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DOI 10.25789/YMJ.2025.92.32

УДК 616.127-004

A CASE REPORT OF FREDERICK'S SYNDROME: COMPLETE TRIFASCICULAR BLOCK WITH ATRIAL FIBRILLATION

This article presents a clinical case of a patient with newly diagnosed Frederick's syndrome, in this case persistent atrial fibrillation and complete trifascicular block, including proximal complete AV block, anterior hemiblock, and complete right bundle branch block. The possible mechanism of development of this condition and the treatment provided at Regional Clinical Hospital No. 1 in Tyumen, Russian Federation, are discussed.

Keywords: Frederick's syndrome; electrocardiogram; atrial fibrillation; trifascicular block; complete heart block; anterior hemiblock; complete right bundle branch block

For citation: Vedernikov A.A., Mezhonov E.M. A case report of Frederick syndrome: complete trifascicular block with atrial fibrillation. Yakut Medical Journal, 2025; 92(4): 130-133. https://doi.org/10.25789/YMJ.2025.92.32

Introduction. As is often the case in medicine, Frederic's syndrome (FS) is an eponym. In 1904, the Belgian physiologist L.L. Frederick, during an experiment, established that in animals with atrial fibrillation (AF), the intersection of the His bundle causes regular contractions of the ventricles, despite the persistent arrhythmia in the atria [7]. In humans, FS is characterized by a combination of complete atrioventricular (AV) block and AF or atrial flutter, which leads to a complete cessation of impulse conduction from the atria to the ventricles. Under these conditions, the ventricles are excited by the pacemaker from the AV node or ventricular conduction system, while chaotic contractions of individual muscle fibers occur in the atria. On an electrocardiogram (ECG), this manifests itself as both an f-wave and regular ventricular contractions. FS occurs in 0.6–1.5% of patients with AF [1,4]. Data on the epidemiology of FS are outdated and require updating. FS is mentioned in isolated English-language publications, which suggests the use of this eponym by physicians in the post-Soviet space rather than the extreme rarity of this pathology. The clinical presentation of FS may include episodes of loss of consciousness (Morgagni–Adams–Stokes attacks), dizziness and weakness, as well as bradycardia.

Objective: to describe a clinical case of a patient with FS, with a trifascicular block against the background of AF.

Materials and Methods: A retrospective analysis of the medical records of an inpatient in the arrhythmology department of the Tyumen Regional Clinical Hospital No. 1 was conducted. Data from clinical observation, laboratory tests, instrumental diagnostics, and the surgical protocol were used.

Case Report. A 74-year-old man presented to our emergency department with complaints of severe general weakness, dizziness, hypotension for 3 months, and syncope for 1 year. The last brief, unattended loss of consciousness, without bladder or bowel movement, occurred 2 months ago. There was no previous histo-

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ry of similar complaints. He discontinued his cardiotonic medications (losartan, indapamide, bisoprolol) 3 months ago due to low blood pressure as self-monitored. His cardiac history revealed hypertension and persistent AF for 5 years, for which he regularly takes apixaban. He did not report taking any other medications.

Patient history. No family history. He denies any bad habits. His allergy and epidemiological history are unremarkable. He has a sigmoid colon tumor. He underwent surgery in 2011, which resulted in a temporary stoma. No other treatments were performed. He was removed from the oncologist's list in 2022. Does not report any other chronic diseases.

Objective data. Examined in a horizontal position. The body constitution is abnormal - congenital deformities of the limbs, fusion and rotation of the toes on the left foot, as well as on the right and left hands. Consciousness is clear. Mucous membranes are clean, moist. The skin is clean, somewhat pale. The lymph nodes are intact. Moderate pastosity of the lower third of the shins and feet. The number of breaths per minute is 18 per min. SpO2 98%. On auscultation, breathing is vesicular, conducted in all parts, no wheezing. The shape of the chest is normosthenic. Heart rate is 32 beats per minute. Pulse is rhythmic. Blood pressure on the right arm is 150/80 mmHg, on the left arm 150/70 mmHg. Heart sounds are clear, rhythmic. Heart murmurs are not auscultated. Pulsation in the peripheral arteries is determined. The abdomen is not distended and is soft. The liver is at the costal margin. Urination and defecation are unremarkable.

Laboratory diagnostics. Troponin I is 18 ng/L (reference range up to 22 ng/L), sodium uretic peptide is 1492 pg/mL (reference range up to 440 pg/mL), glomerular filtration rate (MDRD) is 95.03 mL/min, and there is mild normocytic anemia (Red blood cells (RBC) 4.32 10*12/L; Hemoglobin (HGB) is 118 g/L; Mean corpuscular volume (MCV) is 86.2 fl). Other laboratory tests showed no significant abnormalities.

The patient was admitted to the intensive care unit due to severe bradycardia and complete heart block leading to syncope.

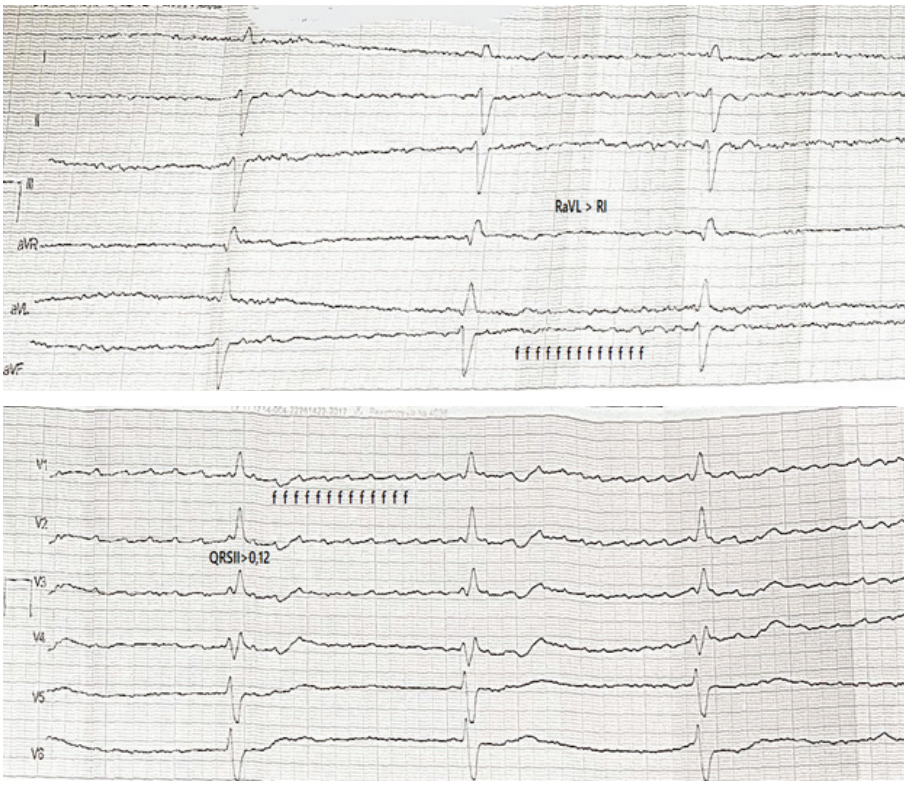
Instrumental diagnostics. Figure shows the patient's ECG. Echocardiography (table 1) reveals moderate right heart dilation, signs of pulmonary hypertension, marked left atrial dilation, and moderate concentric left ventricular (LV) myocardial hypertrophy. Grade 1 aortic and mitral valve regurgitation was present. Against the background of diffuse myocardial hypokinesis, areas of local contractility impairment were not reliably identified. Global contractility of the LV myocardium is reduced, ejection fraction is 32%.

Coronary angiography (CAG) was performed - no significant obstructive lesion of the coronary arteries was detected. According to ultrasound dopplerography of the brachiocephalic arteries, hemodynamically insignificant atherosclerosis is present. Triglycerides are 1.11 mmol/L, high-density lipoproteins are 1.22 mmol/L, cholesterol is 4.16 mmol/L, and low-density lipoproteins are 2.8 mmol/L.

Surgery was then performed. An Ingevity 7842 59 cm endocardial electrode was inserted into the right ventricle via the subclavian vein and positioned in the middle third of the interventricular septum. The electrode was connected to the Essentio SR pacemaker in VVI mode. Pacemaker monitoring data are presented in table 2. During subsequent hospital observation, syncope was absent, episodes of hypotension were not recorded, and a neurologist examined the patient. Neurogenic causes of syncope were excluded. For technical reasons, stress tests for coronary artery disease were not performed.

The final clinical diagnosis was coronary artery disease. Cardiac rhythm and conduction disturbance: third-degree atrioventricular block secondary to atrial fibrillation (Frederick's syndrome). CHA2DS2-VASc 3 points. HAS-BLED 2 points. Implantation of the Essentio SR (VVI) pacemaker with an endocardial electrode on August 12, 2025. Stage 1 CHF (RKO 2023), CHF IIA (according to Vasilenko-Strazhesko), low EF-32%, FC 2. Stage III hypertension. Target blood pressure level not achieved. Risk 4 (very high). The patient was discharged in a satisfactory condition. Upon discharge, the following medications were recommended for regular use: Dapagliflozin 10 mg, 1 tablet once a day. Bisoprolol 2.5 mg, 1 tablet in the morning. Apixaban 5 mg, 1 tablet 2 times a day. Valsartan 80 mg, 1 tablet in the morning. Amlodipine 10 mg, 1 tablet in the evening. Spironolactone 25 mg, 1 tablet in the morning. Atorvastatin 20 mg, 1 tablet in the evening. An appointment with a cardiologist qualified to program pacemakers is scheduled in two months.

Discussion. The main period of studying the cardiac conduction system, mostly in animal models, occurred in the 20th century, but today, various conduction pathologies continue to be investigated by the medical community. Of interest is the newly identified Bayes syndrome, characterized by the association of severe interatrial block and atrial arrhythmias, in particular AF, with an increased risk of dementia, stroke, and mortality [6]. Also recently, a rare case of combined cardiac conduction disor-



ECG of the patient upon admission (recording speed 50 mm/s, amplitude 10 mm/mV)

der was described, in which alternating bundle branch block was accompanied by second-degree AV block [10]. FS, like the above-described Bayes syndrome and alternating block, are an uncommon cardiac conduction disorder in the daily clinical work of a cardiologist, but this fact does not reduce its severity and significance. In routine practice, when reading an ECG, it is necessary to increase physician alertness to changes in the characteristics of the pacemaker and slowing of impulse conduction through various parts of the myocardium.

The causes of FS are pronounced sclerotic, degenerative, or inflammatory changes in the myocardium due to severe organic heart diseases, such as coronary artery disease (CAD), acute myocardial infarction, myocarditis, cardiomyopathy, or other structural disorders. Factors that provoke manifestation may be excessive physical activity, taking medications that have negative chronotropic, dromotropic and bathmotropic effects, as well as electrolyte imbalance and stress [3]. There is also evidence that CAD and hypertension are the main cause of the combination of right bundle branch block and left anterior fascicular block [9].

The presence of significant cardiac rhythm and conduction disturbances and the patient's advanced age may suggest infiltrative myocardial diseases, particularly amyloidosis. The echocardiographic picture (absence of both asymmetrical pronounced hypertrophy and dilation of the heart, absence of signs of restriction, absence of significant damage to the aortic valve, absence of myocardial heterogeneity), as well as the absence of clinical signs of a systemic pathological process, namely, damage to the gastrointestinal tract, nervous system, eyes and kidneys in addition to the heart, speak against this diagnosis in this patient. However, there is evidence that trifascicular block may be a primary manifestation of cardiac amyloidosis [11].

In the present case, we observe prominent AF f-waves and regular, infrequent ventricular contractions, as well as signs of anterior hemiblock and complete right bundle branch block. AF f-waves are not always visualized on the ECG, which creates difficulties in differential diagnosis. The deformation of the QRS complexes on the ECG in this patient is moderate and is associated with a block along the left and right branches of the His bundle, with the pacemaker located in the AV junction, indicating a proximal type of block, often not accompanied by a disturbance in the biomechanics of cardiac contraction [2, 5, 8]. Distal AV block is characterized by widened, deformed ventricular-type QRS complexes.

Table 1

Echocardiography data

Parameters	Value
Left Atrium EDV ICV	46 mm 89 ml 50 ml/m^2
Right atrium	41x59 mm
Right ventricle	36 mm
Anterior wall of the right ventricle	6 mm
Aorta	not dilated, walls are compacted, in the ascending section - 39 mm
Aortic valve leaflets Systolic leaflet opening Leaflet divergence Vmax Pgmax Regurgitation	compacted unlimited 18 mm 1.6 m/s 11 mmHg 1st degree
Left ventricle: EDV EDV ESR ESV Simpson ejection fraction Local contractility Myocardial mass: LVM LVMI TCR	 51 mm 124 ml 43 mm 83 ml 32% диффузный гипокинез 241 g 137 0,47
Mitral valve: Customers Diastolic valve opening peak E: Vmax Regurgitation	 not thickened, compacted unlimited 0.7 m/s 1st degree
Interventricular Septum diastolic Posterior Wall of the Left Ventricle diastolic	12 mm 12 mm
Tricuspid valve: Customers Diastolic valve opening Vmax Regurgitation Pgmax	 not thickened unlimited 0.55 m/s Physiological 1.2 mmHg
Pulmonary valve: Cutlets Systolic leaflet opening Vmax Pgmax Regurgitation	 thin unlimited 1.4 m/s 8 mmHg 1st degree
Collapse of the IVC during inspiration IVC Signs of pulmonary hypertension: pulmonary artery diameter PAS	less than 50% 20 mm 28 mm 47 mmHg

As mentioned above, such cardiac rhythm and conduction disturbances in patients may be associated with CAD. Initially, a large volume of atherosclerotic coronary lesions, possibly multivessel plaque localization, can be suspected. Also, given the high incidence of AV block in inferior myocardial infarction, the physician should assess for patterns of vascular accident in the leads of the corresponding location. In our case, there were no electrocardiographic signs of myocardial infarction, and the intact coronary arteries according to invasive coronary angiography and a negative troponin test in the patient rule out acute forms of coronary artery disease as a cause of FS.

The development of complete AV block in the context of AF is associated with significant cardiac remodeling, the development of various complications, and a worsening prognosis, which is often underestimated by doctors. The ab-

Table 2

Pacemaker examination

Parameters	Meaning
Model of pacemaker	Essentio SR
On the monitor	rhythm from pacemaker in VVI mode 60 bpm
Stimulation mode	VVI
Base rate	60 bpm
Amplitude RV	3.5 V
Duration RV	0.4 ms
Threshold RV	0.4 V
Sensitivity RV	2.5 mV
Voltage of wave RV (R)	14.9 mV
Impedance RV	678 Ohm
Expected service life	more than 8 years
Statistics	AsVs: 4 %; AsVp: 96 %

sence of atrial systole in AF, as well as the presence of AV dyssynchrony with a marked decrease in heart rate, lead to a decrease in the atrial and ventricular contribution to LV filling during diastole. As a result, the degree of mitral regurgitation and pulmonary artery wedge pressure increases, the load on the right ventricle increases, which over time causes its dysfunction and expansion, as well as dilation of the fibrous rings of the AV valves. Similar echocardiographic characteristics are observed in our patient, who likely has arrhythmogenic cardiomyopathy.

The first step in treating patients with bradycardia is discontinuing medications that lower the heart rate, primarily beta-blockers and digoxin. A detailed patient history is helpful in this regard. The next step, if bradycardia persists and the accompanying symptoms described above appear, is pacemaker implantation. The literature describes the practice of implanting a permanent pacemaker with bundle-His pacing in FS, which ensures the most physiological conduction of the impulse through the cardiac conduction system [5]. Our clinic does not yet use this technique. To date, there is no scientific data on the comparison of different pacemaker modes for FS and their effectiveness.

Conclusion

This clinical case contributes to expanding knowledge about FS. Due to the difficult diagnosis and treatment of this combined arrhythmia, especially in elderly patients, increased attention is required to the possible development of FS. Early detection of cardiac symptoms allows for timely administration of drug therapy and/or referral of the patient for pacemaker implantation, which will contribute to an improved prognosis and reduced mortality among patients with such a common arrhythmia as AF.

The authors declare no conflicts of interest.

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