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RESEARCH ARTICLE

ADDISON'S CRISIS SECONDARY TO STERIOD WITHDRAWAL: A CASE STUDY

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ABSTRACT

Addison's disease is an autoimmune condition causing the destruction of adrenal cortex leading to the production of deficit amount of adrenal hormones. The condition is very severe and rare because the adrenal hormones i.e. Glucocorticoids and Mineralocorticoid that are involved in modulating salt, energy and fluid homeostasis are being altered. Addison's Crisis secondary to withdrawal of steroid therapy is usually infrequent and said to be due to inappropriate discontinuation or excessive of this therapy. The symptoms happen to correlate with the pathological changes of adrenal antibodies with the endocrine gland and account for its severity. The following manifestations are drawn from it: fatigue, muscle weakness, loss of appetite, fever, nausea, vomiting, diarrhea, hypotension, hypoglycemia and hyper pigmentation of the skin. The diagnosis is usually confirmed by measuring the cortisol level or adrenocorticotropic hormone concentration. Lastly, the treatment involves the replacement of hormones.

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INTRODUCTION

Endocrine disorders are usually remained undetected due to lack of awareness among the clinicians and limited facilities. Addison crisis is the life-threatening complication and can prove fatal if not treated appropriately. The condition is caused by the deficiency of glucocorticoids (cortisol) and mineralocorticoids (aldosterone) which have a major role to play in maintain the homeostasis (Iliopoulou *et al*., 2013). Adrenal androgen depletion is also seen to a less extent. Cortisol, a steroid hormone, is synthesized and secreted by the adrenal cortex of the suprarenal gland. The secretion of which is governed by the three disseminating regions of the body, the suprarenal glands, the pituitary glands and the hypothalamus. The hormone cortisol helps in the maintenance of homeostasis. It is also called as stress hormone as it secreted with response to stress by suprarenal gland. Certain alteration such as the changes in the blood sugar levels, anti-inflammatory actions, immune responses, metabolism, salt and water balance, blood pressure and central nervous system activation are governed by hormone cortisol (Barthel, 2016). The inability of the adrenal cortex to synthesize and secrete cortisol and aldosterone, the hypothalamus releases corticotropin-releasing hormone, which stimulates the pituitary gland to secrete adrenocorticotropic hormone (ACTH) to stimulate the suprarenal glands (Løvås, 2013).

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The deficiency of hormone cortisol is usually caused either by the destruction or dysfunction of both the adrenal cortices resulting in primary hypoadrenalism that refers to a condition called Addison's disease or primary adrenal insufficiency (Nieman, 2006). A condition called secondary adrenal insufficiency were the glucocorticoid production is the insufficient while, the mineralocorticoid production is close to normal, as the latter is modulated by salt and water metabolism while the former is modulated by the ACTH (Alebiosu, 2003). Hence, secondary adrenal insufficiency occurs as a result of ACTH deficiency. The signs and symptoms are mainly nonspecific such as fatigue, nausea, abdominal pain, weight loss, and hyperpigmentation. ACTH, being more potent stimulator of melanogenesis than alpha melanocyte stimulating hormone, the hyperpigmentation is observed in patients with primary adrenal insufficiency (Choudhary *et al*., 20011). Vitiligo and alopecia areata may be present in the patients with autoimmune aetiology because of auto immune destruction of melanocytes and hair follicles. The corticosteroid is considered as the mainstay of therapy followed by use of pressor agents or antibiotics as clinically indicated.

Case Reports

A 40 year old male patient fetched to the emergency department in a condition of extreme pain and suffering. The reason behind this pain and suffering is the sudden withdrawal of steroid use.

As he have a history of arthritis and was on NSAIDs (Ibuprofen) and steroid (Prednisone) since 2 years which he abruptly withdraw. On hospital admission, he was in a condition of breathlessness, nausea, hiccups, vomiting, and anuria, which he was unable to relate the cause of it. On physical examination, he was found to be conscious and coherent. A pinpoint pupil was observed. The systemic examination showed the following, Blood Pressure-90/60mmHg (120/80mmHg), Pulse Rate-88beats/min (72beats/min), Respiratory Rate-18breaths/min (22breaths/min), General Random Blood Sugar (GRBS)-70mg/dl and Temperature- 99 degree Fahrenheit. All these clinical manifestation were taken into consideration and appropriate treatment is further given.

Treatment

Emergency airway management: Airway support was provided by administrating the nasal oxygen at a rate of 2 liters/minute to facilitate the breathlessness.

Corticosteroid Therapy: Injection Hydrocortisone-100mg in Ringer Lactate solution, which helps in relieving the steroid withdrawal symptoms and breathlessness as well.

Pressor Agents: Injection Dopamine-1mg/minute Intravenous Infusion, is administered to counteract the symptoms of mood alteration, hyperactivity, insomnia, and psychosis which are seen by prolonged use of corticosteroid.

Electrolyte balance: Fluids such was Calcium Gluconate-100mg/dl (10%) which contain 93mg/ml of elemental calcium mainly administrated to treat hyperkalemia, Dextrose Normal Saline (DNS)-70%, Ringer Lactate (RL)-70% and Normal Saline (NS)-100%

Ulcer protective agents: Injection Ranitidine-25mg/ml (prophylactic therapy) and Injection Ondansetron-2mg/ml (Nausea and Vomiting).

Antibiotic Therapy: Injection Ceftriaxone-1gm/50ml as an injectable solution as it is clinically indicated in adrenal crisis condition.

Day 1: On day 1, the Blood Pressure was 90/60mmHg, Pulse Rate- 88beats/minute, Respiratory Rate- 18breaths/minute and temperature- 99 degrees F was observed. The physician advised the following lab investigations to done:

- Complete blood picture
- Serum electrolyte
- Liver function test
- Ultrasound of abdomen and Pelvis

The following medication was prescribed and are listed below in Table 1

Table 1. Medication chart

SI.No	Drugs	Dose	Route
1.	Inj. Hydrocortisone in RL	100mg	Intravenously
2.	Inj. Dopamine	1mg/min	Intravenously
3.	Inj. Ranitidine	25mg/ml	Intravenously
4.	Inj. Ondansetron	2mg/ml	Intravenously
5.	Inj. Calcium Gluconate	100mg/dl	Intravenously
6.	Inj. Ceftriaxone	1gm/50ml	Intravenously
7.	IVF Normal Saline	100%	Intravenously
8.	IVF Ringer Lactate	70%	Intravenously
9.	IVF Dextrose Normal Saline	70%	Intravenously

Day 2: On day 2, the patient was found to be responding to medication and vitals were improving. The Blood Pressure was 100/80 mmHg, Pulse Rate- 82 beats/minute, Respiratory Rate-20 breaths/minute and temperature- 98.5 degrees F was observed. The physician advised the following reports to done:

- Serum electrolyte
- Liver function test
- Complete Blood Picture was found to be normal

Day 3: On day 3, no fresh complaints were noted.

Day 4: The Same treatment was continued for four days and the abnormal laboratory investigations of day 1,2,3,4 are shown below in table 2, 3, 4

Ultrasound of Abdomen and Pelvis

Impression

- Liver- normal
- Gallbladder- normal
- Pancreas- normal
- Spleen- normal
- Kidney: Right kidney-8.2*4.4cm and Left Kidney-8.0*4.2cm

A simple renal cortical shift of 1.2*1.2cm is sited in inferior pole of the right kidney Grade 2 Fatty Liver.

Day 5: Discharge Medication

- Tapering dose of Prednisone given at the time of discharge
- 50mg is given for 7 days after that 40mg for 7 days similarly 10mg is reduced until a 5-10mg dose is reached.

DISCUSSION

Thomas Addison first described the clinical features associated with primary adrenal insufficiency which are the result of the variety of pathological processes. Causes of Primary adrenal insufficiency include the Autoimmune adrenalitis, Infections (tuberculosis, systemic fungal infections, AIDS), Metastasis (from lung, breast, kidney) (rare), lymphoma, Congenital adrenal hyperplasia, Adrenomyeloneuropathy/ adrenoleukodystrophy, Bilateral adrenal hemorrhage and Bilateral adrenalectomy. Secondary adrenal insufficiency includes the Pituitary or metastatic tumor, Other tumors (craniopharyngioma, meningioma), Pituitary surgery or radiation, Lymphocytic hypophysitis, Head trauma, Pituitary apoplexy/Sheehan's syndrome, Pituitary infiltration (sarcoidosis, histiocytosis), Empty-Sella syndrome. And Glucocorticoid-induced adrenal insufficiency includes the Long-term exogenous glucocorticoid use (Puar *et al*., 2016). The clinical presentation is usually manifest in group of patients as hyperpigmentation (85%), weight loss (66%), abdominal pain (22%), diarrhoea (18%), gastrointestinal complaints (>80%), body aches (18%), hypotension (90%), hyponatremia (78%), hyperkalaemia (53%), hypercalcemia (6%) and hypoglycaemia (18%) (Widows, dec 7, 1895). Diagnosis is based on cortisol level and to a lesser extent by identification of large adrenal gland through imaging techniques.

Table 2. Serum cortisol test

SI.No	Laboratory Data	Day 1	Day 2	Day 3	Day 4	Normal Level
1.	Serum Cortisol	7.8mcg/dl	9.0mcg/dl	10.2mcg/dl	11mcg/dl	10-15mcg/dl

Table 3. Serum Electrolytes

SI.No.	Laboratory Data	Day 1	Day 2	Day 3	Day 4	Normal value
1.	Sodium	130mmol/l	138mmol/l	140mmol/l	-	135-145mmol/l
2.	Potassium	5.83mmol/l	5.02mmol/l	4.2mmol/l	-	3.5-5.0mmol/l
3.	Chloride	98mmol/l	100mmol/l	100mmol/l	-	95-105mmol/l
4.	Serum Creatinine	2.12mg/dl	1.9mg/dl	1.3mg/dl	-	0.6-1.5mg/dl

Table 4. Liver function test

SI.No.	Laboratory Data	Day 1	Day 2	Day 3	Day 4	Normal value
1.	Total protein	6.6gm/dl	-	-	-	6.0-7.5gm/dl
2.	Albumin	4.2gm/dl	-	-	-	3.5-5.0gm/dl
3.	Total bilirubin	1.7mg/dl	-	-	-	0.2-0.8mg/dl
4.	Direct bilirubin	2.2mg/dl	-	-	-	0 0-0.2mg/dl
5.	SGOT	30U/L	-	-	-	05-45U/L
6.	SGPT	38U/L	-	-	-	05-45U/L
7.	Alkaline Phosphatase	84U/L	-	-	-	28-88U/L

This help in detection of infiltrating disorders, malignancy or haemorrhage. The treatment aims at replacing the deficient hormone with appropriate hormone and treat the reversal causes of the adrenal crisis. The therapy include the Glucocorticoids (hydrocortisone-10-20mg), Mineralocorticoids (fludrocortisone-0.1mg) and Adrenal Androgen (DHEA-dehydroepiandrosterone) (Brooke, 2013). The challenge of adrenal crisis arises because of it unspecific presentation and diagnosis (Brain, 2008).

Conclusion

With the advances in the medicine the adrenal insufficiency is treatable but, due to the failure of diagnosis and unpredictable clinical manifestations leads to the life-threating complication. Steroid withdrawal syndrome usually causes the reduction of glucocorticoids (cortisol). The appropriate replacement of cortisol helps in maintaining the overall health of the patients. Adequate monitoring of dose reduction is done by regulating the steroid hormone levels. Hence the understanding of unspecific signs and symptoms by clinicians helps the patients in his/her wellbeing.

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