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Case report

Microscopic polyangiitis complicated with massive intestinal bleeding

Shigehiko Ueda[1], Masami Matsumoto[1], Tatsuichi Ahn[1], Satoru Adachi[1], Kazumi Oku[1], Masahiro Takagi[1], Hiroshi Fukui[2], and Masahide Yoshikawa[3]

[1] Department of Internal Medicine, Nara Prefectural Gojo Hospital, 197 Nohara, Gojo, Nara 637-8511, Japan
[2] Third Department of Internal Medicine, Nara Medical University, Kashihara, Japan
[3] Department of Parasitology, Nara Medical University, Kashihara, Japan

Microscopic polyangiitis (MPA) is associated with renal dysfunction, in most cases, and occasionally with pulmonary hemorrhage. However, massive intestinal bleeding is a rare manifestation. We report a case of MPA in a man who developed arterial bleeding in the small intestine. A 74-year-old man was admitted after enduring a fever for 4 weeks. Laboratory examination revealed leucocytosis with neutrophil predominance, and renal dysfunction was noted. He did not respond to treatment with antibiotics, and the worsened renal function soon required hemodialysis. On the seventh hospital day, he suddenly developed massive melena. A colonoscopic examination suggested bleeding in the small intestine, and abdominal angiography demonstrated that the bleeding originated in a branch of the ileal artery. Hemostasis was achieved by transarterial embolization (TAE), but the patient suffered a massive pulmonary hemorrhage 2 days after the TAE. Although he responded well to a combination treatment with corticosteroid and cyclophosphamide, recurrence of pulmonary hemorrhage led to death, on the 87th hospital day. MPA in this patient was associated with three serious complications; deteriorating renal function, massive melena, and pulmonary hemorrhage. Of the various manifestations associated with MPA, arterial bleeding in the gastrointestinal tract, although rare, should be considered as one of the serious complications in MPA.

Key words: antineutrophil cytoplasmic autoantibody (ANCA), small vessel vasculitis, corticosteroid, intestinal bleeding, pulmonary hemorrhage

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Reprint requests to: S. Ueda

Introduction

Microscopic polyangiitis (MPA) is one form of small vessel vasculitis (SVV).[1–5] Renal manifestations are common in MPA, and about 10% of patients with MPA are reported to require dialysis because of severe renal dysfunction found at the time of diagnosis.[5,6] Moreover, MPA patients with renal insufficiency facing hemodialysis are known to be at high risk of alveolar hemorrhage.[6] These renal and pulmonary complications are representative life-threatening determinants,[7,8] while massive gastrointestinal bleeding is also a serious, but rare, complication. We report here a patient with MPA who developed arterial bleeding in the small intestine in addition to other serious complications, including rapidly progressive renal insufficiency and pulmonary hemorrhage.

Case report

A 74-year-old man was admitted to our hospital on March 20, 1997, after enduring a fever for 4 weeks. He had a history of acute glomerulonephritis, in his twenties, and had had a cholecystectomy for cholelithiasis and common bile duct stones at age 43 years. His height was 165 cm and his body weight was 62 kg at the original examination, although he had recently lost 5 kg during the course of the illness. Body temperature was 38.7°C and pulse was regular at 92 beats/min, with a blood pressure reading of 150/70 mmHg. No skin rash or superficial lymphadenopathy was observed. His complexion showed pallor, and his sclera was not icteric. Findings on examination of his heart and lungs were normal. On abdominal examination, the liver, spleen, and kidneys could not be palpated, and there was an operation scar in the middle of the abdomen. There was no peripheral edema, and no neurological abnormalities in the extremities.

Table 1. Laboratory data on admission

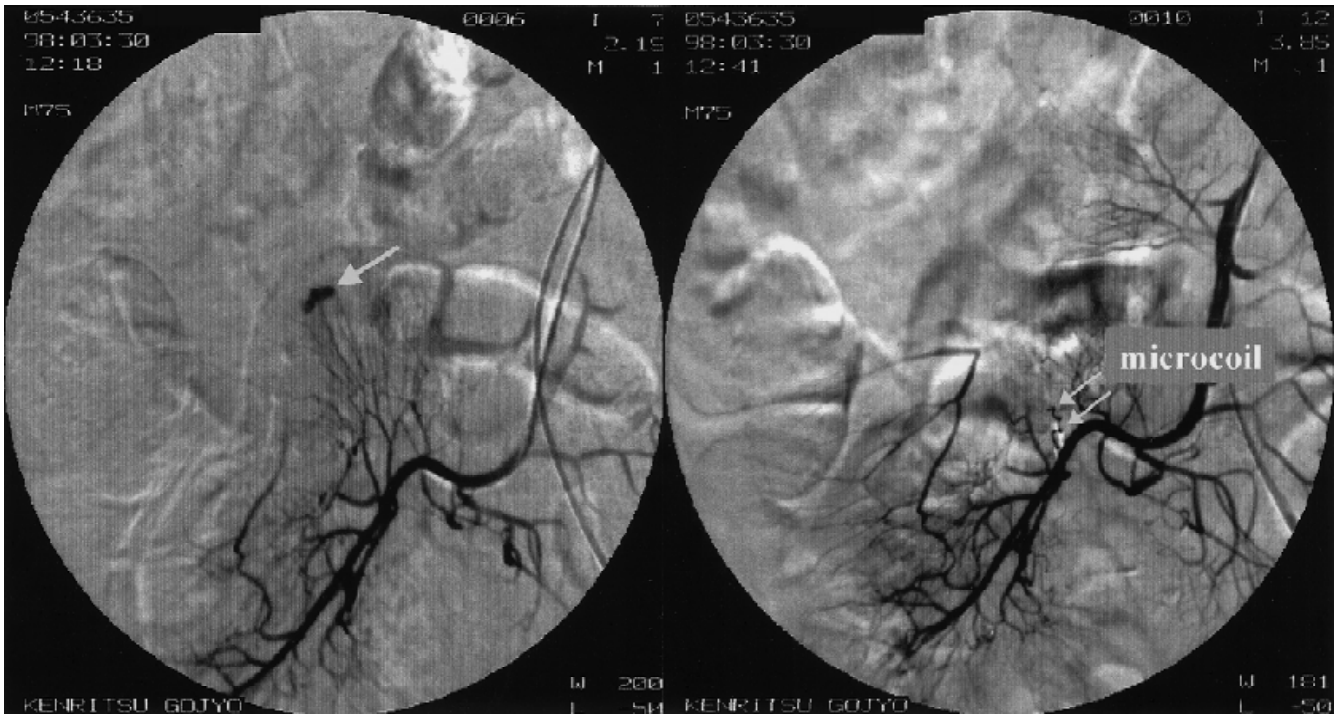
Urinalysis		Chemistry		Coagulation	
Protein	100 mg/dl	T-Bil	1.33 mg/dl	BT	2.0 min
Glucose	0.25 g/dl	ZTT	15.9 KU	PT	67.7%
Ketone	(−)	TP	7.4 g/dl	APTT	33.9 s
Urobilinogen	0.1 Eu/dl	Alb	2.6 g/dl		
Occult blood	3+	AST	138 IU/l	Serology	
Sediment		ALT	149 IU/l	HBsAg	(−)
RBC	20–29/HPF	LDH	422 IU/l	HCV-Ab (3rd)	(−)
WBC	30–49/HPF	ALP	984 IU/l	CRP	25.61 mg/dl
Epi.	(−)	γ-GTP	317 IU/l	IgG	2928.2 mg/dl
Feces		Ch-E	97 IU/l	IgA	412 mg/dl
Occult blood	(+)	T-chol	92 mg/dl	IgM	229 mg/dl
Peripheral blood		Amy	95 IU/l	CH_{50}	37.2 U/ml
RBC	309×10^4/mm^3	BUN	60.5 mg/dl	ANA	×160
Ht	27.9%	Cr	3.8 mg/dl	Anti-DNA Ab	(−)
Hb	8.8 g/dl	UA	8.1 mg/dl	Anti-RNP Ab	(−)
WBC	23 240/mm^3	Na	134 mEq/l	Anti-Sam Ab	(−)
neu.	88.2%	K	5.2 mEq/l	LE test	(−)
eosi.	0%	Cl	100 mEq/l	Immunocomplex	(−)
mono.	6.4%	Ca	7.5 mg/l	CEA	1.9 ng/dl
lym.	4.2%	Glu	186 mg/l	CA19-9	6 U/ml
baso.	0.3%				
Plt	38.9×10^4/mm^3				
ESR	110 mm/h				

Normal ranges: AST, 8–40 IU/l; ALT, 4–35 IU/l; LDH, 50–450 IU/l; ALP, 80–280 IU/l; γ-GTP, 3–50 IU/l; Ch-E, 170–410 IU/l; Amy, 60–200 IU/l; IgG, 1070–2200 mg/dl; IgA, 140–530 mg/dl; IgM, 80–430 mg/dl; CH_{50}, 30–40 U/ml; CEA, 0.6–5.0 ng/dl; CA19-9, <38 U/ml; HPF, high-power field

The results of laboratory tests are shown in Table 1. The patient's urine was positive for protein (100 mg/dl) and glucose (0.25 g/dl). The sediment contained 20–29 red blood cells (RBC) and 30–49 white blood cells (WBC) per high-power field on microscopy. A fecal sample was positive for occult blood. Peripheral blood analysis revealed normochromic normocytic anemia and leucocytosis, with neutrophil predominance. Blood chemistry demonstrated liver and renal dysfunction, showing aspartate aminotransferase (AST), 138 IU/l; alanine aminotransferase (ALT), 149 IU/l; alkaline phosphatase (ALP), 984 IU/l (normal range, 100–300 IU/l), γ-glutamyl transpeptidase (γ-GTP), 317 IU/l (normal range, 5–22 IU/l), cholinesterase (Ch-E), 97 IU/l (normal range, 320–720 IU/l); blood urea nitrogen (BUN), 60.5 mg/dl; and creatinine (Cr), 3.8 mg/dl. Serological examinations showed an elevated C-reactive protein (CRP) level, increased serum IgG amount, and positive results for antinuclear antibody. Abdominal ultrasonography (US) and radiological examinations, including chest X-ray and abdominopelvic computed tomography (CT), found no abnormalities. An endoscopic examination of the upper gastrointestinal tract revealed no ulcer or cancer.

We originally suspected that the patient might be suffering from some infection in the biliary tract or in the urinary tract, along with an underlying chronic renal disease, such as chronic glomerulonephritis. However, no pathogenic organisms were detected in culture specimens from urine or blood. The patient did not respond to a 5-day treatment with antibiotics, while his rapidly worsened renal function required the introduction of hemodialysis. We then decided that he was likely suffering from systemic vasculitis rather than an infection. Accordingly, we performed a checkup for antineutrophil cytoplasmic autoantibody (ANCA), a useful serological marker in diagnosing some forms of SVV.[1–5] We later obtained laboratory data on blood examinations for our patient that had been performed by a nearby doctor on May 12, 1996, these data revealed normal renal and liver function, except for slightly elevated γ-GTP (78 IU/l).

On the seventh hospital day, the patient suddenly developed massive melena. An emergent upper gastrointestinal endoscopic examination did not reveal any lesions in the esophagus, stomach, or duodenum. A subsequent colonoscopy showed large quantities of blood in the colon, which appeared to have come through the ileocecal canal. Although a precise observation could not be made, no definite lesions were present in the colon, suggesting that the bleeding seemed to have originated in the ileum. Abdominal angiography, performed sequentially after these endoscopic examinations, disclosed the extravasation of contrast material from a branch of the ileal artery (Fig. 1a), with no aneurysm observed. As a therapeutic intervention, embolization of the ileal artery was performed, using a microcoil. After this transarterial embolization (TAE),



a b

Fig. 1a,b. Digital subtraction angiography of the ileal artery. **a** Extravasation of contrast material from a branch of the ileal artery was found (*arrow*). **b** After transarterial embolization, the extravasation of contrast material was not detected

extravasation of contrast material was not detected (Fig. 1b), and the melena stopped. The progression of anemia also ceased after the TAE, and the patient had transient relief of his symptoms, including the fever. We could not define whether this arterial bleeding into the gastrointestinal tract was a complication related to systemic vasculitis or whether it was caused by any other diseases, such as Meckel's diverticulum.

However, 2 days after the TAE, in the morning, sudden hemoptysis occurred, and the patient expectorated about 200 ml of blood. Emergency radiological examinations, including a chest X-ray and CT, revealed diffuse alveolar infiltrates bilaterally in the lungs (Fig. 2a,b). This episode of pulmonary hemorrhage convinced us that the patient was suffering from systemic vasculitis, and we understood that the other complications, including the fever, the rapidly progressive renal insufficiency, and the arterial bleeding into the gastrointestinal tract, were all related to an underlying systemic vasculitis. We immediately instituted corticosteroid therapy, beginning with the intravenous (i.v.) administration of methlyprednisolone (mPSL), at 1.0 g/day, for 3 days. Prednisolone (PSL) was then given, orally, at a daily dose of 60 mg, in combination with 100 mg/day of cyclophosphamide (CP), after the mPSL treatment. Two days after starting the mPSL treatment, we obtained the laboratory report stating that ANCA reacting with myeloperoxidase (MPO-ANCA) was positive, at 99 enzyme-linked immunoassay units (EU). We made a diagnosis of MPA, a type of ANCA-associated vasculitis, according to the recent international nomenclature definitions of systemic vasculitis.[4] ANCA reacting with a serine proteinase called proteinase 3 (PR3-ANCA) and anti-glomerular basement membrane antibody were both negative.

The patient responded well to the PSL/CS combination therapy. In order to confirm the diagnosis and to confirm that the treatment we were providing was appropriate, we hoped to conduct a biopsy on the kidneys or other organs when he returned to a stable condition. However, we were not able to obtain consent. He died of pulmonary hemorrhage on the 87th hospital day, and an autopsy was not permitted. The clinical course of the patient is shown in Fig. 3.

Discussion

In 1994, the Chapel Hill Consensus Conference for the Nomenclature of Systemic Vasculitis agreed on the names and definitions of many vasculitides that affect the kidneys.[4] MPA was defined as follows:

> Necrotizing vasculitis with few or no immune deposits, affecting small vessels, i.e., capillaries, venules, or arterioles. Necrotizing arteritis involving small- and

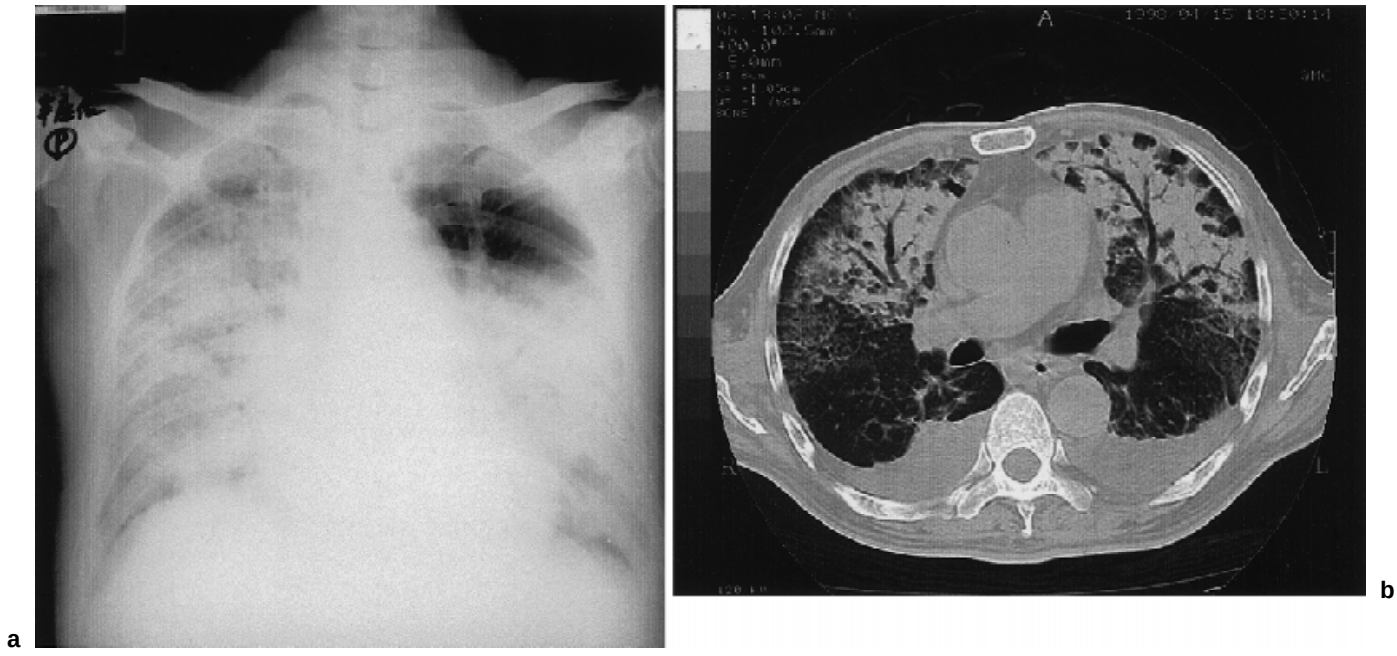


Fig. 2. **a** Chest X-ray and **b** chest computed tomography scan showed infiltrative shadows bilaterally in the lungs

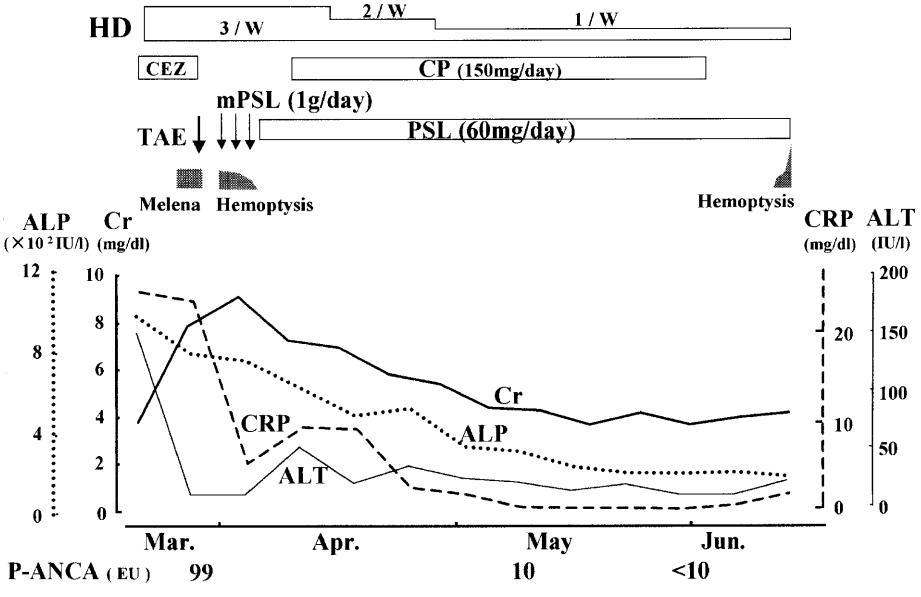


Fig. 3. Clinical course of the patient. *HD*, Hemodialysis; *CEZ*, cefazolin; *mPSL*, methyprednisolone; *PSL*, prednisolone; *CP*, cyclophosphamide; *W*, week; *TAE*, transarterial embolization; *Cr*, creatinine; *CRP*, C-reactive protein; *ALT*, alanine aminotransferase; *ALP*, alkaline phosphatase; *P-ANCA*, perinuclear-antineutrophil cytoplasmic autoantibody

medium-sized arteries may be present. Necrotizing glomerulonephritis is very common. Pulmonary capillaritis often occurs.

However, the conference report also commented that direct demonstration of certain pathologic processes noted in the definitions was not necessarily required in order to make a diagnosis. In our patient, the rapidly worsened renal function with the presence of proteinuria and hematuria suggested necrotizing glomerulonephritis with small-vessel involvement in the kidneys, and the pulmonary hemorrhage indicated the involvement of pulmonary capillaries. Based on definitions agreed by the consensus conference, we diagnosed our patient as having MPA without histopathological demonstration. The presence of ANCA was very helpful for diagnosing MPA, because ANCA is known to be frequently found in the following three types of

Table 2. Summary of patients with PAN or MPA with massive gastrointestinal bleeding

Patient no.	Author	Age (years) /Sex	Diagnosis[a]	Site of gastrointestinal bleeding	Other manifestations in the clinical course	Outcome	Findings of abdominal angiogram	
							Multiple aneurysms	Extravasation of contrast medium
1	Cabal and Holz[17]	43/M	Periarteritis nodosa	Duodenum	Retroperitoneal hemorrhage; rupture of the left kidney	Died	Hepatic artery (a.) Pancreaticoduodenal a. Splenic a. Superior mesenteric a.	Detected
2	Painter[16]	70/F	Polyarteritis nodosa	Ileum		Alive	Not performed	
3	Han et al.[18]	50/F	Polyarteritis nodosa	Colon	Intraabdominal and retroperitoneal bleeding; jaundice, prominent transaminase elevation	Died	Hepatic a.; superior and inferior mesenteric a.	Detected
4	Shin and Ho[19]	41/M	Necrotizing angiitis	Colon	Hypertension; renal insufficiency	Died	Superior mesenteric a.	Not detected
5	Roikjaer[20]	23/M	Polyarteritis nodosa	Colon	Renal insufficiency; peritonitis due to perforation of the cecum; colon necrosis; sepsis, positive for ANCA	Died	Not performed	
6	Kotiloglu et al.[21]	13/F	Microscopic PAN	Stomach	Intractable diarrhea; *Salmonella* infection; necrotizing glomerulonephritis	Died	Not performed	
7	Yazici et al.[22]	16/M	Polyarteritis nodosa	Jejunum	Hemobilia	Alive	Hepatic a.; superior mesenteric a.	Detected
8	Inaguma et al.[23]	54/M	ANCA-related vasculitis	Ileum	Pulmonary hemorrhage; duodenal ulcer; rapidly progressive glomerulonephritis; perforation of the gastrointestinal tract	Alive	Not detected	Detected
9	Mocan et al.[24]	10/M	Polyarteritis nodosa	Not described	Perforation of the cecum; uncontrollable hypertension; intraabdominal hematoma; perforation of the gastrointestinal tract	Died	Not performed	
10	Our patient	74/M	MPA	Ileum	Pulmonary hemorrhage; renal insufficiency; positive for ANCA	Died	Not detected	Detected

[a] Shown as the name described in the report (cases 5, 6, and 8 are considered to be MPA)
PAN, Polyarteritis nodosa; MPA, microscopic polyangiitis; ANCA, antineutrophil cytoplasmic autoantibody

SVV;[3–5] MPA, Wegener's granulomatosis (WG), and Churg-Strauss syndrome. In addition, analysis of the antigen specificity was useful in differentiating between WG and MPA with respiratory tract involvement. The ANCA in patients with MPA may be either MPO-ANCA or PR3-ANCA, while the ANCA in patients with WG is almost always PR3-ANCA. The presence of MPO-ANCA in our patient supported the diagnosis of MPA. According to the Revised Japanese Criteria for the Diagnosis of MPA put forward by the National Study Group of Angiitis in 1998, the diagnosis of "definite MPA" is made either (1) when MPO-ANCA is detected in patients presenting with both rapidly progressive glomerulonephritis (RPGN) and pulmonary hemorrhage, or (2) when histological evidence is demonstrated in patients presenting with more than any two of the following three symptoms: (i) RPGN, (ii) pulmonary hemorrhage, and (iii) other symptoms, such as purpura, subcutaneous hemorrhage, gastrointestinal bleeding, and mononeuritis multiplex. The present patient satisfied the criteria (1) conditions of definite MPA. Some nephrologists believe that the presence of ANCA has a high sensitivity and specificity for so-called pauci-immune necrotizing and crescentic glomerulonephritis.[9] Therefore, the presence of MPO-ANCA and the histological demonstration of small-vessel involvement are of almost equal value in diagnosing MPA.

MPA was initially recognized as a particular type of polyarteritis nodosa (PAN). PAN is now defined as vasculitis affecting medium-sized or small arteries without small-vessel involvement and usually negative for ANCA, whereas MPA may be associated with medium-sized vessel involvement, as in our patient, in whom medium-sized vessel involvement was confirmed by bleeding seen on the abdominal angiograms. According to a report examining the clinical features of PAN and MPA,[10] patients with either PAN or MPA with medium-sized vessel involvement were likely to have gastrointestinal symptoms comparable to those of MPA without medium-sized vessel involvement. The gastrointestinal involvement has been reported in 20%–79% of patients with PAN[11–14] and is also considered to occur at similar frequencies in patients with MPA with medium-sized vessel involvement.[15] However, massive gastrointestinal bleeding is extremely rare. As a result of a computer search of the literature describing gastrointestinal bleeding in PAN and MPA, severe gastrointestinal hemorrhaging was found to be documented in only nine patients[16–24] (Table 2). Most of these patients had other serious complications and showed a poor prognosis. A characteristic feature in those who underwent angiographic examinations was the presence of multiple aneurysms. The rupture of an aneurysm was considered to be pathognomonic for gastrointestinal bleeding. Although aneurysm formation was not found in our patient, active bleeding was detected on the periphery of the ileal artery, indicating the involvement of a medium-sized vessel. In five patients, including ours, extravasation of contrast material was seen on abdominal angiograms. Therefore, we believe abdominal angiography is of great importance, not only for establishing a diagnosis of PAN or MPA with medium-sized vessel involvement but also for detecting the site of bleeding in patients presenting with severe gastrointestinal bleeding, especially when upper and lower endoscopic examinations have failed to show the bleeding source. In addition, recent advances in interventional radiology may provide a safe and effective means of hemostasis. Inaguma et al.[23] reported the first case of MPA in a patient in whom massive gastrointestinal bleeding was stopped by TAE, although there was a recurrence of massive intestinal bleeding 3 days later. Fortunately, complete hemostasis was achieved in our patient.

Necrosis or perforation of the gastrointestinal tract is another rare, but serious, potential complication in systemic vasculitis, in addition to massive gastrointestinal bleeding.[25–28] It occurs as a result of impaired circulation in the gastrointestinal tract wall caused by a mesenteric aneurysm, or its rupture. The small intestine is frequently involved in this complication. A few patients with PAN or MPA who developed colonic perforation have also been reported.[20,29,30] Our patient did not have an intestinal perforation; however, this possibility should always be considered when patients with PAN or MPA present with abdominal pain or gastrointestinal bleeding.

In conclusion, we have reported a patient with MPA who presented with rapidly progressive renal insufficiency, massive melena, and pulmonary hemorrhage, in succession, shortly after hospitalization. It should be noted that gastrointestinal bleeding is an important and serious complication, in addition to deteriorating renal function and pulmonary hemorrhage, in patients with MPA.

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