Chapter 2
New Onset Heart Failure: Which Side Is It, Right or Left?

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Case

A 65-year-old woman presented with no reported past medical history as she had avoided medical care for the past 20 years. She has had mild-to-moderate (1–2+) lower extremity edema up to her knees for at least the past 10 year. During which time, she slept in a recliner, and was able to complete her activities of daily living (ADLs) with intermittent breaks to rest. In the past 6 months, she has needed to rest more frequently while performing her ADLs and usually took 2–3 naps per day. Her abdomen had become firmer than normal,

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L. Tsao, M. E. Afari (eds.), *Clinical Cases in Right Heart Failure*, Clinical Cases in Cardiology,
https://doi.org/10.1007/978-3-030-38662-7_2

with an associated 15 lb weight gain. She was morbidly obese with a BMI of 45 kg/m^2 and stated that she primarily ate frozen or take-out meals on a daily basis. For a number of years, she has complained of palpitations that seemed to gradually worsen. She has become increasingly reliant on her children to assist in household chores due to progressive fatigue. At her daughter's insistence, she finally established care with a primary care provider, who promptly referred her to the cardiology clinic for evaluation and management.

2.1 What Is the Clinical Presentation of Chronic Right Heart Failure?

This patient presented with clear evidence of volume overload and suspected heart failure (HF), but the distinction between right heart failure (RHF) and left heart failure (LHF) based on the history alone is often difficult. Many of the symptoms of RHF are indistinguishable from LHF. The absence of orthopnea, paroxysmal nocturnal dyspnea (PND), and shortness of breath may suggest a right-sided etiology but is not diagnostic. Early signs and symptoms of RHF typically include fatigue and lower extremity edema. The distinction between RHF and LHF is often made after left-sided pathologies are excluded, and the underlying pathology is identified.

In the setting of chronically elevated central venous pressure (CVP), chronic vascular congestion of end organs can lead to progressive dysfunction. As RHF progresses, the forces of ventricular interdependence skew in favor of right ventricle (RV) predominance in both pressure and volume overload, which in later stages may lead to a reduced cardiac output and end organ ischemia [1]. The hepatic, renal, and gastrointestinal systems are primarily affected by these deleterious effects.

In the early stages of hepatic congestion, right upper quadrant discomfort from liver capsule stretching and nausea may be present, which may indolently progress to early satiety and anorexia. It is important to note that these symptoms are indistinguishable from primary hepatic conditions such as

cholestasis with the determination of volume status usually being the delineating factor [2].

Decreased urine output and/or increasing diuretic requirements may be early signs of renal involvement [3]. As renal dysfunction progresses, reports of fatigue, nausea, anorexia, and ultimately confusion may develop. The development of cardio-renal syndrome is associated with a poor prognosis [4].

Increased intra-abdominal pressures due to ascites may develop as systemic congestion overwhelms the capacitance of the splanchnic vasculature, which can lead to renal dysfunction secondary to compression of the renal vasculature and reduced renal perfusion [5]. Effects on gastric and colonic function leading to early satiety, anorexia, and constipation are also seen.

Upon further interrogation, our patient denied any history of orthopnea, PND, or chest pain. She admitted to symptoms of daytime somnolence and a history of morbid obesity since she was a teenager. Her husband and daughter both confirmed the presence of loud night-time snoring and choking spells. She denied any history of tobacco, alcohol, or illicit drug abuse, but admitted to drinking nearly a pot of coffee every day.

2.2 What Is the Next Step in Evaluation?

When a patient presents with signs and/or symptoms of HF, after a comprehensive history, the initial diagnostic approach always begins with a thorough physical exam.

Her vitals were significant for a pulse of 135 beats per minute, blood pressure of 130/80 mmHg, and a respiratory rate of 20 breaths per minute. Using the bell of the stethoscope with the patient sitting in the upright position, her cardiac exam was significant for a holosystolic III/VI murmur best heard at the middle left sternal border. The murmur was louder with inspiration. Her rhythm was tachycardic and irregular. The first heart sound (S1) was not appreciated, the second heart sound (S2) was variable, and a right-sided S3 was present. Her lungs were clear with equal inspiratory and expiratory times. An estimation of her jugular venous pressure (JVP) was at least 15 cm H_2O. No right ventricular heave

was present but the apical impulse of the left ventricle (LV) was displaced laterally. Hepatomegaly, a pulsatile liver, and a slightly tender right upper quadrant were present in the absence of an abdominal fluid wave. Her lower extremity edema was pitting, 3+, and symmetric. Figure 2.1 depicts an example of a patient with RHF.

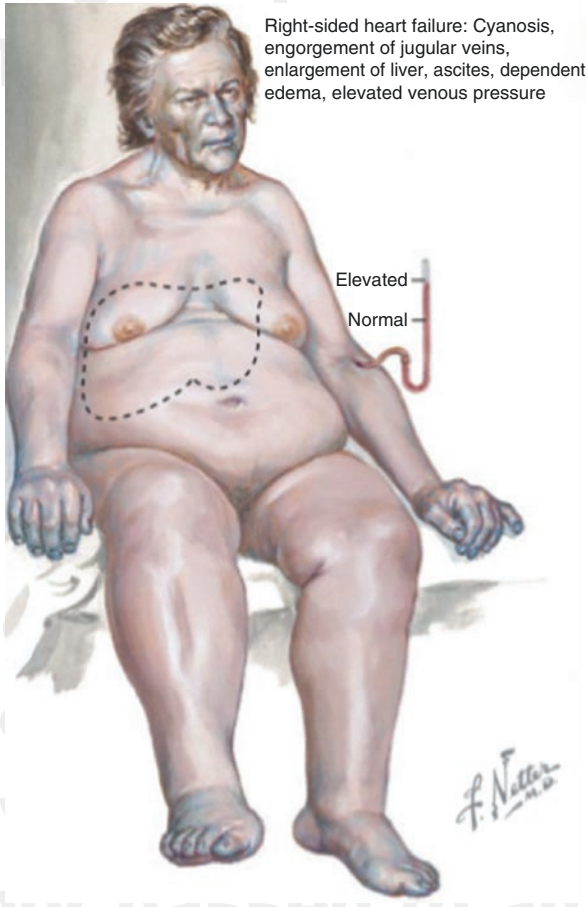

FIGURE 2.1 Schematization of a patient with a classic presentation of right heart failure. Netter illustration used with permission of Elsevier Inc. All rights reserved. www.netterimages.com

2.2.1 Cardiac Findings

Cardiac auscultation, when the time is invested to perform correctly, can identify significant valvular pathologies. It is important to listen directly on the skin with both the bell and the diaphragm as well as to purposefully listen for the presence of each of the possible murmurs. One of the most important auscultatory findings to distinguish left versus right-sided murmurs is how it varies with respiration. Deep inspiration lowers intrathoracic pressure and increases venous return to the right side of the heart. Therefore, right-sided murmurs will typically *increase* in severity with inspiration and *decrease* with exhalation, commonly referred to as Carvallo's sign. This sign was present in our patient and suggestive of tricuspid regurgitation (TR).

Palpation for an RV heave is performed by placing the heel of the hand on the left sternal border and is present when the heel of the hand is lifted off the chest with each systole. This typically represents significant RV hypertrophy, but in rare cases may also represent marked right atrial enlargement.

2.2.2 Pulmonary Findings

Her clear lung sounds may reduce one's suspicion of a left-sided pathology. However, this cannot absolutely distinguish between right and left-sided HF as patients with chronic LHF can also lack pulmonary edema if they are well compensated and euvolemic. Due to compensatory mechanisms including increased lymphatic drainage [6], increased thickness of alveolar basal membrane reducing capillary filtration [7], and enhanced alveolar fluid clearance [8], lung fields could be clear.

2.2.3 Jugular Vein Assessment

Estimation of a patient's CVP is commonly performed via jugular venous pressure (JVP) assessment and is an important tool to assist in quantifying the degree of volume overload. Typically, this evaluation begins with the patient supine and the head of

the bed elevated at a 45° angle. With the head turned towards the left shoulder, the right side of the neck should be closely inspected to observe for the leading edge of the distended jugular vein. A vertical measurement in centimeters is then taken from this point to the Angle of Louis, to which 5 cm is added (an estimation of the depth of the right atrium from the angle of the sternum). Sometimes the leading edge of the distended jugular vein cannot be appreciated. If the right atrial pressure is low then the jugular venous distension is located below the clavicle. Gentle pressure applied to the right upper quadrant/liver may bring the vessel into view, commonly referred to as hepatojugular reflux. If the right atrial pressure is markedly elevated, the patient may be asked to sit upright with the feet dangling off the exam table. This allows the blood to pool in the lower extremities to lower the venous waveforms in the neck below the angle of the jaw for measurement. The right-sided pressures vary based on the respiratory cycle and typically fall with inspiration. Kussmaul's sign is a rise, or failure to fall, of venous pressure with inspiration and represents right-sided volume overload as well as reduced ventricular compliance.

If the CVP cannot be estimated by the jugular vein, then assessment of peripheral venous collapse may be considered. With the patient supine and the head of the bed at a 45° angle, if the veins are distended on the dorsum of the hand, then the arm may be passively elevated until the hand veins are no longer visible. If this transition point occurs when the arm is elevated above the level of the sternal angle, then the CVP is likely elevated. Similarly, the Anthem or Rizkallah sign may be employed during which the patient is in the same position, but instead of the arm being passively raised it is instead placed directly over the sternum. If the hand veins remain distended then an elevated CVP is suspected [9].

2.2.4 *Hepatic Findings*

The majority of patients with RHF develop hepatic congestion with resultant hepatomegaly. The liver may feel firm and is often tender to palpation. Splenomegaly is characteristi-

cally absent. The increase in venous pressure causes perisinusoidal edema and hepatocyte atrophy leading to impaired diffusion of oxygen and nutrients in the liver [10]. The "backward" failure is also responsible for the compression of lymphatics and the impaired capability of lymph drainage may lead to ascites in 25% of the patients [11]. In patients with considerable TR, a prominent systolic pulsation of the liver may be appreciated due to an enlarged right atrial 'v' wave. A presystolic pulsation of the liver, attributable to an enlarged right atrial 'a' wave, can occur in tricuspid stenosis, constrictive pericarditis, restrictive cardiomyopathy involving the RV, and pulmonary hypertension. The slow flow within hepatic sinusoids favours thrombosis within the hepatic venules and portal tracts, promoting fibrosis and ultimately leading to cirrhosis [12].

2.3 What Is the Pathophysiology of Chronic RHF?

The RV is connected to the low impedance, highly distensible pulmonary circulation that allows efficient transfer of blood to maintain the same stroke volume as the LV. Since the RV and LV are connected in series, the cardiac output is essentially the same, but the afterload is significantly different.

The RV adaptation depends on the presence of the pressure or volume overload that it encounters. Because of its greater compliance, the RV can adapt tremendously and increase its contractility by up to fivefold in response to rising afterload [13]. When faced with an even higher load, the RV starts to dilate in order to maintain adequate cardiac output and ventriculo-arterial coupling. Under normal conditions, the RV impact on the LV is minimal but as the RV pressure rises, ventricular interdependence results in movement of the interventricular septum to the left. This causes mechanical inefficiency which impairs LV diastolic filling and stroke volume [13]. Dilatation of the RV results in tricuspid regurgitation, which further increases preload. Finally, as the wall tension continues to rise, molecular changes of RV myocyte

loss and fibrosis occur that will eventually result in right ventricular failure [14].

The most common cause of RHF is LHF secondary to post-capillary pulmonary hypertension and chronically elevated right ventricular afterload. Causes of chronic right ventricular dysfunction can generally be divided into three categories: (1) Increased afterload, (2) Increased preload, and (3) Primary RV cardiomyopathies. The etiologies of RHF are outlined in Table 2.1.

Since the RV is coupled to a high compliance, low impedance pulmonary system, it is better suited to handle volume overload (preload) than pressure overload (afterload). An

Table 2.1 Causes of right heart failure

Increased afterload	Increased preload	Primary cardiomyopathy
• Left heart disease	• Tricuspid regurgitation	• Ischemia/infarct
• Pulmonary hypertension	• Pulmonic regurgitation	• ARVC
• Acute PE	• ASD	• Myocarditis
• Pulmonic stenosis (valvular or subvalvular)	• TGA	• Amyloidosis
• ARDS	• ToF	• Sarcoidosis
• COPD	• Ebstein anomaly	• Dilated CM
• CTEPH	• Anomalous pulmonary venous return	• Hypertrophic CM
• Pulmonary artery stenosis		• Cardiotoxic medications

PE pulmonary embolism, *ARDS* acute respiratory distress syndrome, *COPD* chronic obstructive pulmonary disease, *CTEPH* chronic thromboembolic pulmonary hypertension, *ASD* atrial septal defect, *TGA* transposition of the great arteries, *ToF* Tetralogy of Fallot, *ARVC* arrhythmogenic right ventricular cardiomyopathy, *CM* cardiomyopathy

increase in afterload causes an exaggerated work load on the RV by impeding forward flow and reducing RV stroke volume. Common causes of increased RV afterload include pulmonary hypertension, acute pulmonary embolism, pulmonic stenosis (valvular or sub-valvular), as well as lung pathologies such as acute respiratory distress syndrome, chronic obstructive pulmonary disease, and chronic thromboembolic pulmonary hypertension (CTEPH).

Congenital heart disease such as the presence of an atrial septal defect, transposition of the great arteries, Tetralogy of Fallot as shown in Chap. 4. Ebstein anomaly, anomalous pulmonary venous return as well as pulmonary and tricuspid regurgitation can lead to an increased preload.

Primary RV cardiomyopathies can result in reduced contractility and include viral myocarditis and arrhythmogenic right ventricular cardiomyopathy (ARVC), the latter of which is reviewed in Chap. 5. Impaired contractility of the RV due to an ischemic cardiomyopathy can result in a decrease in LV preload leading to systemic hypotension and a reduction in cardiac output. This can be seen in the post-cardiotomy patient or RV myocardial infarction patient discussed in Chap. 9. Restoration of blood flow is the mainstay of treatment which improves both right ventricular systolic and diastolic function.

2.4 What Is the Recommended Diagnostic Work Up?

2.4.1 Laboratory Tests

There are no laboratory tests that are specific for RHF. On the initial presentation of a patient with previously undiagnosed HF, routine labs are checked including a complete blood count, comprehensive metabolic panel, thyroid stimulating hormone, a B-type natriuretic peptide (BNP) or N-terminal pro-B-type natriuretic peptide (NT-proBNP), as well as an assessment for ischemia through troponin level.

Depending on the clinical history, specialized testing may be considered such as a d-dimer to assess for the presence of a DVT and/or PE. This patient's labs are listed below.

Hemoglobin/ Hematocrit	10.3/33.1	(ref 11.8–15.8 g/dL/35–47%)

Her labs were significant for a mildly reduced hemoglobin and hematocrit, which may be at least in part dilutional from volume overload. Anemia and iron deficiency can also be associated with cardiorenal syndrome [15]. The risk of mortality is also increased in HF associated with anemia [16].

Bilirubin 1.8 (ref 0.0–1.0 mg/dL), AST 30 (ref 0–37 U/L), ALT 35 (ref 0–40 U/L), Alkaline phosphatase 248 (ref 39–117 u/L), γ-glutamyl transpeptidase (GGT) 65 (ref 9–48 U/L)

Jaundice is not commonly reported, with total bilirubin levels rarely exceeding 3 mg/dL (ref 0.0–1.0 mg/dL) [17]. A cholestatic pattern is significantly more prevalent than elevated transaminases, with elevated GGT and alkaline phosphatase most closely correlating with adverse outcomes in RHF [18]. Protein-losing enteropathy is classically associated with patients who have undergone Fontan surgery, but rarely can be seen with pericardial and valvular etiologies such as severe tricuspid regurgitation and can lead to cardiac cachexia [19]. In advanced disease, liver synthetic function may also become impaired as suggested by reduced albumin levels and an elevated international normalized ratio. Jaundice may become evident suggesting cardiac cirrhosis.

Creatinine	1.7	(ref 0.50–1.30 mg/dL)

As previously noted, passive venous congestion and or compression of the renal vasculature from increased abdominal pressures can contribute to abnormal kidney function. Several non-hemodynamic pathways can also exacerbate

cardiac or kidney injury, which include the persistent activation of the renin–angiotensin–aldosterone system and chronic inflammation. This leads to imbalance in the proportion of reactive oxygen species/nitric oxide production, elevated circulation levels of tumor necrosis factor-α (TNFα), interleukin-1 (IL-1), and interleukin-6 (IL-6) [20]. An increase in blood urea nitrogen (BUN) and serum creatinine level can occur, which are independent markers of adverse outcome and results in diuretic resistance [21]. In some cases, diuretics may lead to further worsening of renal function and present a challenge in treatment.

Troponin T	0.08 ng/mL	(ref 0.000–0.030 ng/mL)

If the ECG is not suggestive of an acute coronary syndrome, then once the patient is stabilized an ischemia workup can be pursued. It is common for patients with volume overload to have a mildly elevated troponin when superimposed on reduced renal clearance in the setting of acute and or chronic kidney disease.

NT-proBNP	1845 pg/mL	(ref 0–900 pg/mL)

Elevated levels of BNP and NT-proBNP are supportive, but not necessarily diagnostic of HF. These biomarkers do not distinguish RHF from LHF. BNP has emerged as a useful marker of prognosis in RHF accompanying pulmonary arterial hypertension (PAH) [22–24]. It is important to note that the NT-proBNP, more so than BNP, may be inappropriately elevated due to reduced renal clearance in the setting of significant kidney dysfunction or paradoxically low in obese patients [25].

2.4.2 *Electrocardiography (ECG)*

Her ECG on presentation demonstrated atrial fibrillation at 135 beats per minute with low voltage throughout and a right

bundle branch block (RBBB).The QT interval was normal with no significant ST-T wave abnormalities. Q waves were not present.

ECG is a quick and simple tool to aid in the assessment of RV dysfunction. The RBBB is suggestive of right ventricular strain. Other prominent findings may include right axis deviation, RV hypertrophy, and/or inferior Q waves suggestive of prior RV infarct. Atrial fibrillation is also common in patients with right ventricular dysfunction.

2.4.3 Imaging

Her echocardiogram showed a normal LV ejection fraction (LVEF) of 65% without mitral or aortic valve pathologies, mild LV diastolic function, no LV hypertrophy. The RV was moderately dilated with abnormal systolic function. Moderate-to-severe tricuspid regurgitation was present as well as a flattened interventricular septum during systole and diastole, and a fixed and dilated inferior vena cava (RA pressure of at least 15 mmHg).

Doppler and 2D echocardiography is recommended as the first imaging modality. This can help to assess for left-sided pathologies and determine the size and function of the RV, along with the assessment of right ventricular and pulmonary pressures. Tricuspid annular plane systolic excursion (TAPSE) is an M-mode derived measurement of RV longitudinal motion. A low TAPSE has been shown to predict prognosis related to right ventricular dysfunction, especially in patients with pulmonary hypertension [26]. However, 2D echocardiography is limited in its' ability to comprehensively assess right ventricular dysfunction due to the RV's thin wall, peculiar morphology, and anterior location.

Although not currently indicated in this patient, cardiac magnetic resonance (CMR) imaging provides an advantage over echocardiography due to its superiority in anatomic, volumetric, and quantitative analysis of the RV. It is also more sensitive in the assessment of congenital heart diseases,

pulmonary hypertension, and evaluation of intra-cardiac shunts. CMR is crucial in the diagnosis of ARVC, since there is a lack of established and definite "gold standard" test [27]. Chapter 3 elaborates on the role of diverse imaging tools in RHF.

2.4.4 *Invasive Testing*

A right heart catheterization (Swan-Ganz) may be considered if her creatinine does not improve or actually worsens with diuresis, or as a way to identify the underlying pathology causing RHF. This tool provides a more accurate assessment of right-sided filling pressures as well as determines cardiac output and systemic vascular resistance. PAH can be diagnosed and concurrently assessed for response to treatment, evaluate for intracardiac shunts, and provide an estimation of the left atrial pressure.

Table 2.2 outlines findings that may suggest right ventricular dysfunction and RHF. It is important to note, however, that not all of the listed findings will be present in every patient nor does the presence of one or a few of these findings dictate that right-sided dysfunction is the sole pathology. Rather, these findings may support the diagnosis when combined with the history, physical exam, and multiple data points.

2.5 How Is Chronic RHF Managed?

Generally, the treatment of RHF is focused on treating the underlying pathology. However, the first step is to stabilize symptoms. In the case of our patient, this involves a trial of diuresis and rate control of her atrial fibrillation. The current HF guidelines primarily focus on the management of LV dysfunction. There is a paucity of data demonstrating efficacious therapies directed towards treatment of isolated RV dysfunction. The mainstay of therapy for RV dysfunction is to identify and treat the underlying disorder [3].

TABLE 2.2 Diagnostic findings that support right heart failure

Test	Findings to support right ventricular dysfunction or RHF
Electrocardiogram	• RBBB • RV hypertrophy • Right atrial enlargement • Right axis deviation • S1Q3T3 in acute pulmonary embolus • ST elevation, more elevated in lead III than lead II (acute RV infarct)
Chest X-ray	• RV and/or pulmonary artery (PA) enlargement • Hyperinflated lungs (COPD) • Absence of pulmonary vascular congestion or Kerley "B" lines • Wedge-shaped absence of pulmonary vasculature (pulmonary infarct)
Echocardiography	• Absence of left-sided pathologies • Dilated RV and/or reduced systolic function (low TAPSE, S′, or RV fractional area change) • Elevated RV systolic pressure • RV free wall hypertrophy • Tricuspid stenosis/regurgitation • Pulmonic stenosis/regurgitation • Hepatic vein flow reversal • Ventricular septal interdependence (volume and/or pressure overload) • McConnell's sign or apical "wink" (acute PE) • Atrial septal defect • Ventricular septal defect
Cardiac MRI	• Dilated RV • Reduced RV systolic function/ejection fraction • Increased late gadolinium enhancement (LGE) • RV free wall hypertrophy • Myocardial edema

TABLE 2.2 (continued)

Test	Findings to support right ventricular dysfunction or RHF
Right heart catheterization	• Pulmonary hypertension (pre- and/or post-capillary) • Constriction (prominent 'x' and 'y' descents with a square root sign, elevation and equalization of diastolic pressures) • Elevated RA pressure (equivalent to JVP) • Kussmaul's sign • Ventricular interdependence (requires concurrent LV pressure monitoring) • Shunt run may show a "step up" in oxygenation levels in the right heart chambers, suggestive of a left-to-right shunt
CTPA	• Acute and/or chronic pulmonary emboli • COPD/emphysema
Pulmonary function tests or high resolution CT of the chest	• COPD/emphysema
Labs	• Elevated BNP or NT-proBNP • Abnormal liver function tests • Elevated INR • Reduced albumin • Elevated Creatinine and BUN • Elevated D-dimer

RBBB right bundle branch block, *RV* right ventricle, *COPD* chronic obstructive pulmonary disease, *TAPSE* tricuspid annular plane systolic excursion, *PE* pulmonary embolus, *RA* right atrium, *JVP* jugular venous pressure, *CTPA* computerized tomography pulmonary angiogram, *CT* computerized tomography, *INR* international normalized ratio, *BNP* brain natriuretic peptide, *NT-proBNP* N-terminal pro-B-type natriuretic peptide, *BUN* blood urea nitrogen

2.5.1 *Diuretics*

Loop diuretics such as furosemide, torsemide, and bumetanide are the key to treatment in both acute and chronic RHF. Gastrointestinal absorption of furosemide is reduced in the setting of significant gut edema, often prompting escalation to torsemide or bumetanide due to their increased absorption and better oral bioavailability. Patients who do not respond to escalating doses of oral diuretics may require intravenous diuretics. Diuretic resistance may also result from chronic and or acute renal disease, and low cardiac output resulting in renal arterial hypoperfusion combined with renal venous congestion, and/or intense neurohormonal activation.

Intermittent dosing of thiazide diuretics, such as metolazone or chlorothiazide, may help to "prime" the kidneys in an effort to augment the diuretic response by being administered approximately 30 min prior to the loop diuretic. The use of an aldosterone antagonist in conjunction with loop or thiazide diuretics can also be effective. There is a growing body of evidence regarding the role of aldosterone antagonism in patients with RV dysfunction [28, 29]. Renal replacement therapy with either continuous veno-venous hemofiltration (CVVH) or hemodialysis can be the last resort in patients who are resistant to escalating doses of diuretics.

2.5.2 *Digoxin*

Digoxin has been shown to contribute to improvements in acute hemodynamic abnormalities in RHF but no data exists on the long-term benefits [30].

2.5.3 *Invasive Therapies*

In the presence of ischemia, coronary revascularization should always be considered. As noted above, evaluation with

a PA catheter can help to diagnose an underlying disorder (such as PAH or a shunt), as well as assess for a response to treatment.

2.5.4 *Pulmonary Vasodilators*

Pulmonary vasodilators have been shown to improve WHO functional status and mortality in patients with group I PAH. However there have been no proven therapies to reduce mortality in patients with group II, III, IV, or V pulmonary hypertension. This is discussed in detail in Chap. 7.

2.5.5 *Surgery*

In the setting of symptomatic RHF, tricuspid valve surgery may be considered for primary tricuspid regurgitation if unresponsive to medical therapy, or in the setting of at least moderate right ventricular dilatation and/or systolic dysfunction [31].

Case Conclusion: Her symptoms and physical exam responded well to aggressive oral diuresis. Her labs were closely monitored, demonstrating improvement in her creatinine and LFT abnormalities. She was then started on a low-dose beta blocker for rate control. Her cardiac stress test did not show evidence of ischemia. A sleep study was performed that revealed an Apnea–Hypopnea Index (AHI) of 45, which is consistent with severe obstructive sleep apnea, and she was promptly started on continuous positive airway pressure (CPAP) therapy. The patient was provided extensive education and reinforcement on HF management throughout this process. After treatment, her follow-up echocardiogram showed normalized RV systolic function but remained moderately dilated. Her TR improved to moderate severity. The cause of her sleep apnea was likely related to her morbid obesity, and she was subsequently referred to the local weight loss center for further evaluation and management, including consideration for bariatric surgery.

Clinical Pearls

- Right heart failure is most commonly caused by left heart disease.
- The right ventricle can adapt to volume overload better than pressure overload.
- Assessment of right ventricular failure requires a careful history and physical examination as well as a high index of suspicion.
- Achieving euvolemia and treating the underlying disorder are key to the management of RHF.
- The presence of right ventricular dysfunction, independent of the etiology, is associated with an increased mortality.

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