Addison’s disease –

Anatomy & Physiology Gerald McGraw January 27, 2012 Addison’s Disease A disorder that takes place when your body produces an insufficient amount of certain hormones that are produced by the adrenal glands is called Addison’s disease. With Addison’s disease, the adrenal glands do not produce enough cortisol and often times an insufficient amount of aldosterone as well. Doctors sometimes refer to Addison’s disease as the illness of chronic adrenal insufficiency, or also called hypocortisolism. In 1855, Thomas Addison first described adrenal insufficiency, which was subsequently named after him. The basis of Addison disease has dramatically changed since its initial description. Originally, the disease usually resulted from an infection of the adrenal gland; the most common infection was tuberculosis, which is still the predominant cause of Addison disease in developing countries. Currently, in developed countries, Addison disease most commonly results from nonspecific autoimmune destruction of the adrenal gland. The most important function of cortisol is to aid the body in its response to stress. Cortisol also helps the body regulate its use of protein, fat, and carbohydrates; maintain cardiovascular function and blood pressure as well as control inflammation. Aldosterone helps the kidneys regulate the amount of salt and water in the body, which is the main way to keep blood pressure under control. When the body’s aldosterone level drops too low, the kidneys have a hard time keeping salt and water levels in balance, which makes your blood pressure drop. Addison’s disease has two forms.

If the adrenal glands themselves have a problem, it’s called primary adrenal insufficiency. If it is the adrenal glands being affected by a problem starting

somewhere else, for example in the pituitary gland, it can be called a secondary adrenal insufficiency. Symptoms for Addison’s disease are nonspecific and usually develop slowly, often times over the course of several months, they may include weakness, fatigue, nausea, anorexia, diarrhea, abdominal pain, gastroenteritis, weight loss with a decreased appetite, low blood pressure, which may include fainting, salt cravings, and depression.

A person should visit their doctor for the diagnosis of this disease if they have severe fatigue, muscle or joint pains, salt cravings, unintentional weight loss, and gastrointestinal problems, such as vomiting and abdominal pain and nausea. The cause of Addison’s disease is the result of the adrenal glands being damaged and producing insufficient amounts of cortisol and aldosterone as well. The adrenal glands are located just above the kidneys. Being part of the endocrine system, the adrenal glands produce hormones that give instructions to almost every organ and tissue in the body. The adrenal glands are made up of two different sections.

The interior (medulla) which produces hormones similar to adrenaline. The (cortex) outer layer has the role of producing a group of hormones that are called corticosteroids; these include mineralocorticoids, glucocorticoids and the male sex hormones known as (androgens). Addison’s disease takes place when the cortex is damaged and does not produce hormones in adequate quantities. Doctors often refer to this condition that involves damage to the adrenal glands as a primary adrenal insufficiency. When the adrenal glands fail to produce adrenocortical hormones it is commonly a result of an autoimmune disease where the body is attacking itself.

For an unknown reason the immune system sometimes views the adrenal cortex as being foreign, something for it to attack and destroy. Sometimes less commonly, Addison’s disease is not the failure of the adrenal glands. This condition is known as secondary adrenal insufficiency, and may be caused by a problem with the pituitary gland and the hypothalamus, which is located at the center of the brain. These glands produce hormones to the rest of the body. Tests and diagnosis for Addison’s disease may include a blood test, to measure the levels of sodium, cortisol, potassium and ACTH in your blood.

An ACTH stimulation test which involves measuring the level of cortisol in the blood before and after an injection of a synthetic ACTH. ACTH is what signals your adrenal glands to make cortisol. An Insulin-induced hypoglycemia test, this is done occasionally if a pituitary disease is the possible cause of adrenal insufficiency. (secondary adrenal insufficiency). This test checks your cortisol and blood sugar levels at various intervals after receiving an insulin injection. For healthy people the glucose levels fall and the cortisol levels increase.

Imaging tests may also be done to check the size of the adrenal glands and look for any other abnormalities that may give an insight to the cause of an adrenal insufficiency. Doctors may also suggest an MRI scan of the pituitary gland if other tests indicate that there may be a secondary adrenal insufficiency. Once Addison’s disease has been diagnosed the treatment plans mostly involve hormone replacement therapy which helps to correct the levels of steroid hormones the body is not producing. Some different options include: Oral corticosteroids, these may be prescribed as fludrocortisones to replace aldosterone.

Prednisone, hydrocortisone or cortisone acetate could be prescribed to replace cortisol. Corticosteroid injections may be prescribed if you are vomiting and cannot retain any oral medications. Androgen replacement therapy may be used to treat an androgen deficiency in women. People with Addison’s disease are recommended to intake an ample amount of sodium, especially following heavy exercise, if the patient has gastrointestinal upsets, or when the weather is hot. If the optimum dosage is used glucocorticoids are generally used with a minimal adverse effect. The under dosage of glucocorticoids could result in continued adrenal insufficiency.

The overdosing of glucocorticoids results in increased blood pressure, osteoporosis and weight gain. “ The resolution of symptoms and the correction of electrolyte abnormalities are the customary signals in determining the adequacy of replacement. In patients at risk for osteoporosis, monitor serum and urine cortisol levels; this method appears to be the best available assessment of steroid dosing. The titration of mineralocorticoid replacement is achieved by monitoring electrolyte levels and plasma rennin concentrations and by evaluating clinical findings such as dizziness or weight gain.

Weakness, decreased diastolic blood pressure, low serum sodium levels, and increased plasma rennin concentrations occur with an under dosing of fludrocortisone. Overdosing is difficult to determine. Decreased serum potassium levels may be seen. Increased levels of atrial natriuretic peptide have been proposed to be more accurate in determining an overdose. ” The report of Addison’s disease incidence in the United State is 5 to 6 cases per million of the population per year, this is with a prevalence of 60-110 cases

per million of the population. The mortality rate for Addison’s disease is as many as 1. deaths per million cases a year. Addison’s disease is more commonly found in women with the occurrence in men and women having a ratio of 1: 1. 5 to 3. 5. Addison’s disease may occur in a person of any age, however it is more common in people between the age of 30 to 50 years old. “ The expression of adrenal cortex antibodies (ACAs) in patients without symptoms of Addison disease represents a significant risk of progression to adrenal insufficiency. The risk varies with age; children have a high risk of progression compared with adults, in whom the expression of ACAs represents a 30% risk of progression to Addison disease. Adrenal crisis has a lower diagnosis rate in the United States that has been demonstrated in 31% of patients admitted to the hospital as compared to Addison’s disease that has an incidence of 19-54% of patients. Works Cited http://www. webmd. com/a-to-z-guides/understanding-addisons-disease-basics http://www. mayoclinic. com/health/addisons-disease/DS00361/DSECTION= tests-and-diagnosis http://www. nlm. nih. gov/medlineplus/ency/article/000357. htm http://emedicine. medscape. com/article/116716-overview#a0199