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*Letters to the editor*

# Four-year follow-up of the first case of gastroesophageal reflux disease treated with endoluminal gastroplication in Japan

*To the Editor:* Various endoscopic treatments have been available to treat gastroesophageal reflux disease (GERD) in Western countries.[1] In Japan, a clinical trial of the endoluminal gastroplication (ELGP) method is presently in progress. We succeeded in performing ELGP for the first time in Japan in August 2002. Although results of approximately 2 years of follow-up studies are available in the literature, no other information about a more than 4-year follow-up is available. In this paper, we report on the first use of the ELGP method in Japan, from which we have obtained valuable information during a 4-year follow-up period.

The patient was a 53-year-old man with esophagitis (Los Angeles criteria grade A) diagnosed by esophagogastroduodenoscopy (EGD). Although administration of a proton-pump inhibitor (PPI, omeprazole 20 mg/day for 2 years) mitigated the symptoms, the patient strongly wished to stop the medication and was admitted for ELGP using an EndoCinch (C. R. Bard, Murray Hill, NJ, USA), which was performed in August 2002. Symptoms disappeared immediately after ELGP without treatment with a PPI. Subsequently, esophageal pH monitoring revealed that the fraction of time with pH below 4.0 did not exceed 4% during the subsequent 2 years of follow-up examinations. No symptoms recurred at 1, 2, 3, or 4 years, and the patient followed a good course with no PPI treatment. An EGD showed that one of the two plications had been dropped by 3 months postoperatively, but the other plication remained intact even after 4 years (Fig. 1).

Cases of long-term failure of ELGP have been attributed to the dropping of plications.[2] The likelihood of plications being dropped is proportional to the depth of suturing. Some authors draw attention to suction pressure. In the present case, we performed two plications. One of the two plications remained detectable by EGD in the fourth year. These findings can be considered to reflect the success of suturing of the plication, which was sustained for 4 years within or in the vicinity of the muscle layer. Additionally, not only the onset of new symptoms but also the recurrence of reflux esophagitis can be prevented provided that at least one plication remains. GERD is generally characterized by lesser severity in Japanese than in Western peoples,[3] and acid secretion is lower in Japanese as well.[4] With these facts in mind, it can be postulated that endoluminal surgery, like drug therapy,[5] is more likely to be effective in Japanese than in Western peoples. Our patient represents the first instance of ELGP performed in Japan, and hence provides data from the longest follow-up available. The patient has been followed up and has not taken oral antacids for

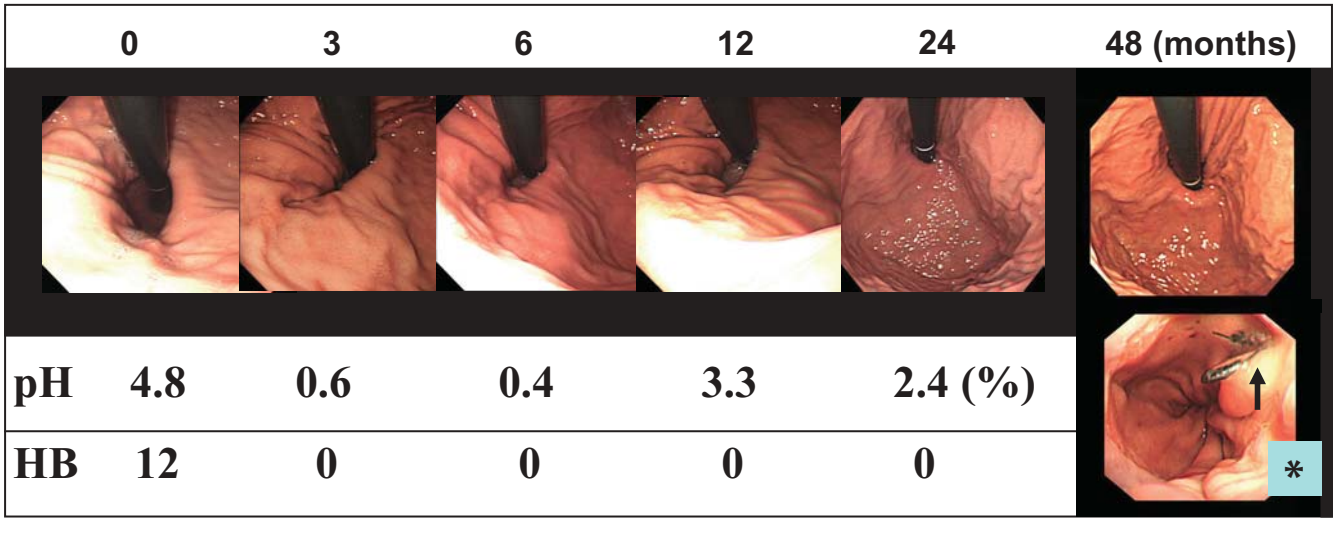


**Fig. 1.** Four-year follow-up of the first Japanese use of the endoluminal gastroplication (ELGP) method for gastroesophageal reflux disease diagnosed by esophagogastroduodenoscopy (EGD). Esophageal pH monitoring (percentage of time with pH below 4.0) and the heartburn score reveal a remarkable improvement during the 4 years after ELGP. *pH*, percentage of time with pH below 4.0 during esophageal pH monitoring; *HB*, heartburn score (heartburn frequency × heartburn severity). *EGD revealed that one plication (*arrow*) remained intact even after 4 years

4 years. EndoCinch is expected to provide an improved long-term prognosis in Japanese patients. It is hoped that a nationwide survey will be undertaken to obtain extensive data on its long-term follow-up.

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## Influence of phlebotomy on iron-related gene expression levels in the livers of patients with chronic hepatitis C

*To the Editor:* Excess iron accumulation is often seen in patients with chronic hepatitis C (CH-C),[1] and iron reduction is an effective treatment option for decreasing hepatocellular injury,[2–4] but the underlying mechanisms of iron overload are still unclear. Over the last few years, a number of genes participating in the regulation of iron homeostasis have been newly identified, and there is now substantial evidence that the liver plays a central role in determining iron balance in the body.[5] Therefore, we evaluated the influence of phlebotomy on iron-related gene expression levels in the liver of CH-C patients.

The study included seven chronically hepatitis C virus (HCV)-infected patients (Table 1). Phlebotomy was performed biweekly, with 200 or 400 ml of blood removed, depending on the patient's response. When serum ferritin levels reached 10 ng/ml, the initial period of phlebotomy was considered complete, and if the serum ferritin rebounded, maintenance phlebotomy was performed as needed to maintain the serum ferritin level below 20 ng/ml. Liver tissue was obtained by percutaneous needle biopsy at the start and after the phlebotomy, and mRNA was extracted from a portion of biopsy samples. HAMP (encoding hepcidin), SLC40A1

**Table 1.** Profile, phlebotomy, and changes of laboratory data and hepatic iron-related gene expression levels in patients with chronic hepatitis C

| Patient no. | Age (years)/ Sex | Phlebotomy months/ volume (ml) | Hemoglobin (g/dl) | | Ferritin (ng/ml) | | ALT (IU/l) | | Hepatic messenger RNA expression levels (/GAPDH) ($\times 10^2$) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | HAMP | | SLC40A1 | | TFRC | | TFR2 | |
| | | | Before | After | Before | After | Before | After | Before | After | Before | After | Before | After | Before | After |
| 1 | 49/M | 12/4000 | 14.8 | 13.6 | 269 | 33.0 | 94 | 38 | 180 | 28 | 53 | 57 | 0.63 | 1.0 | 37 | 59 |
| 2 | 33/M | 13/3600 | 15.1 | 13.3 | 171 | 20.5 | 102 | 68 | 69 | 11 | 38 | 44 | 0.93 | 0.94 | 28 | 24 |
| 3 | 59/M | 14/2800 | 15.3 | 13.5 | 397 | 31.2 | 128 | 79 | 220 | 17 | 29 | 17 | 0.59 | 1.1 | 45 | 48 |
| 4 | 45/F | 12/2400 | 14.5 | 12.9 | 210 | 19.2 | 145 | 87 | 73 | 14 | 58 | 37 | 0.68 | 0.78 | 34 | 58 |
| 5 | 65/F | 12/2000 | 13.7 | 12.4 | 274 | 18.6 | 59 | 47 | 150 | 8.1 | 36 | 41 | 0.66 | 1.3 | 37 | 40 |
| 6 | 62/M | 6/2000 | 14.2 | 13.0 | 168 | 42.8 | 115 | 39 | 50 | 23 | 24 | 16 | 0.69 | 0.98 | 16 | 71 |
| 7 | 60/F | 12/1800 | 12.9 | 12.0 | 71 | 8.0 | 66 | 35 | 33 | 19 | 36 | 39 | 0.85 | 1.1 | 27 | 35 |
| Mean ± SD | | | 14.4 ± 0.8* | 13.0 ± 0.6* | 223 ± 103* | 24.8 ± 11.6* | 101 ± 31* | 56 ± 21* | 110 ± 72** | 17 ± 6.9** | 39 ± 12 | 36 ± 15 | 0.72 ± 0.12** | 1.00 ± 0.16** | 32 ± 9.3 | 48 ± 16 |

ALT, alanine aminotransferase; GAPDH, glyceraldehyde-3-phosphate dehydrogenase
*Statistically significant different at $P < 0.01$ (paired *t* test)
**Statistically significant different at $P = 0.01$ (paired *t* test)