BioCore
Exploring Scientific Community

International Journal of Pediatrics and Neonatal Health

ISSN 2572-4355

Research Article Open Access

Type 2 Diabetes in a Nigerian Adolescent: Diagnostic and Management Challenges in a Resource Poor Setting

Oluwayemi IO*[12],Airemionkhale A[2]

[1]Department of Paediatrics, Faculty of Clinical Sciences, College of Medicine, Ekiti State University, Ado-Ekiti, Ekiti State, Nigeria
[2]Department of Paediatrics, Ekiti State University Teaching Hospital, Ado-Ekiti, Ekiti State, Nigeria

Corresponding Author: Oluwayemi IO, Department of Paediatrics, Faculty of Clinical Sciences, College of Medicine, Ekiti State University, Ado-Ekiti, Ekiti State, Nigeria. E-mail: dareoluwayemi@gmail.com

Citation: Oluwayemi IO et al. (2017), Type 2 Diabetes in a Nigerian Adolescent: Diagnostic and Management Challenges in a Resource Poor Setting. Int J Ped & Neo Heal. 1:3, 73-75. DOI: 10.25141/2572-4355-2017-3.0074

Received: May 08, 2017; **Accepted:** June 10, 2017; **Published** July 06, 2017

Abstract:

Type 2 diabetes in children and adolescents is an emerging clinical problem globally in the last three decades. Previously it was thought that type 2 DM does not affect children and adolescents. There is need for high index of suspicion especially in obese children and adolescents who have positive family history of type 2 diabetes. We present a case of a newly diagnosed type 2 DM in a female adolescent previously thought to have type 1 DM. She presented with weight loss, polyuria, polydipsia and polyphagia. She had a positive family history of type 2 DM. She was previously managed as Type 1 DM but when hyperglycaemia was not responding to insulin therapy and fasting serum C-Peptide was within normal limit, a diagnosis of type 2 DM was made and she has since been doing well on oral hypoglycaemic agent

Keywords: Type 2 Diabetes, Adolescents, Diagnosis, Challenges, Resource-Poor Setting

Introduction:

Diabetes mellitus is a group of metabolic disorder characterized by hyperglycaemia, resulting from defects in insulin secretion, inaction or both. Type 2 DM has heterogeneous etiology with social, behavioral and environmental risk factors unmasking the effect of genetic susceptibility.1 The hallmark of type 2 DM is obesity.2 Previously thought to be a rare disorder in children and adolescents, researchers began to report increasing incidence of type 2 DM among Paediatrics age groups in the last three decades worldwide.3 This observation followed increased prevalence of obesity among children and adolescents globally.4,5 Overweight is presently becoming a major health challenge among Paediatrics age group in both developed and developing countries.5,6 Often there is family history of type 2 DM in first degree relatives among 74% – 100% of children and adolescent having type 2 DM.2 There is paucity of data on type 2 DM among African children and adolescents whereas the prevalence of childhood and adolescent type 2 DM in the United State7 is 12:100000 and in Europe8 2.5:100000

Case report

We report the case of newly diagnosed type 2 diabetes in a 12 year old girl who presented at a private hospital owned by a Paediatric Endocrinologist in Ekiti State, South West, Nigeria.

She was referred from another private hospital where she was noticed to have high blood glucose with difficulty controlling the blood glucose with subcutaneous insulin. She presented with 3 month history of excessive thirst, excessive hunger, frequent urination and weight loss. Body weight before illness was greater than 70 kg (obese; >95th percentile for age and sex) but at presentation she was weighing 54kg (between 75th & 90th percentile). Her mother is diabetic (Type 2 DM). There was no history of autoimmune diseases in the family. She was the 3rd of three children in a monogamous family setting. Elder siblings (23 year old male and 19 year old female were alive and well). Her father was a 50 year old business man, while mother was a 50 year old business woman.

Essential findings at presentation were: not acutely ill looking girl, afebrile (36.3ºC), no sign of dehydration, no acanthosis negrican.

She had a low normal pulse rate of 86/min and BP of 90/50, normal heart sounds, sexual maturity rating stage II for both breasts and pubic hair. She was conscious and alert, well oriented in time, place and person.

RBG= 21.8mmol/L, blood ketone ++++, urinalysis: glucose ++++, protein nil, pH 5.0.

Hyperglycemia was corrected with soluble insulin infusion at 0.1 unit/ kg/ hr and intravenous fluid (IVF) at 8% deficit plus maintenance. The initial IVF was normal saline till blood glucose reduced to < 13.8mmol/L; then, it was changed to 5% Dextrose saline and insulin infusion reduced to 0.05 unit/ kg/ hr. When RBG was < 8.3mmol/L, insulin infusion was reduced to 0.025 unit/ kg/ hr.

Blood glucose normalized within 10 hours of insulin infusion. Thereafter, patient was commenced on subcutaneous insulin and she was commenced on diabetic education, self-blood glucose monitoring, and how to adjust insulin dose and correct hyperglycemia with subcutaneous soluble insulin.

She was managed as type 1 DM and discharged home after 3 days on admission on Mixtard 70/30 at 0.8 unit/ kg/ day (45 units/ day; 30 unit am, 15 unit pm).estimated from total soluble insulin needed to achieve euglycaemia in the patient.

On review 2nd day after discharge, patient was observed to be having hyperglycemia in the range of 16.7 -22.2mmol/L the previous day which was corrected at home with subcutaneous soluble insulin.

At this point a possibility of type 2 DM was entertained and patient was commenced on oral hypoglycemic agent (Metformin 500mg bd) and blood sample was sent for C-Peptide which was within normal range 1.2ng/ml (0.7 - 1.9ng/ml) suggesting that patient was producing insulin. The diagnosis of Type 2 DM was therefore established and patient was managed as such; subcutaneous insulin was discontinued. The patient has since been well controlled on oral hypoglycemic agent, diet and exercise.

Discussion

Type 2 DM is 1.7 times more likely in girls than in boys9 and the peak age for presentation in children is mid-puberty.10 Puberty appear to play a major role in the pathogenesis of type 2 DM in adolescents.10 During puberty there is increased growth hormone secretion which causes increased resistance to the action of insulin resulting in hyperinsulinaemia. Also it has been documented that insulin mediated glucose disposal is 30% lower in adolescents between Tanner stage II and IV compared to pre-pubertal children and adults.11 Our patient is a female, and with sexual maturity rating Tanner stage II.

Obesity is the hallmark of type 2 DM and most patients have 1st degree relatives with type 2 DM.2 Our patient's weight was > 95th percentile at the onset of the symptoms and was nearly overweight at presentation. Her mother was also having type 2 DM.

Unlike in type 1 DM where there is deficiency or absolute lack of insulin, in type 2 DM there is resistance to insulin and this was demonstrated by persistent hyperglycaemia despite administration of insulin until Metformin was given. Also C-peptide, a branch of insulin chain was demonstrated to be within normal limit in the index patients. Other investigations (such as fasting insulin, β cell auto antibodies) to rule out type 1 DM could not be done because of financial constraint. In most patients with type 2 DM diagnosis can be made reliably on clinical presentation and course as with our index patient.2

Type 2 DM can be asymptomatic in the mildest form when it can be detected by routine fasting blood glucose12, 13 and urinalysis in at risk group (obese adolescents with family history of type 2 DM, and acanthosis negrican, a sign of insulin resistance).2 And in the severe form patients presents with classical symptoms of weight loss, polyuria, polyphagia, polydipsia like index patient.

Treatment of type 2 DM in children and adolescents is mainly by lifestyle modification, exercise and use of oral hypoglycemic agent (Metformin)13, 14 which also has additional benefit of weight reduction and decrease in lipids without risk of hypoglycemia.13 Our index patient has remained euglycemic and clinically stable on Metformin and lifestyle modification. Subcutaneous insulin may also be indicated if hyperglycaemia persists.13 Emphasis must however be on prevention especially in resource poor countries with limited resources for early diagnosis, treatment and management of complications.

References

1. Kiess W, Böttner A, Raile K, Kapellen T, Müller G, Galler A, Paschke R, Wabitsch M. Type 2 diabetes mellitus in children and adolescents: a review from a European perspective. Horm Res 2003; 59 Suppl 1: 77-84 [PMID: 12566725 DOI:10.1159/000067829]

2. Rosenbloom AL, Silverstein JH, Amemiya S, Zeitler P, Klingensmith GJ. ISPAD Clinical Practice Consensus Guidelines 2006-2007. Type 2 diabetes mellitus in the child and adolescent. Pediatr Diabetes 2008; 9: 512-526 [PMID: 18694453 DOI: 10.1111/j.1399-5448.2008.00429.x]

3. Arslanian S. Type 2 diabetes in children: clinical aspects and risk factors. Horm Res 2002; 57 Suppl 1: 19-28 [PMID:11979018 DOI: 10.1159/000053308]

4. Lobstein T, Frelut ML. Prevalence of overweight among children in Europe. Obes Rev 2003; 4: 195-200 [PMID:14649370 DOI: 10.1046/j.1467-789X.2003.00116.x]

5. Han JC, Lawlor DA, Kimm SY. Childhood obesity. Lancet 2010; 375: 1737-1748 [PMID: 20451244 DOI: 10.1016/ S0140-6736(10)60171-7]

6. Oluwayemi I.O. Pattern of BMI Percentiles among Adolescents in a semi-urban Community in South-Western Nigeria. Pakistan Journal of Nutriti2015; 14 (9): 570-574

7. Dabelea D, Bell RA, D'Agostino RB, Imperatore G, Johansen JM, Linder B, Liu LL, Loots B, Marcovina S, Mayer-Davis EJ, Pettitt DJ, Waitzfelder B. Incidence of diabetes in youth in the United States. JAMA 2007; 297: 2716-2724[PMID: 17595272 DOI: 10.1001/jama.297.24.2716]

8. Schober E, Holl RW, Grabert M, Thon A, Rami B, Kapellen T, Seewi O, Reinehr T. Diabetes mellitus type 2 in childhood and adolescence in Germany and parts of Austria. Eur J Pediatr 2005; 164: 705-707 [PMID: 16012857 DOI: 10.1007/s00431-005-1709-9]

9. America Diabetes Association; type 2 diabetes in children and adolescents. America Diabetes Association. Diabetes Care. 2000; 23 (3):381-9

10. Arslanian SA. Type 2 diabetes mellitus in children: pathophysiology and risk factors. J Pediatr Endocrinol Metab 2000; 13 Suppl 6: 1385-1394 [PMID: 11202214]

11. Pinhas-Hamiel O, Lerner-Geva L, Copperman NM, Jacobson MS. Lipid and insulin levels in obese children: changes with age and puberty. Obesity (Silver Spring) 2007; 15: 2825-2831 [PMID: 18070774 DOI: 10.1038/oby.2007.335]

12. I. O. Oluwayemi, S. J. Brink, E. E. Oyenusi, O. A. Oduwole, M. A. Oluwayemi. Fasting Blood Glucose Profile among Secondary School Adolescents in Ado-Ekiti, Nigeria. Journal of Nutrition and Metabolism. Volume 2015 (2015), Article ID 417859, 4 pages. http://dx.doi.org/10.1155/2015/417859

13. Reinehr T. Type 2 diabetes mellitus in children and adolescents. World J Diabetes 2013; 4(6): 270-281

14. Jones KL, Arslanian S, Peterokova VA, Park JS, Tomlinson MJ. Effect of metformin in pediatric patients with type 2 diabetes: a randomized controlled trial. Diabetes Care 2002; 25: 89-94 [PMID: 11772907 DOI: 10.2337/diacare.25.1.89]