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Guest Editors

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ABSTRACTS:

MEDAL AND MEMORIAL LECTURES:

M1. THE IBN-SINA MEDAL LECTURE 2009:

CONTEMPORARY MANAGEMENT OF GRAVES' DISEASE. Mahmoud Benbarka, Endocrinology Division, Department of Medicine, Sheikh Khalifa Medical City, Abu Dhabi, UAE. mbenbarka@skmc.gov.ae

Graves' disease is an autoimmune syndrome characterized by hyperthyroidism, a particular ophthalmopathy, and pretibial myxedema. Usually goiter and symptoms of excessive thyroid hormone are the main features of the illness. The presenting symptoms may be caused by the autoimmunity per se, including the exophthalmos, thyroid enlargement, and the dermal changes. On the other hand, excess thyroid hormone causes thyrotoxicosis, or hyperthyroidism. Thyrotoxicosis of Graves' disease does not differ from that induced by any other cause of excess thyroid hormone. The diagnosis of Graves' disease is usually easily made. The combination of eye signs, goiter, and any of the characteristic symptoms of hyperthyroidism provides strong indication of Graves' disease. Once the question of thyrotoxicosis has been raised, laboratory data are required to verify the diagnosis, help estimate the severity of the condition, and assist in planning therapy. A sensitive TSH assay may be most cost-effective and specific and TSH should be suppressed in significant thyrotoxicosis due to Graves' disease. Serum Levels of T4 and T3 are usually elevated. Measurement of TSH receptor antibody helps confirm the presence of autoimmune Graves' disease. Three forms of primary therapy for Graves' disease are in common use today: (1) destruction of the thyroid by radioactive iodine (2) blocking of hormone synthesis by antithyroid drugs; and (3) partial surgical ablation of the thyroid.

Selection of therapy depends on many factors including adherence to a strict medical regimen, availability of a competent surgeon, undue emotional concern about the hazards of radioiodine therapy. All three methods provide satisfactory outcomes in over 90% of patients. If antithyroid drug therapy is selected, treatment duration is usually 12-24 months with approximately 40% remission rate. Treatment with radioiodine results in permanent hypothyroidism and the need for lifelong thyroid hormone replacement therapy. Surgical therapy is reserved to cases with massive goiters, those who did not respond to antithyroid drugs and fear radioiodine therapy, and those with concomitant thyroid nodules raising suspicion of malignancy.

M2. AL FITOURI MEDAL LECTURE 2009:

PANCREAS AND ISLET TRANSPLANTATION. Elmahdi A. Elkhammas, Department of Surgery, College of Medicine, The Ohio State University, Columbus, Ohio USA. elmahdi.elkhammas@osumc.edu

Even though the first human pancreas transplant was done in the 1960's, the procedure remained behind in comparison to kidney transplantation. In the 1980's it progressed rapidly because of improvement of surgical techniques and advances in immunosuppressive medications. Currently pancreas transplantation has become an option for the treatment of diabetic patients. Simultaneous pancreas-kidney transplantation is actually the procedure of choice for type 1 diabetics with ESRD. At the Ohio State University, we have about 20 years experience with this procedure. We have performed over 800 pancreas transplants. We use a bladder drainage for exocrine function and systemic drainage for the endocrine function of the graft. Others have used enteric drainage and some have done portal drainage. Islet cell transplantation is being promoted to be the procedure of choice one day. It is less invasive. Islet cell transplantation has several issues that kept it in the category of "great idea that does not work" for a long time. My talk will include a general view of pancreas and islet cell transplantation. I will go over our center outcome and review the current thinking of both procedures. I will discuss the indications and the complications and outcome of both procedures.

M3. AHMED HASSUNA MEMORIAL LECTURE:

DIABETES IN PREGNANCY: SCREENING, DIAGNOSIS, AND MANAGEMENT AFTER HAPO.
Amna Salhin, Diabetes and Glandular Disease clinic, San Antonio, Texas USA. asalhin@hotmail.com

Pregnancy is a state of progressive insulin resistance, significant metabolic and hormonal changes in order to ensure adequate fuel supply to the fetus throughout pregnancy. Diabetes in pregnancy includes preexisting diabetes (type 1

and type 2 DM), and gestational diabetes (GDM) which is defined as any degree of glucose intolerance first diagnosed during pregnancy. The prevalence of diabetes in pregnancy has increased as the result of a growing epidemic of obesity and type2 diabetes worldwide. However, there are various differences in guidelines among professional organizations on screening, diagnosis of GDM and on glycemic targets in pregnancy. The Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) Study was designed to ultimately determine what level of glucose during pregnancy should be treated. HAPO Study, an international multicenter epidemiological study, examined the impact of the mother's glucose levels below those diagnostic of diabetes on variable outcomes including birth weight > 90th percentile, primary cesarean section, neonatal hypoglycemia and cord-blood C peptide >90th percentile. Based on the HAPO study, an international group of experts representing several professional organizations is working on developing a worldwide consensus on screening and diagnostic criteria for GDM. Maternal hyperglycemia carries an increased risk of adverse pregnancy outcomes such as neonatal macrosomia and its associated risk of fetal and maternal birth injuries. Specifically women with preexisting diabetes are at increased risk for spontaneous abortion and major congenital malformations. Furthermore, infants of diabetic mothers are at increased risk for obesity and diabetes in later life. Managing diabetes in pregnancy is a significant challenge for physicians and patients. It requires a multidisciplinary team to provide comprehensive diabetes and maternity care including risk assessment, screening for vascular complications. It is crucial that all pregnant women with diabetes have access to health care and the essential resources to optimize diabetes management and achieve the recommended glucose target for GDM and preexisting diabetes.

PLENARY LECTURES:

PL1. KEY NOTE ADDRESS: Prediabetes: How Serious? Tarek Fiad, Department of Endocrinology, Dudley Group of Hospitals, Pensnett Road, Dudley, West Midlands, DY1 2HQ, UK. Tarekfiad@doctors.org.uk

The term prediabetes encompasses events associated with glucose metabolism abnormalities, which take place before the emergence of overt diabetes, namely, impaired glucose tolerance (IGT), and impaired fasting glucose (IFG). The expert committee on the diagnosis and classification of diabetes defined IGT as a post-OGTT glucose level of 7.8 – 11 mmol/L (140 – 199 mg/dL) and IFG as a fasting glucose of 5.6 – 6.9 mmol/L (100 – 125 mg/dL). The relevance of the prediabetic state stems from the association of this condition with vascular complications and the progression to overt diabetes. Accordingly, the metabolic syndrome is also a prediabetic condition. The annual rate of progression of IGT and IFG to diabetes is 8 – 10% and 10 – 12% respectively. Furthermore, the risk of future diabetes increases as the fasting plasma glucose increases within the limits of the conventional normal fasting glucose range. The Australian diabetes, obesity, and lifestyle study (AusDiab) reported greater mortality risk of 50 – 60% in subjects with IGT and IFG, mainly accounted for by increased cardiovascular disease (CVD) mortality. The Framingham Heart Study showed that both the 1997 and 2003 definitions of IFG were associated with increased risk of CHD in women but not in men. Clinical trials have demonstrated the beneficial effect of lifestyle modifications and to a lesser extent, pharmacological agents in the prevention of diabetes and CVD risk factors. Acknowledging that failure of late treatment of diabetes, the case can be made for targeted screening for IFG, IGT, the metabolic syndrome, and diabetes with structured implementation of lifestyle measures. Individuals with prediabetes at a higher CVD risk should also be considered for metformin therapy. Integral part of the intervention in the prediabetic state includes treatment of dyslipidaemia and hypertension to same targets recommended for overt diabetes.

PL2. THE DIABETES PANDEMIC: GENES VS ENVIRONMENT 2009

Don Chisholm, Diabetes and Obesity Research Program, Garvan Institute of Medical Research, The University of New South Wales and St Vincent's Hospital, Sydney, Australia.

There is an alarming worldwide increase in the prevalence of Type 2 Diabetes with Middle Eastern countries having both a high current prevalence and one of the highest rates of expected increase. In fact at the present time six of the ten countries in the world with highest diabetes prevalence are in the Middle East. As genes do not change over the short term in populations, it is clear that environmental factors explain the rapid increase in populations. However, genetic predisposition influences the severity in any given population, and individual susceptibility. Various population studies indicate that physical inactivity; excess energy intake and obesity are the major noxious environmental factors. Worryingly, the increasing prevalence of diabetes and glucose intolerance is associated with a corresponding increase in cardiovascular risk factors i.e. the Metabolic Syndrome – suggesting a future progressive increase in cardiovascular disease.

After decades of difficulty in identifying genetic determinants of ordinary Type 2 Diabetes, the last 3 years has seen an explosion of information generated from large population studies using high-density genotyping arrays ("SNP chips"). There is now solid evidence of about 20 different genetic contributors to Type 2 Diabetes, many of which would never have been suspected on known function e.g., TCF7L2 which is probably the strongest determinant in Caucasian populations. Interestingly the majority of the genetic determinants whose function is known contribute to β-cell dysfunction and only one (PPARγ) to insulin resistance. Thus, the β-cell dysfunction of Type 2 Diabetes is strongly genetically determined while the insulin resistance appears to be generated mainly by environmental factors. However, it is known there is a strong genetic determination of visceral adiposity, so future identification of genetic determinants of visceral adiposity will undoubtedly identify genetic determinants of insulin resistance.
What will we do with this genetic information? Because there are so many genetic determinants with relatively small effects, determination of genetic risk alleles adds little value to standard clinical indicators of susceptibility to Type 2 Diabetes. However, elucidation of the function of some of these genetic determinants will increase our understanding of pathophysiology and may lead to development of new pharmacological agents or an improved ability to tailor therapeutic agents to the individual (pharmacogenomics).
In 2009 we cannot yet change our genes but we can change the environment – more exercise and less calories. Let us do so and lessen the pandemic of diabetes.

PL3. THE ANNUAL LADE LECTURE 2009: HYPERTRIGLYCERIDAEMIA: DOES IT REALLY MATTER?
Hawa Sharief, Endocrine Unit, Department of Medicine, Tripoli Medical Center, Tripoli, Libya.

Hypertriglyceridemia is defined as an abnormal concentration of triglyceride in the blood. According to the National Cholesterol Education Program Adult Treatment Panel (NCEP ATP III) guidelines, a normal triglyceride (TG) level is <150 mg/Dl. Hypertriglyceridemia may be Familial or secondary in nature. Familial Syndromes with High TG include Familial chylomicronemia, Familial combined hyperlipidemia (type IIb), Familial dysbetalipoproteinemia (type III), and Familial hypertriglyceridemia. Secondary causes of High TG should be excluded before diagnose FHTG. Evaluation of patient with elevated triglyceride levels should include Fasting lipid profile, exclusion of secondary causes of High TG and search for other components of the metabolic syndrome:- Fasting hyperglycemia, hypertension, abdominal obesity, and low HDL levels. Other laboratory investigations are TSH, serum urea nitrogen, creatinine, and urinalysis, LFT (before starting medication), Amylase and lipase levels (If there is a clinical suspicion of pancreatitis). Hypertriglyceridemia correlates strongly with the presence of Small, dense particles of LDL-C and Reductions in the HDL2 component of HDL cholesterol, both of which are known to be associated with premature coronary artery disease. Predictor of cardiovascular risk in patients who have hypertriglyceridemia are non-HDL-C= TC- HDL-C:-Measure of atherogenic lipoproteins & Relates to prognosis. High TG/HDL ratio: - Correlates with the LDL phenotype B & relate to prognosis. apolipoprotein B:-Measure of the atherogenic lipoproteins, have prognostic role. Hypertriglyceridemia in an individual with diabetes and metabolic syndrome should never be considered a benign phenomenon.

STATE OF THE ART LECTURES:

SA1. GLYCAEMIC CONTROL AND VASCULAR COMPLICATIONS. Don Chisholm, Diabetes and Obesity Research Program, Garvan Institute of Medical Research;, The University of New South Wales and St Vincent's Hospital, Sydney, Australia.

Both the DCCT study in Type 1 Diabetes and the UKPDS study in Type 2 clearly demonstrated a major reduction in microvascular disease with better glycaemic control. Both of these studies failed to show a significant reduction of macrovascular events at the end of the intervention though there was a borderline reduction for myocardial infarction in UKPDS. However long term post-intervention follow up in both studies showed maintenance of microvascular benefit and a highly significant reduction in macrovascular disease. For DCCT/EDIC this was an approximately 50% reduction in major cardiovascular events and in UKPDS a 15% reduction in myocardial infarction. Disappointingly in the last 12 months we have learned that 3 major glycaemic intervention studies in type 2 diabetes failed to show a significant benefit in macrovascular events; ADVANCE, ACCORD and VADT each showed a small but non-significant reduction (6%, 10% and 12% respectively). Moreover, in ACCORD there was a small but significant increase in mortality, which leads to cessation of the glycaemic arm of the study. It is important to recognise the shorter study duration and difference in the populations between these 3 studies and UKPDS; in UKPDS patients were newly diagnosed while in ADVANCE, ACCORD and VADT the mean duration of diabetes at entry to the study was 8-10 years and in each case glycaemic control had

been relatively poor prior to entry. In ADVANCE and VADT there was some reduction in microvascular disease but this was confined to albuminuria and less impressive than in UKPDS. Thus it would seem that better glycaemic control clearly reduces microvascular disease, but with a greater effect early in the course of the disease. With macrovascular disease, the data also points to a benefit, which is likely to be greater if good glyceamic control is introduced early in the course of the disease, and the benefit may take a long time to become evident. In patients with long-standing diabetes and a heavy burden of disease, it is possible that very aggressive glycaemic control could have an adverse effect on mortality.

Most consensus recommendations continue to favour an HbA1c target of 7% or less, with stricter control desirable in younger patients with shorter duration of the disease, and with less strict targets (e.g. HbA1c 7.5%) in older patients with established macrovascular disease. Of course, glycaemic control should be combined with the proven benefits of cessation of cigarette smoking, blood pressure control, and lipid control.

SA2. RECENT ADVANCES IN THE CLINICAL MANIFESTATIONS AND GENETIC BASIS OF IDIOPATHIC HYPOGONADOTROPHIC HYPOGONADISM, INCLUDING KALLMANN'S SYNDROME. Richard Quinton, Royal Victoria Infirmary, Newcastle Upon Tyne, UK.

Idiopathic hypogonadotrophic hypogonadism (IHH) arises from congenital deficiency (or more rarely resistance to the action) of gonadotrophin-releasing hormone (GnRH). Numerous non-reproductive phenotypic abnormalities can be associated, of which by far the most frequent is anosmia, found in around 50% of cases and constituting Kallmann's syndrome (KS). Historic studies that are unlikely to be replicated have given a male population prevalence of around 1-in-4,000, with female cases being 3-5 times less common. Although affected individuals are necessarily subfertile, which limits the size of IHH kindreds, three separate monogenic Mendelian modes of inheritance were recognised even prior to the molecular biology revolution: X-linked recessive, autosomal recessive and autosomal dominant with variable penetrance. The marked male:female excess initially suggested that X-linked inheritance was likely to underlie the majority of cases. Indeed, *KAL1*, the gene implicated in X-linked KS was the first to be identified; studies of mouse and human fetuses demonstrating that the condition arose from embryonic failure of GnRH neurons arising in the olfactory placode to migrate to the hypothalamus, due to developmental disruption of their migratory scaffold, the olfactory nerve fascicles. Clinical studies of IHH patients had shown that (a) they were unable to complete puberty without exogenous (or stimulated endogenous) sex steroids, (b) the endocrine deficit was lifelong and (c) a primary defect of exogenous GnRH secretion could be inferred from the complete neuroendocrine and clinical responsiveness to exogenous pulsatile GnRH therapy. Over the last decade, these assumptions have been overturned one by one, leading us to appreciate both the fragility and the robustness of the human hypothalamic-pituitary-gonadal axis in different aspects of health and disease. First, it soon became clear that *KAL1* mutations occurred in less than 10% of IHH males, implying that the cause of the male preponderance must lie elsewhere. Second, the discovery that some IHH patients harboured mutations of the gene encoding the GnRH receptor meant that the syndrome could result not just from deficient GnRH secretion, but from resistance to its action. Earlier investigators had been misled by their ability to overcome this defect and stimulate gonadotrophin secretion with supraphysiological doses of GnRH. The number of genes known to cause IHH is now nearly in double figures (with over 50% of cases still of unknown genotype). Intriguingly, similar gene defects can result in widely varying reproductive and non-reproductive phenotypes, both within and between kindreds. Thus, the phenotypic distinction between anosmic (KS) and normosmic forms of IHH is becoming increasingly irrelevant to the new genetic reality. Moreover, in a proportion of cases there is remarkably found to be more than one heterozygous gene mutation, different mutant alleles of the same gene or of entirely separate genes. As more genes continue to be discovered and more patient DNA is screened, such oligogenic IHH cases are likely to become increasingly recognized. Yet unrecognized second or even third allelic mutations might thus explain some of the phenotypic diversity in IHH patients harboring identical single gene mutations.

The implication of oligenic inheritance is that a variety of mutant IHH alleles is present in the normal population at sufficiently high frequency for successful mating to occur between carriers. The question therefore arises as to what effect, if any, a single mutant allele might exert on an individual carrier? Likely impacts would be on timing and tempo of pubertal onset, and propensity to develop reproductive axis shutdown in the face of critical illness, chronic disease, strenuous exercise, prescribed or abused drugs (opiates, androgens, and anabolic steroids) and caloric restriction. This finding may also underpin the biological basis of two striking new reproductive variants of IHH and KS described in the last 10 years, reversal of IHH and adult-onset IHH. In the former syndrome, a patient with apparently classical KS is seen

to undergo complete normalization of the hypothalamo-pituitary-adrenal axis, sometimes even decades after their original diagnosis. In the latter syndrome, a patient who underwent normal puberty, with full development of secondary sexual characteristics and of proven fertility, inexplicably undergoes complete reproductive axis shutdown in later life. Both these variants of classical IHH have been associated with genetic mutations.
In summary, a combination of genetic research and careful longitudinal clinical phenotyping over the past decade has significantly advanced our knowledge of IHH & KS, overturning a number of long-cherished assumptions in the process. This new information has the potential to also transform our understanding of normal human reproductive development.

SA3. HYPOGLYCAEMIA IN DIABETES: A CLINICAL REVIEW. Salem El Habroush, Tripoli Diabetes Center, Faculty of Medicine, Tripoli, Libya.

Intensive treatment regimen to control hyperglycemia was proven to be of more benefit to prevent the development of complication of diabetes, especially microvascular complication, compared with conventional treatment regimens. One of the barriers for intensive blood glucose control is the development of hypoglycemia, which is found to be 3 times more common in people on intensive treatment regimens for controlling their blood glucose compared with people on conventional treatment regimens. Hypoglycemia is an important acute complication of diabetes and it may lead to a variety of symptoms and if left untreated may leads to disturbance of cognitive function and coma. There are at least four physiological lines of defense to counteract hypoglycemia, in order to increase the blood glucose and to maintain the glucose within ranges close to normal. These counter-regulatory mechanisms might be affected in one way or another in people with diabetes, especially those with T1DM, may lead to defect of counter-regulation and the possibility for the development of hypoglycemia unawareness, and HAAF, in this presentation the method of evaluation of this important clinical situation as well as its treatment will be discussed.

ABSTRACTS OF CLINICAL SYMPOSIA:

SYMPOSIUM 1: ADVANCES IN DIABETES MANAGEMENT

S1.1 "ETHICS" AND "ETIQUETTE" IN DIABETES CARE. Soad Bosseri, Department of Diabetes and Endocrinology, Tripoli Medical Center, Tripoli, Libya

The human touch is being forgotten in modern medicine as the balance is increasingly tilted in favor of technology. The human relationship comes with the patient comes before the professional responsibility; especially on dealing with people with chronic diseases such as diabetes. Healthcare professionals who do not themselves live each day with diabetes they may not have a true understanding of what happens on a daily basis to manage this disease. They sometimes do not know right thing to say to their patients or how to deal with their problems. Care of people with diabetes requires tact, understanding, and consideration; these are tricky skills to master because every person with diabetes has different opinion. They involve respect the right of the patient regarding choice of treatment, privacy, access to information, informed consent, and protection from nosocomial infections and providing psychological support to the family because they are also victims of the illness because they are anxious and worried. The healthcare professional must be cheerful, merciful, kind, clean and dress appropriately, to look serious, organized, and disciplined. The patient who gets psychological and social support is more likely to be cooperative in taking medications, eating or drinking, therefore making diabetes easier to handle can be accomplished just by having good diabetes etiquette.

S1.2 NEWER ORAL ANTI-DIABETIC DRUGS: INCRETIN-BASED THERAPY. Kamal Abouglila, Endocrine & Diabetes Unit, University Hospital North Durham, United Kingdom. abouglila2000@yahoo.com

Type 2 diabetes is a chronic metabolic disorder with progressive beta-cell dysfunction, impaired insulin actions, and various other abnormalities. Glucagon-like peptide 1 (GLP-1) is an incretin hormone released by the gut in response to meal intake that helps to maintain glucose homeostasis through coordinated effects on islet alpha- and beta-cells, inhibiting glucagon output, and stimulating insulin secretion in a glucose-dependent manner. Biological effects of GLP-1 include slowing gastric emptying and decreasing appetite. In type 2 diabetes, there is progressive decline of these incretins level, GLP-1, and glucose dependent insulinotropic polypeptide (GIP). These peptides are rapidly degraded by endogenous proteases, dipeptidyl peptides-4 (DPP-4) giving a very short half-life of 2-3 minutes. Currently available anti-diabetic drugs do not address these arms of glucoregulatory dysfunction of type 2 diabetes. Incretin mimetic are a new

class of anti-diabetic medication that mimics the actions of GLP-1. They exhibit several properties, including glucose-dependent stimulation of insulin secretion, suppression of glucagon secretion, slowing of gastric emptying and induction of satiety, which result in improvements in glycemic control with weight loss in patients with type 2 DM. DPP-4 resistant incretin mimetic (e.g. exenatide, liraglutide) that have been developed by substitutions in the polypeptide chain. DPP-4 inhibitors incretin enhancers (e.g. sitagliptin, vildagliptin) prevent the degradation of endogenous GLP-1 and GIP, thereby potentiate their actions. Recent 3 years data with exenatide, have demonstrated long-term sustained reductions in haemoglobin A1C with progressive weight loss. Glycemic and weight benefits were also recently reported in the LEAD trial with administration of liraglutide. Incretin mimetic significantly lower haemoglobin A1C, body weight, and postprandial glucose excursions in humans and significantly improve beta-cell function in vivo (animal data). Incretin enhancers have generally neutral effect on weight and improve glycemic control. SUMMARY: The ability of incretin mimetic and enhancers to improve glycemic control and reduce body weight is a unique property that fills an important void in the treatment of patients with type 2 diabetes.

S1.3 ADVANCES IN INSULIN THERAPY 2009. Mohsen Eledrissi. Department of Medicine, Division of Endocrinology, National Guard Medical City, Eastern Province, Saudi Arabia. eledrisim@ngha.med.sa

Type 2 diabetes is characterized by both insulin resistance and insulin deficiency. As diabetes progresses, most patients will require drug therapy including insulin to achieve adequate glycemic control. Too often, insulin therapy either is delayed or is suboptimal. The choice of an insulin regimen depends primarily on glycemic control. It is important to recognize the glucose-lowering power of the various hypoglycemic drugs; this will influence the physician's decision on adding oral drugs or considering insulin. Timely interventions and transition to other drug classes with early initiation of insulin therapy if needed is paramount to achieve glycemic targets. If HbA_1c is less than 10 %, adding a once- daily injection of basal insulin (intermediate- or long-acting) to current oral hypoglycemic agents is a simple and occasionally an effective strategy. For patients who do not achieve glycemic targets on once-daily insulin and those with baseline HbA_1c level of more than 10 %, an insulin regimen that involves basal and prandial components (premixed or split-mixed) is usually required. Intensive insulin therapy using multiple daily injections may be required in certain patients. Insulin analogues-prandial and basal- offer less incidence of hypoglycemia, but at a higher cost and no proven benefit in terms of glucose control. In addition to levels of HbA_1c, patient's lifestyle, comorbid conditions, competence, and personal preferences should be considered in determining the choice of insulin therapy.

SYMPOSIUM 2: MODERN MANAGEMENT OF PITUITARY TUMORS

S2.1 IMAGING OF THE PITUITARY GLAND: TIPS TO THE CLINICIANS. Abdel Salam Abougrara, Tripoli Medical Center, Tripoli, Libya.

(No abstract submitted)

S2.2 Contemporary Management of Prolactinomas. Salah Gwaider, Department of Medicine, Tripoli Central Hospital, Tripoli, Libya.

(No abstract submitted)

S2.3 ACROMEGALY 2009. Tarek Fiad, Department of Endocrinology, Dudley Group of Hospitals, Pensnett Road, Dudley, West Midlands, DY1 2HQ, UK. Tarekfiad@doctors.org.uk

Acromegaly is a rare disease associated with premature mortality, predominantly from cardiovascular disease. It is invariably caused by a GH-secreting pituitary adenoma and results in disabling systemic symptoms, the severity of which tends to be higher in patients with longer disease duration. Disease control is achieved with normal IGF-I level (normalized for age and gender) and Nadir GH < 0.25 mg/L during OGTT (employing sensitive GH assays). Transsphenoidal surgical resection remains the treatment of choice for patients with somatotroph adenomas, which are small, large but still respectable, or large and cause visual impairment. Surgery can also be considered for large tumors to provide debulking of at least 75% of initial tumor size. Patients' with microadenoma achieve a remission rate of 60 – 90% with less favorable outcome in macroadenomas, remission rate 40 – 60%. Medical therapy with long-acting somatostatin analogues is offered to patients with persistent disease following surgery, which was shown to control 60 – 70% of patients when given as an adjuvant therapy after unsuccessful surgery. Primary medical treatment with long-acting somatostatin analogues can be considered for patients not fit for surgery or as an alternative to surgery for

tumors that are too large for complete resection. Data showed 45% normalization of IGF-I and similar rate of tumor shrinkage. Dopamine agonist therapy can be used in a combination with somatostatin analogues but is not effective as a single treatment. The new GH-receptor antagonist, Pegvisomant controls IGF-I in up to 97% of patients resistant to previous treatment modalities. Radiotherapy is reserved for patients with continued increase in adenoma size despite previous surgical and medical therapies. Systemic evaluation includes colonoscopic surveillance and comprehensive control of cardiovascular risk factors.

S2.4 CUSHING'S DISEASE: INVESTIGATIONS AND MANAGEMENT. Fellani A. M. Zwei,Regina General Hospital, Regina, Saskatchewan,Canada. fellani55@hotmail.com

Cushing disease, was initially described by Harvey Cushing in 1912 in his book entitled "The pituitary body and its disorders" still the most common cause of spontaneous or endogenous Cushing syndrome. It is almost always caused by solitary (probably monoclonal) corticotroph adenoma in about 65-70% of all the cases of Cushing syndrome. The vast majority of these tumors are intrasellar Microadenomas (<1cm in diameter) rarely Macroadenomas, in <10%, the cause of these tumors remains unknown. In this disease hyper secretion of ACTH by the pituitary adenoma leads to excessive production of cortisol from the adrenal cortex, this in turn will lead to the usual musculoskeletal and metabolic abnormalities. Unlike the normal pituitary corticotroph, the cells of these pituitary adenomas are relatively resistant to feedback inhibition from hypercorticolism. The duration as well as the severity of hypercorticolism may lead to inappropriate interpretations of the biochemical results, which may lead to inaccurate diagnosis and management. An over view of how to establish the diagnosis of Cushing syndrome, with more focus on the diagnosis and differential diagnosis of Cushing disease will be discussed. The limitation of different laboratory values and the overlap between the normal and abnormal ones, the different anatomical and functional imaging modalities, and finally appropriate strategy of management and follow up will be also discussed.

SYMPOSIUM 3: CURRENT PERSPECTIVES IN PEDIATRIC DIABETES AND ENDOCRINOLOGY

S3.1 DIABETES MELLITUS IN CHILDREN; NOT JUST TYPE 1… Asma Ali Deeb, Imperial College London Diabetes Center, Abu Dhabi, UAE.

In the recent years, it has been recognized that a child presenting with diabetes is not necessarily a type 1 diabetes mellitus (T1DM) patient. Various forms of monogenic diabetes have now been recognized. This has been due to the advances in molecular genetics, which have led to identification of genes associated with many clinically identified subgroups of diabetes. The identification of genes has explained clinical heterogeneity of many conditions with special features of diabetes. The main examples experienced in Paediatrics are; neonatal diabetes and maturity onset diabetes of the young (MODY). In addition to monogenic diabetes, type 2 diabetes mellitus (T2DM) in children and adolescent is becoming an increasingly important public health concern throughout the world. Because of the relatively recent recognition of the problem in this age group, many children with new-onset T2DM may be misclassified as having T1DM. Conversely, as the population becomes heavier, overweight adolescents with autoimmune diabetes may be misdiagnosed as having T2DM. The main emphasis of the talk is to discuss various types of diabetes in children and adolescents and to highlight the special characters of individual diabetes subtype in this population.

S3.2 ENDOCRINOPATHY IN NON ENDOCRINE DISEASES. Faten Ben Rajab, The Diabetes and Endocrine Unit, Al Jala Children's Hospital, Tripoli, Libya.

Clinical endocrinology has rapidly expanded from a minor field devoted to small group of ductless glands to embrace a whole variety of clinical entities, and seems ever ready to incorporate very much more. Endocrinopathy is a disorder of an endocrine gland with associated disturbances of hormone levels and secondary physiological effects. So there are many diseases with variety of clinical manifestations including endocrine symptoms & signs, there is strong link between endocrine systems and autoimmunity, also many studies have shown that genetic factors involved in host susceptibility to autoimmune diseases. Celiac disease one of autoimmune diseases affecting small bowel associated with endocrine disorders as type one diabetes ,hypothyroidism, both hypoparathyrodism & hyperparathyroidism, Addison's disease and infertility. Arthropathy can be a manifestation of endocrinopathies, musculoskeletal signs and symptoms frequently occur in endocrinopathies. Appreciating these clinical Connections will help clinicians avoid misdiagnosis of primary rheumatic diseases and can lead to prompt treatment of a primary endocrine disorder. Slipped capital femoral epiphysis may be associated with hypothyroidism and other endocrinopathies, patients who are on or below tenth percentile for

height at the time of presentation should be screened for a possible endocrine abnormality using measurement of TSH&FT4 as screening test. Chronic mucocutaneous candidiasis does not represent specific disease, but rather a phenotypic presentation of a spectrum of immunogenic, endocrinologic, and autoimmune disorders. The incidence of endocrinopathies in pediatric brain tumor patients is high even before radiotherapy and including patients with tumors not adjacent to the hypothalamic pituitary unit, base line endocrine function should be determined for brain tumor patients before therapy. Neurodegenerative diseases correlate to multiple endocrinopathies due to identification of a large-scale mitochondrial DNA deletion; it should be considered in those who presented with uncommon endocrinopathies with neurological disorders. Iron overload associated endocrinopathies and low bone mass are most frequently reported complications of chronic transfusion therapy in patients with thalassemia (less frequent in SCD). Hypothyroidism is commonly seen in congenital nephrotic syndrome patients due to reduction of serum TBG secondary to massive proteinuria, this condition reversed after nephrectomy. Polyglandular autoimmune syndrome comprises a group of autoimmune disorders of endocrine glands and failure to produce their hormones. Patients could present with other systemic diseases as pernicious anemia, vitiligo, celiac disease, & rheumatoid arthritis. Conclusion: Endocrinopathy associated with many other diseases, it is built upon understanding basic scientific principles so no surprise to witness the rapid union of endocrinology and molecular biology which answer many scientific questions addressed in clinical endocrinology.

S3.3 GROWTH HORMONE DEFICIENCY IN CHILDHOOD AND ADOLESCENCE: DIAGNOSIS AND MANAGEMENT. Abdel Hadi Mohamed Habeeb, Endocrine & Diabetes Unit, Madinah Maternity & Children Hospital, Madinah, KSA.

When a short child referred to clinic, a question is often raised on whether he/she could have growth hormone deficiency (GHD). Although GH plays a central part in linear growth the majority of short children are not GH deficient. Out of the endocrine causes of short stature, GHD is the commonest. The condition has multiple aetiology and wide clinical spectrum. It can be either isolated or part of multiple pituitary hormone deficiency (MPHD). The diagnosis of GHD is based on a combination of auxological and clinical criteria and confirmed with biochemical evidence of abnormal GH secretion. MRI imaging of the hypothalamic pituitary area can also be a useful diagnostic tool. Children with severe GHD often present early with short stature, typical phenotype, and evidence of MPHD making the diagnosis rather simple. Those on the mild end of the spectrum can be a challenge. Once the diagnosis has been made patients should be commenced on daily SC injections of recombinant GH and have regular follow up by a paediatric endocrinologist. The aim of treatment is to normalize growth during childhood and achieve acceptable final height. The side effects of GH therapy are very rare and can be easily detected by clinical evaluation. As the diagnostic criteria and treatment strategy of adult GHD is different not all individuals with childhood GHD will automatically receive GH during adulthood. Once children with GHD reach, their final height further assessment is needed to decide whether treatment into adult life is needed. In order to achieve the maximum somatic and metabolic benefits of GH treatment, both paediatric and adult endocrinologist need to ensure that adolescents with GHD have proper hand-over and smooth transition to adult care. The presentation will provide an overview of GHD with more focus on the diagnosis and treatment.

ABSTRACTS OF FREE COMMUNICATIONS:

ORAL COMMUNICATIONS:

OC1. A PREDICTION RULE TO IDENTIFY ADULTS WITH DIABETIC KETOACIDOSIS WHO ARE AT INCREASED RISK OF DEATH. Rafik R Elmehdawi, Department of Medicine, Al-Arab Medical University and Benghazi Center for Diabetes and Endocrinology, Benghazi, Libya.

Background: according to the American Diabetes Association (ADA), the severity of diabetic ketoacidosis (DKA) is determined by the pH level, bicarbonate level, and mental status. The association recommends that severe DKA should be managed at intensive care unit as it carries a higher risk for mortality and complications. However, this method in defining the DKA severity classifies patients according to the degree of acidemia only and it does not take in account other operating factors that might put patients at an increased risk of death regardless of their arterial pH level. These risk factors include; older age, coexisting diseases, the degree of dehydration and hemodynamic instability. **Aims and Objectives:** the purpose of the study was; to develop a simple and precise prediction model for DKA severity in order to identify DKA patients who are at a higher risk for mortality. **Patients and Methods:** the author derived a prediction rule that stratifies patients into two groups with respect to the risk of death during hospitalization. Then it was applied to 225 randomly selected adult patients with diabetic ketoacidosis who were admitted to different Benghazi hospitals

during the period 2004 -2007. The prediction rule allocates points based on eight variables; age, duration of diabetes, the presence of co-morbidity, abnormal vital signs (pulse rate of ≥100 per minute, systolic blood pressure < 100mmHg or diastolic blood pressure < 60mmHg), and abnormal laboratory results (pH <7, serum urea ≥ 50 mg per deciliter). The patients were then stratified into 2 risk groups as regard to mortality; low risk group (non severe DKA) which includes patients who scored < 4 points and high risk group (severe DKA) which includes patients who scored ≥ 4 points. **Results:** Mortality ranged from 0% in patients who scored less than 2 points to 100% in patients who scored 8 points. There was a significant difference in mortality among the low and high risk groups (5.45% vs. 51.6%, p= 0.000). the new prediction rule showed 77.5% sensitivity and 84.4% specificity in predicting patients` mortality which were significantly better than the sensitivity (45%,p=0.002) and specificity (80.2%, p= 0.000) of using arterial Ph and mental status as prediction means . The positive predictive value of the new scoring system was significantly better than that of arterial Ph (51.6% vs. 36%, p= 0.000). **Conclusions:** The described prediction rule identifies patients with diabetic ketoacidosis who are at high risk for death more accurately than the arterial pH. This prediction rule may help physicians make more proper decisions about managing their patient.

OC2. ASSESSMENT OF DIABETES PATIENT EDUCATION IN BENGHAZI. Suhair Jaber Aubaker and Ahmed Swalem. Department of medicine, 7th October Hospital, Benghazi Libya.

Background: Since diabetes is a lifelong disorder, diabetes education for patients and their families is probably the most important obligation of clinicians providing a diabetes patient's medical care. Knowledge about the illness and how to deal with will have great impacts on the outcomes and prognosis. **Objectives**: 1) To evaluate how much knowledge diabetic patients have about their illness. 2) Identify areas of deficiencies in order to help design the most effective diabetic patient education programs to prevent further morbidity, improve quality of life and to use resources efficiently**. Method and Patients Characteristics**: a total of 360 diabetic Patients were interviewed during the period of January through September 2006. (47% males and 52% females), mean age 54y±12.55, mean duration of diabetes 10.6y±9.7 range from (1y – 33y). About 60% of females were homemakers, and 54% of males were retired. Results: Patient's level of education; primary school (30.5%), secondary school (12.7%), high school and university (8.8%), illiterate (47.7%). Awareness of hyperglycemic symptoms: the most common three known hyperglycemic symptoms were general fatigue (78%), polyuria (56%), and polydipsia (50%). while delayed wound healing was only known to (4.4%) and about 20% of the studied group could not name any hyperglycemic symptoms. Awareness of hypoglycemic symptoms: the most common three known hypoglycemic symptoms were shivering (46%), inability to concentrate (44%), and sweating (36%). The least two known features of hypoglycemia were blurred vision (8%), confusion (7%), and about (14%) of patients did not know any hypoglycemic symptoms. Diabetic diet knowledge: only 58% of patients were trying to control their diet. Knowledge of diabetes medications and insulin: 38 patients were familiar with insulin only while 236 patients knew insulin and oral hypoglycemic agents. 198 (72%) of them knew the injection technique, and (71.8%) inject themselves, of those (9%) keep insulin unrefrigerated. Follow up visits: (87%) of patients visit diabetic clinic regularly (monthly), (41%) check blood pressure (BP) regularly, 19% visit eye clinic regularly, and 21% regularly visit a dentist. Foot care education: 71% of patients did examine their feet daily, 6% infrequently and 23% never checked their feet. About 84% wear perfect shoes. Pregnancy and diabetes: (96%) of the total 170 female patients in the study agreed for the need of diabetes control before conception, and were aware of increased risk of fetal malformation and complicated labor with uncontrolled diabetes. 134 of them (78%) knew that there is a difference between preexisting diabetes and gestational diabetes (GDM) and 118 patients (69%) selected insulin for diabetes treatment during pregnancy while (16%) 52 selected OHA. **Conclusion:** Poor diabetes education was strongly seen among diabetic patients regarding symptoms, complications, foot and dental care and follow up visits. Implementing diabetic patient education programs that are easy for patients and their families to understand and follow could effectively improve the knowledge among Libyan diabetics in Benghazi.

OC3. THE PREVELANCE OF DEPRESSION IN ADULTS WITH DIABETES MELLITUS. Monsef Alokali*, Omar El Shourbagy and Samir Wasef***, *Internal Medicine, **Family and Community Medicine Dep. Faculty of Medicine, Omar Almukhtar University, Libya. *** Community Medicine Dep. Faculty of Medicine, Zagazig University.**

Background: Diabetes mellitus has a strong association with the presence of depression. Depression occurs at high rates among individuals with diabetes mellitus and several studies suggest that diabetes doubles the risk of depression. The

rate of complications in diabetic patients may be increased with comorbid depression. **Objective:** To assess the severity of depressive symptoms in adult diabetic patients using Beck Depression Inventory (BDI). **Methodology:** This descriptive study consisted of 88 diabetic patients (age 23–72 years) attending the diabetes outpatient center of Derna, Libya from June 2008 to December 2008. Depressive symptoms were measured using the Beck Depression Inventory, Arabic version by Ibrahem and Alansary (2001). **Results:** The mean age of patients was 52.18 + 13.7 years, 59.1% were males. Severe depression score was diagnosed in 16 (17.4%); moderate depression score in 36 (40.9%); and mild normal depression score in 36 (40.9%) of diabetic patients. More men than women with diabetes reported symptoms of severe to moderate depression (61.5% vs. 35.7%, $P < 0.01$). A significant association was found between depression and complications of diabetes ($r = 0.29$, $P < 0.01$). All patients with severe depression score (17.4%); had retinopathy and neuropathy complications. **Conclusion:** The prevalence of depression in Derna diabetics is high. There is growing recognition within the medical community that depression has a strong influence on health outcomes. Screening for depression in patients with diabetes is recommended.

OC4. OUTCOME OF GROWTH HORMONE THERAPY IN SHORT STATURE CHILDREN. Faten Ben Rajab, Naima Dafer and Zeinab Hashieshi, Endocrine clinic, Tripoli Children's Hospital, Omar Mokhtar Street, Tripoli, Libya.

Background: Growth hormone (GH) deficiency can be managed successfully by growth hormone, which was first introduced in 1958. **Aims:** This study aimed to determine the effect of a one-year therapy of growth hormone on the height, to determine short-term side effects of the drug, and what is the best therapeutic dose to achieve normal height velocity. **Material and Methods:** Review of all medical records of patients that presented with short stature and treated by growth hormone in the endocrine clinic at Tripoli children hospital (2000 – 2004). **Results**: all treated patients with therapeutic doses of growth hormone had good response. They achieved mean height velocity of 7.45 cm/year, the best response was to the doses range from 0.7 to 1 unite / kg weekly in divided doses. There were no short term side effects for GH use. **Conclusion:** Growth hormone is safe for treating children with short stature. The best therapeutic dose ranges from 0 .7 to 1 unit/k /.week in divided doses. **Recommendations**: the long-term side effects of the use of rhGH should be explored through future studies.

OC5. FLEXIBLE INSULIN THERAPY IS MORE USEFUL THAN CONVENTIONAL INSULIN THERAPY IN GLYCAEMIC CONTROL OF TYPE 1 DIABETIC CHILDREN: THE AL-JAL HOSPITAL EXPERIENCE. Faten Ben Rajab, Salha Gliwan, Asma Moktar, Endocrine clinic, Tripoli Children's Hospital, Tripoli, Libya.

Back ground: The management of type 1D.M has changed dramatically over the past 40yrs in particular, new insulin strategies have improved the ability to maintain near normoglycemia. Aims: This study aimed to compare two different strategies of insulin therapy(twice daily insulin & multiple daily injections) by assessment of patient's growth , level of HbA1c and occurrence of acute complications in each group (DKA & hypoglycemia). Patients and method: Retro-prospective study includes the patients who were diagnosed as type 1 D.M for at least 1yr at endocrine clinic in Tripoli children's hospital (2006- 2007), patients who were on twice daily insulin injections & who changed to MDI after 1-4yrs on twice daily insulin were reviewed. Also we choose others with poor diabetic control (HbA1c > 9) all were switched to MDI 3 doses of Premeal insulin (Regular insulin) & 2 doses of basal insulin asNPH or 1dose as glargine; then All followed up for 1 yr. Insulin dose was 0.7-1.5 u/k/d according to requirement of individual patient & dietary advice was given at most of clinic visits. Data were analyzed according to Wt , Ht ,BMI, Hb A1c and occurrence of hypoglycemia(either symptomatic or if BS <60 mg/dl) or DKA. (We considered that HbA1c <7% tight control, 7-8 accepted,>9 poor control). Results: Our patients were 50, out of them 48% have diabetes for 1-4 years, 48% are diabetic for 5-8 years,& 4% are diabetic for 9-12 years, their age ranged from 2 to 16 years with mean 8.2 + 3.2. We found the age when those patients require switching on MDI ranged from 8 to 16 with mean 13 + 2.4. Seventy percent of them had good growth by normal BMI (mean 20 + 3.6), 24% were overweight &6% under weigh on MDI Therapy. Regarding Mean HbA1C on twice daily therapy is 10.4+2% & mean of HbA1c on MDI therapy is 7.7%+1% which is statistically significant (p=0.000). The occurrence of hypoglycemia reduced from 26% with twice daily injections to only 2% with MDI. There is no patients suffered from DKA on MDI therapy compared to 14% of patients developed DKA on twice daily insulin therapy. Conclusion: Most of our patients who were receiving MDI therapy have optimal growth , better glycaemic control (better HbA1c) and less occurrence of hypoglycemia , no DKA was recorded. Recommendation: Offer MDI for those children who are older than 8yrs with poor control diabetes on insulin therapy to achieve good control and to avoid complications.

OC6. A LIBYAN FAMILY WITH FAMILIAL HYPERCHOLESTEROLEMIA. Maisoon N EL-Hemri (1), Rafik R Elmehdawi (2), Fawzia AL-Fazani(3) and Nuri M Shembesh (4), 1Medicine department, 7th October teaching hospital, 2 Department of Medicine, and 4 Department of Pediatrics, Al-Arab Medical University and 3 Al-Fatah Pediatrics Teaching Hospital, Benghazi, Libya.

Introduction: Familial hypercholesterolemia is the deficiency of the low-density lipoprotein receptors resulting in hypercholesterolemia leading to atherosclerosis, corneal arcus, xanthelasma and tendon xanthomas. Patients die prematurely especially from myocardial infarction. Homozygous familial hypercholesterolemia is a rare disorder, affected children have no low-density lipoprotein receptors in the liver and they have extremely elevated low density lipoprotein cholesterol concentration and massive deposition of lipid in the arterial walls, and the skin. **Case report:** A 12 years old Libyan male child was admitted to the pediatrics teaching hospital in Benghazi with huge, ugly looking, non-painful swellings in the extensor surface of the joints. The child had these swellings since the age of three, in which they enlarge gradually and never disappeared. Both his father and his late brother had the same swellings, his brother died suddenly at the age of 7 years and autopsy showed that he had hepatomegaly. His two grandmothers are sisters and both of them have hyperlipidemia, one died because of coronary artery disease. The child examination was unremarkable apart from firm, non tender swellings over the extensor surface of the joints. His laboratory investigations were normal apart from his fasting lipid profile, which was as following: total cholesterol 673 mg\dl, Low density lipoprotein: 519 mg\dl, triglycerides: 182 mg\dl and the High density lipoprotein: 52 mg\dl. His abdominal ultrasound, cardiac echogram and stress electro cardiogram all were normal. **Comment:** We reported a rare and potentially fatal familial disease. Not enough attention was paid to this from both the patient/family and the physicians. Thus, familial hypercholesterolemia would not have been recognized if not for the cosmetic concerns of the patients himself about the clinical stigmata of his disease.

POSTERS PRESENTATIONS:

P1. BLOOD GLUCOSE CONTROL AND ITS EFFECT ON PATIENTS OUT COME IN A MEDICAL ICU. Adela Elamami and Zaid H. Department of Medicine, Faculty of Medicine, Al-Arab Medical University, Endocrine Unit and Intensive Care Unit , the 7th of October hospital Benghazi , Libya.

Background: Until the end of the past millennium relatively little attention was given to the control of blood sugar level in critically ill patients. Despite the controversy in the results of many trials regarding tight blood sugar control (110mg/dl) as a goal in medical ICU, most of these trials address the bad effect of hyperglycemia on outcome. **Objective**: To assess the outcome of patients in relation to blood sugar control, and to the effect of diabetes and dexamethasone therapy in medical ICU setting. **Material & Method**: a prospective follow up study involving all patients admitted to medical ICU of 7th October hospital for more than four days during the period of January- November of 2008 regardless of their APACHE score. These patients were assessed in regards to their age , sex , history of DM , blood sugar at admission , mean fasting or early morning blood sugar , mean of all blood sugar reading , number of hyperglycemic attacks (=/>200mg/dl) ,number of hypoglycemic attacks (<50mg/dl), dexamethasone therapy , duration of stay , and their outcome .Data were analyzed using a statistical package of the social science SPSS version 11. **Results**: a total of 101 patients were studied (48 males), 52 patients were diabetics, and 55 were on dexamethasone therapy. Only 3 patients were treated with intravenous insulin infusion as their diagnosis was DKA; all other patients were on conventional sliding scale insulin. Blood sugar usually measured 6 hourly for DM and 12 hourly for non DM. the mean admitting blood sugar for all sample was 165.2+/-110SD , mean age 63+/-17SD , mean fasting blood sugar 138.9+/-53SD, the mean of all blood sugar readings 151+/-57SD, mean number of hyperglycemic and hypoglycemic attacks 5.7+/-9SD, and 0.6+/-1.3SD respectively. No significant difference in mean age, duration of stay, and number of hypoglycemic events among diabetics and non diabetics, survived versus died groups, and dexamethasone versus non dexamethasone treated patients. However, there was significant difference in mean fasting blood sugar between diabetics(167+/-54SD) and non diabetics (109+/-33 SD) (P=0.001) , survived 120.6+/-39SD versus died 143+/-55SD (0.04) and dexamethasone treated 152.4+/-57SD/non dexamethasone 122.7+/-44SD(p=0.004), and in mean of all sugar reading DM 185+/-54SD versus non DM 114.6+/-32SD (p=.000), survived 127.3+/-43SD /died 156.2+/-59SD (0.01), dexamethasone 164.8+/-59SD / non dexamethasone 134+/-50SD (0.005), and in number of hyperglycemic events for DM 10.4+/-10SD /non DM0.8+/-1.5SD(0.000),survived 3.2+/-5SD/ died 6.3+/-9SD (0.04) in), dexamethasone group 7.3+/-10SD / non dexamethasone group 3.9+/-7SD (P= 0.03). The survival rate among diabetic group was 13.4% / non

DM 24.5%, and among patients on dexamethasone 12.7% /non dexamethasone 26.1%. **Conclusion**: blood sugar control does affect the outcome of patients in medical ICU. Diabetes and dexamethasone therapy both decrease survival rate in patients admitted to medical ICU for more than four days and treated with conventional sliding scale insulin. We need more attention to blood sugar control and monitoring.

P2. THE EFFECT OF SITAGLIPTIN IN SUBOPTIMALLY CONTROLLED TYPE 2 DIABETES PATIENTS ON METFORMIN: A MIDDLE EASTERN EXPERIENCE. Ali B Khalil, Salem A Beshyah, Mahmoud M Benbarka and Jeanette DaBell. Center for Diabetes and Endocrinology, Sheikh Khalifa Medical City, Abu Dhabi, UAE.

Background: Commonly used anti-diabetic medications such as sulfonylureas, thiazolidinediones and insulin are all associated with significant weight gain. However, newer agents such as incretin-based therapies reduce HbA1c without weight gain. The first oral DPP-inhibitor, Sitagliptin, became available in the UAE shortly after its approval by the FDA in 2006. **Objectives:** Because of its cost and unknown long term safety profile, we elected to gather our own local experience and to observe retrospectively the impact of Sitagliptin added to failed Metformin therapy. **Subjects and Methods:** We assessed the glycaemic control and changes in body weight of 53 patients with type 2 diabetes who failed to reach their HbA1c target of <7% on Metformin therapy. Sitagliptin 100 mg daily was added to Metformin. We retrospectively examined the changes produced in HbA1C and body weight. Patients with combinations other than Metformin and Sitagliptin were excluded. The mean age of patients was 52 (range 21-73) years. They were 26 men and 27 women. Mean duration of diabetes was 8 years. **Results:** Mean values of HbA1c, Body Weight, and BMI (Average (sd)), before, and after treatment are shown in the table both for the whole sample and for those patients with at least one non-missing value before and one non-missing value after treatment.

Parameters	Patients with non-missing values (n=44)		All patients (n=53)	
	Before	After	Before	After
HbA1c	8.3 (1.5)	7.7 (1.2)	8.2 (1.6)	7.8 (1.4)
Body Weight	88.0 (18.8)	87.1 (19.3)	88.3 (18.6)	87.0 (18.6)
BMI	33.8 (7.7)	33.5 (8.1)	33.6 (7.3)	33.1 (7.5)

From the repeated measurements analysis, the mean difference in HbA1c between before treatment and after treatment is 0.56% (95% CI 0.24% - 0.87%). This estimate did not changed by adding body weight, BMI and duration of diabetes to the model. The trend in the drop HbA1c was statistically significant and there was a trend for weight reduction similar in time to HbA1c the drop in weight. **Conclusion:** In our real life experience, this group of patients, the first to be reported from the Middle East, reproduced the literature of Sitagliptin's advantageous effect on glycaemic control associated with a neutral effect on body weight.

P3. SERUM PROLACTIN LEVEL IS ASSOCIATED WITH LUPUS ACTIVITY. Fathia Ehmoda and Rafik Ramadan Elmehdawi. Department of Medicine, Faculty of Medicine Al-Arab Medical University and 7th October Hospital, Benghazi, Libya.

Background: Recent studies have shown that prolactin is also produced by cells of the immune system and that it also serves as an up regulator of immune function and promotes autoimmunity. Hyperprolactinaemia occurs in approximately 15–31% of patients with Systemic Lupus Erythematosis. **Objective:** To determine the relationship between serum prolactin levels and lupus activity. **Patients and Methods;** a cross sectional study of 37 patients diagnosed with systemic lupus erythematosus according to the criteria of American College of Rheumatology. Serum prolactin was checked more than once in all patients and the correlation between prolactin level and various clinical and serological markers of disease activity was studied by linear regression with the least squares method, Pearson's correlation coefficient was calculated and statistical significance was defined as $P< 0.05$. **Results:** The mean age of all patients was 19±39 years (20-58 years), with 94.4% of the patients were females. The mean disease duration was 56± 64 months (6-120 months), hyperprolactinaemia was observed in 37.1% of patients who had all other possible causes for hyperprolactinemia excluded. The mean prolactin level was 857 mIU/l, with 38.5% of patients had mild hyperprolactinaemia [≤ 800 mIU/l], and 61.5% of patients had high hyperprolactinaemia [>800 mIU/l] with normal MRI study of pituitary. Around 68.6% of the patients found to have active disease as defined by SLEDAI, out of which 62.5% had high SLEDAI score (≥10) and 37.5% had low SLEDAI score (1-9).The mean prolactin level in patients with high SLEDAI score was 870 mIU/l while it was 760 mIU/l in patients with low SLEDAI score, There was a significant correlation

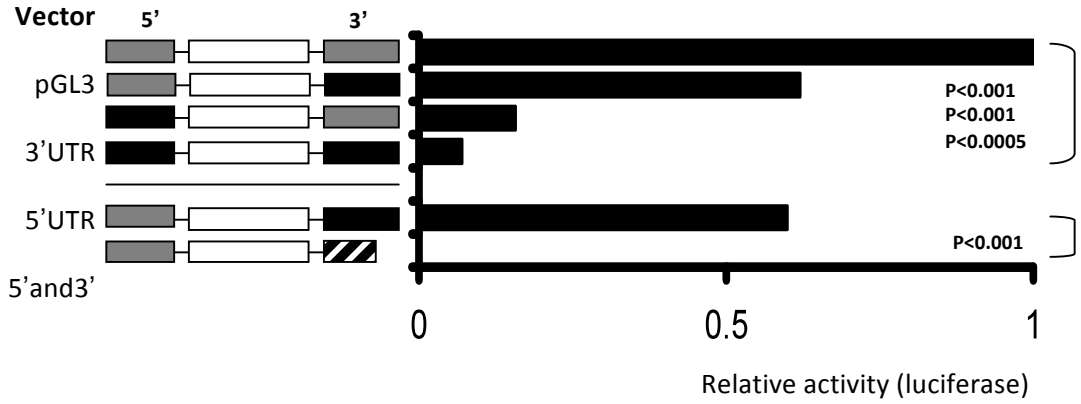
between prolactin level and SLEDAI score [r = 0.312, p=0.037] , while there was no significant correlation between the prolactin level and dsDNA titer [r =0.137, p=0.433]. **CONCLUSION;** Hyperprolactinemia was observed in 37.1% of patients with Systemic lupus erythematosis and it was associated with disease activity, our findings suggest that prolactin may play a role in the activation of systemic lupus erythematosus.

P4. SERUM SELENIUM IN DIABETES. Yakout Abdel Fattah*, Abdel-Monsef Alokali ** and Omar El Shourbagy* *Biochemistry Department Faculty of Medicine, Benha University, Egypt, **Neurology Department and ***Community Medicine Department, Derna Faculty of Medicine, Omar Almukhtar University, Derna, Libya.**

Background: Selenium, an essential trace element, is involved in the complex system of defense against oxidative stress and might prevent the development of diabetes. **Objective**: To assess serum levels of selenium in diabetic patients and normal healthy subjects. **Methodology:** This case control study included 96 patients with diabetes and 130 participants without diabetes who participated in the Derna diabetic care center. Diabetes was defined as the presence of fasting plasma glucose 126 mg/dl, a self-report of a physician diagnosis of diabetes, or current use of insulin or oral hypoglycemic medication. Information about age, sex, education, smoking, physical activity, and use of vitamin/mineral supplements was based. Serum selenium was measured by using flame atomic absorption spectrometers. **Results:** Patients with diabetes mellitus had significantly lower serum selenium levels (66.3±22.5µg/l, P<0.05) than the control groups (96.2±26.4 µg/l). Serum selenium did not differ significantly between the patients with NIDDM and IDDM. A significant correlation between serum selenium and total-cholesterol was observed (r = 0.33, P<0.05). Serum selenium were not statistically correlated with serum triglycerides, GOT and GPT in diabetic patients (P>0.05). **Conclusions:** There is increasing evidence that selenium deficiency may have several serious short- and long-term medical implications.

P5. TRANSFORMING GROWTH FACTOR BETA-1 5' AND 3'-UNTRANSLATED REGIONS CONTRIBUTES TO ITS POSTRANSCRIPTIONAL REGULATION IN PROXIMAL TUBULAR EPITHELIAL CELLS. Rasha Bennagi , A Krupa, J Martin, R Jenkins, AO Phillips and DJ Fraser. Institute of Nephrology, School of Medicine, Cardiff University, Cardiff, United Kingdom, CF14 4XN.

Problem: Transforming Growth Factor Beta-1 (TGF beta) is the principle stimulus for renal fibrosis in chronic kidney disease, among which Diabetic Nephropathy (DN) is the commonest cause. The importance of post-transcriptional regulation of gene expression by the 5' and 3' untranslated regions (UTRs) of mRNA is increasingly recognised. TGF beta synthesis is controlled post-transcriptionally in the kidney, and the contribution of the 5'UTR to this has previously been examined. *In silico* analysis also supports regulation by the 3'UTR. **Purpose:** To evaluate the contribution of the5'UTR and 3'UTR to post-transcriptional regulation of TGF beta synthesis, to investigate potential interactions between the UTRs, and to identify *in cis* and *in trans* regulatory elements. **Design:** *In vitro* studies of the TGF beta 5' and 3'UTRs in the human proximal tubular cell line HK-2. **Findings:** Novel reporter vectors incorporating the 5' and 3' UTRs of TGF beta showed that both inhibit translation, with a synergistic effect when both were included (Figure).



MicroRNAs (miRs) are small, non-coding RNAs that bind to the 3'UTR of specific transcripts and regulate their translation. miR LAT, a miR of viral origin, inhibits neuronal TGF beta synthesis, but the role of endogenous miRs in TGF beta synthesis has not previously been examined. Endogenous PTC miR expression was assessed using miRVANA microarrays. 147 of 640 miRs examined were expressed by PTC. Relative expression data from the arrays was validated

by Q-RT PCR, with high concordance between the two methodologies. We have selected miR16, highly expressed by PTC according to our data, for initial study and to validate techniques for future experiments. Mutation of the TGF beta 3'UTR to include a perfect miR16 homology site leads to substantial knockdown of signal. **Conclusions:** TGF beta mRNA is post-transcriptionally regulated by its 5'UTR and 3'UTR in PTC, with preliminary evidence for an interaction between the 3'UTR and the known inhibitory element in the 5'UTR. PTC miR expression has been characterised, and techniques developed for analysis of specific miRs in post-transcriptional regulation of TGF beta. Future experiments will examine PTC-expressed miRs identified in this study, and will characterise the 5'/3' UTR interaction suggested by this work.

P6. USE OF PHAGE DISPLAY TO ISOLATE PEPTIDES THAT BIND AND INHIBIT THE FUNCTION OF A MOUSE MONOCLONAL THYROID STIMULATING ANTIBODY. Nagat M Saeed and Phil F Watson* Al-Fateh University, Faculty of Medicine, Department of Pharmacology, Tripoli, Libya *University of Sheffield, Faculty of Medicine, Section of Endocrinology and Reproduction, Royal Hallam Shire Hospital, Sheffield S10 2RX, UK.

Background: The clinical features of autoimmune hyperthyroidism (Graves' disease) are caused by the action of agonistic antibodies directed against the thyrotropin receptor (TSHR). Patient autoantibodies recognise highly conformational epitopes on the extracellular domain of the TSHR, and identification of thyroid stimulating antibody binding site remains an important research objective, such data may provide the basis of novel therapeutic strategies. Phage display random peptide libraries have been used to map both linear and conformational epitopes and here we investigate the use of phage display to isolate peptides that bind and antagonise the action of a mouse monoclonal thyroid stimulating antibody (KSAb2), a recently isolated murine TSAb monoclonal. This mAb was isolated from an adenovirus-immunized mouse and was shown to have significant thyroid stimulating activity. **Methods:** Phage display nonapeptide libraries consisting of cysteine-constrained peptides were used. Phage libraries were enriched for 4 rounds with KSAb2 mAb, and the specificity of selected phage evaluated by ELISA. Random clones of the final enriched libraries were sequenced and the corresponding peptides analysed for homology with TSHR using the BLAST algorithm. To investigate the activity of a candidate phage peptide, the peptide sequence corresponding of phage peptide sequence was chemically synthesised and tested in a bioassay for their ability to inhibit cAMP induced by KSAb2 mAb. **Results:** The majority of enriched clones expressed the consensus sequence CQLLPSGRSLC. The motif (SGXSL) was observed in the TSHRecd (residue 226-230), suggesting that the region (SGPSL) may contribute to the KSAb2 epitope. The synthetic peptide CQLLPSGRSLC was shown to inhibit the stimulation of cAMP by KSAb2 IgG in the TSHR bioassay. The results suggest that SGXSL is an important motif for this TSAb monoclonal. **Conclusions:** The isolation of epitopes recognised by KSAb2 mAb could be useful in identification of peptides that are capable of stimulating an immune response direct against TSHR as well as for development of drug for treatment of Graves' disease.

P7. CORD BLOOD COPPER, ZINC, CALCIUM, PHOSPHORUS AND MAGNESIUM IN INFANTS OF DIABETIC AND NON-DIABETIC MOTHERS. Abdelatif Amnina*, Yakout Abdelfatah El Sinosi, Foad Elmagri* and Omar El Shourbagy*** *Pediatric, **Biochemistry and ***Community Departments, Derna Faculty of Medicine, Omar Almukhtar University, Derna, Libya.**

Background: Infants of diabetic mothers (IDMs) have many problems; they are liable for certain metabolic disorders and alteration in the homeostasis of certain elements. **Objectives:** To estimate the cord blood levels of copper, zinc, calcium, phosphorus, and magnesium in infants of diabetic and nondiabetic mothers. **Methodology:** This case control study included 80 neonates in the obstetric department at Alwahda hospital, Derna-Libya during 2007.Forty neonates (l7 males and 23 females) of controlled and poor control (HbA1c>9%) diabetic mothers. In addition to 40 neonates born to healthy mothers used as a control group. Complete history/clinical examination and determination of cord blood copper, zinc, total calcium, phosphorus, and magnesium (Cu, Zn, Ca, Mg, P) evaluated by atomic absorption spectrophotometry were done. **Results:** The gestational age in IDMs (38.17±0.78 wks) was significantly lower than in the normal neonates (39.37±1.14 wks), P<0.001. The percentage of macrosomia was higher in IDMs (42.5%) as compared to normal neonates (17.5%). The mean weight of poor controlled IDMs (4.41±0.31kg) was significantly higher than the controlled IDMs (3.88±0.49 kg) and the normal neonates (3.51±0.60 kg), P<0.05. The mean values of cord blood levels of Cu, Ca, Mg and P in IDMs (33.3±1.1μg/dL, 2.45±0.56 mEq/L, 1.4±0.45 and 6±0.23 mg/dL) were significantly lower than the normal neonates (39.8±1.2μg/dL, 3.78±0.57 mEq/L, 2.1±0.48 and 6.29±0.25 mg/dL, P< 0.05).

Conclusion: Cord blood status of some essential trace elements in gestational diabetes patients could have influences on the health of the fetus and newborn.

P8. EVALUATION OF HYPOGLYCEMIC AND HYPOLIPIDEMIC EFFECTS OF "COENZYME Q10" IN STREPTOZOTOCIN-INDUCED DIABETIC RATS. Mohamed Bendary* and Abdel-Monsef Alokali **, *Physiology Department Faculty of Medicine, Minoufiya University, Egypt, **Neurology department Faculty of Medicine, Omar Al Moukhtar University, Derna, Libya.

Background: Oxidative stress is implicated in the pathophysiology of diabetes mellitus. Coenzyme Q10 (CoQ10), an endogenous antioxidant and a component of the mitochondrial electron transport chain, is recently advocated as an adjuvant in diabetes. **Objectives:** To evaluate the effect of CoQ10 on blood glucose level and lipid profile in streptozotocin-induced diabetic rats. **Methodology:** In the present study, 36 adult male albino rats were used. They were divided into. Control group, rats were subjected to single intraperitonial (I.P) injections of 0.5 ml sodium citrate buffer solution. CoQ10-treated control group, rats were supplemented orally with aqueous CoQ10 solution (10 mg/kg BW daily for 8 weeks). Untreated diabetic group, rats injected I.P 60mg/kg BW streptozotocin. CoQ10-treated diabetic group, diabetic rats were treated orally with CoQ10 solution (10 mg /kg BW daily for 8 weeks). Fasting serum glucose, insulin, and lipid profile, plasma malondialdehyde were measured. In addition, total body weight was determined. **Results:** CoQ10-treated control group showed a non-significant differences in all measured parameters as compared to normal control group. Serum glucose, triglycerides, total cholesterol, LDL, and malondialdehyde in diabetic group were significantly higher (262.2±12.4, 120.3 ±6.5, 106.8 ±12.6, 77.7±5.4 mg/dl and 10.6 ±0.1 umol/L) than the control group (77.4±6.2, 81.5 ±6.3, 79.8 ±5.1, 34.2 ±2.6 mg/dl and 31.1±2.9 umol/L, $P<0.05$). Insulin, HDL and percentage of body weight change in diabetic group were significantly lower (6.3±0.1 uU/ml, 7.6±0.1mg/dl and -22.8%) than the control group (22.7±2.3 uU/ml, 11.4 ±1.0 mg/dl and +44.2%, $p<0.05$). When diabetic group was treated with CoQ10, a significant improvement was exhibited in almost all parameters. **Conclusion:** Oral CoQ10 administration has a hypoglycemic and hypolipidemic effects in experimentally induced diabetic rats, probably through its antioxidant and bioenergetics potentiation effects. This beneficial effect of Coenzyme Q10 provides an impetus for further clinical trials.

P9. FASTING OF RAMADAN IN PEOPLE WITH DIABETES IN BENGHAZI. Rafik R Elmehdawi, NA Mukhtad, NI Allaghi and SJ Elmajberi. Department of Medicine, Faculty of Medicine, Al-Arab Medical University and The Benghazi Diabetes and Endocrine Center, Benghazi, Libya.

Introduction: Diabetics who fast during Ramadan represent a challenge to their physicians. **Aim and objectives:** To define the frequency of fasting and the potential complications of fasting in Libyan diabetic patients during Ramadan. Patients and methods: we interviewed 493 consecutive diabetic patients who attended Benghazi diabetes and endocrine center during the first week after Eid-ul-fiter and a standard proforma. **Results:** Out of the 493 studied patients 95% were type-2 diabetics and 50.3% were males. The mean age was 59+/- 12 years. They had had diabetes for 11.3+/-10 years and their mean HbA1c before Ramadan was 7.8±1.6. About 27% of the patients had no treatment changes during Ramadan fasting. The mean fasting was 28.5±4.6 days with 70,4% completed 30 days of fasting and 97.5% fasted at least 15 days. The mean reason of breakfasting was hypoglycemia (43.4%) followed by hyperglycemia (27%). About 14.6% of patients experienced mild hypoglycemia, 3.2% had severe hypoglycemia, 11.2% had hyperglycemia (≥ 300mg/dl), and only 5.1% were admitted during the study period. There was no significant difference between type-1 and type-2 diabetics regarding fasting rate, frequency of hypoglycemia hyperglycemia or admission rate during Ramadan period. The incidence of hypoglycemia during Ramadan was 31 episode/100 patients, while the incidence of hyperglycemia was 17 episode/100 patients. Patients who experienced hyperglycemia had a significantly higher baseline HbA1c than other patients (8.4+/-1.1% vs. 7.6+/-1.6%, $p=0.02$) also they had a significantly lower mean fasting 26.9+/-5.7% vs. 29+/-4, $p=0.001$). **Conclusion:** More diabetic patients in our study manage to fast compare to published observations from other Muslim countries, Three quarters of the patients had some treatment changes during Ramadan. The commonest complications that occurred were hypoglycemia and hyperglycemia.

P10. USE OF INSULIN PUMP THERAPY IN MOSLEM PATIENTS WITH TYPE 1 DIABETES DURING RAMADAN FASTING. Mahmoud Benbarka, Ali B Khalil, Salem A. Beshyah, Suhad Marjei and Samar Abu Awad. Center for Diabetes and Endocrinology, Sheikh Khalifa Medical City P.O. Box 51900, Abu Dhabi, United Arab Emirates.

Introduction: Although Moslem patients with diabetes may be exempt from fasting during Ramadan, many chose to fast. There are no data on insulin pump therapy during Ramadan; although, some professionals; anecdotally argue

against fasting. We report our experience with type 1 diabetes patients on insulin pump during Ramadan 2008 (29 days). **Patients and Methods:** A total of 64 patients were evaluated. Forty nine patients fasted and fourteen elected not to fast. Those who fasted were 22+ 7 years of age (Mean+SD), 25 males and 25 females and had diabetes for 9.6+ 5.6years. Patients were Medtronic Minimed 722 model and had been using pump therapy for 20+10 months. Outcome measures included number of days fasted, hypoglycemia, hyperglycemia and emergency hospital visits. **Results:** Thirty one patients (62%) fasted the whole month with no problems. nine (18%) fasted 27-28 days, eight (16%) fasted 24-25 days and two (4%) fasted 23 days. Fifty percent of the patients decreased their basal insulin rate by 5-50%. Seventeen patients had hypoglycemia needing breaking the fast. Fasting was broken on 55 out of 1450 potential fasting days (3.8%). No severe hypoglycemia was reported by any patient. Hyperglycemia was reported in nine patients (18%). Hospital visits were reported only in patient for hyperglycemia (a 16 year old girl who disconnected her pump). Twelve patients had fructosamine level obtained both before and immediately after Ramadan. Pre-Ramadan fructosamine level was 4.0+ 0.6 and post-Ramadan 3.6+ 0.6 mmol/l (p= 0.007). **Conclusions:** Fasting during Ramadan is feasible in patients with type 1 diabetes on insulin pump with adequate counseling and support.

P11. LESSON OF THE WEEK: NOT JUST ANOTHER "HAIRY WOMAN"! Huda Ezzeddin Mustafa and Salem A Beshyah. Centre for Diabetes & Endocrinology, Sheikh Khalifa Medical City, Abu Dhabi. United Arab Emirates.

INTRODUCTION: Rare causes of common symptoms may be missed by the unwary and may result from less careful assessment and inadequate investigations. We present an example of such clinical scenario. **CASE STUDY:** A 30 year old, single female presented in May 2008 with oligomenorrhoea, excessive hair growth, thinning of the scalp hair and weight gain since the age of 17 that worsened over the previous 5 years. Menarche was the age of 15 and her last 3 menstrual periods were in April 2008, February 2008, and November 2007. Past medical history was non contributory. She received treatment with Dianette, Aldactone and Metformin intermittently for 2 y till 4 years ago. Family history was significant for type 2 diabetes but no history of excessive hair growth. Physical examination revealed weight of 100 kg, height of 164 cm and body mass index of 37 kg/m². Blood pressure was 107/66 mmHg. A moderate degree of facial hirsutism (with evidence of shaving) was evident. Severe body hirsuitism on the chest, abdomen, and back was present. Her Ferriman and Gallwey score was 30/36. Excessive sweating, Balding and severe acanthosis nigricans (in the neck, axillae and back) were also noted. There was no goiter and she was clinically euthyroid and no striae. Rest of the system examination was negative. There was a significant clitoromegaly. Her Investigations revealed total serum testosterone of 53 nmol/l, 17-hydroxy progesterone was 2.7 nmol/l, serum LH and FSH were 6.4 and 5.3 IU/l respectively. DHEAS was 6.43 umol/l. Respeated investigations confirmed the same trend and in addition, thyroid functions and serum cortisol were normal, insulin level was very high. Pelvic Ultrasound (May 08) CT Abdomen revealed normal suprarenal glands, enlarged both ovaries, the right was larger but not typical for polycystic ovary. An MRI of the pelvis showed a right adnexal complex mass 4.6 cm in diameter, normal adrenals. Patient was offered a gynecological referral but she opted to seek care elsewhere. She returned in September having had laparoscopic right salpingo-opherectomy in India in July 08. The histology reported as well-demarcated yellowish orange tumour nodule (3.5 x 3 x 3 cm) of steroid cell tumour with no features of malignancy. Postoperatively, her serum testosterone dropped from 328 to 90.5 ng/dl and remained so in september 2008 (1.88 nmol/l). Further management included Minoxidil (Regain), Metformin and oral combined contraceptive pill that was recommeded for 1 year. **COMMENTS**: This is an example of a rare cause of a common symptom. Delay in making the diagnosis could have been due to more complacent approach in dealing with her symptoms and attributing them to the most likely most common cause namely PCOS. However several features are present and should have alerted the clinician to look for other underlying pathology earlier (clitoromegaly and very high testosterone). The patient was relatively lucky given the benign nature of the tumor.

P12. THE INFLUENCE OF DIABETES ON ACETOAMINOPHEN® INDUCED HEPATOTOXICITY IN ALBINO WISTAR RATS. Khadija A Debri, Mohammed R. Hamid, Soad Bosseri and Zammzam M. Burkan. Department of Pharmacology and Clinical Pharmacy, Faculty of Pharmacy, Al-Fatah University Of Medical Sciences. Tripoli, Libya.

Introduction: Uncontrolled diabetes is associated with formation of ketone bodies, which are known to cause hepatotoxicity. The aim of this study was to investigate the effect experimentally induced diabetes on susceptibility to acetoaminophen hepatotoxicity. **Methods:** Seventy-eight adult albino male wistar rats of 140-180 gm body weight were divided into 4-main groups. Group I; untreated (control). Group II; received acetoaminophen® in oral serial doses of 50, 100, 150, or 300 mg/kg.. Group III (experimental diabetes); two subgroups; one Injected by alloxan 10 mg/kg

subcutaneously (s.c), the other injected by streptozotocin 150mg/kg, intraperitoneally (i.p). Group IV. diabetic rats were injected by acetoaminophen (150 mg/kg p.o). Their blood sugar were frequently measured, desired hyperglycemia state was detected on day-14 following diabetogenic agent administration. In addition, serum (ALT) was measured a day after acetoaminophen administration. **Results:** Acetoaminophen was found to produce dose-dependent increase in the serum ALT- levels. This effect is worsened in the diabetic status (Table 1).

Table 1. Effect of co-treatment by acetoaminophen®on *s*ALT levels in diabetic rats

No	**Treatment Design**	***s*ALT Level**	
		Mean	±SE
1	*Untreated (control)*	10.82	0.53
2	*Acetoaminophen®*	23.50	1.10
3	Alloxan	27.78	1.79
4	Streptozotocin	18.66	0.39
5	*Acetoaminophen® plus* Alloxan	46.78	0.34
6	*Acetoaminophen®* plus Streptozotocin	59.10	0.50
***Significant at p value < 0.005, 0.005 in comparison to untreated (control) rats or *Acetoaminophen alone (150 mg /kg)**			

Comments: The experimental chemically induced diabetes either by alloxan or by streptozotocin produced significant elevation of serum ALT levels. Treatment by acetoaminophen® to those diabetic rats resulted in significant elevation in the serum ALT to much higher levels than those produced by experimental diabetes or by acetoaminophen® alone. This elevation in the serum ALT- levels represents an additive effect in case of alloxan-diabetes and a potentiate effect in case of streptozotocin-diabetes. We conclude that careful assessment of the interaction between experimental diabetes and hepatotoxic drugs should be highly considered.

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