Gastritis Caused by Ingestion of Eggs of Puffer Fish¹: A Case Report

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Tetrodotoxin is a neurotoxin, so gastrointestinal symptoms are very rare; these described in the literature are merely nausea and vomiting. Severe complications in the gastrointestinal tract caused by tetrodotoxin have not been radiologically reported. US and CT show thickening of the gastric wall and contraction of the lumen, and upper gastrointestinal series show shortening, lobulation and irregularity of the lesser and greater curvature of the body and antrum similar to the findings of corrosive gastritis.

Index Words: Gastritis, Stomach, US, Stomach, CT, Barium

Tetrodotoxin is a neurotoxin usually concentrated in the eggs of puffer fish; their accidental ingestion results in neurologic symptoms such as paresthesia and may lead to death when the amount of toxin is sufficient (1–4).

In the literature, there are a few descriptions of gastrointestinal symptoms after poisoning by tetrodotoxin (2, 3), but there has been no report concerning the radiological findings.

Several authors have attempted experimental studies concerning gastric mucosal damage by arterial infusion of tetrodotoxin and have documented the injury on gastric mucosa (5, 6); these were not, however, clinical studies.

We encountered a case of gastritis caused by ingestion of the eggs of puffer fish, and report this unusual case.

CASE REPORT

A 26-year-old man presented with vomiting and abdominal pain, symptoms which together with nausea he had shown since immediately after the accidental ingestion of puffer fish eggs one month earlier. Four of six persons attending that gathering had suffered from similar acute gastrointestinal symptoms and had been admitted to another hospital, where for ten days they underwent conservative treatment for toxic symptoms. After discharge, this patient persistently complained for about two weeks of continuing abdominal pain and nausea, and was then admitted to our hospital, but the other patients recovered uneventfully.

On admission, sonography showed a distended stomach containing retained food, circumferential wall thickening, and contraction of the gastric antrum, suggesting gastric outlet obstruction (Fig. 1a). Gastroscopic findings suggested corrosive injury at the gastric mucosa and the result of gastroscopic biopsy was chronic ulcer. Abdominal CT showed diffuse wall thickening and contraction of the gastric body and antrum, and visible small perigastric lymph nodes (Fig. 1b). Examination of the upper gastrointestinal tract showed shortening of the lesser curvature of the stomach, scalloping and irregularity of the gastric body, marked narrowing of the antrum, and a deformed duodenal bulb (Fig. 1c). Staining of sputum for acid-fast bacilli was negative and a serologic test for syphilis was non-reactive. The patient was supportively cared for but the symptoms were not relieved and vomiting persisted.

He underwent subtotal gastrectomy and gastrojejunostomy. On surgery, the stomach showed diffuse contraction, especially at the lesser curvature of the body and antrum, and there were multiple diffuse superficial ulcerations. The wall of the antrum was

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Fig. 1. Gastritis by ingestion of eggs of puffer fish in a 26-year-old man.
a. Transverse sonogram of the stomach shows distended body with retained foodstuff, and circumferential wall thickening and contraction of antrum (arrows).
b. CT scan at mid-abdomen shows wall thickening and contraction of gastric low body with small left paraaortic lymph nodes.
c. Upper gastrointestinal series show shortening, lobulation and irregularity of lesser curvature of high body and greater curvature of low body. The antrum reveals markedly narrowed lumen (arrows).
d. Pathologic specimen shows multiple mucosal ulceration with marked wall contraction. The submucosal and muscular layers of antrum are markedly thickened (arrows).

severely thickened and the pylorus was nearly obstructed. Pathologic examination of the resected specimen showed diffuse variegated mucosal ulceration with marked wall contraction; the submucosal and muscular layers were hypertrophied (Fig. 1d). Microscopic examination revealed acute ulcer with necrosis, infiltration of acute and chronic inflammatory cells, and diffuse infiltration of histiocytes with hemosiderin-laden macrophages. There was submucosal fibrosis and muscular thickening, and diagnosis based on a pathologic specimen was acute and chronic ulcers. The patient was discharged one week later and remained in good health.

DISCUSSION

Tetrodotoxin is a nonprotein, heat-stable neurotoxin, concentrated in the ovaries, kidneys, intestines and eggs of the puffer fish (1). This toxin possesses a local anesthetic property that disrupts sodium conductance by blocking the fast sodium channel during cell depolarization (2). The fish muscle during the nonreproductive season is nontoxic and in Oriental countries is considered a delicacy. Accidental ingestion of the eggs or intestines of the puffer fish produces neurologic symptoms. The signs and symptoms of tetro-
dotoxin intoxication begin with paresthesia and vomiting (2, 3), and as ascending paralysis develops, respiratory depression and cardiac dysrhythmia follow, and muscle twitching and convulsion may occur. In serious poisoning, hypotension, bradycardia, depressed corneal reflex and dilated pupil may be seen (2, 4). Mortality has been reported to be as high as 60% (2).

Tetrodotoxin is a neurotoxin, so gastrointestinal symptoms are very rare; those described in the literature are merely nausea and vomiting (2, 3). Severe complications in the gastrointestinal tract caused by tetrodotoxin have not been reported. In our case, gastritis was most likely induced by tetrodotoxin; before intoxication the patient showed no symptoms of an ulcer and gastrointestinal symptoms developed just after intoxication, as in the case of the others attending the gathering. On upper gastrointestinal examination and endoscopy, the findings were typical of corrosive gastritis. Except for eating eggs of the puffer fish, there were no other induced factors leading to gastritis. The pathologic findings were different from the usual pattern of gastric ulcer: in this case, the submucosa and muscle layers of the gastric wall were atypically hypertrophied and there was fibrosis; these findings are not usual in cases of gastric ulcer. The authors therefore concluded that this case was the first radiologically reported case of gastritis caused by the ingestion of eggs of the puffer fish.

Some authors have reported investigations of gastric mucosal damage caused by arterial infusion of tetrodotoxin (5, 6). Holzer et al. (5) explained the mechanism of gastric mucosal damage by tetrodotoxin. Disruption of the gastric mucosal barrier is quickly followed by an increase in gastric mucosal blood flow, which is thought to be a defense reaction to prevent further injury but is blocked by local arterial infusion of tetrodotoxin to the stomach. Inhibition of blood flow is associated with exaggeration of gross and histological injury to the mucosa, and this is permanently damaged. Esplugues et al. (6) reported the role of the platelet-activating factor in the action of tetrodotoxin; the administration of this alone does not induce any detectable mucosal damage, indicating that inactivation of the mucosal neuronal mechanism does not directly disrupt the mucosa. Local intra-arterial infusion of tetrodotoxin only augments the mucosal damage induced by the platelet-activating factor.

In our case, gastric mucosal damage was induced not by intravascular injection of tetrodotoxin but by eating puffer fish eggs containing tetrodotoxin. Gastric mucosal damage is therefore rather unlikely and we believe that this case of gastritis caused by ingesting the eggs of the puffer fish is very unusual.

REFERENCES

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