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Dexamethasone intake for different periods related to Blood Sugar rate in Diabetes Mellitus type 2 Patients in Thi-qar province / Iraq

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Abstract

Dexamethasone widespread use as anti-inflammatory drugs. In Diabetes mellitus type 2 patients using of Dexamethasone has a crucial side effects including increase blood sugar rate. 106 patients involved in this study, all of them take Sulfonylurea tablets to control glucose level in the blood. They divided into 3 groups (depending upon period of Dexamethasone used) group A patients intake drug for one week (30 patient), group B take the drug more than one month (30 patient) and third group don't take the drug (46). Random blood sugar rate taken for all patients after finishing drug prescription. Results of three groups statically analysis. The mean of three groups were 242.8, 317 and 217 for A,B and C respectively. There were a significant differences in level (P<0.5%) between the two groups (A,B) and control group also between group A and B .Females were prodomenet suffer from diabetes they reached 78.6% while males were 21.4%. This the study showed that patients were un educated or negligible with Dexamethasone side effects and took the drug without prescription.

Key words: Dexamethasone, Diabetes Mellitus type2, Glucose, Thi-qar

تناول عقار الدكساميثازون لفترات مختلفة وتأثراته على مستوى السكر لدى مرضى السكري النوع الثاني في محافظة ذي قار/ العراق

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الخلاصة

يعتبر عقار الدكساميثازون من الأدوية المضادة للالتهابات خصوصاً التهاب المفاصل ويستعمل بشكل واسع لدى المصابين بالسكري من النوع الثاني حيث يسبب ارتفاع في مستوى السكر بالدم. شملت الدراسة ١٠٦ مريض مصاب بالسكري النوع الثاني وكانوا جميعا يتناولون علاج السلفانيوليوريا كمنظم لمستوى السكر لديهم .قسم المرضى إلى ثلاث مجاميع (اعتمادا على فترة تناول عقار الدكساميثازون) المجموعة أ شملت (٣٠) حيث تناولوا عقار الدكساميثازون لمدة أسبوع. المجموعة الثانية ب شملت (٣٠) حيث تناولوا العقار لمدة تزيد عن الشهر. المجموعة الثالثة ج (٤٦) اعتبرت كمجموعة سيطرة حيث لم يتناولوا عقار الدكساميثازون . بعد نهاية فترة العلاج تم قياس مستوى السكر بالدم حيث أظهرت النتائج أن معدل مستوى السكري في المجموعة الأولى كان ٢٤٢.٨ في حين أن المجموعة الثانية ب فكان مستوى السكر فيها ٢١٧ . وأظهرت نتائج التحليل الإحصائي أن هنالك فرق معنوي على مستوى احتمالية ($p<0.05$) بين المجموعة أ و ج والمجموعة ب و ج كذلك بين المجموعة أ و ب. حيث كان معدل مستوى السكر بالدم (242.8, 317 217) للمجموعات أ و ب و ج على التوالي. كما بينت الدراسة أن نسبة النساء أكثر من نسبة الرجال الذين يتعاطون العقار حيث وصلت النسبة إلى ٧٨.٦% بينما نسبة الرجال ٢١.٢%.

Introduction

Glucocorticoids have widespread use as anti-inflammatory and immunosuppressive agents (1). However, chronic glucocorticoid therapy is often associated with adverse and serious side effects, including Cushing's syndrome, Osteoporosis, Gastrointestinal bleeding, and Dyslipidemia (1). More importantly, both excess endogenous (2,3) and exogenous (4,5) glucocorticoids impair insulin sensitivity, contributing to generation of the Metabolic Syndrome, including insulin resistance, Obesity, and Hypertension. Incidence of this syndrome is closely linked to increased mortality from cardiovascular diseases (6). Besides the possible effect of Dexamethasone, several other factors may have contributed to the increase in blood glucose concentration, of which surgical stress is probably the most important. That blood glucose concentration has previously been shown to increase significantly over the course of surgery in patients who did not receive Dexamethasone may by itself explain the results, whether patients had Diabetes or not (7,8). Factors secreted by adipose tissue, referred to as adipokines, also act to modify insulin sensitivity of tissues. One of these, adiponectin, promotes insulin sensitivity in tissues and is suppressed by glucocorticoid treatment, serving as yet another component in the insulin resistance seen with glucocorticoid therapy (9). Glucocorticoids also promote proteolysis, lipolysis, free fatty acid production, and fat accumulation in the liver, which can contribute to insulin resistance (9). In addition, glucocorticoids directly promote hepatic gluconeogenesis, increasing hyperglycemia. Pancreatic β cell dysfunction allows further hyperglycemia to develop, likely contributing to the progression to frank diabetes sometimes seen with chronic glucocorticoid therapy (9). In this issue of the JCI, Brennan-Speranza and colleagues provide evidence for a new mechanism of glucocorticoid-induced insulin resistance in which glucocorticoid action on bone decreases the levels of the osteoblast-derived peptide osteocalcin, which in turn contributes to the insulin resistance and metabolic derangements associated with glucocorticoid therapy (10). Strategies are already being explored to attempt to circumvent the side effects of glucocorticoids while maintaining the therapeutic benefits. One strategy for targeting endogenous glucocorticoid-mediated insulin resistance is inhibition of 11β-hydroxysteroid dehydrogenase-1, which is responsible for converting cortisone to the more biologically active form cortisol. Preliminary studies of one such inhibitor have demonstrated positive effects in the treatment of type 2 diabetes in humans (11), but this needs to be validated in other, larger studies. Also, such an inhibitor would not be useful to block the adverse metabolic effects of pharmacologic glucocorticoid therapy, as it would also block the immunosuppressive effects of the drugs. Another approach is to develop selective glucocorticoid receptor modulators. These take advantage of the fact that many of the genes associated with side effects (e.g., osteocalcin; ref. 17) are modulated through glucocorticoid receptor binding to glucocorticoid response elements, whereas many of the therapeutic effects occur through interactions with glucocorticoid receptor–associated transcription factors, such as AP1 (12). A number of selective glucocorticoid receptor modulators have been studied (12).

Methods

The study period extent from November 2014-April 2015 in Thiqar province. Investigation to people of Diabetes mellitus type 2 among different ages and sex based on a questioners of patients about Dexamethasone tablets (dose 5 mg) intake and period of medicine taken. patients divided into three groups. Group A (30 patient) which include patients whom take Dexamethasone for one week, second group (B) (30 patient) patients whom taken drug more than one month. Third group C (46) patients do not taken this medicine. 92 patients tested in this study in different ages (30-65 years), all of them take Sulfonylurea tabletsto control blood glucose rate. Calculated of the blood sugar rate by Random blood sugar test. A blood sample will be taken at a random time after end of medicine taken. Blood sugar values are expressed in milligrams per deciliter (mg/dL), by using advice for sugar rate calculating (True result company, Germany).A hematological standard was measured by using hematology analyzer in the laboratories of the blood bank in the city of Nasiriyah , Thi-Qar province which included : red blood cells (RBC), hemoglobin (Hb) ,packed cell volume (PVC), mean corpuscular hemoglobin (MCH) ,mean corpuscular hemoglobin concentration (MCHC) , mean corpuscular volum (MCV) and white blood cells (WBC).

Results

Results obtained show that females more predominant than males which suffering from diabetes mellitus type 2, percentage was 78.6% while male 21.4%. Most of patients taken Dexamethasone without

physician prescription for enhanced appetite or get moon face like, they did not known the side effects of this medicine. 30 patients involving in the group (A) how take Dexamethasone for one week

Table (1) :Glucose rate of patients treated with Dexamethasone for one week

Sex / Female	Glucose rate mg/dl
S1	400
S2	181
S3	211
S4	175
S5	128
S6	116
S7	185
S8	479
S9	116
S10	107
S11	478

Sex / Female	Glucose rate mg/dl
S12	402
S13	478
S14	200
S15	216
S16	139
S17	200
S18	177
S19	190
S20	188
S21	400
S22	478

S = sample

Sex / Male	Glucose rate mg/dl
S1	252
S2	181
S3	197
S4	488
S5	175
S6	180
S7	245
S8	300

Mean = 242.8

Table (2): Glucose rate in patients who take Dexamethasone more than one month

Sex / Female	Glucose rate mg/dl	Sex / Male	Glucose rate mg/dl
S1	486	S1	364
S2	406	S2	306
S3	419	S3	306
S4	311	S4	235
S5	478	S5	185
S6	402	S6	310
S7	187	S7	353
S8	317		
S9	350		
S10	410		
S11	315		
S12	188		
S13	356		
S14	225		
S15	148		
S16	368		
S17	310		
S18	156		
S19	542		
S20	388		
S21	457		
S22	400		
S23	315		
Mean		317	

Table (3) : Glucose rate of patients do not taken Dexamethasone

Sex / Female	Glucose rate mg/dl	Sex / Male	Glucose rate mg/dl
S1	265	S1	145
S2	313	S2	140
S3	186	S3	222
S4	146	S4	181
S5	270	S5	205
S6	216	S6	206
S7	127	S7	200
S8	164	S8	164
S9	175		
S10	260		
S11	315		
S12	200		
S13	197		
S14	158		
S15	200		
S16	181		
S17	206		
S18	178		
S19	184		
S20	157		
S21	200		
S22	207		
S23	194		
S24	207		
S25	200		
S26	332		
S27	204		
S28	190		
S29	196		
S30	182		
S31	175		
S32	305		
S33	317		
S34	191		
S35	143		
S36	133		
S37	190		
S38	133		
Mean		217	

S= sample

Discussion

The role of beta cell function and other tissues' sensitivity to insulin may be different depending on whether the glucocorticoid effect is acute or chronic. One study compared an acute single dose of prednisolone (75 mg) with 30 mg of prednisolone daily for 15 days. The acute treatment inhibited several parameters of beta cell function. Conversely, prolonged glucocorticoid exposure showed partial recovery of beta cell function but similarly impaired glucose tolerance, suggesting additional factors are important in SIDM other than beta cell dysfunction [13]. From this study we conclusion that most of patients took Dexamethasone without physician prescription, because they negligible with the side effect of the drug, while those whom took it with prescriptions the suffering from ostiomuscular system problems mainly inflammation. Proposed risk factors for steroid-induced diabetes beyond cumulative dose and longer duration of

steroid course include traditional risk factors for type 2 diabetes: older age, family history, high body mass index and impaired glucose tolerance [14]. The association with family history of diabetes is not well defined. Simmons et al. compared the demographics and clinical characteristics of patients with new onset SIDM with those with type 2 diabetes with and without steroid treatment. Those individuals who developed new onset SIDM had significantly less family history of diabetes when compared with individuals with type 2 diabetes mellitus and glucocorticoid treatment [15]

Glucocorticoids were shown to cause various degrees of β-cell dysfunction, reducing insulin sensitivity and impairing β-cell function [16], by acting through glucocorticoid receptors which are also expressed on pancreatic β-cells. Glucocorticoids may also impair the uptake and the metabolism of glucose in β-cells through genomic actions (i.e., modulation of gene expression by nuclear glucocorticoid receptor) which lead to a decrease in the efficacy of cytoplasmic Ca2+ on the exocytotic process of insulin secretory vehicles [17]. It was also recently reported that short-term exposure to glucocorticoids reduced the insulinotropic effects of GLP-1 [18]. Among most populations worldwide, the prevalence of diabetes is similar among female and male members of the population (19). However, among American Indians, the prevalence of diabetes may be higher among female than male members of the population (20-23). The prevalence of previously diagnosed diabetes among Zuni Indians aged ≥5 years (n = 1,503) was higher among female Zuni Indians (16.7% [95% CI 14.1–19.3]) than male Zuni Indians (9.7% [7.4–12.1]) (24). The above results agreed with our study which estimated that number of female suffering from diabetes more than male also they used Dexamethasone more, this may be to give them attractive view on their faces because one of the side effects of Dexamethasone is moon face.

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