Bartter syndrome

THICK ASC. LOOP CELL



(TAL)



3NA-



BLOOD



- autosomal recessive disease due to a defect on any of the pumps and channel on the pic, looking "like being on furosemide all the time"
- Presentation: metabolic alkalosis with hypoK-emia
- Diagnostic of exclusion based on labs; genetic testing rarely done (definitive diagnostic).
- Difference w/ surreptitious vomiting by high urinary CI; difference w/surreptitios diuretic abuse by higher level of urinary CI than diuretics, also by a urine assay for diuretics
- It's a secondary hyperaldosteronism. Difference w/ primary hyperaldosteronism by low/ normal serum Na -> normal blood pressure and high plasma renin (due to volume loss).
- Treatment: NSAIDs (renal prostaglandins are produced in Bartter sd) and K sparing diuretic (spironolactone, amiloride)

LUMEN

CA, MG

Labs analysis

	Urinary CI	Plasma renin	Plasma ALDO	Serum K	Serum HCO3	Serum Na
Diuretic use	↑ > 20					/ normal
Vomiting	↓ < 10					
Bartter/ Gitelman	<u> </u>				1	/ normal
Primary H- ALDO	↑↑ > 40	1			1	
Renin secr. tumor	> 40					
Factitious diarrhea	***					/ normal

*** in diarrhea, Urinary CI can varies, being increased in case of metabolic acidosis

Bartter vs Gitelman

	Bartter sd. (like being on LOOP diuretic all the time)	Gitelman sd. (like being on THIAZIDES all the time)			
genetic	aut recessive	aut recessive			
sign	POLYURIA, POLYHYDRAMNIOS	POLYURIA			
LABS	h-K-emia metab.alkalosis	h-K-emia met.alkalosis			
Urinary Ca or urine Ca/creatinine	Ca-URIA normal->high	Ca-uria low			
Pathophysiology	NaCl& water is lost in urine by defects on Na,K, 2Cl cotransporter or other pumps on TALH-> activates Ren,Ang,ALDO system	NaCl and water is lost in urine by defect on Na,Cl pump on DT -> activates Ren,Ang,ALDO system			
Tx	NSAIDs and K sp.diuretics	K sp diuretics, ACEI			

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