CASE REPORT

Antimitochondrial antibodies-positive myositis accompanied by cardiac involvement

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SUMMARY

We report a 55-year-old man who experienced proximal muscle weakness accompanied by the atrial flutter (AFL) with 1:1 conduction. Detailed examination revealed elevated antimitochondrial antibodies (AMA) and creatine kinase (CK). AFL was converted to sinus rhythm by cardioversion. He was diagnosed as AMA-positive myositis-associated AFL and was treated by prednisolone. Although his muscle weakness and CK level improved, AFL with 1:1 conduction reappeared. Therefore, radiofrequency catheter ablation (RFCA) was needed to treat the AFL, resulting in maintenance of sinus rhythm. This case report describes cardiac involvement in a patient with AMA-positive myositis.

BACKGROUND

It is known that cardiac involvement can occur with inflammatory myopathy, and this combination has been reported in antimitochondrial antibodies (AMA)-negative cases,^{1–11} and AMA-positive cases.^{12–20}

AMA-positive myositis was first reported in 1973 by Sherlock and Scheuer²¹ as an inflammatory myopathy.

According to recent reports,^{12–22} AMA-positive myositis cases are more likely to be complicated by

cardiac involvement than AMA-negative myositis cases.

Therefore, cardiologists must recognise the existence of this condition.

There remain many unknown aspects, such as the characteristic image findings associated with cardiac involvement caused by this disease, the response of cardiac involvement to myositis treatment, and prognosis, which need to be addressed in future studies in a larger number of patients.

CASE PRESENTATION

The patient was a 55-year-old man who had no relevant medical or family history. He had experienced difficulty to abdominal muscle exercise since January 2015, but did not undergo an examination at a medical facility. The results of a 12-lead ECG performed during health check-ups in 2013 and 2014 revealed a normal sinus rhythm and intraventricular conduction disturbance (IVCD). At a health check-up in August 2015, the same ECG findings were noted, and he was instructed to undergo an examination at a medical facility. In December 2015, he was examined at the other hospital, where the ECG revealed a heart rate of ~120 bpm, a common atrial flutter (AFL) with 2:1 conduction ratio, left axis deviation and IVCD (QRS width 132 ms; figure 1A). Holter ECG revealed that the

Figure 1 (A) The ECG on admission revealed a heart rate of ~120 bpm, a common AFL with 2:1 conduction ratio, left axis deviation and IVCD (QRS width 132 ms). (B) The ECG after cardioversion revealed normal sinus rhythm, a heart rate of 60 bpm, left axis deviation and IVCD (QRS width 135 ms). (B, C) Holter ECG revealed that the basic rhythm was AFL rhythm with 2:1 conduction, and the 1:1 conduction was observed mainly during exertion for a few minutes. AFL, atrial flutter; IVCD, intraventricular conduction disturbance.

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Figure 2 (A, B) The TTE revealed diffuse mildly decreased left ventricular wall motion approximately at LVEF 52%, and no other significant abnormal findings, with wall thickness and left cavity size within the normal range. (C) The biopsy of the quadriceps femoris muscle revealed muscle cell degeneration with mild mononuclear cell infiltrates indicated inflammatory myopathy (H&E stain). (D) A liver biopsy revealed chronic non-suppurative destructive cholangitis was noted, indicated primary biliary cirrhosis (H&E stain). (A) systolic phase, (B) diastolic phase. LVEF, left ventricular ejection fraction; TTE, transthoracic echocardiography.

Figure 3 (A, B) Stenosis of coronary artery was not found in coronary angiography. (A) Left coronary artery, (B) right coronary artery.

basic rhythm was an AFL rhythm with 2:1 conduction (figure 1C), and for a few minutes, the 1:1 conduction was observed mainly during exertion (figure 1D).

Therefore, the patient was hospitalised in December 2015 to undergo detailed testing and treatment. Physical findings on hospitalisation included blood pressure of 136/92 mm Hg and heart rate of 124 bpm, which was regular with no abnormal heart or respiratory sounds. The patient did not suffer from any muscular pain. A manual muscle testing (MMT) 4 showed muscle weakness symmetrically on the left and right sides of the neck region, trunk and proximal muscles of the four limbs. Blood testing revealed mildly elevated aspartate aminotransferase/alanine aminotransferase and brain natriuretic peptide at 67/58 IU/L and 118.6 pg/mL, respectively. Creatine kinase (CK) was elevated to 2188 IU/L, with muscle-type CK elevation at 90%. Chest X-rays revealed no cardiac dilation, with a cardiothoracic ratio of 48%. Moreover, there were no findings indicating congestion or pleural effusion. The ECG revealed no marked changes from the patient's state at the original hospital (figure 1A).

Although left ventricular wall motion diffusely and mildly decreased at left ventricular ejection fraction (LVEF) 52%, transthoracic echocardiography (TTE) indicated no other significantly abnormal findings including wall thickness and valvular function (figure 2A, B).

As the proximal muscle strength had significantly decreased and a muscle-type fraction-dominant CK elevation was noted, we suspected a muscular disease, such as muscular dystrophy, polymyositis or dermatomyositis. Therefore, we performed detailed examination for muscular disease. Results indicated that the patient was negative for anti-Jo-1 antibodies, and anti-aminoacyl tRNA synthetase antibodies were within the standard range at 7.9 U/mL (standard value <25 U/mL). Meanwhile, the antinuclear antibody results showed that the granular type was 640-fold higher, and the results were positive for AMA at 183 U/mL (standard value <7 U/mL). Needle electromyogram performed on the triceps brachii and quadriceps femoris muscles revealed myogenic changes. The results of the muscle biopsy of the quadriceps femoris muscles revealed muscle cell degeneration with mild mononuclear cell infiltration, which indicated inflammatory myopathy (figure 2C). These findings suggested that our patient probably had AMA-positive myositis.

Blood tests revealed that liver synthesis function was maintained and no morphological findings indicating liver cirrhosis were noted. As AMA results were positive, a liver biopsy was performed. Chronic non-suppurative destructive cholangitis was noted (figure 2D), and the patient was also found to have primary biliary cirrhosis (PBC). The stenosis of coronary artery was not found in coronary angiography (figure 3A, B). Gadolinium contrast-enhanced cardiac MRI (CMRI) revealed that the patient

Figure 4 (A) Gadolinium contrast-enhanced CMRI performed before starting PSL administration revealed that the patient was positive for LGE in the middle layer of the almost entire circumference wall. (B) Gadolinium contrast-enhanced CMRI performed 8 weeks after starting PSL administration revealed no marked changes in LGE. CMRI, cardiac MRI; LGE, late gadolinium enhancement; PSL, prednisolone.

Table 1 The literature of the AMA-positive myositis accompanied by cardiac involvement

N	Age	Sex	Arrhythmia	LVD/CHF	Tx	CC of CI	ATx	PBC	Reference
1	54	F	AF	+	—	Arrhythmia worsening	—	+	(12)
2	49	F	AF, PSVT	—	ND	ND	—	+	
3	48	M	AVB	+	PSL	Arrhythmia worsening	—	+	
4	54	F	NSVT	+	—	Arrhythmia worsening	PM	+	
5	59	M	AF	+	AZP	Worsening (—)	CA	+	
6	58	F	AF	+	PSL	No change	—	—	
7	46	F	AF	—	PSL	No change	—	—	
8	54	M	CRBBB, PVC	+	PSL	No change	CA	—	
9	55	M	—	+	PSL	No change	—	+	(13)
10	42	M	AF	—	ND	ND	ND	+	(14)
11	58	F	AF	+	PSL	ND	—	+	(15)
12	44	F	AFL	—	PSL	ND	—	+	(16)
13	58	F	NI	—	ND	ND	ND	+	
14	37	F	PAC, PVC	+	PSL	No change	—	NE	(17)
15	51	M	AF, sustained VT	+	PSL	Arrhythmia worsening	ICD	+	(18)
16	65	F	AVB	+	PSL	Improved LVEF	—	+	(19)
17	48	F	AF	+	ND	ND	ND	+	(20)

AF, atrial fibrillation; ATx, additional therapy; AVB, atrioventricular block; AZP, azathioprine; CA, catheter ablation; CC of CI, clinical course of cardiac involvement; CRBBB, complete right bundle branch block; HF, heart failure; ICD, implantable cardioverter defibrillator; LVD/CHF, left ventricular dysfunction/congestive HF; ND, no data; NE, not examined; NI, no information; PM, pace maker; PSVT, paroxysmal supraventricular tachycardia; PVC, premature ventricular contraction; Tx, therapy; VT, ventricular tachycardia.

was positive for late gadolinium enhancement (LGE) in the middle layer of almost the entire circumference wall (figure 4A). Regarding the cause of the mildly decreased LVEF, we considered the possibility of tachycardia-induced cardiomyopathy. As the patient was positive for LGE, we believed that he might have cardiomyopathy as a background factor that could cause LGE positivity and arrhythmia. Therefore, we performed tests to investigate the possibility of such diseases, including dilated cardiomyopathy, hypertrophic cardiomyopathy, sarcoidosis, amyloidosis and drug-induced cardiomyopathy. However, no findings strongly suggestive of any of these diseases were detected. Based on these findings and the fact that AMA-positive myositis was observed, we believed that the cause of the cardiac involvement including AFL, IVCD and LGE positivity was cardiomyopathy associated with AMA-positive myositis.

Holter ECG performed before hospitalisation indicated sporadic AFL with 1:1 conduction (figure 1C). After hospitalisation, AFL was noted with a basic rhythm of ~120 bpm. As this was not initially detected following hospitalisation, the dosage of bisoprolol was gradually increased to 5 mg once daily.

Subsequently, the patient's AFL conduction ratio temporarily decreased, and the pulse rate became stable at ~60 bpm. However, on hospital day 33, AFL with 1:1 conduction ratio accompanied by presyncope occurred occasionally; thus, amiodarone was initiated at 200 mg twice daily on hospital day 34. Moreover, a 50J (biphasic) cardioversion was performed on hospital day 37. Sinus rhythm was then achieved (figure 1B). After 3 days of steroid pulse therapy with 1000 mg/day of methylprednisolone from hospital day 46 to treat the AMA-positive myositis, oral administration of 50 mg/day of prednisolone (PSL; 1 mg/kg) was initiated. On hospital day 48, amiodarone was initiated at 100 mg twice daily.

Thereafter, improvement to MMT 5 was observed in muscle strength at the sites where muscle weakness had been observed; furthermore, CK decreased to ~300 IU/L, and sinus rhythm was maintained. Gadolinium contrast-enhanced CMRI performed 8 weeks after starting administration of PSL revealed no marked changes in LGE (figure 4B). TTE performed on hospital day 60 (24 days after cardioversion and 14 days after starting PSL) revealed that systolic function had clearly improved to LVEF 62%.

AFL accompanied with 1:1 conduction reappeared on hospital day 91 and resulted in presyncope. At this point, blood concentration of amiodarone and mono-N-desethylamiodarone were considered sufficient at 566 and 673 ng/mL, respectively. Despite this, the appearance of AFL of 1:1 conduction ratio could not be inhibited; therefore, catheter ablation was performed for common AFL on hospital day 99. Results for the ventricular tachycardia induction tests (which were simultaneously performed) were negative.

Thereafter, amiodarone was discontinued but the once daily administration of 5 mg bisoprolol was continued. The patient's sinus rhythm was maintained, and LVEF remained within the recovered level in the range of 62% according to TTE. PSL was carefully reduced to 30 mg/day, and the patient was discharged on hospital day 120 when his decreased muscle strength had improved to approximately MMT 5.

OUTCOME AND FOLLOW-UP

Thereafter, MMT and LVEF were maintained while improved, and AFL did not reappear 6 months after discharge, even if dosage of PSL was reduced to the 10 mg per day gradually. On the other hand, improvement of IVCD was not observed.

DISCUSSION

AMAs are typical autoantibodies noted in cases of PBC and are known to appear in ~95% of PBC cases,^{23 24} AMA-positive myositis was first reported in 1973 by Sherlock and colleagues as a disease involving PBC and inflammatory myopathy.²³ In 1974, Uhl *et al*²⁵ first reported myositis accompanied with PBC in detail. Subsequently, it has been recognised that myositis could occur even without the comorbidity of PBC, as cases of myositis that were positive for AMA but did not exhibit PBC were reported.^{26 27} AMA-positive myositis occurs at a male-to-female ratio of 9:15–1:6 with age of onset ranging from 30 to 70 years, and most cases occur in middle-aged women.^{12 22} It is known that cardiac involvement can occur with inflammatory myopathy, and this combination has been reported in both AMA-negative cases^{1–11} and, similar to our patient, AMA-positive cases^{12–20} (table 1). Thus, in case of inflammatory myositis, cardiac involvement as a complication should be considered.

In our patient LVEF decreased (52%) and AFL accompanying a sporadic 1:1 conduction ratio was noted. The cause of this cardiac dysfunction was not coronary stenosis, and ischaemic heart disease was also ruled out. As the patient had no history of hypertension and exhibited no left ventricular hypertrophy, hypertensive heart disease was also ruled out. TTE findings were not suggestive of hypertrophic or dilated cardiomyopathy. Moreover, there were no findings indicative of sarcoidosis, which was ruled out according to the Diagnostic Standard and Guideline for Sarcoidosis, 2015.²⁸ Heart amyloidosis was ruled out as TTE did not indicate any ventricular wall hypertrophy; furthermore, no M protein was detected, and systemic findings were not indicative of the disease. Drug-induced cardiomyopathy was also ruled out as the patient had no history of drug use that could cause cardiomyopathy. Results of the ECG on admission revealed common AFL and tachycardia with a heart rate of ~120 bpm. On hospital day 37, AFL was converted to normal sinus rhythm and the heart rate decreased to ~60 bpm by cardioversion. TTE was performed 24 days later when cardioversion indicated that the LVEF had improved to ~62%. This suggested that tachycardia-induced cardiomyopathy may have been involved in the decreased cardiac function. However, because LGE of CMRI is usually unobserved in tachycardia-induced

cardiomyopathy,^{29 30} we cannot explain the LGE positivity in our patient. In addition, as IVCD was noted 3 years earlier, we presume that the patient may have experienced cardiomyopathy that caused latent myocardial damage. Our patient was diagnosed with AMA-positive myositis, and we cannot conclude whether it was responsible for the noted left ventricular dysfunction. However, we believe at least the AMA-positive myositis was a contributing factor to the development of cardiomyopathy that could cause LGE positivity and IVCD.

Various forms of complicating arrhythmia have been reported for this disease;^{12 14 15 17–20} however, the mechanism underlying the onset of arrhythmia is currently unknown. Nevertheless, we observed positive LGE on CMRI examination in the present case, indicating that the arrhythmia may have been caused by myocardial damage. Therefore, we believe that the arrhythmia in the present case was not an incidental finding.

We were unable to perform myocardial biopsy as the patient did not provide consent. There is only one previously reported case of this disease in which myocardial biopsy was performed.¹⁹ According to this report, the biopsy revealed interstitial fibrosis. This finding appears to be consistent with the CMRI in the present case. However, it is currently unknown whether there are specific biopsy findings leading to the present diagnosis.

As there are a limited number of reports of myocardial biopsies for this disease, further studies are necessary.

The findings in our patient were consistent with those of previous reports,¹² as proximal muscle weakness had improved following PSL induction. However, as AFL recurred after cardioversion despite treatment with PSL, it was necessary to perform radiofrequency catheter ablation (RFCA). No improvement in IVCD and LGE was noted. While LVEF was previously ~52%, it had improved to 62% by hospital day 60 (24 days after cardioversion and 14 days after starting PSL). However, it is unclear whether this improvement was due to PSL, as it may have resulted from an improvement in the tachycardia-induced cardiomyopathy. While improvement in cardiac involvement with recovery of muscle strength induced by PSL has been reported,¹⁹ there have also been some reports of cases in which cardiac involvement remained unchanged,^{13 17} and cases in which arrhythmia did not improve and actually worsened, requiring a pacemaker and catheter ablation,¹² or implantable cardioverter defibrillator implantation.¹⁸ From these reports,^{12 13 17–19} and the present case, in this disease, cardiac involvement might be hard to respond to PSL than myositis.

However, as there is only a limited amount of data available, several points remain unknown regarding whether cardiac involvement also improves as does muscle strength. Further investigations should be conducted based on data from a larger number of cases. Moreover, there are no past reports investigating IVCD and LGE, making our patient the first such case in the world.

Considering the literature of AMA-positive myositis accompanied with cardiac involvement,^{12 13–20} (table 1) and this case, we believe that there are three points regarding AMA-positive myositis in cardiovascular medicine that should be recognised.

The first point is that AMA-positive myositis cases are more likely to be complicated by cardiac involvement than AMA-negative myositis cases. In an investigation of 24 AMA-positive myositis cases out of a series of 212 myositis cases, Maeda *et al*¹² found that cardiomyopathy was significantly ($p<0.005$) more common in AMA-positive cases (8/24; 33%) than in AMA-negative cases (17/188; 9%). These findings suggest that when myositis is suspected in an AMA-positive

case, the presence of cardiac involvement should be proactively investigated. Furthermore, in AMA-positive myositis cases, those with PBC are significantly more likely to exhibit cardiac involvement than cases that do not have PBC (71% vs 18%, $p < 0.05$).¹² Our patient also exhibited AMA-positive myositis and PBC as well as cardiac complications. Thus, it appears that when PBC is noted in a case of AMA-positive myositis, there is a high likelihood of cardiac involvement.

The second point is the fact that this disease may be diagnosed as a result of the appearance of arrhythmia or symptoms of heart failure, rather than myositis. There have been reports of cases diagnosed as a result of atrial fibrillation and the detection of heart failure symptoms,²⁰ and Maeda *et al*¹² reported that 2 out of 24 (8%) AMA-positive cases of myositis were first detected with arrhythmia. Our patient was hospitalised as a result of an AFL noted on his ECG findings, and he was subsequently diagnosed following an AMA evaluation performed due to mildly abnormal AST/ALT results noted on hospitalisation. This suggests that when cases of cardiac involvement of unknown origin are accompanied with liver function and/or myositis symptoms, the differential diagnosis for AMA-positive myositis should be considered.

The third point is the fact that cardiac involvement may appear and/or exacerbation if the condition is left untreated. In the 15 out of 24 cases,¹² in which the course of treatment was followed up, 11 cases were treated with PSL and 1 case with azathioprine. In 11 of these cases, CK normalised within 3 months and in 6 of these cases, muscle strength improved within the observation period. However, follow-up observations of the three cases that refused treatment revealed that two cases had newly developed arrhythmia during the observation period, one of whom had a pacemaker implanted. Thus, one must be cautious to the fact that even if cardiac involvement is not apparent at first, there are cases in which the condition may become exacerbated, with arrhythmia reappearing during the period of non-treatment.¹²

In conclusion, cardiac involvement of AMA-positive myositis does not appear to be widely recognised in the field of cardiovascular medicine. However, as in the case of our patient, this disease may be diagnosed as a result of the appearance of cardiovascular disease (eg, arrhythmia), and AMA-positive myositis should be more carefully observed with respect to cardiac involvement than AMA-negative myositis. As cardiologists, we must recognise the existence of this condition. In addition, there remain many unknown aspects, such as the characteristic image findings associated with the cardiac involvement caused by this

disease, the response of cardiac involvement to myositis treatment, and prognosis, which need to be addressed in future studies in a larger number of patients.

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Learning points

- ▶ Antimitochondrial antibodies (AMA)-positive myositis cases are more likely to be complicated by cardiac involvement than AMA-negative myositis cases.
- ▶ This disease may be diagnosed as a result of the appearance of arrhythmia or symptoms of heart failure, rather than myositis.
- ▶ Cardiac involvement of this disease may appear and/or exacerbation if the condition is left untreated.
- ▶ If cases of cardiac involvement of unknown origin are accompanied with liver function and/or myositis symptoms, differential diagnosis for AMA-positive myositis should be considered.

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