Clinical Case no. 13

37-year-old female

Long-term medical history

Nothing worthy of note.

Recent medical history

For a number of years the patient had experienced episodes of dyspepsia, pyrosis and epigastralgia. These disturbances progressively intensified over the 2 years prior to presenting to us, and included retrosternal pain, nausea, vomiting of bile and food, regurgitation of biliary liquid (she specified that in the morning the pillow was soiled with bile), slow digestion, hypo-anorexia, headache, and asthenia. Diagnostic tests (radiography and gastroesophageal-duodenal endoscopy) did not reveal any noteworthy alterations, and various therapies (gastric and prokinetic antisecretory agents) led to no appreciable results. During the last 10 months before presentation the patient had lost approximately 20 kg.

Physical examination

The patient was in poor general conditions of both nutrition and sanguification. Evident alterations of organs and systems were undetectable. Blood chemistry tests showed hypochromic anemia and hyposideremia. Hepato-duodenal-biliopancreatic ultrasound (US) and radiological examination of the esophagus, stomach and duodenum were negative. Esophago-gastro-duodeno-scopy revealed a moderate hyperemia of the distal esophagus and slight signs of antritis. Biopsy findings were irrelevant.

A functional study of the upper digestive tract was therefore planned:

- 24-hour esophageal-gastric pH monitoring revealed substantial mixed type duodeno-gastric and gastroesophageal refluxes (**see video - 1**);
- Gastric emptying studied by US was noticeably delayed, still incomplete beyond the sixth hour;
- Esophageal-gastro-duodenal manometry showed serious hyperdyskinesia of the duodenum, with diminished antral activity that, while still valid, was insufficient compared to the functional obstacle of the duodenum (**see video - 2**).

Diagnosis

On the basis of the patient's history and results of functional tests, the diagnosis was **gastroesophageal disease from alkaline reflux due to hyperdyskinesia of the duodenum (DGERD)**

Therapy

The purely functional nature of the disorder, that is, lacking any organic alterations above all at the gastric level, gave the precise indication for an **extramucosal duodenal myotomy - Nissen fundoplication**. The operation was performed according to the procedures described in Lecture no. 15 available in this web site and illustrated in the video attached thereto.

The post-operative course was regular, and the patient was released on a schedule of outpatient follow-up visits.

Outcome

A noticeable improvement of the patient's conditions was already evident soon afterwards: disappearance of pyrosis and epigastric pain, and a progressive and ultimately complete reduction of all other symptoms. At two months after surgery the patient was able to eat regularly and presented weight gain of approximately four kilos.

Functional tests were carried out ten months after surgery:

- Combined 24-hour gastroesophageal pH monitoring showed a marked reduction of duodeno-gastric reflux and the absence of gastroesophageal reflux (**see video - 3**);
- Gastric emptying times were within normal limits;
- Esophageal-gastro-duodenal manometry showed a noticeable reduction of pressure values in duodenal waves even after prostigminic stimulation, and recovery of the prevalence of antral over duodenal motility with resumption of motility coordination (Fig. 1).

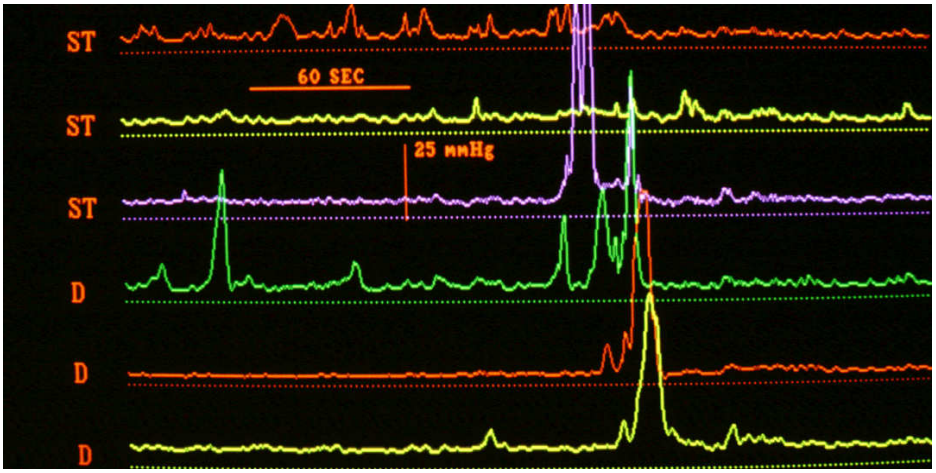

Fig. 1

After 24 months the patient returned to her normal weight and all symptoms had disappeared. Functional tests were repeated, and these confirmed - showing even further improvement - findings obtained at ten months following surgery.

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Remarks

This case regards a relatively young patient who had long complained of disturbances in the upper digestive tract, namely dyspepsia and regurgitation of biliary material. The factors characterizing her condition that commanded attention were the accompanying anorexia and significant weight loss. Even with such a serious clinical picture, common morphological examinations (radiography, endoscopy, etc.) were negative; medical therapy led to no appreciable

results. Before presenting to us her symptoms had further worsened, and physical conditions, as well as basic tests, confirmed the entity of the clinical picture.

At this point it must be said that the array of symptoms should have oriented her first physicians towards the diagnosis, or if nothing else at least towards the premise of a correct diagnosis. The triad of biliary regurgitation, dyspepsia and deterioration, beyond the other symptoms and in the absence of organic lesions, should have prompted first of all and without any shadow of a doubt the diagnosis of cardial incompetence and, secondly, the suspicion of a functional disorder within the antral-pyloric-duodenal complex. Indeed, the characteristics of the refluxate, without requiring any particular examinations, was already in itself a diagnostic indicator of alkaline gastroesophageal reflux that could depend exclusively on duodenal-gastric reflux. Functional tests (pH monitoring, esophageal manometry) confirmed these assumptions and led to the diagnosis of duodenal-gastroesophageal reflux disease (D-GERD).

As is well know, gastroesophageal reflux disease is relatively frequent in western populations, but this is generally thought to be acidic reflux. If, however, we examine the data emerging from patients undergoing long-term pH monitoring, while situations of manifest alkaline reflux (as in the case presented here) are fairly rare, the incidence of so-called mixed reflux is by far higher. This finding is often not interpreted for what it is worth, since the pH curve is easily readable as normal.

Alkaline gastroesophageal reflux may be asymptomatic, but nonetheless progressively harmful to the esophageal mucosa. Pathological duodenal-gastric reflux (duodenal-gastric reflux with normal antral clearing is entirely physiological) both dependent on antral adynamia and due to the prevalence of duodenal over antral motility, may be multi-symptomatic, above all in early stages of disease. It is nonetheless able to exert a deleterious action on the antral mucosa, gradually developing by degree into atrophy, metaplasia or dysplasia. Each of these in the long run can ultimately acquire neoplastic potential. The concomitant cardial incompetence, as shown by this case, is to be viewed as a favorable event, inasmuch as it exposes the presence and nature of the disorder even sooner.

Our experience (University of Genoa Surgical Clinic) is based only on later-stage cases of the gastro-duodenal motility disorder. It is quite possible that in early stage disease, as long as it is recognized, medical (prokinetic agents, proton pump inhibitors, etc.) and dietary approaches are able to restore coordination to the complex. Unfortunately, our series abounds with cases in which D-GERD had already induced serious and irreversible damage to the antral mucosa. Under these circumstances treatment consisted of total duodenal diversion, which, as is well-known, entails subtotal gastric resection.

In those patients, however, in whom the disorder is still in a functional phase (such as the case presented here), extramucosal duodenal myotomy (EDM) allows the surgeon to normalize antral-pyloric-duodenal coordination, restoring at the same time the prevalence of antral over duodenal motility. In this way correct gastro-duodenal function recovers both gastric egestive and antral clearing activity.
