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Original Article

Endoscopic Manifestations and Clinical Characteristics of Localized Gastric Light-Chain Amyloidosis

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To determine the endoscopic and clinical features of localized gastric amyloid light-chain (AL) amyloidosis, we retrospectively examined the characteristics of nine patients (eight men and one woman) encountered by the hospitals in our network. Lesions were predominantly flat and depressed with surface vascular dilatation (n = 5); others were characterized by subepithelial lesions (n = 2), mucosal color change (n = 1), and a mass-like morphology with swollen mucosal folds (n = 1). Colonoscopy (n = 7), video capsule enteroscopy (n = 2), serum (n = 5) and urine immunoelectrophoresis (n = 4), and bone marrow examination (n = 3) were performed to exclude involvement of organs other than the stomach. As treatment for gastric lesions of AL amyloidosis, one patient each underwent endoscopic submucosal dissection (n = 1) and argon plasma coagulation (n = 1), while the remaining seven patients underwent no specific treatment. During a mean follow-up of 4.2 years, one patient died 3.2 years after diagnosis, but the cause of death, which occurred in another hospital, was unknown. The remaining eight patients were alive at the last visit. In conclusion, although localized gastric AL amyloidosis can show various macroscopic features on esophagogastroduodenoscopy, flat, depressed lesions with vascular dilatation on the surface are predominant.

Key words: esophagogastroduodenoscopy, gastric lesion, amyloidosis, light chain

An amyloid is an insoluble and self-assembled fibrous protein that is structurally dominated by β-sheet structures. Thirty-six types of amyloids have been observed and reported to date, including amyloids consisting of immunoglobulin light-chain (AL), amyloid A (AA), transthyretin (TTR), and β_2-microglobulin ($A\beta_2M$) [1,2] proteins. Amyloid deposition in the human body is called amyloidosis and may be localized or systemic. Deposition of amyloids in multiple organs is called systemic amyloidosis, whereas deposition limited to a certain organ is classified as localized amyloi-

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dosis [3]. Although amyloidosis affects organs and tissues throughout the body, the most common sites affected by localized AL amyloidosis are the eyelids, larynx, bronchi, skin, and urinary tract [4]. Additionally, the gastrointestinal tract is sometimes involved in amyloid deposition. One study investigating 2,334 patients with amyloidosis revealed that 3.2% of patients (n = 76) had biopsy-proven amyloid deposition in the gastrointestinal tract [5]. Among them, the characteristics of AL amyloidosis localized to the stomach were not sufficiently investigated owing to the rarity of this disease.

The purpose of the present study was to determine the endoscopic features and clinical background of gastric AL amyloidosis from cases encountered in our hospital network. We also review previously reported cases of this disease entity and discuss its endoscopic manifestations.

Methods

Letters of inquiry from the Department of Gastroenterology and Hepatology, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences were sent to collaborating institutions regarding patients with histologically diagnosed gastric AL amyloidosis. Histological diagnoses had been made based on endoscopic biopsy, mucosal resection, submucosal dissection, and/or surgical resection. Amyloid deposition was detected using hematoxylin and eosin staining as an acidophilic nonstructural material and was subsequently validated using Congo red or a direct fast scarlet. AL amyloidosis was diagnosed based on positive staining for light chain κ or λ. Because we initially planned to retrospectively accumulate patients with primary localized gastric AL amyloidosis, cases were excluded if i) immunostaining for light chain κ and λ was never performed, ii) multiple myeloma was present in the bone marrow, and iii) organs other than the stomach were involved. Because of the study's retrospective and observational nature, the diagnosis of gastric localization was based on the clinical judgment of the treating physician; tests to exclude the involvement of organs other than the stomach were variably performed among subjects.

We identified nine patients diagnosed with gastric AL amyloidosis between March 2012 and April 2022. The patients were retrospectively enrolled in this study. One of the patients examined was described in our previous case report [6]. We retrospectively examined each patient's sex, age at diagnosis, endoscopic and histological features, treatments, and prognoses.

This study was approved by the Ethics Committees of Okayama University Hospital and other institutions and adhered to the Declaration of Helsinki. The requirement for written informed consent was waived due to the observational, non-interventional, and retrospective design of the study. All investigations were performed in accordance with relevant guidelines and regulations.

Results

The clinical characteristics of the enrolled patients are summarized in Table 1. This study included eight men and one woman. The average age at diagnosis of gastric AL amyloidosis was 58.0 years (range, 39-85 years). Immunostaining revealed pathological light-chain λ in five patients and light-chain κ in four patients. Representative endoscopic and pathological images are shown in Fig. 1-3. Gastric lesions were reddish (n = 5), whitish

Table 1 Characteristics and endoscopic features of patients with gastric AL amyloidosis

Case no.	Sex	Age	Type of amyloid	Endoscopic features	Diameter of the gastric lesion (mm)
1	M	58	ALλ	A reddish, flat, depressed lesion with vascular dilatation	25
2	M	59	ALλ	Multiple whitish, flat, depressed lesions with vascular dilatation	20
3	M	41	ALλ	A reddish mass with swollen mucosal folds	40
4	M	39	ALκ	A subepithelial lesion with multiple flat, depressed areas	15
5	M	85	ALκ	An yellowish, flat, elevated, subepithelial lesion	20
6	F	74	ALκ	A reddish patch	10
7	M	50	ALλ	A whitish, flat, depressed lesion with vascular dilatation	20
8	M	51	ALλ	A whitish, flat, depressed lesion with vascular dilatation	35
9	M	62	ALκ	A reddish, flat, depressed lesion with vascular dilatation	15

(n = 3), or yellowish (n = 1). With respect to morphology, flat, depressed lesions were predominant (n = 5), and all depressed lesions presented vascular dilatation on the surface. Other macroscopic features included a subepithelial lesion-like morphology (n = 2), mucosal color change (*i.e.*, patch, n = 1), and a mass with swollen mucosal folds (n = 1).

Magnifying observation using narrow-band or blue laser imaging was performed on four patients with depressed lesions, as well as the one with mucosal color change, and the one with a mass exhibiting swollen mucosal folds. Upon magnifying observation, all depressed lesions displayed a partial absence of microstructural patterns and emphasized dilation of the microvasculature (Fig. 1E and 1G). Conversely, lesions exhibiting mucosal color changes and masses with swollen mucosal folds showed regular patterns of microvascular and microstructural features.

Endoscopic ultrasonography was performed in three patients with depressed lesions and in the patient with a mass lesion with swollen mucosal folds. In patients with depressed lesions, amyloid deposition in the stomach was visualized on endoscopic ultrasonography as hypo- to-isoechogenic areas extending linearly along the mucosal layers, mainly within the deeper layer of the lamina propria mucosa (Fig. 1C). In contrast, the

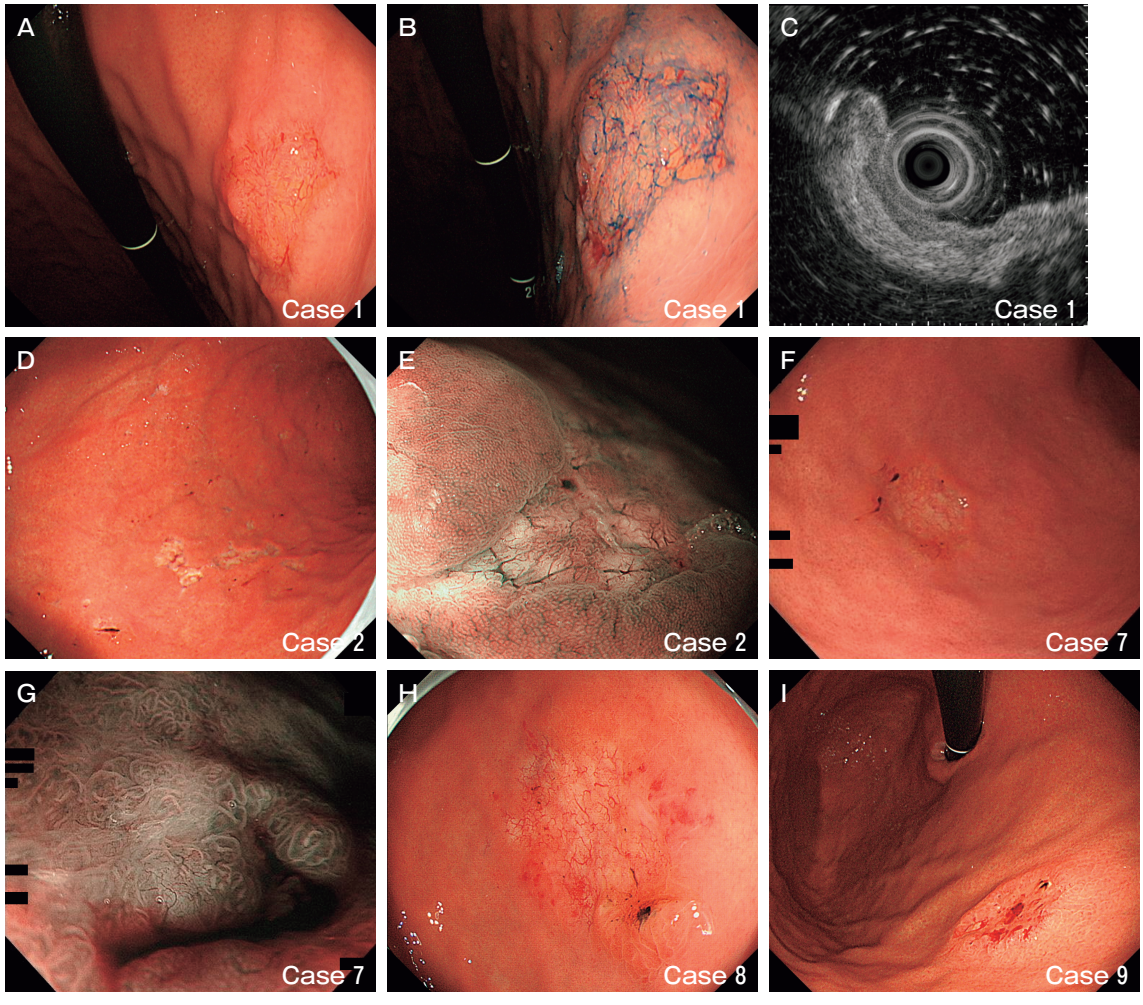

Fig. 1 Representative endoscopic images of flat, depressed lesions of localized gastric AL amyloidosis. A reddish, depressed lesion with vascular dilatation was observed in the gastric body (Case 1; **A**, white light; **B**, after indigo carmine dye spraying). Endoscopic ultrasonography showed a low echogenic area in the deeper layer of the lamina propria mucosae (**C**). A whitish, depressed lesion was seen in the gastric body (Case 2; **D**). Vascular dilatation was emphasized on narrow band imaging observation (**E**). Cases 7 (**F**, white light; **G**, narrow band imaging), 8 (**H**), and 9 (**I**) also showed flat, depressed lesions.

mass lesion exhibiting swollen mucosal folds spanned the entire depth of the lamina propria mucosa and protruded extensively into the submucosa; it also was hypo- to isoechogenic in itensity (Fig. 2C).

Table 2 shows the patients' underlying diseases, tests performed to exclude involvement of organs other

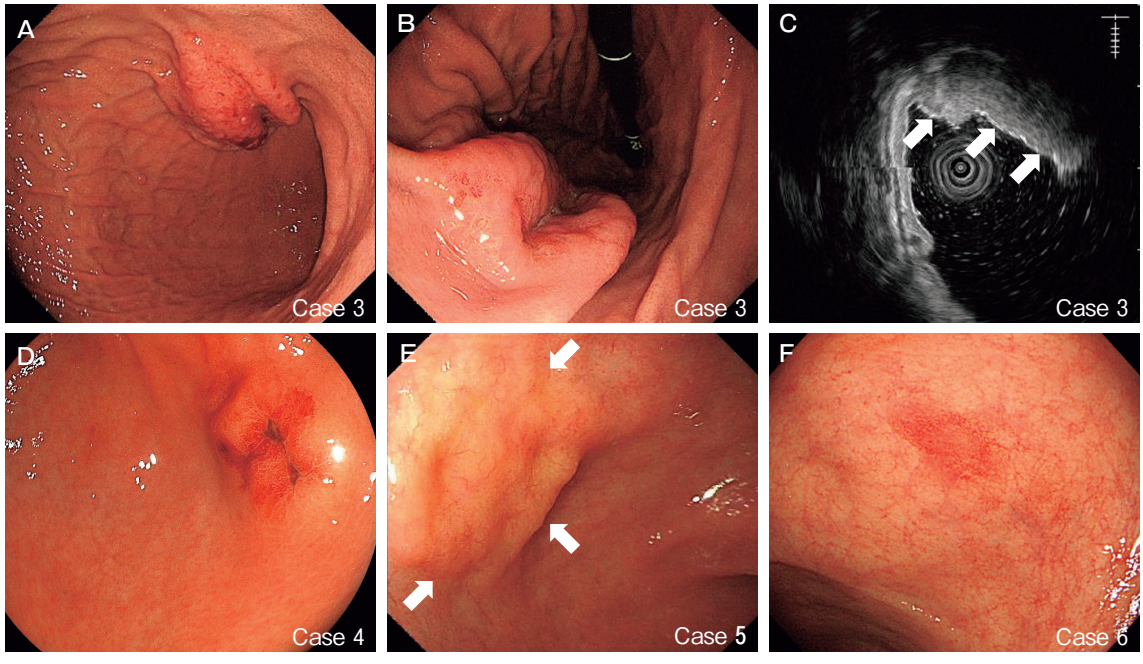

Fig. 2 Representative endoscopic images of localized gastric AL amyloidosis. A reddish mass with swollen mucosal folds was observed in the gastric body (Case 3, **A**&**B**). Endoscopic ultrasonography showed isoechogenic areas (**C**, arrows). Localized gastric AL amyloidosis also presented as a reddish subepithelial lesion with multiple depressed areas (Case 4, **D**), a yellowish, flat elevated subepithelial lesion (Case 5, **E**), and a reddish patch (Case 6, **F**).

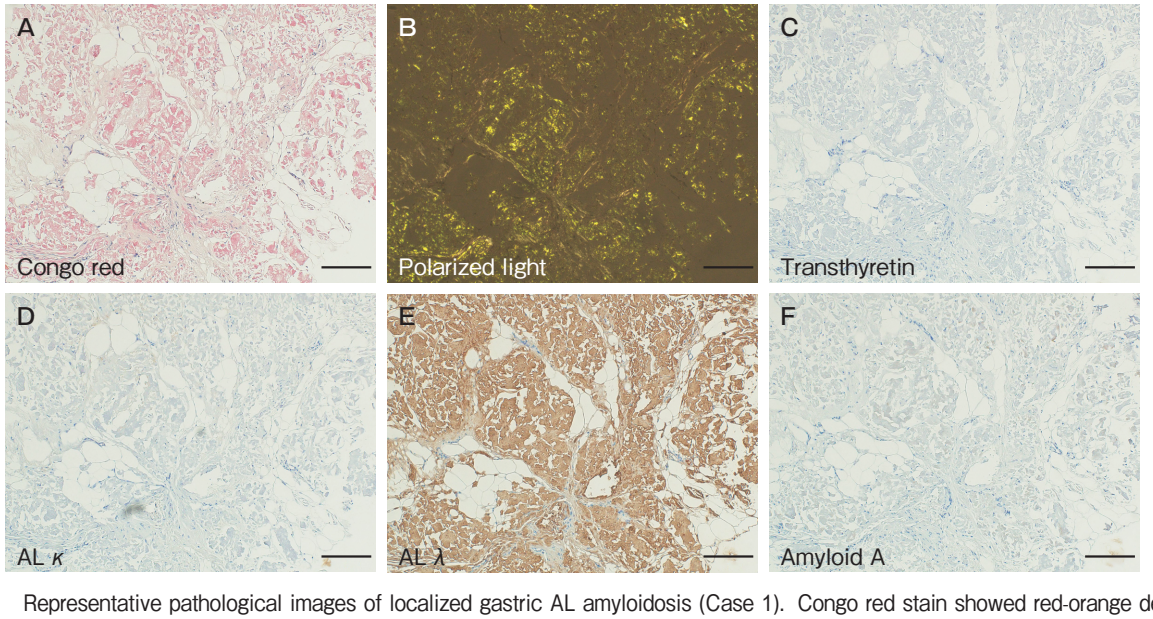

Fig. 3 Representative pathological images of localized gastric AL amyloidosis (Case 1). Congo red stain showed red-orange deposits under non-polarized light (**A**). Observation under polarized light revealed apple-green birefringence in deposits (**B**). Amyloid was negative for transthyretin (**C**), AL κ (**D**), and amyloid A (**F**), while it was positive for AL λ (**E**). Scale bars: **A**-**F**, 200 μm.

Table 2 Tests to exclude involvement of other organs and prognosis of the patients

Case no.	Underlying diseases	Colonoscopy	Video capsule enteroscopy	Serum immuno-electrophoresis	Urine immuno-electrophoresis	Bone marrow examination	Treatment for gastric amyloidosis	Follow-up period (years)	Outcome
1	None	Done	Not done	Done	Done	Done	None	0.4	Alive
2	None	Done	Not done	Done	Done	Done	None	3.9	Alive
3	None	Not done	Not done	Done	Done	Done	Endoscopic submucosal dissection	0.3	Alive
4	None	Done	Not done	Not done	Not done	Not done	Argon plasma coagulation	3.3	Alive
5	Alzheimer's disease, hypertension, dyslipidemia	Not done	Not done	Not done	Not done	Not done	None	3.2	Dead
6	Hypertension, dyslipidemia, early gastric cancer	Done	Not done	Done	Not done	Not done	None	8.8	Alive
7	None	Done	Not done	Done	Not done	Not done	None	5.0	Alive
8	None	Done	Done	Not done	Done	Not done	None	10.1	Alive
9	None	Done	Done	Not done	Not done	Not done	None	3.0	Alive

than the stomach, and their prognosis. Seven patients had no specific underlying diseases, while one patient had Alzheimer's disease, hypertension, and dyslipidemia. The other patient had hypertension, dyslipidemia, and a history of early gastric cancer, which had been curatively resected via endoscopic submucosal dissection. As described in Methods, tests to exclude involvement of organs other than the stomach were diversely performed among patients. Colonoscopy was performed in seven patients and surveillance of the small intestines was performed in two patients using video capsule enteroscopy. No involvement with amyloidosis was found in the small or large intestines. Serum immunoelectrophoresis was performed in five patients and demonstrated monoclonal IgG-κ proteins in one patient, while monoclonal gammopathies were not identified in the serum of the other four patients. Four patients underwent urine immunoelectrophoresis tests, but no cases showed secretion of free monoclonal κ or λ chains (*i.e.*, Bence Jones protein). Bone marrow examination performed in three patients revealed no abnormalities including plasma cells.

Gastric AL amyloidosis was resected via endoscopic submucosal dissection in the patient (case 3) with a mass lesion for both diagnostic and treatment purposes. Pathological analysis of the resected specimen revealed substantial amyloid deposition, 10 mm in thickness, mainly in the deeper layers of the lamina propria mucosa and submucosa. One patient with a reddish subepithelial lesion and multiple depressed areas (case 4) underwent argon plasma coagulation for bleeding from the gastric lesion. No specific treatment was initiated for gastric AL amyloidosis in the remaining seven patients. Patients were followed up for 4.2 ± 3.3 years (range: 0.3-10.1 years) after the diagnosis of gastric AL amyloidosis. One patient died 3.2 years after diagnosis at another hospital; the cause is unknown to us. The remaining eight patients were alive at their last visits.

Discussion

This study collected data from the largest number of patients with localized gastric AL amyloidosis of any study to date, based on our literature search. Analysis of the nine patients revealed flat, depressed lesions with vascular dilatation on the surface in five; this constituted the most common gross appearance (5/9, 55.6%). Localized gastric AL amyloidosis is an infrequent disorder, and cases of this disease are rarely reported. Lin *et al.* reviewed 22 cases of localized gastric amyloidosis reported between 1978 and 2019, including 13 cases of AL amyloidosis [7-19]. One of those patients was

included in this study [6]. We searched PubMed and found four additional cases [20-23]. A total of 16 previously reported cases of localized gastric AL amyloidosis (excluding our case [6]) are summarized in Table 3. Despite the clear male predominance (8/9, 88.9%) in our study, there were seven men and five women in the previously reported cases. The average age was 63.0 years (range, 47-80 years). Flat, depressed lesions were the most common morphology (n=6), followed by subepithelial (n=4), swollen mucosal folds (n=2), ulcerative (n=2), elevated (n=2), and patchy lesions (n=1). A review of previous reports and our study results indicate that although localized gastric AL amyloidosis has various macroscopic features on esophagogastroduodenoscopy [7,20,23], flat depressed lesions are the most predominant.

Takahashi *et al.* reported a patient with localized gastric AL amyloidosis in whom the gastric lesion morphology changed during their 10-year follow-up [21]. Initially, the lesion appeared as a reddish, flat, and depressed lesion, but it had become a subepithelial lesion 10 years after the diagnosis. This case indicates that gastric AL amyloidosis can grow vertically over time, probably due to increased amounts of amyloid deposits within the gastric wall. However, because no apparent change in morphology was reported in other cases, we speculate that alteration of gross appearance is an infrequent event in gastric AL amyloidosis.

The characteristic pathological features of intestinal AL amyloidosis have been documented to encompass prodigious deposition involving the muscularis mucosae, submucosa, and the proper muscle layer, which often results in polypoid protrusions or thickening of

Table 3 Reported cases of localized gastric AL amyloidosis

Author (et al.)	Year	Sex	Age	Type of amyloid	Endoscopic features	Diameter of the gastric lesion (mm)	Treatment
Björnsson	1987	F	60	AL (κ&λ)	Swollen mucosal folds with bleeding*	NA	Surgical resection
Yanai	1991	F	52	AL (λ)	A flat, depressed lesion	25	NA
Shibukawa	2004	F	51	AL	An ulcerative lesion resembling advanced gastric cancer	NA	Surgical resection
Ebato	2012	F	77	AL	A flat, depressed lesion	46	ESD
Sawada	2012	F	72	AL (κ&λ)	Various features including ulcerative lesion, subepithelial lesion, and intramural hematoma	NA	NA
Kamata	2012	F	76	AL	Reddish, swollen mucosal folds with erosions and bleeding	NA	None
Yamaguchi	2015	M	49	AL	A subepithelial lesion with depressed area	15	NA
Kobara	2015	M	80	AL	A reddish granular, elevated lesion	20	NA
Kagawa	2016	M	73	AL	A whitish, flat, depressed lesion with vascular dilatation	15	None
Ahn	2018	F	55	AL (κ&λ)	A whitish, flat, depressed lesion with vascular dilatation	20	None
Savant	2018	M	64	AL (λ)	An elevated lesion*	36	NA
Kinugasa	2020	M	64	AL (λ)	A subepithelial lesion	40	NA
Furukawa	2020	M	56	AL (κ)	A whitish, flat, depressed lesion with vascular dilatation	40	None
Takahashi	2021	F	71	AL (λ)	A reddish, flat, depressed lesion**	NA	ESD**
Singh	2021	F	47	AL (κ&λ)	Yellowish patch with bleeding	30	ESD
Hashimoto	2021	M	49	AL (λ)	A subepithelial lesion with depressed area	20	None

NA, not available; ESD, endoscopic submucosal dissection.
*Endoscopic images are not shown. **The lesion changed morphologically to exhibit a subepithelial lesion 10 years after the initial examination and was removed via ESD.

the Kerckring folds that can be observed by endoscopic examination [24]. Such endoscopic and pathological features were observed in one patient in this study (case 3). In contrast, in three patients with depressed lesions, endoscopic ultrasonography revealed linear amyloid deposition along the mucosal layers, mainly within the deeper layer of the lamina propria mucosa. Thus, we speculate that AL amyloid is deposited primarily in the deeper layers of the lamina propria mucosa of the stomach, leading to flat depressed lesions. Difference in the layers where the amyloid is typically deposited in the intestine and stomach may be attributed to the different structures of their gastrointestinal walls. Nevertheless, our speculations warrant further investigation as pathological evaluation of the resected specimen was conducted in only one patient; other patients were diagnosed solely based on endoscopic biopsy specimens.

As previously mentioned, the predominant endoscopic manifestation of primary gastric AL amyloidosis is with flat, depressed lesions with surface vascular dilatation. In some cases, patients with gastric cancer or extranodal marginal zone lymphoma of the mucosa-associated lymphoid tissue (MALT lymphoma) present with flat, depressed lesions in the stomach. We consider that vascular dilatation, which can be emphasized on magnifying observations using narrow-band or blue laser imaging, may be an indication to distinguish gastric AL amyloidosis from gastric cancer because the latter typically lacks this feature. Differentiation between gastric MALT lymphoma and AL amyloidosis remains difficult, owing to the fact that vascular dilatation on the surface is a common feature of the two diseases [25]. This morphological resemblance probably stems from pathological similarities that primarily affect the mucosal layer beneath the superficial epithelium.

Asymptomatic patients with localized gastric AL amyloidosis are believed to have a favorable prognosis and require no specific treatment [20, 26]. Although several patients in previously reported cases and our research participants underwent surgical or endoscopic resection for diagnosis and/or treatment [8, 10, 11, 21, 22], no intervention was initiated in most patients, and none died of gastric amyloidosis. These results reinforce the favorable long-term outcome of this disease and verify the validity of our observations [20, 26]. However, confirmation of gastric localization of AL amyloid deposition is essential because plasma cell proliferative diseases and involvement of other organs such as the heart, liver, kidney, and nerves require different management strategies [7].

Although screening for plasma cell proliferative diseases and involvement of other organs is important for the diagnosis of localized gastric AL amyloidosis, a uniform diagnostic strategy has not been established. Generally, exclusion tests include biopsy of the bone marrow, endoscopic biopsy of the gastrointestinal tract on esophagogastroduodenoscopy and colonoscopy, ultrasound of the heart and kidney, and immunoelectrophoresis of serum and urine proteins [27, 28]. In the present study, diagnostic tests were variably performed between patients based on the clinical judgment of the treating physician. Insufficient performance of exclusion tests may reflect a lack of recognition of these tests among gastroenterologists, highlighting the importance of educating attending physicians on how to exclude systemic involvement when treating patients with AL amyloidosis.

Our study had several limitations. First, as described above, exclusion tests were unevenly performed, which might have resulted in overestimation of the gastric localization of AL amyloid deposition. Such differences between patients were inevitable because of the retrospective and observational nature of the study. Raising awareness regarding exclusion tests would improve the management of patients with this disease. Second, the follow-up period (average, 4.2 years) was relatively short to investigate the prognosis of localized gastric AL amyloidosis. Further investigations with longer follow-up periods are necessary to determine the outcomes.

In conclusion, we report nine patients with localized gastric AL amyloidosis and revealed that flat, depressed lesions with vascular dilatation comprised the predominant endoscopic feature. These results indicate that although gastric cancer and MALT lymphoma are representative diseases presenting as flat depressed lesions in the stomach [29], localized AL amyloidosis should also be considered as a differential diagnosis. In addition, gastroenterologists should be aware of the importance of surveillance of other organs when AL amyloid deposition is detected in the stomach.

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