Thiazide Diuretics

Thiazide diuretics are often one of the first line treatments for hypertension in the United States. Thiazides are sulfa drugs that can cause an allergic reaction in patients with a sulfa allergy. These drugs inhibit NaCl reabsorption by blocking the Na/Cl cotransporter in the early distal tubule. Increased sodium excretion can lead to hyponatremia and increased delivery of sodium to the collecting ducts can lead to hypokalemic metabolic alkalosis. Other effects include hyperglycemia and hyperlipidemia in patients with long time use. Hyperuricemia is also a relatively common finding which can cause gout. An important distinction between thiazide diuretics and loop diuretics is that thiazide diuretics can lead to hypercalcemia due to decreased urinary calcium excretion while loop diuretics "lose" calcium and can lead to hypocalcemia.

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Early Distal Tubule
Disco Tube
The distal convoluted tubule is a portion of the kidney nephron between the loop of Henle and the collecting duct system. Thiazide diuretics inhibit Na/Cl reabsorption from the distal convoluted tubule by blocking the thiazide-sensitive Na/Cl cotransporter.

Inhibits Na/Cl Reabsorption
Tied-up Salt-shaker and Chlorine-dispenser
Thiazide diuretics inhibit reabsorption of sodium and chloride ions from the distal convoluted tubules in the kidneys by blocking the Na/Cl cotransporter.

Sulfa Drug
Sulfur-match
Thiazide diuretics including hydrochlorothiazide contain a sulfonamide chemical moiety and can cause an allergic reaction in patients with a sulfa allergy.

Hyperglycemia
Hiker-glue
Thiazide use is associated with hyperglycemia. One possible mechanism of action is that thiazides are thought to decrease insulin release. Because insulin plays a role in the uptake of glucose from the blood into the cells, decrease in insulin release can cause hyperglycemia.

Hyperlipidemia
Hiker-lips
Thiazide use is associated with hyperlipidemia. One possible mechanism of action is that thiazides are thought to decrease insulin release. Because insulin plays a role in the storage of fats in adipose tissue, decreased insulin release leads to hyperlipidemia.

Hypercalcemia
Hiker-calcified-cow
The mechanism of hypercalcemia during thiazide use is multifactorial. Distal tubule cells have a Na/Cl cotransporter on the luminal side and a Na-Ca exchanger on the basolateral side. When thiazides inhibit Na-Cl transport, the Na concentration in the cell drops. This causes increased activity of the basolateral Na-Ca exchanger which brings Na from the blood into the cell, while extruding Ca into the blood, leading to hypercalcemia. Thiazides also induce a state of hypovolemia which increases Na and water absorption in the proximal tubule, which passively increases Ca reabsorption as well.

Hyperuricemia
Hiker-unicorn
Hyperuricemia is a relatively common finding in patients treated with a loop or thiazide diuretic. These diuretics reduce urate excretion by both directly and indirectly increasing urate reabsorption and decreasing urate secretion. Hyperuricemia may lead to gout.

Hyponatremia
Hippo-salt-shaker
Because thiazide diuretics block reabsorption of sodium in the distal convoluted tubule, thiazide use can lead to hyponatremia.

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Hypokalemic Metabolic Alkalosis

Hippo-banana Metal-ball Elk-loser

Increased delivery of sodium to the collecting ducts causes increased cellular uptake of Na from the lumen by apical Epithelial Na Channels (ENaCs), which causes the basolateral Na/K exchanger to more actively exchange Na for K, resulting in K loss. The increased delivery of K to the collecting ducts also facilitates the exchange of K for H by the H/K exchanger on the intercalated alpha cells resulting in loss of H causing a metabolic alkalosis. Further loss of potassium is also stimulated by activation of renin-angiotensin-aldosterone system.

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