

# Treatment of Pulmonary Arterial Hypertension Associated with Pregnancy

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## Introduction

Pulmonary arterial hypertension is indicated by 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 (PAH). Pregnancy-related morbidity and mortality from PAH are considerable, with mortality rates as high as 30 to 50 percent recorded. Although the development of new PAH drugs has improved prognosis recently, pregnancy is still not advised in people with PAH, according to standards, making it one of the most risky medical illnesses for both mother and child.

PH is a class IV heart ailment, according to the World Health Organization (WHO), and should be avoided when pregnant. Among the foetal problems include miscarriage (5.6%), foetal loss (2%), preterm delivery (21.7%), foetal growth restriction (19%), and neonatal mortality (0.7%) [1-3]. In PAH, pregnancies do happen despite these dangers. The goal of PH management during pregnancy should be to optimise the central pathophysiology of the condition and provide the maximum amount of achievable physiologic reserve in anticipation of the physiologic stresses involved in pregnancy, labour, and delivery, just like with the management of other cardiovascular disorders in pregnancy. This case study series aims to illustrate our recommended methods for caring for pregnant PAH patients.

## Description

Normal physiological changes that occur during pregnancy include an increase in blood volume and cardiac output. Both the pulmonary and systemic vascular resistances are lowering. The rise in cardiac output is influenced by both an increase in heart rate and a rise in stroke volume. By the third trimester, blood volume has grown by 40% over the initial value. A relative hypercoagulable condition is also present. Increased right ventricular afterload makes it more likely for right sided cardiac filling pressures to rise while limiting the potential of intravascular volume expansion and declining systemic vascular resistance to recruit cardiac stroke volume in PAH patients. The interpretability of functional class and 6MWD may be constrained as pregnancy continues, independent of PAH or RV function. BNP or N-terminal-pro-BNP, which have been connected to risk and prognosis in PAH, are crucial biomarkers that accurately depict cardiac strain and heart failure.

Without treatment, right heart dilatation takes place, which frequently results in a rise in TR, displacement of the interatrial and interventricular septa to the left, and reciprocal reductions in the size of the left atrial and left

ventricular cavities. Hepatic and renal venous congestion may worsen as a result, and cardiac output, central venous pressure, and stroke volume may all decrease. Due to auto-transfusion, increased blood volume from uterine contraction, and enhanced sympathetic nervous system activation, cardiac output increases during labour and delivery. Cardiac preload can drop rather quickly as a result of bleeding [4,5]. Additionally, there could be related acidosis and hypercarbia, which would raise PVR.

Together, these results indicate a substantial risk of morbidity and mortality during pregnancy for both the mother and the foetus. The focus is on contraception and pregnancy prevention when PAH is discovered in women of reproductive age. According to the European Registry on Pregnant and Cardiac Disease, maternal mortality rates in idiopathic PAH can reach 43%. PH has been established as a predictor of pregnancy heart failure. Patients with PAH who are pregnant are frequently recommended to abort their pregnancy because to these concerns. But because of maternal preferences or gestational age at presentation, it may not be possible to terminate a pregnancy in PAH patients.

## Conclusion

The main objective of managing PAH is to bring back normal or nearly normal right ventricular size and function by reducing the PVR substantially. A PVR decrease of 60% or more from baseline resulted in substantial reductions in RV size and normalisation of RV systolic function. Right heart reverse remodelling and functional recovery happened as a sigmoid function of PVR reduction. Enhanced WHO functional class, submaximal exercise capacity, and achieving low-risk clinical status are all considerably improved by greater PVR reduction and improvements in RV size and function.

## Acknowledgement

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## Conflict of Interest

There are no conflicts of interest by author.

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