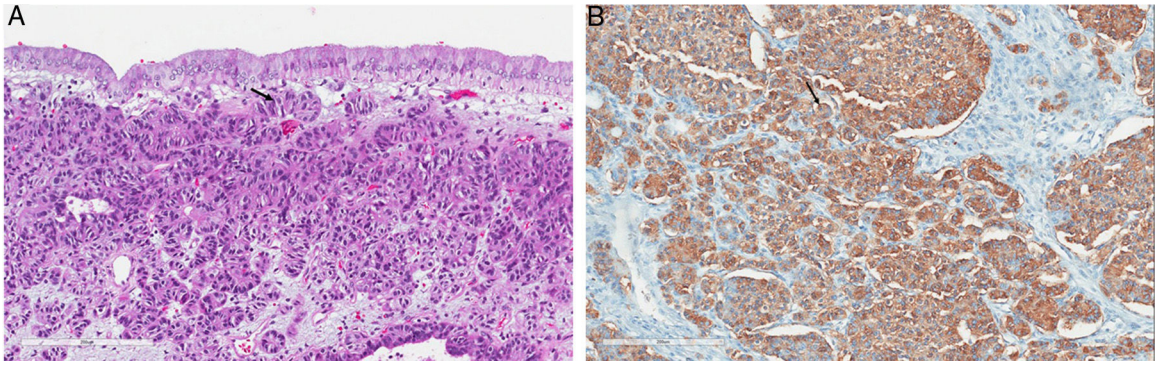

Figure 1 A) The tumour grows beneath the surface epithelium, forming small nests and trabeculae. The cells are of uniform size, with slightly eosinophilic cytoplasm and an ovoid nucleus, with no significant atypia (H&E). B) Immunohistochemical staining for chromogranin: intense cytoplasmic positivity is seen in the neoplastic cells.

on regional lymphadenectomy or IVb-V bisegmentectomy. Adjuvant therapy is recommended in patients with a Ki-67 value of >20–55%, which is more effective for the small cell subtype. Specific prognostic factors have not been identified in NET-GB, but the proliferative index, differentiation and tumour size are global prognostic factors.[1,4]

According to the NCCN Guidelines, serum chromogranin or 5-HIAA in urine should be analysed and a CT or MRI should be performed between three and 12 months post-resection and every 12–24 months.[5]

The five-year survival rates for advanced disease are currently 77–95% when treated with primary tumour resection and adjuvant therapy. However, more aggressive subtypes have been reported, with a fast clinical course in which survival and prognosis are considerably worse.[1]

In conclusion, NET-GB are uncommon and generally non-functioning. They tend to be diagnosed in the histopathological study of the gallbladder after cholecystectomy due to cholelithiasis. The cystic node should be studied to decide whether to use the lymphadenectomy or IVb-V bisegmentectomy approach and adjuvant therapy should be considered based on the proliferative index.

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Joana Aylhin López Marcano*, José Manuel Ramia Ángel, Roberto de la Plaza Llamas, Alba Manuel Vázquez, Raquel Latorre Fragua

Unidad de Cirugía Hepatobiliopancreática, Servicio de Cirugía General y Aparato Digestivo, Hospital Universitario de Guadalajara, Guadalajara, Spain

* Corresponding author.
E-mail address: aylhin10@gmail.com (J.A. López Marcano).

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Endoscopic submucosal dissection for localised gastric amyloidosis mimicking malignancy

Disección submucosa endoscópica para la amiloidosis localizada gástrica que simula una malignidad

Amyloidosis is characterized by the extracellular deposition of insoluble amyloid fibrils, which commonly results in systemic organs dysfunction. A single organ or tissue involved is uncommon, especially regarding the gastric localised amyloidosis. The lesion may manifest as erosions, ulcerations, nodular mucosa or polypoid protrusions. However, the specific treatment for localized amyloidosis has not been established. We present a case of gastric localized amyloidosis mimicking malignancy, in which endoscopic submucosal dissection (ESD) was successfully done with the guidance of multiple endoscopic imaging techniques.

A 54-year-old man presented with occasional postprandial epigastric discomfort for several months. Esopha-

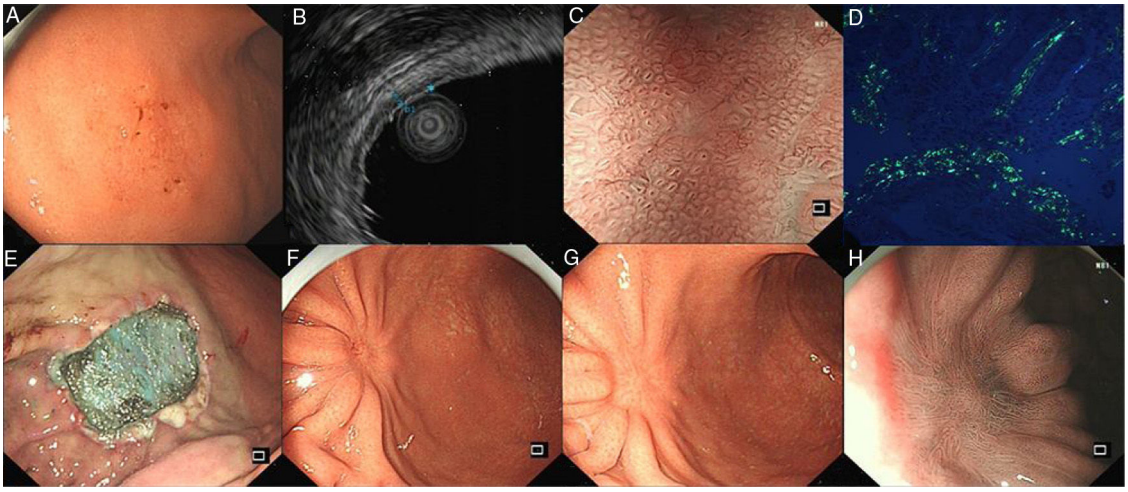

Figure 1 (A) EGD showed the flat depressed lesion in the stomach. (B) EUS revealed the first layer was thinned and the second layer was thickened. (C) NBI+ME showed the mucosal pit pattern and microvessels were obviously changed. (D) Apple green birefringence of amyloid deposition under polarized light. (E) Endoscopic submucosal dissection. (F,G,H) EGD revealed a scar formation and NBI+ME showed sparse glandular tube structure in 3 months and 1 years after surgery.
EGD, Esophagogastroduodenoscopy; EUS, Endoscopy ultrasound evaluation; NBI+ME, Narrow-band imaging with magnified endoscopy.

gogastroduodenoscopy (EGD) showed a 2.5 cm × 3.0 cm flat depressed lesion in the gastric somatic and antral junction (Fig. 1A). The lesion resembled early gastric cancer with interrupted mucosal folds, spontaneous oozing on the surface and disappearance of partial border glandular structure. Endoscopy ultrasound evaluation (EUS) revealed the lesion originated from the mucosal layer. The first layer was thinned and the second layer was thickened with 1.4 mm in the thickest part (Fig. 1B). Narrow-band imaging with magnified endoscopy (NBI + ME) showed the margin of the lesion was well-defined. The mucosal pit pattern was distorted, dilated and partially sparse, and the microvessels were obviously distorted, expanded or partially disappeared (Fig. 1C). However, histopathologic examination revealed an amorphous eosinophilic material deposition that extended from the lamina propria to the muscularis mucosae without malignant findings. The amyloid proteins were identified by Congo red staining and apple-green birefringence under polarized microscopy (Fig. 1D). Immunohistochemical staining was positive for κ and λ chain. Neither gastric fundus nor colorectal biopsy specimens showed amyloid deposition. The serum and urine immunoelectrophoresis were normal and there were no signs of amyloidosis in the kidney, heart and liver. A diagnosis of gastric localized amyloidosis with κ and λ light chain co-expression was made.

Subsequently, the ESD operation was performed with the guidance of NBI + ME and EUS (Fig. 1E). The postoperative specimens further confirmed amyloid deposition without malignant findings. There were no complications associated with the ESD. Three months and a year postoperatively, EGD was repeated (Fig. 1F–H). Mucosal biopsies from the centre and edge of the scar showed no amyloid deposition. The patient was curatively treated with symptom-free during the subsequent 12-month follow-up period.

Localized AL amyloidosis has a good prognosis, but 1–2% of patients exhibited systemic progression and 44% had progression at the primary site.[1,2] A recent study ever revealed more than half of the patients with localized laryngeal amyloidosis developed localized recurrence, which required revision surgery in the first 4 years.[3] 22% of patients with GI localized amyloidosis may have localized progression,[2] but it remains unclear whether localized progression affects disease prognosis. Although there are no standardized guidelines for the treatment of localized AL amyloidosis in gastrointestinal tract, the excision of the amyloid deposits and follow-up are the most common suggestions. GI bleeding, obstructive or abdominal pain may be a trigger for interventions in gastrointestinal deposits for symptomatic improvement.[2] In a retrospective study, two symptomatic patients with GI localized amyloidosis were all relieved symptoms after the excision of lesions.[4] Similarly, our patient was troubled by the occasional abdominal pain, which was gone after ESD.

Our case showed a type IIc lesion in the stomach which resembled early gastric cancer. Although the biopsy specimen showed the amyloid deposition, it could not rule out the possibility of biopsy bias. ESD has been widely accepted and used not only as a curative treatment for the early cancer in the gastrointestinal tracts but also as a diagnostic strategy for some confused cases.[5] Gastric carcinoma was excluded finally in this case and the patient achieved curative treatment with the help of ESD. This case demonstrates that the thorough resection by ESD with guidance of NBI + ME and EUS is probably a safe and successful therapeutic choice for gastric localized amyloidosis mimicking malignancy in a symptomatic patient.

Conflicts of interest

The authors declare no conflicts of interest.

Acknowledgements

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Tao Yang[a], Jia-Wei Lu[a], Li-Hong Teng[a], Jing-Jing Feng[b], Jia-Guo Wu[a], Wei-Li Liu[a,*]

[a] *Department of Gastroenterology, Sir Run Run Shaw Hospital, Zhejiang University School of Medicine, Hangzhou, China*
[b] *Zhejiang Chinese Medical University, Hangzhou, China*

* Corresponding author.
E-mail address: 3307006@zju.edu.cn (W.-L. Liu).

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Hepatic tuberculosis mimicking metastatic colorectal cancer☆

Tuberculosis hepática simulando metástasis de cáncer colorrectal

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis complex*,[1] the target organs of which are the lungs. It primarily affects the populations of developing areas, causing 2,000,000 deaths per year.[1,2] Extrapulmonary tuberculosis is derived from the pulmonary disease and primarily affects immunosuppressed individuals, particularly HIV patients.[1,3] The most commonly affected extrapulmonary tissues are the pleura, the lymph nodes and the urinary system.[1] The rarity of liver involvement coupled with its nonspecific symptoms make it difficult to diagnose, meaning that histological confirmation is often required.[1,2,4] As in our case, it can be confused with primary or metastatic liver tumours.

We present the case of a 69-year-old male with a history of smoking and alcoholism, diabetes mellitus, hypertension, dyslipidaemia and a rectal adenocarcinoma in 2013 who received neoadjuvant chemotherapy and radiotherapy, followed by surgery with a pathological finding of T3N0M0, receiving adjuvant therapy with six cycles of Xeloda®. The patient subsequently remained free of clinical and radiological disease and, at the fourth year of follow-up, exhibited a hypodense low attenuation lesion on the CT scan (Fig. 1A) in segment VI of the liver. Given his oncological history, liver metastasis of colorectal origin was the first possibility to be considered. The study was extended with an abdominal ultrasound, which revealed multiple nodular lesions measuring up to 2 cm in diameter, and an MRI (Fig. 1B), which showed hyperintense, non-calcified bilobar lesions in T1 Fat-Sat with contrast in arterial phase, in segments II, IVA, IVB, VIII and VI, all suggestive of liver metastases, and CEA at the upper limit of normal. The case was submitted to the digestive tumour committee, and it was decided to start systemic chemotherapy with six cycles of FOLFIRI plus cetuximab. New CT and MRI scans at four months showed partial response to chemotherapy with a reduction in the size of the hepatic space-occupying lesions (SOLs), except for one in segment II and one in segment VI. It was decided to perform rescue surgery on the liver, finding five subcapsular lesions suggestive of subcentimetric metastases in segments IVA, IVB, V, VI and VIII, as well as two intraparenchymatous lesions in segments II and IVB. Metastasectomy of the subcapsular lesions was performed and microwaves were applied to the intraparenchymatous lesions. The histological result of all the samples showed granulomatous nodular lesions centred by necrosis of a caseous appearance surrounded by epithelioid histiocytes, some multinucleated giant cells and, peripherally, fibrosis with Ziehl-Neelsen

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