Severe Early-Onset Pre-Eclampsia Revealing Primary Hyperaldosteronism: Contrasting Outcomes Across Two Pregnancies

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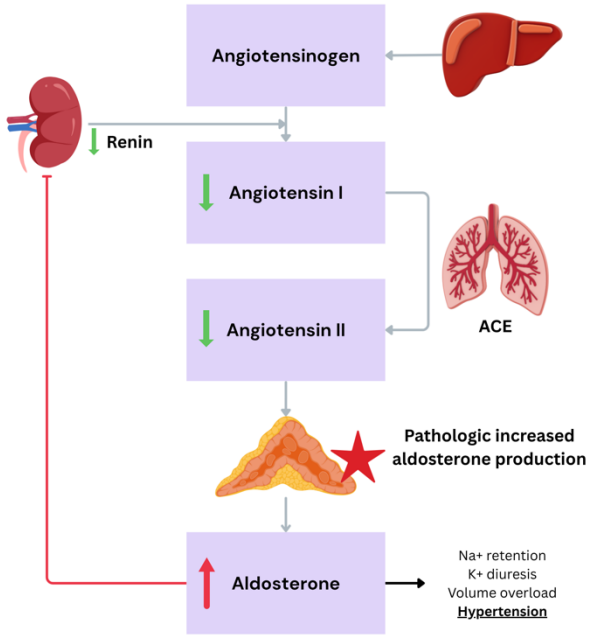
Background

Severe early-onset pre-eclampsia with fetal growth restriction is associated with significant maternal and perinatal morbidity. When hypertension develops early in pregnancy or is resistant to treatment, secondary causes should be considered. Primary hyperaldosteronism is an uncommon but important cause of chronic hypertension that may be under-recognised in pregnancy.



Pathophysiology of Hyperaldosteronism[1]

Pregnancy 1

A woman with chronic hypertension diagnosed, a previously elevated aldosterone:renin ratio and a family history of primary hyperaldosteronism presented in her first pregnancy requiring early escalation of antihypertensive therapy. At 20 weeks gestation, she developed severe early-onset pre-eclampsia with fetal growth restriction, necessitating transfer to a tertiary centre. She subsequently developed severe hypertension with acute pulmonary oedema requiring ICU admission, non-invasive ventilation, frusemide, intravenous antihypertensives and magnesium sulphate. Medical termination of pregnancy was performed at 20+1 weeks for maternal indications. Postpartum investigations and multidisciplinary review confirmed primary hyperaldosteronism.

Pregnancy 2

Following the diagnosis of primary hyperaldosteronism, the patient underwent preconception counselling and multidisciplinary management through a tertiary high-risk obstetric clinic in collaboration with her regional team. Aspirin 150 mg nightly and calcium 1200 mg daily were commenced preconception, alongside labetalol 200 mg three times daily and nifedipine XR 30 mg twice daily. Serial fetal growth assessments, demonstrated normal fetal growth throughout pregnancy. She developed late-onset pre-eclampsia without severe features at 36 weeks. Following admission, antihypertensive therapy was escalated to labetalol 400mg three times daily, methyldopa 500mg three times daily and nifedipine XR 60mg twice daily. She underwent emergency caesarean section at 37+3 weeks following unsuccessful induction of labour, delivering a healthy 2.3kg infant. Although postpartum hypertension required intravenous labetalol, magnesium sulphate and ongoing antihypertensive titration, she avoided ICU admission and severe maternal morbidity, representing a marked improvement compared with her first pregnancy.

Discussion

This case highlights the importance of investigating secondary causes of hypertension in women presenting with severe, early-onset hypertensive disease, particularly when hypertension predates pregnancy or is resistant to multiple antihypertensive agents. Although an elevated aldosterone:renin ratio had been identified several years before conception, definitive investigation occurred only after the index pregnancy. Establishing the diagnosis of primary hyperaldosteronism enabled preconception optimisation, targeted antihypertensive therapy, multidisciplinary care and intensive maternal and fetal surveillance, resulting in progression from pregnancy termination at 20 weeks to delivery of a healthy term infant.

Conclusion

- Severe early-onset pre-eclampsia should prompt investigation for secondary causes of hypertension.
- Primary hyperaldosteronism should be considered in women with resistant chronic hypertension or suggestive family history.
- Early diagnosis, preconception optimisation and multidisciplinary care can substantially improve maternal and neonatal outcomes.

[1]Reincke M, et. al. *Diagnosis and Treatment of Primary Aldosteronism.* The Lancet Diabetes & Endocrinology. 2021; 9(12), 876–892.

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