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IMAGING VIGNETTE

INTERMEDIATE

CLINICAL VIGNETTE

Cardiac Metastatic Melanoma Causing Ventricular Arrhythmias Through Impaired Coronary Artery Vasodilation

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ABSTRACT

Melanoma is an aggressive malignant disease with a high rate of cardiac metastasis. There is a reported association between myocardial tumor invasion and ventricular arrhythmias. We present a case of cardiac metastatic melanoma causing ventricular arrhythmias through a novel mechanism of encasement of coronary arteries leading to reduced myocardial perfusion. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;18:101914)

A 68-year-old man with malignant melanoma metastatic to the bone, lung, and heart was admitted for nausea and emesis and subsequently developed ventricular arrhythmias. He previously received immunotherapy with ipilimumab and nivolumab; however, this therapy was discontinued as a result of checkpoint inhibitor colitis and gastritis. During his admission, he experienced multiple episodes of polymorphic ventricular tachycardia (VT) (**Figure 1A**) requiring emergency cardioversion and antiarrhythmic agents. Initial work-up for an ischemic cause included an electrocardiogram and troponin level, both of which were normal. His arrhythmias were thus attributed to hypokalemia and hypomagnesemia; however, there was arrhythmia recurrence despite correction of these deficiencies. Cardiac magnetic resonance was obtained, which demonstrated septal T2-weighted and late gadolinium enhancement, findings stable from 6 months earlier. A repeat echocardiogram demonstrated a stable ejection fraction of 53% with akinesis of the basal septum to the midseptum. A vasodilator nuclear medicine positron emission tomography (myocardial perfusion scan revealed stress-induced myocardial ischemia in the territory typical of the proximal and middle left anterior descending (LAD) artery (**Figure 1B**). A computed tomography coronary angiogram was performed, which demonstrated tumor encasement of the septal branches of the LAD artery without stenosis (**Figures 1C and 1D**). On the basis of these findings, it was determined that the tumor was preventing vasodilation of the LAD septal perforators, thereby resulting in stress-induced ischemia in the interventricular septum and anterior wall. To shrink the tumor, treatment with dabrafenib and trametinib was initiated. The response to this therapy is pending outpatient imaging, but the patient has had no recurrence of VT.

Although primary and secondary cardiac tumors are rare, secondary tumors are more frequent, and melanoma has a high predilection to metastasize to the heart. Most cases of metastatic melanoma to the heart are clinically silent. When symptoms are present, they vary depending on where the tumor has spread. Patients may present with arrhythmias resulting from myocardial involvement and subsequent conduction

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ABBREVIATIONS AND ACRONYMS

LAD = left anterior descending

MINOCA = myocardial infarction with nonobstructive coronary arteries

VT = ventricular tachycardia

abnormalities or clinical symptoms of heart failure caused by involvement of the values, ventricular cavity, or pericardium.[1]

The number of reported cases of VT in the setting of metastatic melanoma is low.[1] Sheldon et al[2] reported possibly the earliest case demonstrating the association between a ventricular tumor secondary to metastatic melanoma and sustained VT. There were 3 hypothesized mechanisms, which included tumor invasion causing a macro-re-entry circuit, altered tissue architecture causing localized repolarization and then a micro-re-entry circuit, and localized compression of myocardial fibers or a surge of humoral catecholamines leading to abnormal cardiac automaticity.[2] A more recently identified entity that may contribute to altered tissue architecture is myocardial infarction with nonobstructive coronary arteries (MINOCA) because microvascular tumor compression can lead to impaired perfusion and myocardial scarring.[3] Despite overlapping mechanisms, MINOCA is unlikely to explain this current case given the lack of infarction.

We present a case of cardiac metastatic melanoma leading to ventricular arrhythmias through a novel mechanism involving the tumor invading the myocardium and then encasing coronary vasculature. Tumor encasement presumably led to impaired vasodilation during periods of increased myocardial demand that resulted in localized myocardial ischemia, causing VT.

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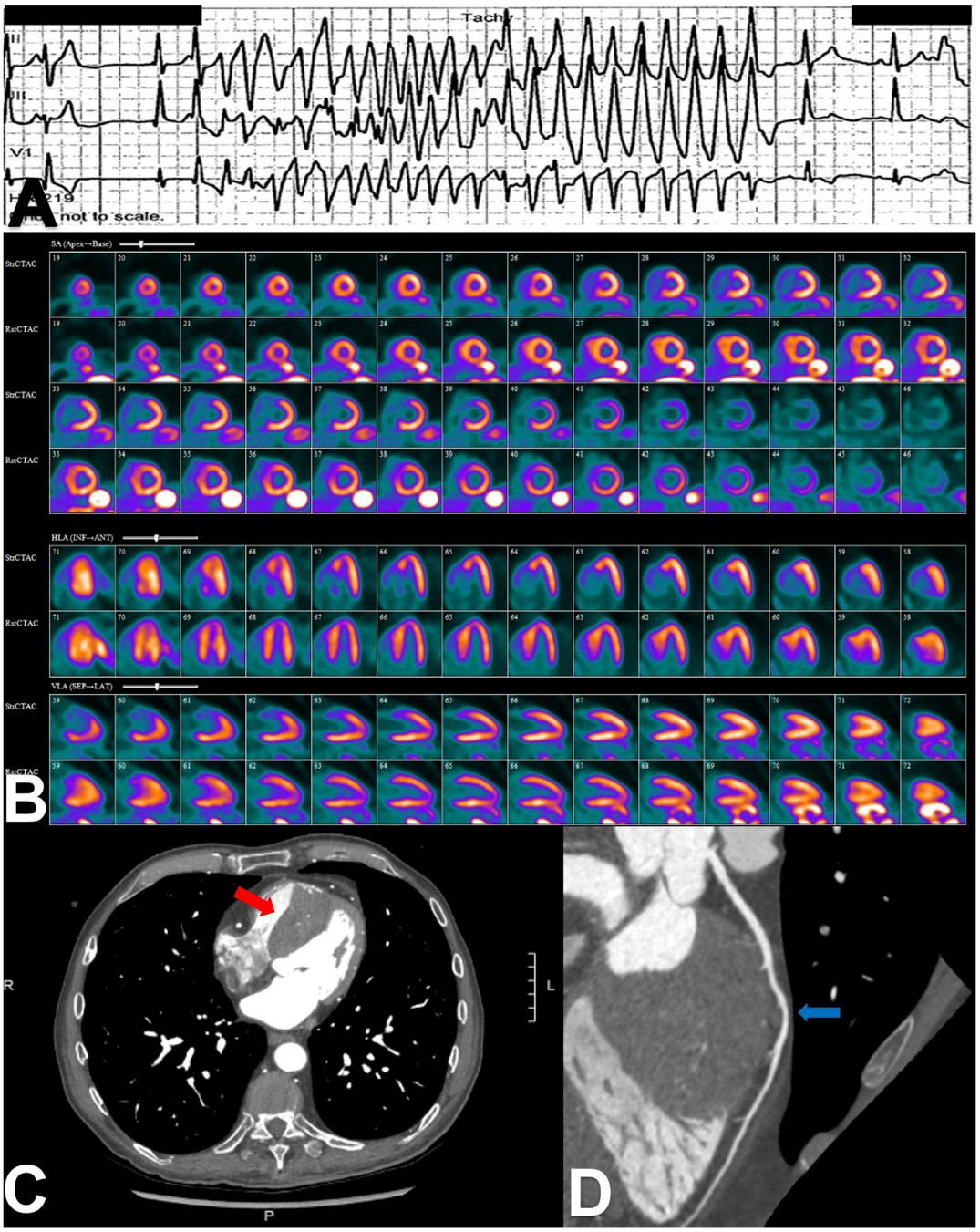
FIGURE 1 Electrocardiogram and Multimodal Cardiac Imaging



(A) Electrocardiogram revealing an episode of nonsustained ventricular tachycardia (Tachy). **(B)** Positron emission tomography images with rubidium-82 radiotracer demonstrating normal resting perfusion of the right and left ventricles with septal ischemia in the midseptal to basal anterior, anteroseptal, and inferoseptal walls during stress. **(C)** Computed tomography demonstrating metastatic infiltration of the interventricular septum with a patent left anterior descending (LAD) artery running along the interventricular groove. Pictured are septal perforators, which are encased by metastatic melanoma **(red arrow)**. **(D)** Computed tomography coronary angiogram demonstrating patency of the left anterior descending artery **(blue arrow).** Septal perforators can be seen extending into the thickened septum. ANT = anterior; HLA = horizontal long axis; INF = inferior; LAT = lateral; L = left; P = posterior; R = right; RrtCTAC = rest CT-based attenuation correction; SA = short axis; SEP = septal; StrCTAC = stress CT-based attenuation correction; VLA = vertical long axis.