EKGs and Pacemakers

Belhassen Ventricular Tachycardia: A Case Study

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PRIME POINTS

• Many patients with Belhassen ventricular tachycardia experience no signs or symptoms with episodes of tachycardia.

• Belhassen ventricular tachycardia occurs more often in males 15 to 40 years than in females of those ages.

• Nursing care includes monitoring for dysrhythmia and its effects, assisting patients with treatment procedures, administering medication and monitoring its effects, and providing discharge education.

A patient who had experienced ventricular tachycardia was admitted to the coronary care unit (CCU) in normal sinus rhythm. According to the admitting physician’s notes, the dysrhythmia presumably was caused by myocardial ischemia. The dysrhythmia was described as paroxysmal sustained ventricular tachycardia with a left posterior fascicular block. After coronary artery disease was ruled out, an electrophysiologist diagnosed Belhassen ventricular tachycardia.

The prevalence of Belhassen ventricular tachycardia is not known, and little is available in the literature on the care of patients with this abnormality. In this case study, we summarize the information in the literature, present the case study, and describe the nursing care provided.

Summary of the Literature

Belhassen ventricular tachycardia is a unique ventricular tachycardia (also known as idiopathic fascicular ventricular tachycardia, verapamil-sensitive ventricular tachycardia, and reentrant left ventricular tachycardia) first described by Bernard Belhassen and colleagues in 1981.1 Belhassen ventricular tachycardia occurs more frequently in males 15 to 40 years old than in females of the same ages, and it may occur more commonly with exercise than at rest.2 Although approximately 90% of ventricular tachycardias occur in patients with anatomically abnormal hearts,2 most patients with Belhassen ventricular tachycardia have anatomically normal hearts—hence one of the names: idiopathic fascicular ventricular tachycardia.

The dysrhythmia may involve slow-calcium-channel–dependent tissue or a reentry mechanism of the distal fascicles of the left bundle branch.3

Patients with Belhassen ventricular tachycardia may experience no signs or symptoms. Those who do have signs or symptoms most often report shortness of breath, fatigue, or dizziness. Syncope and sudden death are “extremely rare.”2

The diagnosis is based on the unique electrocardiographic (ECG) features of the ventricular tachycardia: paroxysmal, usually with a left posterior fascicular block, a right
bundle branch block, and left axis deviation (Figure 1).

Treatment can be ablation or pharmacological therapy. The ventricular tachycardia may be difficult to induce during electrophysiological studies, with or without pharmacological provocation. Some investigators5-7 suggest that induction of Belhassen ventricular tachycardia may be more difficult after pharmacological intervention and recommend ablation as the initial treatment. If the ventricular tachycardia can be induced, radiofrequency ablation may be performed. For younger patients, because of the potential need for long-term antidysrhythmia therapy and the possible side effects of the medications used, ablation is the preferred treatment.5 Typical pharmacological treatment is verapamil. Intravenous verapamil has been effective in treating the ventricular tachycardia, and oral verapamil has had variable results in preventing recurrences.5 Propranolol (Inderal) has been effective both in treating Belhassen ventricular tachycardia and in preventing recurrences. Vagal maneuvers may also be effective. Use of adenosine has not been successful.

**Case Study**

A 73-year-old man came to the emergency department because of intermittent dizziness and near-syncope. Some of the symptomatic episodes occurred with activity, but others occurred while he was sitting in a chair. For 2 to 3 weeks before he came to the hospital, he had experienced shortness of breath after as little as 15 minutes of walking. He had been experiencing the dizziness and near-syncope for 1 to 2 days. He described associated symptoms of 2/10 chest pressure, nausea, shortness of breath, and paresthesias of both upper extremities. Vital signs were normal, with heart rate 80/min, blood pressure 134/72 mm Hg, respirations 14/min, and oxygen saturation 99% by pulse oximetry.

The patient’s medical history included hypertension, hyperlipi-
demia, osteoporosis, gastroesophageal reflux disease, benign prostatic hypertrophy, allergic rhinitis, and childhood asthma. He had smoked 2 packs of cigarettes a day for 25 years until he quit 30 years ago. His surgical history included knee replacement, hip replacement, cervical spine surgery, cataract surgery, and 3 transurethral resections of the prostate. Before this admission, he had worked 3 days a week. He said he had experienced episodes of dizziness for at least the previous 50 years; he thought the dizziness was related to the hypertension. His medications included the following:

- Alendronate (Fosamax) 70 mg/wk
- Amlodipine (Norvasc) 5 mg/d
- Lisinopril 10 mg/d
- Olmesartan (Benicar) 40 mg/d
- Simvastatin (Zocor) 20 mg/d
- Aspirin 81 mg/d
- Calcium supplements, dose unknown

The patient was allergic to chocolate, lobster, and strawberries. The only reported family history of note was the death of his father at age 50 years due to myocardial infarction.

In the emergency department, cardiac monitoring showed ventricular tachycardia. The initial treatment was intravenous lidocaine. The cardiac rhythm changed to sinus bradycardia at 50/min to 60/min with bigeminy. The patient was asymptomatic. Findings on chest radiography were normal. The results of laboratory studies were within the normal range except for slightly low serum levels of calcium and magnesium (see Table).

The patient reported that because of his shortness of breath, he had had cardiac nuclear imaging a few weeks before admission. The results of that imaging indicated inferior wall scarring.

Three hours after he came to the emergency department, the patient was admitted to the CCU; the diagnosis was acute coronary syndrome and dysrhythmia. ECG monitoring revealed normal sinus rhythm with frequent nonsustained ventricular tachycardia. The patient was receiving a continuous intravenous infusion of lidocaine at 2 mg/min. His heart rate was 70/min to 90/min in normal sinus rhythm, and all other vital signs were stable. The patient reported continued chest pressure, which was relieved with 1 sublingual nitroglycerin tablet.

On the basis of the earlier nuclear imaging study, the cardiologist attributed the patient’s dysrhythmias and other symptoms to ischemia; therefore, the patient was treated for acute coronary syndrome. Lidocaine was discontinued, and heparin and eptifibatide (Integrilin) intravenous infusions were started. The patient was prepared for and consented to cardiac catheterization with possible percutaneous coronary intervention. Cardiac catheterization revealed that the coronary arteries had no obstructive disease; left ventricular ejection fraction was 65% (normal 55%-75%).

During the next 24 hours of continuous cardiac monitoring in the CCU, nursing staff documented numerous episodes of ventricular tachycardia. The duration of the tachycardia ranged from as few as 4 beats to many hours. The patient

### Table Results of laboratory studies

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal range</th>
<th>Patient’s value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematocrit, male, %</td>
<td>42-52</td>
<td>40.5</td>
</tr>
<tr>
<td>Hemoglobin, male, g/dL</td>
<td>13-17</td>
<td>14</td>
</tr>
<tr>
<td>White blood cell count, male, cells/µL</td>
<td>3500-10 800</td>
<td>7400</td>
</tr>
<tr>
<td>Platelets, male, x10³/µL</td>
<td>140-400</td>
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</tr>
<tr>
<td>Sodium, mEq/L</td>
<td>136-146</td>
<td>128</td>
</tr>
<tr>
<td>Potassium, mEq/L</td>
<td>3.5-5.3</td>
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</tr>
<tr>
<td>Chloride, mEq/L</td>
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<td>94</td>
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<tr>
<td>Carbon dioxide, mEq/L</td>
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</tr>
<tr>
<td>Urea nitrogen, mg/dL</td>
<td>8.0-20</td>
<td>18</td>
</tr>
<tr>
<td>Serum creatinine, mg/dL</td>
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<tr>
<td>Glucose, mg/dL</td>
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<td>127</td>
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<tr>
<td>Calcium, mg/dL</td>
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<tr>
<td>Magnesium, mEq/L</td>
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<td>Troponin I, ng/mL</td>
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<td>Qualitative 0.02</td>
</tr>
<tr>
<td></td>
<td>Stat 0.2</td>
<td>Qualitative 0.03</td>
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<tr>
<td>International normalized ratio</td>
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</tr>
</tbody>
</table>

* SI units: To convert urea nitrogen to mmol/L, multiply by 0.357; serum creatinine to µmol/L, multiply by 88.4; glucose to mmol/L, multiply by 0.0555; calcium to mmol/L, multiply by 0.25; magnesium to mmol/L, multiply by 0.50.*
did not experience symptoms during the dysrhythmic events. An electrophysiologist was consulted.

A 12-lead ECG recorded during a sustained episode of ventricular tachycardia revealed a wide-complex tachycardia with a right bundle branch block and a -155° axis deviation (Figure 2). The diagnosis was Belhassen ventricular tachycardia. The physician chose to treat the patient with a β-blocker because the drug could provide effective treatment of the ventricular tachycardia and prevent recurrences (rather than verapamil, which would provide treatment of the ventricular tachycardia but was less likely to prevent recurrences). Metoprolol 5 mg intravenous push was administered, and the ventricular tachycardia converted to normal sinus rhythm (Figure 3). The patient was then treated with metoprolol 12.5 mg orally every 6 hours. After 24 hours, no further ventricular tachycardia was observed.

An electrophysiologic study was performed to evaluate the cardiac conduction system. The patient also consented to ablation of areas diagnosed as contributory to the ventricular tachycardia. The electrophysiologic study revealed no evidence of conduction abnormality. Ventricular tachycardia could not be induced despite multiple attempts. The patient was offered the option of stopping treatment with the β-blocker and repeating the electrophysiologic procedure. He had not experienced side effects with the β-blocker and chose not to have the electrophysiologic study repeated. The dose of metoprolol was changed to 25 mg/d, and the patient was discharged.

Nursing Care

The patient was admitted to the CCU with a diagnosis of acute coronary syndrome and possible myocardial infarction. Morphine, supplemental oxygen, and aspirin had been administered previously per protocol. In the emergency department, the patient was given intravenous nitroglycerin, and a 12-lead ECG was performed. Nursing care in the CCU continued to include supplemental oxygen, intravenous nitroglycerin, and continuous ECG monitoring for ST-segment changes and dysrhythmias. The nurses assigned to the patient assessed respiratory rate and rhythm, oxygen saturation via pulse oximetry, heart rate, blood pressure, pain, and urine output every hour and as needed. Blood pressure and heart rate were within the reference ranges at admission, so an intravenous β-blocker (metoprolol) was administered. Within the first 24 hours,
an angiotensin-converting enzyme inhibitor (captopril) was also administered, with no complications. Breath sounds, neurological status, heart sounds, peripheral pulses, and skin color and temperature were assessed every 4 hours and as needed per CCU protocol. Serum levels of cardiac enzymes (eg, troponin I, creatine kinase MB) were determined every 6 hours until the levels peaked.

Nursing considerations after cardiac catheterization included, but were not limited to, assessing vital signs, pedal pulses, and the groin site every 15 minutes for the first hour, every 30 minutes for the next hour, and every hour for at least 2 hours thereafter. Checks of the groin site included assessing the femoral artery site used for the cardiac catheterization to ensure that the site was soft and without bleeding or hematoma. The patient was maintained on bed rest for 4 hours. Staff nurses were prepared to report to a physician and to treat any abnormal findings, including swelling, bleeding, loss of pedal pulse, and pain.8

Education on the importance of keeping the leg that had been accessed for the catheterization straight for 3 to 6 hours after the procedure and the head of the bed elevation at 15° or less was explained. The patient was taught how to splint the site by holding slight pressure over the groin if he felt the need to cough, sneeze, or vomit.

When acute coronary syndrome and myocardial infarction were ruled out on the basis of the cardiac catheterization, the primary diagnosis became dysrhythmia. Nursing care then focused primarily on assessing for any ECG changes, monitoring for ventricular dysrhythmias, assessing vital signs, and monitoring results of electrolyte assays (especially potassium and magnesium levels). Electrolytes were replaced as needed to maintain normal levels, and antidysrhythmics were administered. The ECG was monitored continuously for desired effects as well as side effects.

The patient was taught about the importance of adhering to his medication schedule and follow-up appointments with his physicians. Signs and symptoms of the dysrhythmia (eg, lightheadedness, syncope, fatigue, palpitations) and how to access the emergency medical system were reviewed. He was taught how to take his pulse, and he demonstrated that he could do so accurately.
Although the emphasis on education was directed toward the patient, his wife was included to ensure that members of his immediate support system knew about the new changes in his regimen and specific signs and symptoms.

Discussion

The patient’s history was typical for Belhassen ventricular tachycardia: male sex and beginning in his late teens, intermittent dizziness and rare syncope. This admission was the first time health care providers were able to document a dysrhythmia. The patient’s hemodynamic status was stable, in either normal sinus rhythm or ventricular tachycardia. The 12-lead ECG obtained during ventricular tachycardia showed an irregular wide-QRS-complex tachycardia with a right bundle branch block and a -155° axis deviation (normal, 0° to +90°), making it difficult to differentiate between right and left axis deviation. The diagnostic workup revealed an anatomically normal heart.

The cardiologist ordered a β₁-selective intravenous β-blocker (metoprolol) because of the patient’s history of childhood asthma. Treatment with the drug converted his cardiac dysrhythmia to normal sinus rhythm with a right bundle branch block and a slight left axis deviation (-4°). The patient then was treated with metoprolol orally. In the electrophysiology laboratory, the dysrhythmia could not be induced, so no ablation was performed. Because he was tolerating the β-blocker and had had no further episodes of the ventricular tachycardia, the patient elected to continue his current pharmacological treatment and defer a repeat electrophysiologic study. The health care team was supportive of the patient’s decision. Before discharge, the patient and his family were taught about medications, emergency management, and follow-up care with physicians.

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None reported.

References
