

- Abdominal imaging.

TREATMENT

- Surgical adrenalectomy
- Spironolactone

DISEASES MIMICKING HYPERALDOSTERONISM

1, LIDDLE SYNDROME

- Decreased degradation of sodium channels** in collecting tubules due to genetic mutation; **autosomal dominant**
- Presents as child with HTN, hypokalemia, and metabolic alkalosis, but with **low aldosterone and low renin**
- Diagnosed by **genetic testing**
- Treatment is potassium-sparing diuretics (e.g., **amiloride or triamterene**), which block tubular sodium channels; **spironolactone is not effective**.

2, SYNDROME OF APPARENT MINERALOCORTICOID EXCESS (SAME)

- 11 β -hydroxysteroid dehydrogenase 2 (11 β -HSD2) deficiency** allows cortisol to activate renal aldosterone receptors; **autosomal recessive**
- Presents as child with HTN, hypokalemia, and metabolic alkalosis, but with **low aldosterone and low renin**
- Diagnosed by **low urinary free cortisol** and **genetic testing**
- May also arise with licorice (**glycyrrhizic acid**), which blocks 11 β -HSD2

HYPERCORTISOLISM (CUSHING SYNDROME)

This disorder is caused by conditions that produce **elevated glucocorticoid levels**.

CAUSES

ACTH INDEPENDENT CAUSES

- **Iatrogenic cause** (most common cause) which includes exogenous glucocorticoids administration as a result of this **bilateral adrenal atrophy** occurs.
- **Primary adrenal adenoma, hyperplasia, or carcinoma** which causes **low ACTH** resulting in **contralateral adrenal atrophy**.

ACTH DEPENDENT CAUSES

- ACTH-secreting pituitary adenoma (**Cushing disease**). It is the **most common endogenous cause**.
- **paraneoplastic ACTH secretion** (eg, small cell lung cancer, bronchial carcinoids)

Both of these causes result in **bilateral adrenal hyperplasia**. In this **skin hyperpigmentation is seen due to high ACTH**.

SCREENING/ DIAGNOSIS

- **Serum cortisol & CBG** is not recommended due to fluctuations.