pit or a fissure-like invagination. The ruminal epithelium with serve hyperkeratosis showed an uneven or rough contour. In the multiple lesions, hyperplastic changes of the ruminal epithelial layer were fairly evident.

b) Histologically, in the experimental group subjected to rocking stress, ruminal ulcers occurring in 17 out of 38 rats or about 45%. In general the ruminal ulcers were accompanied with various degrees of hyperplasia and hyperkeratosis, especially neutrophil leucocytes infiltration near the ulcerative site of the epithelial layer. Inflammatory changes occurred in the underlying tunica propria, tunica muscularis mucosae, and tunica submucosa. In the histopathologic changes of the epithelial layer of the gastric rumen, 15 out of 17 ruminal ulcers showed hyperplasia (Fig. 2, 3), and hyperkeratosis in all instances near the ulcerative sites.

The epithelial cells were markedly proliferated and gave rise to papilloma extending to the tunica propria and tunica muscularis mucosa. Remarkable edematous changes were observed in an incidence of 9 out of the 17 ruminal ulcers (Fig. 4).

In the underlying tunica propria and tunica submucosa of the ruminal ulcer sites, typical inflammatory changes were found; for instances, infiltration of neutrophils and eosinophils, and occasionally lymphocytes, and congestion of the blood vessels.

DISCUSSION

Singer (1913) reported that experimental ruminal gastric ulcers were produced in rats by feeding wet bread mixed with sawdust.

Rats fed on a mixture of white bread and minced intestines of other rats also developed ulcers in about the same percentage. He concluded that the lesions were due to infection, but the causative agent was not identified. Pappenheimer and Larimore (1924) reported that ruminal ulcers were produced by feeding a complete diet with chopped hair added, and multiple ulcers with marked epithelial hyperplasia in the gastric rumen were produced by feeding with an alkaline diet, and more or less epithelial hyperplasia only about the marginal ridge by feeding an acid diet. Hueper (1934) has shown that there are many instances in which epithelial tissue is more sensitive to proliferating stimuli when its sulphydryl content is reduced. Sharpless (1937, 1940) produced extreme hyperplasia in the rat's gastric rumen fed with a purified low protein diet and prevented such hyperplasia by
feeding cystine. In the present study the authors found that all cases of the ulcerative lesions were accompanied by more or less hyperplasia in the vicinity of the lesions. Additionally the authors assumed that rocking stress might cause a nutritional disturbance to the gastric ruminal epithelium followed by hyperplasia and ulcerative lesions.

Chen (1941) by starving for 7 days or longer, Shay et al. (1945) by fasting for a few days and ligation of the pyloric region, Robert and Nezamis (1958, 1959) by fasting for 4 days, Brodie and Hanson (1960) by the application of restraint for 18 hours each day for 3 or 4 days, Moon (1964) feeding with vitamine “A” deficient diet associated with rocking stress, Lee (1964) by feeding with riboflavin deficient diet associated with rocking stress, and Park (1967) by feeding with low protein diet associated with rocking stress all produced ulcerative lesions in the gastric rumen of the experimental animals. The authors applied more severe rocking stress than that used by Moon (1964), Lee (1964), and Park (1967) to the young albino rats, and produced significant or typical gastric ruminal ulcers.

Pappenheimer and Larimore (1924) described pathological changes in the tunica propria in the gastric ruminal ulcer of the rat produced by feeding with vitamine-A deficient diet and pointed out the pathological changes of exfoliation of the keratin layer particularly near the esophagus, of hyperplasia of the epithelial layer, accompanying with blunt processes of papillomatous processes growth extended to the tunica muscularis mucosae or to the tunica submucoosa through the tunica muscularis mucosae and of the inflammatory reaction associated with more or less edema in the tunica propria and submucoosa. The pathological pictures of the same layers in the experimental cases were fairly similar to the others.

In the characteristic histological changes of the tunica propria, Robert and Nezamis (1958) reported that the lesions are characterized first by a tremendous edema of the rumen wall in which the inflammatory cells or polymorphonuclear and round cells are contained and only after this has progressed for sometime do the ulcers develop. Also they added that the ulcers probably appear because the walls of the blood vessels are being compressed by the pronounced edema, and a state of malnutrition of the surface epithelium, which is already a vascular, develops. In 8 cases of 17 ruminal ulcers, the typical ulceration was found without the pronounced edema which might cause the ruminal ulcers according to the results of Robert and Nezamis (1958) and frequently the authors found that the small blood vessels and carpi-

REFERENCES

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**LEGEND FOR FIGURES**

**Fig. 1.** Control group. No histopathological changes were found. H-E, 100×.

**Fig. 2.** Experimental group. Severe hyperplasia of the epithelium and slight edematous change of the tunica propria were visible. H-E, 100×.

**Fig. 3.** Experimental group. Hyperplasia of the epithelium occurred in the area of hyperkeratosis. The inflammatory cells were infiltrated into the underlying tissue of the ulcer site.

**Fig. 4.** Experimental group. Severe edematous change of the tunica propria is visible. While the ulcer is formed in the area of hyperplasia. H-E, 100×.