Abstract PO-0062 Table 1

Group Statistic							
	Group	N	Mean	Standar deviation	Mean Standar error	U Mann Whitney	T Student Levene0,3
Age now	control	12	13,5342	1,80952	,52236		
	intervention	12	14,7092	1,24086	,35821		
Evolution time	control	12	6,1833	3,79014	1,09412		
	intervention	12	8,2333	4,47424	1,29160		
HbA1c before Accucheck	control	12	8,0750	,30488	,08801		
	intervention	12	8,1167	,83212	,24021		
dif_0_3m	control	12	-,0667	,68799	,19861	,799	,685
	intervention	12	-,1667	,48492	,13999		
dif_3_6m	control	12	-,0917	,84473	,24385	,291	,502
	interventionn	12	-,3000	,63389	,18299		
dif_0_6m	control	12	-,1583	,36794	,10621	,266	,196
	intervention	12	-,4667	,71138	,20536		

95% IC 0,40-0,60

95% IC 0,42-0,84

95% IC 0,18-0,79

PO-0064 CYSTIC FIBROSIS RELATED DIABETES IN ADOLESCENTS

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Background Cystic fibrosis related diabetes (CFRD) is a combination between reduced insulin secretion and peripheral insulin resistance that only people with cystic fibrosis can get. CFRD is associated with a decline in lung function, poor nutritional status and high mortality rate.

Aim Study of the clinical course and therapeutic management in adolescents with CFRD.

Methods We present 2 cases of CFRD in a female patient (15 years old) and a male patient (17 years old) hospitalised at the CF Centre. The diagnosis of CF was confirmed by positive sweat test (Macroduct USA), identification of CFTR mutation (F508del/F508del), small amounts of elastase in stool. Confirmation of diabetes was achieved by a blood glucose test, blood glucose profile, glycosylated Hb, C peptide, glucose and ketones in urine.

Results On the background of pulmonary exacerbation, both patients had hyperglycemia (9.8/14.1 mmol/l). Subsequently, it triggered clinical syndromes suggestive for diabetes - polydipsia, polyuria, weight loss. Glycemic profile variations 7.8–15.8 mmol/l in boy and 10.4–21.0 mmol/l in girl. Glycosylated Hb values were high (7.3/14.1%) and C-peptide values were low (0.624/0.513). Glucose concentration in urine was 7.3/37.1 g/l and ketones was not detected. Diabetes treatment was performed with Insulin (Glargine, Aspart, Human), which produced clinical benefits by achieving glycemic and clinical syndromes control.

Conclusions Patients with CFRD shows a specific clinical framework and require a strict medical diet control and surveillance

of the insulin therapy, case that differs from the other types of diabetes. If early introduced, it significantly improves life expectancy towards these patients.

PO-0065 VITAMIN D STATUS IN CHILDREN WITH TYPE 1 DIABETES

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Background and aims Serum vitamin D (sVD) deficiency may contribute to the development and progression of diabetic nephropathy (DN). Research has shown urinary vitamin D binding protein (uVDBP) excretion is increased after renal injury, and is associated with tubulointerstitial damage. Animal studies suggested that sVD deficiency may be associated with urine loss due to kidney damage. The aim was to test associations of sVD with levels of uVDBP, urinary vitamin D (uVD).

Methods 42 children aged 6–17 years with type 1 diabetes were examined: 24 normoalbuminuric patients (1st group) and 18 microalbuminuric (2nd group). 15 healthy children were included in controls. We measured serum and urine 25(OH)D levels, uVDBP concentrations and tested their correlations.

Results sVD levels were decreased in the patients of the 1st and 2nd groups, compared with controls ((22.03 (17.23; 24.44) and 14.42 (12.02; 19.63), compared with 30.65 (28.45; 35.05) ng/ml, respectively) ($p < 0.001$)). uVDBP levels were elevated in the patients of the 1st and 2nd groups, compared with controls ((179.5 (174.0; 189.0) and 219.0 (216.0; 222.0), compared with 125.0 (116.5; 136.0) ng/mg, respectively) ($p < 0.001$)). uVD levels were increased in the patients of the 1st and 2nd groups, compared with control group ((3.2 (2.9; 3.3) and 3.9 (3.7; 4.1),

compared with 2.2 (2.1;2.6) ng/mg, respectively) ($p < 0.001$). The correlations between the levels of sVD and uVD ($r = -0.74$, $p < 0.01$), sVD and uVDBP ($r = -0.64$, $p < 0.01$) were determined.

Conclusions These data suggest that, theoretically, one of the causes of VD deficiency in patients with DN is a urine loss.

PO-0066 CIRCULATING OXIDISED LDL AND INSULIN RESISTANCE AMONG OBESE SCHOOL STUDENTS

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Circulating oxidised LDL (ox-LDL) is associated with obesity, insulin resistance (HOMA), metabolic syndrome, and cardiovascular disease in adults. Little is known about relations in children.

Aim To assess association of ox-LDL with fat distribution and insulin resistance in a group of obese Egyptian children.

Methods Study is cross-sectional consisted of 68 obese children, mean age 9.96 ± 1.32 . Each underwent a complete physical examination; blood pressure (SBP, DBP) and anthropometric measurements (weight, height, BMI; waist, hip circumferences, waist/hip ratio), biochemical tests of fasting blood glucose (FBS), insulin levels; lipid profile (TC, LDL, HDL, TG) and ox-LDL; calculated HOMA. Sample was classified according to waist/hip ratio into: group I with and group II without central obesity.

Results ox-LDL showed significant positive correlation with LDL and TC in all groups of obesity. After adjustment for age and sex, significant positive correlation was detected between ox-LDL with SBP, DBP, TC, LDL, insulin, and HOMA in group II and with TC and FBS in group I. Insignificant association was detected between ox-LDL and other anthropometric parameters including BMI in any group of obese children ($p > 0.05$).

Conclusions ox-LDL, as a marker of oxidative stress is not correlated with BMI among all studied obese children (aged 6–12 years). Increased oxidative stress has causal effects on insulin resistance in obese children without central obesity and on fasting blood sugar in those with central obesity. These findings emphasise the importance of obesity during childhood and suggest that the metabolic complications of obesity and body fat distribution are detectable early in life.

PO-0067 WITHDRAWN

PO-0068 IMPROVING METABOLIC MEMORY WITH INTENSIVE EARLY MANAGEMENT IN NEWLY DIAGNOSED TYPE 1 DIABETES IS A FOUNDATION TO IMPROVE SHORT AND LONG TERM CONTROL

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Objective To demonstrate variations in HbA1C (glycosylated haemoglobin) levels between two paediatric cohorts with different approach in diabetes management in blood glucose monitoring (routine and intense) and insulin administration.

Design A follow up cohort study of 34 children and adolescents in a large district hospital with diagnosis of Type 1 diabetes between 2005 and 2011.

Results In the first group (G1=14), without intensive blood sugar monitoring, mean HbA1C post diagnosis was 8.67% (95% CI 7.87–9.48%). 11 out of 14 (79%) of them had HbA1C above target level (7.5%). In the second group (G2=20) with intensive monitoring, mean HbA1C post diagnosis was 7.87% (95% CI 7.29–8.44%). 12 out of 20 (60%) had HbA1C above target level (7.5%) Table 1

G1a/b: Group without intensive initial monitoring'2005/12

G2a/b: Group with intensive initial monitoring'2011/12

Abstract PO-0068 Table 1

	Mean HbA post diagnosis quarter (95% CI)	HbA >7.5% (Numbers/%)	Reduction of HbA levels achieved (%)
G1a	8.67(7.87–9.48)	11(79)	21
G1b	10.45(9.10–11.81)	13(93)	7
G2a	7.87(7.29–8.44)	12(60)	40
G2b	7.81(7.33–8.29)	13(65)	35

Abstract PO-0068 Table 2

	HbA trends (%)	95% CI (%)
G1(a vs. b)	+1.78	0.24–3.32
G2(a vs. b)	-0.06	0.52–0.40

CI-Confidence Interval

Conclusions No significant difference of HbA in two groups post diagnosis; mean HbA1C level ($p = 0.082$). Comparing the changes over time in the two groups, an increase of HbA1C of 1.78 (Table 2) percentage points in G1 (without intensive monitoring) was significantly greater than the decrease of 0.06 percentage points in G2 ($p = 0.008$).

Discussion Intensive initial management of type 1 Diabetes can significantly reduce future HbA1C. We aim to follow G2 over next 5 years to establish that an improved metabolic memory could reduce HbA1C levels over longer periods.

PO-0069 TERMINAL DELETION OF CHROMOSOME 15Q RESULTING IN HAPLOINSUFFICIENCY OF THE IGF-1 RECEPTOR AND MARKED ELEVATION OF IGF-1

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Introduction Ten to fifteen percent of small for gestational age (SGA) infants demonstrate failure of catch-up growth. Haploinsufficiency of the insulin-like growth factor-1 receptor (IGF1R) gene due to monosomy 15q is an extremely rare cause with 16 cases reported in the literature. We describe the phenotype of such a patient including biochemical findings, auxology and management.

Case A three-year-old female with global developmental delay and autistic spectrum disorder was evaluated for short stature. Height was 82 cm (-3.7 SDS) and weight was 12.5 kg (-1.3 SDS). She was born at term weighing 2.88 kg and was microcephalic and dysmorphic. Array CGH revealed an unbalanced translocation resulting in trisomy of the terminal portion of