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Management of Pregnancy Related Pulmonary Arterial Hypertension

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Introduction

The presence of pulmonary hypertension (PH) (mean pulmonary artery pressure greater than 20 mmHg), a pulmonary arterial wedge pressure of 15 mmHg, and pulmonary vascular resistance (PVR) of 3 mmHg/L/min defines pulmonary arterial hypertension (PAH). PAH causes significant morbidity and mortality during pregnancy, with mortality rates as high as 30-50% reported. The introduction of new PAH medications in the field has improved prognosis in recent years, but it remains one of the most dangerous medical conditions for both mother and baby; in fact, pregnancy is contraindicated in patients with PAH, according to guidelines.

The World Health Organization (WHO) classifies PH as a class IV heart disease that should be avoided during pregnancy. Miscarriage (5.6%), foetal loss (2%), preterm delivery (21.7%), foetal growth restriction (19%), and neonatal mortality (0.7%) are among the foetal complications [1-3]. Pregnancy does occur in PAH despite these risks. As with the management of other cardiovascular disorders in pregnancy, the goal of PH management during pregnancy should be to optimise the central pathophysiology of the condition and provide the greatest amount of achievable physiologic reserve in anticipation of the physiologic stresses inherent in pregnancy, labour, and delivery. The goal of this case series is to demonstrate our best practices for managing PAH patients during pregnancy.

Description

During pregnancy, normal physiological changes include an increase in blood volume and cardiac output. The systemic and pulmonary vascular resistances are both decreasing. An increase in heart rate and stroke volume both contribute to an increase in cardiac output. Blood volume has increased by 40% over baseline by the third trimester. There is also a relative hypercoagulable state. Increased right heart afterload limits the ability of intravascular volume expansion and falling systemic vascular resistance to recruit cardiac stroke volume in PAH patients, while increasing the propensity for rising right sided cardiac filling pressures. Functional class and 6MWD may be limited as pregnancy progresses, independent of PAH or RV function, limiting the interpretability of these parameters. As a result, BNP or N-terminal-pro-BNP is important biomarkers that objectively represent cardiac strain and heart failure and have been linked to risk and prognosis in PAH.

Without treatment, right heart dilation occurs, which often leads to an increase in TR, leftward interatrial and interventricular septal displacement, and reciprocal reductions in left atrial and left ventricular cavity size. As a result, cardiac stroke volume, cardiac output, central venous pressure, and hepatic and renal venous congestion may decrease. During labour and delivery, cardiac output increases due to auto-transfusion, increased blood volume from uterine contraction, and increased sympathetic nervous system activation. Bleeding can cause relatively rapid decreases in cardiac preload [4,5]. Furthermore, there may be associated acidosis and hypercarbia, resulting in an increase in PVR.

These findings, taken together, point to a high risk of morbidity and mortality for both the mother and the foetus during pregnancy. When women of childbearing age are diagnosed with PAH, the emphasis is on contraception and pregnancy prevention. The European Registry on Pregnancy and Cardiac Disease has identified PH as a predictor of pregnancy heart failure and has reported maternal mortality rates of up to 43% in idiopathic PAH. Because of these risks, pregnant patients with PAH are frequently advised to terminate their pregnancy. Pregnancy does occur in PAH patients, however, and termination may not be an option due to maternal preference or gestational age at presentation.

Conclusion

The primary goal of PAH management is to reduce the PVR sufficiently to restore normal or near-normal right ventricular size and function. Right heart reverse remodelling and functional recovery occurred as a sigmoid function of PVR reduction, with a PVR reduction of 60% or more from baseline resulting in dramatic reductions in RV size and normalisation of RV systolic function. Greater PVR reduction and improvements in RV size and function, in turn, are associated with significantly improved WHO functional class, submaximal exercise capacity, and attaining low-risk clinical status.

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Conflict of Interest

There are no conflicts of interest by author.

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