A case of adrenal insufficiency during the course of multisystem inflammatory syndrome in children (MIS-C)

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ESPE 2021
The 59th Annual Meeting Online
22-26 September 2021

INTRODUCTION

Multisystem inflammatory syndrome in children (MIS-C) associated with pediatric COVID-19, has variable treatment options, one of which is glucocorticoids .

Although the effects of COVID-19 infection on the endocrine system is not yet fully understood, it has been reported that COVID-19 may rarely affect adrenal function and cause both primary and secondary adrenal insufficiency.

Possible mechanisms include a direct effect of infection on the hypothalamic-pituitary-adrenal (HPA) axis, mediated through cytokines such as interleukin 6 or via toll-like receptors, or indirect effects because of glucocorticoid use during treatment that modifies the inflammatory effect during MIS-C.

Here we present an 11-year-old boy who was followed up with MIS-C and developed adrenal insufficiency.

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CASE REPORT

An 11-year-old boy was referred with a five-day history of fever, vomiting and rash. He had Covid-19 one month previously and had recovered within two-days. At presentation hypotension (80/50 mmHg), tachypnea, tachycardia and rashes that faded under pressure were evident. C-reactive protein and erythrocyte sedimentation rate were elevated (378 mg/L and 82 mm/h, respectively). Serum glucose was 85 mg/dL, sodium was 124 mmol/L, potassium 3.19 mmol/L, while other biochemistry was normal.Covid-19 IgM was 1.83 (<10), IgG was 6.1 (<1.4). He was evaluated by rheumatology and infectious diseases and was diagnosed with MIS-C. Normotension was achieved with fluid and inotropic treatments. He was started on 60 mg/day methylprednisolone after a dose of intravenous immunoglobulin. The glucocorticoid regimen was changed on the third day of treatment to 500 mg methylprednisolone daily. After receiving 500 mg/day methylprednisolone for three days, the dose was reduced to 60 mg/day by the sixth day of treatment. On the eighth day of treatment, while on 40 mg of methylprednisolone/day, sodium decreased (from 140 to 129 mmol/L), and potassium increased (from 4.3 to 5.2 mmol/L). Serum adrenocorticotropic hormone was 18 pg/mL (0-45), cortisol 1.34 ug/dL (6-22), aldosterone 4.9 ng/dL (3.7-31) and plasma renin activity 0.02 ng/mL/h (0.06-4.69) which indicated relative adrenal insufficiency. Adrenocorticotropin stimulation test was not performed because of glucocorticoid therapy. Methylprednisolone was discontinued and hydrocortisone was started at 300 mg/m /day. Serum sodium and potassium normalized within two days (138 and 3.6 mmol/L, respectively) and hydrocortisone dose was gradually reduced to 7 mg/m^2 /day (Table).

After four months of hydrocortisone treatment, the morning serum ACTH level was 27 pg/ml and cortisol level was 10 mcg/dL, so hydrocortisone was discontinued, and a low-dose ACTH stimulation test was performed. During the test, the peak cortisol value was below 18 mcg/dL (16.6 mcg/dL), that indicated the HPA axis had not fully recovered by this time.

Therefore, physiological dose hydrocortisone treatment was restarted at a dose of 5 mg/m^2 daily. At the sixth month of treatment, a repeat re-evaluation of the HPA axis is planned.

Time	Serum sodium (mmol/L)	Serum potassium (mmol/L)	Treatment
Day-1	124	3.19	IVIG, MP 60 mg/day
Day-5	138	3.88	MP 500 mg/day
Day-8	129.9	5.23	MP 2x20 mg/day
Day-9	131.8	4.37	HC 300 mg/m^2/day
Day-12	139.6	3.5	HC 100 mg/m^2/day
Day-18	139	3.7	HC 7 mg/m^2/day
Fourth month	138	4	HC 5 mg/m^2/day

Table: Therapy modalities and biochemical variables during the follow-up.

CONCLUSIONS

COVID-19 may cause both primary and secondary adrenal insufficiency, due to its effects, individual hypersensitivities, and treatment of steroids. Clinicians should be aware that the adrenocortical response to COVID-19 infection may be significantly impaired. The HPA axis should be evaluated, especially in COVID-19 patients with unexplained hypotension and hyponatremia.