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Pathological Diagnosis of Small Intestinal Ischemic Lesions: An Analysis of 194 Surgically Resected Cases

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Abstract

We analyzed pathomorphological appearances of surgically resected specimens in 194 cases of small intestinal ischemic lesions（number of disease types at the final diagnoses was as follows: 148 cases of strangulation, 17 of mesenteric arterial thrombosis, 3 of mesenteric venous thrombosis, 10 of idiopathic ischemic enteritis, and 16 of other causes）. Most of small intestinal ischemic lesions had almost the same pathomorphological findings, irrespective of disease type. Therefore, among the 194 cases, the disease types of 137 cases（70.6%）were finally diagnosed by referring to the descriptions in the medical record or by increasing the number of tissue sections examined. Disease types in remaining 57（29.4%）cases（35 cases of strangulation, 8 of mesenteric arterial thrombosis, 3 of mesenteric venous thrombosis, 8 of idiopathic ischemic enteritis in chronic stage, and 3 of disseminated intravascular coagulation）were diagnosed only by pathomorphological findings. In conclusion, not only pathomorphological findings but also clinical information is essential when searching for and identifying the origin and cause of small intestinal ischemic lesions.

Key words: Small intestinal ischemic lesions, Strangulation, Mesenteric arterial thrombosis, Mesenteric venous thrombosis, Idiopathic ischemic enteritis, Pathological diagnosis

Introduction

Hemorrhagic, necrotic, or ulcerating lesions of the small intestine caused mainly by circulation disturbance are referred to as small intestinal ischemic lesions and include a variety of disease types from transient ischemic enteritis with a relatively benign prognosis to extensive infarction with, currently, poor treatment outcome. Their causes vary from occlusion of the mesenteric artery or mesenteric vein, primary vascular lesion, or non-obstructive factors to strangulation.[1)-7)] Thus, pathomorphological images of small intestinal ischemic lesions vary according to the cause, degree, extent, and stage of the ischemia. Therefore, we analyzed diverse pathomorphological images of surgically resected specimens of small intestinal ischemic

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lesions (194 cases/ 194 lesions).

Materials and Methods

Patients

Among 487 patients with surgical resection of the small intestine whose histopathological data were searched in the pathology division of Fukuoka University Faculty of Medicine from January 2003 to the end of July 2016, 194 cases/ 194 lesions diagnosed preoperatively as small intestinal ischemic lesions comprised the cases to be reviewed. The macroscopic and histologic images of every lesion of the resected specimens were sufficiently compared.

This retrospective study protocol was approved by the Fukuoka University-Medical Ethics Review Board (Approval Number 16-11-05).

Specimen handling

Every resected specimen was adequately spread out, and after being immersed and fixed in formalin, the lesion (nearly the entire area or the central area) was cut parallel to the long axis of the intestinal tract in widths of about 5 mm. Each cut-section was embedded in paraffin, sliced (5-μm thickness), and then hematoxylin-eosin (HE) staining for microscopic examination. Modified Masson's trichrome staining, Berlin blue staining, Elastica van Gieson staining, and immunohistochemical staining using anti-desmin antibody were performed when necessary, and each section was examined.

Clinical items

Age, sex, clinical diagnosis, underlying disease, and lesion site were based on the descriptions in the medical record.

Staging

In accordance with Iwashita et al. [8], we classified the stage in which a histologically apparent fibrotic lesion was recognized in the lesion as the chronic stage and a stage without a histologically apparent fibrotic lesion as the acute stage.

Classification of disease type

According to the clinical information (e.g. clinical course, radiologic findings, and operation record), the cases were broadly classified into two groups, the group with known etiology of ischemia and the group with unknown etiology of ischemia. The former included a broad spectrum of conditions, which differ in onset, duration, cause of damage, and type of vessels involved (below for details). The latter was diagnosed provisionally as "idiopathic ischemic enteritis". The state of being strangulated is defined as pathological constriction or compression of blood flow. In general, mechanical causes of strangulation included different conditions such as bands, incarcerated hernia, volvulus, adhesions, and intussusception. Mesenteric arterial thrombosis was defined as a sudden reduction of intestinal blood supply due to alterations in mesenteric arterial circulation. Just like this, mesenteric venous thrombosis was defined as a sudden reduction of intestinal blood supply due to alterations in mesenteric venous circulation. If thrombi were not detectable in any cut-sections by histologic examination, we preferentially adopted preoperative diagnosis based on imaging studies (e.g. computed radiography and barium meal examination).

Length of the lesion site

The length of the lesion site along the long axis of the formalin-fixed resected intestine was used.

Classification of infarction

Tissue necrosis caused by ischemia was defined as infarction, with infarction accompanied by remarkable bleeding within the infarct area defined as hemorrhagic infarction and infarction with unremarkable bleeding defined as anemic infarction.

Subclassification of the associated ulcers

The gross morphology of each ulcer was subclassified into any one of circular, annular, or circumferential girdle-shaped. In addition, among intestinal circumferential ulcers, if the short-axis length of the ulcer site was longer than the long-axis length, it was judged as annular, and if the short-axis length was the same as or shorter than the long-axis length, it was judged as circumferential girdle-shaped. Furthermore, an ulcer with a circumferential girdle shape that was accompanied by a luminal tubular stenosis and a highly thickened intestinal wall at the lesion site was classified as a girdle-shaped scar.

Ulcer depth

The depth of the associated ulcer or ulcer scar was evaluated according to the conventional classification used for gastric ulcers. [1]

Results

Details of disease types

As shown in Table 1, among the disease types in the 194 cases, 148 (76.3%) were of strangulation, which was the largest number, followed by 17 (8.8%) of mesenteric arterial thrombosis, 16 (8.2%) of other causes, and 10 (5.2%) of idiopathic ischemic enteritis. The type with the least number of cases was mesenteric venous thrombosis with 3 (1.5%).

Strangulation

As shown in Table 2, patient ages ranged from 1 to 98 years, and the male-to-female ratio was 52:96. The disease was in the acute stage in all cases, and the ileum was the most common lesion site with 119 cases, followed by the jejunum with 21 cases and both sites with 8 cases. The length of the lesion was 2 to 160 cm, with the length in 104 cases (70.3%) 50 cm or below and that in 44 cases (29.7%) more than 50 cm.

As shown in Table 3, cord-like substance, which is usually called a band, was the most common cause of strangulation in 104 cases (70.3%), followed by incarcerated hernia in 27 cases (18.2%), volvulus in 12 cases (8.1%), and intussusception in 5 cases (3.4%). Of the 104 cases of small intestinal strangulation most commonly caused by cord-like substance, 59 cases (56.7%) had a history of laparotomy, and the remaining 45 cases (43.3%) had no history of laparotomy. The cord-like band was fibrous connective tissue mixed with a small amount of adipose tissue.

Of the 27 cases of incarcerated hernia, all were external hernias and included groin hernia (femoral hernia in 15 and internal inguinal hernia in 1 case), obturator hernia in 6 cases, and abdominal incisional hernia in 5 cases. Among the 15 cases of femoral hernia, 2 cases were of Richter's hernia in which part of the intestinal wall was incarcerated.

Among the 5 cases of intussusception, 3 cases were of ileo-cecal-type intussusception diagnosed in 1- to 3-year-old female infants and were due to hyperplasia of lymphoid tissue in the distal ileum. The remaining 2 cases were ileo-ileal-type intussusception diagnosed in a 96-year-old woman (unknown etiology) and a 5-year-old girl (with underlying disease of IgA vasculitis).

Based on the macroscopic images, 105 cases (70.9%) showed hemorrhagic infarction (Fig. 1A), 6 cases (5.7%) anemic infarction, and 31 cases (20.9%) mixed-type infarction. Further, 6 cases (4.1%) showed no clear infarct area. An associated ulcer was recognized in 48 cases (32.4%), of which circular-shaped ulceration was observed in 45 cases and annular ulcers were observed in 3 cases. There were no cases of luminal tubular stenosis.

Based on the histology, as shown in Table 4, fresh bleeding, submucosal edema, vasodilation, necrosis of the mucosa and muscularis propria, and inflammatory cell infiltration were found in all cases. Especially, submucosal edema and vasodilation were severe. Various findings were observed in the mucosa, from degeneration of intestinal villi epithelia to coagulation necrosis throughout the mucosal layer (Figs. 1B, C, D). When the ischemic injury became more severe, the structure of the intestinal villi almost disappeared and a framework of intestinal crypts alone was recognized. Then, in the muscularis propria, smooth muscle fibers swelled to varying degrees, and rupture of the muscle fascicles occurred through which, edema and bleeding were observed (Fig. 1E). Inflammatory cells were mainly neutrophils and were conspicuous around the blood vessels. In cases of severe inflammatory cell infiltration, neutrophil infiltration was also observed from the subserosal tissue into mesenteric adipose tissue. The depths of the above-mentioned associated ulcers are as follows: UL-I in 23 cases, UL-II in 15 cases, UL-III in 2 cases, and UL-IV in 8 cases. Among these cases, granulation tissue developed in the ulcer bed in 2 cases. In all cases

Table 1. Classification of small intestinal ischemic lesions

Type of lesions	No. of cases
Strangulation	148 (76.3)
Mesenteric arterial thrombosis	17 (8.8)
Mesenteric venous thrombosis	3 (1.5)
Idiopathic ischemic enteritis	10 (5.2)
Others	16 (8.2)
Total No. of cases	194

Parentheses indicate percentage.

Table 2. Clinicopathologic data of small intestinal ischemic lesions

	Strangulation *n*=148	Mesenteric arterial thrombosis *n*=17	Mesenteric venous thrombosis *n*=3	Idiopathic ischemic enteritis *n*=10		Others *n*=16
	Acute *n*=148	Acute *n*=17	Acute *n*=3	Acute *n*=2	Chronic *n*=8	Acute *n*=16
Age, years (mean)	1 ～ 98 (62.7)	49 ～ 92 (66.3)	55 ～ 88 (69.7)	19 ～ 83 (63.8)		0 ～ 86 (60.0)
Gender						
Male: female	52: 96	9: 8	1: 2	5: 5		11: 5
Site of lesion						
Jejunum	21	1	1	0	4	5
Jejunum and ileum	8	9	0	0	0	1
Ileum	119	7	2	2	4	10
Length of lesion, cm (mean)	(48.5)	(110.9)	(75.0)	(32.1)		(41.6)
≦ 25	62	0	0	0	6	9
26-50	42	0	2	2	1	3
51-75	12	1	0	0	1	1
76-100	16	3	0	0	0	0
≧ 101	16	13	1	0	0	3
Infarct						
Absent	6	0	0	0	0	0
Hemorrhagic	105	3	3	2	0	9
Anemic	6	5	0	0	0	0
Mixed type	31	9	0	0	0	7
Shape of ulcer						
Absent	100	16	3	2	0	9
Round	45	1	0	0	0	7
Annular	3	0	0	0	0	0
Girdle-like	0	0	0	0	8	0
Stenosis	0	0	0	0	8	0
Depth of ulcer *						
UL-I	23	1	0	0		5
UL-II	15	0	0	3		1
UL-III	2	0	0	5		0
UL-IV	8	0	0	0		1

n indicates number of lesions. * : The depth of the associated ulcer or ulcer scar is evaluated according to the classification of gastric ulcers.

Table 3. Subclassification of strangulation

Subtype	No. of cases
Bands	104 (70.3)
Incarcerated hernia	27 (18.2)
Volvulus	12 (8.1)
Intussusception	5 (3.4)
Total No. of cases	148

Parentheses indicate percentage.

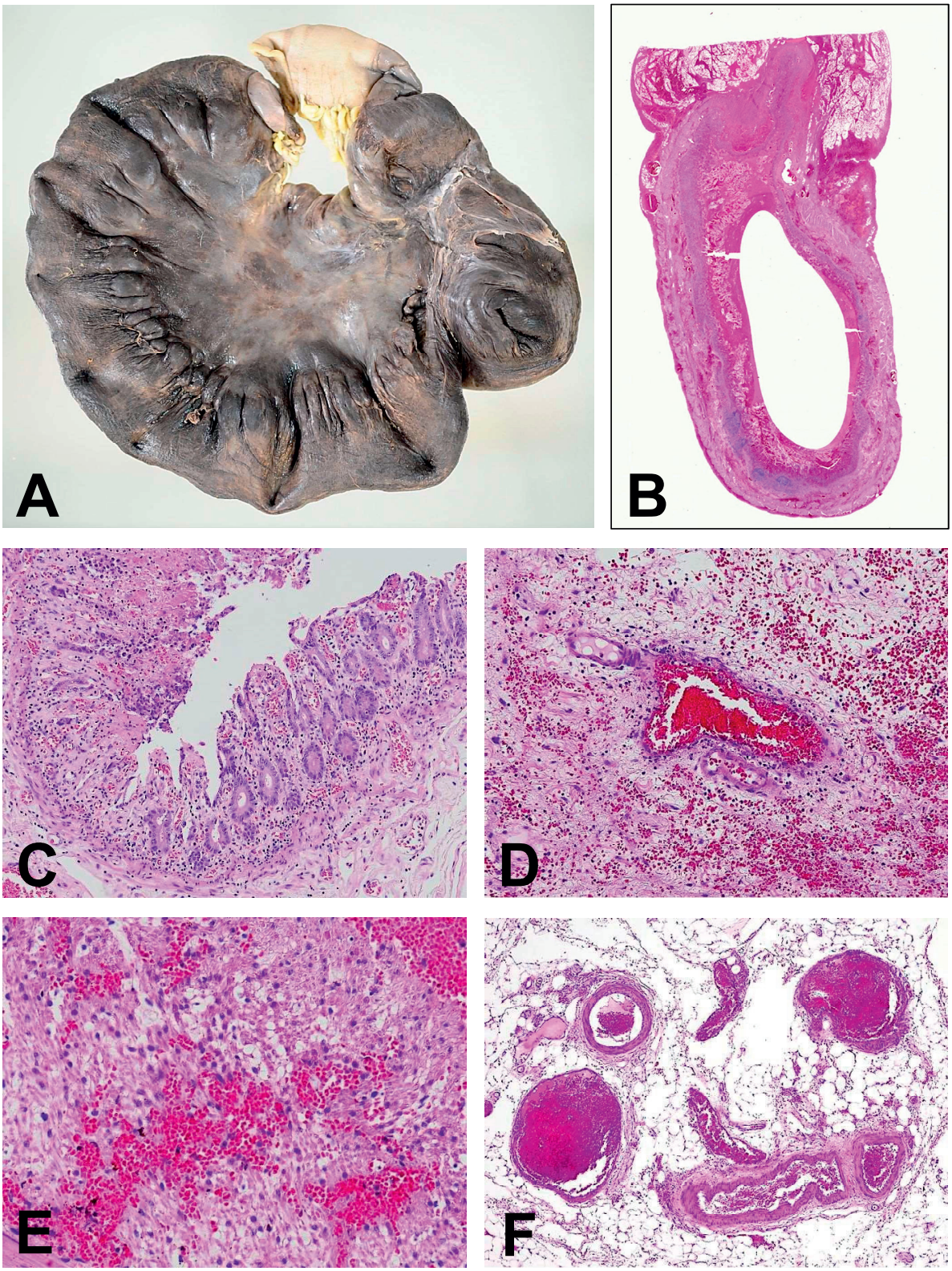

Figure 1 Pathological features of strangulation.
The serosal surface of the resected small intestine appears blue-black（A）. The whole-mount view of the small intestinal wall shows full-thickness coagulative necrosis with marked congestion and hemorrhage（B）. Loss of the epithelium lining almost the full length of the crypt is seen. Necrotic debris and fibrin deposition are also seen in the lamina propria mucosae（C）. Submucosal edema and hemorrhage are also present（D）. The muscularis propria shows hemorrhagic coagulative necrosis（E）. Vascular congestion and hyperemia are also seen in the mesenteric tissue（F）.

of perforation of UL-IV depth, all layers of the bowel wall showed necrosis and were associated with high-grade bleeding and neutrophil infiltration. Bacterial aggregates were observed in the lesions of the perforation cases, and the lesions showed signs of gangrene. Vascular dilatation associated with hyperemia and congestion was also seen in the mesenterium (Fig. 1F).

Mesenteric arterial thrombosis

As shown in Table 2, patient age ranged from 49 to 92 years, and the male-to-female ratio was 9:8. Among the 17 total cases, 15 had such underlying lifestyle diseases as cerebral infarction, arteriosclerosis obliterans, and abdominal aortic aneurysm, and 4 cases had a history of diabetes for more than 15 years. The disease in all cases was in the acute stage, and in terms of lesion site, there were 9 cases in which the lesion extended to the jejunum and ileum, the most frequent case, and 7 cases in the ileum. In 13 of these 17 cases, ischemic lesions developed not only in the small intestine but also in the colon, which is a region controlled by the mesenteric artery. Therefore, the lesion sites were long with the lesion exceeding 100 cm in length

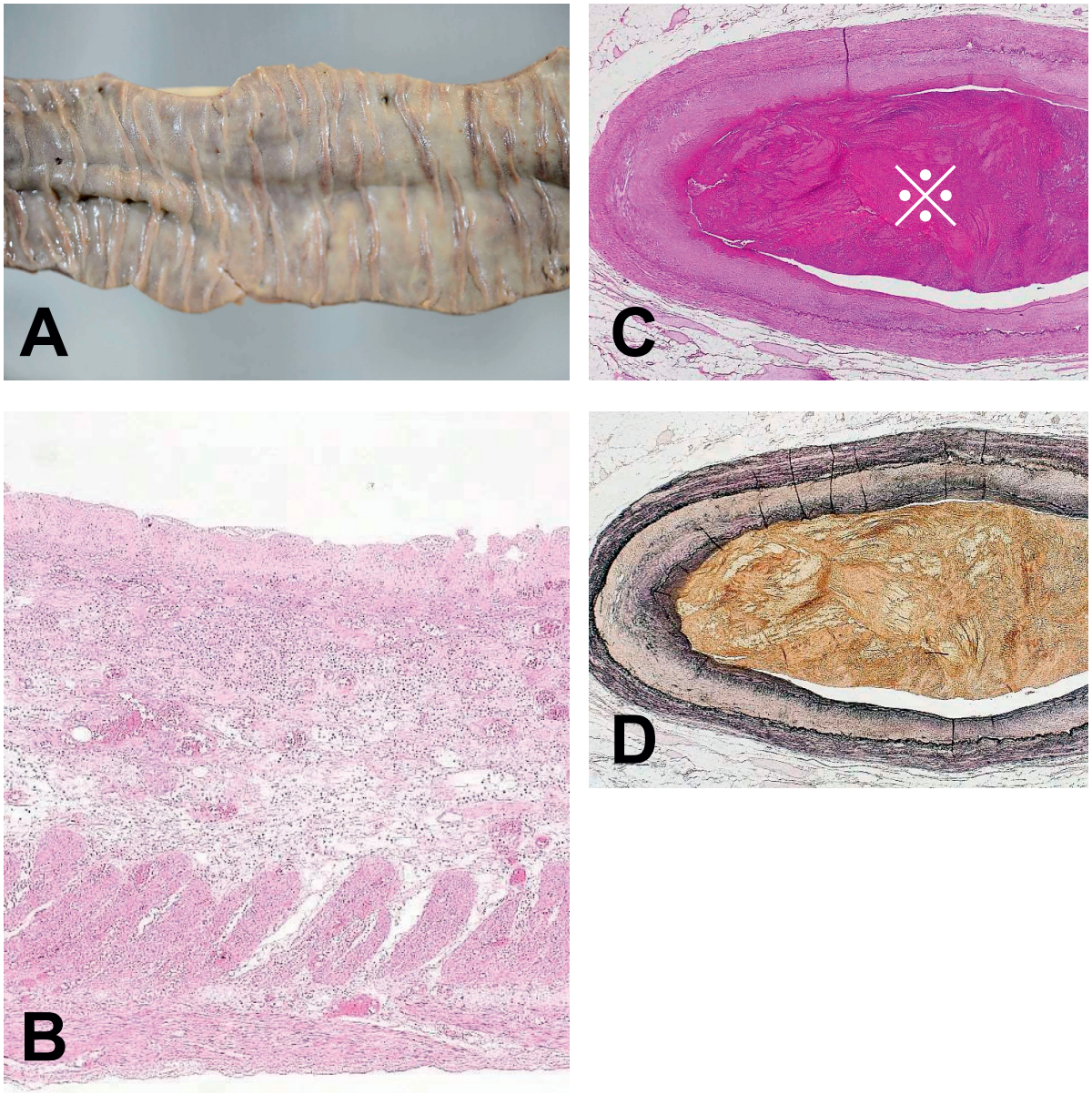

Figure 2 Pathological features of mesenteric arterial thrombosis.
The mucosal surface of the resected small intestine appears dusky blue-brown. This appearance is consistent with anemic infarct (A). The whole-mount view of the lesion shows transmural extensive coagulative necrosis. Moderate neutrophilic infiltration is present in the submucosal tissue (B). Thrombus (※) identified in the branch of superior mesenteric artery (C). The same muscular type artery stained with an Elastica van Gieson method (D).

in 13 cases (76.5%), and the longest lesion was 470 cm.

Based on the macroscopic images, 3 cases (17.6%) showed hemorrhagic infarction, 5 cases (29.4%) anemic infarction (Fig. 2A), and 9 cases (52.9%) mixed-type infarction. Only one case was associated with a shallow ulcer.

Based on the histology, as shown in Table 4, mild- to high-grade fresh bleeding, submucosal edema, vasodilation, necrosis of the muscularis propria and mucosa, and inflammatory cell infiltration were observed in all cases (Fig. 2B). Fibrosis or fibromusculosis was not recognized. Inflammatory cells were mainly neutrophils and were distributed throughout all layers of the bowel wall. When attention was paid to the vascular system, thickening of the arterial wall in the subserosal tissue was observed in 13 cases (76.5%) and was mild in every case. Thrombus was found in 8 cases (47.1%), of which 7 cases showed fresh thrombus of the mesenteric artery (Figs. 2C, D) and the remaining 1 case showed fresh thrombus of an artery in the intestinal wall along with the mesenteric artery.

Mesenteric venous thrombosis

As shown in Table 2, patient age ranged from 55 to 88 years, and the male-to-female ratio was 1:2. As an underlying disease strongly related to this disease type, polycythemia was present in only 2 cases. The disease was in the acute stage in all cases, and the lesion site was in the jejunum in 1 case and in the ileum in 2 cases. The length of lesion sites ranged from 40 to 110 cm and was 50 cm or less in 2 cases.

The macroscopic images in all of these cases showed dark red hemorrhagic infarction without apparent associated ulcers (Fig. 3A).

Based on the histology, as shown in Table 4, fresh bleeding, submucosal edema, vasodilation, necrosis of the muscularis propria and mucosa, and inflammatory cell

Table 4. Histopathologic findings of small intestinal ischemic lesions

	Strangulation *n*=148	Mesenteric arterial thrombosis *n*=17	Mesenteric venous thrombosis *n*=3	Idiopathic ischemic enteritis *n*=10		Others *n*=16
	Acute *n*=148	Acute *n*=17	Acute *n*=3	Acute *n*=2	Chronic *n*=8	Acute *n*=16
Hemorrhage						
Absent: mild: marked	0: 21: 127	0: 7: 10	0: 0: 3	0: 0: 2	8: 0: 0	0: 3: 13
Mucosal necrosis						
Absent: present	6: 142	0: 17	0: 3	0: 2	8: 0	0: 16
Muscular necrosis						
Absent: present	6: 142	0: 17	0: 3	0: 2	8: 0	1: 15
Fibromusculosis						
Absent: present	148: 0	17: 0	3: 0	2: 0	0: 8	16: 0
Inflammatory infiltrate						
Absent: mild: marked	6: 39: 103	0: 2: 15	0: 0: 3	0: 2: 0	0: 8: 0	0: 1: 15
Edema of submucosa						
Absent: present	0: 148	0: 17	0: 3	0: 2	2: 6	0: 16
Dilatation of blood vessels						
Absent: present	0: 148	0: 17	0: 3	0: 2	2: 6	0: 16
Granulation tissue						
Absent: present	146: 2	17: 0	3: 0	2: 0	0: 8	16: 0
Siderophages*						
Absent: present	148: 0	17: 0	3: 0	2: 0	4: 4	16: 0
Thickening of arterial wall						
Absent: present	140: 8	4: 13	3: 0	2: 0	4: 4	16: 0
Thrombosis of artery						
Absent: present	148: 0	9: 8	3: 0	2: 0	6: 2	16: 0
Thrombosis of vein						
Absent: present	148: 0	16: 1	0: 3	2: 0	8: 0	16: 0

n indicates number of lesions. *: Siderophages are defined as hemosiderin-laden histiocytes.

infiltration were observed in all cases, and especially, the amount of fresh bleeding was significant (Figs. 3B, C, D). Further, siderophages were not recognized. Inflammatory cells were mainly neutrophils. Apparent fresh thrombus as a diagnostic criterion was detectable in the mesenteric veins of all 3 cases.

Idiopathic ischemic enteritis

As shown in Table 2, patient age ranged from 19 to 83 years, and the male-to-female ratio was 5:5. The disease

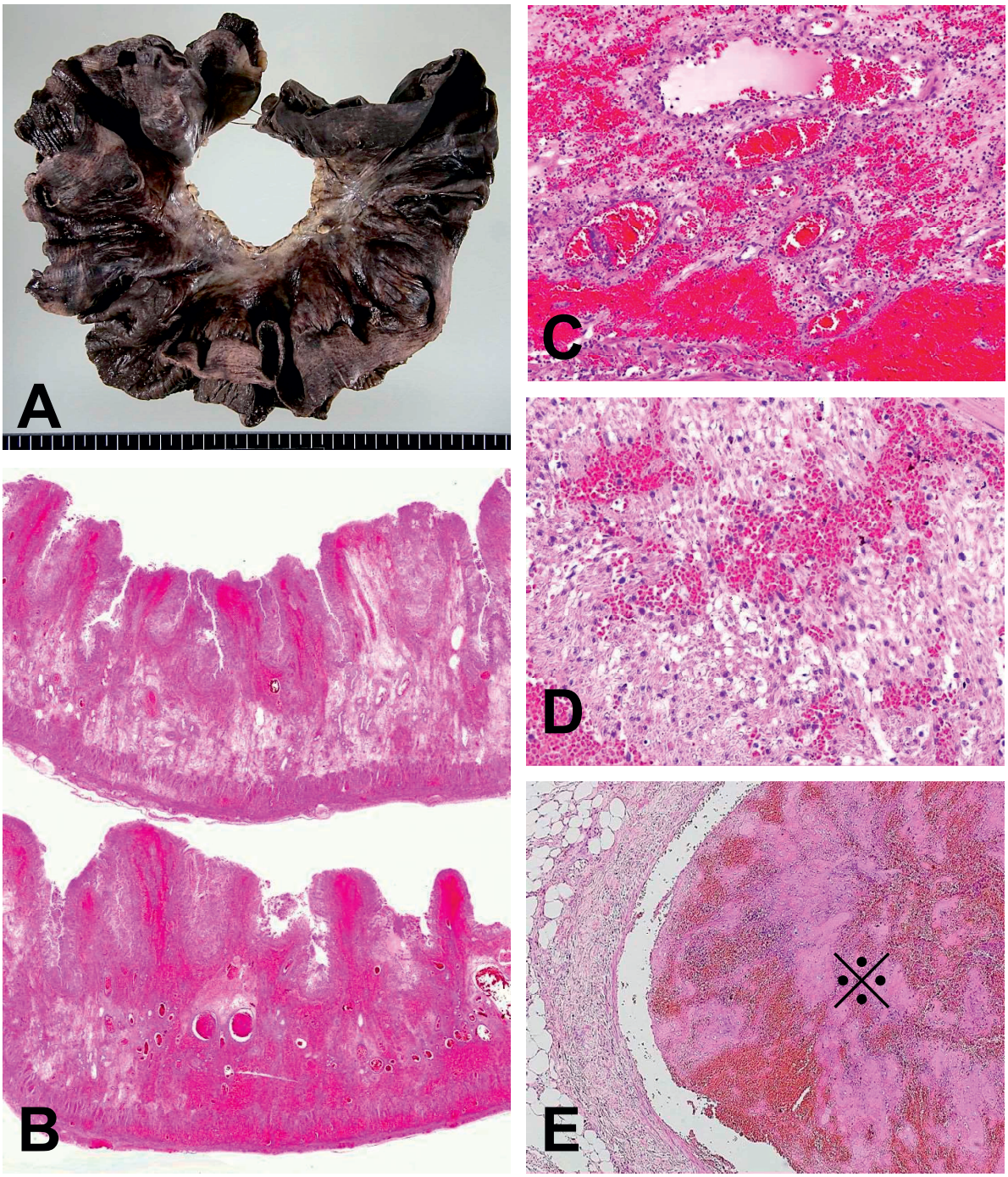

Figure 3 Pathological features of mesenteric venous thrombosis.
The mucosal surface of the resected small intestine appears marked blue-black. This appearance is consistent with hemorrhagic infarct (A). The whole-mount view of the lesion shows full-thickness hemorrhagic coagulative necrosis (B). Submucosal hemorrhage and congestion are also seen (C). The muscularis propria shows hemorrhagic coagulative necrosis (D). Thrombosed vein (※) is detected in the major branch of mesenteric vein (E).

was in the acute stage in 2 cases and in the chronic stage in 8 cases. The lesion site was in the ileum in 6 cases and the jejunum in 4 cases. The length of lesion site was 35 to 45 cm in acute-stage cases and 4.5 to 65 cm in the chronic-stage cases. The average length of the lesions in 7 of the chronic-stage cases was 13.3 cm except for 65 cm in the longest case, and it was shorter than that in the acute-stage cases.

Based on the macroscopic images, 2 acute-stage cases showed hemorrhagic infarction, but no associated ulcer was found. In contrast, all 8 of the chronic-stage cases showed luminal tubular stenosis associated with a circumferential girdle-like ulcer（Figs. 4A, B）. A large proportion of the ulcer was scarred and showed a granular

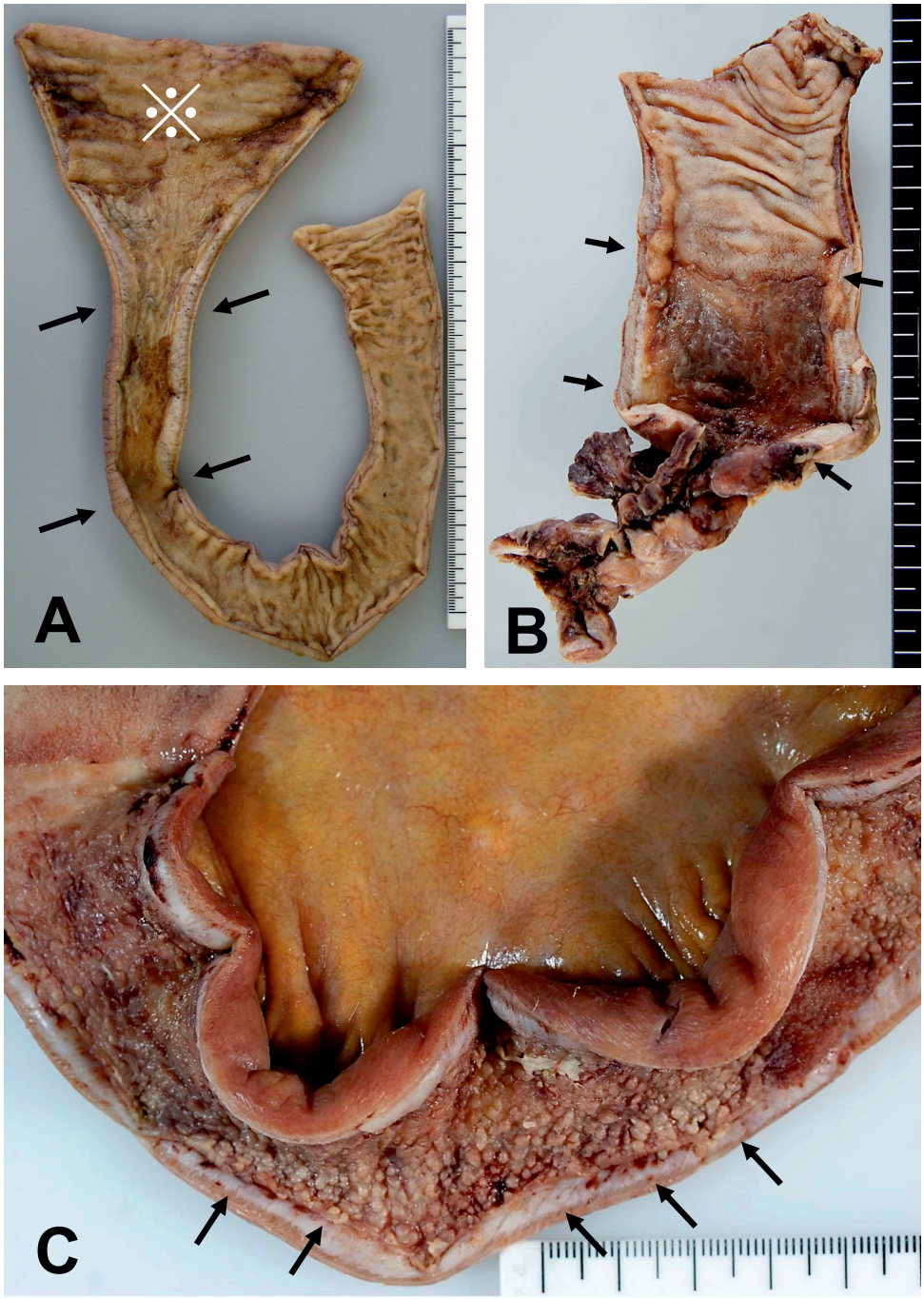

Figure 4 Macroscopic features of idiopathic ischemic enteritis in chronic stage.
The resected small intestine shows luminal tubular narrowing with girdle-like ulcer（arrows）. Note the thickening of the intestinal wall with diffuse and marked submucosal fibrosis（A, B, C）. Funnel-shaped dilated lumen（※）is also seen in the proximal side（A）. The mucosal surface of the tubular narrowing area appears coarsely granular（C）.

surface with prominently thickened intestinal wall (Fig. 4C). Additionally, the non-affected intestinal tract proximal to the lesion was significantly dilated, and the border between the narrowed area and the non-affected area was funnel-shaped (Fig. 4A).

Based on the histology, as shown in Table 4, there was no great difference between the 2 acute-stage cases and the other disease types, but inflammatory cell infiltration was relatively mild. Meanwhile, in the 8 chronic-stage cases, no images showed necrosis, and the depths of the associated

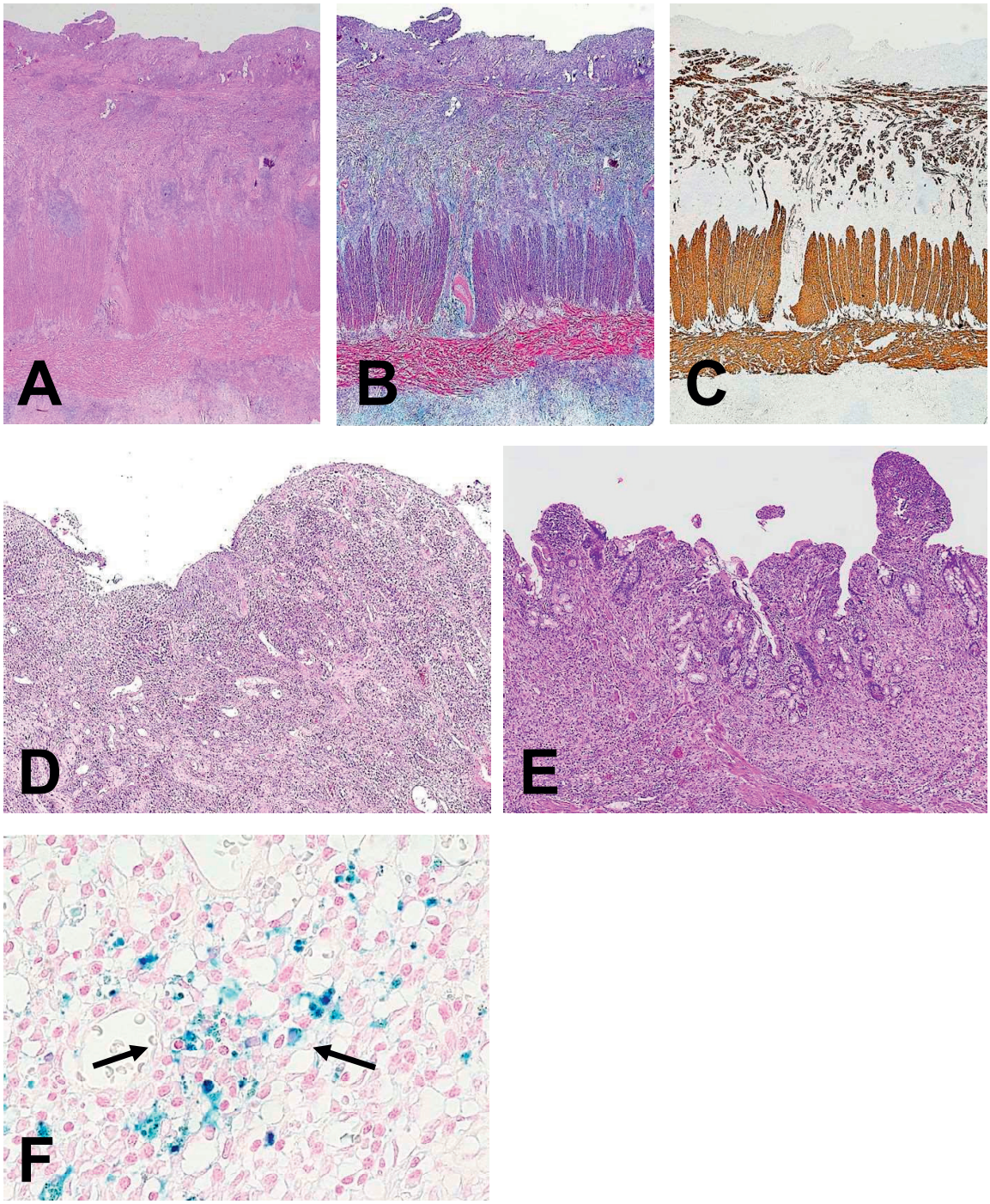

Figure 5 Microscopic features of idiopathic ischemic enteritis in chronic stage.
Low-power view of the tubular narrowing area shows extensive and marked fibrosis and/or fibromusculosis in the submucosal tissue (A). Modified Masson's trichrome stain and immunohistochemical stain for desmin, highlighting submucosal fibrosis and/or fibromusculosis (B, C). Inflammatory granulation tissue and regenerative changes are also present in the rough-surfaced mucosa (D, E). Note hemosiderin-laden histiocytes (arrows) in the submucosal tissue (F).

ulcer scars were UL-IIs in 3 cases and UL-IIIs in 5 cases, which indicated deeper ulcer scars. The surface layer of the lesion site was composed of vascular-rich granulation tissue or immature or mature regenerating epithelium, and the lesion site corresponded to the granular changes previously mentioned (Figs. 5 A-E). Also, a moderate to higher level of fibrosis or fibromusculosis was observed diffusely in the submucosal tissue (Figs. 5 B, C). Inflammatory cells were mainly lymphocytes and plasma cells. Siderophages were found in 4 cases mainly in the submucosal tissue. Thickening of the arterial wall in the subserosal tissue was found in 4 cases (Fig. 5 F). Organized thrombus accompanied by arterial recanalization was observed in 2 cases.

Others

Other than strangulation or thrombosis, the causes of small intestinal ischemia included disseminated intravascular coagulation (3 cases), neonatal necrotizing enterocolitis (1 case), increased intraluminal pressure due to fecaliths filling the lumen (4 cases), congenital jejunoileal atresia (7 cases), and blunt abdominal trauma (1 case). The lesion sites were located in the jejunum in 5 cases, the ileum in 10 cases, and the jejunoileum in 1 case, and the lengths ranged from 20 to 230 cm. The lesion length was longest in the case of disseminated intravascular coagulation in which the cecum was resected together with the ascending colon.

Based on the macroscopic images, as shown in Table 2, all cases showed hemorrhagic infarction, and 4 cases were accompanied by a round ulcer. In the case of neonatal necrotizing enterocolitis, the thinned intestinal wall was perforated.

As shown in Table 4, in terms of the histology there was no great difference between these cases and the other disease types. In the cases of disseminated intravascular coagulation, fibrin thrombi were observed mainly in the capillaries of the lamina propria mucosae.

Discussion

The ischemic small intestinal lesion is essentially a circulation disturbance, referred to as ischemia, and its origin and causes are wide ranging.[2)-7)] Therefore, clinical presentations and pathomorphological images vary. This suggests that pathological diagnosis of the relevant lesion as an ischemic lesion is easy, but clarifying the origin and cause only from pathomorphological findings is quite difficult.[8)-9)] Actually, among the 194 cases targeted in this study, there were a few (29.4%) in which the diagnosis of disease type was achieved only by pathomorphological findings as follows: small intestinal ischemia due to strangulation in 35 cases, mesenteric artery thrombosis in 8 cases, mesenteric vein thrombosis in 3 cases, disseminated intravascular coagulation in 3 cases, and idiopathic ischemic enteritis (chronic stage) in 8 cases, i.e., 57 cases in total . Most of acute ischemic small intestinal lesions had almost the same pathomorphological findings, irrespective of disease type. Therefore, the disease types of the remaining 137 cases (70.6%) were finally diagnosed by referring to the descriptions in the medical record or by increasing the number of tissue sections examined.

In this study, we used surgically resected specimens diagnosed in a single facility as small intestinal ischemic lesions and re-examined the pathomorphological images, from which the following results were obtained.

① Ischemic lesions caused by intestinal strangulation were all in the acute stage, and submucosal edema and vasodilation were remarkable.

② Ischemic lesions caused by mesenteric artery thrombosis were all in the acute stage, and the lengths of the diseased bowel were usually long, with all cases resulting in necrosis through all layers of the bowel wall. The morphology of infarction included hemorrhagic, anemic, and mixed-type infarctions. Furthermore, these ischemic lesions were associated with thickening of the arterial wall in the subserosal tissue.

③ Ischemic lesions caused by mesenteric vein thrombosis were all in the acute stage, and the images in all cases showed hemorrhagic infarction through all layers of the bowel wall with remarkable bleeding and vasodilation.

④ Among the cases of idiopathic ischemic enteritis, those in the chronic stage showed luminal tubular stenosis associated with circumferential girdle-like ulcer. The ulcer depths were Ul-IIs to UI-IIIs, and the formation of both granulation tissue and fibromusculosis was conspicuous.[10)] These results were thought to be useful and influential clues when examining surgically resected specimens pathologically. In addition, there is no clear agreement on the range or the total number of excised sections of the surgically resected samples of small intestinal ischemic lesions that should be prepared, but sections of the surgically resected

diseased bowel, the associated ulcer area, the mesenteric tissue, and of the non-affected border region are considered to be essential for the examination. [11)]

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