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Brugada Pattern in Diabetic Ketoacidosis: A Case Report and Scoping Study

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Abstract Brugada syndrome is a rare cardiac arrhythmia which is associated with right bundle branch block pattern (RBBB) and ST-segment elevation in right precordial leads. SCNA5 mutation is the most common genetic abnormality associated with Brugada syndrome. Brugada pattern not related to genetic mutations has been previously reported in the setting of fever, metabolic conditions, lithium use, marijuana and cocaine abuse, ischemia and pulmonary embolism, myocardial and pericardial diseases. Multiple isolated cases of Brugada pattern associated with diabetic ketoacidosis (DKA) have been previously reported. We here present a case of type 1 Brugada pattern in a 23 year-old-male who presented with DKA. Brugada pattern in DKA is attributed to acidosis and multiple electrolyte abnormalities including hyperkalemia which alter ion channel expression in the heart thus leading to Brugada pattern which subsequently resolved with treatment of DKA. In such patients, Brugada pattern is not reproducible on procainamide induction cardiac electrophysiology study (EPS). Our scoping study demonstrates male predominance 20/22 cases of (DELETE this highlighted area) Brugada pattern in DKA, a finding that is consistent with prevalence of this disease among males.

Keywords: *Brugada pattern, diabetic ketoacidosis*

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1. Introduction

Brugada syndrome is a rare cardiac arrhythmia which is associated with right bundle branch block pattern (RBBB) and ST-segment elevation in right precordial leads [1]. Brugada Syndrome is associated with life-threatening arrhythmias and/or sudden cardiac death in the absence of underlying structural heart disease [1]. The overall prevalence of a Brugada type electrocardiogram in the United States is approximately 0.43%. It is a familial syndrome that is more likely to present in males. Interestingly male to female disease presentation was noted to be higher in Japan compared to the United States at 9: 1 and 2: 1 ratios respectively [2,3,6]. Multiple genetic mutations have been isolated in cardiac sodium, potassium, calcium, and protein regulatory channels; it is most commonly attributed to one gene: SCN5A, which encodes an α-subunit of a sodium channel, in an autosomal dominant pattern of inheritance [4,5].

Brugada pattern can be "induced" by administration of sodium channel blockers such as flecainide, procainamide, and ajmaline [6]. One international registry lists Brugada pattern in patients with metabolic conditions, mechanical compression, ischemia and pulmonary embolism, myocardial and pericardial disease, and ECG modulation [7]. Furthermore, Brugada pattern has been noted in patients in Diabetes Ketoacidosis (DKA) in the setting of extreme electrolyte disturbances including hyperkalemia, hyponatremia, hyperglycemia, and acidosis; it resolves with the appropriate and timely management of the DKA [8]. Flecainide, a potent class IC antiarrhythmic, failed to successfully "induce" Brugada in a case which initially presented with this pattern in the setting of DKA [8]. We here present a case of DKA with a Brugada pattern on initial ECG which resolved with treatment of DKA and underlying electrolyte disturbances, serial ECGs performed showed a rapid resolution of this pattern.

2. Case Report

A 23-year-old Afro-Caribbean male with a past medical history of tobacco use and type 1 Diabetes Mellitus since nine years of age presented with DKA. Patient's diabetes

was managed by continuous insulin infusion for nine years however he reported non-adherence due to insulin pump malfunction over the past week. He reported nausea, several episodes of vomiting, fatigue, polydipsia, and a non-productive cough at home without any polyuria or dysuria at the time of presentation.

On arrival patient's vitals presented as blood pressure of 103/52 mm Hg, heart rate of 110 beats per min, afebrile, respiring at 30 breaths per minute saturating 92%. On physical exam, the patient appeared lethargic with labored breathing. The laboratory findings on the day of admission are summarized in Table 1.

Admission electrocardiogram revealed atrial fibrillation, right bundle branch block (RBBB), and coved ST-segment elevations in leads V1-V3 consistent with a Brugada pattern (image 1). A bedside echocardiogram showed no wall motion or valvular abnormalities, no pericardial effusion, and the ejection fraction was estimated to be 60%.

The patient was treated for DKA with subsequent resolution of hyperkalemia, hyponatremia, hyperglycemia, and metabolic acidosis. Initial labs revealed sodium 103 mmol/L, potassium 8.6 mmol/L, random blood sugar 1313 mg/dl, bicarbonate 5.5 mmol/L, blood urea nitrogen 62 mg/dl, and serum creatinine as 2.69 mg/dl. Repeat labs within 6-9 hours of admission showed significant improvement as shown in Table 1. ECG performed within two hours demonstrated complete resolution of atrial fibrillation and the Brugada pattern. The day after presentation the patient's ECG has returned completely to baseline with complete resolution of all changes and his labs improved further to Na 134 mmol/l, K 4.1 mmol/l, random blood sugar 259 mg/dl, bicarbonate 11 mmol/l, blood urea nitrogen 48 mg/dl, creatinine 1.78 mg/dl. He did not have any life-threatening arrhythmias on telemetry monitoring and subsequent imaging with a transthoracic echocardiogram showed no structural changes. CHA_2DS_2-VASc Score for atrial fibrillation stroke risk was calculated as one and therefore anticoagulation was not initiated after discussing risks and benefits with the patient.

3. Discussion

Brugada pattern is a unique entity, unlike Brugada syndrome, where certain physiologic changes can predispose to life-threatening arrhythmias and sudden cardiac death in the absence of known underlying genetic mutations and/or structural heart disease. Though it is not clear why patients in DKA develop a Brugada pattern, the proposed mechanism revolves around inactivation of the cardiac sodium channels secondary to hyperkalemia and acidosis [9]. Male predominance can be attributed to a more prominent outward current-mediated action potential found in the right ventricular epicardium of males [9]. The pattern resolves immediately with the treatment of the underlying disturbance associated with DKA and was seen within two hours of treatment in our case. Table 3 lists a number of similar cases where Brugada pattern presented after an initial presentation of DKA.

Table 1. Laboratory trends from admission to 24 hours post treatment

	0 (hrs)	6-9 (hrs)	24 (hrs)
Venous blood pH	6.95	7.36	7.44
Bicarbonate (mmol/L)	5.5	6.0	11.0
Sodium (mmol/L)	103	124	134
Potassium (mmol/L)	8.6	4.7	4.1
Random Blood Glucose (mg/dl)	1313	612	259
Blood Urea Nitrogen (mg/dl)	62	58	48
Creatinine (mg/dl)	2.69	2.04	1.78
B-hydroxybutyrate (mmol/L)	>8.00	-	-
Troponins (mmol/L)	0.02	-	0.02

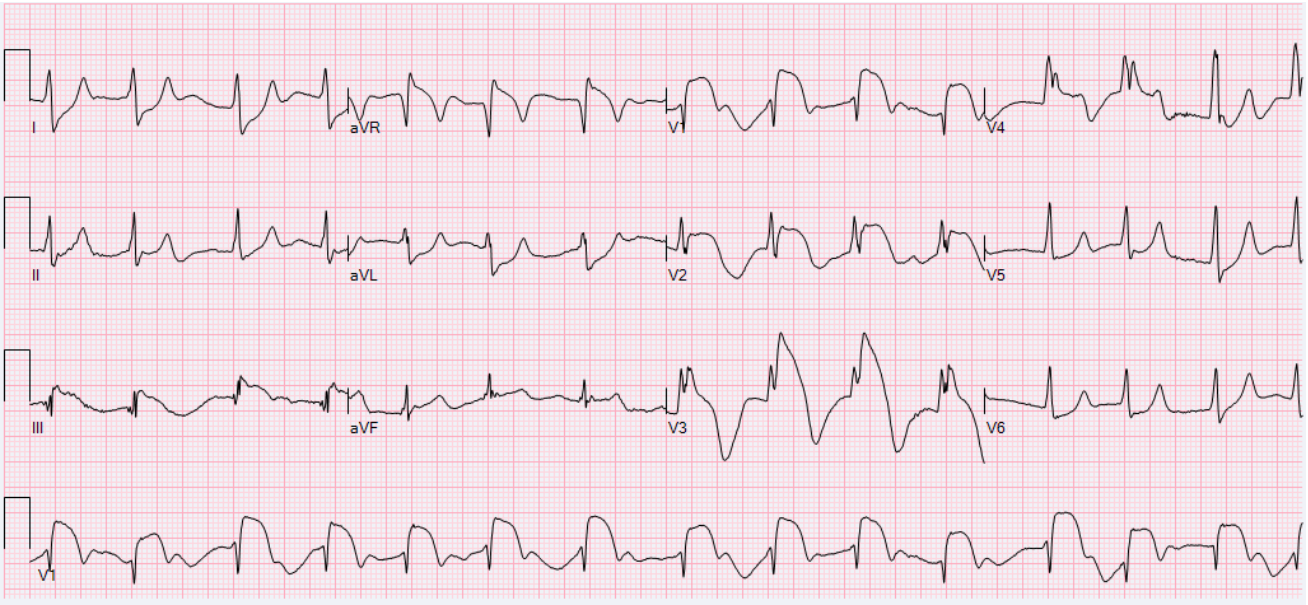

Image 1. Patient's initial ECG: Atrial fibrillation and coved ST-segment elevations with RBBB consistent with a Type 1 Brugada pattern

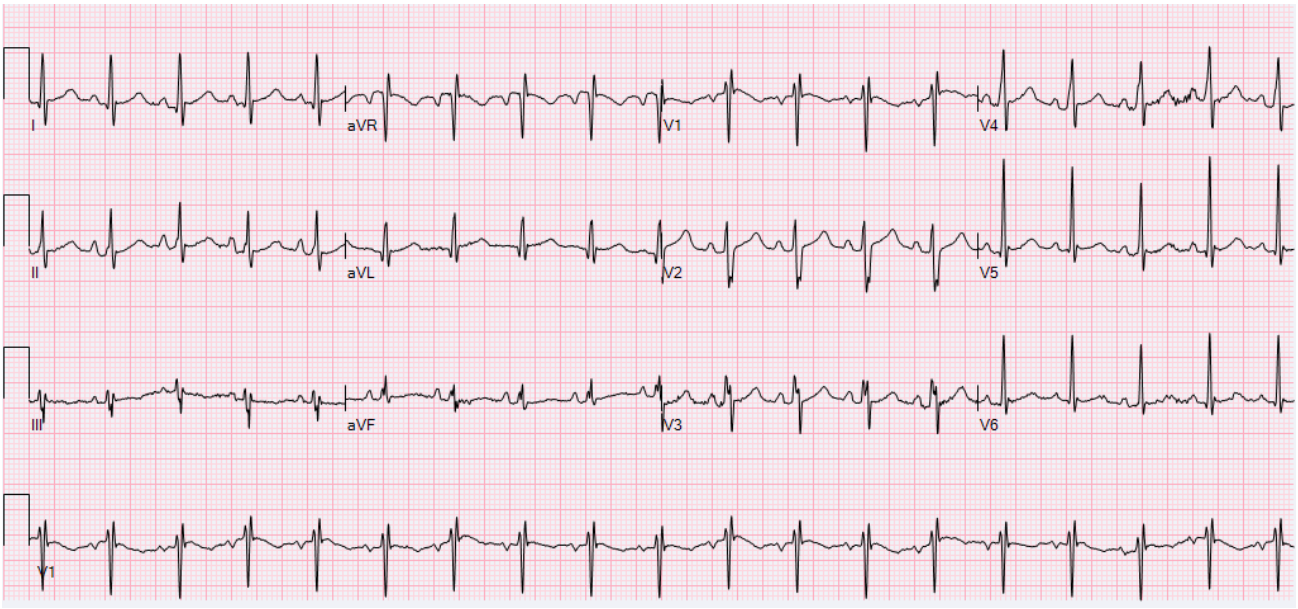

Image 2. ECG obtained two hours after initiation of DKA management showing conversion into normal sinus rhythm and resolution of Brugada pattern

Table 2. Other Precipitating Factors in Literature

Schizophrenia [10]
Fever [11-16]
Illicit Drugs: Cocaine Abuse [17,18] Marijuana [19,20,21,22]
Medications: Lithium [23,24,25], SSRI [26,27], Diphenhydramine [28[, TCA [29]
Anatomic Compression - Rheumatoid Arthritis [30]
Adrenocortical insufficiency [31]

Table 3. Literature review of 22 cases of Brugada-type Pattern in DKA

Author	Age	Sex	pH	Glucose (mg/dl)	Sodium (mmol/L)	Potassium (mmol/L)	Bicarbonate (mmol/L)	Echo
1978, Chawla et al. [32]	48	Male	7.09	840	-	8.3	6.8	-
1981, Cohen et al. [33]	38	Male	6.94	1206	123	7.5	4	-
1983, Johnson et al. [34]	48	Male	7.17	900	130	5.8	5	-
1988, Simon et al. [35]	59	Male	7.06	1664	127	8.1	5	-
1992, Kamimura et al. [36]	45	Male	7.01	1827	127	7.2	5	-
1996, Sweterlitsch et al. [37]	46	Male	6.97	1586	132	7.9	<5	-
1998, YH Lim et al. [38]	59	Female	6.74	1021	133	7.2	<5	-
2003, Milionis et al. [39]	65	Male	7.07	485	102	8.3	13.7	-
2004, Kovacic et al. [8]	38	Male	6.96	1232	105	7	<5	normal
2004, Wang et al. [40]	38	Male	7.21	839	-	7.9	-	-
2005, Cook et al. [41]	60	Male	-	543	128	8.4	19	-
2011, Postema et al. [42]	59	Male	7.21	828	-	6.9	7.3	normal
2012, Ersan et al. [43]	26	Male	7.2	620	-	7.7	-	normal
2013, Franco et al. [44]	44	Male	-	501	-	7.4	6	Normal LV systolic function and wall motion
2015, Arunothayaraj et al. [45]	35	Male	6.97	-	-	7.6	-	-
2016, Maheshwari et al. [46]	65	Male	-	-	-	6.8	-	-
2017, Omar et al. [47]	31	Male	7.05	1090	126	8.4	2.8	-
2017, Dendramis et al. [48]	72	Male	7.35	-	130	8.7	22.6	Normal wall motion, EF: 60%
2017, Mehta et al. [49]	62	Female	6.87	634	-	-	2	-
2018, Alanzalon et al. [50]	18	Male	6.97	1128	120	7.7	<7	-
2018, Alanzalon et al. [50]	7	Male	7.14	313	124	4.4	<7	-
2018, Abrahim et al. [51]	47	Female	-	-	-	6.7	-	-

Table 4. ECG findings from the 22 reported cases

Author	ECG
1978, Chawla et al. [32]	ST-segment elevation in V1-V2 and AVL, tall peaked T waves in V3 -V6
1981, Cohen et al. [33]	irregular rhythm, no P waves, peaked T waves I, II, AVL, V4-V6, Q waves in V3R to V2, broad R wave V3R to V2 w/ wide S waves in left precordial, and ST elevation V3R to V3
1983, Johnson et al. [34]	sinus tachycardia, right axis deviation, broadened QRS, right bundle branch block pattern, peaked T waves and ST elevation in AVL, V1-V3
1988, Simon et al. [35]	Normal sinus rhythm, ST elevation in V1-V3, peaked T waves, broad flat P waves, right bundle branch block, and LAFH
1992, Kamimura et al. [36]	sinus tachycardia, QRS widening, right bundle branch block, Q waves V1-V2, ST elevation in V1-V3, peaked T-waves in V4-V6
1996, Sweterlitsch et al. [37]	ST elevation anteroseptal leads. No Q waves. An intraventricular conduction similar to a right bundle-branch block with a delayed rightward voltage
1998, YH Lim et al. [38]	Sinus tachycardia. pathologic Q-waves with ST elevation in V1-V3
2003, Milionis et al. [39]	Absent P waves widened QRS "sine wave" in leads: I, II, V5-V6, ST depression segments, and tall peaked T waves V3-V6
2004, Kovacic et al. [8]	Irregular supraventricular rhythm, right axis deviation, right bundle branch block, ST elevation precordial leads
2004, Wang et al. [40]	Sinus tachycardia and ST elevation in leads V1-V3. Peaked T waves in II, III, aVF, and V3-V6
2005, Cook et al. [41]	No p-waves accelerated junctional rhythm. ST elevation in V1-V2 and ST depression in I, aVL, and V4-V6. ST-depression and T-wave inversion in the inferior wall leads.
2011, Postema et al. [42]	Convex ST elevation in right precordial leads
2012, Ersan et al. [43]	Right bundle branch block, peaked T waves, ST elevation in V1–V3
2013, Franco et al. [44]	“coved” ST elevation in V1 and V3 and “saddleback” ST elevation in lead V2
2015, Arunothayaraj et al. [45]	Brugada pattern, ST elevation in V1-V2, peaked T and flat P waves
2016, Maheshwari et al. [46]	>2 mm coved ST elevation with T-wave inversion in leads V1 and V2
2017, Omar et al. [47]	Hyperacute T waves, Brugada type 1, prolonged QRS, ST elevation inV1-V2, downsloping ST, and symmetric T wave inversions.
2017, Dendramis et al. [48]	Sinus bradycardia and a “coved” type-1 Brugada pattern in V1- V2 and a “saddleback” type-2 in V3-V4
2017, Mehta et. al. [49]	Type 1 Brugada pattern in V1-V2, with ST depressions in the anterolateral leads
2018, Alanzalon et al. [50]	Type 1 brugada pattern
2018, Alanzalon et al.[50]	Type 1 brugada pattern
2018, Abrahim et al. [51]	ST elevation V1-V3 and associated T wave inversions

Table 5. Mean and median values of the collected data from table 3a

	Mean	Median
Age (years)	45.90	46.5
pH	7.054	7.055
Glucose (mg/dl)	958.7	870
Sodium (mmol/L)	123.6	127
Potassium (mmol/L)	7.424	7.60
Bicarbonate(mmol/L)	7.7	5.5

Other potential triggers of Brugada pattern are summarized by cases in Table 2. There has been a high prevalence in a patient diagnosed with schizophrenia, however, the association was not found to have a correlation with sodium channel blocking medications often prescribed in schizophrenia [10]. Febrile states have been theorized to modulate sodium channels and provoke Brugada syndrome perhaps through missense mutations of T1620M [11] and several cases have been reported to date [12,13,14,15,16]. Illicit drugs such as cocaine and marijuana have been listed as triggers. Cocaine a potent sympathomimetic was isolated as a trigger in two case reports with one case repeatedly presenting with Brugada pattern within 2-5 days of cocaine use. Mechanisms have been proposed to involve both increases in overall norepinephrine and sodium blocking properties predisposing to arrhythmias [17,18]. Marijuana’s proposed mechanism is due to vagal stimulation by cannabis which has previously seen to trigger Brugada Syndrome [19,20,21,22]. Medication use with SSRI and Lithium as triggers have been reported as well. Both medications are potent blockers of cardiac sodium channels and can provoke Brugada syndrome in patients [23,24,25,26,27]. Medication overdose including diphenhydramine and tricyclic antidepressant has been isolated as triggers of arrhythmias and proposed mechanism is attributed anticholinergic overactivity [28,29]. One reported case has found a rare complication of hemopericardium and tumor formation from underlying rheumatoid arthritis which compressed the ventricle and unmasked a Brugada pattern [30]. Adrenocortical insufficiency which is commonly associated with hyperkalemia can decrease resting membrane potential inactivating cardiac sodium channels and produce similar findings [31].

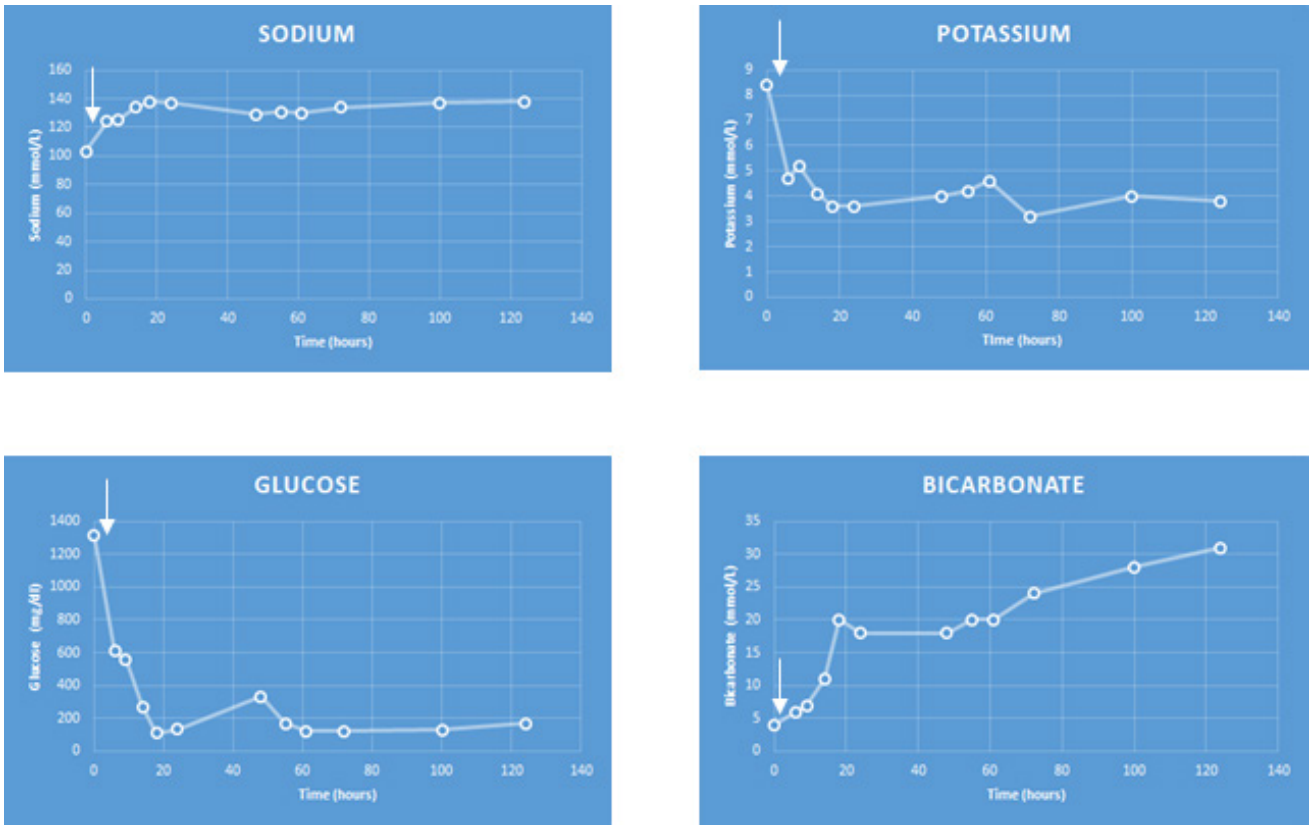

Figure 1. Trends of sodium, potassium, glucose, and bicarbonate levels (white arrow indicating time line at which Brugada pattern resolved)

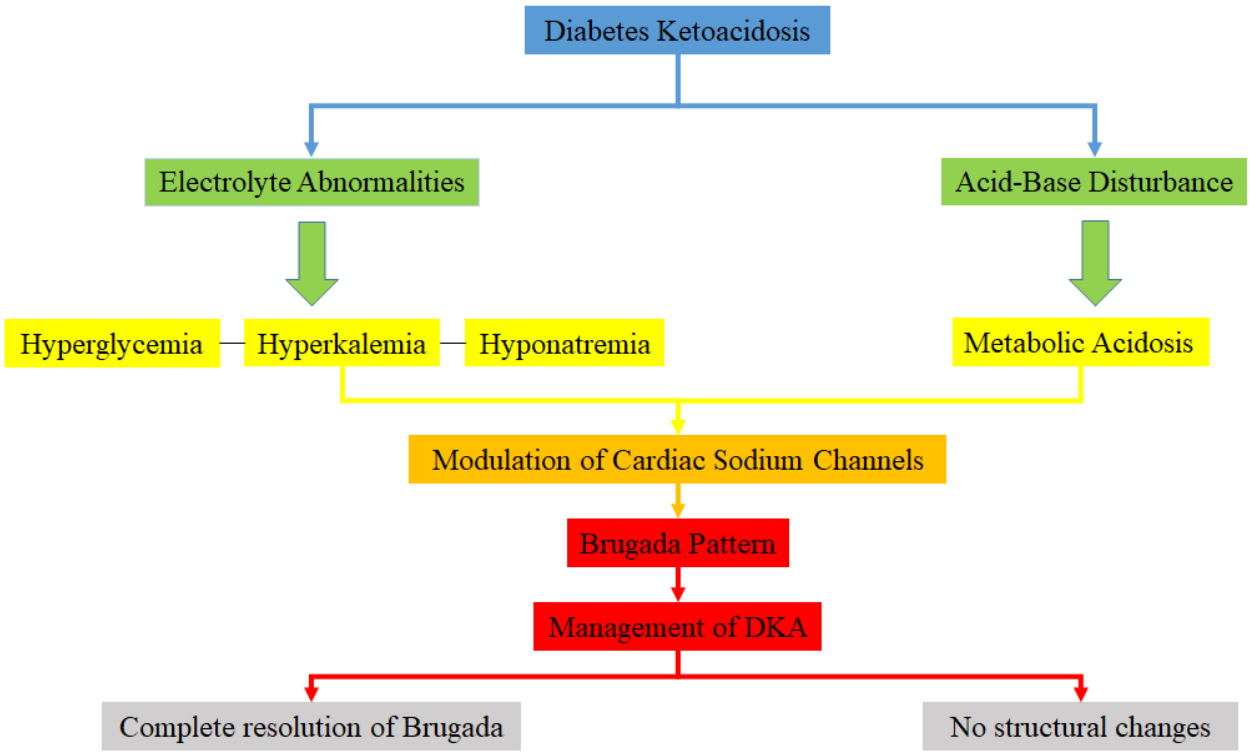

Figure 2. Etiopathogenesis of Brugada pattern in DKA

In our case, the Brugada pattern was elucidated in diabetic ketoacidosis with hyperkalemia and acidosis. In Table 3, we have reviewed 22 case reports that presented with a Brugada pattern in DKA initially concerning for a myocardial infarction. Similar to our case, the pattern resolved with the timely management of the underlying electrolyte disturbances. Furthermore, our review re-demonstrated a male predominance at a ~7.33: 1 male-female ratio with ages ranging from 7 to 72. Mean and median values were calculated for age, pH, glucose, sodium, potassium, and bicarbonate levels. The average age was found to be ~45.9 years, average pH on admission

at 7.054, average glucose 958.7 mg/dl, average potassium for 7.424 mmol/L, average sodium 123.6 mmol/L, and average bicarbonate to be 7.7 mmol/L. Our case showed that our patient was lower than average in age, pH, and bicarbonate levels and greater than average in potassium and glucose levels. Some of the cases evaluated the cardiac function with transthoracic echocardiograms and they demonstrated no structural or valvular abnormalities which were consistent with our own echocardiogram findings. Others performed cardiac catheterization with normal coronaries. All cases had complete recovery once the DKA was treated and they bring to attention great importance in the close outpatient follow-up of diabetic management.

4. Conclusion

DKA may precipitate Brugada pattern in middle-aged men with no genetic predisposition. Brugada pattern will completely resolve on appropriate management of DKA. Acidosis and hyperkalemia in DKA promote electrophysiologic changes in the cardiac sodium channels producing a Brugada pattern/ coved ST-elevation mimicking acute myocardial infarction on an ECG. Brugada induction in electrophysiology laboratory often fails to induce Brugada pattern in such patients suggesting the Brugada pattern in these patients is due to DKA.

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