1 | INTRODUCTION

We herein report a case that encompasses three uncommon, yet important electrophysiological entities, Wolff-Parkinson-white (WPW), Brugada phenocopy, and Flecainide toxicity in a patient with acute myeloid leukemia. A brief review of the literature is discussed.

2 | CASE PRESENTATION

A 54-year-old male with history of hypertension, diabetes, and acute myeloid leukemia (AML) underwent matched unrelated donor stem cell transplant in May 2015; as his disease recurred, he underwent stimulated donor lymphocyte infusion in January 2016 complicated by graft versus host disease manifesting with oral, skin, ocular, liver, and pericardium involvement; subsequently, the patient underwent pericardial window in April 2017.

He continued to be on chemotherapy with Decitabine, his disease course was also complicated by recurrent deep venous thrombosis and pulmonary embolism in April 2018 and a left femur fracture in May 2018.

In July 2017, he presented with multiple episodes of supraventricular tachycardia (SVT), Figure 1, and he was diagnosed with WPW syndrome with intermittent pre-excitation (right posteroseptal accessory pathway as evident by negative delta wave in V1, QRS complex transition at V1-V2, and negative delta wave in inferior leads, I, II AVF, Figures 2 and 3. EKG several years ago was reported as normal with no evidence of pre-excitation (image not provided). Patient preferred to try medical treatment and to avoid electrophysiological study with ablation, he responded well to flecainide and metoprolol.

In July 2018, the patient was admitted to the bone marrow service after an episode of unresponsiveness concerning for seizure along with shortness of breath in the setting of recently diagnosed pneumonia, heart failure exacerbation, and progression of cardiac leukemia.

Physical examination was remarkable for scattered lung rales bilaterally and traces pedal edema. Patient was afebrile.

A laboratory panel on admission found the following abnormal results: hemoglobin 10.1 g/dL; platelet count: 107 000; BUN: 37; creatinine: 2.35; aspartate transaminase (AST): 94; alanine transaminase (ALT): 29; albumin: 2.1 g/dL; lactate dehydrogenase: 901 IU/L; lactic acid: 2.5 mm/L; and troponin I levels: 0.0-0.09 ng/mL.
All electrolytes including potassium were within normal ranges.

Home medications were as follows: flecainide 100 mg twice daily; amlodipine; dabigatran; doxycycline; duloxetine; Lasix; metoprolol XL; pantoprazole; and albuterol inhaler.

He was noted to have intermittent polymorphic ventricular tachycardia (PMVT) and pauses on telemetry, along with progressive widening of the QRS complex on EKGs.

On his second day of admission, he developed cardiopulmonary arrest and asystole.

In view of worsening kidney and liver functions, he was suspected to have flecainide toxicity, amiodarone, which was started during resuscitation, was discontinued, he was started on IVF and sodium bicarbonate (NaHCO₃), epinephrine and atropine were given, and he regained his pulse with no further pauses or PMVT. His pauses and PMVT did not recur.
FIGURE 4  Twelve leads EKG showing persistent Brugada pattern (July 2018)

FIGURE 5  Parasternal view of 2-D echocardiogram showing remarkable thickening of the right ventricle and right ventricle outflow tract from the infiltrative tumor involvement (July 2018)

FIGURE 6  Modified parasternal view of 2-D echocardiogram showing a tumor mass involving the juncture of right ventricle and right ventricle outflow tract (July 2018)
and he remained in sinus rhythm, his QRS became narrower, Figure 4, with persistent Brugada pattern, and his fleca

de level was supratherapeutic at 1.32 μg/mL (normal range 0.2-0.99 μg/mL).

A transthoracic echocardiogram revealed evidence of in
drative tumor in his RA, RV, RVOT, and septum concern-
ing for progression of disease, Figures 5 and 6.

During the course of his admission his respiratory status
decompensated and he was started on mechanical ventilation.
Patient then developed mixed shock in the setting of infil-
trative tumor, heart failure, and sepsis which did not respond to
broad-spectrum antibiotics with meropenem and vancomy-
cin, in addition to vaspressors including norepinephrine and
vasopressin.

In the setting of worsening shock and progression of disease,
family meeting was held and decision was made to transition to
comfort care. For the few days before he expired, he remained
free of pauses or other arrhythmias, including PMVT.

3 | DISCUSSION

This case encompasses three uncommon, yet important elec-
trophysiological entities, the first is WPW. No electrocardio-
gram prior to the diagnosis of AML showed any evidence of
pre-excitation, a routine EKG later showed the WPW pattern,
and this was followed by episodes of SVT.

It is possible that the accessory pathway in our patient
became manifest or intermittent as the conduction proper-
ties of his AV node became slower with aging; however,
another possible mechanism is that there is a growth of
tissue in the form of infiltrative leukemia involving the
RA and RV where the cancerous tissues were clearly iden-
tified. While this was not reported before in the litera-
ture to our knowledge, we think that it can be a possible
mechanism.

Acquired accessory pathway has been described rarely,
especially after Fontan procedure.1-3 The mechanism of
which has been assumed to be due to the presence of pre-
viously unapparent congenital AP and that changes in the conduction
properties of the atrial myocardium after surgical
intervention and surgical injury to the AV node could slow
conduction, allowing a previously unapparent AP to become
manifest.2,3 However, surgically created accessory connec-
tions at the atroiofundibular anastomosis are another cause
of WPW syndrome after the Fontan procedure. The growth
of myocardial cells or excitable tissues across the suture line,
or the presence of electronic transmission through this line, is
the main explanation for this finding.1

Another interesting feature of this case is that it represents
a form of Brugada phenocopy, in which the mechanical com-
pression of the RVOT by infiltrative tumor may have led to
an EKG identical appearance to Brugada pattern which per-
sisted after flecainide was discontinued.

Brugada phenocopy as a new EKG phenomenon that has
recently been described as a cause of identical EKG appear-
ance but etiologically distinct clinical entity.4,6

Flecainide at supratherapeutic level in our patient is
thought to have caused the conduction abnormalities man-
ifesting as pauses, broad QRS, and PMVT. It is essential
to have high index of suspicion for flecainide toxicity
when encountering these arrhythmias in patients taking
the drug.7

Sodium bicarbonate (NaHCO₃) is commonly used to
reverse drug-induced sodium channel blockade caused by
different agents including flecainide.8 While there are no
definitive guidelines for management of severe flecainide
intoxication, intravenous fat emulsion (IFE) and extracor-
poreal membrane oxygenation (ECMO) have been success-
fully used.9-11

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

EKA: data collection, writing the paper. MB: data collection,
writing the paper. SCV: writing the paper. PAS: writing the
paper.

ORCID

Eyad K. Alhaj https://orcid.org/0000-0003-3246-8954

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