A Case of Submucosal Hematoma of The Esophagus and Stomach, Possibly Caused by Fish Bone Ingestion

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Submucosal hematoma of the esophagus is a rare disorder. We encountered a 70-year-old woman with an extremely large submucosal hematoma of the esophagus that extended from the cervical esophagus to the gastric angle. It was likely that her hematoma was caused by accidental ingestion of a fish bone, and her condition improved with conservative treatment alone. In this article, we summarize the reported cases of submucosal hematoma of the esophagus in Japan, and discuss the case with a review of the literature.

Key words: esophageal submucosal hematoma, foreign body, fish bone

INTRODUCTION

Submucosal hematoma of the esophagus is a rare disorder and is classified into idiopathic and traumatic types. The former is thought to be associated with a high intraluminal pressure caused by nausea, emesis and/or blood coagulation abnormalities, while the latter is caused by a direct wound in the esophagus by foreign objects, or other trauma, such as external injuries, cardiac massage, endoscopic injection sclerotherapy for esophageal varices. We encountered a patient with submucosal hematoma of the esophagus that appears to have been caused by a fish bone. This was thought to be a very rare case in which the hematoma extended broadly from the cervical esophagus to the gastric angle. In this article, the case is presented with endoscopic and computed topography (CT) images according to the time course from onset to cure, and is discussed based on the reported cases of submucosal hematoma of the esophagus in Japan.

CASE REPORT

Patient: 70-year-old woman

Chief complaint: Hematemesis

Onset and Course: A yellowtail bone became lodged in the throat of the patient while she was eating dinner. Upon drinking large amounts of water to flush the bone from her throat, she felt an unpleasant sensation move to her chest. She subsequently vomited a small amount of blood and was transported to the emergency room of our hospital.

Past history: The patient had hypertension and arteriosclerosis from about the age of 60, for which she was being treated with medication by a local doctor.

Family history: Noncontributory

Personal history: Alcohol (–), Tobacco (–),

Medications: Nifedipine (20 mg), 1 tablet twice a day; aspirin (81 mg), 1 tablet once a day

Physical examination: Height, 147 cm; weight, 63 kg; blood pressure, 140/90 mmHg; pulse, 86 beats/min (regular); body temperature, 36.4°C; SpO_2, 97% (room air). Conjunctiva was not icteric or pale. Heart sounds were regular without murmur and breath sounds were clear. Abdomen was flat and soft and bowel sounds were normokinetic. No tenderness, rebound tenderness or guarding was observed in the abdomen. She had no pretibial pitting edema.

The results of tests in admission showed almost no abnormalities, with no anemia and no findings of inflammation (Table 1). On thoracoabdominal CT scan, no fish bone but conspicuous thickening of the esophageal wall was observed from the cervical to the lower esophagus. Raised shapes were observed to protrude on the esophageal lumen side, which seemed to narrow the lumen. With regard to the stomach, there was also thickening in the submucosal layer with low CT density from the cardiac to the body regions of the stomach (Fig. 1). On upper gastrointestinal endoscopic examination, there was a point of submucosal bleeding in the posterior wall of the middle pharynx that might have been caused by the penetration of the fish bone, and a submucosal hematoma about half of the circumference of the esophagus was seen mainly in the left wall that continued from the cervical esophagus to the esophagogastric junction with blood seeping from some portions. The fish bone itself could not be confirmed by examination (Fig. 2). The patient was diagnosed with bleeding from a submucosal hematoma of the esophagus that occurred together with ingestion of food, and the lesion was sprayed with 10,000 U of thrombin. The course was observed with conservative treatment based on intravenous drip management with nothing by mouth except for drugs; aspirin was discontinued after the informed consent on the risk of bleeding from the lesion comparing with that of possible progression of arteriosclerosis, and 10,000 U of thrombin, 4 times a day for 3 days, and 20 ml of

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Table 1 Laboratory Data on Admission.

WBC	7,800/μl	BUN	15 mg/dl
Hb	13.0 g/dl	Creatinine 0.7 mg/dl	
Platelets	18.8×10^4/μl		
		Na	140 mEq/l
		K	3.3 mEq/l
Total protein	7.2 g/dl	Cl	102 mEq/l
Total bilirubin	0.7 mg/dl		
AST	21 U/l	CRP	< 0.09 mg/dl
ALT	13 U/l		
LDH	297 U/l	PT	109%
ALP	309 U/l	APTT	24 s
γ-GTP	10 U/l		

AST: aspartate aminotransferase; ALT: alanine aminotransferase;
LDH: lactate dehydrogenase; ALP: alkaline phosphatase;
γ-GTP: γ-glutamyl transpeptidase; BUN: blood urea nitrogen;
PT: prothrombin time; APTT: activated partial thromboplastin time.

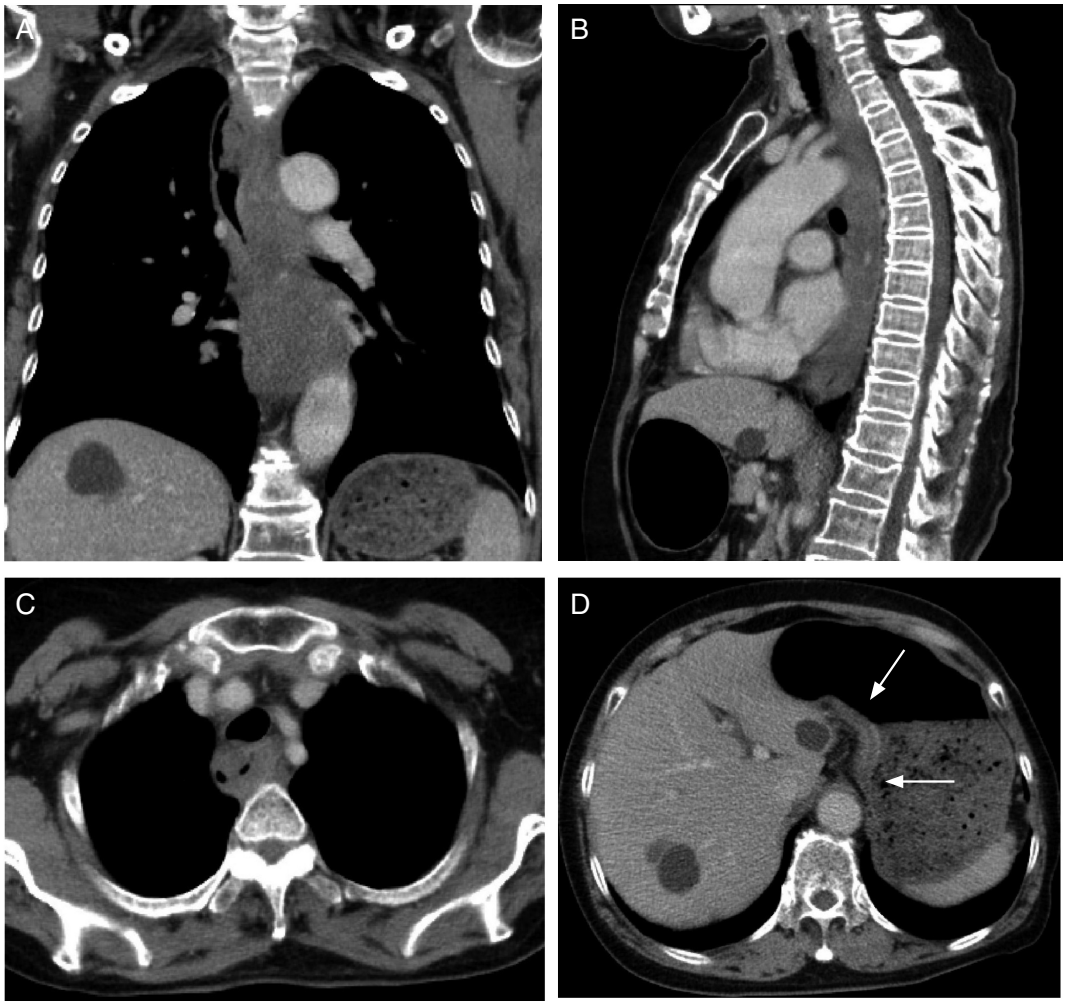


Fig. 1 Initial thoracoabdominal CT findings.
No fish bone was found. (A, B, C) Conspicuous thickening of the esophageal wall is seen continuing from the cervical esophagus to the lower esophagus. Raised shapes are seen protruding on the esophageal lumen side, and there is narrowing of the lumen. (D) Wall thickening that shows low density is seen in the submucosa from the cardiac to the body regions of the stomach (arrows).

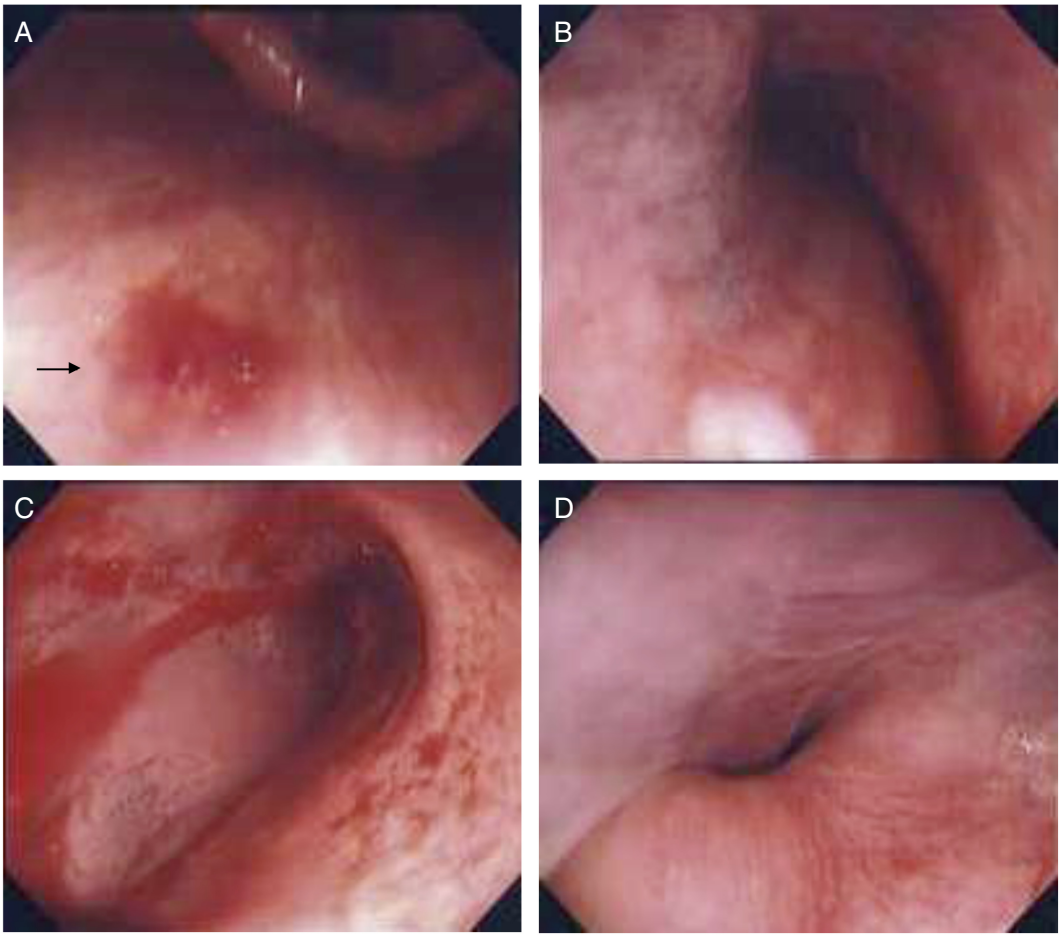


Fig. 2 Initial upper gastrointestinal endoscopy findings.
(A) Point of submucosal bleeding that might have been caused by a fish bone piercing the posterior wall of the middle pharynx (arrow). (B, C, D) A submucosal hematoma about half of the circumference of the esophagus, mainly in the left, wall continued from the cervical esophagus to the esophagogastric junction, with blood seeping from some portions.

sodium alginate, 4 times daily, were used. Drip infusion containing carbazochrome sodium sulfonate 100 mg/day, tranexamic acid 1,000 mg/day, cimetidine 800 mg/day and cefmetazole 2 g/day was administered. The patient had a fever of 38°C on the day 3 in hospital, but this resolved spontaneously on the following day. She presented with melena for the first several days, but anemia was mild and no blood transfusions were given. On upper gastrointestinal endoscopic examination on the day 6 in hospital, the hematoma only remained in the lower esophagus, however, broad submucosal dissection with partial ulceration and a pocket-like structure with blind end on the anal side was observed. In addition, there was a change to a dark purple color with swelling, flare, and erosion that suggested submucosal bleeding from the gastric angle through the cardia and gastric fundus (Fig. 3). The thickening of the esophageal wall improved on a thoracoabdominal CT scan on day 9 in hospital, and the lumen had enlarged. Bilateral pleural effusion was seen, but there was no accumulation of air or fluid in the mediastinum (Fig. 4). On esophagography on the same day, passage was good with no leakage, and ulceration only remained in the left wall (Fig. 5). The pharyngeal and thoracic pain disappeared from the day 11. Upper gastrointestinal endoscopy examination on day 12 confirmed that the hematoma was no longer present and thin ulcers after submucosal dissection in the hematoma lesion remained, showing a tendency for re-epithelialization (Fig. 6). She began eating on the same day. There was no marked change in course after she started eating, and she was discharged from the hospital on day 16. Scarring of the ulcerated area and disappearance of the color change in the gastric body were subsequently confirmed by upper gastrointestinal endoscopy examinations during outpatient visits. She returned to the local doctor and started the medications including low-dose aspirin, and there have since been no recurrences.

DISCUSSION

Submucosal hematoma of the esophagus is a rare disorder and is considered to be a form of esophageal injury. According to Shima *et al.* [1] and Furukawa *et al.*, [2] esophageal injury may be divided broadly into mechanical and chemical injuries. Mechanical injury is further classified into traumatic and spontaneous types; the former is caused by accidental ingestion of foreign objects or medical procedures such as endoscopic examination and procedures, intubation of nasogastric tube and dilatation of constrictions; the latter accompanies increased intraluminal pressure

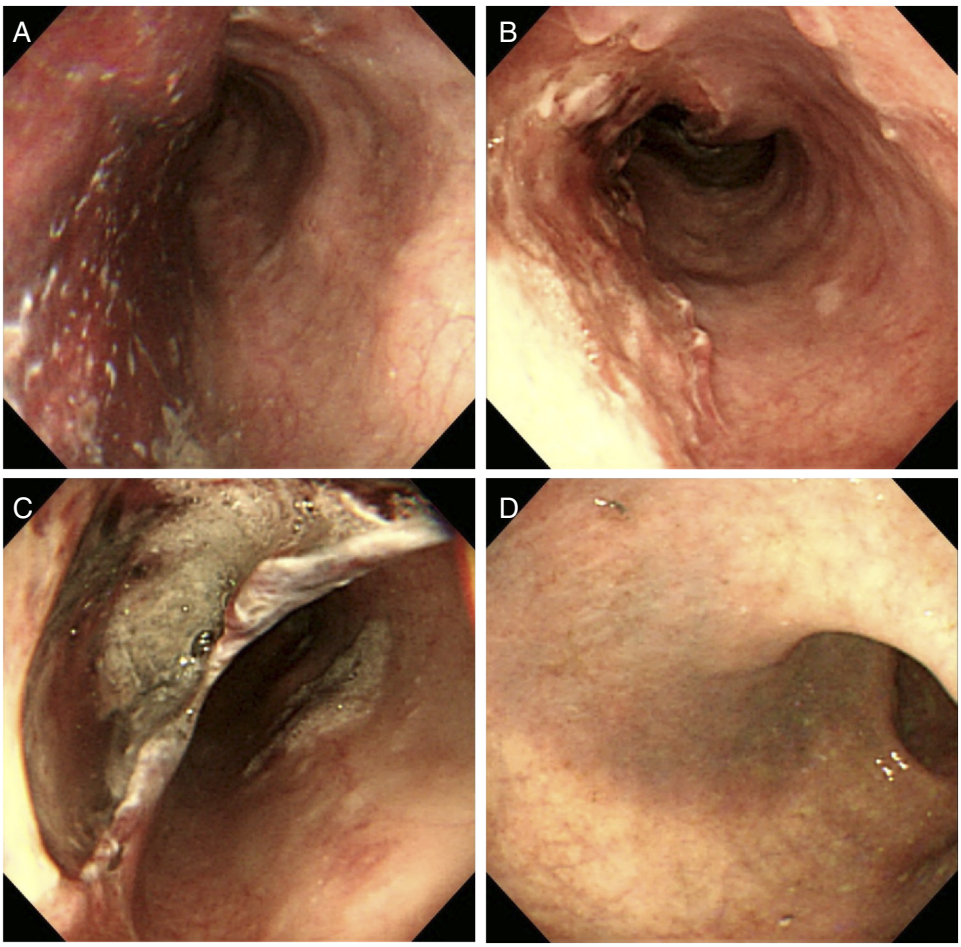
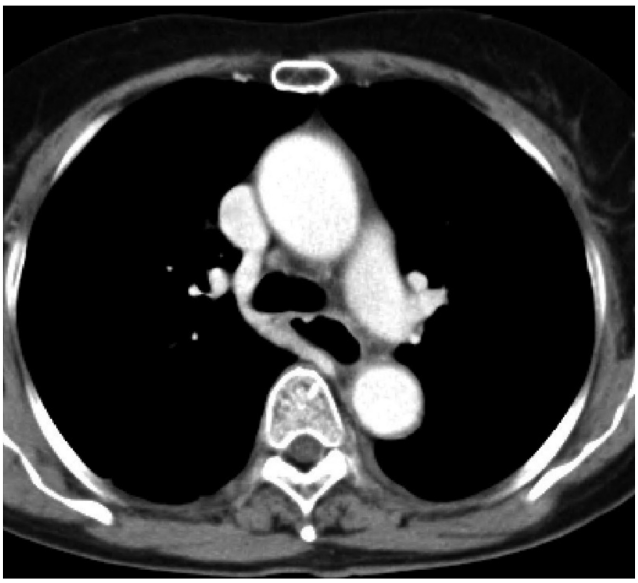


Fig. 3 Upper gastrointestinal endoscopy findings on day 6 of hospitalization.
(A) Hematoma remaining in the lower esophagus. (B) Ulceration is seen behind submucosal dissection of the esophagus in the left wall 25 to 35 cm from the incisors. (C) A pocket is formed in the submucosa in the bottom portion. (D) A change to a dark purple color with swelling, flare, and erosion is seen that suggests submucosal bleeding from the gastric angle through the cardia and gastric fundus.



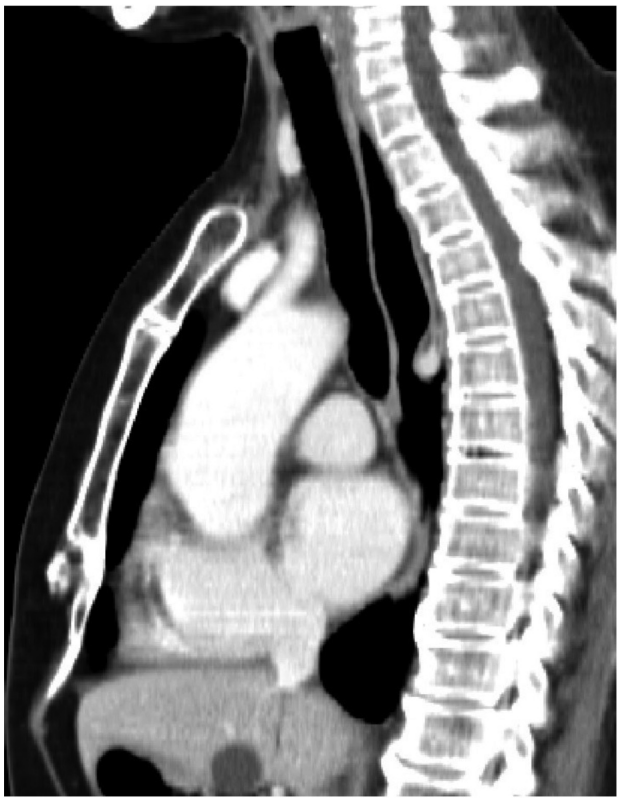


Fig. 4 Thoracoabdominal CT findings on day 9 of hospitalization.
There is improvement in the thickening of the esophageal wall and expansion of the lumen.

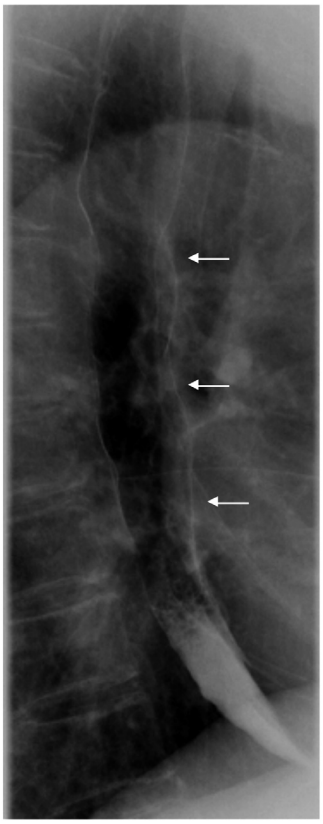


Fig. 5 Esophagography findings on day 9 of hospitalization.
Ulceration mainly of the left wall is seen from the esophagus in the central to lower chest (arrows). There is no leakage, and good passage.

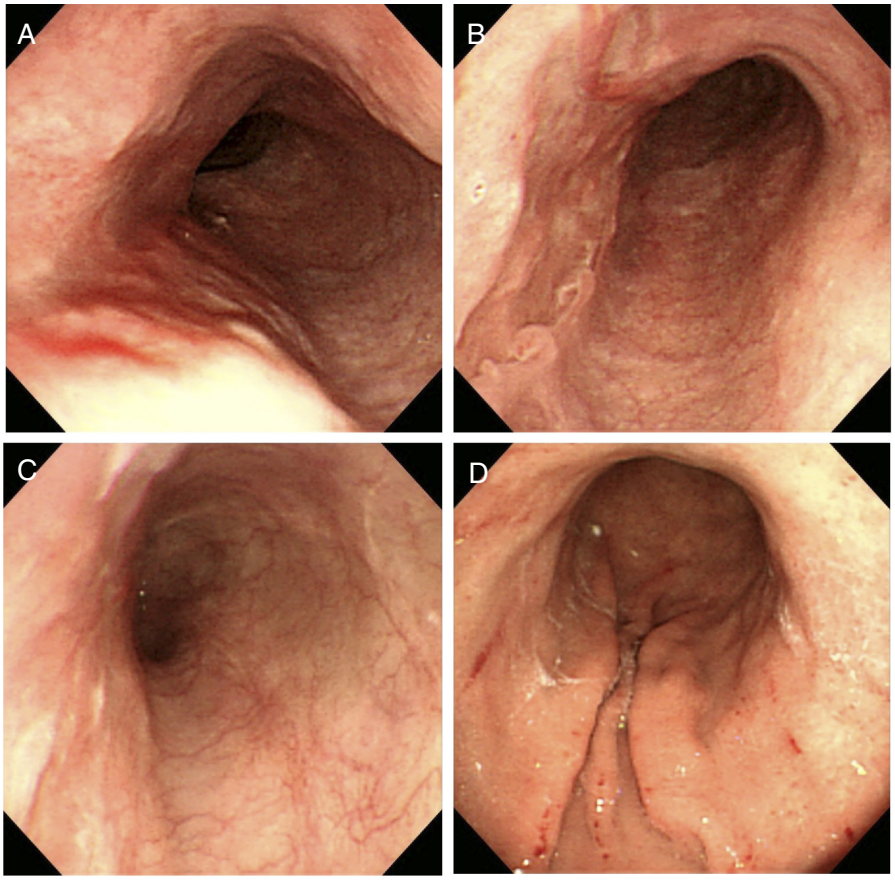


Fig. 6 Upper gastrointestinal endoscopy findings on day 12 of hospitalization.
(A, B, C) Esophageal hematoma is no longer seen. Formation of shallow ulcers behind the hematoma mucosal dissection mainly in the left wall, and a tendency for epithelialization of the ulcer surface, is seen 18 to 35 cm from the incisors. (D) The area of dark coloration in the gastric mucosa has also decreased.

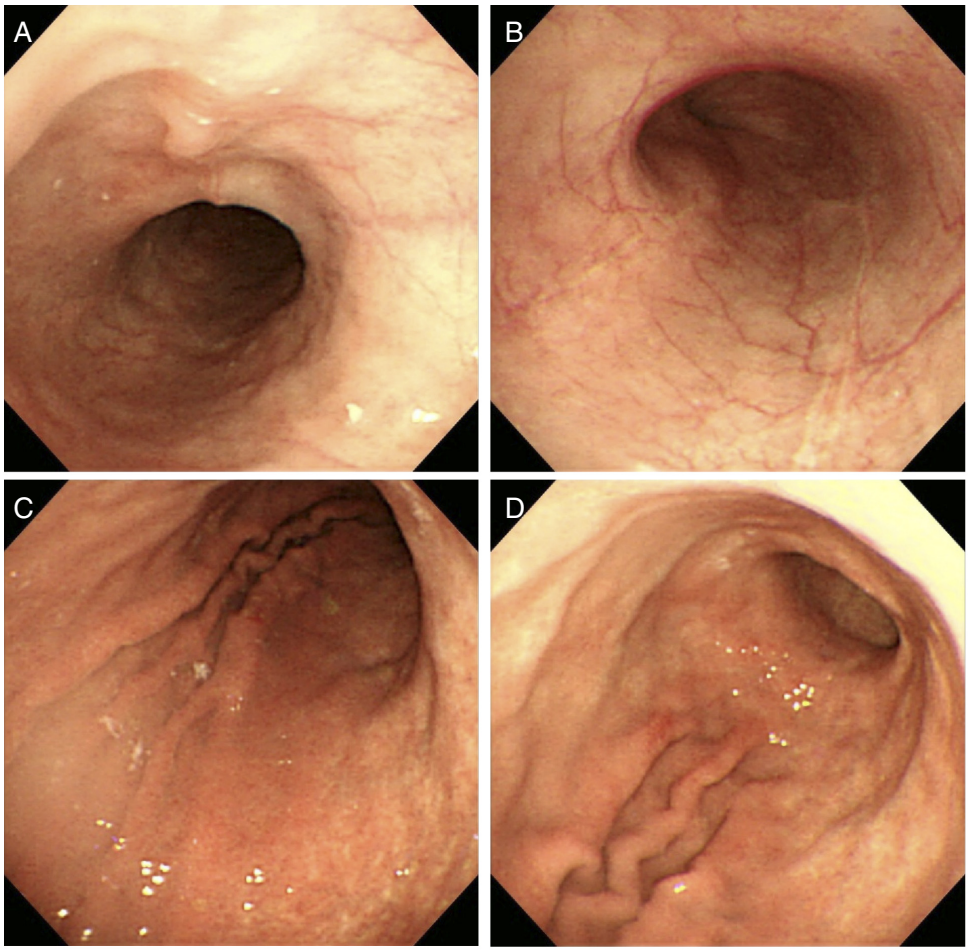


Fig. 7 Upper gastrointestinal endoscopy findings on day 45 after admission.
(A, B, C) Scarring is seen in the area where the esophageal ulcers formed. (D) The color change, flare, and erosion of the gastric mucosa have also disappeared.

in the esophagus due to nausea, emesis and/or blood coagulation abnormalities. Representative spontaneous injuries include spontaneous rupture of the esophagus, reported by Boerhaave in 1724 as tearing of all layers of the esophagus [3], Mallory-Weiss syndrome, reported by Mallory and Weiss in 1929 as bleeding from local tears in mucosa of the esophagogastric junction [4], and spontaneous submucosal dissection of the esophagus, reported by Williams in 1957 for hematomas found in the esophageal submucosa [5]. Since the report by Williams, this condition has been referred to by various names, including submucosal hematoma, submucosal dissection and intramural hematoma of the esophagus. Spontaneous submucosal hematoma of the esophagus is also broadly divided into 2 subgroups according to the presence of factors causing a tendency to bleed, such as drugs or underlying disease [6].

With regard to the onset mechanism in the present case, the hematoma is thought to have occurred as a result of traumatic factors related to a fish bone and there was no forced vomiting stimulation. Therefore, the case is classified as a mechanical, traumatic type. In addition, the patient was taking an antiplatelet agent, which may have led to extensive submucosal hematoma formation from the cervical esophagus past the esophagogastric junction to the gastric angle. To date, there has been only one report in Japan of submucosal hematoma of the esophagus extending all the way to the gastric angle [7], and the present case is thought to be extremely rare.

In order to clarify the clinical features of this disorder in Japan and to compare them with this case, we searched Pub-Med and Ichushi-Web with the keywords of "esophageal submucosal hematoma" or "intramural hematoma of esophagus" and only the Japanese cases were selected from the results. The number of the cases from 1982 to 2009, including meeting abstracts and the present case, and excluding cases of direct iatrogenic esophageal injury, is 79 (Table 1). [1, 7–75] The patients included 35 men and 44 women, and the ages ranged from 32 to 84 years (mean age, 61.6 years), with a tendency toward older patients. The chief complaint was hematemesis in 60 cases, followed by chest pain in 26 cases, chest discomfort in 20 cases, and epigastralgia in 8 cases. There were only 8 cases with findings from the esophagus to the stomach, which consisted of 2 cases of hematoma with Mallory-Weiss syndrome complications reported by Shinozawa *et al.* [35] and Ichida *et al.*, [64] a case of hematoma to the fornix in by Kubota *et al.*, [37] 3 cases to the cardia reported by Karasawa *et al.*, [53] Sakai *et al.* [68] and Kawashima *et al.*, [70] and 2 cases to as far as gastric angle reported by Koyama *et al.* [7] and in the present paper. Comorbidities included chronic renal

Table 2 Cases of Esophageal Submucosal Hematoma in Japan.

No.	Author	Year	Sex	Age	Location	Chief complaint	Complications	Impaired hemostasis	Food-relaled injury	Ref
1	Hasegawa	1982	F	82	Ut-Mt	Hematemesis, Epigastralgia	Thrombocytopenia	Aspirin	N.M.	8
2	Shima	1984	M	43	Ce-EGJ	Hematemesis, Chest pain	(–)	Aspirin	N.M.	1
3	Hasebe	1984	F	40	Ut-Mt	Hematemesis	(–)	(–)	(–)	9
4	Konagaya	1986	F	51	Ce	Hematemesis	CRF, HD	Heparin	(–)	10
5	Konagaya	1986	F	42	Ce	Hematemesis	(–)	(–)	(+)	10
6	Frusawa	1986	M	58	Ut-EGJ	Hematemesis	(–)	(–)	N.M.	11
7	Harada	1987	M	40	Mt	Nausea	CRF, HD	Heparin	N.M.	12
8	Tsukioka	1988	F	44	Ce-Ae	Epigastralgia, Dysphagia	(–)	(–)	(+)	13
9	Ooshika	1991	M	63	?	Epigastric discomfort	CRF, HD	Heparin	N.M.	14
10	Matoba	1992	F	38	Ut	Hematemesis	(–)	(–)	(+)	15
11	Furukawa	1992	F	44	Mt-Lt	Chest pain, Hematemesis	(–)	(–)	N.M.	16
12	Shimada	1993	F	41	Ut-Lt	Sore throat, Chest pain, Hematemesis	(–)	(–)	(+)	17
13	Zeniya	1994	M	41	Ce-Ut	Hematemesis	(–)	(–)	(+)	18
14	Zeniya	1994	M	45	Ce-EGJ	Hematemesis	(–)	(–)	N.M.	18
15	Takami	1994	M	43	Ce-EGJ	Chest pain, Emesis	(–)	(–)	(+)	19
16	Yamada	1995	M	64	Ut-Lt	Emesis, Chest pain, Hematemesis	LC	(+)	N.M.	20
17	Nagata	1995	M	75	Mt-EGJ	Emesis, Hematemesis	Ileus	(–)	(–)	21
18	Ousaka	1995	M	50	Ut- Lt	Emesis, Hematemesis	(–)	(–)	(+)	22
19	Koyama	1996	F	73	Ut-EGJ	Hematemesis	(–)	(–)	N.M.	7
20	Nagayama	1996	F	57	Ut	Emesis, Hematemesis	(–)	(–)	(+)	23
21	Hasebe	1997	F	51	Mt-Lt	Hematemesis	CRF, HD	Heparin	(–)	24
22	Hasebe	1997	M	32	Lt	Emesis, Hematemesis	(–)	(–)	(+)	24
23	Tanaka	1997	F	57	Ut	Hematemesis	(–)	(–)	(–)	25
24	Tanaka	1997	F	60	Ut-EGJ	Chest pain	(–)	(–)	(–)	25
25	Shinogi	1997	M	71	Ut-EGJ	Chest pain, Epigastralgia, Hematemesis	Cerebral infarct	Anticoagulant therapy	(+)	26
26	Matsui	1998	M	44	Mt-Lt	Chest pain	(–)	(–)	(+)	27
27	Amagai	1998	F	60	Ut-EGJ	Emesis, Hematemesis	(–)	(–)	(+)	28
28	Endoh	1998	F	67	Ce-EGJ	Epigastralgia, Hematemesis	(–)	(–)	(+)	29
29	Endoh	1998	F	49	Ce-Ut	Emesis, Hematemesis	(–)	(–)	N.M.	29
30	Sanaka	1998	F	57	Ce-Ut	Uncomfortable sensation in the throat	(–)	(–)	(+)	30
31	Ohara	1999	F	59	Ut- Lt	Chest pain, Hematemesis	(–)	(–)	(+)	31
32	Ishii	1999	F	56	Ut-EGJ	Chest pain	(–)	(–)	(+)	32
33	Ishii	1999	M	55	Mt-Lt	Chest pain, Hematemesis	(–)	(–)	(+)	33
34	Yamamoto	1999	F	70	?	Chest and back pain	(–)	(–)	N.M.	34
35	Shinozawa	1999	M	49	Mt-EGJ M-W (+)	Emesis, Hematemesis	(–)	(–)	N.M.	35
36	Masaki	2000	F	67	Ut-EGJ	Hematemesis	(–)	(–)	(+)	36
37	Kubota	2000	M	76	Ce-EGJ -Fornix	Hematemesis	Cerebral embolism Af	Thrombolytic therapy	N.M.	37
38	Uehara	2000	F	48	Mt- EGJ	Chest pain, Hematemesis	CRF, HD	Heparin	N.M.	38
39	Handa	2000	F	73	Ut-EGJ	Chest pain, Hematemesis	(–)	(–)	(–)	39
40	Shimomura	2000	F	55	Ut	Epigastric discomfort	CRF, HD	Heparin	N.M.	40
41	Yamashita	2000	F	67	Mt-Lt	Hematemesis	Cerebral aneurysm	Heparin	N.M.	41
42	Noma	2001	F	57	?	Chest discomfort, Hematemesis	(–)	(–)	N.M	42

43	Kise	2001	F	84	Mt-Lt	Chest discomfort, Hematemesis	Dissecting aortic aneurysm	(–)	N.M.	43
44	Yamamoto	2002	F	52	Ut-EGJ	Upper abdominal pain	ITP	(+)	N.M.	44
45	Nishinakagawa	2002	F	57	Ut	Emesis, Hematemesis	(–)	(–)	(+)	45
46	Nishinakagawa	2002	M	66	Ut-EGJ	Chest discomfort, Hematemesis	(–)	Antiplatelet therapy	(+)	45
47	Nakazawa	2002	F	71	Ut-Mt	Hematemesis	CHF	Aspirin	(+)	46
48	Tokuhiro	2002	F	75	Ut-EGJ	Chest pain, Hematemesis	(–)	(–)	(+)	47
49	Kouzu	2002	F	56	Ut-EGJ	Chest pain	(–)	(–)	(+)	48
50	Inomata	2002	F	67	Ce-Lt	Dyspnea	Esophageal achalasia	(–)	N.M.	49
51	Inomata	2002	F	74	Ce-Mt	Hematemesis, Chest discomfort	(–)	(–)	(+)	49
52	Murayama	2003	M	74	Hypo pharynx -EGJ	Hematemesis	OMI Pulmonary infarct DVT	Antiplatelet & Warfarin	(+)	50
53	Akashi	2003	M	56	Mt-Lt	Hematemesis	Artificial valve replacement	Warfarin	N.M.	51
54	Mitogawa	2003	F	67	Mt	Chest pain	(–)	(–)	(+)	52
55	Karasawa	2003	F	78	Ut-EGJ cardia	Epigastralgia, Hematemesis	(–)	(–)	(–)	53
56	Kato	2003	M	63	Mt-EGJ	Chest discomfort, Hematemesis	Post op	(–)	(–)	54
57	Yabu	2003	M	51	Ut-Lt	Chest pain, Emesis, Hematemesis	(–)	(–)	(+)	55
58	Wakiyama	2003	F	61	Ut-Lt	Chest pain, Hematemesis	RA	(–)	N.M.	56
59	Oonita	2004	F	67	Ut-Lt	Epigastralgia, Emesis, Hematemesis	(–)	(–)	(+)	57
60	Hata	2004	M	83	Ut	Chest pain	(–)	(–)	N.M.	58
61	Nagai	2004	M	66	Ut-Lt	Emesis, Hematemesis	(–)	(–)	(–)	59
62	Nakazawa	2004	M	63	Ut-EGJ	Hematemesis	CRF	(–)	N.M.	60
63	Nakae	2005	M	66	Ut-EGJ	Chest pain, Abdominal pain, Hematemesis	Cerebral arteriostenosis	Ticlopidine Aspirin Heparin	(–)	61
64	Okamura	2005	M	84	Mt-Lt	Emesis,Upper abdominal pain	Cerebral infarct Af	Aspirin	N.M.	62
65	Saito	2005	M	75	Ce-EGJ	Sore throat, Chest pain, Hematemesis	Angina pectosis	Ticlopidine Aspirin	(+)	63
66	Ichida	2006	F	63	Ce-EG JM-Wtear	Epigastralgia, Hematemesis	(–)	(–)	N.M.	64
67	Kosedo	2006	M	67	?	Sore throat, Chest pain, Hematemesis	Artificial valve replacement	Warfarin Aspirin	N.M.	65
68	Iwamoto	2007	M	72	Mt	Emesis, Chest pain, Hematemesis	Arteriostenosis obliterans Angina pectosis	Aspirin	(–)	66
69	Noumi	2007	F	58	Mt-Lt	Emesis, Hematemesis	(–)	(–)	N.M	67
70	Sakai	2007	M	69	Mt-EGJ Cardia	Dysphagia, Emesis	(–)	Aspirin	N.M	68
71	Inamura	2007	M	67	Ut-EGJ	Chest pain	(–)	(–)	N.M.	69
72	Kawashima	2008	F	74	Ce-EGJ Cardia	Sore throat	Angina pectosis	Aspirin	N.M.	70
73	Kawashima	2008	M	64	Ce-EGJ	Epigastralgia, Sore throat, Hematemesis	CRF, HD	Heparin	N.M.	70
74	Oota	2008	M	83	Ut-EGJ	Chest pain, Hematemesis	Angina pectosis	Aspirin	(+)	71
75	Enomoto	2008	M	84	Ce-EGJ	Chest discomfort, Emesis, Hematemesis	(–)	(–)	N.M.	72

76	Tobari	2008	F	69	Ce-EGJ	Chest discomfort, Hematemesis	Cerebral infarct	Antiplatelet	(+)	73
77	Hara	2009	F	81	Ce-EGJ	Emesis, Chest discomfort	(–)	(–)	(+)	74
78	Hirotani	2009	M	77	Ce-EGJ	Chest discomfort, Hematemesis	Angina pectosis	Aspirin Ticlopidine	N.M.	75
79	Present case	2009	F	70	Ce-EGJ Stomach	Sore throat, Chest discomfort, Hematemesis	(–)	Aspirin	(+)	

Ce: cervical esophagus; Ut: upper thoracic esophagus; Mt: middle thoracic esophagus; Lt: lower thoracic esophagus; Ae: abdominal esophagus; EGJ: esophageal gastric junction; AGML: acute gastric mucosal lesion; M-W tear: Mallory-Weiss tear; CRF: chronic renal failure; HD: hemodialysis; ITP: idiopathic thrombocytopenic purpura; LC: liver cirrhosis; Af: atrial fibrillation; N.M.: not mentioned; ?: location not specified.

failure in 8 cases (7 cases receiving hemodialysis), 6 cases of cerebrovascular disorder, 5 cases of angina pectoris, 2 cases following heart valve replacement, 1 case of hepatic cirrhosis, and 1 case of idiopathic thrombocytopenic purpura, and the latter 2 cases had the underlying disease with a tendency for increased bleeding. Twenty-seven patients were receiving medications that tended to cause bleeding to treat coexisting conditions (antiplatelet agents, 14 patients; heparin, 8 patients; warfarin, 3 patients; combined antiplatelet agent and heparin, 1 patient; combined antiplatelet agent and warfarin, 1 patient; thrombolytic therapy, 1 patient). Therefore, 27 cases (34.2%) had factors that increased the tendency to bleed. An antiplatelet agent was also being used in the present case. In many patients (33 patients), onset followed eating. Onset was not associated with meals in 12 cases, and was of unknown cause in 34 cases. Among the cases with onset after eating, there were 3 cases, including the present case, in which the possible cause was a fish bone. [36, 48] The present patient drank large amounts of water after ingesting the fish bone, and this might have increased the pressure within the esophagus, possibly exacerbating the hematoma.

Although patients often exhibit hematemesis or melena, bleeding is usually temporary and relatively minor [1, 76]. Subjective symptoms and hematoma generally disappear in 7 to 14 days with conservative treatment using intravenous drips with nothing by mouth but mucoprotective agents. The disorder is, therefore, thought to be a condition with good prognosis [77]. In only 2 reports by Hasebe *et al.* [9] and Hata *et al.*, [58] has recurrence been reported. In both cases, the size of the hematoma was small in both of the original and recurring ones. In the present case, the hematoma covered a very large area, but the patient recovered steadily with conservative treatment alone, which is in agreement with previous reports. Cases vary substantially from small to very large (such as in the present case); thus, CT scans, endoscopic examination, and gastrointestinal series tests are important in making a differential diagnosis, in addition to evaluation of the lesion.

Among the reported cases in our search, 8 had extensive hematomas reaching from the esophagus to the stomach. Excluding 2 of these cases in which the patient also had Mallory-Weiss syndrome, 4 of the 6 patients were taking medications that caused a tendency to bleed. The submucosal layer of the stomach is denser than that of the esophagus and it is therefore more difficult for the hematoma to extend from the esophagus to stomach. In cases without mechanical damage to the esophagogastric junction, such as Mallory-Weiss syndrome, it is possible that medications resulting in an increased tendency to bleed may be related to extension of the hematoma to the gastric submucosa. In this case, low-dose aspirin might relate to the extension of the large hematoma but probabaly did not with the development of the disorder. Therefore, she started low-dose aspirin again after the recovery and no recurrence has been observed.

In aging societies, people taking antiplatelet agents and anticoagulants, in addition to cases with traumatic factors such as ingestion of foreign objects and endoscopic examination/treatments, are increasing; thus, more attention should be focused on this rare condition.

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