A case of hypokalemic paralysis, onset of primary hyperaldosteronism

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Introduction

Hypokalemic paralysis (HP) is a condition characterized by recurring episodes of sudden loss of strength associated with low potassium serum levels. Inherited and acquired forms of HP exist. Inherited forms are channelopathies linked to mutations in the sodium, calcium and rarely potassium channels; acquired forms, on the other hand, can be caused by a variety of electrolyte imbalances.

Presentation and history

A 55-year-old man, NIDDM treated with OHA (metformin), referred a history of recurring loss of strength in the lower limbs. He reported that many episodes had followed periods of intense stress in the workplace; during one of those, he became severely paraparetic and had to be brought to the emergency ward. In that circumstance, blood test showed severe hypokalemia (2,5 mEq/L);
the administratioin of potassium led to clinical remission.
A week after he was discharged from the E. R., he was admitted to our department for further evaluation.

Neurological examination

Negative

Electroneurography & Electromyography

Negative

Labolatory values

- *Routine blood tests* : normal
- *Sieric electrolytes*: normal
- *Urinary electrolytes*: normal
- *PRL, GH, IGF-1, ACTH, cortisole*: normal
- *Plasmatic Renin*: normal
- ***Plasmatic Aldosterone:* 455 pg/ml**
  (7.5-150)

Recumbent-Saline Infusion Test

Maintaining recumbent position at least 1 h before and during the infusion of 2 l 0.9% normal saline infused intravenously over 4 h. Sampling for renin, aldosterone and potassium drawn before initiation of infusion and after 4 h with continuous monitoring of BP and heart rate
Results: post-infusion renin and aldosterone were normal (Suppressibility)

Imaging

- *MRI brain and spine*: moderate pituitary hyperplasia
- *Abdomen ultrasonography*: normal finds
- ***Abdominal CT scan without and with contrast: bilateral surrenal hyperplasia (FIG.1)***

Discussion & Conclusions

This case highlights the importance of a full evaluation of both neurological and non neurological factors that can be responsible of a sudden loss of strength; in particular, a thorough collection of medical history and an extensive assessment of the electrolyte and hormonal balance can be a necessary step either for solving the diagnostic riddle either for providing an effective measure for treatment. Particular interest in our case seemed to be the strength deficit as the onset of the disease.
In our case increased serum level of aldosterone was suppressed after saline infusion test, as expected in some cases of idiopatic surrenal hyperplasia (suppressibility in false negative)
So our diagnosis was *hypokalemic paralysis caused by primary hyperaldosteronism.*

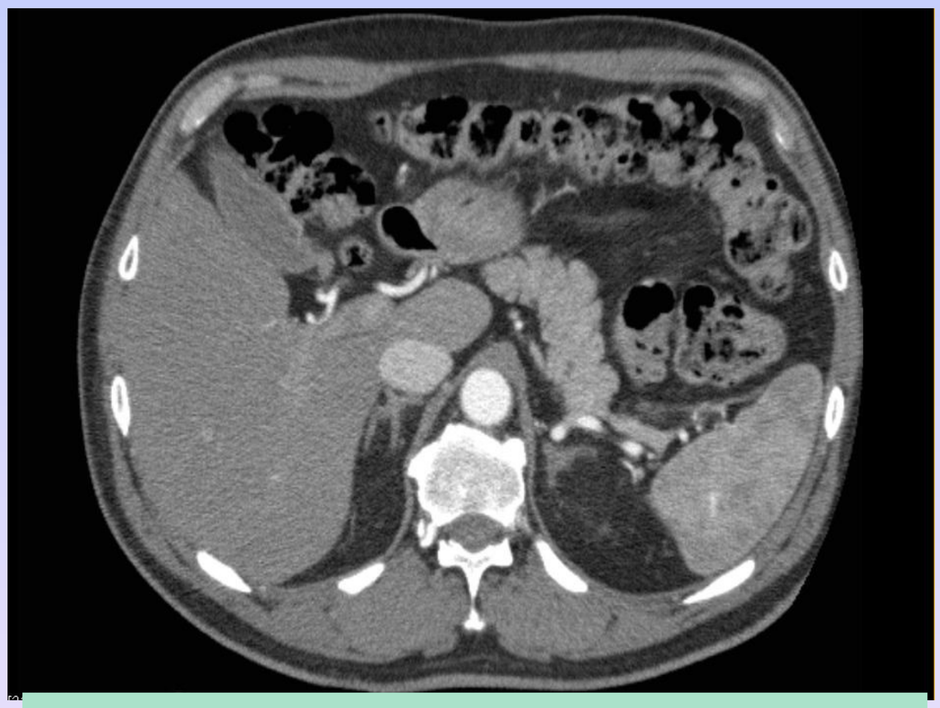

***Fig 1.* Abdominal CT scan with contrast**

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