Labetalol-induced hyperkalaemia in pregnancy: A case report

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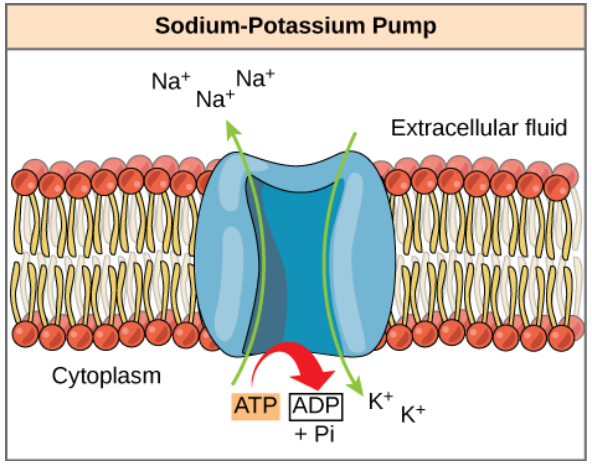
Case:

39 year old female admitted to hospital with vasa praevia for a planned elective Caesarean section. She developed late onset gestational hypertension and was commenced on regular labetalol 200 mg BD. On day 3 of medication administration, patient was noted to be hyperkaelamic.

Patient was noted to be hyperkaelaemia to 5.9. An insulin-glucose infusion infusion was commenced and potassium improved to 4.0. Hyperkalaemia reoccurred despite low potassium diet. After review from the renal medicine team, labetalol was ceased. Nifedipine was commenced in its place to treat hypertension. No further episodes of hyperkaelamia occurred.



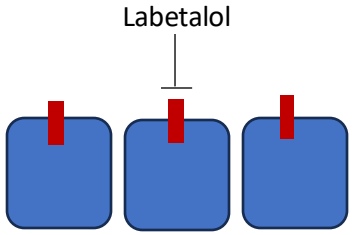
Relevence

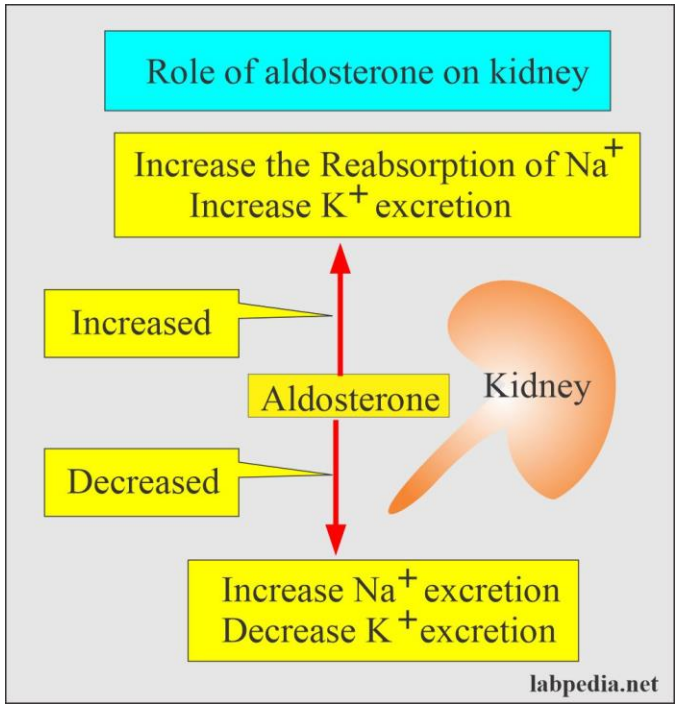
Labetalol is one of the first line therapies used for hypertension management in antenatal patients. It used both in pre-eclampsia and in pregnancy-induced hypertension. Due to its fast acting nature, IV and oral labetalol are used in acute settings such as in hypertensive crisis. As hypertension itself can cause renal impairment, often reflected in a rising creatinine and later hyperkaelamia, it is important to identify that isolated hyperkaelmia can be caused by the therapy being used to treat hypertensive crisis, as opposed to a result of the hypertension itself.

Mechanisms of Beta-Blocker-Induced Hyperkalemia:

1. Reduced Cellular Uptake of Potassium:
- Normally, beta$_2$-adrenergic receptor stimulation leads to increased cellular uptake of potassium through the Na-K-ATPase pump.
- Non-selective beta-blockers competitively inhibit the beta$_2$ receptor, decreasing Na-K-ATPase function and thus reducing potassium uptake by cells.

2. Suppressed Renin Release and Aldosterone Synthesis:
- Beta-blockers can suppress catecholamine-stimulated renin release, which normally leads to aldosterone synthesis.
- Reduced aldosterone synthesis can contribute to hyperkalemia by impairing renal potassium excretion.





Juxtaglomerular cells in the kidney harbour B β1 adrenergic receptors