ASSESSMENT OF THE ADRENAL FUNCTION IN CHILDREN WITH ACUTE LYMPHOBLASTIC LEUKEMIA BEFORE AND AFTER INDUCTION THERAPY WITH CORTICOSTEROIDS



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INTRODUCTION

Corticosteroids are the cornerstone of treatment of childhood acute lymphoblastic leukemia (ALL). Dexamethasone and Prednisolone are synthetic analogues that mimic the natural steroids produced by adrenal glands. The main effects of these drugs are reduction of inflammation; immunosuppression and they have anti-proliferative and cytotoxic effects on cancerous cells.

RESULTS

Thirty-three patients with ALL were recruited; 63% of them were males. The mean age of the cases was 6.18 years + 3.16. Basal cortisol was significantly higher than follow-up values at the end of induction phase (T=2 and P < 0.000). Adrenal suppression was found in 12 patients (36.3%) within the first 2 weeks after discontinuation of prednisolone and persisted later in 5 patients who

were given hydrocortisone replacement with follow up every 4 weeks till full recovery (Table 2).

However, Glucocorticoids can suppress the function of the hypothalamus-pituitary – adrenal axis and consequently induce atrophy of the adrenal gland or secondary adrenal insufficiency.

Therefore, this can disrupt cortisol response to stress and can be a cause of morbidity and even mortality in those children.

OBJECTIVES

To assess the adrenal gland function of children with acute lymphoblastic leukemia before and after induction therapy of corticosteroids.

CONCLUSION

Cortisol level assessment must be obtained after steroids discontinuation for all patients with ALL. Steroids replacement therapy should be started immediately if abnormal levels were detected and follow up is required.

Age	(2-15 years)			mean	Std. Deviation	
				6.179	3.1570	
Gender		No.	%			
	Male Female	21 12	63.6% 36.4 %			
Diagnosis	Pre B ALL	30	91%			

Initial C	NS involvement	Yes No	6 27	18.2% 81.8%
Protocol	UK ALL 2011 F	Regimen A	23	Standard risk protocol
	UK ALL 2011 F	Regimen B	2	Intermediate risk protocol
	UK ALL 2011 F	Regimen C	4	High risk protocol
	AALL 0434		4	T cell ALL
			TABLE	1 : Demographic and clinical data of the
				studied patients

METHODS

Newly diagnosed ALL cases during the period from December 2016 till end of January 2018 were recruited from pediatric hematology/ oncology ward at Sultan Qaboos University Hospital. Basal Adrenocorticotrophic hormone (ACTH) and cortisol levels were done at 7:30 am before and after induction therapy with Corticosteroids. ACTH stimulation test using a standard dose of (250 mcg) was done for those with low cortisol after induction and hydrocortisone replacement was started in cases with abnormal test response with follow up every 4 weeks to check their serum cortisol till recovery of adrenal gland.

Referen	ce range cortisol 7:30	<u>am (83-580 nmol/L)</u>
Cortisol	Mean	Std. Deviation
Baseline Cortisol	431.1304	292.02962
F/U cortisol	218.2106	416.31044
T Value 2		TABLE 2:Comparison of serum cortisol level in the study

			patients be	erore and after the steroid treatment.			
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	<u>Reference range ACTH 7:30am (7.2-63 ng/L)</u>						
	NO.	Mean	std. Deviation				
Baseline ACTH	28	28.0643	22.07647				







