International Journal of Research in Medical Sciences
Snezhitskiy VA et al. Int J Res Med Sci. 2025 May;13(5):2140-2144
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20251323

Case Report

A rare case study: pre-excited atrial fibrillation in Wolff-parkinson-white syndrome associated with partial ventricular septal defect

Viktor A. Snezhitskiy, Marina Matsiukevich, Naveen D. K. N. Direcksze*, Prithvi A. Dineshkumar, M. N. M. Afham

Department of Internal Medicine, Grodno State Medical University, Belarus, Europe

Received: 19 March 2025
Revised: 17 April 2025
Accepted: 21 April 2025

***Correspondence:**
Dr. Naveen D. K. N. Direcksze,
E-mail: ndkndis@gmail.com

ABSTRACT

Wolff–parkinson–white (WPW) syndrome is a congenital disorder affecting the hearts' conduction system, which is recognized by the existence of an accessory pathway (AP) that can lead to pre-excitation of ventricles, paroxysmal supraventricular tachycardias and in severe cases to sudden cardiac death. Thus, AP usually has non-decremental conduction allowing immediate ventricular activation which in case of AF leads to rapid ventricular response with high risk of transformation into ventricular fibrillation and cardiac arrest. Physical examination of this patient revealed the following clinical manifestations of WPW syndrome like severe tachycardia, dizziness and presyncope. The lab findings revealed an increase in the high-sensitive troponin I suggesting the underlying result from atrial fibrillation. And hyperbilirubinemia was shown in the biochemical analysis confirming the concomitant disease of this patient which is Gilbert's Syndrome. The ECGs depicted the presence of a type A WPW pattern and paroxysmal form of atrial fibrillation (AF). The echocardiograms disclosed the presence of ventricular septal defect (VSD) in the membranous part of the interventricular septum. Both the ECGs and Echocardiograms revealed type A WPW pattern and paroxysmal form of atrial fibrillation respectively. The patient's heart rate was monitored routinely in the intensive care unit with the help of 24-hour Holter monitoring device, breathing rate and blood pressure to detect the evaluation the patient's prognosis. This article enhances the difficulties in differential diagnosis, experience of amiodarone uses in acute management, interventional treatment of pre-excited AF and VSD, because this can be a primary manifestation in young patients.

Keywords: Wolff-parkinson-white syndrome, Ventricular septal defect, Atrial fibrillation

INTRODUCTION

The normal cardiac conduction system (CCS) consists of two types of cardiomyocytes such as contractile (98%) and conducting cells (2%). CCS components are classified into fast-conducting pathways including the bundle of His, the branches of right and left bundle, Network of Purkinje fibres and slow conducting pathways including sinoatrial node (SAN) and atrioventricular node (AVN).[1] In contrast, Wolff-Parkinson-White (WPW) Syndrome is recognized to be a congenital disorder with an abnormal electrical conductance between the atria and ventricles, with the assistance of an accessory pathway, called Bundle of Kent.[2] The bundle of Kent is categorized into the following clinical variants such as accessory pathway, concealed accessory pathway, decremental accessory pathway, permanent junctional reciprocating tachycardia and Atri fascicular (nod ventricular Mahaim) accessory pathways.[3] The most common locations of Bundle of Kent are left lateral (50%), posterolateral (30%), right lateral (10%) and right anteroseptal (10%). These pathways are a result of incomplete closure of atrioventricular and fibrous insulator separations during embryogenesis, thus leading to development of preexcitation (early depolarization of ventricles) and malignant arrhythmias.[4] The keystone electrocardiogram criteria (ECG) depict the type A WPW pattern showing a short PR interval, broad QRS complex with a positive Delta wave in a sinus rhythm and type B

reveals negative delta waves, dominant S wave in precordial lead V1 with broad QRS complex and short PR interval.[5] WPW syndrome is also defined as a clinical syndrome with an WPW pattern in ECG and set of clinical symptoms like chest pain, tachyarrhythmia, episodic light-headedness, presyncope, syncope. Increased heart rate also leads to palpitations and may also cause chest pain due to the increased myocardial oxygen demand. The term WPW pattern should not be confused with WPW syndrome, where the WPW pattern is an ECG finding of WPW in asymptomatic patients.[2]

The incidence among the general population is about 1 to 3 for each 1000 individuals (0.1% to 0.3%). A greater occurrence of 0.55% has been reported in blood relatives of patients with accessory pathways.[3] Furthermore, the most common conditions associated with WPW are congenital heart diseases, including atrial septal defect (ASD), ventricular septal defect (VSD), coronary sinus diverticula, coronary abnormalities, hypertrophic cardiomyopathy (HCM), Ebstein anomaly and corrected transposition of great vessels; multisystem diseases such as PKAG2 syndrome, hypokalemia periodic paralysis, Pompe disease, Danon disease, cardiac rhabdomyoma and surgically acquired conditions consisting of corrective surgeries (Fontan procedure), heart transplantation, valve surgeries and Intra-atrial baffles.[6]

The following types of tachycardia are found in WPW syndrome: orthodromic atrioventricular reciprocal tachycardia, oAVRT (most frequent), antidromic atrioventricular reciprocal tachycardia, aAVRT and another supraventricular tachyarrhythmias (atrial fibrillation, atrial flutter and atrial tachycardia).[7]

Ventricular septal defect has been shown to be associated with WPW syndrome. Few case studies have been shown to further accommodate this common prevalence and adding the diagnostic value to physicians in their practice for an efficient prognosis of the patient.[8]

Risk stratification is carried out to examine the risk of sudden cardiac death (SCD) in patients, this is usually done with the help of invasive and non-invasive modalities. Non-invasive modalities usually review patients with low-risk subgroups based on ECG markers, treadmill stress test, Holter monitor, pharmacological tests with adenosine, catecholamine, ajmaline, procainamide and amiodarone, revealing the stable nature of the patient.[9-11] Invasive modalities include mainly the electrophysiological testing (EPT), which is proposed to symptomatic individuals and high-risk individuals.[12]

CASE REPORT

A 23-year-old male patient called an ambulance; because of complaints of severe tachycardia, accompanied by chest pain, palpitations, dizziness and presyncope. Physical examination revealed a very high heart rate which was difficult to count and low blood pressure (100/60 mmHg). In the ambulance the patient was diagnosed with sustained broad complex tachycardia (Figure 1) and extended infusion of amiodarone was given. By the time the patient was delivered to the emergency department of the grodno regional clinical cardiology centre in Belarus by ambulance, sinus rhythm was restored (Figure 2).

From the patient's medical history, it was discovered that previously he had been diagnosed with type A WPW syndrome and a ventricular septal defect of 5.5 mm in the membranous part of the interventricular septum which allows a left to right shunt. Over the past four years, the patient had experienced brief episodes of tachycardia that would spontaneously cease. Patient was hospitalized for additional investigations.

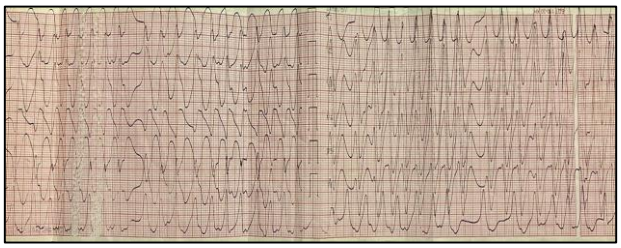

Figure 1: The 12-lead ECG exhibiting irregular wide polymorphic QRS tachycardia without the QRS twisting around the isoelectric baseline with a heart rate up to 320 b.p.m.

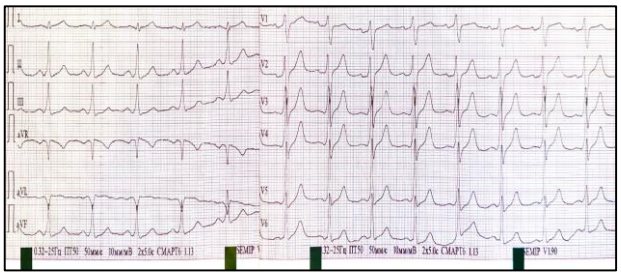

Figure 2: The 12-lead ECG exhibiting Type A WPW pattern consisting of positive delta wave (II, III, aVF and precordial leads), prolonged QRS complex and short PR interval after administration of Amiodarone 300 mg.

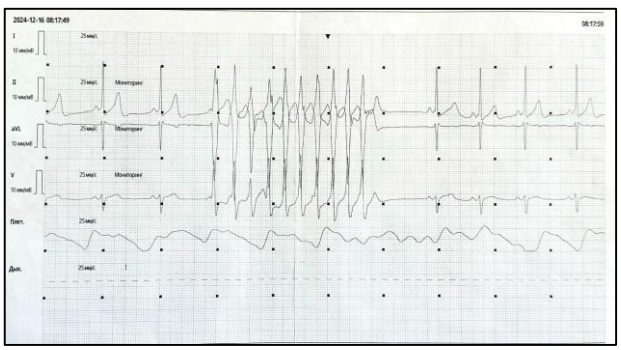

Figure 3: The 24-hours ECG exhibiting paroxysms of antidromic atrioventricular reentrant tachycardia.

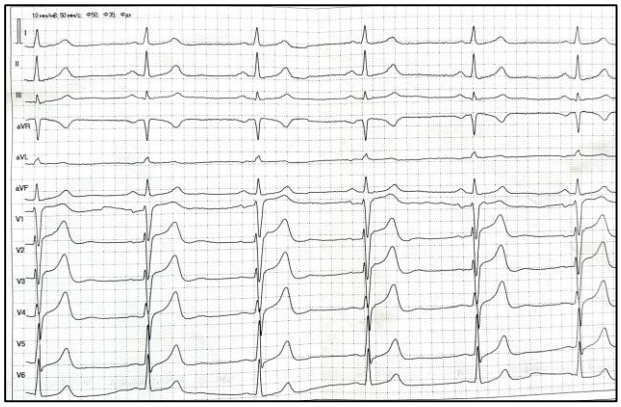

Figure 4: The 12-lead ECG exhibiting absence of delta wave, narrow QRS complex and normal PR interval after radiofrequency ablation of AP in WPW.

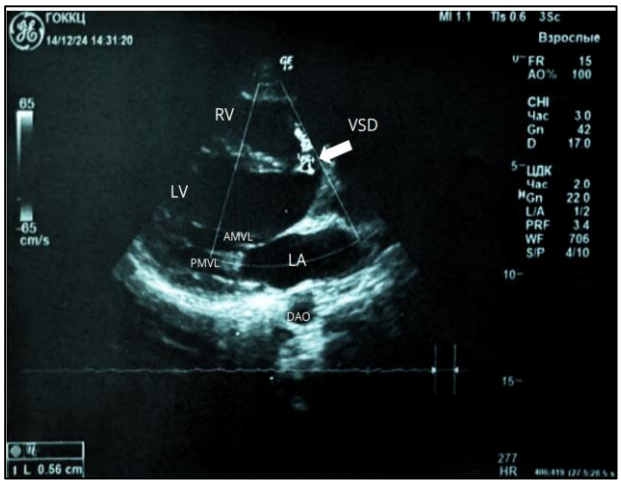

Figure 5: Echocardiography reveals ventricular septal defect (VSD). LA-left atrium, LV-left ventricle, AMVL-anterior mitral valve leaflet, PMVL-posterior mitral valve leaflet, RV-right ventricle, DAO-descending aorta.

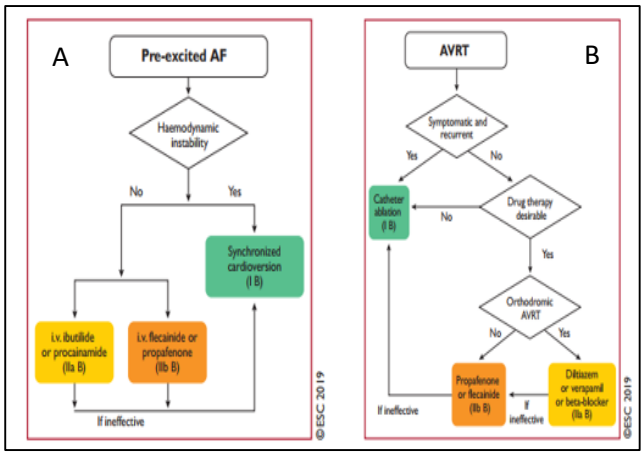

Figure 6: Acute (A) and long-term (B) treatment strategies of pre-excited atrial fibrillation. AF- atrial fibrillation, I.V-Intravenous, AVRT-atrioventricular re-entrant tachycardia.[22]

Lab findings

The complete blood count (CBC) did not reveal any significant deviation from the normal values. Biochemical analysis showed an elevated level of bilirubin, concluding the coexisting concomitant disease (Gilbert's Syndrome) (Table 1). The normal value of troponin I in males according to the laboratory normal range is 0-14 ng/l, the elevation of hs-Troponin I suggests about the myocardium damage due to tachyarrhythmia.

Imaging and instrumental findings

From the unstable nature of the patient's condition, a series of instrumental diagnostics were performed except stress tests. Patient was given Propafenone 150 mg three times a day in combination with Metoprolol 12,5 mg once a day. During 24-hours ECG monitoring were registered paroxysms of aAVRT (Figure 3).

Patient was identified as being at high risk of SCD and after electrophysiological study was performed successful radiofrequency ablation of AP (Figure 4).

The echocardiogram revealed a 5.5 mm opening in the membranous part of the interventricular septum with a left to right discharge of blood with an interventricular gradient of 70 mmHg without signs of right ventricle overload and dilation (Figure 5). For prevention of right ventricular failure was indicated elective surgical repair of VSD. Initially, the patient was treated with pharmacological therapy of drugs such as propafenone 150 mg three times a day in combination with Metoprolol 12.5 mg once a day.

This resulted in a progressive stabilization of the patients' condition, but after the electrophysiological study, this patient was identified as a high-risk category for SCD, therefore this patient was indicated for radiofrequency ablation of the accessory pathway (Bundle of Kent). The WPW pattern in ECG was disappeared after the radiofrequency ablation and the patients' condition was satisfactory. Further follow-ups were recommended by the cardiologist annually to assess the recurrence of WPW syndrome; but this is rare, yet possible.

Table 1: Biochemical analysis.

Creatinine, mcmol/l	**70**
Total bilirubin, mmol/l	39
Indirect bilirubin, mmol/l	31
Glucose, mmol/l	4.5
AST, IU/l	17
ALT, IU/l	13
LDH, IU/l	233
Potassium, mmol/l	4.5
TSH, mu/l	6.27
Cardio markers	
Troponin, ng/l	30.7

DISCUSSION

Wide complex tachycardia (WCT) is defined as a cardiac rhythm with a rate greater than 100 beats/min (bpm) and a QRS complex duration equal or greater than 0.12 seconds in the adult patient. The differential diagnosis of the WCT includes ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant intraventricular conduction; aberrant conduction results from pre-existing or rate-related bundle branch block (BBB), ventricular pre-excitation, or dysfunction of the intraventricular conduction system due to various factors (toxic, metabolic, cardiac injury, infectious, etc.) and pre-excited AF. From the electrocardiographic perspective, the rhythm differential diagnosis is broad, including both relatively benign and life-threatening dysrhythmias furthermore, the diagnosis of specific rhythms is often not possible, at least initially during the early phase of evaluation and management.[13] When it is impossible to identify the WCT the arrhythmia must be treated as VT.

The treatment of AF in WPW patients is completely different from the WPW patients without AF. Cardioversion is obligatory for acute treatment in unstable patients. The basic principle of pharmacological therapy is to elongate the refractory period of the accessory pathway in relation to AV node.[14] The conventional drugs for treatment of AF (Digoxin, Calcium channel blockers and Beta-blockers) are not feasible due to its prolongation of refractory period of AV node, thereby resulting in an increment of transmission through the accessory pathway, the use of procainamide alone can be dismissed due to its side effects on production of conventional AF, thus requiring of the other drugs for the treatment of pre-excited AF.

Pre-excited atrial fibrillation often is unrecognized and mistreated. In the presented clinical case patient was diagnosed with WCT without hemodynamic instability, that's why pharmacological cardioversion with amiodarone was the treatment of choice. It is known that amiodarone is considered to be potentially harmful to patients with pre-excited AF. According to ESC guidelines 2019 from the European heart rhythm association survey revealed insights about the preferred choice of treatment for pre-excited atrial fibrillation in WPW by application of class 1C antiarrhythmic agent. Amiodarone, a class 3 antiarrhythmic agent and beta-blockers were used as a minority.[15]

Amiodarone has a wide spectrum of multichannel pharmacological effects, including not only potassium and sodium channel blockade to lengthen the antegrade refractory periods of AP, but also calcium channel blockade to affect AV node conduction. In this clinical case the positive amiodarone use could be explained by the fact that the antegrade conduction of the AP strongly prevailed which may have reflected the predominant effect of amiodarone on the AP over the AV node conduction.[16] Long term pharmacological therapy can be used for prophylaxis of AF in WPW patients and the combination of flecainide and beta-blockers have shown a greater efficacy in termination of symptomatic tachyarrhythmias in WPW patients.[17] However, use of amiodarone is recommended only for patients with existing structural heart defects, who are warned against catheter ablation.[18,19]

Since paroxysmal form of atrial fibrillation is a major risk contributor for stroke development, the risk of thromboembolic events assessed by CHA2DS2-VA score was 0 and anticoagulation treatment was nao prescribed.[20,21]

The diagram (Figure 6) depicts the recommendation of acute treatment for pre-excited atrial fibrillation, where hemodynamically unstable patients undergo spontaneous synchronized DC cardioversion and hemodynamically stable patients undergo pharmacological therapy according to the severity level of the WPW syndrome.[22]

CONCLUSION

In conclusion, the presented case summarizes the complications of differential diagnosis between pre-excited AF with another WCT. Even though it is a rare pathological occurrence, in patients with VSD, WPW Syndrome and paroxysmal AF, this combination should be classified as a high-risk criterion for hemodynamic instability and SCD can be a primary symptom recorded in young patients. This case illustrates the importance of WPW patients with congenital diseases like VSDs as high priority with diagnostic value for clinicians to elucidate the proper and timely intervention can lead to better prognosis of patients.

Further genetic and clinical studies are warranted to explore the correlation of Wolff-parkinsons-white syndrome with ventricular septal defect in complicated cases with atrial fibrillation.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Bhattacharyya S, Munshi NV. Development of the cardiac conduction system. Cold Spring Harbor Persp Biol. 2020;12(12):37408.
2. Chhabra L, Goyal A, Benham MD. Wolff-Parkinson-White Syndrome. InStatPearls. StatPearls publishing.
3. Sethi KK, Dhall A, Chadha DS, Garg S, Malani SK, Mathew OP. WPW and preexcitation syndromes. J Asso Phys India. 2007;55:10.
4. Jemtrén A, Saygi S, Åkerström F, Asaad F, Bourke T, Braunschweig F, et al. Risk assessment in patients with symptomatic and asymptomatic pre-excitation. Europace. 2024;26(2):36.
5. Saka A. Determination of factors influencing the activities of pr interval on the electrocardiogram (ecg)

in an experiment of the factorial Type. Int J Clin Biostat Biom. 2021;7:041.
6. Vătășescu RG, Paja CS, Șuș I, Cainap S, Moisa ȘM, Cinteză EE. Wolf–parkinson–white syndrome: diagnosis, risk assessment, and therapy—an update. Diagnos. 2024;14(3):296.
7. Murphy SD, Torlutter M. The use of vagal manoeuvres in narrow complex tachyarrhythmias in primary care. South African Family Practice. 2022;64(1):56.
8. Green JR, Bartley TD, Schiebler GL. The Wolff-Parkinson-White Syndrome and Ventricular Septal Defect: A Case Report of Successful Surgery in an 18-Year-Old Man. Diseases of the Chest. 1965;47(6):659-62.
9. Saka A. Determination of Factors Influencing the Activities of PR Interval on the Electrocardiogram (ECG) in an Experiment of the Factorial Type. Int J Clin Biostat Biom. 2021;7:41.
10. Wellens HJ, Bär FW, Gorgels AP, Vanagt EJ. Use of ajmaline in patients with the Wolff-Parkinson-White syndrome to disclose short refractory period of the accessory pathway. Am J Cardiol. 1980;45(1):130-3.
11. Wellens HJJ, Braat SH, Brugada P, Gorgels AP, Bar FW. Use of procainamide in patients with the Wolff-Parkinson-White syndrome to disclose a short refractory period of the accessory pathway. Am J Cardiol. 1982;50:921–25.
12. Khairy P, Van Hare GF. PACES/HRS Expert Consensus Statement on the Recognition and Management of Arrhythmias in Adult Congenital Heart Disease. Can. J. Cardiol. 2014;30:53.
13. Brady WJ, Mattu A, Tabas J, Ferguson JD. The differential diagnosis of wide QRS complex tachycardia. Am J Emerg Med. 2017;35(10):1525-9.
14. Schatz I, Ordog GJ, Karody R. Wolff-Parkinson-White syndrome presenting in atrial fibrillation. Ann Emerg Med. 1987;16:574-8.
15. Svendsen JH, Dagres N, Dobreanu D, Bongiorni MG, Marinskis G, Blomström-Lundqvist C. Conducted by the Scientific Initiatives Committee, European Heart Rhythm Association. Current strategy for treatment of patients with Wolff–Parkinson–White syndrome and asymptomatic preexcitation in Europe: European Heart Rhythm Association survey. Europace. 2013;15(5):750-3.
16. Hamer AW, Mandel WJ, Zaher CA, Karagueuzin HS, Peter T. The electrophysiological basis for the use of amiodarone for treatment of cardiac arrhythmias. Pacing Clin Electrophysiol. 1983;6:784–94.
17. Crozier I. Flecainide in the Wolff-Parkinson-white syndrome. Am J Cardiol. 1992;70(5):26-32.
18. Calkins H, Camm AJ, Campbell WB, Haines DE, Kuck KH, Lerman B. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias-executive summary. European Heart J. 2003;24:1857-97.
19. Ludmer PL, McGowan NE, Antman EM, Friedman PL. Efficacy of propafenone in Wolff-Parkinson-White syndrome: electrophysiologic findings and long-term follow-up. J Am College of Cardiol. 1987;9(6):1357-63.
20. Patel MR, Mahaffey KW, Garg J, Pan G, Singer DE, Hacke W, Breithardt G, Halperin JL, Hankey GJ, Piccini JP, Becker RC. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. New England J Med. 2011;365(10):883-91.
21. Prystowsky EN, Greer S, Packer DL, Thompson KA, German LD. Beta-blocker therapy for the Wolff-Parkinson-White syndrome. The American J Cardiol. 1987;60(6):46-50.
22. Brugada J, Katritsis DG, Arbelo E. supraventricular tachycardia the task force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC) developed in collaboration with the association for European pediatric and congenital Cardiology (AEPC). European Heart J. 2019;41(5):655-720.

Cite this article as: Snezhitskiy VA, Matsiukevich M, Direcksze NDKN, Dineshkuma PA, Afham MNM. A rare case study: pre-excited atrial fibrillation in Wolff-parkinson-white syndrome associated with partial ventricular septal defect. Int J Res Med Sci 2025;13:2140-4.