Does Alcohol Cause Duodenitis?


Duodenal biopsies were taken from 6 patients during an acute episode of chronic alcoholism, and specimens of duodenum, jejunum and ileum were taken from 12 rats given alcohol over long periods of time. Specimens were examined by a combination of histologic, biochemical, and histochemical technics. No evidence of duodenitis was found.

It is held widely that excessive intake of alcohol can cause duodenitis.1-3 This is postulated to be a factor in the production of alcoholic pancreatitis. It has been suggested that duodenal inflammation could cause obstruction of the pancreatic duct,3 or might result in duodenopancreatic reflux.4 Despite these suggestions there is no histologic evidence that alcohol, in fact, does produce duodenitis. This report describes the results of a histologic, histochemical, and biochemical study of the duodenal mucosa in alcoholic patients and in rats given alcohol for long periods of time.

MATERIALS AND METHODS

Experiment 1

The subjects were 6 chronic alcoholics admitted to the hospital during a drinking spree. In the weeks before admission, the average consumption of alcohol had been 590 gm. of alcohol per man per day, usually in the form of a combination of cheap wines, spirits, and beer.

A typical case history was that of 1 patient who for 6 weeks before admission had drunk 2 oz. of rum, 220 oz. of beer and one bottle of port per day.

Peroral biopsies were taken under fluoroscopic control from the second part of the duodenum within 15 hr. of admission. The biopsies were examined under the dissecting and light microscopes with particular attention being paid...
to the amount of infiltration by inflammatory cells and the vascularity of the mucosa. Portions of specimens from 2 patients were taken for estimation of disaccharidase activity.

Experiment 2

A total of 24 male Wistar rats weighing 150–260 gm. were paired by weight and placed on an adequate synthetic diet. The sole source of fluid for 1 rat of each pair was alcohol. This was given in a concentration of 15% (v/v) for 150 days, and 30% for a further 70 days. Controls received water. Pair-feeding was used to insure that the intake of solid food of each rat given alcohol was identical with that of its control.

On Day 100 of the study, 2 pairs of animals were killed, and the remainder were killed at varying intervals after Day 150. Food and fluids were withdrawn 3 hr. before death. Animals were killed by a blow on the head and specimens of small bowel taken from the following areas: duodenum, 2 cm. distal to the pylorus; jejunum, 12 cm. distal to the pylorus; and ileum, 10 cm. proximal to the cecum.

Histology. Histologic examination of the small bowel mucosa was carried out on specimens from all rats. A portion of each specimen was fixed in formalin and stained with hematoxylin and eosin.

Histochemistry. In 3 pairs of rats killed on Day 200, a portion of each duodenal and jejunal specimen was examined histochemically for leucine aminopeptidase activity by the method of Nachlas et al.

Disaccharidase activities. Estimations of disaccharidase activities were performed on specimens from 8 pairs of rats killed after 180 days. In these, mucosal homogenates were prepared from a portion of each small bowel specimen. The mucosa was scraped off with a glass slide and immediately frozen on dry ice. It was then weighed and homogenized in distilled water in a Bellco glass homogenizer, the final concentration of mucosa being 20 mg./ml. The homogenate was placed in polypropylene microcentrifuge tubes, frozen again on dry ice and stored at −4°C.

Maltase, sucrase and lactase activities were assayed by the Tris-glucose oxidase method of Dahlqvist. All enzyme activities were expressed as μ moles of substrate hydrolyzed per minute per gram wet weight of mucosa.

RESULTS

Experiment 1

The appearance of each duodenal mucosal specimen was normal under both the dissecting and light microscopes. No evidence of duodenitis was found (Fig. 1). The disaccharidase activities of duodenal mucosa in the 2 specimens for which it was estimated (Table 1) were within the normal range.