Treatment for Gitelman and Bartter Patients

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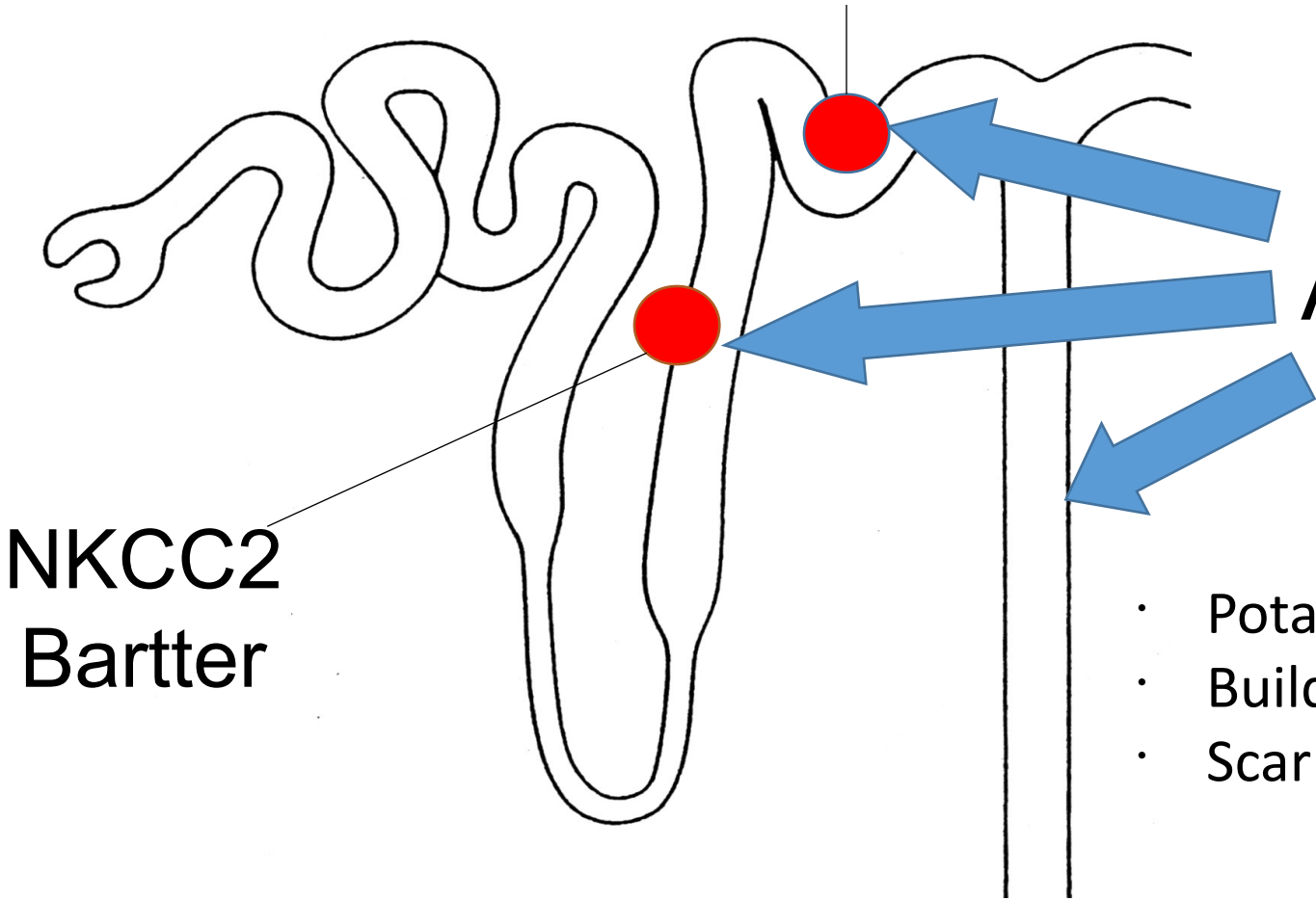
NCC
Gitelman
(Bartter 3)

- Low BP
- Calcium loss in urine (Bartter)
- Calcium build up (Gitelman)
- Magnesium loss in urine (Gitelman)



ALDOSTERONE

NKCC2
Bartter

- Potassium loss in urine
- Build up of alkali
- Scarring in kidney and heart

Aims of treatment

- Make you feel better
- Reduce electrolyte problems
- Stop long term complications

Salt

- The ideal solution
- However, salt loss is vast
- Replacement via diet alone is impossible (induces vomiting)
- Half daily requirement via slow sodium: 10 tablets x3/day

Potassium

Formulations are KCl, $KHCO_3$ or K Citrate

- Slow K
- KCl
- Sando K (KCl and $KHCO_3$)
- Potassium citrate (UroCitK)

May cause gastritis.

Magnesium

Different formulations, lactate, oxide, glycerophosphate

- Magnesium Lactate
- Magnesium Glycerophosphate

All may cause diarrhoea

Drug treatments

Non steroidal anti-inflammatory drugs
Bartter may increased PGE and thus Aldosterone
Blockade with NSAIDs may be very effective, especially in infants

- Indomethacin

Gastritis/ulcers
Decreased renal function

ACE inhibitors

Commonly used blood pressure medicines

- Ramipril
- Lisinopril

Cough

Low blood pressure

Aldosterone blockers

- Spironolactone
- Eplenerone

Breast growth in men
Low BP

- Amiloride

Low BP

Future Directions

Primary problem is salt reabsorption
K and Mg supplementation causes further urinary losses
Treating secondary aldosterone increase causes lower BP and/or kidney toxicity.

Can we increase the salt transporters in the kidney that are the problem?

- 4 Phenylbutyrate; works in cells in the lab
- Tacrolimus increases NCC in the kidney in kidney transplant patients