## A and p case study on addisons disease

**Education** 



Unit 1Case StudyAddison's disease is from a result from damage to the adrenal cortex. These two adrenal glands are located on top of the kidneys, where they are enclosed in a fibrous capsule and a cushion of fat. . The have an outer portion, called the cortex, which is a glandular tissue derived from embryonic mesoderm. The inner portion called the medulla, which is more like a knot of nervous tissue than a gland and it is part of the sympathetic nervous system. The adrenal cortex synthesizes over two dozen steroid hormones called corticosteroids. The Adrenal medulla is part of the autonomic nervous system.

They are often referred to as the suprarenal glands. That damage causes the cortex to produce less of their hormones (corticosteroids) The 3 types of hormone secreted are the glucocorticoid hormone (Cortisol) which maintain glucose control, decrease immune response and help the body respond tostress. The mineralocorticoid hormones (aldosterone) regulates sodium and potassium balance. The 3rd one is the sex hormones, androgen and estrogen, which affect sexual development and sex drive. There are a few things that may cause damage such as, the immune system mistakenly attacking the gland autoimmune disorder), infections such as tuberculosis, HIV or fungal infections. Hemorrhage's, tumors and use of blood thinning drugs are some other causes. Risk factors for the autoimmune type of Addison's disease include chronic thyroiditis, dermatis herpetiforms, Graves' disease, hypoparathyroidism, hypopituitarism, myasthenia gravis, testicular dysfunction, type 1diabetesand pernicious anemia. Usually the destruction of the adrenal gland cortex is often gradual and the symptoms are mild. Addison's often goes undiagnosed until a sudden illness or accident occurs.

The worsening of symptoms is called acute adrenal insufficiency and can be life threatening. Let's go back to the start of symptoms with Addison's disease. Here are a few, changes in heart rate and blood pressure, chronic diarrhea, darkening of the skin (it becomes patchy), irregular menstruation, irritability, paleness, extreme weakness, fatigue, loss of appetite, salt cravings, slow, sluggish movements, weight loss, lesions on the buccal mucosa, nausea and vomiting. Acute adrenal crisis have similar symptoms of course, however you will also see abdominal ain, confusion, dizziness, headaches, joint pain, rapid heart rate, rapid respirations, shaking chills, unusual and excessive sweating on face and/or palms. And in severe cases coma and/or death. The initial diagnosis and decision to treat are based on history, physical examination and lab findings. Lab tests such as ACTH stimulation test, cortisol level, fasting blood sugar, serum potassium and serum sodium. Tests may show an increase in potassium, low cortisol level, low serum sodium. The Adrenal medullae normally secrete 80% epinephrine and 20% norepinephrine. Sympathetic stimulation results in secretion.

Epinephrine is the more potent stimulator of metabolic activities, but norepinephrine has the greater influence on peripheral vasoconstriction and blood pressure. The adrenal cortex produces the 3 hormones listed above. Cortisol is produced from 2 hydroxylations of 17 alpha-hydroxyprogesterone. Cortisol is 90-93% protein bound. Glucocorticoids are nonspecific cardiac stimulants that activate release of vasoactive substances. So in the absence of corticosteroids, stress results in hypotension, shock and even death. Glucocorticoids stimulate gluconeogenesis and decrease cellular glucose use, obilize amino acids and fatty acids, inhibit the effects of insulin, and give

rise to ketone bodies in metabolism, elevate RBC and platelet levels and exhibit anti- inflammatory effects. Adrenal crisis occurs when the adrenal gland is damaged (primary adrenal insufficiency), the pituitary gland is injured (secondary adrenal insufficiency) or that adrenal insufficiency is not properly treated. Treatment with replacement corticosteroids will control the symptoms of this disease, and this usually will require the patient to take these drugs for life. It is receive a combination of glucocorticoids and mineralocorticoids.

The provider may increase the dose in times of infection, injury and stress. With adrenal crisis patients will need an immediate injection of hydrocortisone, either IM, or IV. If the blood pressure is extreme low IV fluids will be helpful. Complications can occur if you take too much or not enough of the adrenal hormone supplement. This complications can arise due to related illnesses such as diabetes, chronic thyroiditis, hypoparathyroidsim, ovarian hypofunction or testicularfailure, thyrotoxicosis and pernicious anemia. Low sodium with Addison's should be carefully corrected, if one to quickly it can lead to brain damage, this is noted in a 2004 article in " AmericanfamilyPhysician" they require immediate but slow administration of saline, which is basically salt water, composed of sodium chloride in water. They list a second step which then be finding the underlying cause. Primary adrenocortical insufficiency is not a common disorder. It's incidence in western populations are near 50 cases per 1, 000, 000. However with the widespread corticosteroid use secondary adrenocortical insufficiency due to steroid withdrawal has become much more common.

Approximately 6, 000, 000 persons in the United States are considered to have undiagnosed adrenal insufficiency, which is significant only during times of physiologic stress. Primary adrenocortical insufficiency does have many etiologies. But it is noted that 70-80% of the cases in the United States are caused by autoimmune adrenal destruction. It is found that about 30% of the time the adrenal damage is due other causes such as TB. In children about 70% of the cases are caused by a congenital disease termed congenital adrenal hyperplasia. Primary adrenocortical insufficiency affects men and women equally, women are affected 2-3 imes more often by the idiopathic autoimmune form of adrenal insufficiency. In idiopathic autoimmune adrenal insufficiency, the diagnosis is most often found in the third to fourth decades of life. This disease however is not limited to any specific age group. "American Family Physician", Kian Peng: 2004 "Robbins and Cotran Pathologic Basis of Disease", Vinay Kumar 8th Ed 2009 http:/emedicine. medscape. com/article/765753-overview http:/labtestsonline.org/understanding/conditions/addisons-disease/ http:/www. nlm. nih. gov/medlineplus/ency/article/000378. htm