ELECTROLYTE ADVICE

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Q. In order for a pediatrician to refer a child to a pediatric endocrinologist, must the blood work show low NA and high K, or can other symptoms suffice? Does someone with Addison’s ALWAYS have low NA and high K? Thanks!

A. Abnormal electrolytes can be a useful sign of adrenal insufficiency, but they are not a necessary clue. A referral should be made if there are significant symptoms and physical findings. Some Addisonians have an abnormal ACTH stimulation test in the absence of electrolyte abnormalities.

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Q. I have had Addison’s disease since 1972. Within the past year, I have had experienced low blood pressure more frequently than before. I have tried water, Gatorade®, increased prednisone and fludrocortisone acetate, and am concerned that I am not getting a long-term solution. Any ideas or confirmation?

A. Low blood pressure is a cardinal feature of Addison’s disease. Unless there is evidence of some other factor, my first response would be to increase the fludrocortisone acetate dose until the blood pressure is normal. I would expect your doctor to be able to document the need for an increased dose of fludrocortisone acetate by finding an elevated level of plasma renin and possibly as elevated serum potassium. I have many patients who need 0.3 mg of fludrocortisone acetate a day.

What Can You Expect at Your Endocrine Examination?

The medical follow-up exam of a patient with Addison’s disease must include a discussion of symptoms that may have occurred that may reflect the patient’s response to therapy: energy, stamina, weight changes, salt craving, dizziness, nausea, diarrhea, muscle cramps, appetite, change in pigmentation, change in mood are just a few of the questions to be reviewed.

A general physical exam should follow, including blood pressure sitting and standing, pulse, observation of skin, thyroid, lungs, heart, abdomen, muscle tone, reflexes.

Basic laboratory studies for a follow up exam should include basic chemistries, electrolytes, CBC. Other tests that should be performed if there are new symptoms or a recent change in medication are plasma renin, thyroid function tests, and vitamin B12 levels. In my opinion, there is no clinical utility in measuring ACTH, serum or urine cortisol or alsosterone once the diagnosis of Addison’s disease has been made and treatment has been started. The assessment of response is based on the clinical features reviewed in the interim history, physical and routine labs suggested above.

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Q. Why do some secondary adrenal insufficient patients (either from pituitary non-function or adrenal atrophy from long-term cortisol prescription use) end up needing to take aldosterone replacement medication?

A. Aldosterone is primarily regulated by the kidney. When blood volume drops, the kidney makes renin, which then stimulates the production of angiotensin, which is metabolized in the lung, and then stimulates the adrenal to produce aldosterone and increase sodium retention and potassium excretion and increase blood volume. This mechanism usually does not require the pituitary, and therefore most people with secondary adrenal insufficiency (who lack ACTH) have only cortisol deficiency, but still maintain adequate aldosterone production, since their adrenals are intact. However, there are some people (about 10% of the population) who do seem to need ACTH stimulation to maintain their renin-aldosterone balance. These people wind up with high potassium levels despite prednisone treatment, and they do respond to fludrocortisone (Florinef®), or may be managed with hydrocortisone in place of just prednisone alone.