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PROPEDYTICS OF CHILDREN'S DISEASES CLINICAL CHARACTERISTICS AND FUNCTIONAL STATE OF THE ADRENAL CORTEX AND THYROID GLAND IN THE RECOVERY PERIOD AFTER COVID-19 IN CHILDREN

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ANNOTATION

In this article, you will learn about the impact of Covid-19 infection on children's adrenals and thyroid. You will learn about the signs that appear in children.

Keywords: Covid-19, adrenal glands, thyroid, kidney failure, prevention and treatment.

This flyer aims to provide guidance on prevention and treatment of COVID-19 in patients with a pediatric endocrine disease. It summarizes fast facts about COVID-19, and what children with endocrine diseases and their parents need to know regarding their health and well-being. These recommendations are based on the latest knowledge and expert opinion. If you still have concerns or questions after reading this information, please contact your physician or healthcare provider.

Children and COVID-19. Children play a minor role in coronavirus spread. Coronavirus infection mostly distributes from adult to adult. Distribution among children or from children to adults is rare. In children, the disease course of COVID-19 is generally mild, mostly they do not get sick or only mildly and for a short time. Often no special treatment is required other than simple supportive measures (drink enough water, use paracetamol). Few children have complaints that are so serious that hospitalization is necessary. Are children with Adrenal Insufficiency at increased risk of COVID-19 infection or severe course in COVID-19. Currently, there is no evidence indicating that patients with Adrenal Insufficiency are at an increased risk for COVID-19 infection. However, several studies have demonstrated that adult patients with Adrenal Insufficiency have a 2-fold to 8-fold higher risk for infection in general, which inherently increases the risk of death from COVID-19. In patients with Adrenal Insufficiency, the innate immune response is impaired, thereby potentially compromising antiviral immune defense mechanisms and increasing patients' susceptibility to respiratory viral infections. Moreover, the life-long requirement for supraphysiologic glucocorticoid replacement using currently available preparations may place patients with Adrenal Insufficiency at an increased risk for infectious diseases.

What should children with Adrenal Insufficiency do in case of an infection.

Infection is a condition of acute stress, which requires an increased dose of glucocorticoids. Since adrenal crises precipitated by infections are the major cause of death in patients with Adrenal Insufficiency, an immediate modification of the glucocorticoid regimen, as indicated in so-called "sick day rules," should be conducted at the beginning of an infection. Whenever patients with.

Adrenal Insufficiency present with cough, sputum, or fever (≥37.5°C), which are symptoms suspicious for COVID-19, they need to immediately double or triple their daily oral glucocorticoid dose and continue with the increased dose until the symptoms resolve in order to avoid adrenal crisis. In addition, patients need to consume more electrolyte-containing fluids. If their condition deteriorates, or they cannot eat due to vomiting or diarrhea, they should be admitted to the hospital to receive intravenous hydrocortisone. Furthermore, patients are advised to obtain sufficient hydrocortisone and fludrocortisone supplementation to prepare for "sick day rules" and "social distancing" during the COVID-19 outbreak in order to maintain the social confinement when required for impeding the COVID-19 outbreak spread.

Do the coronavirus prevention measures differ for children with Adrenal Insufficiency compared to the general population? The coronavirus measures are no different for children with Adrenal Insufficiency than for healthy children. No extra precautions are needed other than the usual advice. They should go to school when permitted by the general coronavirus prevention measures. The coronavirus will be around for a long time; therefore, it is important for children to attend school regularly to allow for their education, as well as normal development and general well-being.

AI, adrenal insufficiency considered as a whole group; GIS, gastrointestinal symptoms; PAI, primary adrenal insufficiency; SAI, secondary adrenal insufficiency; URTIS, upper respiratory tract infection symptoms. *$P < 0.05$ in controls vs AI; #$P < 0.05$ in SAI vs PAI.

Patients with primary hypothyroidism and hyperthyroidism disease should be managed per routine care. Anticipatory guidance should be given to patients taking anti-thyroidal drugs (ATDs) such as methimazole, given the risk of agranulocytosis and secondary bacterial infection. Symptoms to monitor for while on ATDs—including fever, sore throat and cough—do overlap with symptoms of COVID-19 thus prompt medical evaluation should be sought if symptoms arise. Additionally, like other infections, COVID-19 may precipitate thyroid storm in patients with poorly controlled hyperthyroidism. Most patients with thyroid cancer are not at increased risk of infection given that surgical treatment with replacement levothyroxine is standard of care. For the rare patients on chemotherapy, there may be an increased risk of all infections due to immunosuppression. As per the American Thyroid Association and the American Association of Endocrine Surgeons, most thyroid cancer surgeries can be safely postponed without risk for worsening disease course given the typically slow tumour growth. Relationships between COVID-19 and morphological and pathological changes in the thyroid gland. The pathogenesis of the thyroid dysfunction that is induced by COVID-19 has not been characterised. One theory is that the virus has a direct effect on the thyroid gland. SARS-CoV-2 appears to have the ability to infect the gland by direct infiltration from the upper respiratory tract. Post-mortem examinations of individuals who died of COVID-19 have revealed pathological abnormalities in various organs, including the thyroid gland. However, surprisingly, no morphological abnormalities or severe damage to thyroid follicles have been discovered. Histological examination of the thyroid has revealed the absence of a lymphocytic infiltrate but the presence of extensive apoptosis, indicative of destructive thyroiditis, which may be the cause of the thyrotoxicosis. In addition, despite ACE2 being highly expressed in the thyroid, SARS-CoV-2 has not been detected in thyroid tissues by either PCR or

immunohistochemistry.Thus, there may be factors that prevent the virus from infecting thyroid follicular cells. Relationship between COVID-19 and the hypothalamic–pituitary–thyroid axis An infection produced by SARS-CoV-2 has been reported to disrupt the nervous system by damaging the cranial nerves, resulting in the loss of smell and taste. SARS genome sequences have been found in the cytoplasm of many neurons in the hypothalamus, and immunohistochemical analysis of adenohypophyses obtained during the autopsy of five patients with SARS revealed significant reductions in both the number and immunoreactivity of thyroid-stimulating hormone (TSH)-positive cells. According to Ur et al., SARS-CoV-2 can propagate across nerve axons because high ACE2 expression causes a cytokine storm. High circulating concentrations of interleukin (IL)-6, IL-7, tumour necrosis factor (TNF)-α, the soluble version of the IL-2 receptor, and inflammatory chemokines have been detected during the cytokine storm generated by SARS-CoV-2. SARS-CoV-2 has a substantial effect on TSH-secreting cells, resulting in lower TSH concentrations and, as a result, a disruption of pituitary endocrine axis feedback loops. These effects on TSH-secreting cells may involve four different mechanisms: direct damage to the pituitary gland caused by SARS-CoV-2 (central TSH abnormalities caused by virus-related hypophysitis), indirect damage caused by pro-inflammatory cytokines and the cytokine storm, chronic stress caused by hypoxia, and the effects of specific classes of medication, such as glucocorticoids. Chen et al. found that the serum concentrations of TSH and total T3 were considerably lower in patients with COVID-19 than in a control group

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