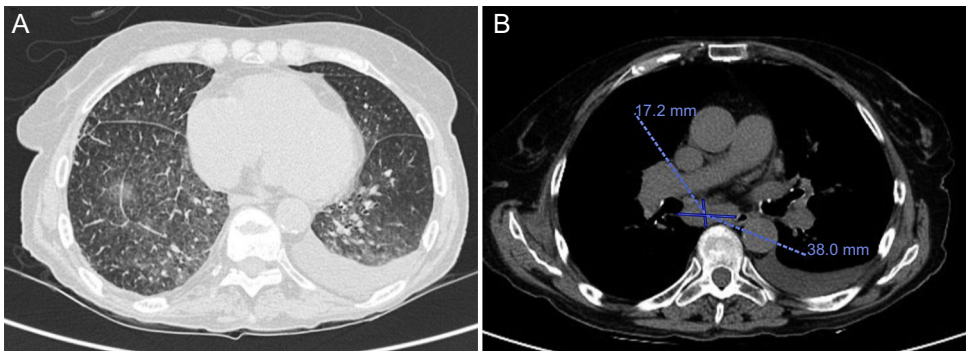

Figure. High-resolution lung computed tomography. A, Nodular ground-glass opacities with a centrilobular distribution and thickening of interlobular septa. B, Subcarinal mediastinal adenopathy, with a diameter up to 38 mm.

a specificity of up to 100%.[2] In addition, a DLCO below 55% has a specificity of 89.5%, and the diagnosis is further strengthened by the right-side catheterization data and the respiratory failure.

The initiation of specific treatment in PVOD patients carries a recognized risk of acute pulmonary edema. Nonetheless, improvements with these therapies have been documented in small series and individual cases, the vast majority with low dose epoprostenol. To date, only minor and transient improvements have been documented with oral vasodilators,[4] and there is only 1 reported positive experience with combined treatment.[5] This is thus the first report of a clear improvement with combined oral therapy and a beneficial effect of macitentan in the treatment of PVOD.

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Late Detachment of an Aortic Prosthetic Valve and Reactivation of Takayasu Arteritis

Dehiscencia tardía de prótesis valvular aórtica y reactivación de arteritis de Takayasu

To the Editor,

Takayasu arteritis (TA) is a chronic autoimmune disease that affects the aorta and proximal segments of arteries arising from the aortic arch, especially the left subclavian artery. The pulmonary artery, its branches, and sometimes the coronary circulation may also be involved. The disease causes dilatation of the affected vessels with or without the formation of aneurysm, and stenosis or occlusion, accompanied by vessel wall thickening and calcification.[1]

The incidence of significant aortic regurgitation is estimated to be between 13% and 25% in patients with TA. The etiology has been associated with aortic root dilatation,[1] as well as with aortic valve leaflet inflammation.[2] Aortic regurgitation, hypertension, and, sometimes, coronary lesions are the main factors associated with the onset of heart failure.[1] Aortic valve surgery entails a significant risk of prosthetic valve detachment and the formation of pseudoaneurysms along the suture line.[1,3,4]

We present the case of a male patient with TA who had received an aortic valve replacement. The immunosuppressive therapy was discontinued, TA reappeared, and late valve detachment then occurred.

The patient was diagnosed with TA and hypertension in 2001, at the age of 16 years. Magnetic resonance imaging showed damage to the ascending aorta, aortic arch and descending thoracic aorta, and stenosis of the left subclavian artery. The ultrasound study revealed mild aortic regurgitation and moderately depressed systolic function. The patient was treated with corticosteroids, methotrexate and antihypertensive medication. In 2003, he had symptoms of heart failure. An echocardiogram showed severe aortic regurgitation, moderately reduced systolic function, and dilatation of the ascending aorta. The patient

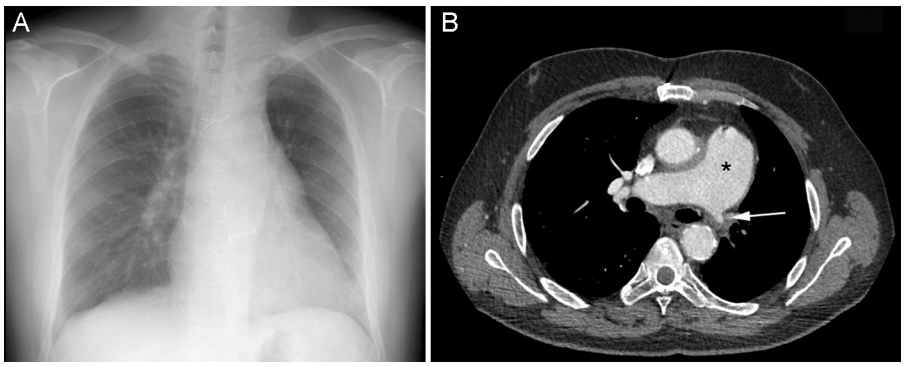

Figure 1. A: chest radiograph showing reduced left lung volume and lack of vasculature. B: computed tomography angiography showing dilatation of the pulmonary trunk (asterisk) and occlusion of the left pulmonary artery (arrow).

received an aortic valve replacement with a mechanical prosthesis, resection of the ascending aorta above the sinuses and placement of a Dacron graft.

Postoperative recovery was satisfactory. The patient remained asymptomatic and continued the immunosuppressive, anticoagulant and antihypertensive therapy until 2012. Yearly ultrasound studies confirmed normal valve replacement function and preserved ventricular function. In 2013, the patient decided to discontinue the immunosuppressive therapy and to stop attending any check-ups, until 2015, when he presented with progressive dyspnea. The echocardiogram showed prosthetic valve detachment, severe regurgitation, and preserved ventricular function. A radiograph showed reduced left lung volume, aneurysmal dilatation of the pulmonary artery trunk, and occlusion of the left pulmonary artery (Figure 1). Aneurysmal dilatation was also found in the brachiocephalic artery, with occlusion of the left subclavian artery, lack of arterial vessels in the left lung, and stenosis of the descending thoracic aorta with a minimum lumen diameter of 12 mm (Figure 2). A diagnosis was made of midaortic syndrome based on the lumen diameter, severe hypertension, and a systolic pressure gradient of 60 mmHg between the upper and lower extremities. In July 2015, an ^{18}F-fluorodeoxyglucose positron emission tomography confirmed active TA in the aortic root. The patient started treatment with corticosteroids and azathioprine, and 8 months later he was referred to our center for valve surgery.

During the procedure we observed that the prosthetic detachment affected two thirds of the circumference. In view of the diameter of the sinus portion of the aorta (42 mm) and the absence of macroscopic evidence of inflammation, we decided to implant a mechanical prosthesis instead of replacing the aortic root with coronary vessel reimplantation.

During anesthesia induction and the immediate postoperative period, we observed a systolic pulmonary pressure of about 75 mmHg, using a Swan-Ganz catheter. Postoperative recovery was satisfactory.

Early and late prosthetic valve detachment in TA is associated with an active inflammatory process.[1,2,4] For this reason it is recommended to prescribe immunosuppressant therapy pre- and postoperatively.[1,2,4] Due to the risk of disease reactivation when the immunosuppressant is reduced or stopped, regular follow-up is recommended in these patients.[1,4] Monitoring with ^{18}F-fluorodeoxyglucose positron emission tomography is useful to assess inflammatory activity.[5] Our patient had 2 rare complications of TA: complete occlusion of a pulmonary artery and midaortic syndrome. In patients with TA after valve problems have been overcome, the prognosis depends on the degree of vascular injury and hypertension severity, which may warrant future surgical interventions.

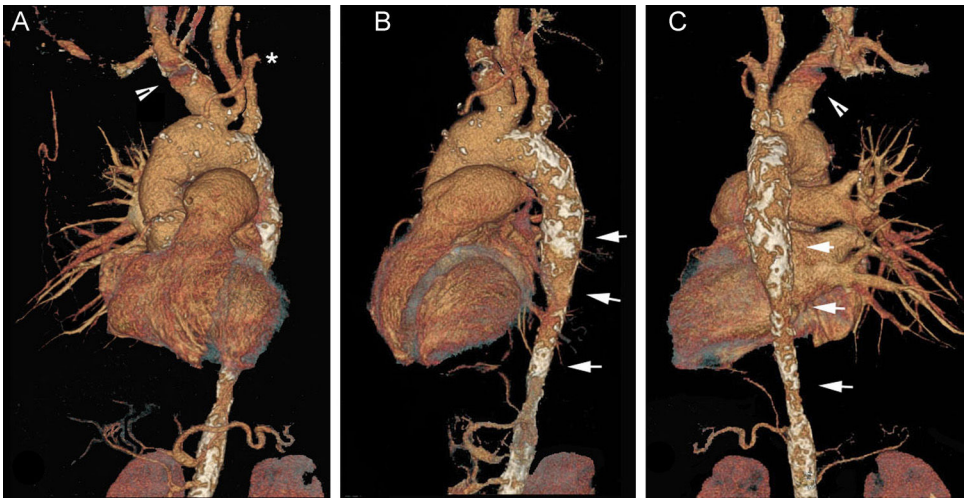

Figure 2. Computed tomography angiography with left anterior oblique (A), left lateral (B) and posterior (C) view reconstruction. Note the occlusion of the left subclavian artery (asterisk), dilatation of the brachiocephalic artery (arrow head), lack of left pulmonary artery, and stenosis of the descending thoracic aorta with calcified wall (arrow).

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Liver and Cardiovascular Disease: What Cardiologists Need to Know About Ultrasound Findings

Hígado y enfermedad cardiovascular: lo que el cardiólogo debería conocer de los hallazgos ecográficos

To the Editor,

Given the anatomical proximity of the liver and gallbladder to the heart, cardiologists should be able to identify certain common conditions affecting these organs that may have significant clinical implications for patient management. Here, we present typical findings with regard to liver echogenicity, size and edges, and focal lesion study.

A normal liver is minimally hyperechogenic or isoechogenic compared with the normal renal cortex[1] (Figure 1A and B). The most common cause of hyperechogenic liver (increased liver echogenicity compared with the renal cortex) in routine practice is steatosis, otherwise known as "fatty liver". This can be either diffuse or focal.

Areas of intact parenchyma are commonly found within diffuse steatosis. These areas are hypoechoic, as they correspond to "islets" of normal liver parenchyma that contrast with the pathological liver (with increased echogenicity due to the fatty infiltration).

Focal areas of fatty infiltration can also be found, ie, areas of increased echogenicity against the background of normal liver parenchyma.[1,2]

Both the intact parenchymal areas within diffuse steatosis and the areas of focal steatosis in normal liver have geographic borders, are not associated with mass effect, change over time (sometimes quickly), and are usually located in subcapsular or perivesical areas, adjacent to the portal bifurcation and the falciform ligament. They can, however, be found in any location. Occasionally, these areas may resemble nodules/solid masses[2] (Figure 1C-F).

Normal liver echogenicity is homogeneous, with fine echoes.[1] One of the main causes of heterogeneous echogenicity of the liver is chronic liver disease/cirrhosis (Figure 1 of the supplementary material). Other common conditions leading to heterogeneous echogenicity are patchy steatosis and diffuse tumor infiltration.[2]

In liver congestion triggered by right-sided heart failure, such as tricuspid regurgitation, a diffuse decrease in liver echogenicity can be observed.

Other typical findings of chronic liver disease/cirrhosis include volume redistribution, with an increase in the caudate lobe, the left lobe, or both, with respect to the right lobe, and liver surface irregularity.[1] These findings can occur in patients with Fontan circulation. The irregularity/nodularity of the liver contour is more obvious if there are ascites, which are often present in decompensated liver disease.

Hepatomegaly and dilation of the inferior vena cava and suprahepatic veins are typical findings in liver congestion, such as in tricuspid regurgitation and Ebstein anomaly.

Ultrasound is useful for distinguishing between cystic and solid lesions, whose management tends to differ significantly.

On ultrasound, lesions are defined as a simple cyst when it is anechogenic, with thin, smooth walls and posterior acoustic enhancement. A cystic lesion is complex if it does not fulfil all these characteristics, ie, if it has a thick or irregular wall, with mural nodules, septations, echogenic content, or calcifications. Hydatid cysts and abscesses are typical examples of complex cystic lesions (Figure 2E-H).

A solid nodule can be hyperechogenic, isoechogenic or hypoechogenic with respect to the adjacent parenchyma, it can be homogeneous or heterogeneous, and flow may be detected in the Doppler study, although a negative result does not rule out a solid lesion. If a solid nodule is detected, it should be compared with any previous studies that may be available to evaluate whether it is a new nodule, is stable, or has grown, and it should be correlated with the patient's medical history (eg, history of tumors, hepatitis B or C virus infection, alcoholic cirrhosis) for a decision to be made on the performance of any additional imaging tests.

The benign nodules most commonly observed in practice include hemangioma, which has a typical appearance of a hyperechogenic nodule with well-defined borders.

The 2 most common types of malignant hepatic lesions are metastasis and hepatocellular carcinoma.

There are generally various metastases and their ultrasound appearance is highly variable (hyperechogenic, isoechogenic or hypoechogenic compared with the adjacent parenchyma, and they can be homogeneous or heterogeneous, either with or without a halo, and may even be cystic or have calcifications[3,4]) (Figure 2A-D).

In most cases, hepatocellular carcinoma is found in a liver with chronic liver disease/cirrhosis. It can present as a solid nodule (or various in the case of multicentric hepatocellular carcinoma) and