BELHASSEN VENTRICULAR TACHYCARDIA- A DIAGNOSTIC CHALLENGE IN EMERGENCY ROOM: CASE REPORT

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ABSTRACT

Fascicular Ventricular Tachycardia (VT) is a unique clinical syndrome, rarely encountered by physicians. It is also known as Belhassen Syndrome, named after a physician who reported the case in 1981. The condition, accounts for 10-15% of total idiopathic VT and the rhythm is sensitive to calcium channel blocker. First described in 1979, the diagnosis of this syndrome remains challenging, as the electrocardiogram (ECG) changes may be incorrectly diagnosed as Supraventricular Tachycardia (SVT) with aberrant conductions.

We described a patient who presented to Emergency Department with palpitation. The difficulty in diagnosis and management is illustrated in the report as he was initially misdiagnosed as SVT with resistance to initial standard treatment. This case report also described wide complex tachycardia algorithms to assist physician in daily clinical practice. Therapeutic options in managing this rare syndrome were also discussed.

INTRODUCTION

Fascicular Ventricular Tachycardia (VT), classified as idiopathic VT, is a rare syndrome encountered by physicians. It was first described in 1979 and accounts for 10-15% of total idiopathic ventricular tachycardia. The diagnosis remains challenging, as the electrocardiogram (ECG) may be classified as Supraventricular Tachycardia (SVT) with aberrant conductions[2]. The mainstay of treatment for termination of fascicular VT is calcium channel blocker [1]. The case below described the challenges in diagnosing Fascicular VT and its treatment.

CASE

A 17-year-old boy presented to Emergency Department(ED) with sudden onset of palpitation which persisted for one hour while he was playing football. It was not associated with chest pain or shortness of breath. Further history revealed he was hospitalised with two similar episodes in the past. He was referred for further cardiac investigations at National Heart Institute, Kuala Lumpur which he had defaulted. On arrival, patient was triaged to the resuscitation area. He was alert with warm peripheries. His vital signs were: BP 100/65 mmHg, HR 202/min, oxygen saturation of 96% under room air and he was afebrile. There were no signs of acute heart failure. Continuous cardiac monitoring showed a wide complex tachycardia with a rate of 220/min. ECG was performed and interpreted as SVT with right bundle branch block (RBBB) (Figure 1) by the emergency resident..

Subsequent to the initial assessment, he was treated with intravenous (IV) Adenosine 6 milligram (mg) and subsequent two doses of 12 mg of IV Adenosine. However the abnormal rhythm persisted. A loading dose of IV Amiodarone 300mg was administered and he was referred to cardiology team for further management. His cardiac enzymes, electrolytes and chest radiograph were normal. He was admitted to Coronary Care Unit (CCU) for continuation of Amiodarone infusion (900mg/23 hours) and close monitoring. Although his symptoms improved, monitored electrograph persistently showed a wide complex tachycardia. As there were no signs of circulatory instability, electrical cardioversion was not performed.

On second day of admission, patient's ECGs were reviewed by the attending cardiologist. A diagnosis of fascicular VT was made and IV Verapamil 2.5mg was administered. He subsequently reverted to sinus rhythm (figure 2 & 3) following IV Verapamil. Echocardiogram was performed which showed a structurally normal heart. He was started on oral Verapamil 40mg thrice a day and discharged on day 3 of admission, with an appointment to cardiology clinic for an electrophysiology study.

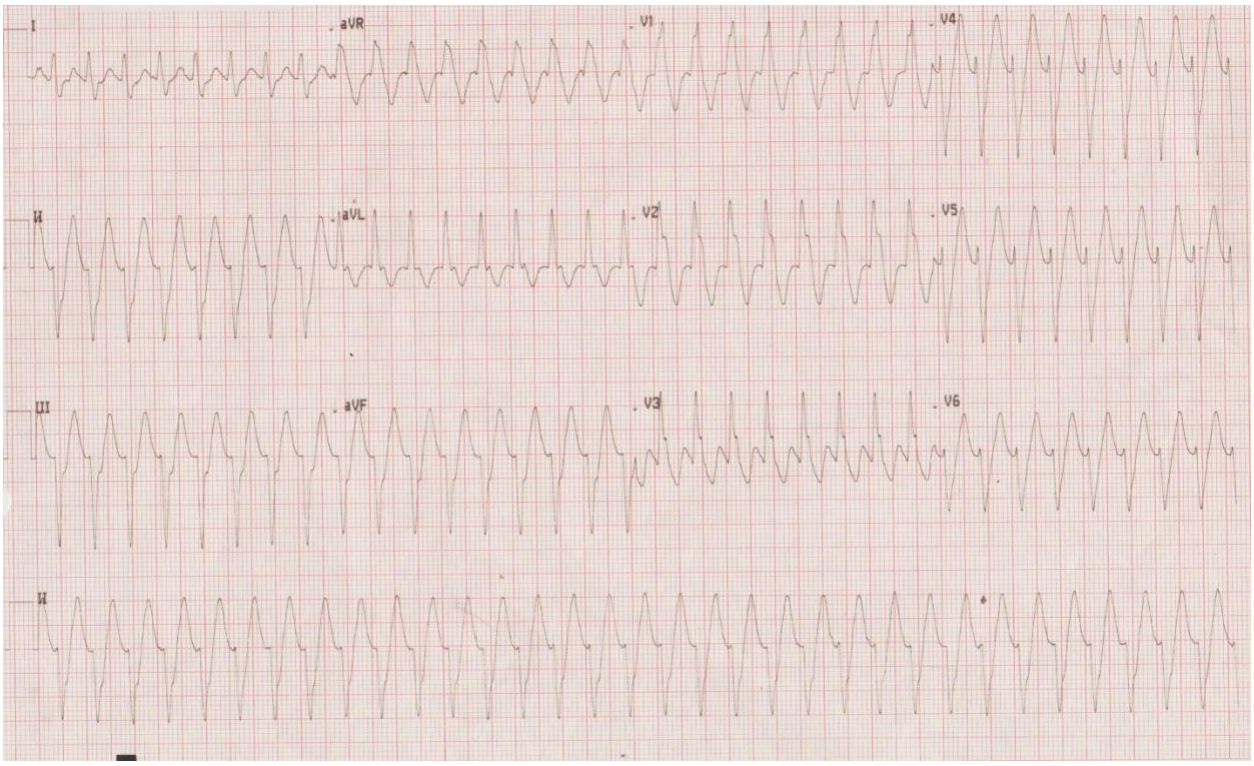


Figure 1: ECG during presentation

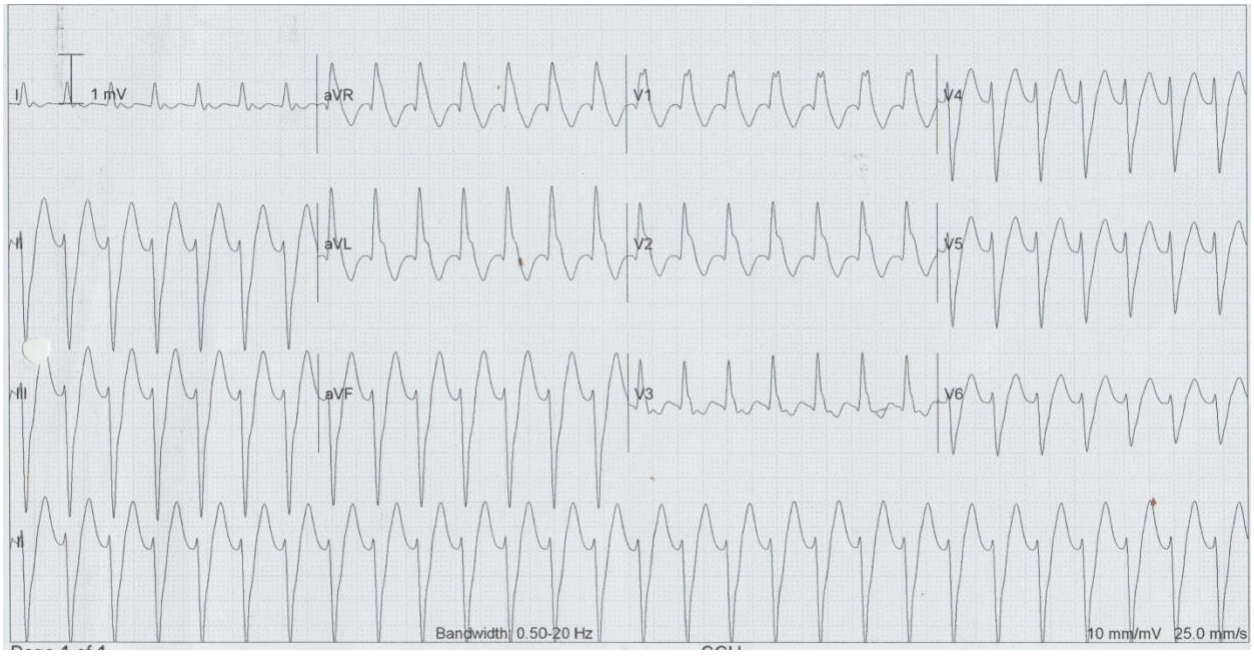


Figure 2: ECG pre IV Verapamil

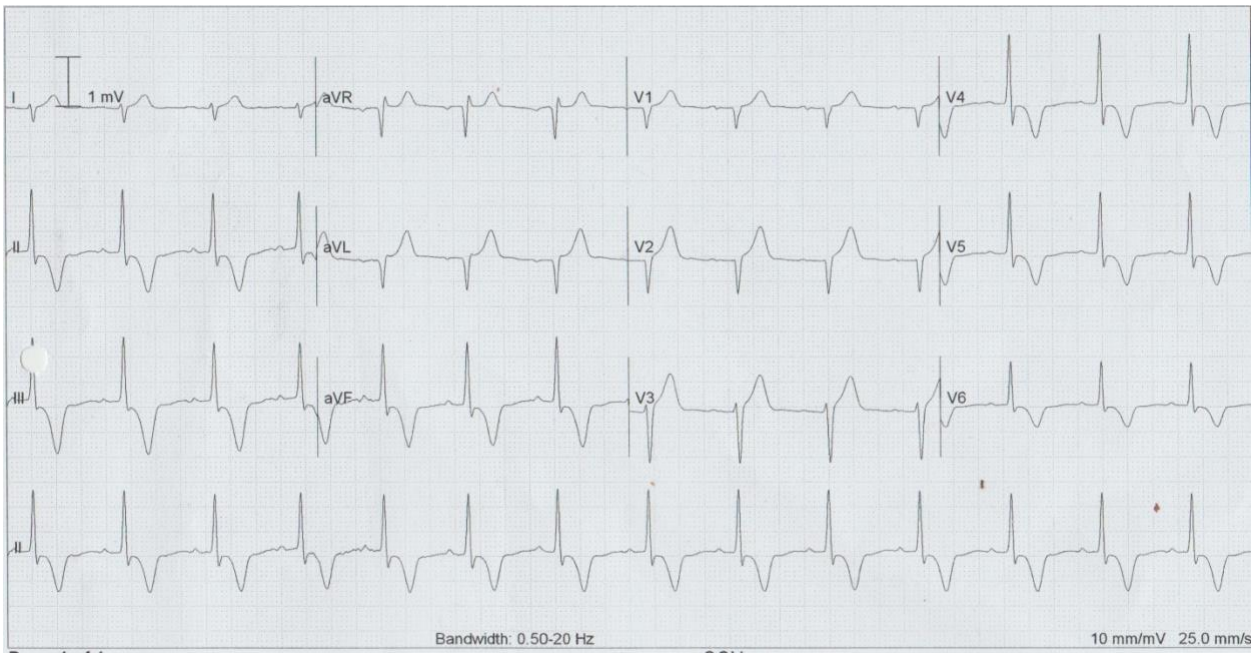


Figure 3: ECG post IV Verapamil

DISCUSSION

Wide complex tachycardia (WCT) is a diagnostic challenge encountered by physicians who have to make a decision on emergent treatment. Two major differential diagnoses for WCT are VT and SVT with aberrant conductions. VT accounted for approximately 80% of those with WCT, followed by SVT with aberrant conductions[3]. As these two diagnoses accounted for almost 95% of total patients with WCTs, the main workup during presentation would be to differentiate them. Differentiating these two entities has important diagnostic and therapeutic implications. For example, in patients presenting with VT and have mistakenly diagnosed with SVT, treating these patients with Verapamil may lead to disastrous consequences such as severe hypotension.

Few algorithms have been produced and validated to differentiate between VT and SVT with abberant conductions based ECG chracteristics . Algorithms that are widely used include Brugada algorithms and Vareckei's AVR algorithms (Figure 4 & 5). However, using these algorithms in clinical management requires practice and additional vigilance. Approximately 10% of patients with WCTs are still misdiagnosed, thus overreliance to these algorithms are potentially dangerous[4]. This conundrum in diagnosing is similar to our patient where his ECGs showed a pattern similar with SVT with aberrant conductions. However his symptoms were resistant to Adenosine, a drug well known to abort episodes of SVT in most patients. The rarity of encountering these abnormal ECGs among physicians showed the importance to understand this uncommon syndrome in avoiding misdiagnosis.

Figure 4: Brugada Algorithm (Reproduced from Andras Vareckei[4])

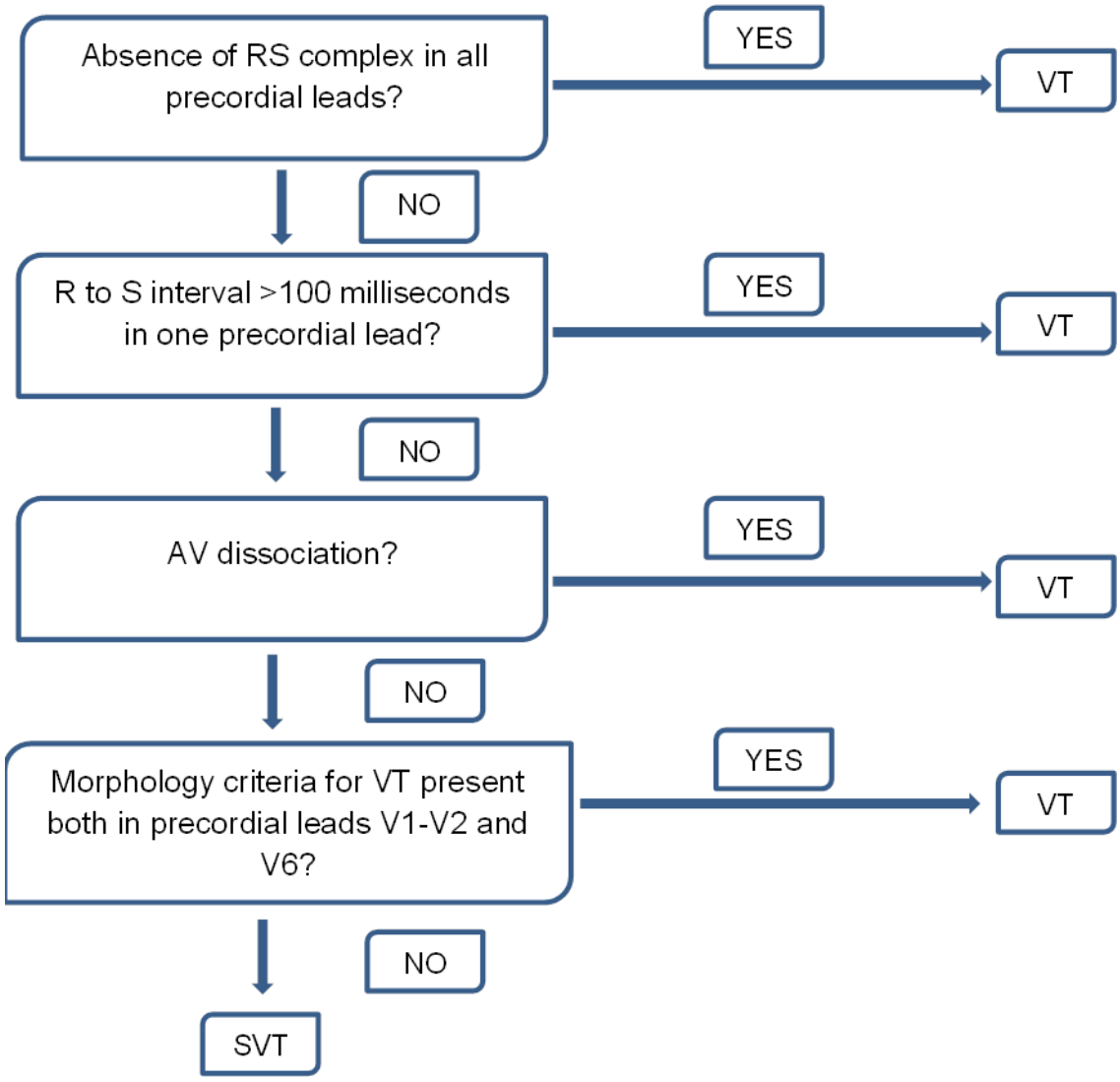
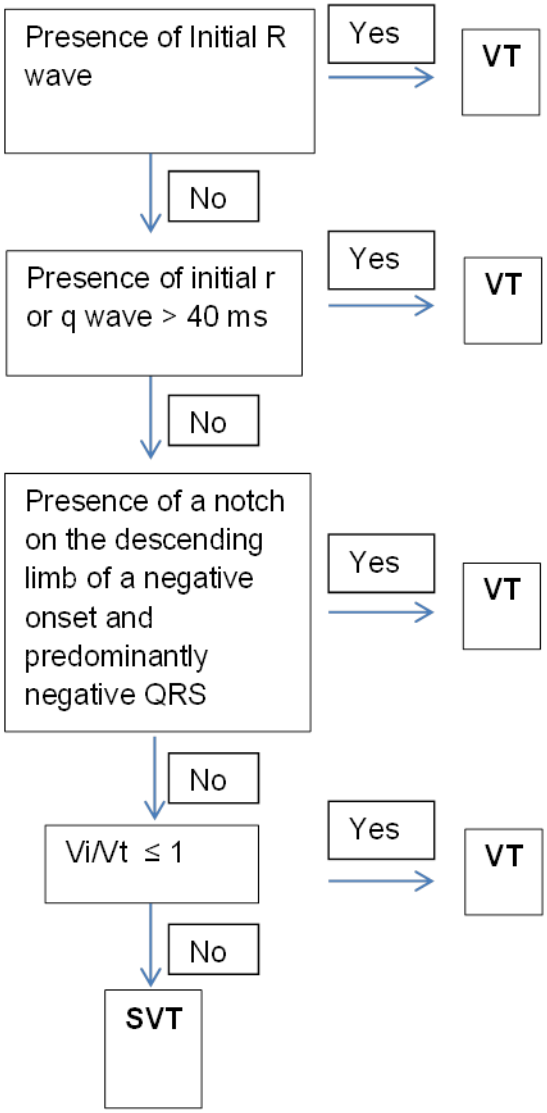


Figure 5: Vareckei's AVR Algorithm (Reproduced from Andras Vareckei[4])



Fascicular VT, as the name suggests, arises from the fascicles of the left bundle branch. It is an idiopathic VT in which the heart structure is normal without myocardial scarring. It accounts for 10-15% of total idiopathic VT[1]. It is also known as Belhassen-type VT or Verapamil sensitive VT. Fascicular VT can be further classified into few types; left posterior fascicular VT, left anterior fascicular VT and left upper septal VT. This differentiation is based on the fascicles involved as well as the corresponding ECG morphology produced. By far the commonest type is the left posterior fascicular VT, representing 90% of fascicular VT[5].

In terms of epidemiology, it is more common in male than female, and affects younger individuals. Similarly, our patient is a young, healthy man without structural cardiac pathology diagnosed prior or during presentation. They usually presented with palpitations, chest discomfort or dizziness which may or may not be triggered by exertions. Rarely, patient may also presents with syncope or associated with tachycardia-related cardiomyopathy.

In 1981, Belhassen was first to report the responsiveness of a patient with no demonstrable structural heart disease presented with ECG pattern suggestive of fascicular VT to IV Verapamil, hence the term Belhassen VT or Verapamil sensitive VT was used to describe this entity[6]. Subsequently few case series of patients presented with similar tachycardia (RBBB and axis deviation pattern) without any bundle branch blocks during sinus rhythms showed comparable responsiveness to calcium antagonists[7, 8]. This leads to further research and the mechanism was found to be due to reentrant circuit. The proposed reentrant circuit consists of an area of slow conduction which is sensitive to Verapamil that forms the orthodromic limb in the LV septum from base to apex, with the retrograde limb using the Purkinje network[9].

Fascicular VT can easily be confused with an SVT with aberrant conduction, even when the wide complex tachycardia algorithms for differentiating VT and SVT are used. The pattern of ECG commonly encountered is RBBB with axis deviation[9]. The QRS complex is relatively narrow. The left posterior fascicular VT which is the commonest type, classically demonstrates RBBB with left axis deviation (arises close to the left posterior fascicle). On the other hand, the left anterior fascicular VT demonstrates an RBBB with right axis deviation (arises close to left anterior fascicle). An incomplete RBBB (QRS duration 100-110ms), or occasionally LBBB and normal axis will be demonstrated in septal VT (arises from region of upper septum) which is the least common type. Similarly in our patient, his initial ECG was clinically misdiagnosed as SVT with RBBB. Presence of AV dissociation, capture or fusion beats in ECG suggests VT instead of SVT with aberrant conduction.

The treatment for the syndrome can be categorised into acute and long term management. In general, the prognosis for patients with fascicular VT is good. In rare cases, patient may have tachycardia-related cardiomyopathy. Catheter ablation therapy has a probable of 90% cure rate[9]. In acute setting, IV Verapamil has been highly effective in terminating the rhythm[6, 7, 8, 9]. In our patient, a small dose of IV Verapamil (2.5mg) was used and successful in reverting to sinus rhythm. In patients who opted for long-term medical therapy in place of catheter ablation, oral Verapamil has been shown to be effective in preventing future recurrence. Adenosine has been ineffective in terminating the rhythm. The rhythm is also unresponsive towards Lignocaine, a drug commonly used for VT. Some studies have reported that Amiodarone to be equally effective in treating fascicular VT, however in our patient, it proved to be futile[10].

CONCLUSION

Fascicular VT is a specific entity that presents with an ECG that can be mistakenly identified as SVT leading to incorrect treatment. Misdiagnosis is common, as illustrated in this case. Therefore, understanding the common clinical presentations, ECG findings as well as demographic trends of the syndrome will lead to correct diagnosis and treatment with good outcome.

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