

Recurrent Parotid Adenocarcinoma Presenting as Diffuse Myocardial Metastatic Disease

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A 64-year-old man who had undergone treatment for left parotid adenocarcinoma presented with progressive exertional shortness of breath. Evaluation revealed metastatic invasion of the myocardium as a rare presentation of recurrent parotid adenocarcinoma. This case highlights the importance of using multimodal imaging methods in diagnostic evaluation and a collaborative multidisciplinary approach in managing patient care. (Tex Heart Inst J 2022;49(3):e207424)

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Neoplastic metastases to the heart and pericardium, which occur more often than primary malignant cardiac tumors do, are found at autopsy in up to 18% of patients with a history of cancer.¹⁻³ Cardiac metastases affect cardiac function in only 30% of cases and tend to be clinically silent. To date, very few cases of parotid adenocarcinoma with metastasis to the heart have been reported.^{4,5} We describe the case of a patient in whom symptomatic metastatic invasion of the myocardium was the primary presentation of recurrent parotid adenocarcinoma.

Case Report

A 64-year-old man presented at our cardiology clinic for routine follow-up evaluation after a recent hospital admission for pericardial effusion with tamponade. His medical history included stage IVA parotid adenocarcinoma, which had been treated by means of resection, chemotherapy, and radiation. He had been in remission for 2 years. Two months earlier, he had been hospitalized and underwent a pericardial window along with a pericardial biopsy. No malignant cells were seen on cytologic or pathologic examination.

In the clinic, the patient was asymptomatic. His physical examination was notable only for mild tachycardia. An electrocardiogram showed sinus rhythm with nonspecific T-wave changes. A transthoracic echocardiogram (TTE) showed mild concentric LV hypertrophy and a left ventricular ejection fraction (LVEF) of 55% (Fig. 1). No pericardial effusion was present; however, tissue-Doppler mode revealed myocardial-pericardial tethering of the LV lateral wall with annulus reversus (Fig. 2). No other findings suggested constrictive physiology.

After this clinic visit, the patient had progressive shortness of breath, and his medical history raised concern about constrictive pericardial disease. At his 2-month follow-up visit, cardiac magnetic resonance (CMR) images revealed diffuse LV hypertrophy with asymmetric involvement; the lateral wall was most affected, with relative sparing of the septum (Fig. 3). In addition, late gadolinium enhancement revealed diffuse, patchy, midmyocardial delayed uptake in the LV, most prominently in the lateral wall (Fig. 3). Other CMR images showed myocardial-pericardial tethering of both the LV lateral wall and the right ventricular (RV) apex (Fig. 4). However, no sign of increased ventricular interdependence was present. An infiltrative myocardial

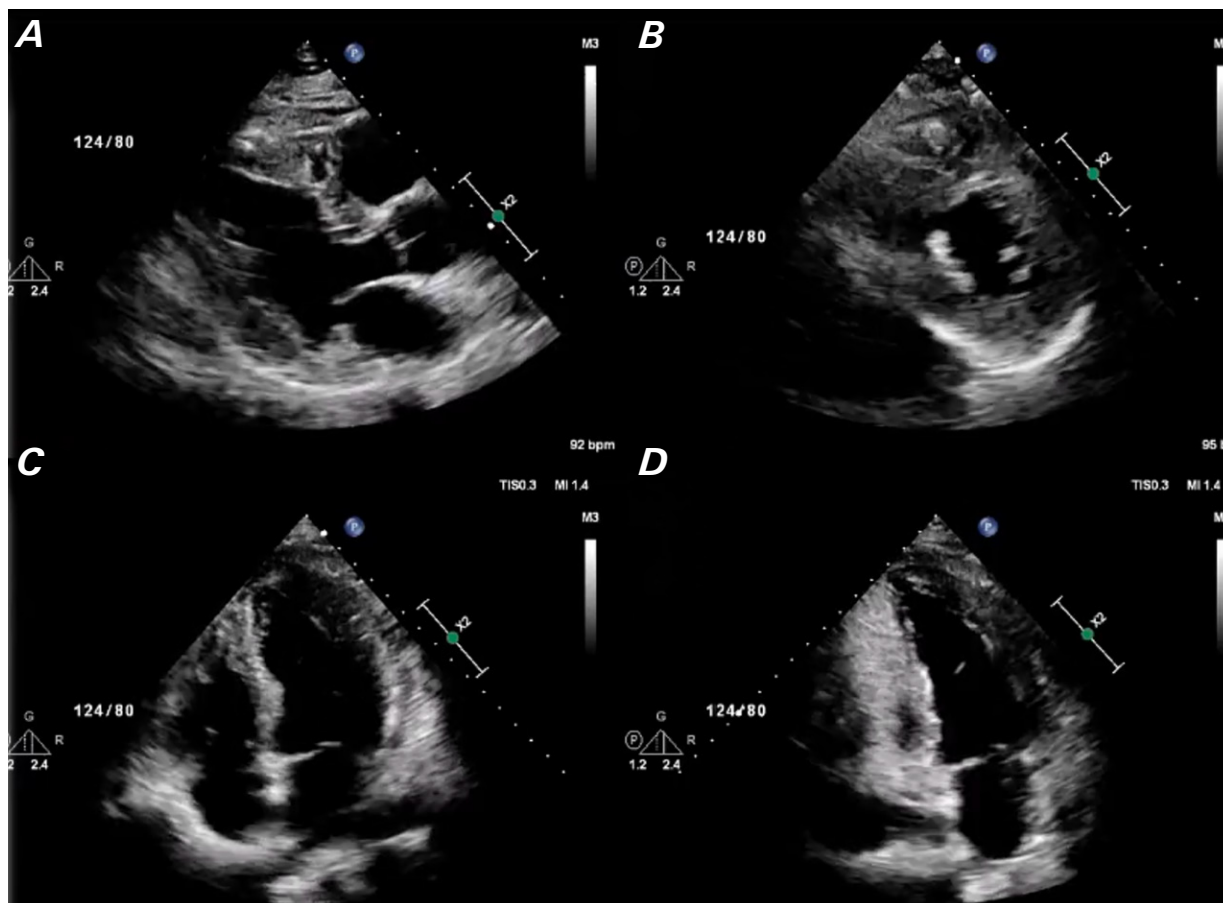


Fig. 1 Transthoracic echocardiograms in **A)** parasternal long-axis, **B)** parasternal short-axis, **C)** apical 4-chamber, and **D)** apical 5-chamber views show mild left ventricular hypertrophy and a normal left ventricular ejection fraction. The accompanying motion image shows lateral wall tethering.

Supplemental motion image is available for [Figure 1](#).

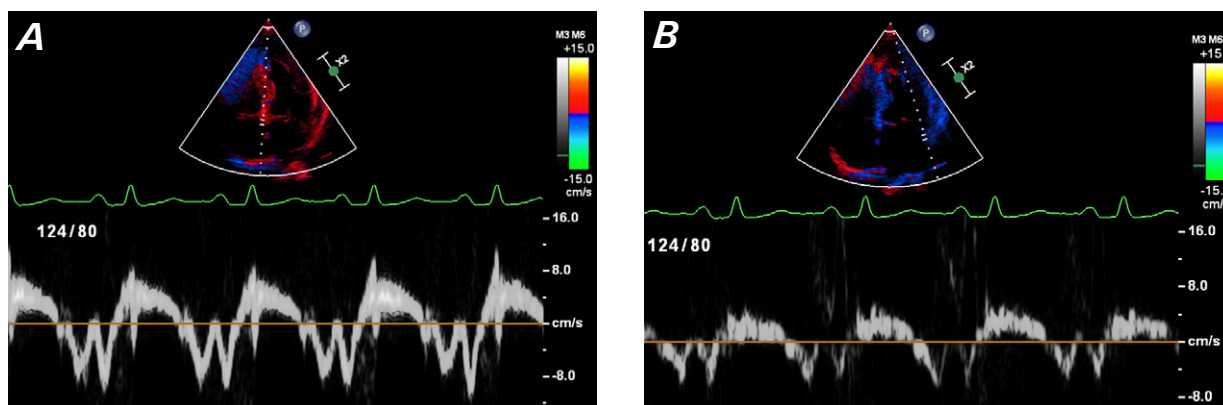


Fig. 2 Transthoracic Doppler echocardiogram shows the **A)** septal and **B)** lateral mitral annulus. Because of lateral wall tethering, the lateral e' velocity is reduced in comparison with the septal e' velocity (annulus reversus).

process, including metastatic disease, was thought to be the most likely cause. The patient was persistently hypercalcemic and had a newly enlarged right supraclavicular lymph node, biopsy of which confirmed recurrent parotid adenocarcinoma. Results of a subsequent positron emission tomographic/computed tomographic

(PET/CT) scan indicated infiltrative myocardial thickening with asymmetric lateral wall involvement, as well as increased nodularity adjacent to the pericardium and in the subepicardial region overlying the RV (Fig. 5). An RV biopsy specimen contained metastatic, poorly differentiated parotid adenocarcinoma.

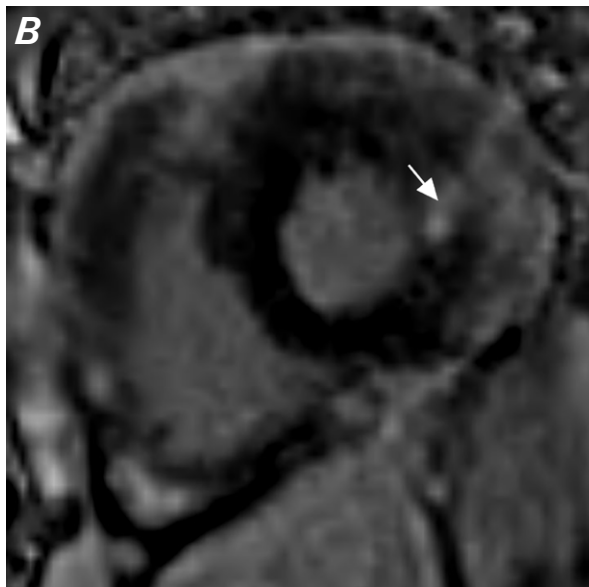
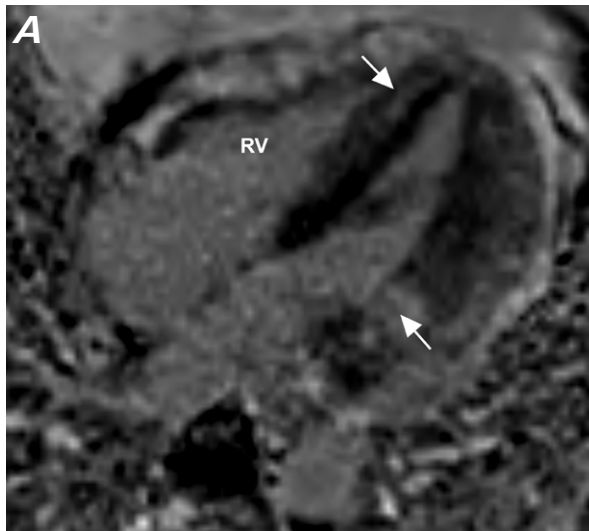


Fig. 3 Cardiac magnetic resonance images with late gadolinium enhancement show diffuse, patchy, mostly midmyocardial uptake throughout both ventricles, most prominently in the lateral walls. **A)** The 4-chamber view shows enhancement along the basal lateral left ventricular septum (arrows), and **B)** the short-axis view shows uptake in the lateral mid myocardium.

The patient's functional status continued to deteriorate, with rapidly worsening dyspnea, palpitations, and lightheadedness. Four months after his initial presentation, a TTE showed decreased LV and RV systolic function (LVEF, 49%) and increased hypertrophy of the LV lateral wall and RV free wall. It also revealed a restrictive filling pattern with signs of substantially reduced stroke volume. An echocardiographic polar map of the LV showed severely reduced systolic strain in the anterior, lateral, and inferior walls (Fig. 6).

The patient's decreased cardiac output and limited filling caused by right atrial compression made fluid management challenging. His multidisciplinary medical team

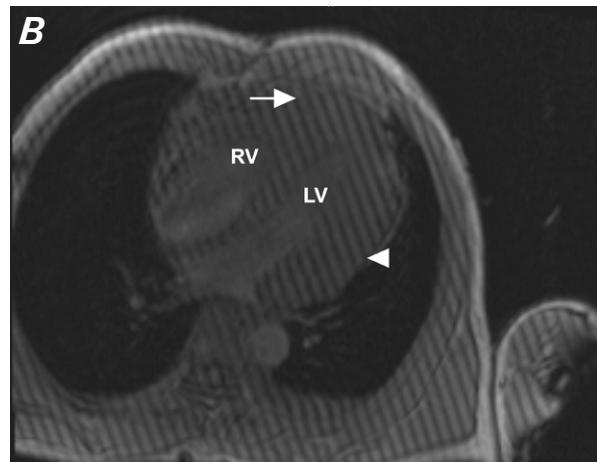
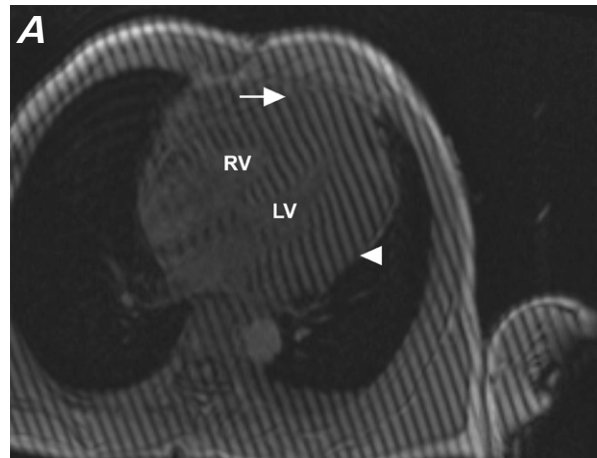


Fig. 4 Cardiac magnetic resonance images in **A)** systole and **B)** diastole show tethering of the left ventricular (LV) lateral wall (arrowheads) and the right ventricular (RV) apex (arrows) with contrasting tag lines not breaking at the RV myocardial-pericardial junction. In patients without cardiac disease, tag continuity is disrupted during cardiac contraction because of shear motion between the visceral and parietal layers.

Supplemental motion image is available for [Figure 4](#).

concluded that he was not a surgical candidate, given the extent of neoplastic myocardial invasion spanning large portions of both ventricles, the severe biventricular dysfunction, and the restrictive cardiomyopathy. He was started on palliative chemotherapy, was later transferred to hospice care, and died 6 months after the onset of the hypercalcemia and dyspnea.

Discussion

Metastatic invasion of the myocardium and pericardium can occur through hematogenous spread, lymphatic spread, or direct invasion.⁶ Cardiac metastases most often originate from carcinomas of the lung, gastrointestinal tract, or breast, or from lymphomas, melanoma, or sarcomas.^{2,3} Although metastatic parotid adenocarcinoma with myocardial infiltration that results in symptom-

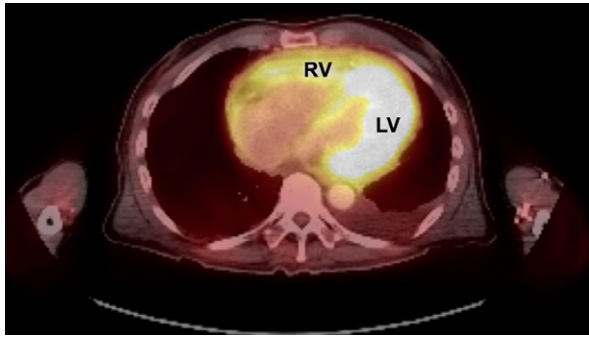


Fig. 5 Positron emission tomographic/computed tomographic scan shows infiltrative myocardial thickening with increased metabolic activity in both ventricles (most pronounced in the left ventricular [LV] lateral wall), as well as increased nodularity adjacent to the pericardium and in the subepicardial region overlying the right ventricle (RV).

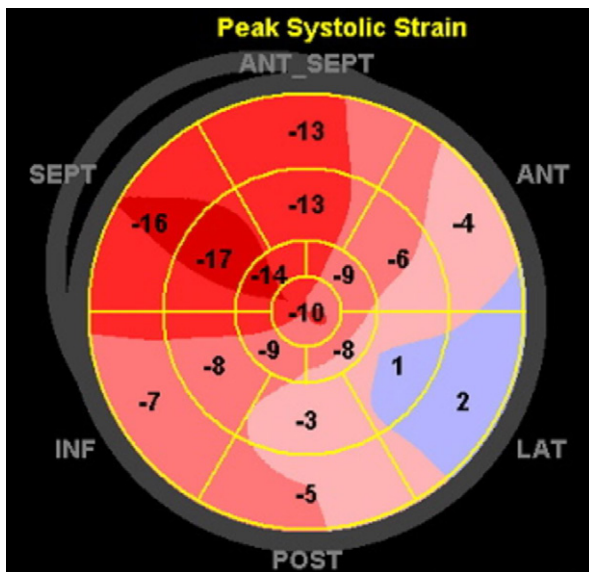


Fig. 6 Polar map from speckle-tracking echocardiography shows bull's-eye representation of left ventricular global longitudinal systolic strain. Severely reduced strain is evident in the anterior, lateral, and inferior walls.

ANT = anterior; ANT_SEPT = anteroseptal; INF = inferior; LAT = lateral; POST = posterior; SEPT = septal

atic heart failure is rare,^{4,5} strong suspicion is important when patients who have a history of advanced cancer present with cardiovascular symptoms.

Our case highlights how multimodal imaging can improve diagnostic evaluation and help to determine the extent of cardiac metastasis. The complementary results of TTE, CMR, and PET/CT contributed to reaching the correct diagnosis. In addition, this case illustrates the importance of a collaborative multidisciplinary approach. The oncology team managed the tumor and advised other specialists. The cardiology and cardio-oncology teams analyzed images and biopsy specimens to reach a diagnosis, and they managed the

patient's cardiac symptoms. The cardiovascular surgery team created the pericardial window and provided surgical input throughout. Finally, the palliative care team established goals of care and assisted with symptom management.

Conclusion

Cardiac metastasis should be considered when cardiovascular symptoms develop in a patient with a known malignancy. The use of multimodal imaging can greatly facilitate diagnosis and help in determining the extent of cardiac involvement. In addition, a collaborative multidisciplinary approach can accelerate the evaluation process so that the best treatment plan can be formulated in challenging cases.

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