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Mucosal Schwann cell hamartoma of the gastroesophageal junction: A series of 6 cases and comparison with colorectal counterpart

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ABSTRACT

Mucosal Schwann cell hamartoma (MSCH) is an uncommon neural lesion characterized by an ill-defined proliferation of S100-positive Schwann cells in the lamina propria, with reported cases exclusively occurring in the colorectum. Here we describe the first series of MSCHs arising in the gastroesophageal junction (GEJ) and discuss their clinicopathologic features in comparison with their colorectal counterparts. We searched the UCLA pathology database from 01/2014 to 12/2018 to identify cases carrying the diagnosis of MSCH. A total of 48 cases (45 in-house, 3 consults) of colorectal MSCHs and 6 cases (1 in-house, 5 consults) of GEJ MSCHs were identified. For GEJ MSCHs, there were 4 males and 2 females with an average age of 70.2 years (range: 57–76 years). Clinical indications for endoscopy included history of gastroesophageal reflux disease (n = 2), heartburn (n = 2), dysphagia (n = 1), and iron deficiency anemia (n = 1). Endoscopic findings at the GEJ were available for 5 patients including irregular Z-line (n = 3), mild nodular carditis (n = 1), and normal (n = 1). None of them showed a polyp or nodule. The mean size of the lesion was 2.8 mm (range: 2–4 mm) microscopically. None of the colorectal or GEJ MSCH cases had an association with inherited syndromes. In conclusion, MSCH of the gastrointestinal tract is predominantly seen in the colorectum, but also infrequently seen in the GEJ. GEJ MSCH shares histologic and immunohistochemical features with its colorectal counterpart, but is usually an incidental finding with no endoscopically visible lesion. As there is no syndromic association with MSCH, additional treatment, work-up and follow-up are unnecessary.

1. Introduction

Mucosal Schwann cell hamartoma (MSCH), variably termed "neurofibroma" or "neuroma" in the past, is a newly recognized neural lesion of the gastrointestinal (GI) tract, first proposed by Gibson and Hornick in 2009 [1]. MSCH is characterized by an ill-defined proliferation of spindle cells within the lamina propria purely composed of S100-positive Schwann cells. Different from neuromas and neurofibromas of the GI tract that are frequently associated with inherited syndromes, such as neurofibromatosis type-1 (NF1) and multiple endocrine neoplasia type 2B (MEN 2B), MSCHs have no known syndromic association but are often incidental findings. The reported MSCHs in the literature virtually exclusively occur in the colon and rectum, predominantly in the rectosigmoid region. In this report, we describe the first series of MSCH arising in the gastroesophageal junction (GEJ) and discuss their clinicopathologic features in comparison with their colorectal counterparts.

2. Materials and methods

We searched the surgical pathology database of Ronald Reagan UCLA Medical Center spanning a period of 5 years from January 2014 to December 2018. Both in-house and consultation cases were included. Relevant clinical data, such as patients' demographics, clinical

Abbreviations: MSCH, mucosal Schwann cell hamartoma; GI, gastrointestinal; NF1, neurofibromatosis type-1; MEN 2B, multiple endocrine neoplasia type 2B; GEJ, gastroesophageal junction; H&E, hematoxylin and eosin; EMR, endoscopic mucosal resection; GERD, gastroesophageal reflux disease; GIST, gastrointestinal stromal tumor; TCLBs, tactile corpuscle-like bodies

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Table 1
Clinical and endoscopic findings of colorectal MSCHs (n = 48).

		n	%
Age (years)		Mean: 60.4; range: 32–81	
Sex	Male	23	47.9
	Female	25	52.1
Indication for endoscopy	CRC screening	38	79.2
	GI/rectal bleeding	5	10.4
	Diarrhea	2	4.2
	Constipation	1	2.1
	Abdominal pain	1	2.1
	CRC follow-up	1	2.1
Location	Appendiceal orifice	2	4.2
	Cecum	3	6.3
	Ascending	4	8.3
	Transverse	5	10.4
	Descending	8	16.7
	Sigmoid	23	47.9
	Rectum	3	6.3
Endoscopic findings	Polyp	17	35.4
	Sessile polyp	29	60.4
	Random biopsy	1	2.1
	Mass base	1	2.1
Size (mm)		Mean: 3.7; range: 1–10	

MSCH, mucosal Schwann cell hamartoma; CRC, colorectal cancer; GI, gastrointestinal.

manifestations and endoscopic findings, were recorded.

Hematoxylin and eosin (H&E)-stained slides and available immunohistochemical stains were reviewed for all cases. Antibodies used for immunostains were as follows: S100 (Cell Marque, clone 4C4.9, 1:400 dilution), EMA (Agilent, clone E29, 1:2000 dilution), SMA (Agilent, clone 1A4, 1:1500 dilution), desmin (Agilent, clone D33, 1:200 dilution), CD34 (Agilent, clone QBEnd-10, 1:50 dilution), CD117 (Agilent, polyclonal, 1:800 dilution), DOG1 (Fisher, clone SP31, 1:100 dilution), and CD68 (Agilent, clone PGM1, 1:200 dilution). The Envision Plus detection system (Dako, Carpinteria, CA) was used for all antibodies. Appropriate positive and negative controls were included throughout.

3. Results

3.1. Clinical and endoscopic findings of colorectal MSCHs

A total of 48 cases of colorectal MSCHs were identified; 45 were in-house cases and 3 were consult cases. The clinical and endoscopic features are summarized in Table 1. Patients comprised 23 males and 25 females. The average age was 60.4 years old (range: 32–81 years). Thirty-eight (79.2%) patients had colonoscopy for colorectal cancer (CRC) screening. The indications for the remaining cases included GI/rectal bleeding (n = 5), diarrhea (n = 2), CRC follow-up (n = 1), constipation (n = 1), and abdominal pain (n = 1). None of them had an association with NF1 or other inherited syndromes. Thirty-one (64.6%) cases were left-sided, including the descending (n = 8) and sigmoid (n = 23) colon. All colorectal MSCHs except for 2 (46/48; 95.8%) were targeted biopsies from polypoid lesions. One patient presented with diarrhea and had random biopsies to rule out microscopic colitis, which incidentally showed a MSCH in the left colon. Another patient had a 2 cm cecal mass that showed a tubulovillous adenoma with extensive high-grade dysplasia on endoscopic mucosal resection (EMR). A biopsy from the mass base following EMR showed a MSCH unexpectedly. For targeted biopsies, all lesions (n = 46) were described as small or diminutive polyps on endoscopy reports, among which 29 (63%) were described as sessile polyps. The mean size of the polyps was 3.7 mm (range: 1–10 mm).

Table 2
Clinical, endoscopic and pathologic findings of GEJ MSCHs.

Case	Age (years)	Sex	Indication	Endoscopic findings	*H. pylori*	Background mucosa	Microscopic size (mm)	Positive IHC	Negative IHC
1	75	F	Iron deficiency anemia	N/A	Negative	N/A	N/A	S100	SMA, CD34, CD117, HHV8
2	57	F	GERD	Mild nodular carditis	Negative	Mild chronic inflammation	3	S100	SMA, CD68, CD1a, DOG1
3	75	M	Heartburn	Irregular and displaced Z-line	Positive	Mild chronic inflammation with superficial erosion	4	S100	Desmin, CD117, SMA
4	72	M	GERD	Irregular Z-line	Negative	Mild chronic and acute inflammation	2	S100	Desmin, CD117
5	76	M	Heartburn	Normal	Negative	Mild chronic and acute inflammation	3	S100	CD68
6	66	M	Dysphagia	Irregular Z-line	Negative	Mild chronic and acute inflammation	2	S100	SMA, CD117

GEJ, gastroesophageal junction; MSCH, mucosal Schwann cell hamartoma; GERD, gastroesophageal reflux disease; N/A, data not available; H. pylori, Helicobacter pylori; IHC, immunohistochemistry.

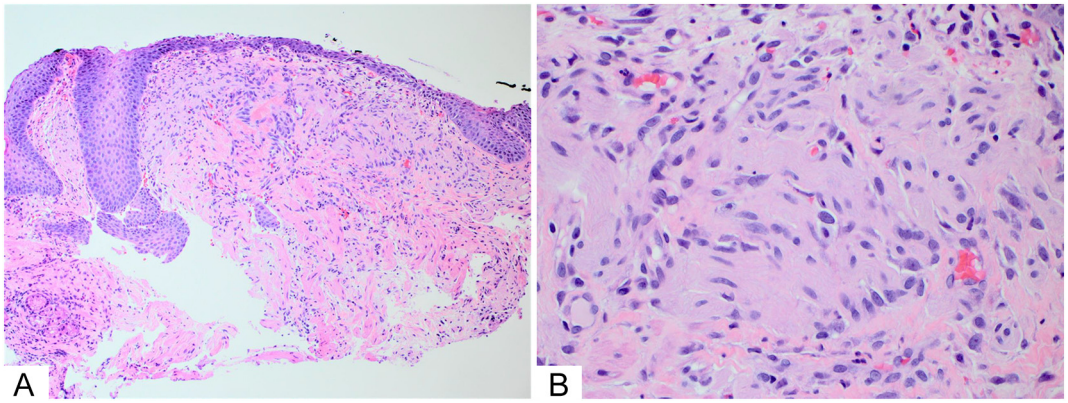

Fig. 1. An example of GEJ MSCH showing proliferative spindle cells in the lamina propria underneath attenuated squamous epithelium (A), suggestive of prior mucosal injury in this area (H&E, 100×). Higher magnification showing bland spindle cells with elongated nuclei, eosinophilic cytoplasm and indistinct cell borders (B). No significant nuclear pleomorphism or mitosis is seen (H&E, 400×).

3.2. Clinical and endoscopic findings of GEJ MSCHs

Six cases of GEJ MSCHs were identified (Table 2), 5 of which were consult cases, one was an in-house case. Four patients were male and two were female. The average age at diagnosis was 70.2 years old (rang: 57–76 years). Two patients had a history of gastroesophageal reflux disease (GERD), 2 had heartburn, one had iron deficiency anemia, and one had dysphagia. None of the patients had a polyp, nodule or other visible lesion at GEJ endoscopically. The mean size of the lesions, measured microscopically, was 2.8 mm (range: 2–4 mm). Among the 5 patients with available endoscopic reports, 3 showed irregular Z-line, one showed mild nodular carditis, and one was documented as normal. *H. pylori* gastritis was documented in one patient (16.7%).

3.3. Pathologic findings

The histologic features of colorectal and GEJ MSCHs were similar. The lesions consisted of un-encapsulated, diffusely distributed spindle cells in the lamina propria. The border between the lesion and the adjacent lamina propria was indistinctive and irregular (Fig. 1A). All the lesions were confined to the mucosa without involvement of the muscularis mucosae or submucosa. Scattered inflammatory cells, such as lymphocytes and eosinophils, were seen admixed with lesional cells. The lesional cells were characterized by relatively uniform, bland spindle cells with elongated nuclei, abundant eosinophilic cytoplasm and indistinct cell borders (Fig. 1B). There was no significant nuclear pleomorphism, but one case from the GEJ showed what appeared to be degenerative nuclear atypia or “ancient change” (Fig. 2A and B). No mitoses were identified. No granulomas, necrosis or ganglion cells were seen.

By immunohistochemistry, the lesional cells in all cases were positive for S100 protein in the nuclei and cytoplasm (Fig. 3A-C). Other markers including EMA, SMA, desmin, CD34, CD117, DOG1 and CD68 were all negative, except for one case biopsied from the descending colon that was also positive for CD34, and one case from the cecum that was focally positive for desmin.

Four of the colorectal MSCHs arose in the setting of a sessile serrated lesion, hyperplastic polyp or inflammatory polyp. The remaining 44 cases were isolated lesions found in unremarkable colonic mucosa (Fig. 4A and B). In comparison, all 5 cases of GEJ MSCHs, for which endoscopy reports and pathology slides were available, arose in inflamed GEJ mucosa. The overlying epithelium was squamocolumnar for 3 lesions, squamous for 1, and columnar (gastric cardic-type) for 1.

4. Discussion

MSCHs of the GI tract are rare benign lesions. In 2009, Gibson and Hornick reported the first series of 26 cases in the colorectum and the name of MSCH was coined [1]. Since then, < 20 case reports have been published [2-12]. The lesion was initially regarded to occur only in the colon and rectum, but recent reports showed that rare cases could also be seen in other parts of the GI tract, such as the stomach [13] and gallbladder [14]. Khanna and colleagues retrospectively examined 500 cholecystectomies for chronic cholecystitis after they incidentally found a MSCH in a gallbladder, and found MSCHs in 20 (4%) cases confirmed by immunohistochemistry [14]. It is worth noting that, in this study, all

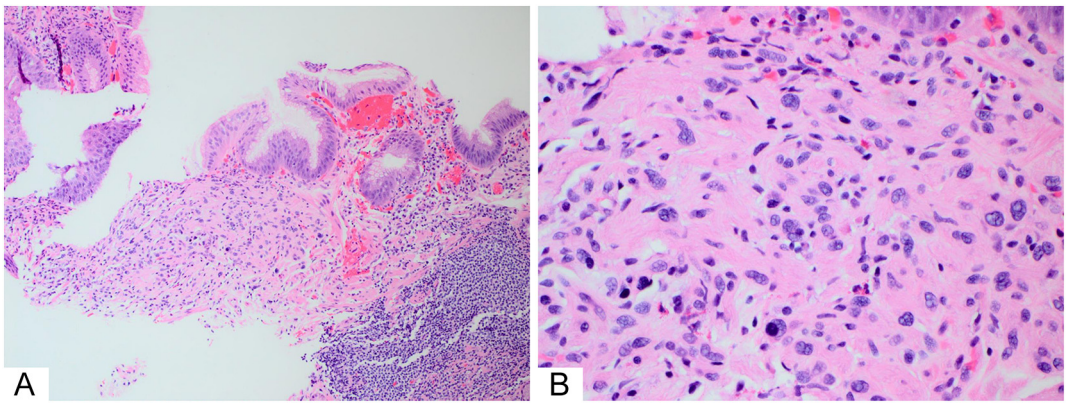

Fig. 2. Another example of GEJ MSCH showing a cluster of spindle cells in the lamina propria at the squamocolumnar junction (A). Note the presence of inflammatory cells in the adjacent mucosa (H&E, 100×). Higher magnification showing “ancient change” featuring enlarged and irregular nuclei (B). However, mitosis is absent (H&E, 400×).

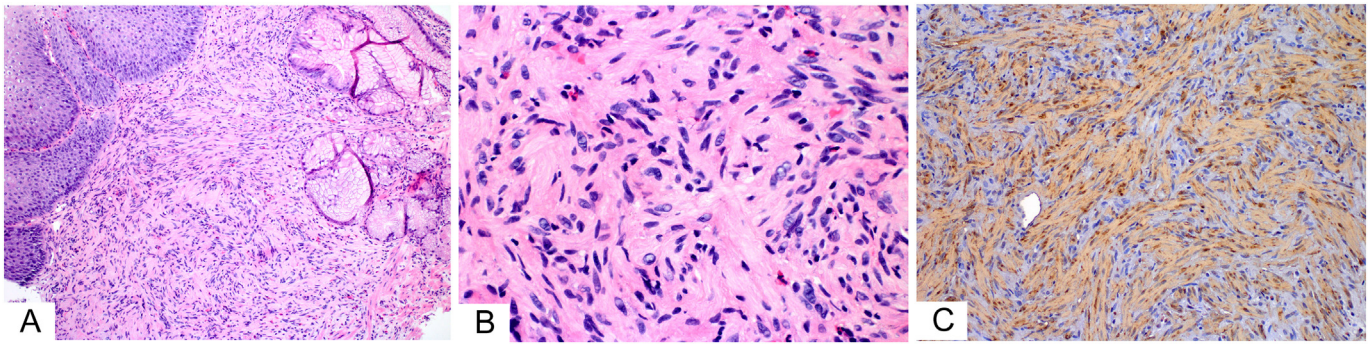

Fig. 3. Another example of GEJ MSCH showing spindle cell proliferation in the lamina propria lined by squamocolumnar epithelium (A; H&E, 100×). Higher magnification showing minimal nuclear pleomorphism and no mitosis (B). Note the presence of occasional eosinophils within the lesion (H&E, 400×). Lesional cells are positive for S100 by immunohistochemistry (C), confirming their Schwann cell origin (200×).

the lesions were missed on the initial examination. The gallbladder lesions were all restricted to the subepithelial mucosal region. In the present study, we analyzed a series of 54 MSCH cases (48 from the colorectum and 6 from the GEJ), which is the largest series of MSCHs in the GI tract and the first with the GEJ as a reported site. The findings in our study along with the retrospective study of Khanna et al. [14], lead us to suspect that MSCHs are probably more common in the GI tract than originally thought; however, they are likely overlooked endoscopically, as they are not always polypoid, as well as microscopically.

As summarized in Table 3, the reported colorectal MSCHs typically present as small polypoid mucosal lesions. Most lesions are incidental findings during screening colonoscopy. None of the cases are associated with an inherited syndrome. Interestingly, different from their colorectal counterparts, none of the GEJ MSCHs in our series presented as a polyp, nodule or mucosal elevation. Instead, 3 cases showed an irregular Z-line and one case showed mild nodular carditis endoscopically.

Histologically, MSCHs are composed of relatively uniform spindle cells in the lamina propria, which expand the space between the glands or crypts. The lesional cells are bland-appearing with elongated, tapering, or wavy nuclei, abundant eosinophilic cytoplasm, and indistinct cell borders. Occasionally, MSCH can have degenerative atypia ("ancient change"), but nuclear atypia is usually minimal (if any) and mitosis is consistently absent. Immunohistochemically, the lesional cells are positive for S100 protein, and negative for CD34, GFAP, EMA, SMA, CD117, DOG1 and CD68.

In the colorectum, MSCHs need to be differentiated from mucosal perineurioma (formerly known as "benign fibroblastic polyp"). This lesion is also characterized by a bland spindle cell proliferation that is limited to the colonic mucosa [15]. In contrast to MSCHs, however, this lesion is frequently associated with serrated colonic crypts and is immunohistochemically positive for EMA and GLUT1 and negative for S100. To our knowledge, mucosal perineurioma has not been described in the GEJ. The most common benign spindle cell lesion in the GEJ is leiomyoma, which is typically much larger in size and can be easily differentiated from a MSCH by its histologic and immunohistochemical characteristics. Gastrointestinal stromal tumor (GIST) can rarely occur at the GEJ and show S100 positivity [16], but this lesion is not mucosa-confined and can be easily diagnosed with positive immunostains for CD117 and DOG1. Localized scar caused by reflux esophagitis or other insults may be seen in the distal esophagus and GEJ, with fibroblast/myofibroblast proliferation in the lamina propria, which may mimic a MSCH. However, the proliferating spindle cells in a scarred area may show positive staining for SMA but should be negative for S100.

Other neural lesions that may enter the differential include Schwannoma, neurofibroma, and ganglioneuroma. Among them, neurofibroma is the most important differential given its strong association with NF1. In NF1 patients, the GI involvement is usually diffuse, commonly involving the stomach and small intestine. Sporadic GI cases are extremely rare [17,18]. Morphologically, neurofibromas have heterogeneous cellular components, including Schwann cells, perineural-like cells, axons and fibroblasts. Although positive for S100, the staining is usually focal and uneven, unlike in MSCHs as they are composed of pure Schwann cells.

Ganglioneuromas are benign lesions composed of ganglion cells, nerve fibers, and Schwann cells, which have been subclassified into two general groups: solitary isolated lesions and multiple lesions (ganglioneuromatous polyposis or ganglioneuromatosis) [19]. The latter group has a significant association with Cowden syndrome or MEN 2B. The majority of solitary ganglioneuromas are found in the colon,

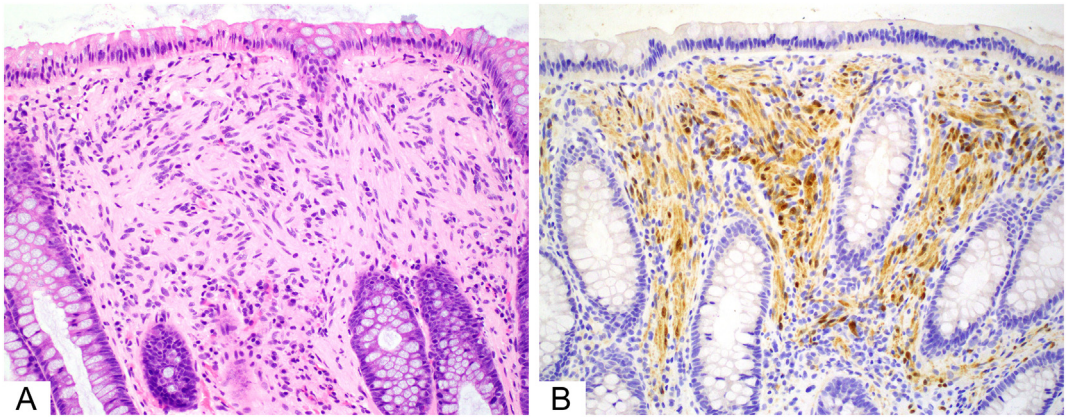

Fig. 4. An example of colonic MSCH showing spindle cell proliferation in the lamina propria expanding the space between unremarkable crypts (A; H&E, 200×). Lesional cells are positive for S100 by immunohistochemistry (B; 200×).

Table 3
Summary of clinical and endoscopic features of reported colorectal MSCHs in the literature and present study.

No. of cases	Age (years)	Sex	Endoscopic findings	Size (mm)	Location	Indication/symptom	Reference
26	Mean 62 (46–88)	M:F = 10:16	Sessile polyp	Mean 2.5 (1–6)	Predominantly in rectosigmoid colon	Screening (most common), diarrhea, lower GI bleeding	Gibson and Hornick, 2009 [1]
1	60	F	Sessile polyp	5	Rectosigmoid colon	Occult blood in stool	Pasquini et al., 2009 [8]
1	67	F	Sessile polyp	3	Sigmoid colon	Asymptomatic	Rocco et al., 2011 [7]
1	40	M	Many small whitish nodules in mucosa		Sigmoid colon	Occult blood in stool	Sagami et al., 2012 [27]
1	41	F	polyp	8	Descending colon	Asymptomatic	Bae et al., 2013 [11]
1	59	M	polyp	3	Sigmoid colon	Underlying ulcerative colitis	Neis et al., 2013 [9]
1	72	M	polyp	5	Sigmoid colon	Asymptomatic	Ferro de Beca et al., 2014 [6]
1	78	F	Polyp, 7 mm along with rectal erythema and inflammation	7	Rectum	Abdominal pain and intermittent tenesmus	Klair et al., 2014 [5]
1	67	M	Polyp	6	Sigmoid colon	Asymptomatic	Kanar et al., 2015 [4]
1	20	M	Polyp and scattered tiny polyp-like mucosal elevations	4	Rectum	Abdominal discomfort and loose stools	Bae et al., 2015 [28]
1	49	M	Tiny polyp-like mucosal elevation	2	Rectum	Asymptomatic	Han et al., 2017 [10]
1	55	F	polyp	5	Ascending	Asymptomatic	Chintanaboina et al., 2018 [2]
48	Mean 60.4 (32–81)	M:F = 23:25	Predominantly polyp (95.8%)	Mean 3.7 (1–10)	Predominantly in left-sided colon	Screening, GI bleeding, diarrhea, CRC follow-up, constipation, abdominal pain	Current study

usually detected as a small sessile polyp. The histologic recognition of ganglion cells enables its differentiation from a MSCH.

In the GI tract, Schwannomas are most commonly seen in the stomach and are rarely found at the GEJ. The lesions are composed of Schwann cells arranged in vague fascicles. GI Schwannomas seldom show nuclear palisading (Verocay bodies), but unlike MSCHs, they are larger, well-demarcated tumors and have characteristic peripheral lymphoid cuffs.

GI granular cell tumors, most commonly seen in the esophagus, are also neurogenic [20,21]. Histologically, tumor cells are large and round to polygonal with abundant eosinophilic and granular cytoplasm, in contrast to the spindle cell morphology seen in MSCHs. In addition to being strongly and diffusely positive for S100, granular cell tumors are often positive for CD68, which is consistently negative in MSCHs.

Tactile corpuscle-like bodies (TCLBs), also called Wagner-Mesissner corpuscles in the skin and sensitive mucosal surfaces [22], are microscopic Schwannian structures that can be found throughout the GI tract including the esophagus, GEJ, stomach and colon. They have the same immunophenotype as MSCHs, i.e., S100 positivity. Morphologically, TCLBs are relatively well-demarcated small clusters, ranging from < 0.1 to 1.5 mm in greatest dimension, of lamellated, concentrically arranged spindle cells in the lamina propria. The cells have eosinophilic cytoplasm and peripherally placed nuclei, which is characteristic [23]. Interestingly, MCSHs may occasionally have tactile corpuscle-like structures [6]. It is thus possible that TCLBs in the GI tract might represent part of the morphologic spectrum of MSCHs.

Most mucosal neuromas of the GI tract diagnosed before the publication by Gibson and Hornick in 2009 are likely to be MSCHs. True mucosal neuromas (other than traumatic neuromas) are considered exceedingly rare in the GI tract, which may be associated with MEN 2B [24]. These lesions are mainly found in the lips, tongue, oral cavity and eyelids. When involving the GI tract, they are almost always multiple, whereas the reported MSCHs are always a single occurrence. Histologically, mucosal neuromas are composed of hyperplastic nerve bundles and have perineurial capsules, which are often EMA-positive. It should be pointed out here that the term of MSCH may not be universally accepted and other terms for this lesion may still be used by pathologists. However, the term “mucosal neuroma” should be avoided mainly because it may signify an association with an inherited syndrome as discussed above.

Although the exact etiology is unknown, MSCHs likely represent a reactive process following mucosal injury. In the gallbladder, the lesions are commonly associated with neuromatoid hypertrophy of the inter-muscular nerve plexus [14]. An association with ulcerative colitis is also reported [9]. In our series of GEJ MSCHs, all cases are seen in the background of inflammation. In the colon, MSCHs are more commonly seen in the rectosigmoid, the segment that is most likely to suffer from mechanical injury. In addition, Schwann cell proliferation is a common finding in obliterated appendices, formerly termed “appendiceal neuroma” [25], which has been linked to a chronic inflammatory process. Stockl et al. reported Schwann cell proliferation in 20% (9/45) of normal adult appendices and as high as 42% (10/24) of appendiceal diverticula cases [26]. We assume that MSCHs in the GI tract may represent the same process as in the appendix and gallbladder. Thus, mucosal Schwann cell “proliferation” may be a more appropriate term than “hamartoma”.

In summary, MSCH is an uncommon mucosal spindle cell lesion of the GI tract. It is most commonly seen in the colorectum, particularly the left colon, but can also be found in the GEJ, stomach and gallbladder. GEJ MSCHs share the same histologic and immunohistochemical features with their colorectal counterparts, but are usually an incidental finding with no endoscopically visible lesion. There is currently no evidence that MSCHs are related to inherited syndromes or malignancies. Additional treatment, work-up or follow-up is thus unnecessary.

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