around common intrathoracic causes of pleural effusion. The effusions responded poorly to thoracentesis and cleared only after splenectomy. Koehler and Jones commented that the mechanism by which the effusions formed was unknown.

Leung et al., studying ureteral obstruction and pleural effusion, expanded on the work of Koehler and Jones and postulated that the effusions were due to compression of the posterior lymphatics draining the thorax. The relationship of subphrenic inflammation and increased filtration of peritoneal fluid into the pleural space has been described previously by Miller and Talman.

To understand how these effusions could occur, it is important to understand the lymphatic drainage of the chest. The costal pleura drains into the internal mammary and intercostal lymphatic system. The visceral pleura drains into the mediastinal lymph nodes. There are also unidirectional transdiaphragmatic vessels which descend and drain the posterior thorax, receiving tributaries from the diaphragm and splenic mesentery. We suggest that the exudative effusion found in our patient with SVT can be explained by a combination of (1) direct compression of the posterior lymphatics by the enlarged infarcted spleen and (2) filtration of hemorrhagic splenic fluid into the pleural space due to increased permeability caused by peri-splenic inflammation.

We carried out a ten-year retrospective review of unexplained left-sided pleural effusions at our institution (a 465-bed tertiary hospital) and found an additional patient with SVT. This was a 77-year-old woman admitted with weakness, near syncopal episodes, and a left-sided pleural effusion. After an extensive evaluation the patient was found to have SVT, documented by abdominal ultrasound and CT. No cause for the SVT or pleural effusion was found. Several months later, a repeat abdominal CT showed no change in splenic defects and only partial resolution of the left-sided pleural effusion. Although not conclusive, this additional case further supports the association which we have proposed.

In summary, to our knowledge, the association of SVT and left-sided pleural effusion has not been previously reported. Although an uncommon cause, we suggest that SVT should be considered in the differential diagnosis of left-sided pleural effusion.

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Transthoracic and Transesophageal Echocardiography in the Diagnosis and Surgical Management of Right Atrial Myxoma

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An asymptomatic patient was discovered to have a large right atrial myxoma by transthoracic echocardiography. Preoperative considerations included the possibility of satellite lesions, left atrial origin, and a question of tricuspid valve involvement. Subsequent operative transesophageal echocardiography demonstrated single-stalk attachment in the right atrial septal wall and no satellite lesions. Doppler and color flow examination immediately following tumor removal aided in the decision not to perform tricuspid annuloplasty as there was no significant tricuspid regurgitation. The combined use of transthoracic and transesophageal echocardiography with Doppler and color flow imaging aids in the preoperative and intraoperative diagnosis and surgical management of right atrial tumors.

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Primary cardiac tumors are extremely rare with a reported incidence ranging from 0.0017 to 0.28 percent in most autopsy studies. Atrial myxomas compose 30 to 50 percent of most pathologic series with right atrial myxomas accounting for less than one quarter of these. Approximately 10 percent of all myxomas have multiple locations and some involve the valvular and subvalvular apparatus. Surgical removal is routinely recommended due to potential embolic phenomena. To our knowledge, this is the first reported use of the utility of transesophageal echocardiography to clarify questions raised by transthoracic echocardiography and to aid in the surgical management of right atrial myxoma.

CASE REPORT
An asymptomatic 51-year-old man was referred for transthoracic echocardiography due to a heart murmur. The patient had a history of heart murmur for 12 years. Transthoracic echocardiography disclosed a large fenestrated mass in the right atrium that prolapsed through the tricuspid valve during diastole (Fig 1). Color flow and Doppler echocardiography disclosed right atrial and right ventricular enlargement with tricuspid regurgitation that could not be quantified due to masking by the calcified myxoma. In addition, unexplained densities were seen on the right ventricular free wall insertion of the moderator band and the left atrial septum. Questions arose as to the degree of tricuspid regurgitation, left atrial origin of the myxoma, and the possibility of satellite lesions in the right ventricle. During subsequent intraoperative transesophageal echocardiography, the right ventricle was interrogated with no indication of satellite lesions. Tricuspid regurgitation was seen and thought to be moderate at most. A single attachment site to the right atrial wall in the region of the fossa ovalis was found (Fig 2). With

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Diagnosis

Atrial myxoma; prolapsing into the right ventricle. RA = right atrium; RV = right ventricle; LV = left ventricle; MYX = myxoma.

Subsequent removal of the myxoma and with resection and patching of the atrial septum, the ventricle was again interrogated for satellite lesions and residual tumor and none was found. Tricuspid regurgitation was noted by Doppler and color flow and was believed again to be mild to moderate (Fig 3). In view of good cardiac output and the limited amount of valvular regurgitation, no repair of the tricuspid annulus was performed. The postoperative course was uneventful. The patient remained asymptomatic with no evidence of clinically significant tricuspid regurgitation at two-month follow-up.

Discussion

While primary cardiac tumors are extremely rare, their diagnosis may be suggested by clinical and physical findings. In this case, a systolic murmur was the only suggestion of a right atrial myxoma. Numerous reports have demonstrated the efficacy of transthoracic echocardiography in detecting the presence of intracardiac tumors. Transthoracic echocardiography has provided definition of the size, mobility, locations, and sites of attachment of cardiac tumors.

Figures 1 and 2: Transthoracic echocardiogram of right atrial myxoma prolapsing into the right ventricle. RA = right atrium; RV = right ventricle; LV = left ventricle; MYX = myxoma.

Figure 3: Transeosophageal view following tumor removal demonstrating mild tricuspid regurgitation by color flow. RA = right atrium; RV = right ventricle; LA = left atrium; TR = tricuspid regurgitation.

Transeosophageal echocardiography has recently been used in the interrogation of left atrial myxomas and has provided superior visualization of the tumor attachment sites due to better resolution of the posterior cardiac structures. In this particular case, transthoracic echocardiography raised the question of (1) possible satellite lesions, (2) a left atrial origin with extension through the fossa ovalis, and (3) the severity of the tricuspid regurgitation. Through subsequent transeosophageal echocardiography, all of these questions were answered. In addition, transeosophageal echocardiography provided useful information in the intraoperative state in regard to the necessity of repair of the tricuspid annulus. Lastly, the integrity of the interatrial patch was demonstrated prior to the patient leaving the operating room.

In conclusion, transeosophageal echocardiography aids in the preoperative and intraoperative evaluation of intracardiac tumors. It also provides useful information following the intraoperative removal of intracardiac tumors regarding tricuspid regurgitation and the integrity of the right atrial patch. It provides valuable information on the extent of the intracardiac tumors, their effect on valvular function, and the ultimate efficacy of the surgical treatment.

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