HYPERGLYCEMIA: „TRICK OR TREAT" – CASE REPORT

Mirela Mogoi[1], I Velea[2], Simona Dumitriu[3], Corina Paul[2], Georgeta Noditi[1]

Abstract

Hyperglycemia is a condition characterized by excessively high levels of glucose in the blood: fasting glucose higher than 7 mmol/l (> 126 mg/dl), or random glucose higher than 11,1 mmol/l (> 200 mg/dl) (1). Temporary hyperglycemia is often asymptomatic, but when glucose levels are extremely high is a medical emergency and can rapidly produce serious complications. Usually when we say „hyperglycemia" we think of diabetes or a prediabetic state. The paper presents the case of an adolescent girl, admitted for pseudoneurological symptoms and abnormal capillary blood glucose values (> 11,1 mmol/l). Her medical history is remarkable for repeated episodes of neurological symptoms accompanied with pathological glucose blood levels. She had two other hospital admissions for impaired glucose tolerance, overweight and loss of consciousness. The initial laboratory tests showed: high levels of fasting blood glucose without glycosuria or ketonuria, abnormal Oral Glucose Tolerance Test (positive for diabetes mellitus) and no clinical symptoms related to the high glycemic levels. Further investigations revealed an unexpected „cause" of hyperglycemia: the somatoform disorder.

Key words: hyperglycemia, somatoform disorder, medically unexplained symptoms, fabricated or induced illness.

Introduction

The origin of the term „hyperglycemia" is Greek: *hyper-* meaning „over, beyound, overmuch, above measure"; *-glycys-*, meaning „sweet"; and *–emia* from „ *haima"* meaning *„of the blood" (2).* So hyperglycemia is a condition characterized by excessively high levels of glucose in the blood: fasting glucose higher than 7 mmol/l (> 126 mg/dl), or random glucose higher than 11,1 mmol/l (> 200 mg/dl) (1). Temporary hyperglycemia is often asymptomatic, but when glucose levels are extremely high is a medical emergency and can rapidly produce serious complications. Usually when we say „hyperglycemia" we think of diabetes or a prediabetic state. The following conditions can also cause hyperglycemia in the absence of diabetes:

a) endocrinopathies (especially those affecting the thyroid or adrenal and pituitary glands);
b) another exocrine pancreas diseases (cystic fibrosis, pancreatitis, trauma / pancreatectomy, haemochromatosis, neoplasia);
c) certain infections , sepsis;
d) terminal stages of many diseases;
e) neurological pathology: encephalitis, brain tumors (especially those located near the pituitary gland), brain hemorrhage, convulsions;
f) prolonged, major surgeries can temporarily increase glucose levels;
g) certain forms of severe stress and physical trauma can increase levels for a brief time as well, yet rarely exceeds 6,6 mmol/l (> 120 mg/dl).

An unusual differential diagnosis of hyperglycemia is a psychiatric disorder. In recent years somatoform disorders gained increasing significance in all medical specialties, although in child psychiatry, publications on this topic are rare.

Case report

We present the case of an adolescent girl, aged 15 years, who was admitted to the Pediatric Clinic, presenting pseudoneurological symptoms (weakness, trembling, sweating, dizziness, headaches) and abnormal capillary blood glucose levels, higher than 11,1 mmol/l (> 200 mg/dl) – random determinations. The patient was checking her glucose level 7 - 8 times a day, using test strips.

She is the first borne of a young, healthy couple, out of an uncomplicated pregnancy, birth age: 38 weeks, normal birth in cranial presentation, birth weight: 3350 grams, birth length: 51 cm, physiologic jaundice, breastfeed until 4 months old, rickets prophylaxis and complete immunization scheme. Menarche at 13 years, regular menses.

She has a healthy younger sister, aged 5 years, and a recently deceased grandfather who had Type 2 Diabetes mellitus.

Personal medical history reveals two other hospital admissions for: impaired glucose tolerance, overweight and loss of consciousness. The first admission was 3 years ago for: loss of consciousness accompanied with trembling, sweating and one episode of emesis without nausea.

[1]II nd Clinic of Pediatrics, Timisoara County Clinical Emergency Hospital.
[2]University of Medicine and Pharmacy "Victor Babes" Timisoara.
[3]Pediatric neuropsychiatry - Children's Hospital "Louis Turcanu" Timisoara.
E-mail: mire_med02@yahoo.com, ivelea56@yahoo.com, dumitriusimona@yahoo.com, paulcorina@yahoo.com, georgetanoditi@yahoo.com

The biological tests performed then were relatively normal except the Oral Glucose Tolerance Test (OGTT) which showed an Impaired glucose tolerance (fasting blood glucose level was 5,3 mmol/l (96 mg/dl) and at 120 minutes the glucose level was 7,05 mmol/l (127 mg/dl). The glycemic profile was in normal range. After 3 years, she was again admitted, in another hospital for: dizziness, weakness, headaches, abdominal pain. Laboratory tests were normal and the OGTT is presented in *Table 1 (The 1st test column)*. Her medical history is remarkable for repeated episodes of neurological symptoms accompanied with pathological glucose blood levels, episodes that cannot be proved with medical records. Also she described one episode, a few days prior admission, when she had the same symptomatology. Then she was taken with an ambulance from school to an Emergency Room, where she did some laboratory tests and she was treated with insulin for hyperglycemia (blood glucose value higher than 22,2 mmol/l (> 400 mg/dl). In the same afternoon she was released from hospital. These affirmations were later infirmed by the Emergency Room doctors.

Physical examination at admission: weight = 73 kg, height = 174 cm, BMI = 24,17 kg/m2 s.c. (> 85% CDC 2000 Growth Charts). Waist circumference = 96 cm. Pale skin with latero-abdominal white striae. Excessive adipose tissue with android disposition. The blood pressure and the rest of the clinical examination were normal for her age.

Laboratory test: fasting glycemia (capillary blood) = 10,2 mmol/l (185 mg/dl), *urine = negative ketonuria, absent glycosuria*. Cortisol (8.00 a.m) = 14,7 µg/dl (N = 6,2 – 19,4 µg/dl).

Table 1 – Oral Glucose Tolerance Test (75 g anhydrous Glucose) – the test was performed after an overnight fast of 8 hours, from capillary blood, using strip test and the pacient's own needle

	1st test	**2nd test**	**Normal**	**IGT**	**DM**
Fasting glycemia	5,38 mmol/l	5,33 mmol/l	< 5,7 mmol/l	<6,1 mmol/l	≥ 6,1 mmol/l
At 30 min.	11,05 mmol/l	**29,7 mmol/l**			
At 60 min.	7,5 mmol/l	**13,6 mmol/l**			
At 120 min.	7,38 mmol/l	**9,8 mmol/l**	< 7,8 mmol/l	≥7,8 mmol/l	≥ 11,1 mmol/l
At 180 min.	5,77 mmol/l	**16,6 mmol/l**			

IGT – Impaired Glucose Tolerance
DM – Diabetes Mellitus

ECG and cardiac ultrasound evaluation: normal.
Abdominal ultrasound evaluation: normal
Sella turcica radiography: normal.
Neuropsychological evaluation: normal. Diagnosis : Lipothymia. Recommendations: No medical treatment.

The patient began a normocaloric diet, divided into six meals, with strict adherence to mealtimes and we were monitoring the glucose blood levels (preprandial, 1 hour postprandial and 2 hour postprandial glycemia – *Table 2*). We have to mention that since she has been admitted the pacient hasn't experienced any symptoms in spite of the high glycemic levels.

Table 2: Pre and postprandial glycemia

Hour / **Day**		*6.30*	*7.30*	*8.30*	*13.30*	*14.30*	*15.30*	*18.00*	*19.00*	*20.00*	*0.00*	*3.00*
1	**Glycemia (mmol/l)**	-	-	-	6,66	8,88	7,16	6,66	9.05	**10,66**	**6,22**	**5,72**
2		10,2	**14,6**	**19,3**	**334**	**18,5**	**16,6**	**13,6**	7,6	7,3	**5,83**	**5,5**

Considering the high glucose blood levels during the day, in spite of the normal glycemic levels during the night when the patient was asleep, we decided to determine the value of: HbA1c, C Peptide, Islet Cell Antibody (ICA) and Glutamic acid decarboxylase autoantibodies (GAD Ab). All these determinations were normal: HbA1c = 5%, (N < 6%); C Peptide = 3,4 ng/ml, (N = 1,1 - 4,4 ng/ml); ICA = negative, GAD Ab = negative.

Then we repeated the OGTT, but only this time from *venous blood and under strict supervision*, the results were: fasting blood glucose = 3,6 mmol/l (66 mg/dl), at 30 min = 7,2 mmol/l (131 mg/dl), at 60 min = 6,1 mmol/l (111 mg/dl), at 120 min = 5,4 mmol/l (98 mg/dl), at 180 min = 4,9 mmol/l (89 mg/dl).

Given the medical history, physical examination and the new laboratory tests results, we decided to ask for another neuropsychological evaluation.

The examination, performed by another neuropsychiatrist, reveals: "Cheerful affective disposition, increased interest in describing the symptoms without associating any concern indicators, group integration difficulties, adaptation problems. Separation anxiety regarding her father, mild social anxiety. Poor ability to resolve problems and low emotional self-regulation. Impulsivity, egocentrism, without any prosexic and memory difficulties, no perception deficit. Mild global executive functions deficit. *Conclusion:* Somatoform disorder.

Tendency to develop disharmonic personality - emotional unstable personality structure"

Discussions

Children and adolescents suspected of having somatisation disorders present a challenge to pediatricians. Somatoform disorders present with somatic complaints and/or dysfunctions that are not under conscious control and for which physical findings are absent or insufficient to explain all complaints. These disorders include body dysmorphic disorder, conversion disorder, hypochondriasis, somatisation disorder, and somatoform pain disorder (3).

Differential diagnosis of pediatric somatisation is made with: 1) Unrecognized physical disease (e.g. multiple sclerosis, endometriosis etc); 2) Unrecognized psychiatric disorder (depression, anxiety); 3) Factitious disorder / Munchausen by proxy syndrome; 4)Psychological factors influencing a medical condition (11). Distinguishing somatoform disorders from factitious disorders can be difficult: in the former, symptoms are associated with *unconscious* conflict, whereas in the latter the unconscious need to be cared for motivates the falsification of symptoms. However, in clinical practice, the boundaries between factitious disorder, somatisation and malingering are often unclear and there is also a strong association with personality disorder.

Treatment of these patients, who repeatedly succeed in subjecting themselves to invasive and sophisticated procedures is difficult because of minimal compliance. Unless the underlying psychopathology is recognized by the treating physician, chronic somatisation may result in high cost to the health care system due to frequent use and unnecessary biochemical and radiographic evaluation.

With appropriate intervention, the prognosis for most somatisation disorders in children and adolescents is very good. Sometimes, somatisation is the „tip of the iceberg", that calls attention to a psychiatric disorder necessitating mental health consultation and treatment. Unfortunated many untreated children risk continuous somatisation as adults.

Conclusions

1. Although the psychiatric pathology is an unusual pediatric differential diagnosis we have to think of it every time we have a patient with medically unexplained symptoms.
2. The interdisciplinary pediatric teams are needed in every hospital. Routine neuropsychological evaluation is a very important part in the overall evaluation.
3. Even the strip tests are easy to use, the OGTT test should be done using venous blood as Global IDF / ISPAD Guideline for diabetes recommends.

References

1. Global IDF / ISPAD Guideline for diabetes in childhood and adolescence, 2011, International Diabetes Federation, ISBN 2-930229-72-1.
2. Online Etymology Dictionary. Retrieved June 24, 2012, from Dictionary.com website: http://dictionary.reference.com/browse/hyperglycemia
3. American Psychiatric Association. Task Force on DSM-IV (2000). Diagnostic and statistical manual of mental disorders: DSM-IV-TR. American Psychiatric Pub. ISBN 9780890420256. pp 485.
4. Garralda ME., Unexplained physical complaints, *Pediatr Clin North Am.*; aug 2011; 58(4):803-13.
5. Gregory K. Fritz, Sandra Fritsch, Owen Hagino, Somatoform Disorders in Children and Adolescents: A Review of the Past 10 Years, *Journal of the American Academy of Child & Adolescent Psychiatry***,** October 1997, Volume 36, Issue 10, Pages 1329–1338.
6. Joel E. Dimsdale, Robert Dantzer, A Biological Substrate for Somatoform Disorders: Importance of Pathophysiology, *Psychosomatic Medicine* 2007; 69:850-854.
7. Libow JA, Child and Adolescent illness falsification, *Pediatrics*, 2000, 105: 336 – 342.
8. Konichezky A, Gothelf D., Somatoform disorders in children and adolescents, *Harefuah*, febr 2011;150(2):180-4, 203.
9. Roman R, Zeitler PS., Oral glucose tolerance testing in asymptomatic obese children: more questions than answers, *J Clin Endocrinol Metab*. nov 2008;93(11):4228-30.
10. Sumathipala A., What is the evidence for the efficacy of treatments for somatoform disorders? A critical review of previous intervention studies, *Psychosom Med*. dec. 2007; 69(9):889-900.
11. Thomas Jose Silber, Somatisation Disorders: Diagnosis, Treatment, and Prognosis, *Pediatr. Rev*. 2011; 32; 56-64.

Correspondance to:

Mogoi Mirela
Str. E.Celebi, nr. 1-3,
Timisoara,
România,
E-mail: mire_med02@yahoo.com