VIDEO CASE REPORT

Effects of endoscopic gastric plication on portal pressure gradient in a patient with nonalcoholic steatohepatitis cirrhosis

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CASE DESCRIPTION

Nonalcoholic fatty liver disease is the most common cause of chronic liver disease worldwide, with the prevalence approaching 90% in patients with obesity. Nonalcoholic steatohepatitis (NASH) is an aggressive form of nonalcoholic fatty liver disease with an increased risk of progression to cirrhosis. To date, the only treatment for NASH is lifestyle modification (LM).[1] Previous studies showed that at least 7% to 10% total weight loss is required for improvement in liver steatosis, inflammation, and fibrosis regression.[2] Nevertheless, less than 10% of patients who undergo LM achieve this threshold, leaving the majority of patients with NASH undertreated.[2]

For patients who have progressed to cirrhosis, the determinant of cirrhosis decompensation and mortality is portal hypertension, defined as a hepatic venous pressure (HVP) gradient of >5 mm Hg.[3] Traditionally, the HVP gradient is measured transjugularly by subtracting the free HVP from the wedged HVP, which is used as a surrogate for portal venous pressure (PVP) (Fig. 1). More recently, an EUS-guided approach to measure the portal pressure gradient (PPG) has been developed, which allows a direct measurement of PVP and HVP (Fig. 2).[4]

This case represents the convergence of endobariatrics and endohepatology. Specifically, in this video, we demonstrate the use of endoscopic gastric plication (EGP) to treat obesity and NASH in a patient with cirrhosis, as well as the application of EUS-guided PPG measurement to monitor its changes after EGP (Video 1, available online at www.giejournal.org).

Our patient was a 68-year-old woman with class III obesity (body mass index of 42.4 kg/m^2) and compensated NASH cirrhosis. Her MELD-Na score was 18 with Child-Pugh class A. Her blood work was notable for alanine aminotransferase of 60 U/L, platelets of 120 k/μL, and international normalized ratio of 1.1. Homeostatic model assessment for insulin resistance, which is a surrogate for insulin resistance, was 8.8. She underwent an attempted Roux-en-Y gastric bypass, which was aborted due to cirrhosis. She denied alcohol use. Transient elastography suggested cirrhosis with liver stiffness of 17 kPa.

Before EGP, she underwent EUS-guided PPG measurement. Specifically, a transgastric transhepatic puncture with a 25-gauge FNA needle into the hepatic vein was achieved. The manometer and needle were flushed with heparinized saline solution to prevent occlusion within the needle (Fig. 3). Turbulent flow was observed,

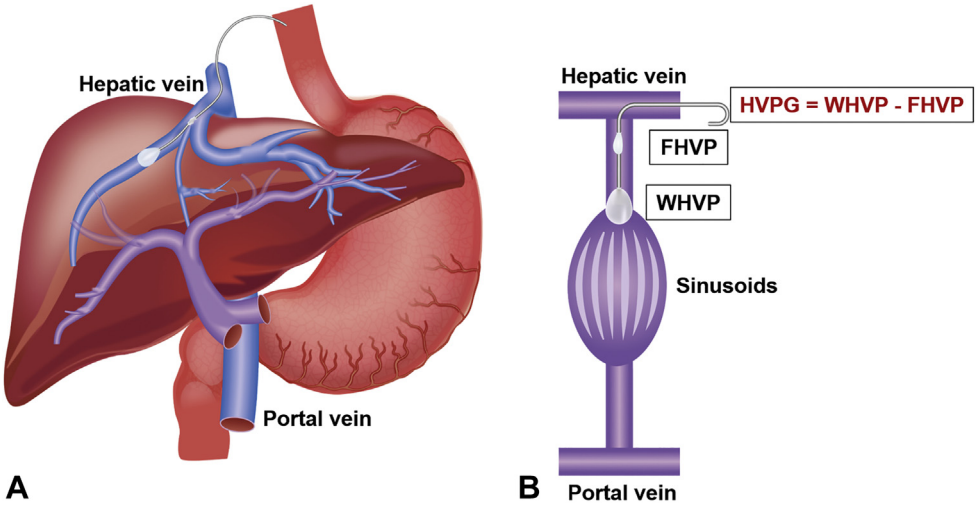

Figure 1. Transjugular hepatic venous pressure gradient (HVPG) measurement. **A,** A balloon-tipped catheter is inserted into a hepatic vein, where retrograde occlusion detects wedged hepatic venous pressure (WHVP), which is a surrogate for portal venous pressure (PVP) in cirrhotic patients, and keeping the catheter "free" in the hepatic vein detects free hepatic venous pressure (FHVP). **B,** HVPG represents the difference between WHVP and FHVP.

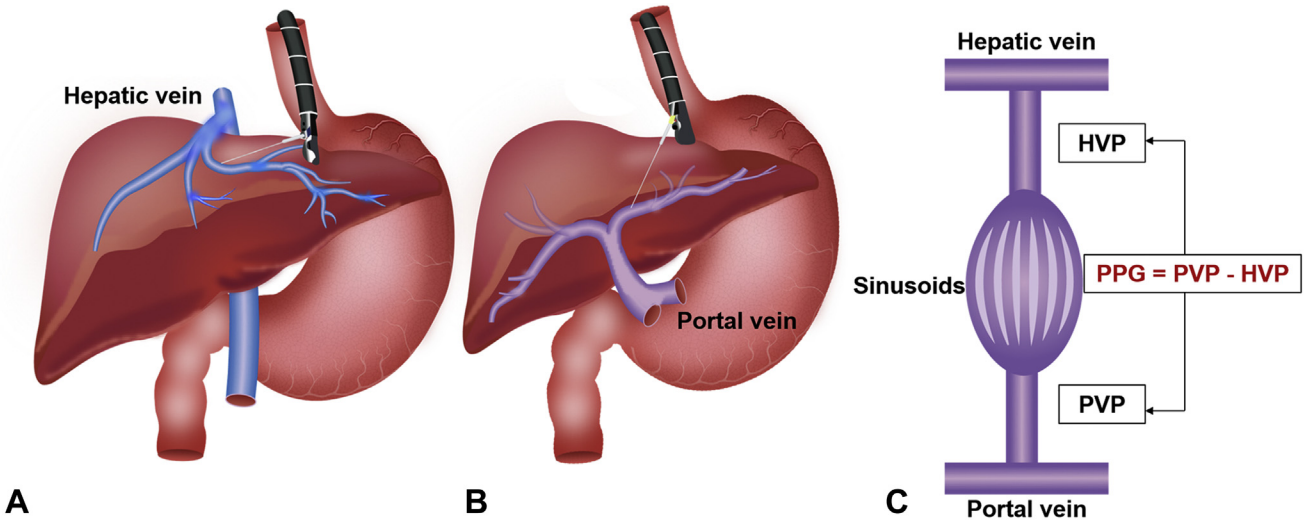

Figure 2. Endoscopic ultrasound-guided portal pressure gradient (PPG) measurement. **A,** Hepatic venous pressure (HVP) measurement. **B,** Portal venous pressure (PVP) measurement. **C,** PPG represents the difference between PVP and HVP.

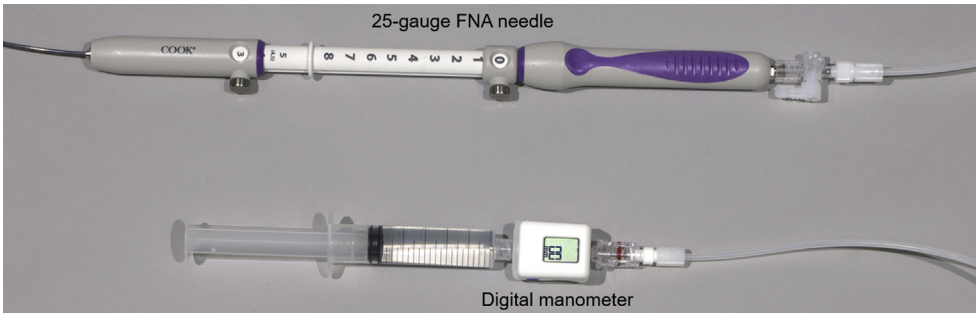

Figure 3. EUS-guided portal pressure measurement device consists of a 25-gauge FNA needle and compact manometer.

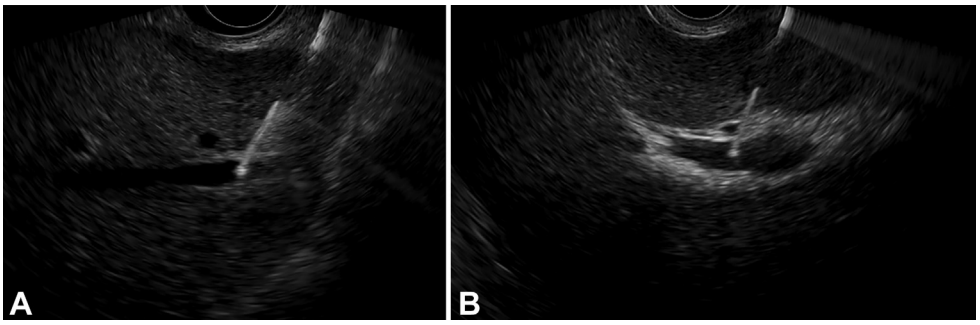

Figure 4. EUS-guided measurement of **(A)** hepatic venous pressure (HVP) and **(B)** portal venous pressure (PVP).

confirming good position within the vessel. The manometer reading rose and eventually plateaued, and this number was recorded. The process was repeated at least 2 more times, and the average number represented the HVP (Fig. 4A). The needle was slowly withdrawn under Doppler visualization to mitigate postprocedural bleeding. Subsequently, the portal vein was punctured, and the same steps were repeated to measure the PVP (Fig. 4B**)**. In this case, the average HVP and PVP were 17 mm Hg and 28.5 mm Hg, respectively. Therefore, her PPG before EGP was 11.5 mm Hg. The procedural time was 17 minutes.

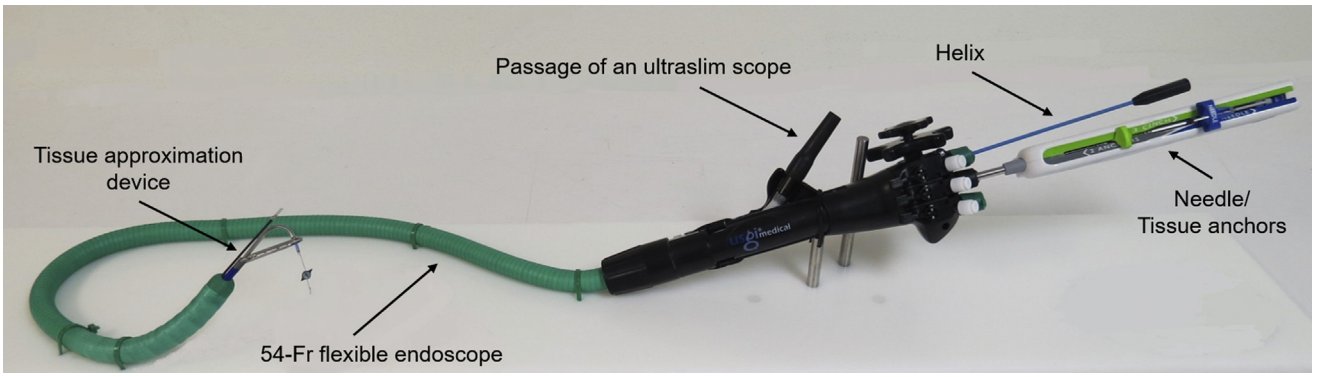

Figure 5. Endoscopic plication device.

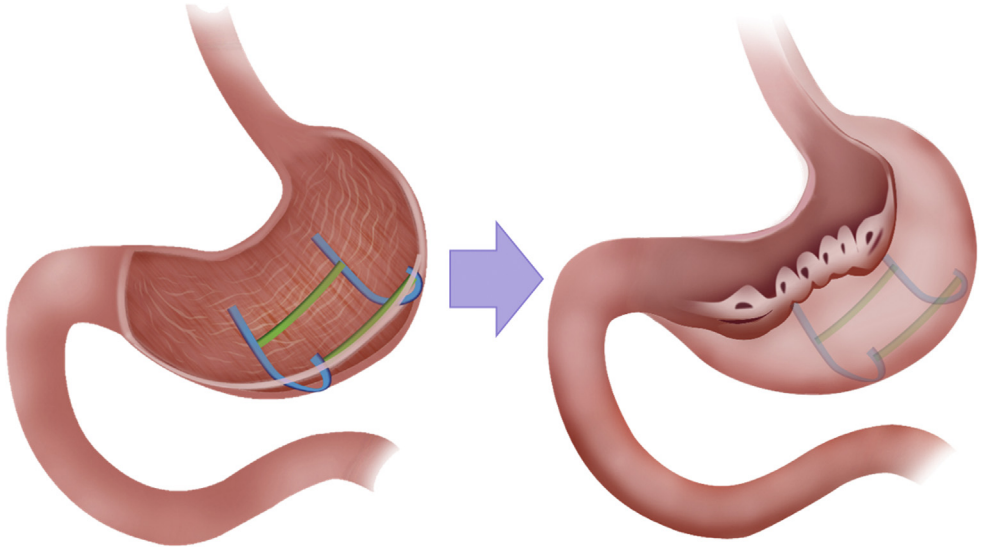

Figure 6. Endoscopic gastric plication procedure. Gastric plications are placed in the gastric body via a belt-and-suspenders approach. *Blue lines* represent belt plications, which reduce the gastric width. *Green lines* represent suspender plications, which reduce the gastric length.

The patient then underwent an EGP. Although the procedure has been shown to be safe and effective in treating obesity,[5] its safety and efficacy profiles in patients with cirrhosis were unknown. During the procedure, an endoscopic plication device (Fig. 5), which has Food and Drug Administration clearance for tissue approximation, was used to place plications using a belt-and-suspenders pattern (Fig. 6).[6] Specifically, the first set of plications was placed in the distal gastric body perpendicular to the length of the stomach to reduce its width. The second and third sets of plications were placed longitudinally to shorten the gastric length. The last set of plications was then placed at the proximal gastric body to further reduce its width. For this case, a total of 17 plications were placed, with 8 being placed in the distal belt, 6 being placed in the suspenders, and 3 being placed in the proximal belt. In this patient, the gastric body was shortened by 19 cm (Fig. 7). The procedural time was 63 minutes with fellow participation.

After the procedure, the patient followed LM as per our protocol by counting daily caloric intake and decreasing carbohydrate consumption. At 9 months, her weight decreased from 279 to 255 pounds, representing an 8.6% total weight loss. Her body mass index decreased from 42.4 to 38.8 kg/m^2. On transient elastography, her liver stiffness decreased from 17 to 7.6 kPa, suggesting regression from stage 4 to stage 2 fibrosis. Homeostatic model assessment for insulin resistance decreased from 8.8 to 4.3. A follow-up PPG test showed a PPG of 8 mm Hg, representing a 30% reduction.

In conclusion, this video demonstrates the safety and efficacy profile of EGP for the treatment of compensated NASH cirrhosis with improvement in liver fibrosis and PPG.

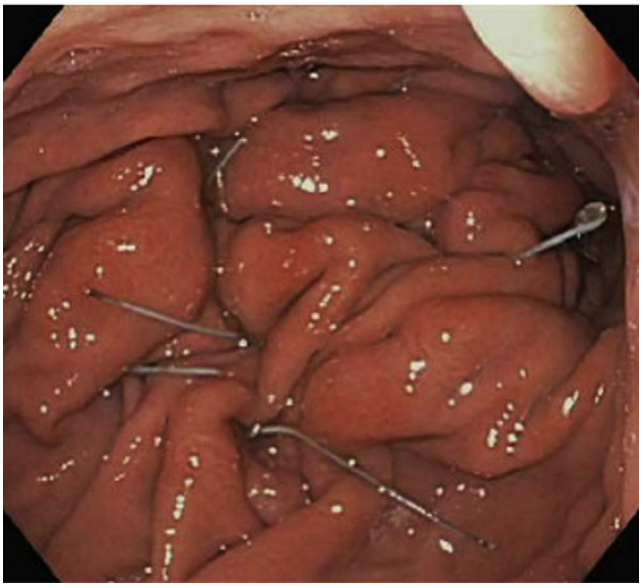

Figure 7. Endoscopic findings at 9 months after endoscopic gastric plication.

DISCLOSURE

All authors disclosed no financial relationships.

Abbreviations: EGP, endoscopic gastric plication; EUS, endoscopic ultrasound; HVP, hepatic venous pressure; LM, lifestyle modification; NASH, nonalcoholic steatohepatitis; PPG, portal pressure gradient; PVP, portal venous pressure.

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