Inside-out tract re-cannulation for G-tube replacement

To the Editor,

A 61-year-old male patient with a history of post-stroke dysphagia, necessitating enteral nutrition through percutaneous endoscopic gastrostomy (PEG) since >12 months presented after his caregiver had noticed tube loss of unclear duration estimated at 8-12 hours. The patient was on apixaban due to atrial fibrillation, and the stoma site appeared unremarkable without significant granulation and/or signs of infection. However, bedside attempts to carefully re-cannulate the stoma opening by a guidewire to replace for a balloon-type PEG tube failed, insomuch so that urgent upper endoscopy was warranted. Esophagogastroduodenoscopy identified the PEG stoma with the internal bumper already having passed (Fig. 1A). As under endoscopic control the external guidewire insertion was impossible, we considered "inside-out tract re-cannulation". Near-view within the stoma cavity succeeded in identifying the orifice of the PEG fistula (Fig. 1B, asterisk), which was selectively cannulated under endoscopic vision using a 0.035-inch guidewire with a hydrophilic tip (Fig. 1C). Next, the puncture cannula included in the new PEG tube system was advanced over the guidewire (Fig. 1D) to allow for placement of the silk thread into the stomach (Fig. 1E). After external extraction of the thread, the new PEG tube was positioned without complications as per standard procedure (Fig. 1F).

Tube dislodgment mostly related to excessive external pulling by mentally impaired patients ranks amongst the most common long-term complications of PEG tubes, ranging from 13-29% and implying significant healthcare costs [1].

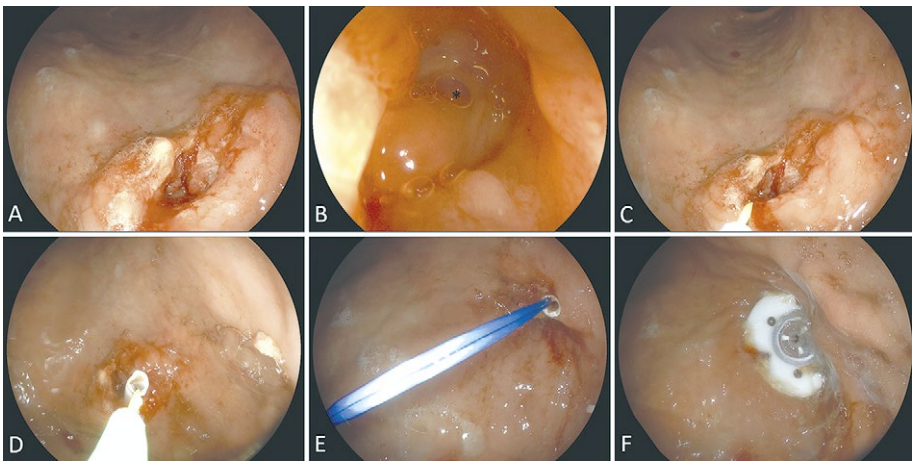

Fig. 1. (A) The stoma site with the internal retention bumper passed. (B) Near-view of the stoma cavity identifying the shrunken orifice to the PEG stoma, (C) which was cannulated from inside-out with a 0.035-in guidewire with a hydrophilic tip. (D) The puncture cannula provided within the new PEG system advanced from externally, (E) over which the rubber thread was next introduced, grasped and orally extracted by scope withdrawal. (F) Final result of the new G-tube placed as per standard procedure in the pull technique.

The recently published European Society of Gastrointestinal Endoscopy (ESGE) guidelines recommend in the case of late (more than 4 weeks) tube dislodgement the insertion of balloon-type tube, bedside ("blind"), through the established tract as soon as possible to prevent tract closure [2]. Oftentimes, a Foley catheter, introduced by the nurse and/or physician, used to maintain the tract patency as a temporary bridge to PEG tube replacement is easily achieved and safe.

However, in general, after the failure of the external reinsertion of a balloon-type tube, the placement of a new PEG tube at a different site is usually pursued. For patients receiving direct oral anticoagulants (DOACs) it is recommended their discontinuation for 48 to 72 hours prior to the primary PEG insertion, relative to the specific drug and renal function. This situation implies the specific risk of PEG tube placement, as well as significant treatment delays with prolonged hospitalization and need for an alternative temporary nutrition access, e.g. by a central venous line [3].

The inside-out tract re-cannulation of PEG stoma may represent a valuable rescue technique with special clinical value, in patients with altered coagulation, that circumvents re-puncture and related bleeding risk. However, this approach has only once been reported in the literature before [4. 5].

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Conflicts of interest: None.
DOI: 10.15403/jgld-3821

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Gastrointestinal AL amyloidosis: a rare but severe masquerader

To the Editor,

We read with great interest the article published by Luigetti et al. [1], which presents a retrospective cohort of patients with transthyretin (ATTR) gastrointestinal amyloidosis (GIA). Of the 39 patients in this single center in Italy, 82% reported at least one gastrointestinal symptom, most commonly weight loss. Upper gastrointestinal symptoms were less common than lower ones in this cohort (66.7% vs. 35.9%, p=0.01). These findings underscore the frequency of gastrointestinal symptoms in GIA, and subsequently, the role of gastroenterologists in evaluating and diagnosing patients with amyloidosis.

Furukawa et al. [2] recently in the *Journal of Gastrointestinal and Liver Diseases* referred to a case with a localized gastric light-chain (AL) GIA. The nuances and challenges of endoscopically diagnosing GIA were features of this case, and the authors highlighted that early diagnosis of local disease could have a favorable prognosis.

We recently cared for a 77-year-old female with a history of hypertension, ruptured brain aneurysm, and chronic kidney disease who presented with 5-months of nausea, vomiting, diarrhea, left lower quadrant (LLQ) abdominal pain, and 20-pound weight loss. She had been previously treated for dyspepsia without relief. On physical exam, she was hypotensive, tachycardic, tender to palpation in the LLQ, and had 1+ pitting pedal edema. Laboratory analyses revealed normocytic anemia (hemoglobin 9.4 g/dL), acute kidney injury (creatinine 1.73 mg/dL, baseline 1.1-1.3), and electrolytes within normal limits. A stool pathogen panel was negative. Esophagogastroduodenoscopy (EGD) noted scattered erosions in the gastric antrum, three duodenal ulcers without stigmata of recent bleeding (Forrest grade III); flexible sigmoidoscopy revealed diffusely friable exudative colonic mucosa with ulceration and mild blood oozing throughout the sigmoid and descending colon (Fig. 1A-B). Pathology revealed transitional type mucosa with focal amyloid deposition within the stomach, the muscularis mucosa of the duodenum, and extensive amyloid deposition within the left colon confirmed by Congo Red staining (Fig. 1C). Computed tomography scan revealed a thickened gastric wall and persistent colonic wall thickening (Fig. 1D). Bone marrow biopsy with flow cytometry demonstrated plasma cell myeloma, confirming the diagnosis of AL GIA. Her hospital course was complicated by multiple segmental and sub-segmental pulmonary emboli requiring systemic anticoagulation. She underwent treatment for multiple myeloma with cyclophosphamide-bortezomib-dexamethasone. She developed recurrent gastrointestinal bleeding and underwent repeated EGDs that noted gastric and duodenal ulcers with stigmata of bleeding. An inferior vena cava filter was placed. Following discharge, her functional status continued to decline. The patient was transitioned to hospice and passed away two months after diagnosis.

Although Luigetti et al. [1] reported that upper gastrointestinal symptoms were less common in their cohort, our patient presented with both upper and lower gastrointestinal symptoms. ATTR amyloid, as presented in the Luigetti et al. [1] cohort, is

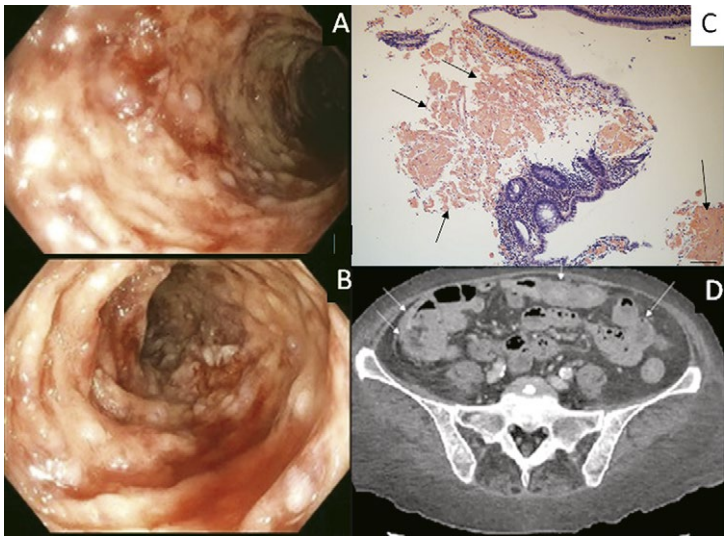

Fig. 1. Endoscopic images demonstrating diffusely friable, exudative mucosa in the descending colon (A) and sigmoid colon (B). Amyloid deposition (C, black arrows) within the muscularis mucosa of the duodenum, with underlying fibrosis (C, black arrows). Axial computed tomography scans revealed diffuse colonic wall thickening (D, white arrows).

much more common than AL amyloid [3-5]. AL amyloidosis is commonly associated with hematologic malignancies, especially plasma cell myeloma [4]. The overall prognosis for AL amyloidosis is worse in those with gastrointestinal involvement [4].

Unlike the Furukawa et al. [2] case, our patient presented with systemic involvement, had a poorer prognosis, and was diagnosed based on histologic assessment from the duodenum and colon instead of the stomach.

Gastrointestinal amyloidosis has an incidence of approximately 1 in 100,000, but biopsy proven GIA accounts for only 3.3% of all amyloid cases [4]. Gastrointestinal amyloidosis is difficult to diagnose due to its variable etiologies and non-specific clinical symptoms [1, 2, 4]. Treatment is dependent on the type of amyloidosis. AL amyloidosis is treated with chemotherapy directed at plasma cell dyscrasia. AA amyloidosis is often treated with anti-inflammatory and immunosuppressive therapies, while single organ involvements such as renal or hepatic amyloidosis may be amenable to organ transplantation [4]. Additional treatment, including dietary modification and pharmacologic therapy can be individualized based on patient's symptoms.

The findings presented by Luigetti et al. [1] and Furukawa et al. [2] highlight the frequency and variety of gastrointestinal symptoms in amyloidosis and that early diagnosis and treatment can minimize morbidity and mortality. Clinicians should keep GIA as a differential diagnosis in middle-age and elderly patients presenting with symptoms including gastrointestinal bleeding, malabsorption, nausea, vomiting, and protein-losing enteropathies, especially in the presence of cardiac and renal diseases. Future studies should assess the relationship between demographics, comorbidities, and mortality rates of GIA in larger cohorts.

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Conflicts of interest: None.
DOI: 10.15403/jgld-3928

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COVID-19 induced haemobilia: a novel entity

To the Editor,

An 81-year-old man admitted with fever, fatigue, and weakness was diagnosed with coronavirus disease 2019 (COVID-19) and hospitalized. During his stay he developed hypoxia and rapid deterioration of clinical course, being admitted to the intensive care unit (ICU), intubated and placed on mechanical ventilation with the diagnosis of acute respiratory distress syndrome (ARDS). After 20 days in ICU, his follow-up in the ward was uneventful until he suddenly started vomiting. Biochemistry at that time showed a 20-

fold rise in alanine aminotransferase (ALT) and aspartate aminotransferase (AST) (ALT=851 IU/L; AST=745 IU/L). Abdominal ultrasound revealed echogenic material in the hydropic gallbladder (Fig. 1A) and dilated intrahepatic and common bile duct. Endoscopic retrograde cholangiography disclosed blood clots in the common bile duct and bleeding from the papilla of Vater; blood clots were evacuated by endoscopic sphincterotomy, and lastly plastic biliary stents were inserted (Fig. 1B).

One week later a 54-year-old man presented with acute onset of vomiting, epigastric and lower right abdominal pain. In his history, he required ICU stay for 53 days due to COVID-19; on the 10th day of ward follow-up, he developed these complaints. Laboratory tests at that time showed increased levels of liver function tests [ALT=368 IU/L, AST=257 IU/L, gamma glutamyl transpeptidase (GGT) = 1128 IU/L]. Initial diagnosis was acute cholangitis on an abdominal ultrasonography and computed tomography scan which revealed high-attenuation blood within the hydropic, thick-walled gallbladder and common bile duct, in addition to dilated intrahepatic bile ducts and common bile duct (Fig. 1C). The patient underwent ERCP procedure that disclosed papilla hemorrhage (Figure 1D). Blood clots were evacuated by sphincterotomy, and plastic biliary stents were inserted.

The main conclusion inferred from these cases is the differential diagnosis among critically ill patients with cholestatic disease containing several clinical conditions that should be taken into regard such as ischemic cholangiopathy, cholestasis in sepsis, drug induced liver injury due to antibiotics which are generally used in ICU and cholangitis due to choledocholithiasis. Ischemic cholangiopathy is a clinical condition in which focal or extensive bile duct injury due to insufficient blood flow occurs including laboratory abnormalities suggestive for cholestasis. Secondary sclerosing cholangitis (SSC) is described as cholestatic liver injury in ICU patients without a history of liver disease [1]. Long-term hypotension, vasopressor administration and mechanical ventilation which lead to ischemic injury of biliary epithelium are involved in the pathogenesis. Since a correlation between severe hypotension and onset of cholestasis is detected, ischemic cholangiopathy remains as the main concept of the underlying mechanism. However, the gallbladder epithelium is involved in the process of COVID-19 extrapulmonary manifestations. To improve our understanding of these cases, coagulation abnormalities as a result of COVID-19 related cardiovascular complications must be focused on. Uncontrolled immune response, direct viral invasion, endothelial damage, and medications given to patients which may interfere with blood thinners and promote thrombosis or bleeding are some proposed mechanisms [2]. Venous and arterial thromboembolism, pulmonary embolism, acute limb ischemia has been described in patients with COVID-19 secondary to a hypercoagulable state [3 ,4]. Other complications due to hypercoagulation might worsen the clinical picture, being the main reason of organ failure in severe COVID-19 cases.

In conclusion, the possibility of haemobilia development after severe COVID-19 illness should be considered. Since timely diagnosis and treatment play an essential role in such life threating complications, clinicians must be aware of early recognition of related symptoms and laboratory indicators of liver damage and should not relate elevated liver enzyme to disease progression.

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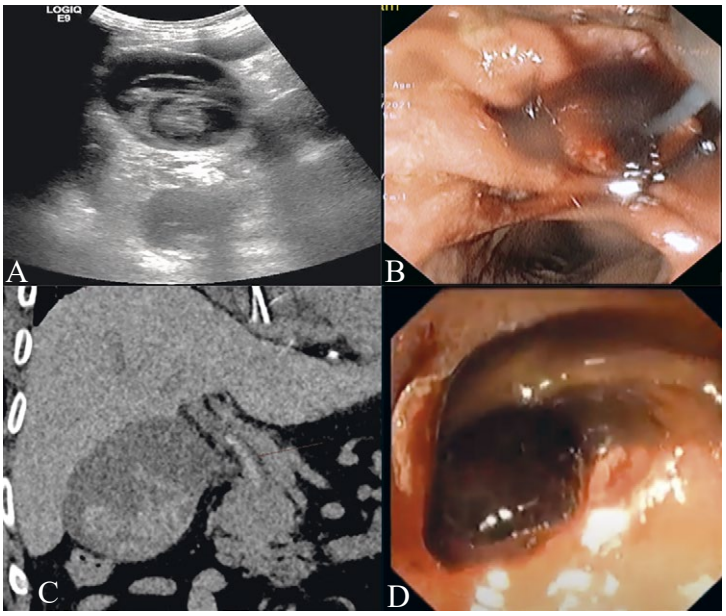

Fig. 1. A) Ultrasound: echogenic material in the hydropic gallbladder; B) Endoscopic retrograde cholangiography (ERCP): clots in the common bile duct and bleeding from the papilla of Vater; C) Computed tomography scan: blood within the hydropic, thick-walled gallbladder and common bile duct in addition to dilated bile ducts; D) ERCP:hemorrhage from the papilla.

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Conflicts of interest: None.
DOI: 10.15403/jgld-4043

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Anticoagulant treatment in cirrhotic portal vein thrombosis – how wide can we open the window?

To the Editor,

We read with great interest the article of Florescu et al., [1] wherein the authors demonstrated that in half of the cirrhotic patients receiving anticoagulant (AC) treatment for non-malignant portal vein thrombosis (PVT) the thrombus regressed and, in some cases partial recanalization was obtained. Overall mortality was lower in patients receiving AC treatment; however, the hemorrhagic complications were higher than previously reported [1, 2]. It is worth mentioning that this is one of the few studies demonstrating a survival benefit of AC treatment after PVT recanalization, and one of the largest cohort of cirrhotic PVT patients receiving AC. This issue is of interest, as the controversies regarding PVT influence on LC mortality are remaining [3]. It is still not clear if PVT is the consequence of end-stage liver disease or a cause of liver cirrhosis (LC) progression [4]. However, the previous studies did not evaluate the role of AC in PVT treatment in compensated or decompensated LC, and they did not take into account the type of PVT: acute or chronic, partial or total [5].

From our experience, the anticoagulant treatment is not so effective and safe for cirrhotic non-malignant total PVT. We prospectively evaluated 54 cirrhotic patients (mean age 58.91±8.98 years, alcoholic etiology: 35%, Child-Pugh class B: 48% and C: 52%) diagnosed with total non-malignant PVT. Twenty-seven were treated with enoxaparin 0.1 UI/Kgc b.i.d. for 6 months and 27 patients received no anticoagulant treatment (control group). The two study groups were matched by age, gender and Child–Pugh class. All patients were evaluated by Doppler abdominal ultrasound and computed tomography. After 6 months follow-up partial recanalization was obtained in 5 patients (18.5%) receiving AC treatment and in 3 patients (11.1%) in the control group. PVT aggravated by extension to superior mesenteric vein in 4 patients (14.8%) receiving AC compared to 7 patients (25.9%) in the control group. The survival rate at 6 months was 76.35% in the treated group vs 79.62% in the control group (p=0.241). There were 9 hemorrhagic events, 5 (18.5%) patients that received AC treatment (3 non-variceal upper digestive bleeding, 1 upper digestive bleeding - post band-ligation ulcers and 1 intracerebral hemorrhage), and 4 (14.8%) patients in the control group (variceal upper digestive hemorrhage). The efficacy of the enoxaparin in cirrhotic patients with decompensated LC and total PVT was low and the hemorrhagic events complicated the disease evolution more frequently during enoxaparin treatment. Our cohort was very small for drawing clear conclusions regarding the indication of enoxaparin treatment for patients with total PVT. We consider that data consisting of real-life efficacy and safety of AC in LC should be reported according to LC staging and PVT classification.

All these results suggest that the AC window should be carefully opened in patients with decompensated LC, and the balance between risk and benefits should be judiciously assessed.

In conclusion, even if, overall, AC treatment seems to be safe and efficient in patients with LC, we still need to refine the AC treatment indication. To identify the patients that will have the best benefits from this treatment, we need prospective studies including large cohorts of patients with PVT. A national registry including cirrhotic patients with PVT could overcome the shortcomings caused by the low prevalence of PVT in LC. We have to demonstrate, using homogeneous cohorts of patients, who is the best candidate for AC treatment, which AC should be used, and when we have to close the "anticoagulation window". The BAVENO VII Consensus which just presented their recommendation could be used as a framework for designing new studies.

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Conflicts of interest: None.
DOI: 10.15403/jgld-4081

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Reply,

To the Editor,

We thank Girleanu et al. [1] for their interest in our manuscript [2] and their comments are more than welcomed. However, we also have several remarks to make. First, the prevalence of portal vein thrombosis (PVT) in liver cirrhosis (LC) is high, up to 26% of liver transplant (LT) candidates [3] and also its incidence is high ranging from 4.6 to 30.5% according to the recent study by Senzolo et al. [4]. A meta-analysis by Zanetto et al. [5] also showed that PVT is common in LT candidates, and it is associated with higher short- and medium-term mortality after LT (1-year mortality 13.5% in recipients with PVT vs 9.9% in those without, p<0.0001).

Another recent paper by Turon et al. [6] identified the independent risk factors for developing PVT: low platelet count, portal blood flow velocity <15cm/sec and history of variceal bleeding, all factors related to the severity of portal hypertension. That is why the Baveno VII statements recommend screening for PVT in all patients who are or may become LT candidates at the time of screening for hepatocellular carcinoma (HCC).

There are several major differences between our study and the results reported by Girleanu et al. [1]. First, the patients included in our cohort were on the waiting list for LT or potential LT candidates; thus, all of them underwent regular abdominal ultrasound for HCC screening (every 3 to 6 months). Our patients have both partial and complete PVT; in our cohort only 20.5% of patients had complete PVT compared to Girleanu et al. [1] cohort where all patients had total PVT. It is already demonstrated that patients with complete PVT have a higher 30-day pooled mortality rate (OR=5.65) and a 1-year mortality rate (OR=2.48) than patients with partial PVT [5]. The second difference is related to the sample size and to the selection of patients for anticoagulation (AC) therapy. The sample size of the colleagues from Iasi was very small and valid conclusions were difficult. In Girleanu et al. study [1], their patients were in Child Pugh C class in a significantly higher percentage (35%) versus 2.8% in our cohort [2]. In our cohort, most of the patients were Child Pugh B (71.9%) compared to 48% in their group of patients. This finding, in relation to the "timing" of initiation of AC therapy may be relevant. "Timing" refers to the time since diagnosis of PVT and to the stage of liver cirrhosis. Our "timing" is shorter because we performed regular screening for PVT for patients on the waiting list (before Baveno recommendations) and we have a significant proportion of patients with partial PVT. The majority of our patients were Child A and B cirrhosis, but with clinically significant portal hypertension.

According to the Baveno VII conclusions [7], AC is recommended in patients with LC and recent (< 6 months), completely or partially occlusive thrombosis of the portal vein trunk with or without extension to the superior mesenteric vein or symptomatic PVT independently of the extension or PVT in patients potentially candidates for LT, independently of the degree of occlusion and extension.

Our results regarding AC therapy in patients with LC and PVT are in agreement with another large cohort of patients presented by Pettinari et al. [8] with more than 50% of on-treatment patients experiencing recanalization of the PVT and in contrast to the small group of treated patients by Girleanu et al. [1]. Anticoagulation therapy is safe and effective leading also to significantly improved survival in both cohorts (ours and the one of Pettinari et al. [8]) and opposite to Girleanu et al [1], the cause being the selection of patients with Child Pugh C class at the beginning of AC as was shown by the Bologna group [8].

According to our results, the highest risk of bleeding during AC therapy is in patients with low thrombocytes <50x10^3/µl. Because these patients are at a higher risk of developing PVT, but also at the highest risk of bleeding events during AC therapy according to Baveno VII statements, caution and balancing risks and benefits in these patients is warranted.

In our cohort, patients receiving AC had a high risk of bleeding events (18.5%), but similar to the Bologna study group (19.7%) [8]; however, most of these bleeding events were related to high portal hypertension and not to AC itself. On the other hand, the PRO-LIVER study group [9] also demonstrated that a previous gastrointestinal bleeding and encephalopathy, but not platelet count <50x103/µl, independently predict overall bleeding events.

We agree with Girleanu et al [1] that large prospective studies are required. However, much emphasis should be placed on screening of PVT development in LC patients, especially LT candidates and the early initiation of AC therapy in order to prevent progression to complete PVT or to obtain recanalization and prevent re-thrombosis and thus increase survival both on the waiting list as well as following LT. In our opinion the "anticoagulation window" is rather large if we properly follow-up all cirrhotic patients with portal hypertension and possibly find models to identify the best candidates for AC therapy with the smallest risk of bleeding related to AC. The "anticoagulation window" should probably include both preventive AC in patients with a very high risk of developing PVT and therapeutic AC in patients with already established partial or total PVT.

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Conflicts of interest: None.
DOI: 10.15403/jgld-3082

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