

Case Report

Transient Atrioventricular Conduction Disturbances in a Case of Severe Fever with Thrombocytopenia Syndrome

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A 79-year-old woman with severe fever with thrombocytopenia syndrome (SFTS) developed progressive atrioventricular (AV) conduction disturbances during hospitalization. On hospital day 6, first-degree AV block progressed to transient complete AV block, causing syncope and a 15-sec asystolic pause. Echocardiography showed preserved ventricular function, and cardiac magnetic resonance imaging did not support clinically overt myocarditis. A temporary transvenous pacemaker was inserted, and AV conduction recovered spontaneously; pacing was discontinued by day 9 without permanent pacemaker implantation. This case suggests that SFTS can cause reversible high-degree AV block and may warrant continuous twelve-lead electrocardiography (ECG) monitoring and timely temporary pacing.

Key words: severe fever with thrombocytopenia syndrome, atrioventricular block, bradyarrhythmia, temporary pacing, myocarditis

Severe fever with thrombocytopenia syndrome (SFTS) is an emerging tick-borne viral infection caused by the SFTS virus and was first described in China in 2011 [1]. Since then, cases have been reported across East Asia, including in Japan and South Korea. SFTS is characterized by fever, cytopenias, gastrointestinal symptoms, and multiorgan involvement, and it is associated with substantial mortality [2]. In addition to systemic manifestations, cardiac complications have been increasingly recognized. Arrhythmias and other electrocardiographic abnormalities have been reported as markers of disease severity and poor prognosis in SFTS [3], with sinus bradycardia and atrial tachyarrhythmias most commonly described [4].

However, atrioventricular (AV) block appears to be rare. One case series reported only two cases among 115 patients, without describing their severity or clinical course, and detailed clinical descriptions of high-degree AV block remain limited [5]. We report a case of SFTS complicated by progressive AV conduction disturbances culminating in transient complete AV block requiring temporary pacing, without findings supporting clinically overt myocarditis.

Case Report

In May 2025, a 79-year-old woman with diabetes mellitus and dyslipidemia presented to her primary care physician with fever that had begun the day before. The

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patient had no prior history of syncope, bradycardia, or conduction disease. She was not taking any atrioventricular nodal blocking medications, including beta-blockers, non-dihydropyridine calcium channel blockers, digoxin, or antiarrhythmic agents. Initial laboratory tests showed a white blood cell count of 5,000/ μ l and platelet count of 174,000/ μ l. Two days later, persistent fever prompted re-evaluation; leukopenia, thrombocytopenia, and elevated aminotransferase levels were noted, and she was referred for admission because SFTS was suspected in the context of reported contact with a pet cat.

On admission, her vital signs were stable (temperature 37.0 °C, blood pressure 138/64 mmHg, heart rate 61 beats/min). Physical examination revealed a tender left inguinal lymph node. No tick-bite marks were identified. Laboratory testing showed marked leukopenia and thrombocytopenia, mild elevation of transaminase and lactate dehydrogenase levels, hypocalcemia, and coagulation abnormalities, with an acute-phase disseminated intravascular coagulation (DIC) score of 4

points. Twelve-lead electrocardiography (ECG) showed sinus rhythm without conduction abnormalities (Fig. 1A). Transthoracic echocardiography demonstrated normal biventricular function without structural abnormalities.

Supportive treatment was initiated with favipiravir as antiviral therapy for SFTS, filgrastim for severe neutropenia, and recombinant thrombomodulin for DIC. Empirical broad-spectrum antibiotic therapy with meropenem was also started given the severity of illness and concern for bacterial coinfection, although no clear bacterial focus was identified. These therapies were not considered likely to have contributed to the atrioventricular conduction disturbance. On hospital day 2, reverse-transcription polymerase chain reaction (RT-PCR) testing of a serum specimen was positive for SFTS RNA, with a viral load of $10^{6.1}$ copies/ml.

On hospital day 6, at approximately 08:00, first-degree AV block was observed on ECG monitoring. Later that day, the patient experienced transient loss of consciousness after defecation, accompanied by complete

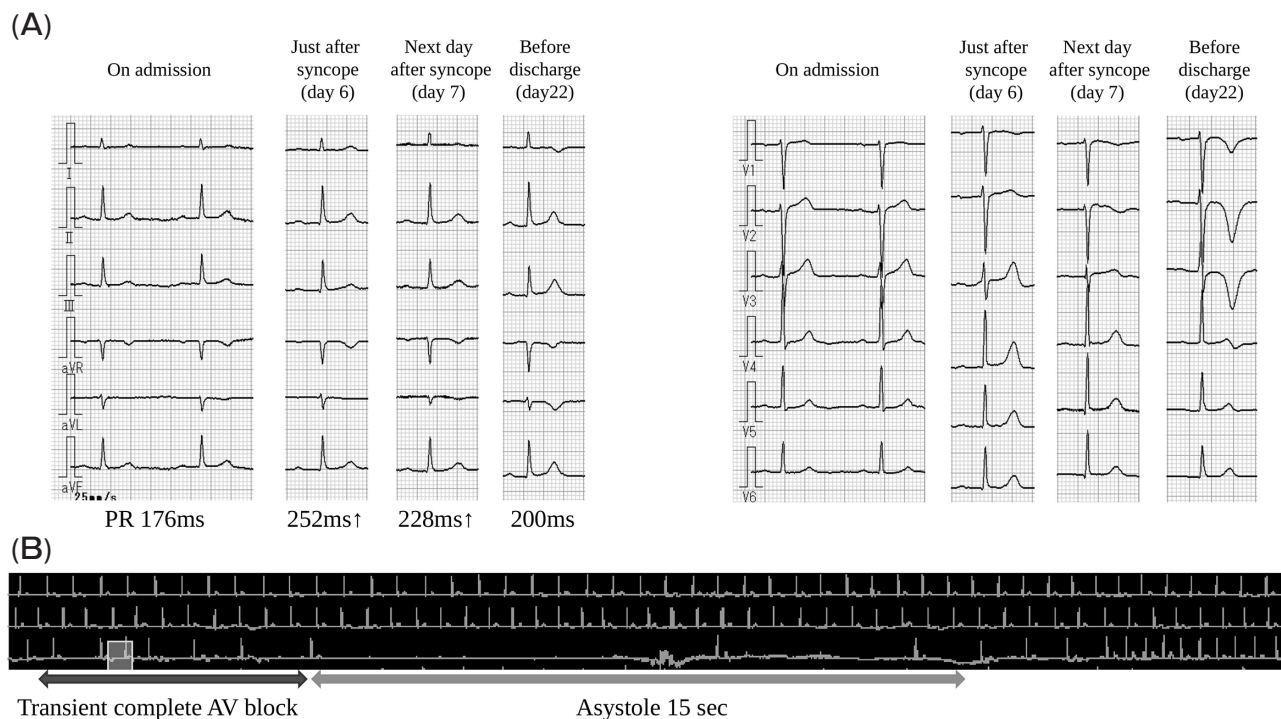


Fig. 1 Electrocardiographic monitoring and 12-lead electrocardiography (ECG) findings during hospitalization. **(A)** On admission, 12-lead ECG showed sinus rhythm with a normal axis and no significant ST-T abnormalities. On hospitalization day 6, immediately after syncope, ECG monitoring showed first-degree atrioventricular (AV) block. On hospital day 22, the first-degree AV block had resolved, and negative T waves were observed in leads I, aVL, and V1-V4. **(B)** At 12:53 on hospital day 6, ECG monitoring showed a 15-second asystolic pause due to AV block.

AV block, which was initially considered possibly related to vagal reflex. At 12:53, she experienced a 15-second asystolic pause during lunch due to recurrent complete AV block (Fig. 1B). A third syncopal episode occurred while arrangements for pacing were being made.

Repeat echocardiography continued to show preserved ventricular function. Cardiac enzymes were not suggestive of myocardial injury (creatine kinase 93 U/l; creatine kinase-MB 8.0 U/l). Troponin I was elevated (50.6 pg/ml). Selected serial laboratory findings during hospitalization, including the period of AV block progression and recovery, are shown in Table 1. Given recurrent syncope and asystolic pauses due to complete AV block, a temporary transvenous pacemaker was

inserted via the right internal jugular vein and set to ventricular demand pacing at 50 beats/min. Monitoring detected a 1.5-min period of consecutive pacing spikes, consistent with prolonged ventricular standstill following complete AV block.

On hospital day 7, the PR interval remained prolonged (228 msec) but shorter than on day 6, and no further pauses occurred. The temporary pacing catheter was removed on day 9. By day 15, ECG showed negative T waves in leads I, aVL, and V1–V4, with a slightly prolonged PR interval but no recurrent first-degree AV block. These T-wave changes were considered to reflect myocardial ischemia related to prolonged pauses or ischemia of the conduction system; cardiac memory after temporary pacing was also considered as a possible benign explanation [6]. Coronary CT angiography performed on hospital day 16 revealed no clinically significant coronary stenosis. Cardiac magnetic resonance imaging performed on hospital day 22, 16 days after the onset of conduction disturbance, showed no myocardial edema or late gadolinium enhancement, arguing against myocarditis. The patient was transferred to another hospital on day 23 in stable condition. A concise clinical timeline of the patient's course is provided in Table 2.

Discussion

We describe a patient with SFTS who developed progressive AV conduction disturbance culminating in transient complete AV block with syncope and brief cardiac arrest. The conduction disturbance emerged

Table 1 Selected serial laboratory findings during hospitalization

	Day 1	Day 6	Day10
White blood cells (/μl)	970	2,700	4,800
Platelets (×10 ⁴ /μl)	6.4	3.9	12.7
AST (U/l)	55	71	29
ALT (U/l)	28	54	29
FDP (μg/ml)	10.7	2.9	3.6
Na (mEq/l)	133	138	140
K (mEq/l)	3.5	3.9	3.9
Ca (mg/dl)	7.7	7.5	–
CK-MB (U/l)	7.9	8.0	5.5
Troponin I (pg/ml)	15.7	50.6	389.4

AST, aspartate aminotransferase; ALT, alanine aminotransferase; CK-MB, creatine kinase-MB; FDP, fibrin/fibrinogen degradation products.

Table 2 Clinical timeline of the patient

Hospital day	Events
Day –3	Illness onset with fever
Day –2	Visited primary care physician
Day 1	Admission; ECG showed sinus rhythm
Day 2	SFTS confirmed by RT-PCR
Day 6	
08:00	First-degree AV block appeared
Later that morning	Syncope after defecation with complete AV block
12:53	Complete AV block with 15-second asystole
Thereafter	Temporary pacemaker inserted
Day 7	PQ interval 232 ms; no pauses
Day 9	Temporary pacemaker removed
Day 15	No recurrent AV block; borderline PR interval with negative T waves
Day 23	Transferred in stable condition

after several days of systemic illness, required temporary pacing, and resolved spontaneously without permanent pacemaker implantation.

High-degree AV block in viral infections can occur through several mechanisms, including myocarditis with inflammatory involvement of the conduction system, ischemia, metabolic and electrolyte disturbances, and autonomic imbalance [7, 8]. In patients with SFTS, cardiac injury and arrhythmias have been reported and may be associated with worse prognosis [3]. A systematic review reported that bradycardia was most frequently observed, followed by atrial tachyarrhythmias and premature atrial or ventricular complexes; only two AV block cases were reported, without detailed description of severity or clinical course [4]. Myocarditis has also been reported in patients with SFTS and should be considered when conduction abnormalities and cardiac biomarker elevation are present [9]. In the present case, however, clinically overt myocarditis was not supported by echocardiography, cardiac biomarker patterns, or cardiac magnetic resonance imaging. The modest troponin I elevation at the onset of AV block, followed by a marked increase later in the clinical course, raises the possibility that myocardial injury may have occurred secondary to severe conduction disturbance or systemic illness, rather than representing the primary cause of AV block. Nevertheless, because endomyocardial biopsy was not performed and cardiac magnetic resonance imaging was obtained 16 days after the onset of conduction disturbance, focal or subclinical myocardial involvement cannot be fully excluded. Therefore, the precise mechanism of AV block in this case could not be definitively established.

Important differential diagnoses for transient complete AV block include acute coronary syndrome, drug-induced conduction disturbance, significant electrolyte abnormalities, and degenerative conduction disease. This patient had no baseline conduction abnormality on admission ECG and demonstrated spontaneous recovery, features that are less typical of degenerative conduction disease. There was no exposure to AV nodal blocking agents. Although hyponatremia and hypocalcemia were present, the temporal relationship and rapid progression to complete AV block suggested that these abnormalities alone were unlikely to fully explain the event. Acute coronary syndrome could not be conclusively excluded because cor-

onary imaging was not performed; however, the preserved ventricular function, the absence of overt ischemic symptoms, and the absence of significant coronary stenosis on coronary CT made a large myocardial infarction less likely.

In SFTS, coagulopathy, including DIC, is common and may contribute to microvascular dysfunction [10]. One potential explanation for reversible high-degree AV block is transient ischemia of the conduction system due to microthrombi or supply-demand mismatch in the setting of severe systemic inflammation. This hypothesis remains speculative without direct objective confirmation. Regardless of the underlying mechanism, the presence of progressive conduction disturbance—from first-degree AV block to complete AV block—on monitoring provided an actionable warning sign. In similar cases, early recognition and timely temporary pacing may be life-saving.

In conclusion, SFTS may be complicated by reversible high-degree AV block even in the absence of supportive evidence for clinically overt myocarditis. Careful ECG monitoring and timely temporary pacing should be considered when progressive conduction disturbances are detected in patients with SFTS.

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