Transcript Details

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The Pathophysiology and Pathoetiology of Heart Failure

Announcer:

You're listening to ReachMD. This medical industry feature is titled "The Pathophysiology and Pathoetiology of Heart Failure." Here is your guest, Dr. Stephen Greene.

Dr. Greene:

Welcome everyone, and thank you for listening today to the second podcast in our heart failure series. My name is Dr. Stephen Greene, and I am an Advanced Heart Failure Specialist at Duke Heart Transplant Clinic and a Cardiologist at Duke Cardiology Clinic in Durham, North Carolina.

Last time, my colleague Dr. Nancy Albert discussed the epidemiology and disease burden of heart failure. Today, we will compare the pathophysiology and pathoetiology of heart failure with *reduced* ejection fraction to that of heart failure with *preserved* ejection fraction.

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One of the most important things we know about heart failure is that is doesn't affect everyone equally. There's quite a difference between heart failure with reduced ejection fraction and heart failure with preserved ejection fraction, which we'll discuss during our time together today.

By definition, heart failure with reduced ejection fraction, which accounts for about 50% of all heart failure cases, is caused by the loss of systolic cardiac function. Here, the heart muscle is weakened, resulting in a left ventricle that—while filling normally—is compromised in its ability to pump blood out. Interestingly, the weakened heart muscle observed in patients with heart failure with reduced ejection fraction is the result of myocardial injury, which causes left ventricular remodeling, dilatation, and impaired contractility.

Conversely, heart failure with *preserved* ejection fraction is the result of endothelial dysfunction, arterial stiffening, and left ventricular abnormalities. Here, the heart muscle is stiff and thickened, resulting in less blood than normal filling the left ventricle, which means that less blood is available to be pumped out.

Let's go into a little more detail about the complex pathophysiology surrounding heart failure and how it differs when ejection fraction is reduced versus when it is preserved.

As I mentioned previously, heart failure with reduced ejection fraction results from myocardial injury, which can be caused by a host of cellular consequences, including oxidative stress in cardiomyocytes, an increase in cardiomyocyte necrosis, apoptosis and/or autophagy, or a decrease in myocardial contractility.

The pathophysiology of heart failure with preserved ejection fraction is less well-understood. Heart failure with preserved ejection fraction is pathologically considered a systemic proinflammatory disease that results from microvascular endothelial inflammation; an increase in interleukin-6, tumor necrosis factor-alpha, and/or soluble suppression of tumorigenesis-2, better known as sST2. It can also result from an increase in cardiomyocyte stiffness and interstitial fibrosis, or an increase in passive stiffness and fibrosis.

Although they are functionally and pathologically distinct diseases, comorbidities, such as diabetes, hypertension, chronic kidney disease, obesity, chronic obstructive pulmonary disease, and aging are shared risk factors for both heart failure with reduced ejection fraction and heart failure with preserved ejection fraction. Therefore, it is important to understand the underlying comorbidities when diagnosing an individual with heart failure.

Thank you for joining me today.

As we have discussed, the pathophysiology and pathoetiology of heart failure with reduced ejection fraction and heart failure with preserved ejection fraction is unique and important to understand in order to fully appreciate the debilitating effects of the disease.

Announcer:

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