# HYPERTENSION AND HYPOKALEMIA IN A 56 YEAR-OLD MALE

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**OBJETIVE:** Describe unusual cause of hypertension.

MATERIAL Y METHODS: Review of a clinical record

## DESCRIPTION

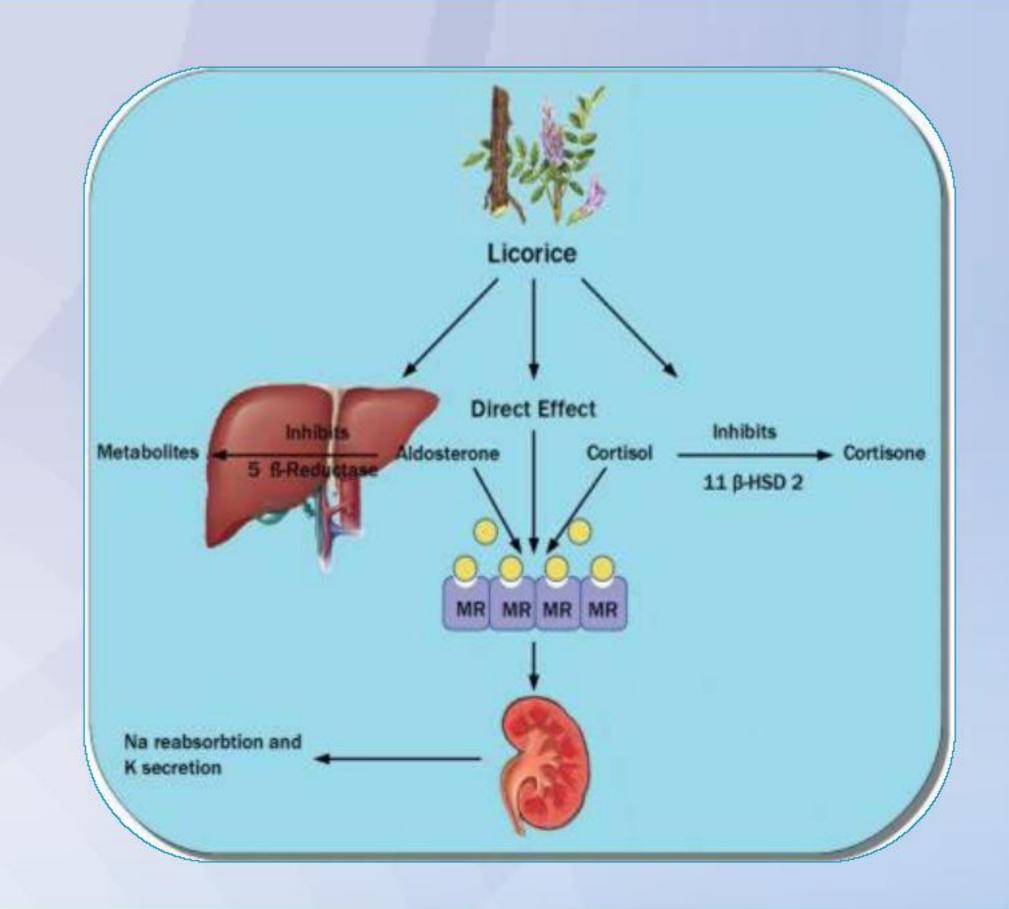
A 56 year-old Swedish male was diagnosed with hypertension, initially attributed to continued use of NSAIDs. These were withdrawn and treatment with 80 mg valsartan was started, but BP remained uncontrolled. A fixed combination (valsartan/hydrochlorothiazide, 80/12.5 mg) was introduced, but blood pressure remained high and hypokalemia (K+ 3.3 mEq/L) developed. The patient was referred to our Endocrinology Clinic for study. Treatment was switched to manidipine 10 mg/12h.

## PHYSICAL EXAMINATION AND COMPLEMENTARY TESTS

Weight 78 kg, height 176 cm, waist 98 cm, BMI 25 kg/m², BP 155/88 mmHg, HR 76 lpm, no additional findings.

Lab tests: CBC, Glucose, Cr, lipid profile, liver enzymes, Na<sup>+</sup>: Normal. K<sup>+</sup> 3.6 mEq/L, aldosterone 12 ng/mL, plasma renin activity (PRA) <0.2 ng/mL/ h.

ECG, chest radiograph, abdominal CT: normal.



## DIAGNOSIS

## Hypertension and hypokalemia probably secondary to pseudohyperaldosteronism

## DIFFERENTIAL DIAGNOSIS

- Primary or secondary hyperaldosteronism (discarded by low aldosterone).
- Liddle syndrome (unlikely because of its rarity, age at diagnosis and no family history)
- Producing tumors or 11-DOCA DOCA (rare, improbable normal CT)
- Congenital enzyme deficiency of 11-β-OHS-DH type 2, 11-α-OHase or aldosterone synthase (improbable age of debut)
- Consumption of inhibitors of 11-β-OHS-DH type 2 as licorice (for its acid content 18-βglycyrrhetinic), carbenoxolone, etc.



## **EVOLUTION**

When directly asked the patient revealed daily intake of 50-75 grams of salmiak (licorice with salt and ammonium chloride). One month after stopping licorice intake and withdrawing manidipine, blood pressure was controlled (136/78 mmHg); K+ (4.8 mEq / L), aldosterone (128 ng/mL) and PRA (0.9 ng/mL/h) were normal. Currently the patient consumes 18-β-glycyrrhetinic acid-free salmiak and his BP and lab tests remain normal.

## CONCLUSION

The patient presented with hypertension, hypokalemia, suppressed PRA and low aldosterone, leading to the diagnosis of pseudohyperaldosteronism. The finding of the habitual high intake of licorice was the key to diagnosis and treatment, since its withdrawal led to the normalization of BP and lab tests. The usual intake of >2 mg/kg/ day of 18-β-glycyrrhetinic acid is a cause of pseudohyperaldosteronism, which can be diagnostic challenge. a

Sontia B, Mooney J, Gaudet L, Touyz RM. Pseudohyperaldosteronism, liquorice, and hypertension. J Clin Hypertens 2008; 10:153-7.







