The authors report a 52 year-old female with a complex medical history including non Hodgkins lymphoma with cutaneous T-cell lymphoma subtype Sezary syndrome for three years. She had received chemotherapy including Doxorubicin as recently as several weeks prior to an admission for a non productive cough, shortness of breath and fatigue after long air travel. She also had a history of hypereosinophilia and hyperlipidemia. Initial work up included a pulmonary CT angiogram that was negative for pulmonary embolus, a chest film that was by report consistent with heart failure or infection and a transthoracic echocardiogram that demonstrated a left ventricular apical mass. Evaluation of the left ventricular mass was then pursued by multiple imaging modalities that in some ways seemed to add to confusion rather than identify what it was. Because multiple different etiologies were considered, multiple antibiotic and anticoagulant regimens were used intermittently over a period of several weeks. She developed scintillating scotomas and was found to have two intracranial lesions on MRI consistent with systemic emboli. Since this was in the setting of a left ventricular mass with mobile components, she was considered high risk for recurrent emboli and the left ventricular mass was removed surgically through a left atrial approach. On histology it was a thrombus with a ‘fibrin rich collagen scar’. Importantly there were no findings of malignancy. The authors conclude that this thrombus resulted from a hypercoagulable state accompanying her multiple medical problems. They also conclude that the left ventricular wall motion abnormalities intermittently noted with several imaging modalities were caused by the thrombus.

Prerequisites for intravascular thrombosis, including intracardiac thrombus, were described by Virchow and include hypercoagulability, hemodynamic abnormality (primarily stasis) and endocardial injury. The authors feel that in this case a hypercoagulable state led to a left ventricular thrombus. Hypercoagulability as the sole cause of intracardiac thrombosis occurs but is rare without either or both of the other components of Virchow's triad. Did the authors consider that dynamic changes in left ventricular function secondary to chemotherapy induced cardiomyopathy in the setting of a hypercoagulable state as a cause of this patients clinical course? Myocardial infarction and stress cardiomyopathy are also possibilities as the authors discuss.

The sequence of events in this case should lead to consideration of
chemotherapy induced cardiomyopathy with decreased left ventricle function presenting with symptoms and signs of congestive heart failure and initial chest film findings also consistent with heart failure. In the setting of a potential hypercoagulable state this could have led to development of thrombus. A thrombus was noted on initial transthoracic echocardiogram but the authors do not report the ejection fraction, if it was reduced, it would support this sequence of events. The course of chemotherapy induced cardiomyopathy frequently includes recovery of function and this could explain an ejection fraction of 68% on the preoperative echocardiogram. In addition improvement in left ventricular function could change the thrombus shape resulting into a protruding, mobile thrombus and even resulting in systemic embolization. Other causes of reversible regional or global left ventricular dysfunction leading to thrombus are myocardial infarction from acute coronary syndrome and stress cardiomyopathy (Tako-Tsubo syndrome). Coronary arteriography was not performed so we do not know if the patient’s coronary arteries were diseased and the trend in troponin I levels and serial ST-T changes were not reported so it is difficult to incriminate either of these etiologies but both are possible.

To consider publication, the authors should provide or discuss:

1. Include congestive heart failure as the cause of the initial hospitalization to explain the symptoms, signs and radiographic findings. Most important here would be the ejection fraction on the initial echocardiogram.
2. The trend in troponin I levels and serial ST-T changes from the initial hospitalization. These could support infarction or stress cardiomyopathy
3. Between the last two imaging procedures was the patient continued on anticoagulant? If so the decrease in size of the mass would have been the clue that this was thrombus rather than tumor.
4. Surgical findings: a. Was the ‘fibrin rich collagen scar’ part of the thrombus or ventricular wall? b. Did the surgeons biopsy the left ventricular lateral wall to evaluate for malignancy?
5. Were follow-up images of the left ventricle performed and if so, did the left ventricular lateral wall and apex look and move normally?
6. The authors feel the left ventricular thrombus caused the wall motion abnormality rather than the other way around but they present no mechanism to explain this and the literature would not support this contention. They should propose a mechanism for their opinion.

The authors conclude that this case highlights the challenges in diagnostic assessment of cardiac masses and their management in the presence of multiple comorbidities. This case indeed demonstrates these challenges despite multiple imaging techniques. I am attracted to the possibility that there was some reversible event that led to decreased systolic performance of the left ventricle and contributed to formation of a thrombus as noted above. The authors feel the left ventricular thrombus caused the wall motion abnormality rather than the other way around but they present no mechanism to explain this and the literature would not support this contention.
Level of interest: An article of limited interest

Quality of written English: Acceptable

Declaration of competing interests:
I declare that I have no competing interests.