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Anti-Neutrophil Cytoplasmic Antibody-Associated Vasculitis Developing Pancreatic Lesion and Diabetes Mellitus: A Case Report and Review of the Literature

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Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) affects small blood vessels and causes severe systemic organ injury commonly affecting the lungs and kidney. However, gastrointestinal, especially pancreatic, lesions are rare. We report the case of a 67-year-old Japanese man diagnosed with myeloperoxidase (MPO) AAV who developed pancreatic lesions and diabetes mellitus. The patient was admitted to our hospital due to fever, cough, and weight loss. He developed progressive glomerulonephritis, lung nodules, and pancreatic swelling and mass. Additionally, laboratory examination revealed positive MPO-ANCA and elevated glycated hemoglobin A1c, which were suggestive of diabetes mellitus. Renal biopsy revealed necrotizing crescentic glomerulonephritis and vasculitis in the small arteries. Endoscopic ultrasound-guided fine needle aspiration of the pancreas was performed, and histological findings suggested the possibility of pancreatic vasculitis and parenchymal injury. The patient was diagnosed with AAV, which was managed with glucocorticoids. This improved the renal function and pancreatic lesions. Furthermore, blood glucose levels improved despite treatment with glucocorticoids. These findings suggest that AAV-related pancreatic lesions worsened glycemic control. However, glucocorticoid therapy improved vasculitis and pancreatic lesions, which resulted in improved glycemic control.

Keywords: anti-neutrophil cytoplasmic antibody; diabetes mellitus; histopathological diagnosis; pancreas; vasculitis
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Introduction

Anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) causes systemic symptoms such as fever, weight loss, fatigue, and small vessel inflammation-induced organ damage (Kitching et al. 2020). AAV is mainly categorized into the following three types: microscopic polyangiitis (MPA), granulomatosis with polyangiitis (GPA), and eosinophilic granulomatosis with polyangiitis (EGPA) (Kitching et al. 2020). AAV primarily targets the kidneys and lungs, which often have severe presentations. The representative kidney lesion is necrotizing glomerulonephritis that causes rapidly progressive glomerulonephritis, resulting in progressive renal failure and end-stage renal disease. In contrast, representative lung lesions include interstitial pneumonitis, alveolar hemorrhage, and nodular and cavitary lesions, particularly in patients with GPA (Kitching et al. 2020). Furthermore, purpura, multiplex mononeuritis, and eye lesions have been reported (Kitching et al. 2020). In addition, patients with GPA often have nasal and ear lesions (Kitching et al. 2020). Previous studies of patients with AAV have reported development of gastrointestinal lesions including ulcer, bleeding, cholecystitis, and gallbladder bleeding; however, these complications are rare (Chetty and Serra 2017). Similarly, pancreatic lesions have been reported with an even lower incidence (Pagnoux et al. 2005).

Here, we report a case of AAV that resulted in a pancreatic lesion and newly diagnosed diabetes mellitus. After treatment with glucocorticoids, the blood glucose level of

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the patient improved. Herein, we discuss the development of pancreatic lesions and diabetes mellitus in a patient with AAV and the prognosis of such patients.

Case Presentation

A 67-year-old Japanese man presented to a local hospital with fever associated with cough and weight loss. No associated upper respiratory tract symptoms were noted. Past medical history revealed hypertension. He was admitted for further evaluation.

Blood examination revealed elevated C-reactive protein and leukocytosis. Additionally, hemoglobin A1c (HbA1c) was 6.7% upon admission and increased to 7.8% one month after hospital admission.

Contrast-enhanced chest and abdominal computed tomography (CT) scan revealed multiple nodular lesions in both lungs (Fig. 1A, B), mass formation in the pancreatic head (Fig. 1C), and pancreatic swelling (Fig. 1D). Considering the imaging findings, pancreatic cancer and IgG4-related disease were suspected. Therefore, endoscopic retrograde pancreatography (ERP) and endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) were performed to obtain a sample from the swollen pancreatic tail. Notably, the presence of the aorta on the puncture route presented challenges in obtaining tissue from the pancreatic head mass. No malignant findings were observed;

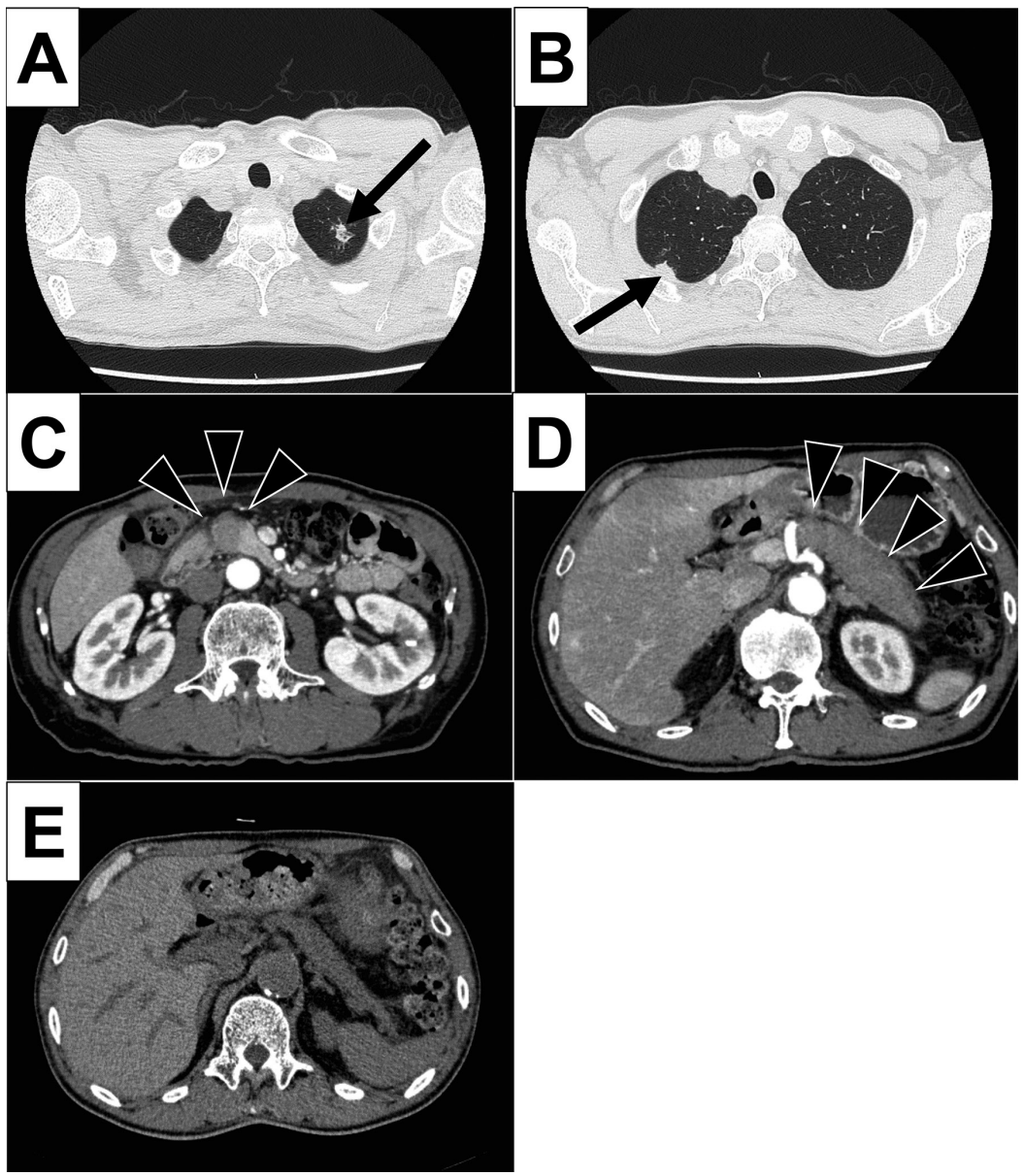

Fig. 1. Findings of computed tomography.
(A, B) Multiple lung nodules are detected in both lungs (arrows). (C) Low-density mass in the pancreatic head (arrowheads) and (D) swelling of the pancreatic body to the tail (arrowheads) are observed in contrast-enhanced CT (early phase). (E) Improvement in pancreatic swelling and disappearance of pancreatic mass formation are observed after glucocorticoid treatment.

however, the presence of non-specific inflammation was noted.

Antibiotic therapy was initiated for the fever and elevated C-reactive protein; however, no improvements were observed in the patient's inflammatory response. Two weeks after admission, serum creatinine level increased from 0.7 to 1.2 mg/dL, which indicated kidney dysfunction; additionally, myeloperoxidase (MPO) ANCA was positive (60 IU/mL). For more comprehensive management, he was transferred to our hospital. Vital signs revealed a temperature of 37.2°C and blood pressure of 95/63 mmHg. Purpuras were detected on the trunk and upper limbs. The abdomen was flat, soft, and non-tender. The pink palpebral conjunctiva indicated the absence of anemia. Furthermore, chest, including lung and heart sounds, and neurological examinations were unremarkable. Joint pain, upper respiratory tract and gastrointestinal symptoms, and edema were not observed.

Representative laboratory data during the patient's admission in our hospital are shown in Table 1. Urinary examination revealed proteinuria (0.75 g/gCr) and hematuria (> 100 per high-power field). Blood examination revealed elevated inflammatory response (white blood cell count, 17,280/μL; C-reactive protein level, 22.50 mg/dL) and kidney dysfunction (serum creatinine level, 1.38 mg/dL). Pancreatic enzyme levels were within normal range (amylase, 18 IU/L; lipase, 15 IU/mL). However, HbA1c increased to 8.3%, and the urinary C-peptide was 29.5 μg/day (reference range: 45-117 μg/day), suggesting a decrease in the insulin secretory ability. The MPO-ANCA level was high (75.7 U/mL). Proteinase 3 (PR3)-ANCA and anti-glomerular basement membrane (GBM) antibodies were negative, and IgG4 levels were within the normal range. Renal biopsy was performed the day after admission, prior to treatment initiation, which revealed pauci-immune necrotizing crescentic glomerulonephritis (Fig. 2A) and vasculitis

Table 1. Main laboratory data.

Urinalysis		(Reference range)	Unit	AST	46	(10-30)	U/L
Protein	2+	(−)		ALT	83	(5-40)	U/L
Protein/creatinine ratio	0.75	(< 0.3)	g/gCr	LDH	139	(110-220)	U/L
Occult blood	3+	(−)		ALP	720	(100-330)	U/L
Hematuria	> 100	(−)	/HPF	γGT	211	(5-35)	U/L
NAG	38	(0.3-11.5)	U/L	Total bilirubin	0.48	(0.2-1.2)	mg/dL
β2MG	8,736	(< 230)	μg/L	Amylase	18	(44-127)	U/L
Granular cast	+	(−)		Lipase	15	(13-55)	U/L
Waxy cast	+	(−)		CRP	22.5	(< 0.1)	mg/dL
Epithelial cast	+	(−)		HbA1c	8.3	(4.6-6.2)	%
White blood cell cast	+	(−)		Glucose	233	(75-110)	mg/dL
C-peptide	29.5	(45-117)	μg/day				
				ANA	-	(−)	
Blood analysis				C3	160	(70-150)	mg/dL
White blood cells	17,280	(3,500-8,000)	/μL	C4	14.5	(15-40)	mg/dL
Hemoglobin	11.3	(11.0-14.5)	g/dL	CH50	70.7	(30-50)	U/mL
Platelets	37.6×10^4	([13-35] $\times 10^4$)	/μL	MPO-ANCA	75.7	(< 3.5)	U/mL
Total protein	7.3	(6.7-8.3)	g/dL	PR3-ANCA	negative	(< 3.5)	U/mL
Albumin	2.1	(3.8-5.3)	g/dL	Anti-GBM Ab	negative	(< 3.0)	U/mL
Blood urea nitrogen	25.3	(8.0-22.0)	mg/dL	IgG4	60	(5-105)	mg/dL
Creatinine	1.38	(0.40-0.80)	mg/dL	Anti-GAD Ab	negative	(0-4.9)	U/mL
Uric acid	5.3	(2.6-7.0)	mg/dL	Anti-IA-2 Ab	negative	(0-0.3)	U/mL
Sodium	132	(135-147)	mEq/L	Insulin Ab	negative	(< 124)	nU/mL
Potassium	3.4	(3.5-5.0)	mEq/L				
Chloride	96	(98-108)	mEq/L				
Calcium	8.6	(8.5-10.2)	mg/dL				
iP	3.1	(2.5-4.5)	mg/dL				

Reference ranges are shown in round brackets.

ALP, alkaline phosphatase; ALT, alanine aminotransferase; ANA, anti-nuclear antibody; Anti-GAD Ab, anti-glutamic acid decarboxylase antibody; Anti-GBM Ab, anti-glomerular basement membrane antibody; Anti-IA-2 Ab, anti-insulinoma-associated protein-2 antibody; AST, aspartate aminotransferase; β2MG, beta-2-microglobulin; C3, complement 3; C4, complement 4, CH50, complement hemolytic activity assay; CRP, C-reactive protein; g/gCr, gram/gram creatinine; iP, inorganic phosphorus; LDH, lactate dehydrogenase; MPO-ANCA, myeloperoxidase-anti-neutrophil cytoplasmic antibody; NAG, N-acetyl-β-D-glucosaminidase; PR3-ANCA, proteinase 3-anti-neutrophil cytoplasmic antibody; γGT, gamma-glutamyl transpeptidase.

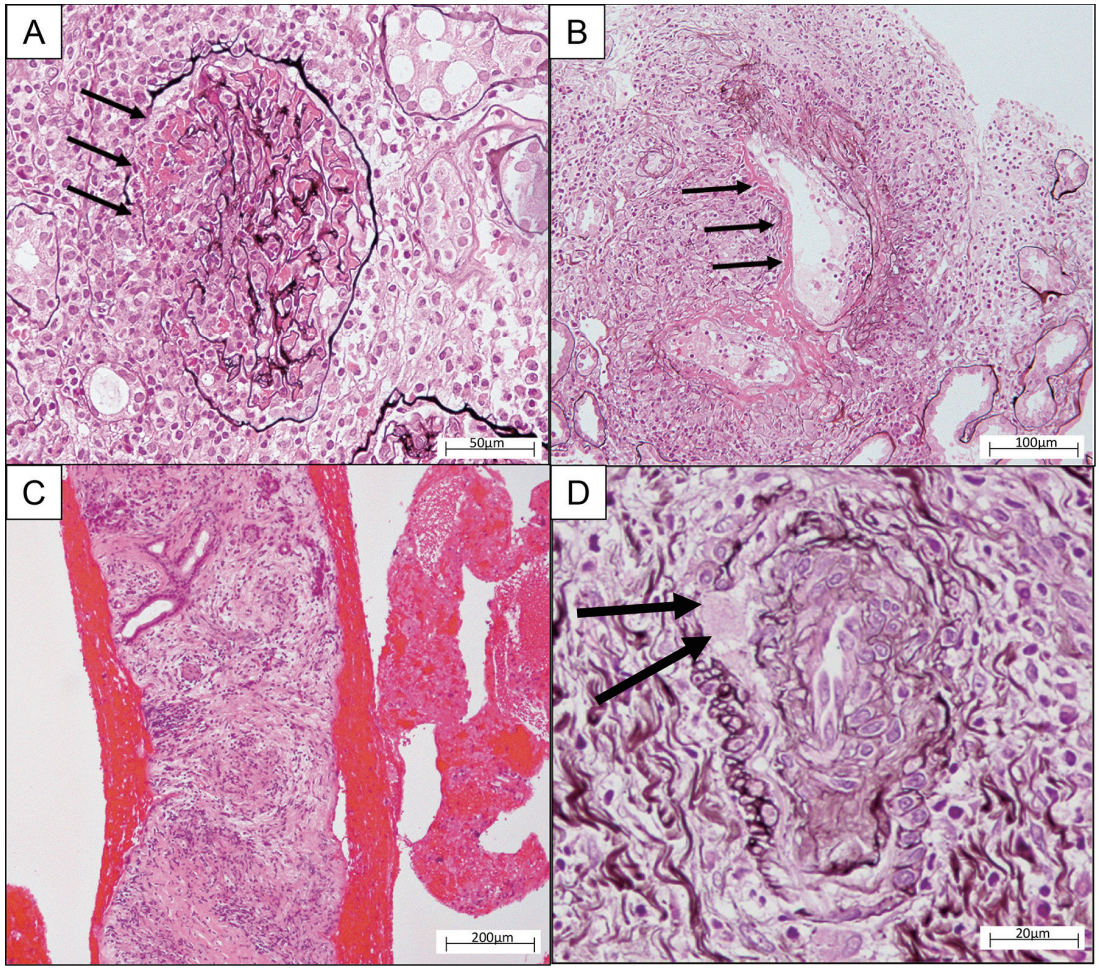

Fig. 2. Pathological findings of kidney biopsy specimens and pancreatic specimen obtained by endoscopic ultrasound-guided fine needle aspiration cytology (EUS-FNA).
(A) Necrotizing crescentic glomerulonephritis is detected in the glomeruli (arrows). (B) Fibrinoid necrosis is detected in the interlobular arteries of the kidney (arrows). (C) Inflammatory cell infiltration and fibrosis observed in the stroma of the pancreas. (D) Irregularly ruptured smooth muscle of vascular wall in small-size-arteries and plasma component exudation (arrows) observed in the blood vessel walls in pancreatic specimens obtained by EUS-FNA.

with fibrinoid necrosis in the small-sized arteries (Fig. 2B); these findings are consistent with AAV. We re-confirmed the finding of EUS-FNA performed at the previous hospital. Inflammatory cell infiltration and fibrosis were observed in the stroma of the pancreas, suggesting inflammation (Fig. 2C). Additionally, irregularly ruptured smooth muscles of small artery vascular walls and plasma component exudation were also observed (Fig. 2D). These findings are indicative of pancreatic vasculitis and are associated with pancreatic parenchymal injury. Regarding the disease activity of AAV, the Birmingham vasculitis active score (BVAS) of the patient was 20 (General 3, Cutaneous 2, Chest 3, Renal 12). Based on these results, he was diagnosed with MPO-ANCA-associated vasculitis and treated with glucocorticoid therapy, specifically, 1,000 mg methylprednisolone for three days, which was tapered to 50 mg/day oral prednisolone (PSL) (Fig. 3). After the initiation of glucocorticoid therapy, his fever and systemic inflammation improved. His serum creatinine level decreased to 1.0 mg/dL, and his proteinuria improved to 0.3 g/gCr. Additionally, improvements were observed in the patient's lung nodules, pancreatic swelling, and mass (Fig. 1E). The MPO-ANCA titer decreased. Insulin treatment had been initiated since the onset of vasculitis; however, a decrease in blood glucose level was observed and the amount of insulin administered could be reduced only after initiation of glucocorticoid treatment. Three weeks after the start of glucocorticoid treatment, insulin treatment was terminated, and the patient was switched to oral administration of repaglinide and linagliptin, and the HbA1c level improved to 6.3%. One month after the initiation of glucocorticoid treatment, the patient was discharged from our hospital. Subsequently, azathioprine was added, and the PSL dose was gradually reduced to 5 mg/day. There were no findings suggestive of recurrence, and the titer of MPO-ANCA remained negative. In addition, he was maintained on oral hypoglycemic agents to control diabetes mellitus.

Written informed consent was obtained from the

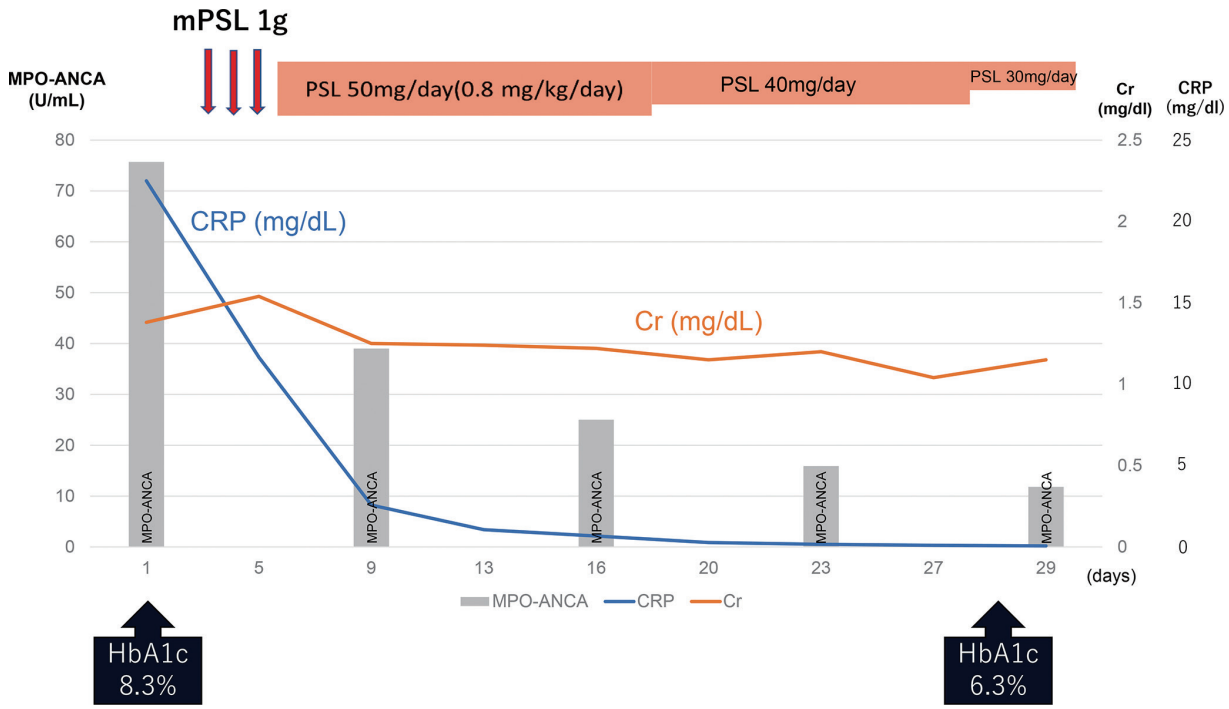

Fig. 3. Clinical course of serum creatinine, C-reactive protein, anti-neutrophil cytoplasmic antibody (ANCA) titers and hemoglobin A1c (HbA1c).
After initiating glucocorticoid therapy, the C-reactive protein level and myeloperoxidase-anti-neutrophil cytoplasmic antibody (MPO-ANCA) titer decreased rapidly, and the serum creatinine level also decreased gradually. Three weeks after the start of treatment, the HbA1c level improved from 8.3% to 6.3%.
Cr, creatinine; CRP, C-reactive protein; mPSL, methylprednisolone; PSL, prednisolone.

patient.

Discussion

We encountered a case of AAV with diabetes mellitus, possibly due to a pancreatic lesion. Previous case reports were reviewed to investigate the clinical course and characteristics of AAV with pancreatic lesions (Table 2).

Including the current case, 23 patients with AAV with pancreatic lesions have been previously reported (Kemp et al. 1990; Pezzilli et al. 1991; O'Neil et al. 1992; Stuckey and Smart 1992; Berney et al. 1997; Matsubayashi et al. 2001; Haraguchi et al. 2005; Reddy et al. 2007; Tinazzi et al. 2007; Joshipura et al. 2007; Abu-Hilal et al. 2008; Chawla et al. 2011; Hamilton et al. 2011; Valerieva et al. 2013; Kontis et al. 2014; De Bie et al. 2015; Iida et al. 2015, 2016; Suzuki et al. 2019; Sowida 2019; Alesaeidi et al. 2021). Most of the patients were elderly with a median age of 60 years, ranging from 20-84 years. The male-to-female ratio was 12:11, which is similar to that in the general population of patients with AAV. Regarding ANCA classification, eight patients presented with MPO-ANCA, nine with PR3-ANCA, four were negative for ANCA, one was positive with unknown type, and one was unknown. Regarding diagnosis, 16 patients were diagnosed with GPA, five with MPA, and two with AAV; however, no cases of EGPA were reported. Among 23 patients, 15 developed renal complications, 14 developed lung lesions, and seven developed nasal and sinus lesions. Additionally, three patients had only their pancreas affected. The average BVAS was 21.2 (range, 9-41), which is close to our patient's score of 20. Most patients were managed with glucocorticoids and cyclophosphamide. Regarding the prognosis of the 23 patients, the renal survival of patients with AAV with pancreatic lesions was 87% (three patients required maintenance hemodialysis), whereas the survival rate was 78% (five patients died). In contrast, the renal survival and survival rates (one year after initiation of treatment) of patients with AAV were 78% and 79%, respectively (Yamagata et al. 2012). Therefore, the prognosis of typical patients with AAV and patients with AAV and pancreatic lesions may be similar.

Among the patients with AAV and pancreatic lesions, 11 patients (including the current patient) presented with signs indicative of diabetes mellitus, including hyperglycemia and elevated HbA1c levels. The current patient developed diabetes mellitus at the onset of AAV; additionally, his blood glucose levels improved despite glucocorticoid therapy. In addition, EUS-FNA findings were indicative of pancreatic vasculitis and parenchymal inflammation. Vascular lesions in the pancreas occur in small arteries and are similar to vascular lesions in the kidney. Therefore, vascular lesions in similar-sized arteries are expected to develop in organs other than the kidney. With regard to the mechanism of development of diabetes mellitus with

Table 2. Summary of reported cases of anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis with pancreatic lesions.

Authors	year	Age, sex	Primary symptoms	BVAS	ANCA type	Diagnosis	Pancreatic enzyme	Pancreatic lesion Imaging	Histological findings of pancreatic vasculitis	Other organ disorders	Treatment	Outcome	Diabetes, BS
Kemp et al.	1990	57, M	epigastric pain, fever	14	ND	GPA(WG)	AMY 4,651	CT: pseudocyst, diffuse swelling	ND	lung	mPSL, PSL, CY	improved	ND
Pezzilli et al.	1991	66, M	fever, epistaxis, hearing loss, abdominal discomfort	20	MPO	GPA(WG)	AMY 277 Lipase 256	US: normal	ND	nose, ear, skin, lung, liver, kidney	CY, mPSL	improved	hyperglycemia (192g/L)
O'Neil et al.	1992	62, M	painless jaundice	35	+	GPA(WG)	AMY 26 (normal)	CT: 3cm mass ERCP: CBD stenosis	CT directed fine-needle aspiration → nondiagnostic	kidney, nose, lung, ear, heart	PSL, CY	improved	Type 1 diabetes
Stuckey et al.	1992	45, M	epigastric pain, jaundice, hematuria asthenia, vomiting,	22	-	GPA(WG)	AMY 55 (normal)	CT: enlargement, sporadic low density lesions ERCP: CBD stenosis	ND	lung, parotid grand	CY, PSL	improved	Insulin dependent diabetes
Berney et al.	1997	32, M	weight loss, oliguria	20	PR3(C)	MPA	Raised of AMY and Lipase	CT: edematous p ancreatitis	ND	kidney	PSL, CY, ope	Improved (ESRD)	ND
Matsubayashi et al.	2001	65, M	Left abdominal pain, constipation	37	PR3	GPA	Trypsin 550 Elastase-I 440 AMY 34 (normal)	CT: enlargement, sporadic low density lesions	+ (autopsy)	kidney, lung, spleen	No	died	HbA1c 9.2%
Haraguchi et al.	2005	84, F	edema, fever, anorexia	20	MPO	MPA	AMY 130	CT: normal	+ (autopsy)	kidney, lung	mPSL, PSL	died	HbA1c 6.9% BS 103 mg/dl
Reddy et al.	2007	34, F	sinusitis, epistaxis	41	PR3(C)	GPA	AMY 273 Lipase 74	CT: 2.5cm mass MRI: inhomogeneous mass	ND	kidney, lung, nose, ear,skin, thyroid, gastrointestinal tract	mPSL, PSL, CY, AZA	improved (ESRD)	ND
Tinazzi et al.	2007	48, F	epigastric pain	9	-	GPA	ND	US: 2cm hypoechoic mass MRI: 2cm mass MRCP: obstruction of MPD	+ (ope)	No	opePSL, CY, MTX	improved	ND
Joshipura et al.	2007	47, M	epigastric pain	22	PR3	GPA	AMY 874 Lipase 1294	US: bulky CT: edematous	ND	nose, eye, kidney	PSL, CY	improved	BS 256mg/dl
Abu-Hilal et al.	2008	20, F	epigastric pain, nausea, loss of appetite	26	PR3	GPA	AMY 20 (normal)	CT: edematous	ND	kidney, lung, skin, intestinal tract	PSL, CY	died	ND
Chawla et al.	2011	60, F	epigastric pain, nausea	24	PR3	GPA	Lipase 1,316	CT: diffiusely edematous, hypoattenuated lesion EUS: hypoechoic lesion	EUS-FNA → nondiagnostic	kidney, lung, heart, pituitary dysfunction	mPSL, PSL, CY, AZA	improved	ND
Hamilton et al.	2011	78, F	arthralgia	20	PR3	GPA	ND	CT: a bulky mass	ND	lung, kidney	CY, PSL	improved	ND
Valerieva et al.	2013	62, F	epigastric pain, nausea, fever, headache	17	PR3	GPA	normal (AMY, Lipase)	US: enlarged, hypoechoic CT: edema MRI: 3cm mass	+ (ope)	ear, spleen	ope, PSL, AZA	improved	BS 148mg/dl
Kontis et al.	2014	57, M	abdominal pain, nausea	9	-	GPA	ND	CT: a bulky soft tissue density mass	+ (ope)	No	ope	improved	ND
Kontis et al.	2014	68, F	abdominal pain, weight loss, jaundice	12	MPO	GPA	ND	CT, MRI: mass	+ (ope)	No	ope	improved	diagnosed with type 2diabetes three months prior to onset
De Bie et al.	2015	57, M	fatigue, weight loss, painless jaundice	20	MPO	AAV	Normal (AMY)	ERCP: CBD stenosis CT: 25 mm mass	ND	kidney, nose, eye	CY, PSL, PE	improved (ESRD)	ND

Iida et al.	2015	64, F	edema		12	MPO	MPA	AMY 543 Lipase 460 Elastase 1857	CT: enlarged	ND	kidney	mPSL, PSL	improved	ND
Iida et al.	2016	72, M	weight loss		20	MPO	MPA	AMY 59 (normal) Lipase 53 (normal) Trypsin 723	CT: enlargement, multiple hypoattenuated lesions	EUS-FNA → nondiagnostic	kidney, lung	CY, PSL	died	HbA1c 10.8%
Suzuki et al.	2019	71, M	right lower abdominal pain		30	MPO	MPA	P-AMY 29 Lipase 294	CT: swelling, tumor, MPD dilation MRI: high intensity	EUS-FNA → unsuccessful	lung, kidney	mPSL, PSL, CY	improved	BS 147 mg/dl HbA1c 6.2%
Sowida	2019	22, F	mouth ulcers, fever, dry cough, weight loss		14	PR3	GPA	ND	CT: low density change MRI: fluid-filled cyst	ND	lung, nose, mouth, ear	mPSL, PSL, AZA, RTX	improved	ND
Alesaeidi et al.	2021	38, F	swelling, pain, nasal congestion		24	-	GPA	ND	CT: 26 × 23 mm mass MRCP: CBD dilation	EUS-FNA → nondiagnostic	nose	PSL, CY	died	ND
Current case		67, M	fever, cough, weight loss		20	MPO	AAV	AMY 18 (normal) Lipase 15 (normal)	CT: low density mass, swelling MRI: diffuse swelling	+ (EUS-FNA)	lung, kidney	PSL	improved	HbA1c 8.3%

AAV, ANCA-associated vasculitis; AMY, amylase; AZA, azathioprine; BS, blood sugar; BVAS, Birmingham vasculitis active score; CBD, common bile duct; CT, computed tomography; CY, cyclophosphamide; ERCP, endoscopic retrograde cholangiopancreatography; ESRD, end stage renal disease; EUS-FNA, endoscopic ultrasound-guided fine needle aspiration; F, female; GPA, granulomatosis with polyangiitis; M, male; MPA, microscopic polyangiitis; MPD, main pancreatic duct; MPO, myeloperoxidase; mPSL, methylpredonisolone; MRCP, magnetic resonance cholangiopancreatography; MRI, magnetic resonance imaging; MTX, methotrexate; ND, No data; ope, operation; P-AMY, pancreatic amylase; PR3, proteinase 3; PSL, predonisolone; RTX, rituximab; US, ultra sound; WG, Wegener's granulomatosis.

ANCA, decreased urinary C-peptide levels are suggestive of decreased insulin secretion ability, which may result in impaired glycemic control. Unfortunately, data regarding insulin secretory ability after treatment for AAV or exocrine pancreatic function were not collected. Since inflammatory cytokines, such as interleukin-6 (IL-6) and/or tumor-necrosis factor α, are known to induce insulin resistance and increase blood glucose levels, the impaired glycemic control in the current patient may also be attributed to insulin resistance due to systemic inflammation. We measured the IL-6 level in the serum obtained at admission, which was within the normal range despite the severe systemic inflammatory response. However, this serum sample had been in storage for an extended period, which may have influenced the result. We cannot elucidate the definitive mechanism of diabetes development via ANCA; however, we suspect that both decreased insulin secretion ability and insulin resistance due to the AAV may have been involved, given the pathological findings of inflammation in the pancreatic parenchyma. A previous patient with AAV developed type 1 diabetes mellitus (Nederstigt et al. 2019). It is known that patients with type 1 diabetes mellitus often have autoimmune diseases (Nederstigt et al. 2019); therefore, the association between AAV and type 1 diabetes mellitus should be considered. However, a representative auto-antibody associated with type 1 diabetes mellitus was not found in our patient. Pancreatic vasculitis-induced diabetes mellitus is extremely rare; therefore, more case reports are needed to clarify the clinical course and relationship between diabetes mellitus and AAV.

In addition, it is important to distinguish pancreatic lesions of AAV from pancreatic tumors because pancreatic lesions of AAV often form a mass in the pancreas. However, it is difficult to preoperatively diagnose pancreatic lesions; therefore, four patients with AAV-associated pancreatic lesions required surgery. Our patient similarly presented with a pancreatic mass. However, surgery was not indicated because MPO-ANCA-related vasculitis was diagnosed using EUS-FNA, and such patients are rare. CT-guided FNA or EUS-FNA of pancreatic masses was attempted in five patients with AAV; however, none led to a successful diagnosis (O'Neil et al. 1992; Chawla et al. 2011; Iida et al. 2016; Suzuki et al. 2019; Alesaeidi et al. 2021). Therefore, it is difficult to diagnose pancreatic lesions using methods other than surgery. In addition, three patients presented with pancreatic lesions only with no other organ involvement. In diagnosing pancreatic masses, patients with AAV should be carefully diagnosed, and pancreatic vasculitis due to AAV is an important diagnostic consideration. In our patient, tissue was obtained from the swollen pancreatic tail, since the aorta led to difficulties in reaching the pancreatic head. The pathological findings of the pancreas were indicative of vasculitis-induced inflammation; additionally, glucocorticoid therapy caused resolution of the pancreatic head mass. These findings were consistent with inflammatory cell infiltration and/or

granulomatous lesions due to AAV.

In conclusion, pancreatic lesions due to AAV are rare; therefore, there is a need for more case reports of this condition. Glucocorticoid therapy is a known aggravator of glycemic control; therefore, clinicians are often hesitant to initiate glucocorticoid therapy in patients with diabetes mellitus. However, the use of glucocorticoid therapy in patients with pancreatic vasculitis-induced diabetes mellitus improves glycemic control. In addition, mass formation in patients with AAV and pancreatic vasculitis necessitates careful consideration when conducting a differential diagnosis.

Conflict of Interest

The authors declare no conflict of interest.

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