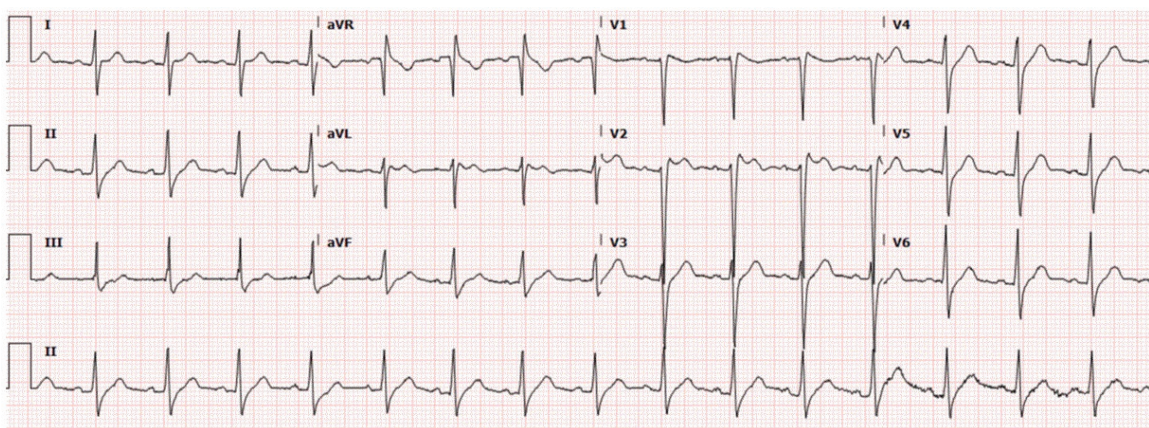
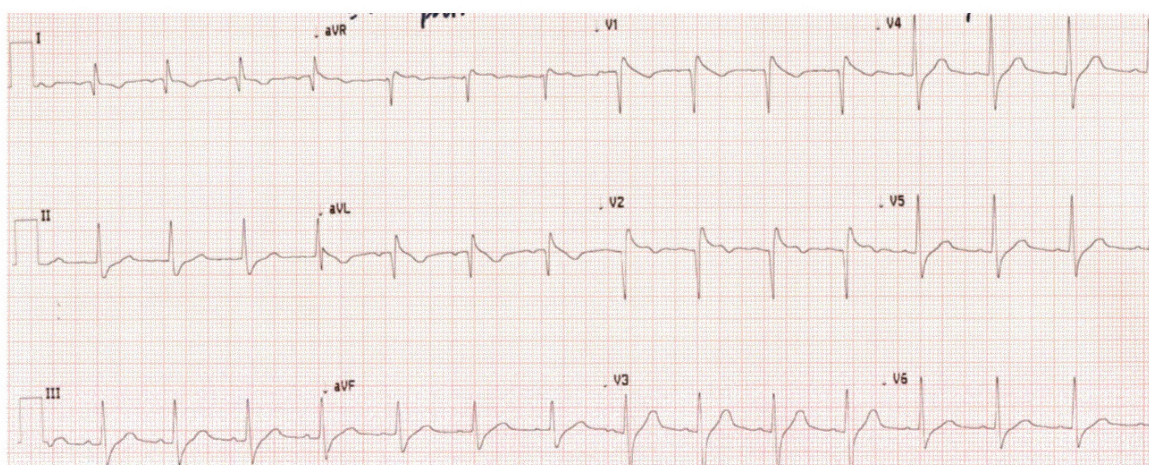


**Figure 1.** Type 1 Brugada pattern on ECG strip of patient upon presentation; BP upon presentation: 204/102 mmHg



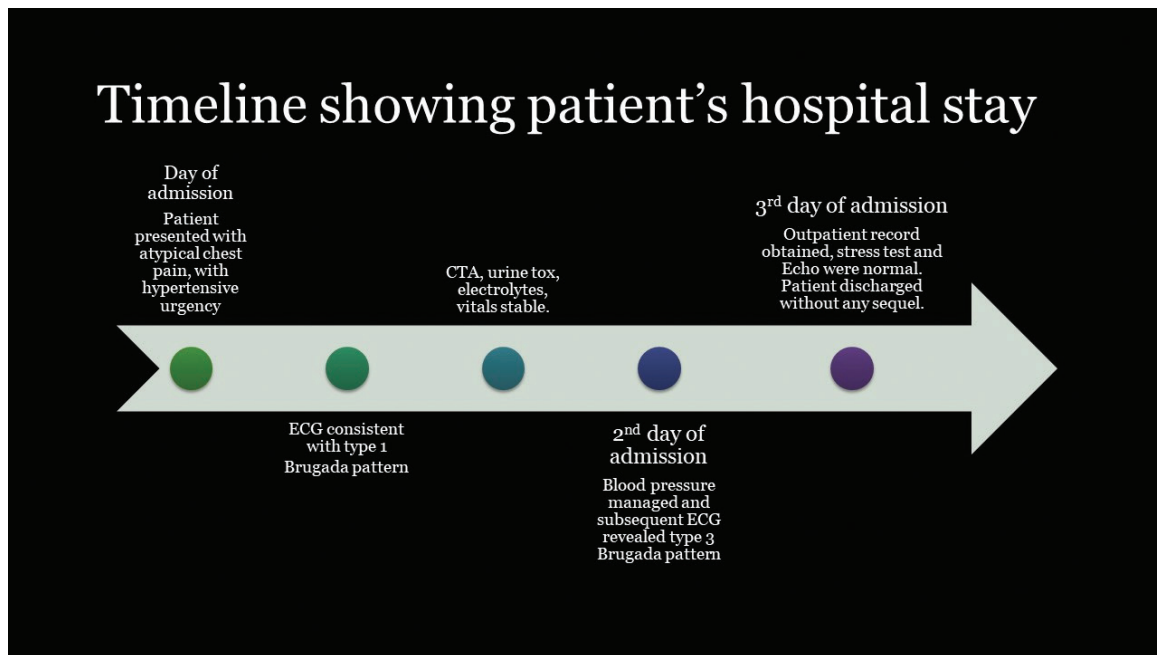
**Figure 2.** Type 3 Brugada pattern on ECG strip of patient upon lowering his blood pressure. BP at the time of ECG: 132/76 mmHg



**Figure 3.** Type 3 Brugada pattern on ECG strip of patient. BP at the time of ECG: 119/77 mmHg.

chest pain resolved as well. Subsequent ECG revealed type 3 Brugada pattern as shown in Figure 2. On further reviewing the patient's outpatient records, he recently had an abnormal ECG showing type 3 Brugada pattern when his blood pressure was 119/77 mmHg as shown in Figure 3. His treadmill stress test was negative for ischemia. Similarly, a transthoracic echocardiogram

demonstrated an ejection fraction of 59% and normal RV systolic function. Moreover, a 30-day event monitor revealed sinus rhythm. He declined provocative testing as well as genetic testing for the *SCN5A* gene mutation. Nonetheless, since BrPh was seen on ECG after lowering his blood pressure, he was discharged from hospital without any sequel. His hospital course is shown in Figure 4.



**Figure 4.** Timeline showing patient's hospital stay.

## Discussion

We presented a rare case of typical type 1 Brugada pattern in ECG in a young man who presented with uncontrolled hypertension and atypical chest pain. ECG findings remarkably changed to type 3 Brugada pattern when the blood pressure was normalized. BrS is an inherited arrhythmic disorder which develops secondary to the *SCN5A* gene mutation, and which encodes for the cardiac sodium channel [1]. Such channelopathy can lead to life-threatening arrhythmias and SCD. Sleep deprivation, fever, antiarrhythmic drugs, tricyclic antidepressants, alcohol and cocaine use, and electrolyte abnormalities, including hypokalemia, hyperkalemia, and hypercalcemia, are some of the common triggers known to incite SCD in patients with BrS [4-6]. Three ECG patterns can be noted in patients with BrS. Classic Brugada ECG (type 1), also referred to as a “coved type” ECG, is described as a RBBB, prominent ST segment elevation ( $\geq 2$  mm) with inverted T-wave in precordial leads V1 and V2 with little to no interruption of the isoelectric line, which is our patient's ECG results [3]. Type 2 Brugada ECG, also known as “saddle back” pattern, has RBBB with ST elevation and biphasic T-wave configuration in the precordial leads. Type 3 Brugada ECG has a similar ECG pattern to that of type 2, the only difference being ST segment is elevated by 1 mm or more in the type 2 pattern and less than 1 mm in the type 3 pattern [3,7]. Furthermore, it is crucial to recognize the conditions that can induce BrPh without the presence of true BrS since BrPh is not associated with life-threatening arrhythmia and SCD [8]. In our patient's case, we believe he had BrPh, which was modulated by his uncontrolled hypertension. A good history is essential to differentiate BrS from BrPh. It is equally important to exclude other causes of ST-segment

elevation. In the absence of electrolyte abnormality, ischemic, or structural heart disease, our patient's ECG during sinus rhythm revealed RBBB, normal QT interval, and persistent ST-segment elevation in precordial leads V1 and V2 during the episodes of elevated blood pressure, followed by resolution of the ECG pattern upon resolution of elevated blood pressure. Since the patient had evidence of type 1 Brugada pattern captured on a 12-lead ECG with no known history of documented ventricular fibrillation, polymorphic ventricular tachycardia, a family history of SCD at <45 years old, coved-type ECGs in family members, syncope, or nocturnal agonal respiration, we concluded that his ECG changes could be a manifestation of BrPh rather than BrS [7]. For similar reasons, an electrophysiology study was not performed to establish a diagnosis. To the best of our knowledge, this is the first case reporting hypertension-induced classical type 1 BrPh. Our case emphasizes the importance of recognizing the Brugada pattern in ECGs in young adults so that the inciting agents could be identified earlier and managed accordingly. While few medications, including quinidine and hydroquinidine, are known to offer some benefit to patients with BrS, the presence of spontaneous Brugada type 1 ECG pattern in the occurrence of symptoms warrants an implantable cardioverter-defibrillator [9]. In conclusion, early recognition and prompt intervention are of utmost importance in patients who demonstrate a Brugada pattern on ECG as it might be the first sign of impending SCD. The management of BrPh, on the other hand, lies in treating the underlying stress leading to the ECG change.

## Conclusion

BrPh is an acquired form of BrS that can resemble Brugada-like ECG pattern in the presence of an underlying

stressor. This is the first case reporting a suspected BrPh as a result of uncontrolled hypertension in which ECG tracing transforms from type 1 Brugada pattern to type 3 Brugada pattern with management of underlying stressors of hypertension.

**What is new?**

BrPh is an acquired form of BrS that can resemble Brugada-like ECG pattern in the presence of an underlying stressor. This is the first case reporting a suspected BrPh as a result of uncontrolled hypertension in which ECG tracing is transformed from type 1 Brugada pattern to type 3 Brugada pattern with management of underlying stressors of hypertension.

**Acknowledgment**

We thank our my co-authors.

**List of Abbreviation**

BrPh Brugada phenotype  
 BrS Brugada syndrome  
 SCD Sudden cardiac death

**Consent for publication**

Informed consent was obtained from the patient to publish this case.

**Ethical Approval**

Ethical approval is not required from the institute for case reports.

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**Summary of the case**

1	<b>Patient (gender, age)</b>	African-American Male, 28 years old
2	<b>Final diagnosis</b>	Brugada phenotype
3	<b>Symptoms</b>	Atypical chest pain
4	<b>Medications</b>	Labetalol, hydrochlorothiazide
5	<b>Clinical procedure</b>	None
6	<b>Specialty</b>	Internal medicine and cardiology