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Case Study: CPET

Announcer:
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Dr. Brownstein:
So let's get started with our case. So we have a 35-year-old female with a history of lupus and PAH who was referred for a level 3 CPET. Her baseline right heart cath 6 months prior to presentation for the CPET. This was her index right heart cath not on any therapy demonstrated an RA pressure of 6, a mean PA pressure of 51, thermodilution cardiac output of 4.1, and giving her a PVR of 9.5 Wood units and wedge pressure 12 and her PA saturation was 70%. Her echocardiogram, as you can see in the middle, demonstrated a normal RV size and function, and estimated PASP of 44, and her baseline pulmonary function tests on the side show that moderately reduced diffusion capacity.

So when she comes in for the CPET, she obtained a resting right heart cath on dual oral therapy with tadalafil and macitentan, showed an RA pressure of 3, mean PA pressure of 31 with a wedge of 7, thermodilution output of 3.9 giving her a PVR of 6. Her VO2 at rest was 290 milliliters per minute, and her direct Fick cardiac output was 4.38 with a direct Fick index of 3 and a direct Fick PVR of 5.5 Wood units. H er PA sat was 69.8%.

So then, the patient underwent incremental cycle ergometry consisting of a 3-minute period of unloaded exercise, followed by a 15 watt per minute ramp to a maximum of 90 watts. And the test was stopped due to dyspnea and achievement of high PA pressures. Her peak RER, or respiratory exchange ratio, of 1.31, and a post-exercise venous lactate of 72 milligrams per deciliter indicate that this was a maximal effort study. The patient only achieved a heart rate, a peak heart rate of 83% predicted, which is mildly low. You can see that her peak VO2 was around 62% predicted, which indicates moderately reduced aerobic capacity. Her VO2 at her anaerobic threshold was 42%, which is low normal. The peak O2 pulse and peak cardiac output are both low, in the 70s. And her aerobic efficiency is abnormal as evidenced by a low VO2 to work rate relationship. Her VE to VCO2 is elevated at 35, indicating abnormal ventilatory efficiency and are her end-tidal CO2 is low at rest and does not increase appropriately with exercise.

This table demonstrates her cardiac output, heart rate stroke volume, and pulmonary pressures throughout the duration of exercise. The RV to pulmonary vascular response to exercise is abnormal, given her PA pressure to cardiac output slope of 12, and her transpulmonary gradient to cardiac output slope of 8.6. As you can see, notably, her stroke volume actually decreases as she achieves peak exercise, PA pressure achieves a maximum of 63 and her PVR remains above 4 Wood units, and peak was around 5.6; whereas, normally with exercise your pulmonary vascular resistance should fall.

This is the nine-plot as we kind of learned about earlier showing with number one, a low peak VO2 and low VO2 to work rate slope, number two with an early plateau and low peak O2 pulse, number three with a low normal anaerobic threshold of 41% predicted, a high VE to VCO2 nadir in number four, and a high VE to VOC2 slope in number five. Number six shows a low end-tidal CO2 that does not increase with exertion, and no pulmonary mechanical limitation in number seven.

And then the simplified four-plot version that we learned about just a little while ago shows the low peak VO2 and a low VO2 to work rate slope in number one – sorry, on the top left, a low normal anaerobic threshold of 41% predicted on the top right, and then a high VE to VCO2 slope in the bottom left.

So in the summary, this level 3 CPET demonstrates moderately reduced exercise capacity. The overall pattern is consistent with precapillary PH, given the high PA pressure, the high peer pressure to cardiac output slope, transpulmonary gradient greater than the wedge pressure, a high PVR, a high VE to VCO2 slope, and a low end-tidal CO2 with abnormal exercise augmentation. She was then started on selexipag with improvement in her functional status.

Announcer:
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