A 23-year-old man is referred by his family physician to a cardiology clinic with a several-month history of progressively worsening episodes of palpitations associated with dyspnea. He reports experiencing brief episodes almost hourly and describes the palpitations as being extremely rapid and the rhythm as regular.

Dr Salehian: Palpitations as a chief complaint are a common issue routinely assessed by family physicians, general internists, emergency department physicians, and cardiologists. The differential diagnosis in such patients is broad and often not related to a primary cardiac issue or arrhythmia. Broadly, symptoms of palpitations may be related to cardiac arrhythmia or structural heart disease, medications, recreational drugs and alcohol, metabolic disorders, high output states, or psychiatric conditions. To help determine the etiology of the patient’s symptoms, a thorough history, physical examination, and basic laboratory investigations are necessary to help to determine which further investigations are required.

The patient reports mild dyspnea with the episodes but denies associated syncope or presyncope, chest discomfort, nausea, or vomiting. The palpitations are not precipitated by exertion, and there are no other identifiable aggravating factors. He is unaware of any physical activity limitations, exertional symptoms, or features suggestive of congestive heart failure. There is no drug or alcohol use and absence of a family history of early coronary artery disease, congenital heart disease, dysrhythmia, or sudden cardiac death. He also denies recent infectious symptoms. On physical examination, he is afebrile, with a blood pressure of 116/72 mm Hg, heart rate of 68 bpm, and respiratory rate of 14 breaths per minute. There is no adenopathy noted, and the jugular venous pressure is not elevated. Carotid upstroke is normal without any bruits. On cardiac examination, apical impulse is not easily palpable, and no obvious heaves or thrills are felt. On auscultation, first and second heart sounds are normal, with no extra heart sounds, murmurs, or rubs. Lungs are clear to auscultation with no adventitious sounds. Abdomen is soft, and organomegaly is not detected. Peripheral pulses are all normal. There is no peripheral edema detected in his extremities. All blood work, including complete blood count, electrolytes, and thyroid function studies, is within normal limits.

Dr Salehian: In this patient, there are no significant worrisome features in the history or physical examination to suggest a sinister arrhythmia as the cause of his symptoms. Basic blood work is reassuringly normal as well. Although not completely ruled out by a benign history, absence of certain features is encouraging. For example, symptoms are not precipitated by exercise (catecholaminergic polymorphic ventricular tachycardia [VT], hypertrophic obstructive cardiomyopathy), and there is no history suggestive of underlying cardiomyopathy (nonischemic or ischemic cardiomyopathy) and no family history of arrhythmia or sudden death (arrhythmogenic right ventricular cardiomyopathy, congenital long QT syndrome, Brugada syndrome, hypertrophic cardiomyopathy). Furthermore, there is no personal history of syncope in the patient to suggest malignant ventricular dysrhythmia. Other cardiac etiologies for palpitations in addition to noncardiac causes need to be considered. A 12-lead ECG would be a useful next investigation, looking for signs of preexcitation (Wolff-Parkinson-White syndrome), conduction system disease, repolarization abnormalities (Brugada syndrome, long QT syndrome, arrhythmogenic right ventricular cardiomyopathy), or features suggestive of subclinical cardiomyopathy.

Initial 12-lead ECG shows sinus arrhythmia with an average heart rate of 58 bpm, unremarkable PR and QRS intervals, normal axis, and no evidence for chamber enlargement or hypertrophy. There are symmetrical and deep T-wave inversions noted in the lateral precordial (V3 through V6), lateral, and inferior leads (Figure 1). Corrected QT is prolonged at 476 ms.

Dr Salehian: These findings are surprising because history and physical examination support a more benign cardiac or noncardiac cause for the patient’s symptoms. However, ECG
features are suggestive of underlying cardiac structural or electric abnormalities. Cardiac diagnoses that should now be considered include myocardial ischemia or infarction and ventricular hypertrophy from processes such as hypertrophic cardiomyopathy. Noncardiac causes of deeply inverted T waves should also be contemplated, including pulmonary embolism or cerebral T waves from intracranial pathologies such as subarachnoid hemorrhage or large ischemic or hemorrhagic stroke. Cardiac ischemia related to atherosclerosis would be unusual in a young patient without risk factors for coronary disease; however, coronary anomalies leading to ischemia could cause similar ECG findings and lead to arrhythmia. Unlike the morphology seen in the patient, persistent juvenile T-wave inversions are generally limited to the right precordial leads (V1 through V3) and are generally not deep or symmetrical. There are no features in this patient to suggest pulmonary embolism or acute neurological insult as the cause of the ECG changes. Finally, QT interval is mildly prolonged, which is likely related to the cause of the underlying T-wave abnormalities, such as ischemia or hypertrophic cardiomyopathy, and not a primary congenital long QT syndrome or acquired prolongation of the QT interval. Subsequent investigations, including 24-hour ambulatory ECG monitoring and transthoracic echocardiography, would be helpful to assess for arrhythmia in the setting of symptoms and underlying structural heart disease, respectively, given the ECG findings. A 24-hour ambulatory ECG monitor is a logical choice in this case given the patient’s frequent symptoms; however, in cases with less frequent symptoms, an event monitor or a loop recorder may provide a higher yield.

The 24-hour ambulatory ECG (Holter) monitor reveals an average heart rate of 64 bpm, numerous premature ventricular contractions, and multiple episodes of nonsustained monomorphic VT, with the longest episode lasting 104 beats at a heart rate of 203 bpm (Figure 2). During these arrhythmias, the patient reports his usual symptoms, including palpitations and mild dyspnea.

**Dr Salehian:** Although the ambulatory ECG report states recurrent nonsustained VT, other diagnoses should be considered. Regular wide complex tachycardia may be related to VT or supraventricular tachycardia with aberrant conduction or preexcitation. There is no suggestion of preexcitation on 12-lead ECG or on Holter recordings, and therefore supraventricular tachycardia with preexcitation over an accessory pathway is unlikely. At first glance, it is difficult to rule out supraventricular tachycardia with aberrant conduction on the basis of the Holter recordings; however, a closer look clearly shows atrioventricular dissociation during the tachycardia episodes, with the same atrial rate as the previous sinus rhythm as well as a capture beat and fusion beat (Figure 2). All of these findings imply a ventricular origin and confirm recurrent VT as the cause of the Holter rhythm abnormalities and the patient’s symptoms. VT is often related to underlying structural heart disease, and therefore a transthoracic echocardiogram is the test of choice to assess for abnormal cardiac structure or function.

**Transthoracic echocardiography** is technically difficult but suggests a large intracardiac mass, near the apex of the left ventricle, visually estimated to be similar in size to the left ventricle. Left and right ventricular size and function are normal, and there are no significant valvular abnormalities or pericardial effusion.

**Dr Salehian:** Echocardiography confirmed a potential structural cause for the patient’s VT and also ruled out more common etiologies for VT such as ischemic or nonischemic cardiomyopathy and hypertrophic cardiomyopathy. The echocardiogram was of relatively poor quality because of technical
and patient factors, and therefore precise assessment of the mass characteristics other than approximate size and location does not appear to be feasible. This issue is becoming more common as technically limited echocardiographic examinations are on the rise related to the increasing prevalence of obesity. More detailed imaging is required because the differential diagnosis for cardiac masses is extensive and includes both malignant and benign etiologies. A cardiac magnetic resonance imaging (cMRI) scan would provide precise anatomic details for the mass and also provide a gross functional assessment of the cardiac chambers and valves while avoiding radiation in this young patient. A cMRI would be preferred over a transesophageal echocardiogram in this case for a few reasons. Although transesophageal echocardiography provides excellent imaging of most structures of the heart, the true left ventricular apex can be difficult to visualize, and lesions in this area may be missed or difficult to accurately characterize. In addition, a transesophageal
The echocardiogram is an invasive test requiring procedural sedation and carries a small risk of adverse events.

The patient is referred for a cMRI study that shows grossly normal biventricular function and no evidence for valvular abnormalities. The cMRI confirms a large mass located in the left ventricular myocardium toward the apex, measuring 7.4×6.1×5.0 cm (Figure 3A and 3B and Movies I and II in the online-only Data Supplement). It displays intermediate T1-weighted signal intensity and high T2-weighted signal intensity. The mass has smooth outer borders and is intramyocardial with a thin rim of what appears to be myocardial tissue surrounding the mass. This rim of tissue separates the lesion from the left ventricular cavity on the inside and also a small, simple-appearing pericardial effusion on the outside. Postgadolinium sequences demonstrate homogeneous enhancement of the lesion (Figure 3C).

Dr Salehian: Given the absence of constitutional symptoms, as well as location and imaging characteristics of the mass, a benign lesion is favored in this case. Gadolinium enhancement on MRI is highly suggestive of a vascular tumor. On the basis of MRI features and tumor location, cardiac hemangiomia, rhabdomyoma, and fibroma would be the most common causes to consider in this young patient. However, malignant tumors with some similar features such as angiosarcoma, rhabdomyosarcoma, or pheochromocytoma need to be excluded. Depending on the location and extent of the cardiac tumor, biopsy may be attempted; however, results are often nondiagnostic, and more invasive open procedures are frequently required to obtain a tissue diagnosis. Given that the patient is highly symptomatic from ventricular arrhythmias and the potential for sudden cardiac death, tumor resection would be recommended in this case. It is hoped that this would provide relief of his arrhythmias and importantly provide a pathological tissue sample to confirm whether the lesion is in fact benign. Preoperative coronary angiography is not necessary given that the probability of significant coronary artery disease is low in this patient on the basis of age, clinical risk factors, and absence of ischemic symptoms.

While a patient awaits surgery, medications can be used in an attempt to suppress episodes of nonsustained VT, thereby reducing symptoms and possibly diminishing the risk for malignant, sustained arrhythmia. There may be a role for medications such as β-blockers or amiodarone in this setting; however, most data suggesting potential benefit in relieving symptoms or prevention of sustained VT or sudden death come from studies investigating patients with structural heart disease related to myocardial infarction. Data from less common structural causes of nonsustained VT (ie, cardiac tumors) are lacking. For symptomatic relief, β-blockers would be a reasonable option in this patient given their favorable safety profile and tolerability.

Urgent referral to a cardiac surgeon is organized, and after assessment of the patient, surgery is scheduled. In the operating room, a standard median sternotomy is undertaken, and with the use of cardiopulmonary bypass under cardioplegic arrest, the large tumor, similar in size to the left ventricle (Figure 4 and Movie III in the online-only Data Supplement), is carefully resected off the heart. Because of tumor invasion of the myocardium, a deep cut into muscle is necessary, entering the left ventricular cavity. A thin layer of myocardium is left behind on the tumor. The distal left anterior descending coronary artery is identified entering
into the tumor region and is ligated. After resection of the tumor, the left ventricle is closed in multiple layers in a manner similar to that used with a left ventricular aneurysm resection. Gross pathological examination reveals a large (9.0 × 6.5 × 3.5 cm) homogeneous mass (Figure 5), with peripheral and deeper resection margins showing myocardium. Microscopic examination shows myocardium with an infiltrating lesion with vascular proliferation, including capillary, cavernous, and venular hemangioma (Figure 6). The lesion is seen infiltrating into adjacent cardiac myocytes. No malignant components are identified. Immunohistochemistry is strongly positive for factor VIII, CD31, and CD34, confirming vascular origins. The final pathological diagnosis is infiltrating cardiac hemangioma.

Dr Salehian: The prognosis in patients after successful cardiac hemangioma resection is favorable, with reportedly low recurrence rates. There also appear to be symptomatic improvement and reduction or elimination of arrhythmias after surgery. Periodic follow-up with clinical assessment aimed at elucidating symptoms related to recurrence of arrhythmias or structural heart disease should be performed postoperatively. Ongoing serial imaging with the use of echocardiography and/or cMRI is also sensible. However, the exact interval and duration for clinical assessment and imaging are not established given the rare nature and variability of the disease.

Postoperatively, the patient made an uneventful recovery and remains symptom free over 12 months of follow-up. In fact, he successfully completed a 50-km cycling race without any issues. Subsequent imaging, including echocardiography and cMRI (Figure 7 A-C), has demonstrated the expected postsurgical changes and has not suggested recurrence of the hemangioma. Follow-up Holter monitoring exhibited no supraventricular or ventricular arrhythmias.

Discussion

This case illustrates the broad differential diagnosis in patients presenting with symptoms of palpitations. History and physical examination suggested a benign cardiac or noncardiac cause of the patient’s symptoms; however, noninvasive testing (ECG, ambulatory ECG monitoring, echocardiography, and cMRI) supported more sinister arrhythmias with an unusual cause. Structural heart disease is a common cause of arrhythmias, both supraventricular and ventricular in origin, and is generally associated with chronic hypertension, valvular disease, or ischemic heart disease. A cardiac tumor as the cause of symptomatic VT is extremely rare, highlighting the importance of cardiac imaging to assess for any structural abnormalities in cases of documented arrhythmia. The patient discussed in this case had VT related to a primary benign cardiac tumor, specifically hemangioma. Symptoms were completely resolved with tumor resection.

Primary cardiac tumors are extremely uncommon and often diagnosed incidentally on imaging performed for unrelated issues or postmortem owing to the typically asymptomatic nature of these tumors. Based on a collection of large series, the frequency of primary cardiac tumors discovered at autopsy is ≈0.02%. The majority of these cardiac tumors are classified as histologically benign, with myxoma accounting.
for the majority. Other benign tumors include lipomas, papillary fibroelastomas, rhabdomyomas, fibromas, hemangiomas, and teratomas. The patient in this case was found to have a cardiac hemangioma, an exceedingly rare benign tumor of the heart. Although they are the most common vascular tumor of the heart, hemangiomas account for only 5% of all benign cardiac tumors in adults.

Hemangiomas are benign, proliferative tumors of vascular origin resulting from increased endothelial cell proliferation. They are located most commonly in the epicardium but have also been described within the myocardium and endocardium. Moreover, they are usually found on the lateral wall of the left ventricle, free wall of the right ventricle, or interventricular septum. Histologically, there are 3 categories of cardiac hemangiomas: (1) cavernous hemangioma typified by dilated thin-walled vessels; (2) capillary hemangioma distinguished by small capillary-like vessels; and (3) arteriovenous hemangioma characterized by dysplastic and malformed arteries and veins. Histological examination of resected hemangioma is often able to identify elements of all categories interspersed with fibrous tissue. The patient in this case demonstrated a less commonly seen apical tumor location in addition to more typical pathology showing all 3 hemangioma types with vascular proliferation consisting of capillary and cavernous-type hemangioma with interspersed dysplastic-appearing, thick-walled vascular spaces. Finally, the tumor stained positive for factor VIII, CD31, and CD34, confirming an endothelial origin.

Occasionally, benign cardiac tumors, including hemangiomas, present with symptoms, including exertional dyspnea, congestive heart failure, outflow and inflow tract obstruction, coronary insufficiency from obstruction or vascular steal, arrhythmias, pericardial effusion, embolic phenomenon, heart block, or even sudden death. In general, symptoms and clinical presentation vary according to the size, location, and mobility of the tumor. The patient presented with frequent and symptomatic episodes of nonsustained VT, which has been described in the setting of benign cardiac tumors, including hemangiomas. These ventricular arrhythmias are most likely related to macroreentry circuits around or within the tumor. There are a few reported cases of benign tumors causing recurrent VT refractory to medical management. In these cases, ventricular arrhythmias were completely alleviated after surgical resection of the tumor, as was the case in our patient.

Cardiac tumors are usually identified initially on echocardiography. However, other modalities such as computed tomography, MRI, ventriculography, and coronary angiography are generally utilized to better characterize the tumors in terms of size, exact location, morphology, and relationship with other cardiac and mediastinal structures. Cardiac hemangiomas are hyperechoic on echocardiography and on computed tomography show intense enhancement with intravenous contrast administration. MRI characteristics of cardiac hemangiomas are similar to those seen in the liver with intermediate or increased signal intensity on T1-weighted images and high signal on T2-weighted images. Because hemangiomas are vascular lesions, there is usually intense enhancement after gadolinium administration; however, the degree of enhancement varies with the amount of calcification and/or fibrous septa that are present. MRI is the best diagnostic modality to identify aggressive characteristics of cardiac tumors, and the use of cMRI for preoperative planning is widely accepted. Cardiac MRI offers superior soft tissue contrast in comparison to computed tomography and echocardiography, offering improved resolution, and it also provides a larger field of view to assess
### Table. Imaging Characteristics of Benign and Malignant Cardiac Tumors

<table>
<thead>
<tr>
<th>Tumor</th>
<th>Macroscopic Examination</th>
<th>Echocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Benign</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myxoma (45% to 50% of benign cardiac tumors)</td>
<td>Spherical or ovoid intracavitary mass, often arising from narrow stalk; lobular, smooth and may have frond-like or villous extensions; usually located in left atrium with attachment to interatrial septum at fossa ovalis</td>
<td>Appears homogeneous but may have central areas of hyperlucency representing hemorrhage and necrosis; characteristic narrow stalk with attached highly mobile tumor</td>
</tr>
<tr>
<td>Papillary fibroelastoma (15% of benign cardiac tumors)</td>
<td>Usually small (&lt;1 cm) and attached to endocardium by short, thin stalk; occur on atrial surface of atrioventricular valves and aortic surface of semilunar valves; majority occur on cardiac valves</td>
<td>Small, mobile masses attached to valves by a short pedicle; may appear as elongated strands or may have well-defined mass at end</td>
</tr>
<tr>
<td>Fibroma (3% of benign cardiac tumors in adults, second most common in children)</td>
<td>Solid, white, fibrous tumors that arise within myocardium; may grow to obliterate ventricular cavity; no foci of cystic, hemorrhagic, or necrotic areas</td>
<td>Large, noncontractile, echogenic intramural mass; may be nodular and discrete or cause hypertrophy of ventricular septum; may show calcification</td>
</tr>
<tr>
<td>Paraganglioma (very rare)</td>
<td>Soft, fleshy tumors that may be encapsulated; necrosis is common, and calcification occurs rarely</td>
<td>Usually large, echogenic left atrial mass; may arise from interatrial septum with a broad base; compression of superior vena cava or encasement of coronary arteries may be seen on TEE</td>
</tr>
<tr>
<td>Lipoma (20% of benign cardiac tumors)</td>
<td>Soft, encapsulated, homogeneous fatty tumors; majority arise from epicardial surface, usually with broad pedicle, expanding into pericardial space</td>
<td>Appearance varies depending on location; in pericardial space, may be completely hypoechoic, have hypoechoic regions, or be completely echogenic; intracardiac lipomas are homogeneous and hyperechoic</td>
</tr>
<tr>
<td>Hemangioma (5% of benign cardiac tumors)</td>
<td>Well-demarcated, lobulated, mural tumors; most commonly originate from epicardium but are also seen as intramyocardial or intracavitary tumors</td>
<td>Variably sized echogenic mass, possibly containing echolucent areas, located within the myocardium or extending into pericardial space or intracavitary space</td>
</tr>
<tr>
<td>Rhabdomyoma (1% of benign cardiac tumors in adults, most common benign cardiac tumor in children)</td>
<td>Originate within the myocardium, usually in ventricles; often multiple tumors present</td>
<td>Intramyocardial well-circumscribed echogenic pedunculated tumor, usually multiple and within ventricle; slightly brighter than surrounding myocardium</td>
</tr>
<tr>
<td><strong>Malignant</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angiosarcoma (most common primary cardiac malignancy in adults)</td>
<td>Large, grossly hemorrhagic infiltrative tumors with areas of necrosis; typically involve the right atrium and/or pericardium</td>
<td>Usually echodense, broad-based right atrial mass near the inferior vena cava; may show pericardial effusion and possibly infringing mass extending along pericardium, may invade myocardial tissue or contiguous structures and alter function or appearance</td>
</tr>
<tr>
<td>Rhabdomyosarcoma (most common primary cardiac malignancy in childhood, 20% of adult cardiac sarcomas)</td>
<td>Gelatinous and friable, firm and cyst-like with large areas of central necrosis; no specific site more common but can affect valves and have multiple sites of involvement</td>
<td>Echodense, bulky mass or multiple masses that commonly invade surrounding myocardium and valves; may extend beyond myocardium, causing polypoid extension into chamber cavity; many have pericardial effusion and pericardial nodular masses</td>
</tr>
<tr>
<td>Osteosarcoma (rare)</td>
<td>Many have dense calcification; usually occur in left atrium; differentiated from myxoma by broad-based attachment, location away from fossa ovalis, and signs of invasion</td>
<td>Echodense, broad-based left atrial mass, usually arising from location other than fossa ovalis on interatrial septum</td>
</tr>
<tr>
<td>Leiomyosarcoma (rare)</td>
<td>Gelatinous, irregular, sessile mass or masses; most commonly found in left atrium and can have multiple sites of involvement; usually arise from posterior wall of left atrium; tend to invade pulmonary veins or mitral valve</td>
<td>Echodense, irregular left atrial mass (may be multiple masses), usually arising from posterior wall, which helps to differentiate from myxoma</td>
</tr>
<tr>
<td>Primary cardiac lymphoma (exceedingly rare)</td>
<td>Multiple firm nodules; commonly involves right side of heart, particularly right atrium; frequent involvement of &gt;1 chamber with invasion to pericardium and extension into inferior and superior vena cava;</td>
<td>May only see pericardial effusion; echodense intracardiac mass or masses, most commonly seen in right atrium; invasion of pericardium common and sometimes pericardial thickening seen</td>
</tr>
<tr>
<td>Mesothelioma (rare)</td>
<td>Tumor arising from mesothelial cells of pericardium; multiple coalescing masses that envelop pericardial space; ultimately obliteration of pericardial space</td>
<td>Pericardial effusion common and may have focal areas of tethering and adherence; pericardial thickening frequent and may be marked; features of pericardial constriction can be seen</td>
</tr>
</tbody>
</table>

N/A indicates not applicable; TEE, transesophageal echocardiography.
<table>
<thead>
<tr>
<th>T1-Weighted</th>
<th>T2-Weighted</th>
<th>After Gadolinium</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypointense</td>
<td>Heterogeneously hyperintense</td>
<td>Heterogeneous pattern of enhancement due to increased vascularity and inflammation</td>
<td>Fibrous areas are hypointense on both T1 and T2; thin stalk usually not seen, but narrow base of attachment can be appreciated</td>
</tr>
<tr>
<td>Not defined because almost never seen as a result of small size and attachment of moving valves</td>
<td>Not defined because almost never seen as a result of small size and attachment of moving valves</td>
<td>Not defined because almost never seen as a result of small size and attachment of moving valves</td>
<td>On cine gradient-echo images may appear as hypointense mobile mass</td>
</tr>
<tr>
<td>Isointense or hypointense</td>
<td>Homogeneously hypointense</td>
<td>Little or no enhancement; occasionally enhancing or isoointense rim with hypointense core</td>
<td>N/A</td>
</tr>
<tr>
<td>Isointense or hypointense</td>
<td>Markedly hyperintense</td>
<td>Intense contrast enhancement but may be heterogeneous with central nonenhancing areas due to necrosis</td>
<td>N/A</td>
</tr>
<tr>
<td>Homogeneously hyperintense</td>
<td>Slightly less hyperintense than T1</td>
<td>No enhancement</td>
<td>Signal suppression on fat-saturated images; signal intensity is similar to adjacent subcutaneous or mediastinal fat</td>
</tr>
<tr>
<td>Heterogeneously isointense or hypointense</td>
<td>Usually hyperintense; occasionally heterogeneous with hypointense areas</td>
<td>Heterogeneously hyperintense pattern of enhancement</td>
<td>N/A</td>
</tr>
<tr>
<td>Isointense to marginally hyperintense</td>
<td>Hyperintense</td>
<td>May be hypointense</td>
<td>N/A</td>
</tr>
<tr>
<td>Heterogeneous appearance with areas of low, intermediate, and high signal intensity</td>
<td>Heterogeneous, predominantly hyperintense</td>
<td>Heterogeneous enhancement with marked surface enhancement and areas of central necrosis</td>
<td>May show pericardial thickening and/or hemorrhagic pericardial effusion</td>
</tr>
<tr>
<td>Isointense</td>
<td>Heterogeneous, predominantly hyperintense</td>
<td>Usually homogeneous enhancement but may have areas of low signal intensity due to central necrosis</td>
<td>N/A</td>
</tr>
<tr>
<td>Heterogeneously hypointense</td>
<td>Heterogeneously hyperintense</td>
<td>Markedly enhancing</td>
<td>N/A</td>
</tr>
<tr>
<td>Isointense or hypointense</td>
<td>Hyperintense</td>
<td>Markedly enhancing</td>
<td>N/A</td>
</tr>
<tr>
<td>Isointense</td>
<td>Heterogeneously hyperintense</td>
<td>Heterogeneous enhancement with areas of low enhancement in center of lesion</td>
<td>N/A</td>
</tr>
<tr>
<td>Homogeneously isointense</td>
<td>Heterogeneously isointense because of areas of necrosis</td>
<td>Markedly enhancing</td>
<td>N/A</td>
</tr>
</tbody>
</table>
surrounding structures for additional clues to the diagnosis. The size, border, calcification, presence of a pericardial effusion, and location are assessed to help to determine whether a tumor is benign or malignant (Table). A typical benign tumor will demonstrate a small size, have smooth, well-defined borders, and show no evidence of calcification. In the patient discussed in this report, rapid tumor enhancement after gadolinium administration indicated that the tumor was highly vascular. As mentioned above, hemangioma, rhabdomyoma, or fibroma was considered most likely given the patient’s age, clinical symptoms, and tumor location. However, a tissue sample for pathological examination is important to differentiate from other potentially more worrisome tumors that exhibit gadolinium enhancement, including angiosarcoma, rhabdomyosarcoma, and pheochromocytoma.

Twelve-lead ECG and Holter monitoring are routine tests ordered in patients presenting with cardiac symptoms, and abnormal results have been reported in patients with benign cardiac tumors, including hemangioma. Recurrent VT was noted in a patient with a large intramural left ventricular fibroma with no recurrence after surgical resection. Abnormalities on resting ECG have been also been described, including anterior T-wave inversion. In the presented patient, 12-lead ECG at baseline was significantly abnormal, with deeply inverted T waves in the lateral and inferior leads (leads V3 through V6, I, II, III, and aVF). Furthermore, a 24-hour ambulatory ECG recording demonstrated multiple runs of monomorphic nonsustained VT. These features were extremely concerning and prompted urgent investigations and ultimately, on discovery of the left ventricular mass, swift referral to a cardiac surgeon.

In patients with cardiac hemangioma, the natural history is difficult to determine because the tumors are often asymptomatic and diagnosed either postmortem or not at all. In patients with known cardiac hemangioma, the natural history is inconsistent and therefore unpredictable. For example, cardiac hemangiomas have been reported to proliferate and grow indefinitely, remain stable for long periods of time, or occasionally regress. Al- though these tumors may remain clinically asymptomatic or minimally symptomatic, given the unpredictability and potentially life-threatening complications, surgical tumor resection is the therapy of choice for patients with cardiac tumors. Furthermore, surgical resection allows for histological confirmation of the tumor type. Complete resection is preferable when technically feasible. With complete resection, the long-term prognosis is extremely favorable, with significant improvement in symptomatic patients, low tumor recurrence rate, and cessation of ventricular arrhythmias. In addition, prognosis appears quite good even after partial or incomplete resection. Periodic follow-up has also been advocated because recurrence or transformation into a malignant tumor has been seen occasionally. In the aforementioned patient, resection was accomplished with care taken to restore the normal geometry of the left ventricle. Postoperatively, the patient remained asymptomatic with no signs of arrhythmia recurrence clinically or on ambulatory ECG monitoring and unremarkable imaging, including cMRI and echocardiography.

Disclosures

None.

References

A Bloody Mass: Rare Cardiac Tumor as a Cause of Symptomatic Ventricular Arrhythmias
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