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Research Article

Keywords: SAIM, perforation, lower gastrointestinal tract, diverticulum

Posted Date: July 13th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10154640/v1>

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Additional Declarations: No competing interests reported.

Segmental Absence of Intestinal Musculature and Its Mimics in Adult Lower Gastrointestinal Perforation: A Review of 100 Cases

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ABSTRACT

Background: Segmental absence of intestinal musculature (SAIM) is an uncommon and/or underrecognized cause of lower gastrointestinal perforation and is often misdiagnosed as diverticular disease or ischemic injury.

Methods: We retrospectively reviewed 100 adult resections for lower gastrointestinal perforation and assessed the presence of histopathological features consistent with SAIM. Cases were classified as SAIM when they showed: (1) preservation of the mucosa surrounding the perforation site; (2) segmental loss of the muscularis propria; (3) histological continuity between the submucosa and subserosa; and (4) absence of mucosal outpouching suggestive of diverticular disease.

Results: The major causes of perforation were diverticular disease (45%) and ischemia (15%). Among the six cases initially diagnosed as SAIM, three were reclassified as non-SAIM due to submucosa–subserosa discontinuity or associated diverticula, while three additional cases were identified, yielding six cases considered compatible with SAIM. Perforations showed a well-demarcated, usually abrupt absence of the muscularis propria, highlighted by desmin immunohistochemistry. SAIM was identified on the oral and/or anal side of the perforation in five cases and only laterally in one case. Elastic fiber stains helped distinguish true submucosa–subserosa continuity from fibrinous pseudo-apposition.

Conclusion: Recognition of SAIM remains challenging in perforated specimens, particularly in the presence of diverticular disease or secondary inflammatory changes. Careful gross examination, appropriate sampling from the oral/anal sides of the perforation, and ancillary stains may facilitate pathological evaluation.

Keywords

SAIM, perforation, lower gastrointestinal tract, diverticulum

Introduction

Segmental absence of intestinal musculature (SAIM) is characterized by focal loss of the muscularis propria without fibrosis, whereas the mucosa and subserosa remain intact. SAIM was first described in neonates [1], but it has increasingly been reported in adults [2-4], most often presenting with perforation, obstruction, or peritonitis [4, 5]. Diagnosis depends on a histopathological examination and is typically made after surgery for an abdominal emergency or suspected neoplasm [4, 6]. The pathogenesis remains uncertain; proposed mechanisms include congenital anomaly, developmental vascular insult, and acquired injury that unmasks a preexisting structural weakness [4].

Although SAIM in adults has become increasingly recognized as a potential cause of intestinal perforation, its diagnostic features remain incompletely defined. Differentiating SAIM from its mimics—such as diverticular disease, ischemic muscle loss, or other causes of perforation with secondary tissue alteration—can be challenging, particularly in limited specimens. In addition, practical guidance regarding gross examination, tissue sampling, and histological assessment remains limited.

We retrospectively reviewed 100 cases of adult lower gastrointestinal perforation to identify lesions showing histopathological features consistent with SAIM and to describe their clinicopathological findings in comparison with other perforation-associated lesions. We specifically evaluated histological patterns, including abrupt or tapering loss of the muscularis propria and apparent continuity between the submucosa and subserosa, and assessed the utility of desmin immunohistochemistry (IHC) and elastic fiber staining. We also examined practical aspects of gross examination and tissue sampling relevant to routine pathological practice.

Materials and Methods

Patient Selection

A total of 100 adult patients who underwent bowel resection for lower gastrointestinal perforation (jejunum to rectum) between January 2007 and April 2022 were included. Cases of perforated appendicitis were excluded. All specimens underwent pathological review, and clinical data were retrospectively obtained from medical records. This study was approved by the Asahikawa Medical University Research Ethics Committee (No. 22123) and conducted in accordance with the Declaration of Helsinki.

Histopathologic Evaluation

Tissue samples were fixed in 10% neutral-buffered formalin and embedded in paraffin. Sections of 4 μ m thickness were stained with hematoxylin and eosin (H&E). Elastica van

Gieson (EVG) or Elastica–Masson staining and IHC for desmin were performed at the time of postoperative diagnosis, and additional stains were applied for the purpose of this study when necessary. For this study, lesions were considered to show histopathological features consistent with SAIM when the following findings were present: (1) preservation of the mucosa surrounding the perforation site; (2) segmental loss of the muscularis propria; (3) histological continuity between the submucosa and subserosa; and (4) absence of mucosal outpouching suggestive of diverticular disease. Findings indicative of pseudo-SAIM included intervening spaces, with or without fibrinous exudate, between the submucosa and subserosa; structural alterations related to diverticular disease; and attenuation or interruption of the muscularis propria due to ischemic injury and subsequent luminal dilatation. Cases showing histological features consistent with SAIM (SAIM group) were compared with the remaining cases (non-SAIM groups) with respect to their clinicopathologic features.

Statistical Analysis

Comparisons between the SAIM and non-SAIM groups were performed using nonparametric tests. The Mann–Whitney U test was used for continuous variables, and Fisher’s exact test was applied for categorical variables. Statistical analyses were performed using EZR (version 1.64), a modified version of R Commander [7], with a two-sided significance level of 5% ($P < 0.05$ was considered statistically significant).

Results

Overall Cohort Characteristics

Among the 100 patients, diverticular disease was the most frequent cause of perforation, occurring in 45 patients (45%), followed by ischemia in 15 (15%) (Table 1). Six cases showed histopathological features consistent with SAIM. After pathological review, three initially diagnosed SAIM cases were reclassified as non-SAIM, whereas three additional cases were newly recognized as SAIM. The details of these cases are described below. The sigmoid colon was the most common site of perforation in both the SAIM and non-SAIM groups, accounting for 50% and 46% of cases, respectively (Table S1). In the comparative analysis, no significant clinical differences were observed between the two groups with respect to age, sex, body mass index, white blood cell count, C-reactive protein level, maximum perforation diameter, postoperative hospital stay, 30-day mortality, or in-hospital mortality (Table S2). No cases showed clinical, intraoperative, or pathological findings suggestive of vascular Ehlers–Danlos syndrome (vEDS).

Clinicopathological Features of SAIM Cases

Clinical Characteristics:

The median age of the six patients was 77 years (range, 68–84 years) and included three male and three female. The most frequent site of perforation was the sigmoid colon (n = 3), followed by the rectum (n = 1), transverse colon (n = 1), and ileum (n = 1). Factors contributing to increased intraluminal pressure included fecal impaction in two cases and colorectal cancer in one case; the factors were unknown in the remaining three (Table 2).

Gross Findings:

The median perforation size was 10 mm (range, 5–40 mm) (Table 2). The perforations appeared round-to-oval or “punched-out” (Fig. 1a). On cross-section, band-like defects of the muscularis propria extended to the oral side (n = 1), anal side (n = 1), or both sides (n = 2) of the perforation site (Fig. 1b). Of the remaining two cases, one had not been examined on cross sections, and in the other, the defect of the muscularis propria was not grossly evident. No diverticula were identified near the perforation, except in one case (case 6). Four SAIM lesions were located on the mesenteric side and two on the antimesenteric side (Table 2).

Histological and Immunohistochemical Findings:

In all six cases, the muscularis propria was completely absent in the affected area, and the subserosa was located directly beneath the submucosa without any intervening space. These findings were observed on the anal side (n = 3), on both the oral and anal sides (n = 1), on the oral and lateral sides (n = 1), and on the lateral side (n = 1). A median of 3 tissue blocks (range, 1–4 blocks) per SAIM case were examined from the perforation site and adjacent bowel wall, and sections meeting all four study criteria for SAIM were identified in a median of 1.5 blocks (range, 1–3 blocks) per case (Table 2). The median size of the SAIM lesion (muscular defect) was 21 mm in length (range, 16–70 mm) and 12.5 mm in width (range, 5–23 mm), although in many cases, either the length or the width could not be assessed because of the sectioning method. Either abrupt or tapered disappearance of the muscularis propria was observed—abrupt in four cases and tapered in one (Fig. 2 and 3; Table 2). In the ileal case, both abrupt and tapered defects of the muscularis propria were present. In case 4, a penetrating vessel was identified at the margin of the muscular defect, whereas no such vessels were identified in the remaining cases. The muscularis mucosae adjacent to the perforation site was preserved in all six SAIM cases. Nerve plexuses were identified in only one case, which exhibited a tapered-off pattern; they were located at the submucosa–subserosa interface and could not be

distinguished as either a residual Auerbach plexus or a Henle's/Schabadasch's plexus (Fig 3b; Table 2). In some cases, elastic fiber staining showed that elastic fibers near the serosal surface were closely apposed to the submucosa at the point where the muscularis propria abruptly disappeared, with the serosal surface located directly beneath the submucosa (Fig. 2d).

As described above, three of the six SAIM cases (Cases 4–6) were identified during pathological review (Table 2). Cases 4 and 5 had originally been diagnosed as perforations of unknown cause but were subsequently considered consistent with SAIM because they fulfilled all four study criteria. Case 6 had initially been diagnosed as a diverticular perforation; however, after re-evaluation, no true diverticulum was identified macroscopically and microscopically, and the lesion was ultimately considered consistent with SAIM (Fig. 4). In this case, diverticula were present near the perforation site (Fig. 4c), and the clinical diagnosis had been diverticular perforation, making the pathological interpretation particularly challenging.

Features of non- and pseudo-SAIM cases

In non-SAIM cases, the subserosa was often observed beneath the submucosa due to contraction of the ruptured muscularis propria. When the submucosa and subserosa were separated by an intervening space, with or without fibrinous exudate, these findings suggested that the two layers had not been contiguous before perforation (Fig. 5a–d). In some cases, the intervening space was narrow and filled with fibrinous exudate, making the layers appear continuous and thereby mimicking SAIM. Such cases were regarded as “pseudo-SAIM,” and this pattern was observed in two cases who had initially been diagnosed with SAIM but were subsequently reclassified as non-SAIM. In both cases, no specific pathological abnormality, including SAIM, was identified at the perforation site. Fecal impaction and barium retention were considered clinical precipitating factors in these cases, respectively. Similar pseudo-SAIM changes were focally observed in some SAIM cases; however, these cases were classified as SAIM because other examined sections fulfilled all four study criteria for SAIM.

Diverticular perforations can also mimic SAIM. In diverticula, the mucosa and submucosa protrude through the muscularis propria. When the diverticular sac extends to the subserosa, the muscularis propria may be absent, resulting in direct apposition of the submucosa and subserosa. In one case that had initially been diagnosed as SAIM but was subsequently reclassified as non-SAIM, one section fulfilled all four study criteria for SAIM (Fig. 5e). However, an adjacent section demonstrated mucosal outpouching toward the subserosa, favoring a diverticular origin rather than SAIM (Fig. 5f).

The muscularis propria may also appear attenuated or interrupted as a result of ischemic injury and subsequent luminal dilatation. In contrast to SAIM, these changes are typically diffuse rather than segmental and lack a well-demarcated muscular defect. However, when inflammation or ischemic injury is severe, distinction from SAIM could be difficult on H&E staining alone. In such cases, IHC for desmin is helpful in delineating the extent and pattern of muscularis propria loss (Fig. 5g).

Discussion

SAIM in adults is an increasingly recognized, though uncommon, cause of lower gastrointestinal perforation. In our retrospective review of 100 adult cases of lower gastrointestinal perforation, six cases showed histological findings consistent with SAIM. Our findings highlight the diagnostic challenges associated with these lesions and emphasize the importance of careful gross examination, adequate tissue sampling, and ancillary staining for their evaluation.

Diverticular perforations may histologically resemble SAIM, and enlargement of the perforation or superimposed inflammation can obscure the underlying diverticular structure. In addition, if the mucosa within a diverticular region is embedded at the same level as the adjacent mucosa during paraffin embedding, the lesion may appear to fulfill all four study criteria for SAIM. Grossly, SAIM typically shows a sharply demarcated, round-to-oval, or “punched-out” perforation, in contrast to the irregular morphology seen in diverticulitis. Diverticula typically arise at sites where penetrating vessels (vasa recta) traverse the muscularis propria, resulting in focal weakness of the colonic wall [8]. In our series, five of the six SAIM lesions did not correspond to penetrating-vessel entry sites, whereas only one case demonstrated a penetrating vessel at the margin of the muscular defect. These findings suggest that the muscular defects observed in most cases were unlikely to be attributable solely to physiological weak points associated with vascular penetration, which are typically implicated in diverticular formation. Nevertheless, we cannot entirely exclude the possibility that a markedly distorted diverticulum was present but obscured by tissue distortion and inflammation.

Proper sectioning and adequate sampling are also crucial. In our series, band-like defects of the muscularis propria were grossly observed on the oral side, anal side, or both sides of the perforation site in most cases. In addition, histological features consistent with SAIM were identified on the oral and/or anal sides of the perforation in five of the six cases. These findings suggest that, in addition to the perforation site itself, careful sampling of both the oral and anal sides is important for the diagnosis of SAIM.

IHC for desmin and elastic fiber stains (EVG or Elastica–Masson) are useful to

recognize the abrupt or tapering disappearance of muscle fibers, which helps differentiate SAIM from inflammatory or ischemic injury. Elastic fiber stains may also demonstrate fiber migration, as elastic fibers near the serosal surface move toward the submucosa after the muscularis propria is torn, making it easier to identify the serosal surface located just below the submucosa.

While SAIM may be underrecognized, our findings suggest that overdiagnosis is also possible when secondary inflammatory and reparative changes are superimposed. Pseudo-SAIM may mimic true muscular defects, particularly in the setting of severe inflammation or necrosis. In our series, two cases initially diagnosed as SAIM were later reclassified as pseudo-SAIM because an intervening space was identified between the submucosa and subserosa, indicating that these layers had not been directly apposed before perforation. Because perforation is followed by rapid fibrin deposition, adjacent tissue layers may become secondarily adherent. Therefore, the presence of fibrin-containing spaces between the submucosa and subserosa suggests secondary apposition rather than true continuity and may argue against SAIM [9, 10]. Likewise, granulation tissue or fibrosis between the submucosa and subserosa favors secondary injury rather than a primary absence of the muscularis propria. Elastic fiber stains are particularly useful for evaluating the continuity between the submucosa and subserosa, because they highlight discontinuity between these layers, especially when intervening fibrinous exudate, granulation tissue, or fibrosis is present.

In our cohort, diverticular disease (45%), ischemia (15%), and SAIM (6%) represented the leading causes of perforation. The proportion of SAIM in our series was lower than the 24% reported by Tsuyuki et al. [3], in which SAIM represented the second most common cause of intestinal perforation. This difference may reflect variations in the histological features used to identify SAIM. Notably, histological continuity between the submucosa and subserosa was not included in their definition, which may have contributed to differences in the proportion of SAIM. In addition, the relatively low proportion of SAIM in our series may partly reflect underrecognition due to limited tissue sampling. In 22 cases, no specific pathological abnormality causing perforation could be identified, and because only a single section from the perforation region was examined in some of these cases, SAIM may have been overlooked.

A comprehensive review of 49 adult cases reported in the literature revealed consistent clinicopathological features (Table S3)[2-6, 11-23]. Most patients were in their seventh to eighth decades of life, although the reported cases ranged from the second to eighth decades, with a slight male predominance. The sigmoid colon and rectum were the most frequently affected sites, followed by the ascending colon and distal small intestine.

Approximately 90% of patients presented with acute abdominal symptoms caused by intestinal perforation, whereas only a minority were diagnosed incidentally during unrelated surgical procedures, such as Whipple operations [6].

As summarized in Table S3, previously published descriptions of adult SAIM vary in the emphasis placed on (i) the extent and configuration of muscularis propria loss (abrupt versus tapered, band-like), (ii) the status of the mucosa and muscularis mucosae, and (iii) how strictly diverticular architecture and perforation-related artifacts are excluded. In our pathological reassessment, we focused on two practical issues in challenging specimens: (1) exclusion of diverticular architecture through careful gross-histological correlation and examination of adjacent sections, and (2) interpretation of submucosa–subserosa apposition in the context of perforation-related secondary adhesion, where an intervening fibrinous layer may create pseudo-continuity. These considerations may help reduce overdiagnosis of SAIM in inflamed perforation specimens, although very focal lesions may still be missed when tissue sampling is limited.

Several mechanisms have been proposed for adult SAIM, including congenital muscular deficiency, acquired ischemic injury during development, mechanical stretching, and subclinical inflammation [24]. Some authors have also suggested that both intrinsic wall fragility and increased intraluminal pressure may contribute to the development of SAIM-related perforation [17, 23]. In addition, a subset of patients has been reported to have heritable connective tissue disorders, particularly vEDS [19, 20]. Although none of our patients had a confirmed diagnosis of vEDS, genetic evaluation should be considered in young adults, in individuals with a family history of connective tissue disorders, or in those with multiple spontaneous bowel perforations.

Recognition of SAIM has clinical implications, as overlooked lesions at anastomotic sites may lead to postoperative anastomotic complications [21]. When a round-to-oval, smooth-edged perforation is encountered intraoperatively and no diverticula are identified, SAIM should be included in the differential diagnosis. In such cases, careful assessment of the intestinal wall adjacent the perforation may be helpful, and construction of an anastomosis in a segment with grossly intact muscularis propria should be considered. Intraoperative frozen section assessment may also be helpful when SAIM is suspected. SAIM has also been reported to involve multiple intestinal segments in the same patient, either synchronously or metachronously [4, 14, 20]. Therefore, in patients diagnosed with or suspected of having SAIM, a cautious management approach may be warranted to avoid marked increases in intraluminal pressure. This includes surveillance for obstructive lesions, control of constipation, and careful endoscopic technique with attention to insufflation.

This study has several limitations, including potential selection bias due to its retrospective, single-center design limited to surgical cases; variability in tissue fixation and specimen sampling; non-standardized immunohistochemical panels; absence of blinded re-evaluation by multiple pathologists; and the potential misclassification of lesions in the context of diverticular disease, as some cases may represent everted or partially everted diverticula and limited tissue sampling may have failed to detect adjacent diverticula. In addition, the histopathological features used to identify SAIM lesions were applied to perforated specimens and have not been validated against non-perforated lesions. Because only six SAIM cases were identified, the comparative analyses of clinical findings (Table S2) are exploratory and should be interpreted cautiously; the study was not powered to detect modest clinical differences between SAIM and non-SAIM perforations.

Conclusion

Recognition of SAIM remains challenging in perforated specimens, particularly in the presence of diverticular disease or secondary inflammatory changes. Careful gross examination, appropriate specimen preparation, and systematic sectioning, including sampling of both the oral and anal sides of the perforation, are important. The use of ancillary stains such as elastic fiber staining and desmin IHC may facilitate pathological evaluation. Larger multicenter studies with standardized sampling protocols are needed to better define clinicopathological correlates and management implications.

Supplementary information

The online version contains supplementary material.

Acknowledgments

We sincerely thank the laboratory staff of the Department of Diagnostic Pathology and Hepatobiliary Pancreatic, Transplantation and Gastrointestinal Surgery at Asahikawa Medical University for their valuable contributions and critical reading of the manuscript.

Author contribution

T. Takeda and S. Yuzawa designed the study. T. Takeda, S. Yuzawa, M. Hayashi, S. Mizukami, Y. Kamikokura, and M. Tanino performed histological examinations. T. Takeda, M. Otani, M. Ohara, C. Tani, T. Shonaka, K. Hasegawa and H. Yokoo provided clinical data. T. Takeda and S. Yuzawa drafted the manuscript, and other authors revised it critically. M. Tanino supervised the project. All authors read and approved of the final

manuscript.

Funding statement

None.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval statement

This study was approved by the Asahikawa Medical University Research Ethics Committee (No. 22123) and conducted in accordance with the Declaration of Helsinki.

Consent for publication

All the authors have approved the manuscript and consent to its publication.

Patient consent statement

Not applicable.

Competing interests

The authors declare no competing interests.

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Table 1. Causes of lower gastrointestinal perforation

Pathological substrate at the perforation site	N (%)
Diverticulitis	45 (45)
Ischemic injury	15 (15)
Segmental absence of intestinal musculature (SAIM)	6 (6)
Malignancy involving the perforation site [†]	5 (5)
Infectious enterocolitis	2 (2)
Amyloidosis	2 (2)
Ulcerative colitis	1 (1)
Endometriosis	1 (1)
Drug-induced	1 (1)
No specific pathological abnormality identified [‡]	22 (22)
Clinical precipitating factors[§]	N (%)
Fecal impaction	5 (5)
Complications of colonoscopy	4 (4)
Barium	2 (2)
Foreign body	2 (2)
Trauma	2 (2)
Anastomotic leakage	1 (1)

[†] In cases with colorectal carcinoma, perforations were classified as “malignancy involving the perforation site” only when the perforation occurred within the tumor; perforations occurring in non-neoplastic bowel were evaluated separately.

[‡] “No specific pathological abnormality identified” indicates the absence of identifiable structural or inflammatory abnormalities at the perforation site.

[§] Clinical precipitating factors were recorded when clinically documented and were not considered primary pathological causes of perforation; accordingly, they were not identifiable in all cases.

Table 2. Clinical and histopathological characteristics of SAIM cases

Case	Age/ Sex	Location	Perforation shape	Type of defects of MP	Side (M/ AM)	Section orientation relative to the bowel axis	Maximum perforation diameter (mm)	SAIM length (mm)	SAIM width (mm)	Side of MP defect (Gross)	Side of MP defect (Histology)	Sections meeting SAIM criteria/Sections from the perforation area	Penetrating vessel	Nerve plexus	Increased intraluminal pressure	Initial diagnosis
1	83/ F	Rectum	Oval	AD	M	Transverse	40	70	5	A	A	3/4	-	-	CRC	SAIM
2	84/ F	S/C	Oval	AD	M	Longitudinal	25	21	NA	O and A	A	1/3	-	-	Fecal impaction	SAIM
3	75/ M	S/C	Oval	TO	M	Transverse	11	NA	23	O	O and L	3/3	-	+	Fecal impaction	SAIM
4	77/ M	T/C	Oval	AD	AM	Transverse	10	NA	10	-	L	1/3	+	-	Unknown	Unknown
5	68/ M	Ileum	Circular	TO and AD	AM	Transverse	10	NA	15	NA	A	2/3	-	-	Unknown	Unknown
6	77/ F	S/C	Circular	AD	M	Longitudinal	5	16	NA	O and A	O and A	1/1	-	-	Unknown	Diverticulum

Abbreviation: SAIM, segmental absence of intestinal musculature; MP, muscularis propria; T/C, transverse colon; S/C, sigmoid colon; AD, abrupt disappearance; TO, tapered-off; M, mesenteric; AM, antimesenteric; NA, not applicable; A, anal; O, oral; L, lateral; CRC, colorectal cancer

Fig. 1

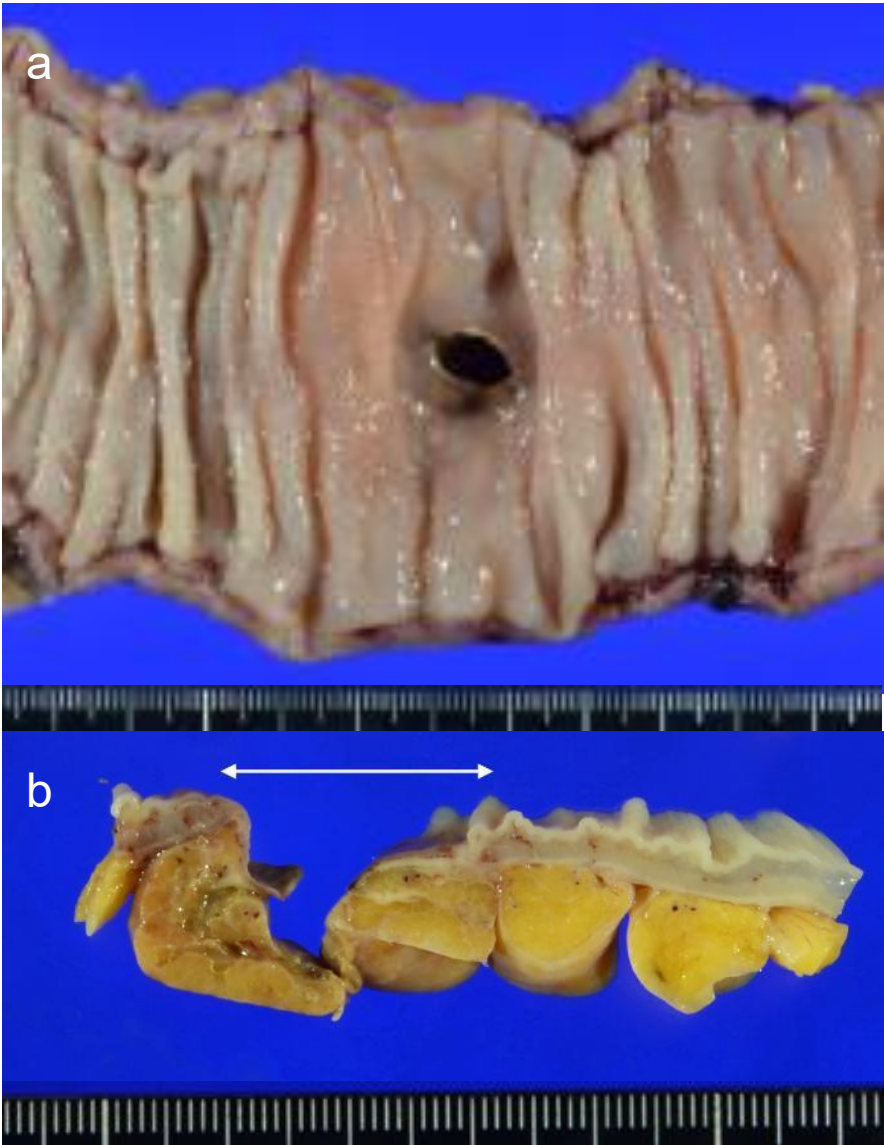


Fig. 2

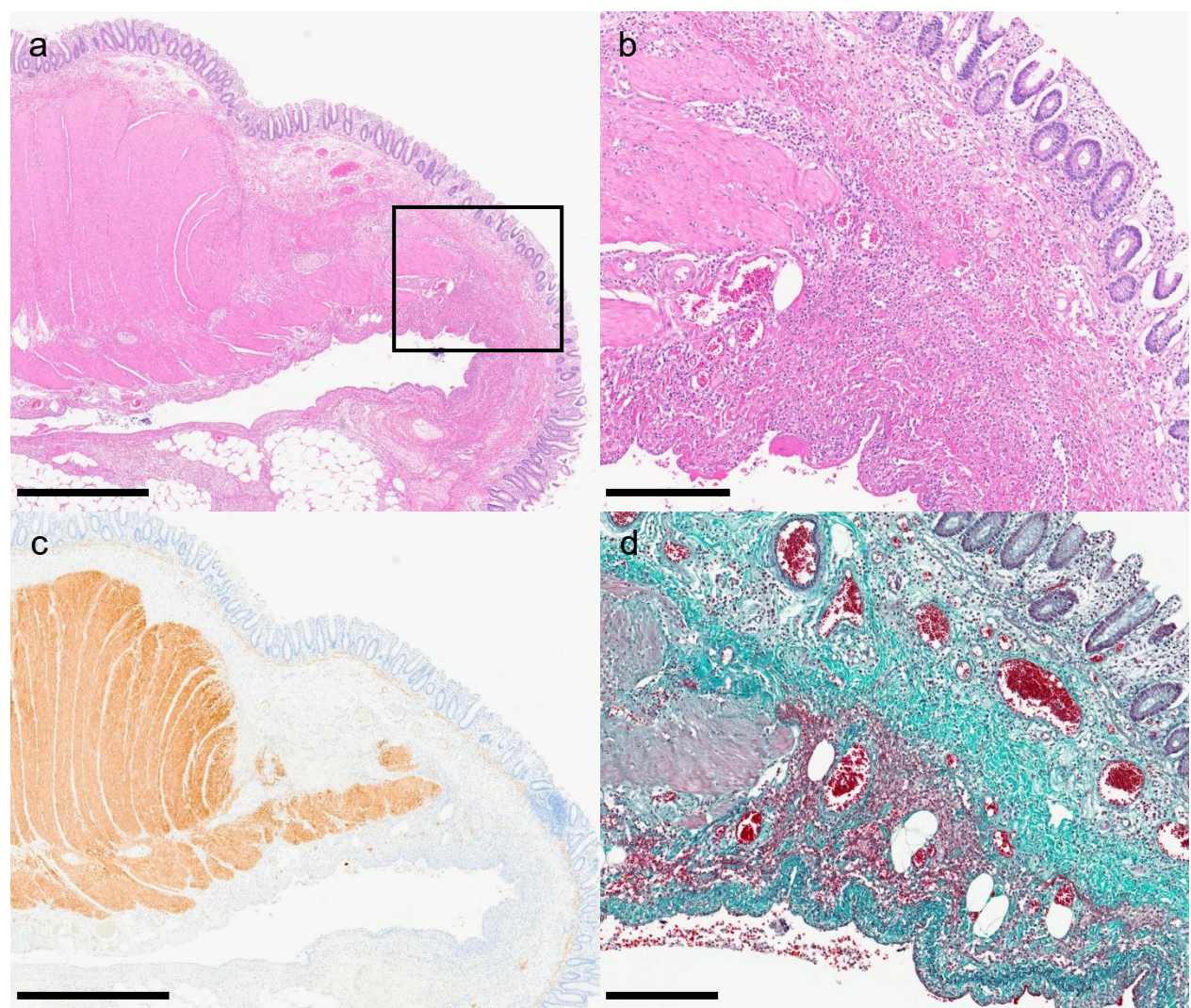


Fig. 3

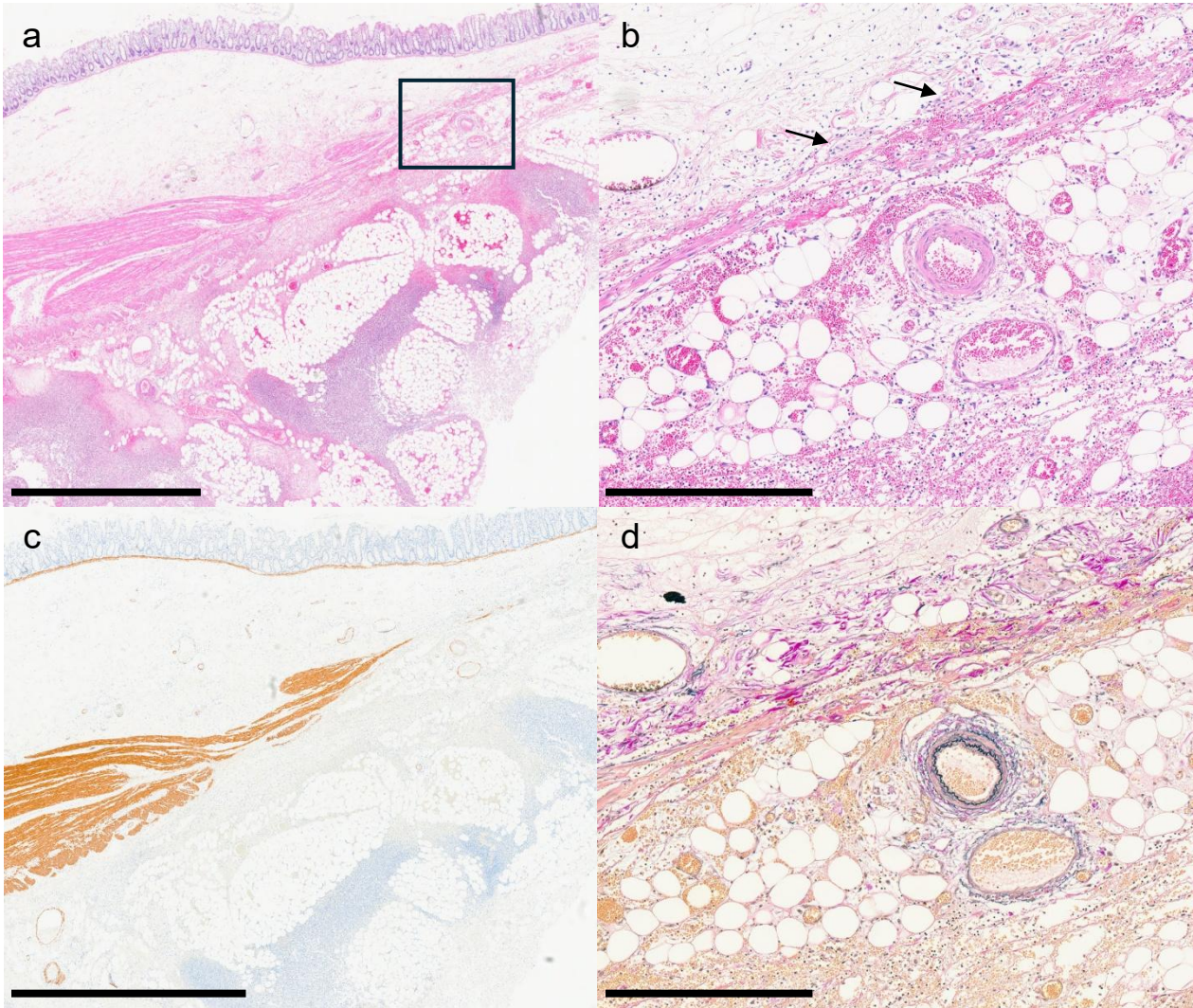


Fig. 4

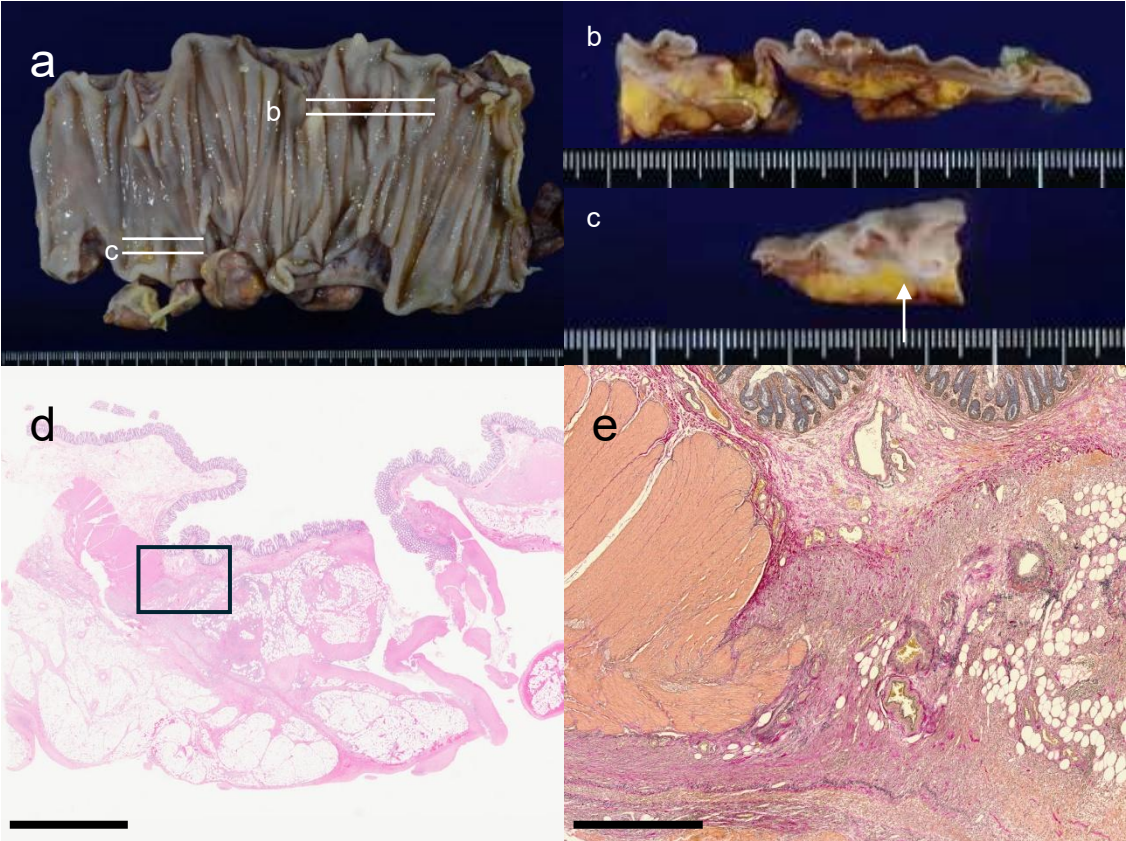


Fig. 5

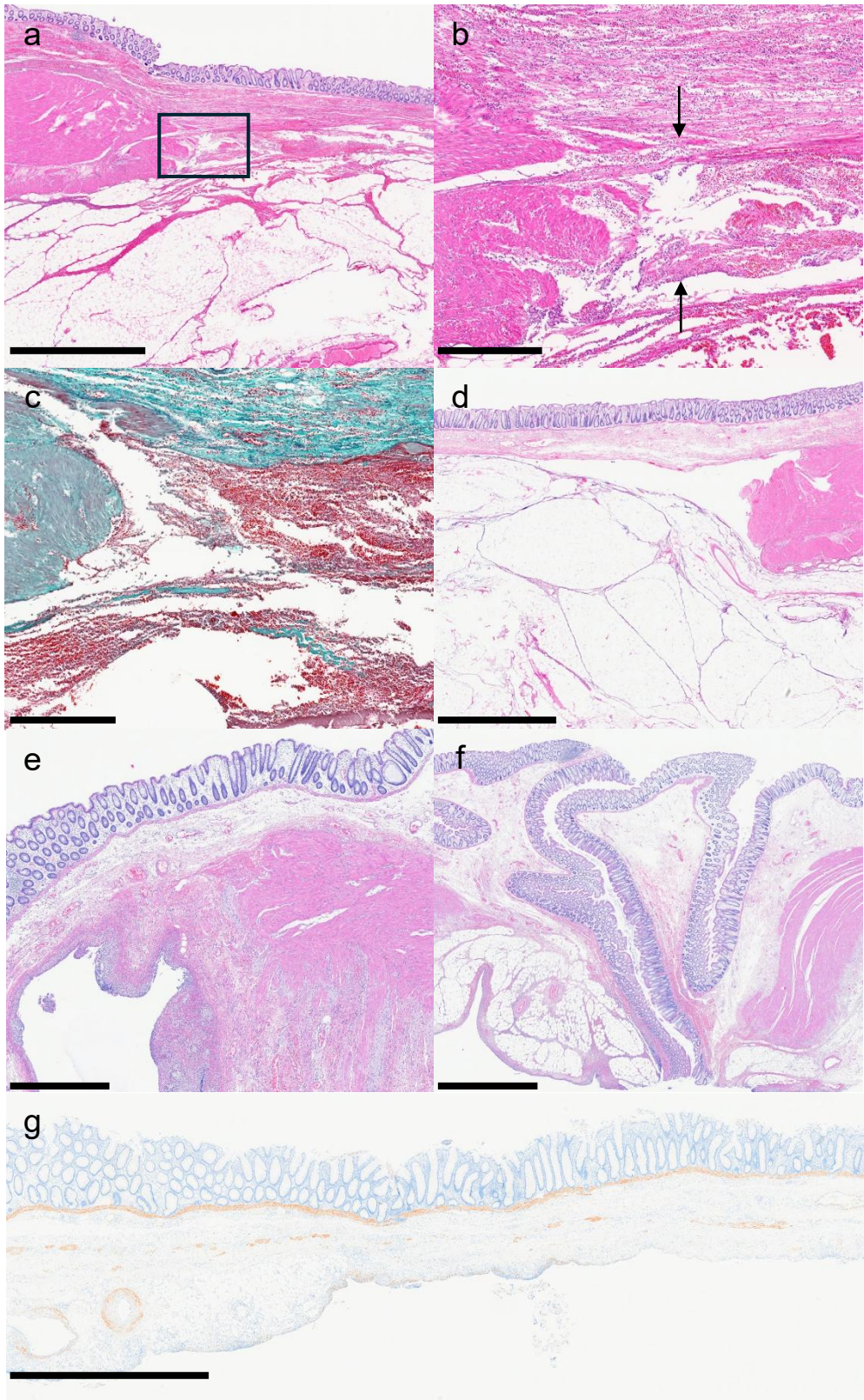


Figure legends

Fig. 1 Gross appearance of SAIM-related perforation. (a) The perforations were round-to-oval or “punched-out” (case 3). (b) On the cross-section, zonal defects of the muscularis propria (white arrows) were observed on both the oral and anal sides of the perforation (case 2)

Fig. 2 Abrupt disappearance pattern (case 2). (a, b) A sharply demarcated, abrupt loss of the muscularis propria is present, with preservation of the mucosa. The subserosa lies directly beneath the submucosa due to the loss of the muscularis propria. Diagnosis on H&E staining alone can be difficult when inflammation is superimposed. (c) IHC for desmin highlights the complete loss of the muscularis propria, with positivity in adjacent intact muscularis propria and well-preserved muscularis mucosae, facilitating confirmation of true muscle loss. (d) Elastica–Masson staining demonstrates elevation of serosal elastic fibers toward the submucosa at the site of muscularis propria loss, and allows recognition of the direct apposition between the submucosa and subserosa. a and b: H&E, c: desmin, d: Elastica–Masson. Scale bars: a and c, 1 mm; b and d, 250 μ m

Fig. 3 Tapered-off pattern (case 3). (a, b) The muscularis propria gradually thins and tapers to absence, while the mucosa remains intact. At the defect, the subserosa lies directly below the submucosa. Nerve plexuses are present at the interface of the submucosa and subserosa (b, arrows). (c, d) IHC for desmin and EVG staining demonstrate disappearance of the muscularis propria and continuity between the submucosa and subserosa. a and b: H&E, c: desmin, d: EVG. Scale bars: a and c, 2.5 mm; b and d, 500 μ m

Fig. 4 A case re-classified as SAIM (case 6). (a–c) The patient had originally been diagnosed with diverticular perforation. The perforation is observed at the proximal side (b), and a diverticulum is identified at the distal side (c). On the cross-section of the perforation site, mucosal outpouching is unclear (b), whereas mucosal outpouching is evident at the site of the diverticulum (c, arrow). (d, e) An abrupt loss of the muscularis propria is observed on both the oral and anal sides, and the subserosa lies directly beneath the submucosa. No diverticulum is identified. a–c: Macroscopic photographs, d: H&E, e: EVG. Scale bars: d, 5 mm; e, 1 mm

Fig. 5 Characteristics of non- and pseudo-SAIM cases. (a) Absence of the muscularis propria is observed; however, at higher magnification (b), spaces with fibrin deposition are present between the submucosa and subserosa (arrows), indicating discontinuity. (c) Elastica–Masson staining makes the lack of continuity more apparent. (d) There is a zonal loss of the muscularis propria and a fibrin-free space between the submucosa and subserosa caused by contraction of the ruptured muscularis propria. The intervening space is focally narrow, and the subserosa lies close to the submucosa. (e, f) A case of diverticular disease mimicking SAIM. An abrupt disappearance of the muscularis propria is observed with preserved mucosa (e); however, another section shows mucosal outpouching, indicating a diverticulum (f). If mucosal outpouching disappears due to enlargement of the perforation and inflammation, distinguishing a diverticulum from SAIM is impossible. (g) A case with intermittent defects

of the muscularis propria, interpreted as ischemic changes. a, b, and d–f: H&E, c: Elastica–Masson, g: desmin. Scale bars: a, d, f, and g, 2.5 mm; b–c, 500 μ m; e, 1 mm

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