

# Transcatheter Aortic Valve Replacement to Treat Liver Decompensation



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## INTRODUCTION

*Cardiac cirrhosis, congestive hepatopathy, and chronic passive liver congestion* are terms used to describe a spectrum of hepatic derangements secondary to cardiac disease. Major determinants of outcome include the severity of the underlying cardiac disease and the degree to which hepatic injury is related to passive congestion or diminished perfusion. In cases of critical aortic stenosis, increased left ventricular (LV) afterload may cause LV dilation, reduction in LV ejection fraction (LVEF), increased LV filling pressures, and an increase in left atrial pressure, resulting in increased pulmonary capillary wedge pressure and pulmonary hypertension. Subsequent right ventricular (RV) remodeling, dysfunction, and elevation of RV filling pressures may ensue, which may cause hepatic congestion in fulminant cases. Chronic hepatic congestion may cause hepatic parenchymal atrophy, necrosis, collagen deposition, perivenular fibrosis, and ultimately cirrhosis. Left untreated, both hepatic and cardiac damage may be irreversible.

We report a case of painless jaundice presenting secondary to biventricular dysfunction and pulmonary hypertension as a consequence of undiagnosed critical aortic stenosis. Definitive treatment was achieved by transcatheter aortic valve replacement (TAVR) that reversed our patient's clinical and biochemical congestive hepatopathy and normalized biventricular function at 8-week follow-up.

## CASE PRESENTATION

A 64-year-old female ex-smoker presented with anorexia, painless jaundice, and a 19-kg weight loss. In her medical history, she had mild chronic obstructive pulmonary disease, previous hysterectomy for dysfunctional uterine bleeding, and hypothyroidism. On examination, she was breathless at rest, in New York Heart Association class III to IV, afebrile, in sinus rhythm, and normotensive. Chest auscultation was consistent with severe aortic stenosis and right pleural effusion, the jugular venous pressure was elevated at 15 cm, and she had bilateral peripheral edema to above her knees. Biochemical testing revealed elevated bilirubin of 66  $\mu\text{mol/L}$  (normal range, 0–20  $\mu\text{mol/L}$ ) and liver alanine transaminase of 98 IU/L (normal range, 8–40 IU/L), with preserved synthetic liver function (albumin 35 g/L [normal range, 35–50 g/L] and prothrombin time 11.9 sec [normal range, 10.2–13.2 sec]). Thyroid function was normal. However, brain

natriuretic peptide level was markedly elevated at 3,568 ng/L (normal level <20 ng/L).

Treatment was commenced with intravenous furosemide 80 mg twice daily and oral spironolactone 25 mg once daily. A right-sided pleural drain was inserted, which drained 1,000 mL of transudate. Liver ultrasound demonstrated a mildly dilated common bile duct at 10 mm, and the patient was referred for endoscopic hepatobiliary ultrasound. Clinical management was altered when transthoracic echocardiography documented critical aortic stenosis in the presence of severe biventricular impairment. The left ventricle was not dilated (end-diastolic dimension 5.0 cm) but was significantly impaired (LVEF 15%–20%; [Figure 1](#), [Videos 1](#) and [2](#)); the right ventricle was dilated (RV base 4.8 cm, RV midcavity 3.3 cm, and RV length 5.7 cm), and tricuspid annular plane systolic excursion was reduced (1.0 cm). Despite reduced LVEF, the peak pressure gradient across the aortic valve was 85 mm Hg, there was Doppler evidence of elevated LV filling pressures (restrictive LV filling pattern with a lateral annular velocity of 5.5 cm/sec and E/E' ratio of 17.0), mild functional mitral regurgitation and tricuspid regurgitation (TR), the inferior vena cava was dilated (2.4 cm) with <50% inspiratory collapse, and estimated pulmonary artery systolic pressure was elevated at 65 mm Hg.

Given the extent and severity of biventricular impairment, our patient was classified as having stage 4 aortic stenosis<sup>1</sup> and transferred urgently to a cardiac center for consideration of aortic valve intervention. A cardiac-gated multislice computed tomographic scan was performed and demonstrated no flow-limiting coronary artery disease, small femoral access vessels (right femoral 5.5 mm and left femoral 5.9 mm) with moderate abdominal atheroma. The aortic annular circumference was 83 mm, and orthogonal cross-sectional diameters measured 29.7 by 23.2 mm ([Figure 2](#)).

The clinical history, relevant images, and severity of biventricular impairment were discussed at a structural heart disease multidisciplinary meeting. The patient was deemed at prohibitively high risk for conventional aortic valve replacement (logistic European System for Cardiac Operative Risk Evaluation score II was 29.98%), and she was accepted for TAVR. As she was deemed to be a high-risk patient, with significantly impaired biventricular function, it was suggested that the TAVR should be performed with the aid of periprocedural circulatory support. Under general anesthesia, cardiopulmonary bypass was achieved through a two-stage venous cannula in the right femoral vein returning to the right subclavian artery. Given the density of aortic valve calcification, a preballoon aortic valvuloplasty was performed with an 18-mm Crystal Balt Extrusion balloon. A surgical cut-down onto the right femoral artery was used to facilitate the implantation of a 26-mm CoreValve Evolut R (Medtronic, Minneapolis, MN) device during a short (2-min) period on full cardiopulmonary bypass. An excellent TAVR position was confirmed on fluoroscopy ([Video 3](#)) and transesophageal echocardiography ([Figure 3](#), [Video 4](#)), and cardiopulmonary bypass was discontinued uneventfully.

There was an immediate reduction in peak aortic valve gradient (from 85 to 10 mm Hg), visual improvement in LV function (with

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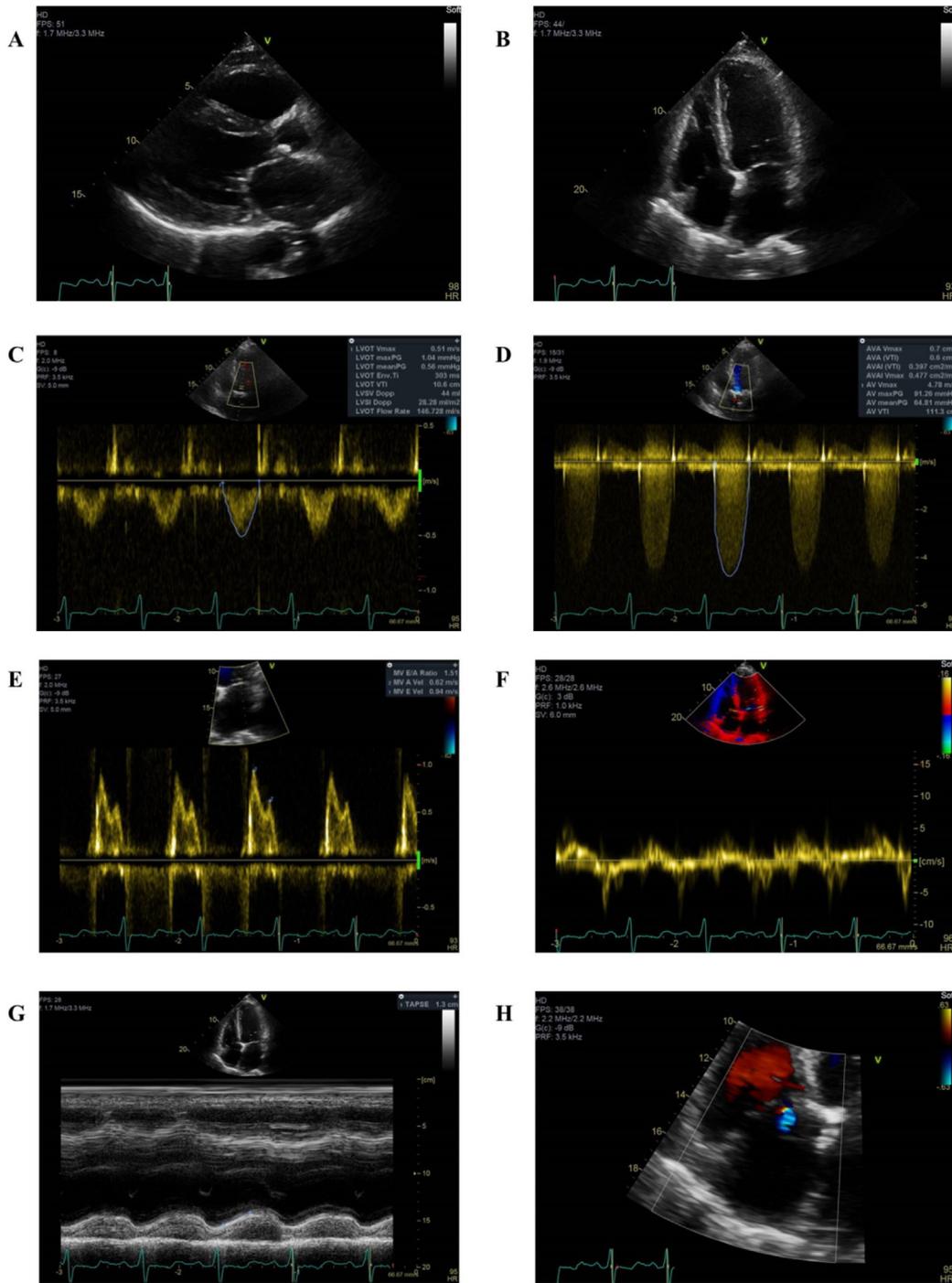
Keywords: Cardiac cirrhosis, Liver congestion, Critical aortic stenosis, Biventricular failure

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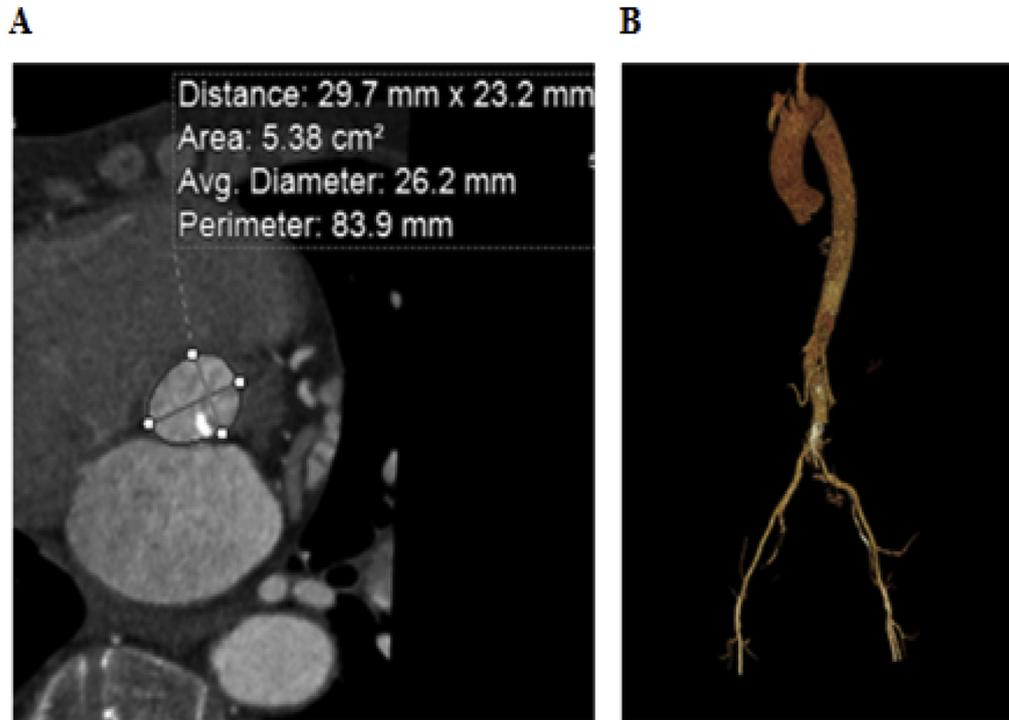


**Figure 1** Transthoracic echocardiographic images demonstrating severe biventricular dilatation (A,B), critical aortic stenosis with subvalvular velocity 0.6 m/sec (C), peak velocity of 4.8 msec<sup>-1</sup>, peak pressure drop of 91 mm Hg, mean pressure drop of 65 mm Hg (D), dominant mitral E-wave velocity and lateral tissue Doppler E' 5.5 cm/sec (E,F), giving a value for E/E' ratio of 17, reduced tricuspid annular plane systolic excursion of 1.0 cm (G), and mild TR on color Doppler (H).

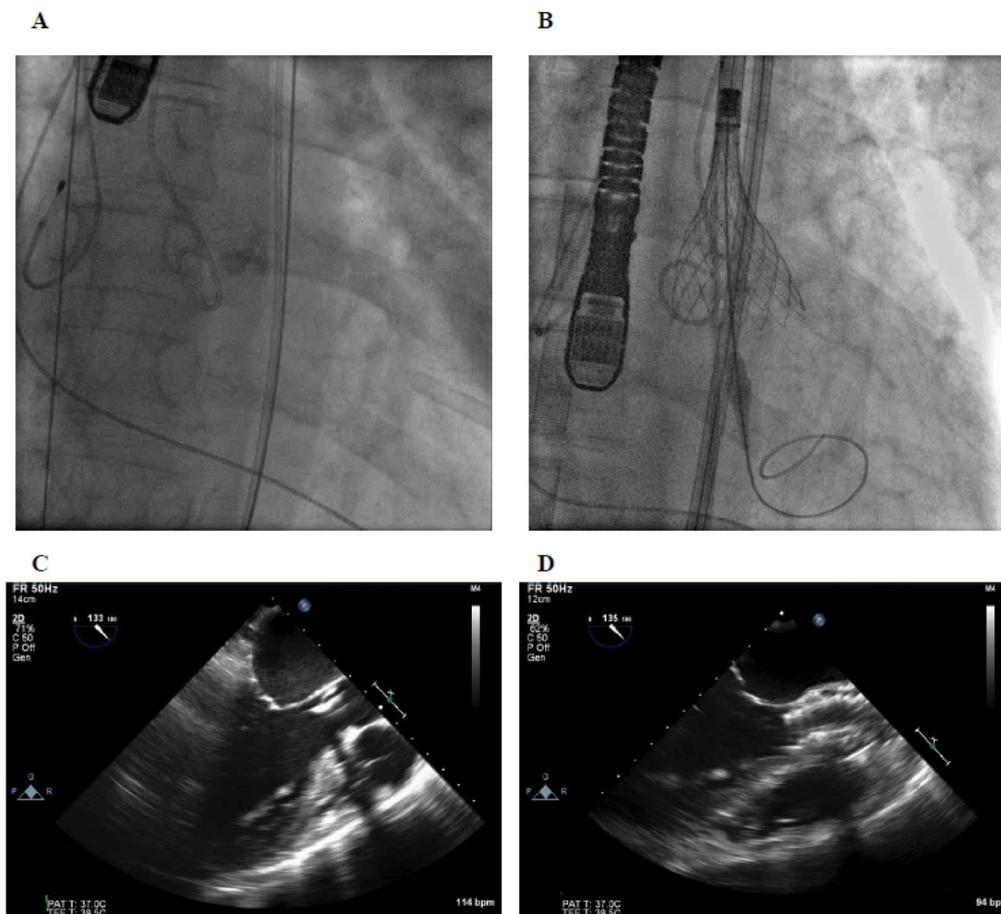
LVEF increasing from 10%–15% to 30%), and reduction in invasively measured LV end-diastolic pressure (from 45 to 20 mm Hg; Figure 4).

The patient was extubated, all inotropes were discontinued within the first 4 hours, and the remainder of the postprocedural period was uneventful. She was discharged on day 4, clinically euvolemic, with a reduction in brain natriuretic peptide level (to 235 ng/L) and normalization of liver function.

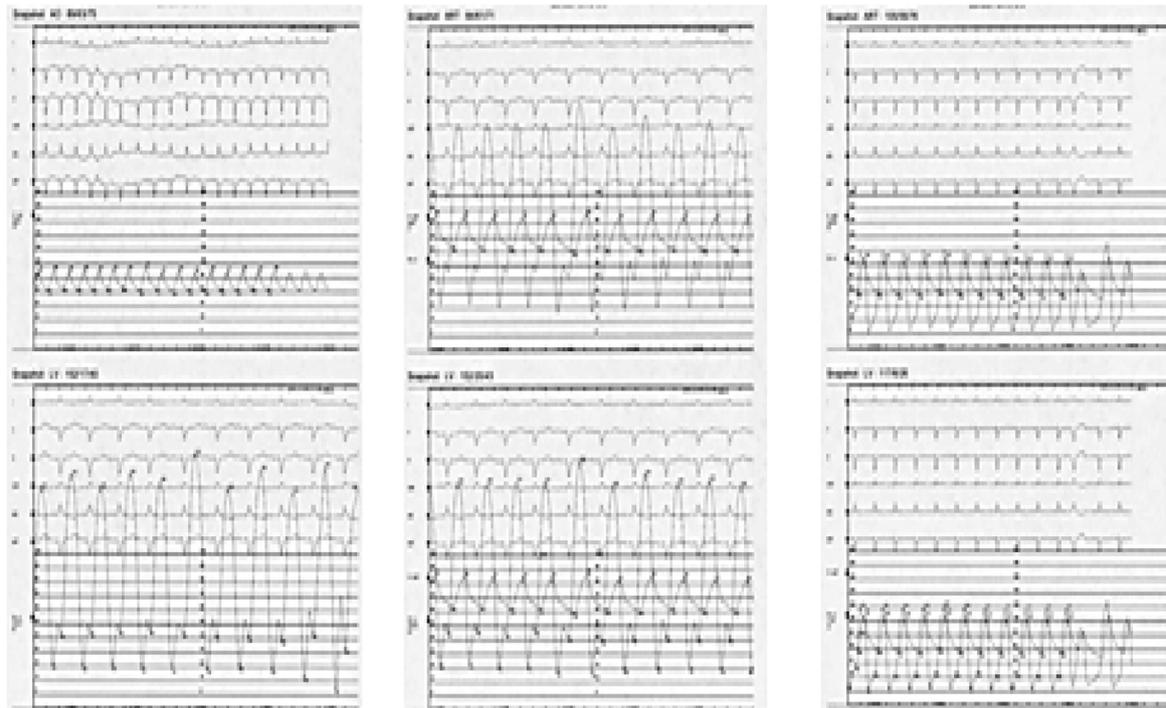
At 8-week follow-up, the patient was independent in all activities of daily living. On echocardiography, LV end-diastolic dimension had reduced to 4.0 cm and LVEF had increased to 60% to 65% (Videos 5 and 6). RV size had decreased (base 3.1 cm, midcavity 2.2 cm, length 5.6 cm), and RV tricuspid annular plane systolic excursion had increased to 1.7 cm (Figure 5). The TAVR device was well seated, with a peak pressure gradient of 25 mm Hg, and there was no



**Figure 2** Cardiac gated multislice computed tomography demonstrated an aortic annular circumference of 83.9 mm **(A)** and small iliofemoral access vessels **(B)**.



**Figure 3** Calcified aortic valve on fluoroscopy before TAVR **(A)**: the final TAVR position confirmed on fluoroscopy **(B)**. Calcified aortic valve on transesophageal echocardiography before TAVR **(C)** and the final TAVR position on transesophageal echocardiography **(D)**.



**Figure 4** Invasive hemodynamics with LV end-diastolic pressure (LVEDP) decreasing from 45 mm Hg immediately before TAVR to 20 mm Hg after TAVR and peak-to-peak aortic valve gradient decreasing from 85 to 10 mm Hg.

paravalvular leak. The change in LV filling pattern (restrictive LV filling pattern became dominant A, and lateral annular velocity increased from 5.5 to 7.2 cm/sec with a resultant decrease in E/E' ratio from 17.0 to 9.7) suggested a reduction in LV filling pressures, there was mild TR and trace mitral regurgitation, and estimated pulmonary artery systolic pressure had decreased to 24 mm Hg. Biochemically, brain natriuretic peptide had fallen to almost within the normal range (59 ng/L).

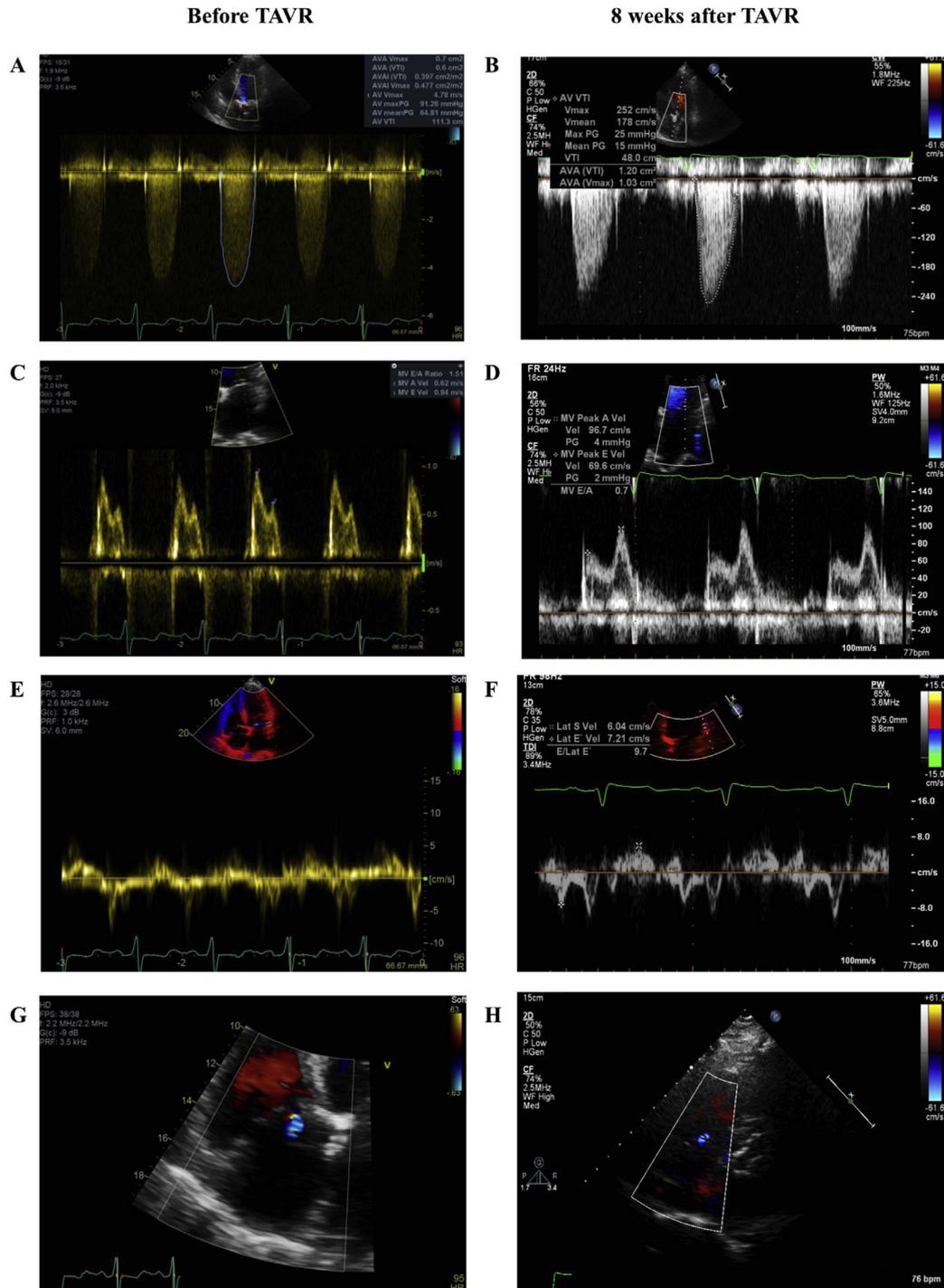
## DISCUSSION

Cardiomyopathy and valvular heart disease are recognized precipitants of cardiac cirrhosis. Despite its name, cardiac cirrhosis rarely satisfies strict pathologic criteria for cirrhosis. The terms *congestive hepatopathy* and *chronic passive liver congestion* are more accurate and describe a spectrum of hepatic derangements of variable severity,<sup>2,3</sup> determined by the blood vessels involved and the degree to which injury is related to passive congestion and/or diminished perfusion.<sup>4,5</sup> The hepatic vasculature is complex and particularly vulnerable to circulatory disturbance: portal venous flow does not have the capacity to autoregulate but is dependent on mesenteric circulation and the gradient between portal and hepatic venous pressure.<sup>6,7</sup> In the case of RV failure, elevated right atrial pressure is transmitted to the liver via the inferior vena cava and hepatic veins. At a cellular level, increased central venous pressure causes sinusoidal dilation of sinusoidal fenestrae and deoxygenated blood stasis,<sup>4</sup> which leads ultimately to exudation of protein and fluid into the space of Disse and further impairs diffusion of oxygen and nutrients to hepatocytes.<sup>5</sup> Left untreated, chronic congestion can result in parenchymal atrophy, necrosis, collagen deposition, perivenular fibrosis, and ultimately cirrhosis. In the setting of severe aortic stenosis, passive congestion coexists

with reduced cardiac output and hypotension, further decreasing hepatic blood flow.

Treatment of congestive hepatopathy due to cardiomyopathy or valvular heart disease involves improving cardiac hemodynamics, optimizing cardiac output, and managing the underlying cardiac condition.<sup>2,3</sup> Diuresis can improve hepatic congestion but requires caution to avoid precipitating hepatic ischemia.<sup>8</sup> Definitive hemodynamic optimization requires aortic valve intervention. Because our patient was at prohibitively high risk for conventional aortic valve replacement, TAVR was performed. That the patient's cardiac function instantaneously improved suggests that the degree of LV impairment before TAVR was most likely driven by afterload mismatch from critical aortic stenosis. Furthermore, as suggested by Dauerman *et al*,<sup>9</sup> the lack of flow-limiting coronary artery disease, prior myocardial infarction, and high baseline aortic valve gradient in our patient were predictors of early recovery of LV systolic function.

Over the past decade, several clinical and echocardiographic parameters have emerged as important markers in risk stratification for TAVR. In particular, recent interest has highlighted the role of the right heart unit and pulmonary vascular indices.<sup>10-12</sup> The presence and severity of TR, pulmonary hypertension, and RV size and function have all been reported to delineate hemodynamic staging of the disease process and improve risk stratification.<sup>12</sup> The presence of mild functional TR in our patient that persisted after TAVR is consistent with findings by Jeong *et al*.<sup>11</sup> That persistent mild TR after surgical aortic valve replacement is not necessarily associated with poor outcome, though the impact of persistent TR after TAVR is less clear. The prevalence of pulmonary hypertension in patients undergoing TAVR varies from 30% to 75%.<sup>11,13</sup> Our patient had severe pulmonary hypertension that resolved with TAVR, but reversibility may be unpredictable,<sup>14</sup> and other hemodynamic markers of pulmonary vascular remodeling (elevated transpulmonary pressure gradients,



**Figure 5** Transthoracic echocardiographic images before TAVR and at 8-week follow-up demonstrate a decrease in aortic gradient (**A,B**) from 91 to 25 mm Hg, a change in LV filling pattern to dominant A (**C,D**) with an increase in lateral tissue Doppler E' (to 7.2 cm/sec) and a reduction in E/E' ratio to 9.7 (**E,F**), suggesting a reduction in LV filling pressures. TR on color Doppler remained mild (**G,H**).

diastolic pressure gradient, and pulmonary vascular resistance) may be useful in assessing potential reversibility of pulmonary hypertension in patients with long-standing severe aortic stenosis and chronic pulmonary venous congestion.<sup>15</sup> Unfortunately, our patient did not have a right-heart catheter study before or after TAVR, and therefore we are unable to report on these measurements in this case. However, our patient did demonstrate RV reverse remodeling and normaliza-

tion of RV dimensions and function after TAVR. The importance of integrating RV size and function in staging of aortic stenosis has been hampered in part by a lack of standardized RV imaging techniques, but this is currently being addressed in an era of increased use of tricuspid annular plane systolic excursion, fractional area change, tissue Doppler, and strain imaging by speckle-tracking in the preassessment stage.

## CONCLUSION

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Undiagnosed critical aortic stenosis may present as painless jaundice secondary to severe biventricular failure. In very high-risk patients, transcatheter aortic valve replacement (with the optimal support of circulatory support if necessary) can reverse the clinical and biochemical changes of congestive hepatopathy and normalize biventricular cardiac function.

## SUPPLEMENTARY DATA

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Supplementary data related to this article can be found at [10.1016/j.case.2018.04.011](https://doi.org/10.1016/j.case.2018.04.011).

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