EDITORIAL

GASTRIC ULCER AND FLUORIDE

In a recent issue Czerwinski and Lankosz reported that seven out of 60 retired workers (12%) in a Polish aluminum plant, who showed evidence of low-grade skeletal fluorosis, were afflicted with gastric ulcer; five of them had undergone gastric resection (1). Such a high incidence raises the question whether or not certain kinds of ulcers in the stomach and upper intestinal tract, encountered in a physician's practice, might be related to fluoride. Could our daily diet contain enough fluoride to induce this disease?

A cardinal feature of acute poisoning from inorganic fluoride compounds is severe vomiting, extreme pain in the epigastrium and gastric hemorrhages. At autopsy, ulcerations of the stomach and the upper intestinal tract are almost always present (2). These ulcers are believed to be induced by the corrosive action of hydrofluoric acid which is formed when a fluoride compound comes in contact with free hydrochloric acid which is normally present in the stomach at a concentration of about 0.1 molar (0.2 to 0.4%). Whitford et al. (3) observed that at a pH range of 1.85 to 5.50, fluoride penetrates the bladder tissue by nonionic diffusion of HF. This action was suggested earlier by Roholm who quoted the observations of Wieland and Kurtzahn that fluoride and silicofluoride, under the influence of the gastric hydrochloric acid, form hydrogen fluoride which penetrates the gastric mucosa in a non-dissociated state including corrosive changes. "For absorption of fluoride from low soluble compounds such as calcium fluoride, fluoroapatite and cryolite, the acidity of the stomach plays a decisive role" (4).

Roholm elaborates further on this phenomenon as follows: "The corrosive action of fluoride upon the skin and mucous membranes is not likely to be mediated by its acidity but by the fact that the non-dissociated HF molecule penetrates the epidermis and the mucosa and thus damages the underlying tissue. Therefore, not only hydrogen fluoride and silicofluoride have a corrosive action but all other acid solutions of fluoride as well, particularly bifluorides and silicofluorides" (5).

The degree of damage to the gastric mucosa i. e. the severity of the ulceration is therefore dependent on the amount of the fluoride compound ingested, the tightness of the bond of the fluoride ion in the molecule of the compound, and the acidity of the stomach of the individual patient. Other factors undoubtedly play a part in this action as, for example, the simultaneous presence of fat in the stomach which impedes absorp-

tion into the bloodstream (6).

In chronic fluoride poisoning from long-term intake of minute amounts of fluoride, sufficient hydrofluoric acid may be formed in the stomach to induce the symptoms of an "irritable stomach" namely, gastric pain and tenderness in the epigastrium, nausea, and vomiting. In about 1% of 1,100 pregnant women and young children receiving fluoride tablets (about 1.0 mg daily) for prevention of tooth decay, Feltman and Kosel observed these manifestations (7). These doses correspond to those ingested from the average daily consumption of fluoridated water. Roholm has observed gastric symptoms in 55 (80.9%) of 68 cryolite workers with skeletal fluorosis (8). Waldbott in 50 out of 52 cases of preskeletal fluorosis due to artificially fluoridated water (9).

The delicate lining of the gastrointestinal tract in young children and infants seems to be particularly susceptible to injury from fluoride as suggested by the occurrence of hemorrhages in the stomach and bowels of five infants who received 0.5 mg of fluoride/day in drops, an amount equal to the intake from 500 ml of fluoridated water. As soon as this medication was discontinued the hemorrhages disappeared (10). Gastric hemorrhages were also described in five newborn infants whose mothers had been exposed, during pregnancy, to fluoride fumes in a Czechoslovakian aluminum factory (11).

I have had occasion to review the record and the microscopic findings of a similar, rather dramatic, instance of fluoride-induced stomach ulcers. On August 24, 1962, the chief surgeon of the Ochsner Clinic in New Orleans consulted me about a nine-year-old boy, W.B.B., Jr. Gastric hemorrhages had necessitated the removal of a large portion of the stomach. After the boy's return home from the hospital, he promptly suffered another hemorrhage so severe that a part of the upper bowel had to be removed. This time, careful questioning revealed that several hours before the second incident, the boy had taken a fluoride tablet (0.5 mg) for prevention of tooth decay. The attending physician was convinced that the fluoride tablet had caused the hemorrhages. The child had been taking the tablets twice daily for at least six months.

The microscopic sections of his stomach revealed another remarkable phenomenon--the presence of so-called teleangiectasis, areas of widened capillary blood vessels below the surface of the stomach. This unusual finding further supports the causal relationship to fluoride, since teleangiectasis also occurs on the skin of patients treated with fluorine-containing cortisone ointments, but not if the cortisone molecule lacks fluorine (12).

The question arises whether or not the minute amounts of fluoride which are present in food and in drinking water would suffice to induce gastric ulcers and whether fluoride should be considered one of its causes, a possibility which, to date, has not received attention in the medical literature.

In the average person, such small amounts of fluoride would probably not suffice to adversely affect the gastric mucosa, particularly if buffered by the presence of non-acid food. But what about the patient with hyperacidity? Spasticity of the stomach associated with hyperacidity is usually interpreted by the radiologist as "irritable stomach" and attributed to strain and nervous tension. In my own experience, this condition is not uncommon in fluoridated communities. When these patients are placed on nonfluoridated water their symptoms subside. Whether or not such persistent, long-term "irritability" of the stomach may eventually lead to ulcers in an individual with hyperacidity should be of interest to the gastro-enterologist.

G.L.W.

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