

CASE REPORT

Type 1 Diabetes Presenting After Bariatric Surgery: A Medical Paradox Leading to a Delayed Diagnosis and Diabetic Ketoacidosis

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Abstract

The relationship between type 2 diabetes and obesity is well established; however, obesity at onset of type 1 diabetes is fairly uncommon. We present a 19-year-old man who was diagnosed with type 1 diabetes after considerable weight loss following bariatric surgery. This unexpected medical paradox may have led to the delayed recognition of the type of diabetes resulting in decompensation into diabetic ketoacidosis.

Key words: DKA, Type 1 diabetes, Diagnosis of Diabetes, Bariatric surgery, Pre-diabetes, Obesity, Autoimmune.

Introduction

The United Arab Emirates (UAE) has one of the highest rates of both obesity and type 2 diabetes (T2DM) in the Gulf and Middle Eastern region (1,2). As a

result of this double jeopardy of diabetes and obesity, bariatric surgery has gained immense popularity as a treatment for obese T2DM patients (3). Interestingly, the procedure has gained momentum even in obese individuals without co-morbid conditions though there are no clear national guidelines on the use of bariatric surgery on obese non-diabetic subjects. This is a case of a young obese non-diabetic Emirati male who underwent laparoscopic sleeve gastrectomy and five months later was admitted in frank diabetic ketoacidosis.

Case Summary

A 19-year-old college student was offered laparoscopic sleeve gastrectomy as treatment for obesity. At the time of surgery he had no concomitant illnesses and had a body mass index (BMI) of 37 kg/m². Prior to the procedure he was screened for T2DM using HbA1c

which at that time was 5.8% compatible with pre-diabetes. His post-operative course was uncomplicated, and he was discharged three days after the procedure. Five months later he presented to the emergency department with marked osmotic symptoms of one week duration associated with generalized malaise, persistent nausea and vomiting. There was no family history of diabetes or exposure to systemic steroids or any other diabetogenic medications. He did not smoke or consume ethanol. Following surgery he had resorted to a fluid diet consisting of fruit juices and milk shakes. He was compliant with the vitamin replacement therapy advised post-operatively. He had lost approximately 40 Kilograms of weight after the surgery.

Physical examination revealed a healthy looking male with stable vital signs. He had no cutaneous markers of insulin resistance. Systemic examination was

unremarkable except for healed surgical laparoscopic scars over the abdomen. The biochemical profile on admission supported the diagnosis of diabetic ketoacidosis (DKA) as shown in table 1. He was managed successfully according to the hospital's local DKA management protocol. He was subsequently started on a subcutaneous basal-bolus insulin regimen consisting of basal insulin glargine at bedtime and prandial boluses of insulin lispro prior to meals. The management of his glycemia proved challenging as he insisted on consuming fruit juices and milk shakes and was non-compliant with the dietary advice offered. This resulted in fluctuating blood glucose levels with recurrent episodes of hypoglycemia. Considering his history of obesity the possibility of type 2 diabetes was also entertained. He was therefore given a trial of gliclazide MR and metformin together with basal glargine while in the hospital under close observation.

Table 1. Results of the laboratory investigations during the presentation of the DKA.

Tests	Patients results
Random blood glucose	327 mg/dl
Urinary ketones	3+
Urea and electrolytes	Na ⁺ 139 mmol/L, K ⁺ 3.9 mmol/L, Urea 28 mg/dl Bicarbonate 14.2 mmol/L
Creatinine	1.4 mg/dL on admission 1.1 mg/dL following hydration.
Full blood count	Hb 16.9 g/dL, WBC 11.2 x 10 ⁹ /L, Platelet: 262 x 10 ⁹ /L, MCV 88.4 fL
HbA1c	8%
Liver function tests	Alb: 5.79 g/dL, ALP: 131 U/L, SGPT: 10 U/L, Bilirubin: 1.2 mg/dL Total protein: 9.5 g/dL
Thyroid function tests	TSH 1.28 uIU/ml, T3: 5.0 pmol/L, T4 18.3 pmol/L
Calcium	9.5 mg/dL
Magnesium	2.37 mg/dL
Vitamin B1	71.2 (66.5-200) nmol/L
Vitamin B12	192 (187-383) pg/ml
Vitamin E	6.8 (5-18) µg/ml
Serum amylase & lipase	Normal

Table 2. Islet cell autoantibodies results

Autoantibodies	Patient' results	Reference ranges
Islet cell antibodies (ICA)	1:160+ titre	<1:10
Glutamatedecarboxylase antibodies (GAD 65)	<10.0IU/ml	<1:10

Table 3. Sensitivity and specificity of the four major islet autoantibodies in the diagnosis of type 1 diabetes mellitus (8).

Autoantibodies	Sensitivity ^a	Specificity
Islet cytoplasmic antibodies (ICA)	70%-80%	>99%
Glutamatedecarboxylase antibodies (GAD)	70%-80%	97%-98%
Insulinoma antigen 2 proteins (IA-2A)	60%	97%-98%
Insulin autoantibodies (IAA)	60% ^b	95%
a. <i>Frequency in new-onset diabetes</i> b. <i>This is for children; IAA are uncommon in adults</i>		

However, a poor response was noted to this regimen and after 3 days of this approach he was switched back to intensive insulin regimen. Two weeks later the reports of his auto-immune work-up were obtained which were positive for islet cell antibodies (ICA) confirming a diagnosis of type 1 diabetes mellitus (T1DM) (Table 2).

Discussion

This case proved to be a diagnostic challenge with regards to the type of diabetes due to the fact that the patient had many atypical features. His background history of obesity was in keeping with T2DM particularly considering the epidemiology of diabetes in the UAE. There has been an increasing trend of T2DM presenting with ketoacidosis. The entity "ketosis-prone atypical diabetes" has therefore emerged and a subset of these patients are auto antibodies negative with partial recovery of pancreatic function after resolution of the acute episode and can be

managed with oral anti-diabetic agents (4). However, he responded poorly to a trial of oral hypoglycemic agents. Another unusual feature of this case was that if we considered him to have type 2 diabetes than it was strange that he should present after he had shown dramatic weight loss post bariatric surgery. Numerous articles have shown that bariatric surgery improves type 2 diabetes even resulting in complete resolution of hyperglycemia to the point of cure (5). In contrast, T1DM is not associated with obesity or insulin resistance and results from cell-mediated autoimmune destruction of the β cells of the islets of Langerhans. Autoantibodies directed against the islets are useful clinical tools that allow the recognition and confirmation of this diagnosis (6).

Islet cell autoantibodies comprise autoantibodies to islet cell cytoplasm(ICA), to native insulin, referred to as insulin autoantibodies (IAA), to the 65-kDa isoform of glutamic acid decarboxylase (GAD65A),

to two insulinoma antigen 2 proteins (IA-2A and IA-2B; also known as phogrin) and to three variants of Zinc transporter 8 (Zn T8A). Autoantibody markers of immune destruction are usually present in 85% to 90% of individuals with type 1 diabetes when fasting hyperglycemia is initially detected. CA are detected in 70-80% of individuals with new-onset type 1 diabetes. GADA are found at similar rates (70%-80%) as ICA. IA-2A is less common at the onset of T1DM (approx. 60%) than either ICA or GADA (7,8) (Table 3).

In this patient the detection of islet cell antibodies in a high titer together with a low C-peptide level was in keeping with a diagnosis of T1DM. The detection of islet autoantibodies in a diabetic individual indicates that the diabetes is of autoimmune origin (viz T1DM). This is true even if the initial phenotype is more like that of T2DM and the term LADA (Latent autoimmune diabetes of adults) was coined for such cases (9). Islet autoantibody testing is indicated when the clinician cannot readily differentiate T1DM from T2DM (10). Differentiation of these conditions is critical because the early introduction of insulin has been shown to preserve β -cell function, and good glycemic control will delay the onset of microvascular and neuropathic complications in T1DM (11). This case is quite unique in that T1DM was discovered following a period of weight loss post-bariatric surgery. We are not aware of any similar encounter that was reported in the literature.

References

1. WHO website. <http://www.emro.who.int/health-topics/obesity/>
2. Sheikh-Ismail LI, Henry CJ, Lightowler HJ, Aldhaferi AS, Masuadi E, Al Hourani HM. Prevalence of overweight and obesity among adult females in the United Arab Emirates. *Int J Food Sci Nutr*. 2009;60(Suppl 3):26-33.
3. Nimeri A, Mohamed A, El Hassan E, McKenna K, Turrin NP, Al Hadad M, et al. Are results of bariatric surgery different in the Middle East? Early experience of an international bariatric surgery program and an ACS NSQIP outcomes comparison. *J Am Coll Surg*. 2013;216(6):1082-8.
4. Balasubramanyam A, Nalini R, Hampe CS, Maldonado M. Syndromes of ketosis-prone diabetes mellitus. *Endocr Rev* 2008;29:292-302.
5. Taylor R. Type 2 diabetes: etiology and reversibility. *Diabetes care*. 2013;36(4):1047-55.
6. Winter WE, Schatz DA. Autoimmune markers in Diabetes, *Clinical Chemistry*. 2011;57(2):168-75.
7. Libman IM, Pietropaolo M, Trucco M, Dorman JS, LaPorte RE, Becker D. Islet cell autoimmunity in white and black children and adolescents with IDDM. *Diabetes Care* 1998;21:1824-7
8. Orban T, Sosenko JM, Cuthbertson D, Krischer JP, Skyler JS, Jackson R, et al Pancreatic islet autoantibodies as predictors of type 1 diabetes in the Diabetes Prevention Trial-Type 1. *Diabetes Care* 2009;32:2269-74.
9. Leslie RD, Kolb H, Schloot NC, Buzzetti R, Mauricio D, De Leiva A, et al. Diabetes classification: grey zones, sound and smoke: Action LADA 1. *Diabetes Metab Res Rev* 2008;24:511-9.
10. Iserman B, Ritzel R, Zorn M, Schilling T, Nawroth PP. Autoantibodies in diabetes mellitus: current utility and perspectives. *Exp Clin Endocrinol Diabetes* 2007;115:483-90.
11. Shah SC, Malone JJ, Simpson NE A randomized trial of intensive insulin therapy in newly diagnosed insulin-dependent diabetes mellitus. *N Engl J Med* 1989;320:550-4.