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Case Report

A Rare Case of Endometrial Adenocarcinoma with both Left and Right Cardiac Metastasis

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Abstract

Endometrial adenocarcinoma rarely metastasizes to the heart. Few reports in the literature have discussed such situations with this type of carcinoma. Most metastases to the heart originate from lung, breast, thyroid, or skin (melanoma). In most cases, only one heart cavity is involved. We report a rare case of a 66 year old woman with endometrial adenocarcinoma who has metastases to both the right and left ventricles.

Introduction

We report a case of a 66 year old woman who was referred to our cardiology clinic for investigation of an intra-cardiac mass. She has a history of coronary artery disease, hypertension, hypercholesterolemia and type II diabetes. She had been diagnosed with stage 3A endometrial adenocarcinoma 16 months earlier with metastases to her ovaries that were treated with surgical resection followed by systemic chemotherapy.

A transthoracic echocardiogram was performed after images from a follow-up abdominal CT scan raised the possibility of an intra cardiac mass. It showed an echo dense, immobile mass measuring $4.0~\rm cm \times 2.3~\rm cm$ attached to the right ventricular free wall. This mass was protruding into the tricuspid inflow tract without inducing any significant flow obstruction across the valve (Figure 1, movie 1). The Right Ventricular Outflow Tract (RVOT), atria and left ventricle appeared to be uninvolved.

We proceeded with a biopsy of the mass which confirmed an adenocarcinoma that originated from the endometrium. The patient was subsequently referred for surgical resection but ultimately refused cardiac surgery. She was started on experimental chemotherapy as palliative treatment.

The patient was sent back to our clinic 4 months later for complaints of shortness of breath and progressive lower extremity edema. Her blood pressure was 122/70 mmHg with a resting heart rate of 82 beats per minute. Physical examination revealed an elevated central venous pressure and a paradoxal pulse of 15 mmHg, while the rest of her physical exam was unremarkable. A repeat TTE showed the right ventricular mass to have significantly increased in size, now measuring 5 cm \times 3.1 cm and almost completely occupying the ventricular cavity (Figure 2a, 2b and movie 2a, 2b). A tricuspid inflow obstruction was now present with a pressure gradient of 3 mmHg across

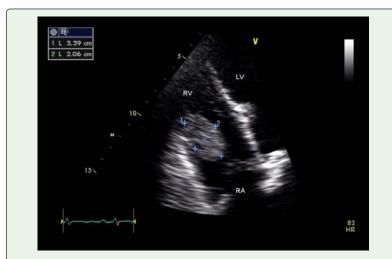
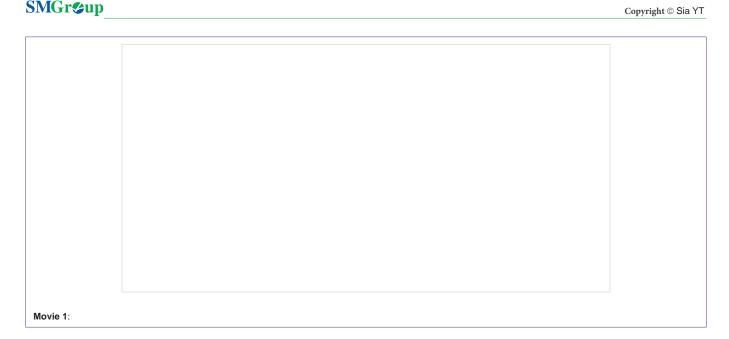


Figure 1: Apical four chamber view showing an echo dense immobile mass in the right ventricle attached to the free wall 12 months after diagnosis of endometrial adenocarcinoma.



the tricuspid valve. No pericardial effusion was seen. In addition, we found a small mobile echo dense mass measuring 1 cm \times 1 cm attached to the mid septal wall of the left ventricle. This second mass had not been apparent on the first TTE done 4 months earlier (Figure 3).

Discussion

Cardiac tumors are very rarely diagnosed. Previous trials have shown that the incidence of primary cardiac tumors ranges between 0.1 to 0.2 % [1,2]. Metastatic tumors to the heart are more frequent. Cancers that most frequently metastasize to the heart are lung and kidney carcinoma, lymphomas, melenoma, breast and thyroid cancers [3]. These cancers may spread to the heart by hematogenous route, growth or spread through the vena cava, or through direct invasion.

To the best of our knowledge this is only the seventh reported case of endometrial adenocarcinoma that has metastasized to the heart, and only the fourth reported case to have actually metastasized into a cardiac cavity (the three other cases had metastasized to the pericardium) [4]. Butany, et al. confirmed this low incidence by reporting that only 2 cases among 113 reproductive system cancers had metastasized to the heart [5]. In contrast, a necropsy series revealed a higher incidence of cardiac metastasis with a reported incidence of 17.2% and 16.7% in cases of small cell and uterine carcinomas respectively [6]. This difference could be related to the fact that most cardiac metastases are either not identified or remain clinically silent pre-mortem.

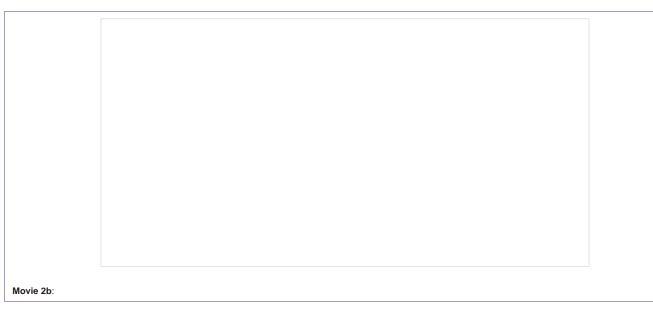
In reviewing the literature, we did not find other cases where both the right and left ventricles were involved in metastatic



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endometrial adenocarcinoma. Although we did not get a biopsy of the left ventricular mass, the appearance of the new lesion in the left ventricle, which had not been present four months earlier, led us to believe that it most probably represented further metastatic spread of the cancer. The mechanisms by which the cancer cells metastasize to the left ventricular myocardium are debatable. The first hypothesis is thought to be direct invasion of carcinoma from the right sided to the left sided. This form of spread is seen in cases of hepatoma or renal cell carcinoma (right side) or angiosarcoma (left side).

However, most of the time, only one side of the heart is involved. To reach both ventricles, spread of cancer cells could also occur via hematogenous seeding. Circulating cancer cells or tumor emboli need to pass through the pulmonary circulation or patent atrial septal defect. In the case of the former mechanism, most metastases are filtered by the pulmonary microvasculature and never reach the left side of the circulation, while in cases of a patent foramen ovale with right to left shunt as in the latter mechanism, left sided metastates or arterial tumor embolization can occur. This phenomenon might account for the very low incidence of left ventricular metastases.

Another interesting aspect of our case is the presence of a pulsus paradoxus. Classically, a paradoxical pulse is present when there is a competition of filling pressures between the two ventricles within the pericardium, also known as ventricular interdependance. In the presence of an external constraint on the myocardium, such as a pericardial effusion with increased intrapericardial pressure, an increase in venous return to right ventricle on inspiration causes a septal shift towards the left ventricle, leading to a reduction of left ventricular stroke volume and decreased pulse pressure. However there was no pericardial effusion detected in this case. We postulate therefore that the large mass in the right ventricle per se acted as a kind of internal restraint with a dynamic obstruction. Cyclical respiratory changes in RV venous return, RV after load or pulmonary vascular capacitance may have modified the inflow pressure gradient, leading to changes in right atrial and RV size and thus ejection volume. Other possible explanations for the pulsus paradoxicus observed could be hypovolemia (reduced venous return on the right due to the obstructive RV mass) or exaggerated inspiratory effort because of anxiety and dyspnea.



Figure 2a: Apical four chamber view showing the same mass 16 months after initial diagnosis of endometrial adenocarcinoma.



Figure 2b: Modified RV inflow view showing a mass in the right ventricle.

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Figure 3: Continuous doppler through tricuspid valve showing a mean gradient of 3 mmHg caused by the mass obstructing the tricuspid flow.

Conclusion

Although gynecological tumors have rarely been reported to metastasize to the heart in living patients, necroscopic reports has shown that cardiac involvement might be more frequent than previously believed. A patient with a known gynecological tumor manifesting new cardiovascular symptoms should prompt the physician to perform a complete cardiac evaluation and physical examination. We don't believe that echocardiography should be included as part of initial gynecological cancer staging workup given the low yield in asymptomatic patients. However, if clinical signs and symptoms suggest cardiac involvement, echocardiography should be considered. This would allow us to better define the true incidence of cardiac metastases from gynecological tumors in living subjects.

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