

Case report

Microscopic polyangiitis associated with subarachnoid hemorrhage

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Abstract

Microscopic polyangiitis (MPA), an anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis, is a systemic disease that damages all organs through predominantly affecting small vessels. However, few cases of MPA are related to aneurysms on medium-sized muscular vessels, and whether subarachnoid hemorrhage (SAH) is associated with MPA is still unclear. An 85-year-old woman with rapid progressive glomerular nephritis caused by MPA complained of sudden severe headache due to SAH 2 days after admission and subsequently underwent surgery. Cerebrovascular disease occurring simultaneously with MPA might result in poor prognosis, and the complications exacerbate the condition and lead to high mortality; thus, physicians should pay more attention to cerebral aneurysms concurrent with MPA. Among patients with MPA, it is important to identify priority cases and investigate the intracranial vessel environment. To the best of our knowledge, this is a rare report about SAH associated with MPA. We recommend that the presence of cerebrovascular disease should be considered in patients with MPA to improve their prognosis.

Key words: ANCA-associated vasculitis, microscopic polyangiitis, subarachnoid hemorrhage, cerebral aneurysm, prognosis

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Introduction

The Chapel Hill Consensus Conference (CHCC) has played an important role in classifying primary vasculitis

based on the size of the affected vessels¹. Under this classification, anti-neutrophil cytoplasmic antibody (ANCA) is a common pathologic factor in vasculitis of small vessels, called ANCA-associated vasculitis (AAV). While granulomatosis with polyangiitis (GPA) is more common in the United States and Europe, microscopic polyangiitis (MPA) is the major AAV in Japan, and it mainly affects the kidneys and lungs^{2,3}. MPA is defined as pauci-immune necrotizing vasculitis predominantly affecting small vessels. Hence, MPA is known to be a systemic disease that damages every organ involved with small vessels, namely the lungs, kidneys, skin, and peripheral nervous system.

In general, few cases of MPA are related to aneurysm formation on medium-sized muscular vessels, although CHCC classification describes the possibility of necrotizing arteritis involving small and medium arteries in MPA¹. As a result, cerebral aneurysms are rarely encountered among patients with MPA. Therefore, whether subarachnoid hemorrhage (SAH) is associated with MPA is unclear.

However, we encountered an 85-year-old woman who had SAH concurrent with MPA, and hypothesized that the SAH might be associated with MPA. Thus, this is a rare report about SAH associated with MPA.

Case Report

An 85-year-old woman complained of anorexia for 1 month prior to admission. She also experienced abdominal pain, leading to lower intake of food and water. She showed extreme dehydration and loss of body weight. Eventually, she was admitted to our hospital for anorexia, abdominal pain, and fatigue, which had resulted in renal dysfunction and hyperkalemia. Her medical history included postoperative uterine myoma, pancreatic neuroendocrine tumor with conservation strategy, grade C–D reflux esophagitis according to the Los Angeles classification 21 months before admission, and right renal cell carcinoma completely excluded

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by operation 13 months prior to the present admission. Her younger brother had died of cholangiocarcinoma, and her mother had leukemia. The patient denied alcohol use, smoking, and any allergies. She was administered esomeprazole and magnesium hydroxide. On admission, her consciousness was lucid; she had a height of 143.3 cm, body weight of 57.5 kg, body mass index of 28.0 kg/m², body temperature of 36.7°C, blood pressure of 158/67 mmHg, pulse of 65 beats per minute, and oxygen saturation of 97% in room air. Physical examination revealed anemia on palpebral conjunctiva, severely dry tongue, and edema of the extremities. The neck, heart, lung, and abdomen revealed no abnormalities. The results of the laboratory tests demonstrated urinary abnormalities, anemia, hypoalbuminemia, renal dysfunction, electrolyte abnormalities, metabolic acidosis, and elevation of C-reactive protein level (Table 1). In comparison to her past renal function with an estimated glomerular filtration rate of 50–59 mL/min/1.73 m² and a normal urinary status 1 month prior to the present admission, the exacerbation of her renal function indicated rapid progressive glomerulonephritis (RPGN). A few days after submission of the laboratory tests, the results revealed that the serum titer of MPO-ANCA was 444.0 IU/mL using the Fluorescence Enzyme Immunoassay (normal < 5.0 IU/mL). Chest radiograph and computed tomography (CT) examinations showed no abnormalities in the lung field (Figure 1a, b). The left kidney was swollen (101 × 59 mm) but revealed no hydronephrosis (Figure 1c). Transthoracic echocardiography showed no vegetation on the cardiac valves, and both sets of blood cultures showed negative results. Renal biopsy was not performed, as the patient had only the left kidney. Based on the CHCC classification¹⁾ and clinical practice guideline for ANCA-associated vasculitis⁴⁾, vasculitides induced by malignancy condition, systemic disease (e.g., rheumatoid arthritis, systemic lupus erythematosus), infections (e.g., infective endocarditis, hepatitis C, and tuberculosis), and drugs (e.g., propylthiouracil, hydralazine, minocycline)⁵⁾ was excluded. Since MPO-ANCA was positive (Table 1) and as “findings of a positive ANCA should suggest the diagnosis of small vessel vasculitis such as MPA, GPA, or eosinophilic granulomatosis with polyangiitis (EGPA)”⁵⁾, the patient was diagnosed with AAV rather than immune complex small vessel vasculitis. After excluding EGPA and GPA, and confirming the RPGN with urinary exacerbation, she was eventually diagnosed with MPA. The severity of her vasculitis was equivalent to 14 points in the Birmingham Vasculitis Activity Score⁶⁾ and 7 points in the Renal Severity Classification, which is equal to grade III according to the clinical guideline for RPGN⁷⁾.

Subsequently, a therapeutic strategy against MPA was planned. However, the patient suddenly complained of severe headache and nausea on the third day of hospital-

ization. Her blood pressure was 180/110 mmHg, and she vomited several times. Cranial CT indicated a high-density area in the basal cistern corresponding to SAH and mild hydrocephalus (Figure 2a). Moreover, digital subtraction angiography revealed a fusiform and a saccular-like aneurysm sharing their neck, arising from the right middle cerebral artery (Figure 2b, c). Based on the location of the subarachnoid hematoma, it was concluded that the SAH was attributed to the rupture of the right middle cerebral artery aneurysm, and emergent neck clipping was performed. Although a premature rupture occurred upon the dissection of the neck of the aneurysm, bleeding was well controlled through application of a temporary clip. The neck clipping was accomplished in the usual manner followed by ventricular drainage owing to the tight posterior fossa. The SAH of the patient was classified as grade II, I, and 3 according to the World Federation of Neurosurgical Societies, Hunt & Kosnik, and Fischer classifications, respectively. Despite the successful operation, her postoperative consciousness was poor, which scored 9 points (E3V1M5) on the Glasgow Coma Scale; unfortunately, we never communicated with her after the event. To introduce remission and avoid further occurrence of any complications from MPA, she was administered intravenous methylprednisolone (0.5 g/day for three doses) followed by oral prednisolone at 30 mg/day (0.5 mg/kg/day). Considering the deterioration of her renal function owing to RPGN, the patient was started on renal replacement therapy (RRT) via a catheter until her renal function recovered. Intermittent hemodialysis was performed owing to her stable hemodynamic condition. After the initiation of steroid therapy and RRT, her renal function gradually recovered, and we were able to withdraw RRT as the titer value of MPO-ANCA became negative (Figure 3). The patient never experienced SAH, or cardiovascular or cerebrovascular events. Prophylaxis against infection was started soon after the immunosuppressive therapy; however, repeated instances of infectious diseases including pneumonia, urinary tract, and catheter-related blood stream infections were observed. Unfortunately, the patient died of infection on Day 135 of hospitalization.

Discussion

From the findings of this study, we confirmed two important clinical issues: 1) cerebrovascular disease concurrent with MPA leads to poor prognosis, and 2) more attention should be paid to cerebral aneurysms complicated with MPA.

First, patients with cerebrovascular disease simultaneous with MPA tend to have poor prognosis. Table 2 demonstrates the poor prognoses in patients with MPA once

Table 1 Laboratory tests upon admission

blood tests	values	<i>(continues)</i>		urinalysis	values
WBC	10400 / μ L	T-Bil	0.4 mg/dL	protein	2+
neutrophils	90.1%	CK	11 U/L	glucose	–
lymphocyte	5.4%	BS	100 mg/dL	gravity	1.012
monocyte	3.1%	HbA _{1c}	5.9 %(NGSP)	pH	5.5
eosiphils	1.2%	AMY	41 U/L	urobilinogen	±
basophils	0.2%	TG	112 mg/dL	bilirubin	–
RBC	283 \times 10 ⁴ / μ L	LDL-C	72 mg/dL	ket	–
Hb	8.6 g/dL	Fe	21 μ g/dL	WBC	2+
Ht	25.50%	TIBC	158 μ g/dL	nitrate	–
MCV	90 fL	ferritin	232.7 ng/dL	occult blood	3+
MCH	30.3 pg	CRP	11.1 mg/dL	bacteria	3+
MCHC	33.70%	KL-6	165 U/mL	RBC	10–19 /hpf
Plt	39.6 \times 10 ⁴ / μ L	SP-D	29.7 ng/mL	WBC	1–4 /hpf
PT	55.90%	(1-3) β -D Glucan	< 6.0 pg/mL	epithelial cells	1–4 /hpf
PT-INR	1.23	free T ₃	1.43 pg/mL	granular cast	1–4 /WF
aPTT	> 130.0 sec	free T ₄	1.06 ng/dL	TP	149 mg/dL
fibrinogen	501 mg/dL	TSH	1.96 μ U/mL	Cr	59.9 mg/dL
D-dimer	10 μ g/mL	BNP	619.5 pg/mL	Na	54 mEq/L
FDP	24 μ g/mL	HBs Antigen	–	Cl	54 mEq/L
AT-III	67.40%	HBs Antibody	85.7 mIU/mL	K	34 mEq/L
TP	6.3 g/dL	HBc Antibody	1.16 INH%	urinary collection	value
Alb	2.2 g/dL	HCV Antibody	–	TP	0.515 g/day
BUN	75 mg/dL	RPR	–	UN	1.698 g/day
Cr	6 mg/dL	TPLA	–	Cr	0.256 g/day
UA	9.5 mg/dL	IgG	1664 mg/dL	Na	43.2 mEq/day
Na	124 mEq/L	IgM	106 mg/dL	Cl	48.6 mEq/day
K	7.4 mEq/L	IgA	339 mg/dL	K	15 mEq/day
Cl	98 mEq/L	C ₃	76 mg/dL	urine volume	600 mL/day
Ca	8 mg/dL	C ₄	14 mg/dL	arterial blood gas	values
iP	5.5 mg/dL	CH ₅₀	52 U/mL	pH	7.367
LDH	252 U/L	cryoglobulin	–	PCO ₂	28.6 mmHg
AST	10 U/L	ANA (homogenous)	20 times	PO ₂	102 mmHg
ALT	7 U/L	Anti GBM Antibody	2.3 U/mL FEIA	HCO ₃ [–]	16 mmol/L
γ GTP	13 U/L	MPO-ANCA	444 IU/mL FEIA	Na ⁺	128 mmol/L
ALP	208 U/L	PR3-ANCA	1 IU/mL FEIA	Cl [–]	102 mmol/L

Hb: hemoglobin, Ht: hematocrit, Plt: platelet, PT: prothrombin time, PT-INR: prothrombin time-international normalized ratio, aPTT: activated partial thromboplastin time, FDP: fibrin degradation product, AT-III: antithrombin III, TP: total protein, Alb: albumin, BUN: blood urea nitrogen, Cr: serum creatinine, UA: uric acid, LDH: lactic acid dehydrogenase, AST: aspartate aminotransferase, ALT: alanine aminotransferase, γ GTP: γ -glutamyltranspeptidase, ALP: alkaline phosphatase, T-Bil: total bilirubin, CK: creatine kinase, BS: blood sugar, AMY: amylase, TG: triglyceride, LDL-C: low-density lipoprotein cholesterol, TIBC: total iron-binding capacity, CRP: C-reactive protein, SP: surfactant protein, TSH: thyroid-stimulating hormone, BNP: brain natriuretic peptide, ANA: antinuclear antibody, GBM: glomerular basement membrane, MPO-ANCA: myeloperoxidase anti-neutrophil cytoplasmic antibody, PR3-ANCA: proteinase 3 anti-neutrophil cytoplasmic antibody. The extension time of aPTT was due to the disoperation of sample distribution.

cerebrovascular accidents occur^{8–16}). In addition, when considering the time to event after admission, we noticed that these events strike during active phases of the vasculitides. Despite successful operations in patients with SAH after the rupture of cerebral aneurysms, patients with MPA tend to experience various complications, as observed in our case,

because most of them tend to undergo immunosuppressive therapy. Under these circumstances, not only SAH but also the complications from immunosuppression (e.g., infectious diseases) would exacerbate the condition and lead to high mortality. Although there is no report about the prognosis of the association between SAH and immunocompromised

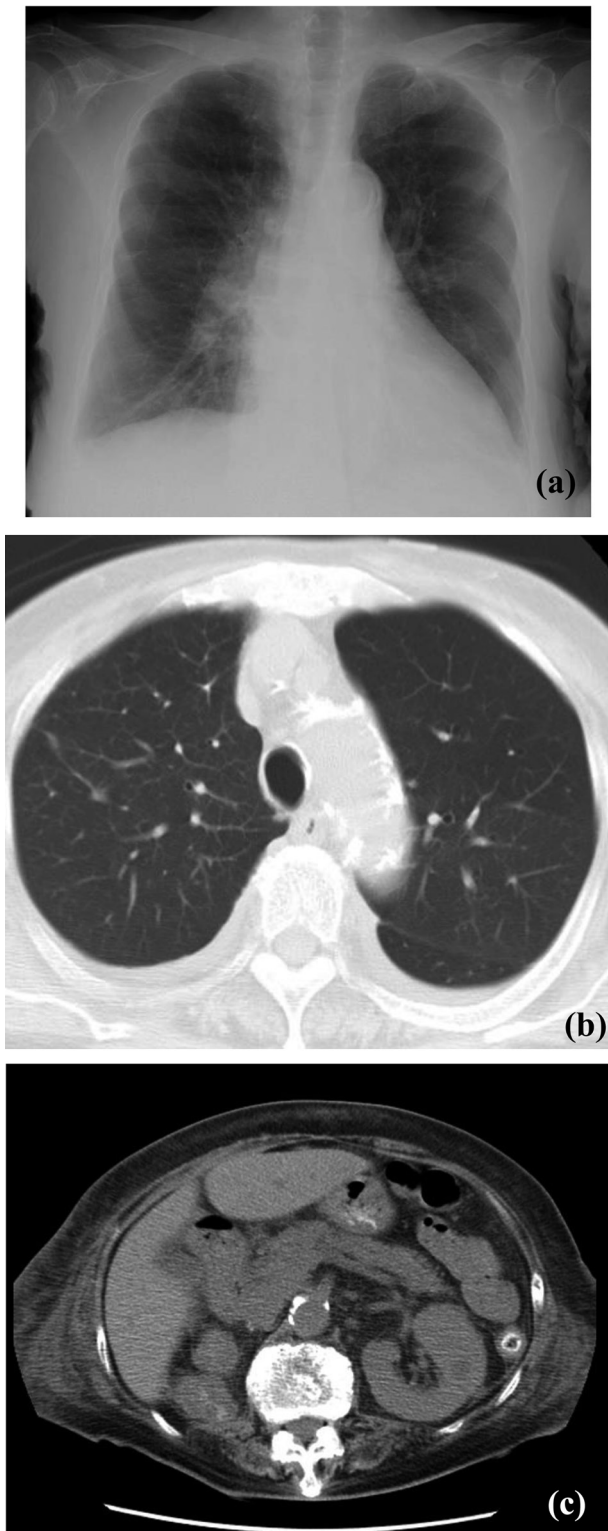


Figure 1 (a) (b) Lung field showed no abnormalities on chest radiography or computed tomography (CT). (c) Left kidney was swollen on abdominal CT.



Figure 2 (a) Computed tomography of the head showed subarachnoid hemorrhage, mainly located in the cisterna magna and mild hydrocephalus. (b) (c) Digital subtraction angiography revealed a fusiform and a saccular-like aneurysm sharing their neck, arising from the right middle cerebral artery (arrows).

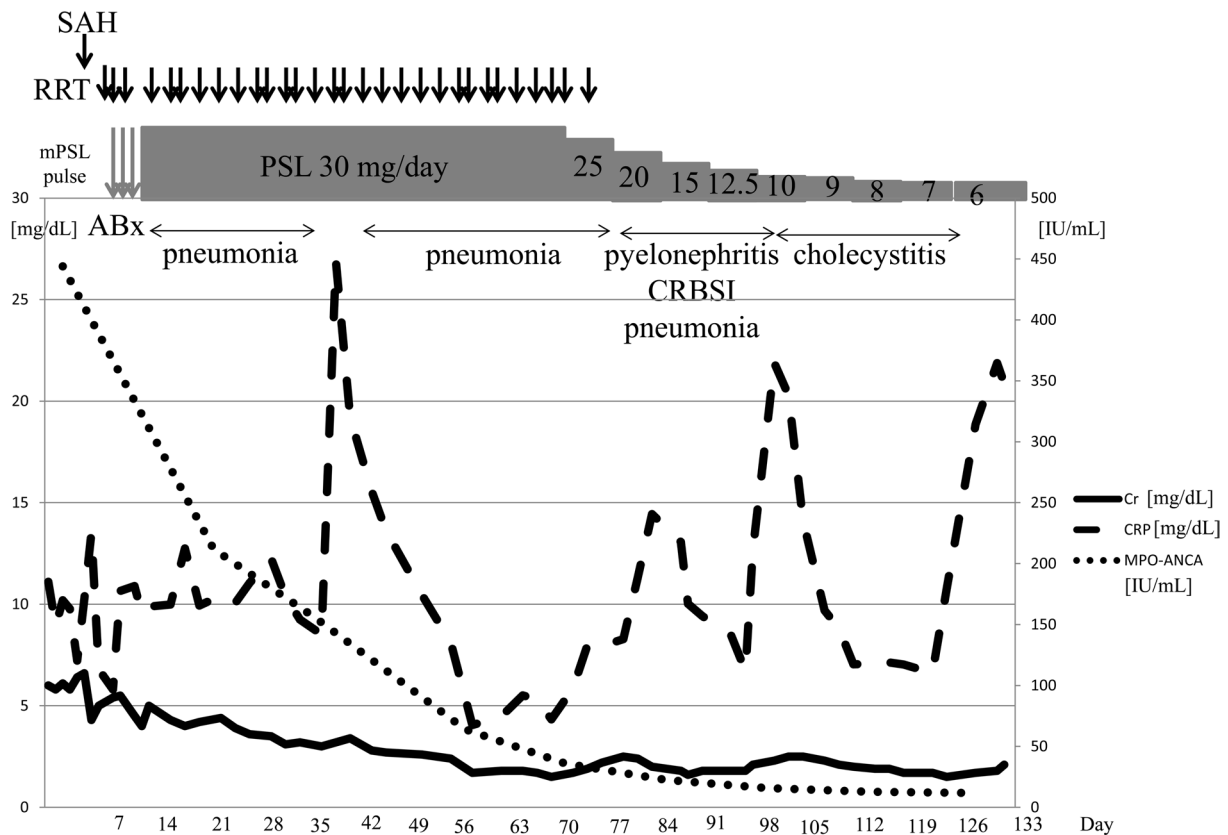


Figure 3 Clinical course after admission to hospital.

SAH: subarachnoid hemorrhage; RRT: renal replacement therapy; m-PSL: methylprednisolone; PSL: prednisolone; ABx: antibiotics; CRBSI: catheter-related blood stream infection; CRP: C-reactive protein; Cr: serum creatinine; MPO-ANCA: myeloperoxidase anti-neutrophil cytoplasmic antibody.

state, we could recognize the poorer prognosis of SAH concurrent with MPA based on our experience. Hence, we recommend that the presence of cerebrovascular disease should be anticipated in patients who show positive results for MPO-ANCA, in order to improve the prognoses of these patients.

Second, more attention has to be paid to cerebral aneurysms associated with MPA. Although aneurysms of medium-sized vessels are rarely reported in MPA¹⁷⁾ and little is known about their accurate pathogenesis and mechanisms in aneurysm formation, MPA could be related to arteritis of both small- and medium-sized vessels and aneurysm formation. Firstly, approximately 23% of MPA cases were reported to be involved with the medium-sized renal artery¹⁸⁾. Secondly, one report hypothesized that debilitating vessel walls and aneurysm formation result from necrotizing changes¹⁹⁾. For instance, Kimura *et al.* reported a case of SAH derived from pseudoaneurysm on the posterior inferior cerebellar artery, which was histologically consistent

with MPA⁹⁾ (Table 2). Two cases of MPA reported both necrotizing changes and intrarenal aneurysms^{20, 21)}, and an autopsy case of a 74-year-old man with MPA leading to intra-abdominal hemorrhage revealed medium-sized arteritis and rupture of a pseudoaneurysm on a branch of the left gastric artery²²⁾. Lastly, CHCC classification specifies that “ANCAs are not associated with polyarteritis nodosa (PAN)” for discriminating from AAV because “PAN and AAV can exhibit clinically and pathologically indistinguishable necrotizing arteritis of medium and small arteries”¹⁾. Therefore, we could consider that the medium-sized vasculitides are derived from MPA when patients show positive results for ANCAs. Despite the low prevalence of cerebral aneurysm formation complicated with MPA, our case indicates an association between SAH and MPA. This will enable the provision of interventions for cerebral aneurysms before they rupture.

In conclusion, cerebrovascular disease occurring simultaneously with MPA might result in poor prognosis; thus, physicians should pay more attention to cerebral aneurysms

Table 2 Previous reports about cerebrovascular disease in patients with MPA

Case	Reference	Age/Sex	Cerebrovascular diseases	Time to event after admission	Aneurysms	Biopsy	Responsible areas	Complications	ANCA	Outcome
1	Isoda <i>et al.</i> ⁽¹⁰⁾	72/M	cerebral infarction and hemorrhage	8 days	Yes (renal artery)	Not performed	right thalamus	RPGN polyneuropathy	MPO-ANCA 330 EU	Survival
2	Kimura <i>et al.</i> ⁽¹¹⁾	44/M	subarachnoid hemorrhage	2 days	Yes (pseudoaneurysm)	fibrin layer inflammatory cell infiltration in aneurysm	posterior inferior cerebellar artery	RPGN	MPO-ANCA 312 EU	Survival
3	Yamashiro <i>et al.</i> ⁽¹²⁾	67/F	cerebral infarction	N/A	No	Not performed	left thalamus	microscopic hematuria alveolar hemorrhage	MPO-ANCA 51.2 U/ml	N/A
4	Sasaki <i>et al.</i> ⁽¹³⁾	78/M	cerebral hemorrhagic infarction	on admission	No	necrotizing angiitis in cerebral vessels	cerebral cortex	RPGN alveolar hemorrhage	N/A	Dead
5	Ito <i>et al.</i> ⁽¹⁴⁾	56/M	cerebral hemorrhage and infarction	5 days	No	hypertrophic endothelial cells perivascular inflammation at perioheral nerve	bilateral corona radiata	polyneuropathy	MPO-ANCA 640 U	Dead
6	Han <i>et al.</i> ⁽¹⁵⁾	43/M	cerebral hemorrhage	9 days	No	leucocytotoxic vasculitis in cerebral vessels	N/A	RPGN	PR3-ANCA 40.0 U/ml	Dead
7	Tang <i>et al.</i> ⁽¹⁶⁾	55/M	cerebral infarction	on admission	No	leukoerythrocytic vasculitis in skin	bilateral basal ganglionic infarction	No	MPO-ANCA 22 U/ml	Survival
8	Iyoda <i>et al.</i> ⁽¹⁷⁾	60/F	cerebral hemorrhage	one day between 2 and 26 days	N/A	necrotizing crescentic glomerulonephritis renal arteriolitis with fibrinoid necrosis	cerebral cortex	RPGN pulmonary hemorrhage	MPO-ANCA 1,230 EU	Dead
9	Ku <i>et al.</i> ⁽¹⁸⁾	66/F	multiple cerebral infarctions	25 days	No	perivascular inflammation with occlusion in peripheral nerve	corona radiata basal ganglia	neuropathy	MPO-ANCA 1,719 AAU	Survival

MPA: microscopic polyangiitis, ANCA: anti-neutrophil cytoplasmic antibody, RPGN: rapid progressive glomerulonephritis, MPO: myeloperoxidase, PR 3: proteinase 3, N/A: not applicable.

concurrent with MPA. As improvement of the prognosis of patients with cerebral aneurysms is possible before the rupture of blood vessels, MPA cases should be carefully investigated to identify the cases that should be prioritized and to determine the environment of intracranial vessels in these cases.

Conflict of Interest: The authors declare that no conflict of interest exists.

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