CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 34-2024: A 69-Year-Old Man with Dyspnea after Old Myocardial Infarction

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PRESENTATION OF CASE

Dr. Patrick Malecha (Medicine): A 69-year-old man with a history of myocardial infarction, ischemic cardiomyopathy, and an implantable cardioverter–defibrillator (ICD) was evaluated because of dyspnea during an urgent visit at an outpatient primary care clinic affiliated with this hospital.

The patient had been in his usual state of health until 2 days before the current presentation, when he felt unwell while working. He had dyspnea on exertion (while moving and stocking boxes and when walking home from work), with baseline exertional dyspnea that occurred after ascending two to three flights of stairs. The next day, the patient had palpitations and felt lightheaded throughout the day. The palpitations continued, and he was evaluated by his primary care physician at an outpatient primary care clinic affiliated with this hospital.

On a review of systems, the patient reported nausea with intermittent retching, anorexia (eating only a slice of cheese for the day), and fatigue. He stated that his right eye was "out of focus," but he also noted that he had had this symptom in the past. He reported no chest pain or pressure. Although he had often had angina in the past, the patient noted that he had not had any episodes in the past 2 years and that he had had only one episode of substernal discomfort, which occurred while he was walking and had lasted 45 seconds. In addition, he had had no presyncope or loss of consciousness, ICD discharge, edema, orthopnea, paroxysms of nocturnal dyspnea, fever, known sick contacts, diarrhea, weight change, abdominal pain or bloating, dysuria, myalgia, arthralgia, rash, bleeding symptoms, or focal neurologic symptoms. He had not missed any doses of medications, and his last ICD check was 3 months before the current presentation.

The patient's medical history was notable for premature coronary artery disease, with a first occurrence of myocardial infarction in his third decade of life. In his fifth decade of life, he had undergone coronary-artery bypass grafting (CABG) and subsequent stenting of the native left anterior descending coronary artery. Coronary angiography, which was performed 2 years earlier for evaluation of angina, showed that the bypass graft and stent were patent but that severe na-

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N Engl J Med 2024;391:1633-41.
DOI: 10.1056/NEJMcpc2402505

tive coronary disease was present (and was unchanged from an earlier study). The patient's medical history also included ischemic cardiomyopathy with chronically reduced ejection fraction and scarring of the inferior and inferoposterior left ventricle, and ventricular tachycardia that had been treated with antiarrhythmic medication and an ICD. The ICD had been placed 3 years before the current presentation during an admission at this hospital for syncope that was followed by in-hospital cardiac arrest due to ventricular tachycardia, which required multiple external defibrillations. Thereafter, the patient received treatment with oral amiodarone; however, device interrogation revealed intermittent ventricular tachycardia at rates of 109 to 117 beats per minute that persisted over the next 8 months, and he underwent endocardial ablation of ventricular tachycardia in the scar of the mid-inferior wall. Six months after ablation, he was readmitted to this hospital because of nausea and dizziness that were due to recurrent sustained slow ventricular tachycardia (110 beats per minute), for which he was treated with intravenous lidocaine and antitachycardia pacing.

In addition, the patient's medical history included bioprosthetic mitral valve replacement concurrent with CABG, which had been complicated by prosthetic valve deterioration with mixed stenosis and mitral regurgitation, and culminated in transcatheter valve-in-valve replacement 2.5 years before the current presentation. He also had a history of hypertension, pulmonary embolism (4 years before the current presentation), iron-deficiency anemia, a colonic polyp, and prolonged encephalopathy after CABG. Medications included aspirin, coumadin, furosemide, metoprolol, lisinopril, dapagliflozin, ranolazine, atorvastatin, ferrous gluconate, paroxetine, and clonazepam. Treatment with amiodarone had previously been associated with elevations in liver-function test results. At the patient's last office visit with an electrophysiologist 6 months before the current presentation, device interrogation showed no ventricular tachycardia and the heart rate was 60 beats per minute; treatment with amiodarone was discontinued.

Dr. Emily K. Zern: A transthoracic echocardiogram obtained at the same office visit with the electrophysiologist showed a left ventricular ejection fraction of 31% with inferior, posterior, and apical territory dysfunction; right ventricular hypokinesis; a left-to-right interatrial shunt; trace-to-mild aortic insufficiency; a well-seated mitral valve prosthesis with no regurgitation and a mean transmitral gradient of 5 mm Hg; and a right ventricular systolic pressure of 31 mm Hg.

Dr. Malecha: At a primary care visit 5 months before the current presentation, the patient reported bloating. The heart rate was 62 beats per minute and the blood pressure 104/64 mm Hg. The thyrotropin level was normal, the N-terminal pro–B-type natriuretic peptide (NT-proBNP) level 646 pg per milliliter (reference value, <900), the hematocrit level 40.9% (reference range, 41.0 to 53.0), and the low-density lipoprotein cholesterol level 74 mg per deciliter (1.92 mmol per liter; reference range, 50 to 129 mg per deciliter [1.29 to 3.34 mmol per liter]).

The patient had had a routine office visit with his cardiologist 18 days before the current presentation, during which the heart rate was 63 beats per minute and the blood pressure 111/69 mm Hg; the weight was 83 kg. No changes were made to his medications.

Dr. Conor D. Barrett: An electrocardiogram (ECG) obtained at the visit with the cardiologist showed atrial pacing, first-degree atrioventricular delay, right bundle-branch block, and evidence of old inferior, apical, and posterior myocardial infarctions (Fig. 1).

Dr. Malecha: The patient was employed in multiple service jobs. He did not drink alcohol or use tobacco or other substances. He lived with several roommates on the fourth floor of an apartment building in an urban area and climbed stairs daily. His family history was notable for coronary artery disease in his father and stroke in his father, mother, and maternal grandmother.

At the current presentation, the temporal temperature was 35.7°C, the heart rate 124 beats per minute and regular, the blood pressure 84/59 mm Hg while the patient was sitting and 99/67 mm Hg while he was in the supine position, and the oxygen saturation 99% while he was breathing ambient air. The weight was 83 kg. The patient appeared diaphoretic and clammy, and the skin was warm to the touch. He was alert, oriented, and lucid. The previous sternotomy and ICD sites were intact. There was no heart murmur; however, a possible S3 heart sound and minimal basilar rales were observed. The jugular venous pulse was difficult to visualize. A healed appendectomy scar was present, but the abdomen was nontender, the liver was nonpulsatile,

and there was no fluid wave. The legs were warm with mild, symmetric pedal edema.

Laboratory test results were obtained 2 weeks before the current presentation. The sodium level was 139 mmol per liter (reference range, 135 to 145), the potassium level 4.1 mmol per liter (reference range, 3.4 to 5.0), the creatinine level 0.91 mg per deciliter (80.44 μg per liter; reference range, 0.80 to 1.30 mg per deciliter [70.72 to 114.92 μg per liter]), the hematocrit 44.0%, the NT-proBNP 669 pg per milliliter, and the international normalized ratio (INR) 2.9 (reference range, 0.9 to 1.1).

Diagnostic tests were performed, and management decisions were made.

DIFFERENTIAL DIAGNOSIS

Dr. Kevin Heaton: This is a 69-year-old man with a complex history of premature coronary disease, systolic heart failure, cardiac arrest due to ventricular tachycardia that led to ICD placement, persistent ventricular tachycardia, endocardial ablation, and slow ventricular tachycardia. He presented to the primary care clinic with a 2-day history of dyspnea, palpitations, lightheadedness, nausea, retching, loss of appetite, and vision changes in his right eye.

DIAGNOSTIC APPROACH IN THE PRIMARY CARE CLINIC

As a general internist seeing this patient in a time-pressured urgent care visit, I would be daunted by this complicated myocardial and electrophysiologic disease. My initial concern would be that the primary care clinic environment lacked the appropriate tools to manage unstable cardiac conditions, and I would quickly determine whether triage is needed for treatment in the emergency department.

My attention would be drawn to this patient's history of ventricular tachycardia and ischemic cardiomyopathy — two dynamic and dangerous conditions. However, I would also be resistant to anchoring only on these alarming features. At this point, I would pause to consider other diagnostic possibilities.

ACUTE DYSPNEA

The differential diagnosis of acute dyspnea is extensive in a patient of this age with known systolic heart failure, malignant arrhythmias, pulmonary embolism, iron-deficiency anemia, and valve replacements. The causes of dyspnea usually fall into one of the following categories: cardiac, pulmonary, hematologic, metabolic, and other causes. On initial review of this patient's case, many possibilities on the differential diagnosis

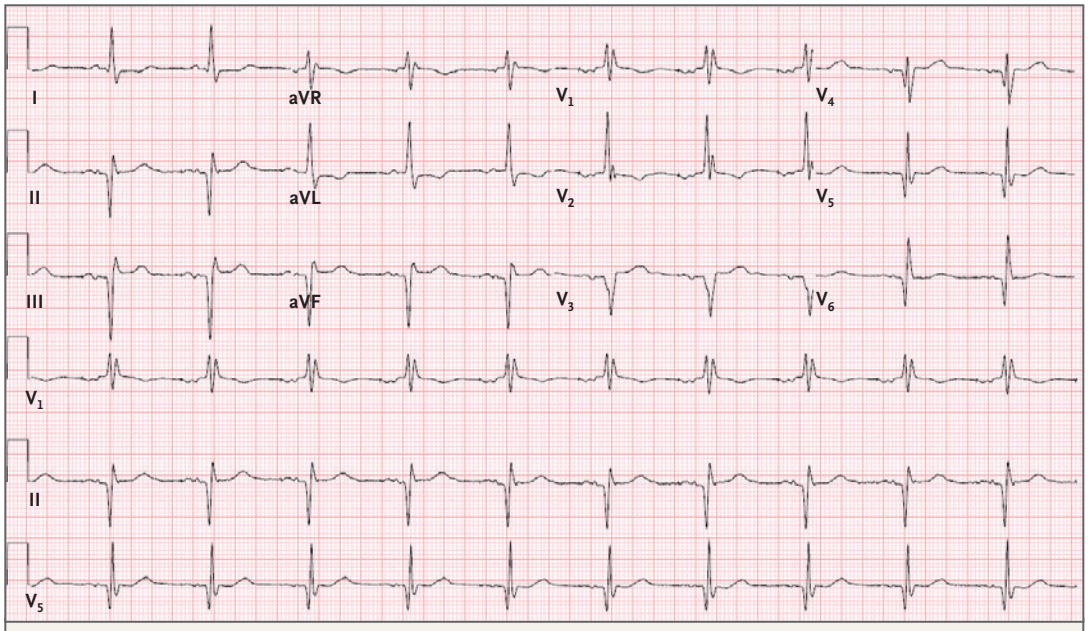

Figure 1. Initial Electrocardiogram.

The initial electrocardiogram, which was obtained 18 days before the current presentation, shows atrial pacing, first-degree atrioventricular delay, right bundle-branch block, and evidence of old inferior, apical, and posterior myocardial infarctions.

remain, with the exception of chronic obstructive pulmonary disease and smoking-related lung disease. However, the rapid progression of illness and the presence of abdominal symptoms narrow the list of possible causes. Pulmonary embolism can probably be ruled out, given the use of anticoagulant therapy, the recent therapeutic INR, and the fact that the patient had not missed any doses of medication. None of the features of this patient's presentation would support a diagnosis of pneumothorax. Acute anemia is an unlikely cause of the acute dyspnea in the absence of any history of overt bleeding. Although acute hemolysis related to the patient's valve replacements could be a possible explanation, he did not report darkened urine or have examination findings consistent with anemia.[1] Pulmonary or cardiac infections are unlikely causes in the absence of fever, cough, sputum production, or chest or pleuritic pain. Decompensated heart failure could have caused this patient's symptoms but typically involves a more gradual onset of symptoms; however, flash pulmonary edema, valve failure, and acute coronary syndrome can lead to a rapid onset of symptoms.

Arrhythmia is a classic cause of acute dyspnea and, given this patient's medical history, the most obvious diagnosis. Dyspnea, we are told, was followed by palpitations, lightheadedness, and fatigue, which further align with this diagnosis. The nausea, retching, and loss of appetite remain unexplained, but if we find no other facts to sufficiently counterbalance the evidence in support of a diagnosis of arrhythmia, it could also account for these symptoms. The discontinuation of treatment with amiodarone 6 months before the current presentation removed an important pharmacologic protection against ventricular arrhythmia. On the basis of this patient's history, there is a moderate-to-high probability of arrhythmia.

Heart failure is another possible diagnosis. Heart failure with visceral malperfusion or congestion could be consistent with this patient's constellation of symptoms.

PHYSICAL EXAMINATION

The physical examination is useful for investigating specific diagnostic hypotheses. In this case, given the patient's elevated heart rate and low blood pressure, I would start the examination by assessing overall hemodynamic stability. This patient was lucid, a finding that suggests adequate cerebral perfusion. In addition, he had no signs of peripheral hypoperfusion, with warm skin and without a narrow pulse pressure, and there were few indications of substantial volume overload with minimal leg edema and minimal rales. There was a possible S3 sound, but this could be chronic. Moreover, I would struggle to be confident in my own ability to auscultate this subtle examination finding in a patient with tachycardia. I would check for cannon A waves as a sign of atrioventricular dissociation compatible with ventricular tachycardia. The absence of a systolic or diastolic murmur argues against notable prosthetic mitral regurgitation or stenosis. We are not told of a goiter, painful thyroid, or exophthalmos, which would suggest thyrotoxicosis. The presence of diaphoresis introduces the possibility of acute coronary syndrome or unstable arrhythmia. I would assess for signs of anemia (e.g., conjunctival pallor). On physical examination of the abdomen and skin, no signs of infection were present.

RECENT CARDIAC STUDIES

The ECG obtained 18 days before the patient's current presentation showed atrial pacing with evidence of extensive myocardial damage from previous infarctions. The last echocardiogram, obtained 6 months before the current presentation, showed known systolic heart failure, a dilated and hypokinetic left ventricle, a well-seated prosthetic mitral valve, and no other relevant abnormalities. Regular tachycardia remains the pivotal objective diagnostic finding.

Overall, the most notable objective findings in this case are hypotension, regular tachycardia, and the absence of signs of overt heart failure, which together are consistent with a diagnosis of arrhythmia. In the primary care clinic, ECG is the first diagnostic test I would request, which would allow me to rule out ST-segment elevation myocardial infarction (STEMI) and to determine whether this patient has an arrhythmia or sinus tachycardia.

ALTERNATIVE DIAGNOSES

The patient's cardiac history could lead a clinician to ignore nonspecific symptoms such as nausea and unfocused vision. It is therefore important to explore other explanations that might account for these symptoms.

The patient had a history of vague gastrointestinal upset, including bloating. At the current evaluation, he presented with acute gastrointes-

tinal symptoms of nausea, retching, and loss of appetite. We also learned that he had had nausea and dizziness that was associated with an episode of slow ventricular tachycardia. It is important to keep in mind the possibility of smoldering intestinal disease. If a repeat ECG were to show sinus tachycardia, this would increase the relevance of these symptoms. However, in the absence of abdominal pain, weight loss, jaundice, or abnormal findings on abdominal examination, it is unlikely that this presentation could be explained by gastrointestinal disease.

The use of amiodarone can cause thyrotoxicosis by activating latent nodules — triggering Graves' disease — or by causing a destructive thyroiditis. Thyrotoxicosis can occur up to 1 year after discontinuation of amiodarone treatment.[2,3] We are told that the patient discontinued amiodarone therapy 6 months before the current presentation and that laboratory tests performed 2 weeks before the current presentation did not include thyrotropin. There is no mention of goiter or exophthalmos on examination. Given the association of amiodarone-induced thyrotoxicosis and multiple arrhythmias, a thyrotropin level should be obtained, especially if a repeat ECG shows sinus tachycardia.

Nausea can also be a manifestation of toxic effects related to many gastrointestinal irritants or medications, right heart failure, and insults to the central nervous system. There is no evidence of medication-related toxic effects or intracranial or intraocular disease. None of the common causes of nausea would explain the dyspnea or palpitations except as mediated through consequent anemia, sepsis, cardiac injury, or perforated viscus.

The patient's report of his right eye being "out of focus" is an intriguing symptom on first evaluation as a potentially specific clue that leads to a unifying diagnosis (e.g., transient ischemic attack from prosthetic valve endocarditis or thrombus or Graves' ophthalmopathy). The fact that this was a chronic symptom suggests that it is unrelated to the current presentation. An eye out of focus is not a common description associated with amaurosis fugax or retinal emboli. Moreover, the patient had no signs of acute mitral valve dysfunction or murmur or other inflammatory or autoimmune findings that would suggest acute or subacute bacterial endocarditis.

After considering alternative diagnoses that could explain this patient's presenting symptoms, I suspect the most likely diagnosis is slow ventricular arrhythmia.

DR. KEVIN HEATON'S DIAGNOSIS

Slow ventricular arrhythmia.

ECG ASSESSMENT AND DIAGNOSTIC TESTING

Dr. Barrett: ECG was performed during the patient's urgent visit at the primary care clinic. The ECG findings contribute to establishing the electrophysiologic diagnosis of ventricular tachycardia (Fig. 2A). On the basis of the patient's history, the pretest probability of ventricular tachycardia is high. Given his history of old myocardial infarctions and a wide-complex tachycardia, our presumptive diagnosis must be ventricular tachycardia until proved otherwise.

The patient had low-normal systolic blood pressure; however, it is important to note that blood pressure does not help to differentiate between ventricular tachycardia and supraventricular tachycardia. The blood-pressure response to arrhythmia depends on a host of factors, including consequences of atrioventricular dyssynchrony, systolic and diastolic ventricular function, and noncardiac coexisting conditions.

Supraventricular tachycardia with aberrancy, which is the competing diagnosis for a patient with a wide-complex tachycardia, would be a most unlikely diagnosis in this case. Supraventricular tachycardia with aberrancy is caused by supraventricular depolarization reaching the ventricle through an accessory pathway or by a supraventricular tachycardia conducted with a bundle-branch block aberrancy. Various algorithms can be used to assess wide-complex tachycardias to differentiate between ventricular tachycardia and supraventricular tachycardia with aberrancy, the most well-known of which is the algorithm developed by Brugada and colleagues.[4]

The Brugada criteria use a stepwise ECG algorithm (Table 1), in which the diagnosis of ventricular tachycardia is confirmed if any criterion is present. The findings from this patient's ECG met several of the criteria, the first of which being the measurement of the time interval from the R wave to the nadir of the S wave in leads V_4 through V_6, which is approximately 130 msec (a longer duration is in keeping with ventricular

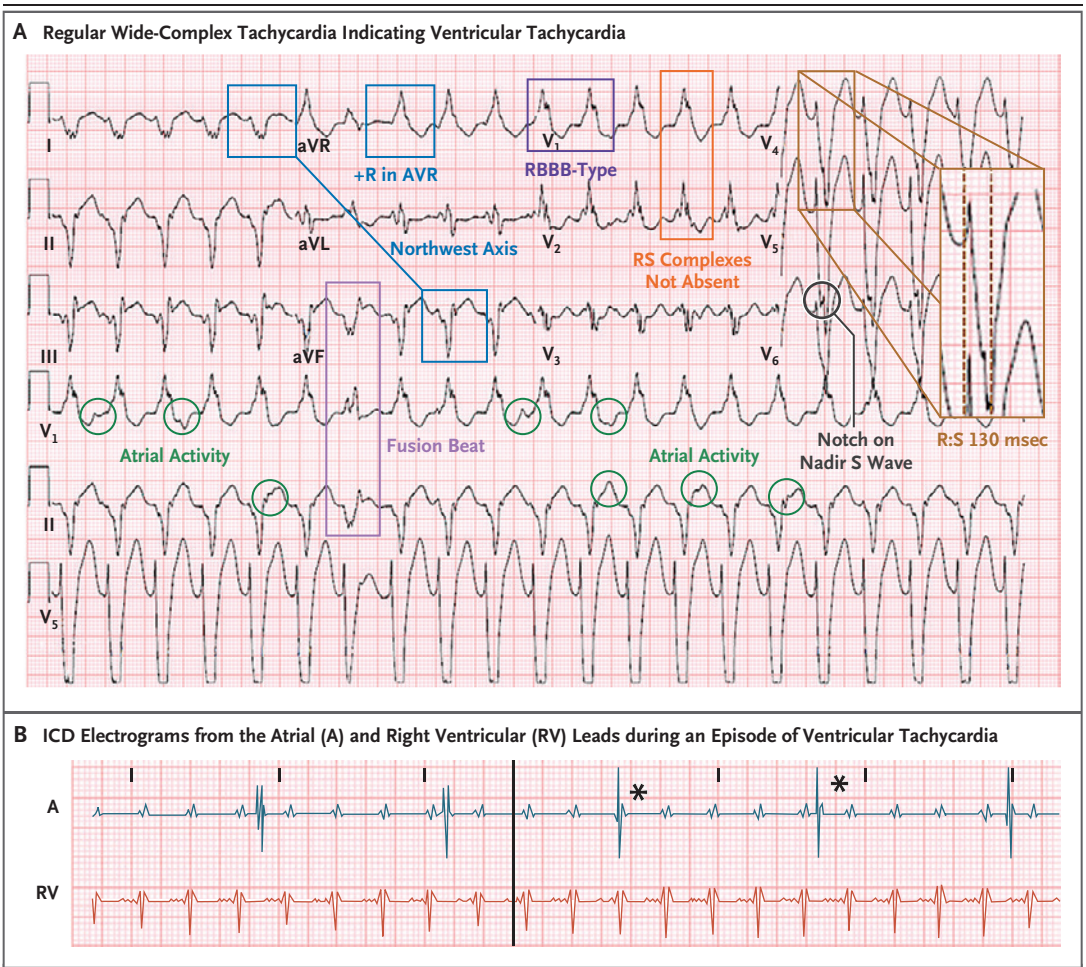

Figure 2. Electrocardiogram Obtained at the Primary Care Clinic.

An electrocardiogram obtained at the time of the primary care clinic visit (Panel A) shows a predominantly regular wide-complex tachycardia that is consistent with ventricular tachycardia. The QRS morphologic feature, also see in the baseline electrocardiogram (ECG), is a right bundle-branch block (RBBB)–type morphology (dark purple box), but the QRS morphology has changed and the duration has increased from 120 to 200 msec from the baseline ECG. The seventh beat in the tracing indicates a fusion beat (Panel A, light purple box), in which there is contemporaneous activation of the ventricular myocardium by the ventricular tachycardia, as well as a sinus beat propagating through the atrioventricular node and His–Purkinje system. The first Brugada criterion is not satisfied because RS complexes are present in precordial leads (Panel A, orange box). The second Brugada criterion confirms ventricular tachycardia if the interval of the onset of the R wave to the nadir of the S wave is greater than 100 msec; in this case, it is approximately 130 msec (Panel A, brown boxes). For academic purposes, it is also noted that atrioventricular dissociation, the third Brugada criterion, is evidenced by atrial activity that is independent of the ventricular activity (Panel A, green circles). This is easily seen during a subsequent and faster episode of ventricular tachycardia on intracardiac electrograms that were obtained from implantable cardioverter–defibrillator (ICD) interrogation (Panel B). This shows sinus rhythm (*) at about 50 beats per minute on the atrial channel with more rapid electrograms during ventricular tachycardia on the RV channel. This atrioventricular dissociation is consistent with those seen on the ECG, albeit at different rates. In addition, the QRS morphologic feature is not the more typical finding of rSR' in a true RBBB, but rather RSr', which is a more common finding in patients with ventricular tachycardia (Panel A, dark purple box); this morphologic feature fulfills the fourth Brugada criterion. Other features consistent with ventricular tachycardia include the extreme (northwest) axis (blue boxes indicate negative QRS in leads I and aVF, with northwest axis triangulation, which is corroborated by the tall positive R wave in lead aVR) and the notching of the nadir of the S wave (Panel A, gray circle).

Table 1. Assessment of Wide-Complex Tachycardia with the Use of Brugada Criteria in This Patient.*

Brugada Criteria	If Yes	If No	Test Characteristics for Ventricular Tachycardia	Present in This Patient
Step 1: Absence of an RS complex in all precordial leads ("negative concordance")?	Ventricular tachycardia confirmed; end analysis	Assess step 2	Sensitivity: 21% Specificity: 100%	No
Step 2: Onset of R wave to nadir of S wave interval >100 msec in one precordial lead?	Ventricular tachycardia confirmed; end analysis	Assess step 3	Sensitivity: 66% Specificity: 98%	Yes; ventricular tachycardia confirmed
Step 3: Presence of atrioventricular dissociation?	Ventricular tachycardia confirmed; end analysis	Assess step 4	Sensitivity: 82% Specificity: 98%	Yes
Step 4: Presence of morphologic criteria for ventricular tachycardia in leads V_1 and V_2 and in lead V_6?	Ventricular tachycardia confirmed; end analysis	Ventricular tachycardia ruled out; consider other wide-complex tachycardias (e.g., supraventricular tachycardia with aberrancy)	Sensitivity: 99% Specificity: 97%	Yes; with an RBBB-type complex, a tall initial R wave in lead V_1 meets the criterion; with an RBBB and R:S >1 in lead V_6 meets the criterion

* If any of the criteria are met, then the diagnosis of ventricular tachycardia is confirmed. Data are from Brugada and colleagues.[4] RBBB denotes right bundle-branch block.

tachycardia because an impulse propagating through the native conduction system, even in the presence of a bundle-branch block, should not have an RS duration that is too long). In addition, evidence of atrioventricular dissociation was present, which also confirms the diagnosis of ventricular tachycardia.

After the patient was transferred from the outpatient clinic to this hospital, the ICD was interrogated. Even though the device had not reported ventricular tachycardia, direct interrogation of the ICD confirmed the diagnosis of ventricular tachycardia, as evidenced by the presence of atrioventricular dissociation, with a ventricular rate that was faster than the sinus rate. This was seen during a subsequent faster episode of ventricular tachycardia (as shown in Fig. 2B, where there are several more electrograms in the right ventricular (lower) channel than in the atrial (upper) channel, in which the large spikes indicate sinus rhythm (indicated by asterisks).

The ICD did not deliver therapies; does this mean that the ICD was not working or that the patient did not have ventricular tachycardia? Before this admission, the patient's ICD had been programmed to deliver therapies for the treatment of ventricular tachycardia at a rate above 124 beats per minute, so the ICD would not have delivered therapies for the wide-complex tachycardia of the patient's rate on presentation. This approach is used to ensure that patients are not shocked erroneously for supraventricular or sinus tachycardia in devices with a single lead or to prevent unnecessary shocks for slower ventricular tachycardias, which may otherwise self-terminate. It is important to note that such programming and discrimination can be difficult in a patient who has a history of slow ventricular tachycardia, as in this case.

ELECTROPHYSIOLOGIC DIAGNOSIS

Ventricular tachycardia.

DISCUSSION OF MANAGEMENT

Dr. Barrett: Advanced cardiac life-support algorithms are used for the treatment of any patient presenting with a stable wide-complex tachycardia and those who have hemodynamic instability. These algorithms advocate the use of synchronized direct current cardioversion if evidence of hypotension, altered mentation, signs of shock, acute heart failure, or ischemic chest discomfort is found. Given this patient's symptoms and his lower blood pressure, treatment with antitachycardia pacing through his ICD was given; antitachycardia pacing at a rate faster than the ventricular tachycardia rate successfully converted the rhythm to sinus bradycardia. The ICD was subsequently reprogrammed to detect ventricular tachycardia at a lower heart rate.

However, during the patient's hospital stay, ventricular tachycardia recurred four times, and

each recurrence led to additional antitachycardia pacing and serial addition of antiarrhythmic drugs, including lidocaine and amiodarone. Given the recurrent ventricular tachycardia in this patient, we obtained the patient's consent to proceed with performing a catheter ablation procedure (an arrhythmia management strategy) to address a likely scar-mediated reentry circuit.[5] The patient had a known scar in the left ventricle with an arrhythmogenic substrate in the inferior wall that had been identified during the previous electrophysiologic study. The next step was to obtain an echocardiogram to assess the left ventricular function and rule out left ventricular thrombus.

Videos showing echocardiographic studies are available at NEJM.org

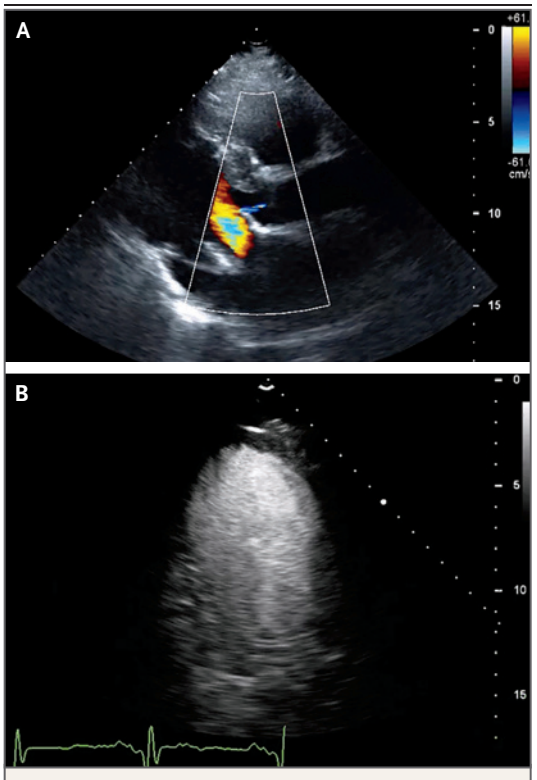

Figure 3. Echocardiogram Obtained before Electrophysiologic Catheter Ablation.

A transthoracic echocardiogram was obtained during hospital admission as part of the evaluation of ventricular tachycardia and for procedural planning; during the imaging study, the patient was in sinus rhythm. The left ventricular ejection fraction was 28% with akinesis of the inferior, inferoposterior, and apical walls; the left ventricle was dilated. In a parasternal long-axis view (Panel A), a bioprosthetic mitral valve shows with no regurgitation and normal transmitral gradients; trace aortic insufficiency is present. No substantial change is observed in left ventricular global or regional function, as compared with the previous transthoracic echocardiogram obtained 6 months before the current presentation. Contrast-enhanced echocardiography was performed 1 day later. An apical four-chamber view (Panel B) shows no echocardiographic evidence of left ventricular thrombus.

Dr. Zern: Transthoracic echocardiography, which was performed while the patient was in sinus rhythm, revealed impaired left ventricular systolic function (ejection fraction, 28%) with akinesis of the inferior, inferoposterior, and apical walls, along with diffusely hypokinetic right ventricular systolic function (Fig. 3A and see Video 1). No substantial change was observed in left ventricular global or regional function, as compared with the previous transthoracic echocardiogram. Contrast-enhanced echocardiography, which was performed 1 day later, confirmed that there was no echocardiographic evidence of left ventricular thrombus (Fig. 3B and see Video 2).

Dr. Barrett: We then performed an electrophysiologic study, which confirmed that the patient had a postmyocardial infarction (scar-related) reentrant ventricular tachycardia.[5] After electrophysiologic mapping and pacing maneuvers with an ablation catheter on the inferior wall of the left ventricle were performed, a critical isthmus of myocardium was identified that was responsible for maintenance of ventricular tachycardia circuits. Catheter ablation was performed at this site, resulting in the successful termination of ventricular tachycardia. Moreover, the patient had no inducible ventricular tachycardia with aggressive pacing maneuvers at the end of the ablation and electrophysiologic study procedure. In addition to catheter ablation, successful long-term management of arrhythmia in patients with ischemic cardiomyopathy also requires meticulous management of the patient's heart-failure therapy.

Dr. Aferdita Spahillari: This patient had heart failure with reduced ejection fraction due to ischemic cardiomyopathy with baseline New York Heart Association (NYHA) functional class II symptoms. He presented with an acute heart-failure exacerbation in the context of sustained ventricular tachycardia. The 2022 heart-failure guidelines of the American Heart Association, American College of Cardiology, and Heart Failure Society of America[6] delineate key evidence-supported strategies for the treatment of heart failure with reduced ejection fraction (guideline-directed medical therapy), with a goal of reducing heart-failure symptoms, morbidity, and mortality. At each encounter, clinicians should assess whether patients are receiving appropriate guideline-directed medical therapy.

Guideline-directed medical therapy for heart failure with reduced ejection fraction currently includes four medication classes. The first group comprises angiotensin receptor–neprilysin inhibitors (for NYHA class II or III functional class) and angiotensin-converting–enzyme (ACE) inhibitors or angiotensin-receptor blockers (for NYHA class II, III, or IV functional class) when angiotensin receptor–neprilysin inhibitors are not available. The second group comprises beta-blockers, the third is mineralocorticoid receptor antagonists, and the fourth is sodium–glucose cotransporter 2 (SGLT2) inhibitors. The initiation of guideline-directed medical therapy leads to a reduction in overall mortality, deaths from cardiovascular causes, and hospitalizations for heart failure.[6] The mechanisms of action of these medications include effects on the adrenergic nervous system, effects on the renin–angiotensin–aldosterone system, and activation of the natriuretic peptide system. SGLT2 inhibitors, in particular, may be involved in several pathways, including improvement in cardiac energetics, reduction of inflammation, direct vasodilation, and renal protection.[7] This patient was taking a beta-blocker (which is also beneficial for patients with ventricular tachycardia), an SGLT2 inhibitor, and an ACE inhibitor. However, previous attempts to transition his medication to angiotensin receptor–neprilysin inhibitor therapy and to add mineralocorticoid receptor antagonists were limited by recurrent hypotension, lightheadedness, and hyperkalemia. There is a growing use of potassium binders for the regulation of potassium homeostasis, which results in an acceptable side-effect profile for guideline-directed medical therapy.[6]

Although the patient in this case had an ICD placed for secondary prevention after a cardiac arrest due to ventricular tachycardia, device therapies such as ICDs should be considered for primary prevention of sudden cardiac death due to arrhythmias for patients with a left ventricular ejection fraction of 35% or less. Cardiac resynchronization therapy should be reserved for patients with heart failure who have a left ventricular ejection fraction of 35% or less and left bundle-branch block characterized by wide QRS intervals. Patients who have signs and symptoms of progressive heart failure despite receiving appropriate guideline-directed medical therapy, as well as those who have recurrent hospitalizations related to heart failure or ventricular arrhythmias that are not amenable to conventional treatments, would benefit from a discussion with their cardiologist about therapies for advanced heart failure, such as heart transplantation.

Dr. Malecha: After ablation of the ventricular tachycardia, the patient had no additional ventricular arrhythmias during hospitalization. At hospital discharge approximately 1 week after admission, treatment with mexiletine was started. At a follow-up primary care visit 2.5 weeks later, he reported 1 second of palpitations but no recurrence of dyspnea or chest discomfort.

The patient continued treatment with mexiletine for 2 years. Four years after the procedure, the patient reported feeling well, he had a stable weight, and was no longer receiving diuretic therapy. His current treatment regimen includes standard, four-pillar guideline-directed medical therapy with a beta-blocker, an angiotensin receptor–neprilysin inhibitor, a mineralocorticoid receptor antagonist, and an SGLT2 inhibitor. Recent device interrogation revealed a single episode of nonsustained ventricular tachycardia at a rate of approximately 150 beats per minute that lasted for 10 beats.

FINAL DIAGNOSIS

Postmyocardial infarction (scar-related) reentrant ventricular tachycardia.

This case was presented at the Medicine Case Conference.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

REFERENCES

1. Case Records of the Massachusetts General Hospital (Case 36-2020). N Engl J Med 2020;383:2066-76.
2. Elnaggar MN, Jbeili K, Nik-Hussin N, Kozhippally M, Pappachan JM. Amiodarone-induced thyroid dysfunction: a clinical update. Exp Clin Endocrinol Diabetes 2018;126:333-41.
3. Daniels GH. Amiodarone-induced thyrotoxicosis. J Clin Endocrinol Metab 2001;86:3-8.
4. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation 1991;83:1649-59.
5. Stevenson WG, Friedman PL, Sager PT, et al. Exploring postinfarction reentrant ventricular tachycardia with entrainment mapping. J Am Coll Cardiol 1997;29:1180-9.
6. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol 2022;79:1757-80.
7. Lopaschuk GD, Verma S. Mechanisms of cardiovascular benefits of sodium glucose co-transporter 2 (SGLT2) inhibitors: a state-of-the-art review. JACC Basic Transl Sci 2020;5:632-44.