

# Early Onset of Type 2 Diabetes Mellitus - An Unusual Presentation- A Case Report

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## ABSTRACT

Diabetes mellitus includes heterogeneous mixtures of metabolic diseases characterized by chronic hyperglycemia. The most common type of DM are type I and type 2 but there are other type, MODY (Maturity onset DM in young) is a clinically heterogeneous diseases characterized by non dependent insulin DM diagnosed early with dominant autosomal transmission and absence of autoimmune antibodies. The present case, a 15 year old girl slightly obese (69 Kg) and BMI higher than normal (25.2), family history of DM, with RBS 640 mg/dl could have been diagnosed as MODY. Sequence analysis of HNF1 $\alpha$ , HNF4 $\alpha$  clearly ruled out this case to be MODY. Absence of autoimmune antibodies showed it is not type I. It is a case of type 2 DM presented at an early age of 15years. The physician should be thoroughly aware of the difference between type I, MODY and type 2 so that the correct diagnosis and treatment can be given to the patient to avoid unnecessary complications.

**Key Words:** Early onset of Diabetes Mellitus, Type 2DM, HbA1c, MODY.

## INTRODUCTION

Diabetes is a global health concern. It is a metabolic disease of multiple etiologies, characterized by hyperglycemia resulting from defect in insulin secretion or action or both causing metabolic disturbances. According to International Diabetic Federation Atlas [1, 5] - India stands second in the world with respect to the prevalence of diabetes mellitus. Diabetes mellitus is broadly classified into Type I and Type 2. Type I diabetes results from cellular mediated autoimmune dysfunctions of  $\beta$ -cells of pancreas [2]. In type 2 diabetes mellitus the body is able to produce insulin but either this is insufficient or the body is unable to respond to its effect resulting in hyperglycemia. It is the most common type and usually seen in adults. There is pancreatic dysfunction, monogenic mutation and secondary forms of diabetes mellitus [3].

MODY is a clinically and genetically heterogeneous group of monogenic disorder causing diabetes

mellitus in the younger population. Sequencing is widely used genetic diagnostic platform for identifying MODY and the technique is 99% sensitive in identifying MODY. Only 3 major genes - HNF1 $\alpha$ , HNF4 $\alpha$  and glucokinase are sequenced. Genetic diagnosis has huge treatment and prognostics benefits [4].

## CASE REPORT

A 15 year old girl came to the hospital OP for gynecological complaint of not getting periods. Routine investigations were carried out. She has height 165 cms, weight 69 kg and BMI 25.3. Her father and grandfather were type 2 diabetic patients. The blood picture showed Hb13.9g%, TC 13600cells /cumm, Poly 56%, Lymph 38%, Eosin 04% and PCV 39.0% Platelet count 3.4 lakhs /cumm, ESR 15mm/hr. Lipid profile, RFT, LFT -normal. HbA1c 16.1%.

On 1<sup>st</sup> July 2019 her GRBS was 636 mg/dl. The clinician started the treatment with a combination of Glycomet and

Humalog mix .The review was on 20<sup>th</sup> July 2019 FBS 104 mg/dl and PPBS 89 mg/dl. On 4<sup>th</sup> August 2019 FBS 108mg/dl PPBS 85mg/dl and HbA1c 11.9%. Hence the diabetes has been controlled .On October 5<sup>th</sup> 2019 HbA1c 5.9%, FBS 95 mg/dl and PPBS 85mg/dl .Insulin was stopped.

Onset of DM at a young age, obesity (69 Kg) and BMI 25.3, slightly above normal limits might lead to the conclusion that it may be MODY [5]. But the MODY calculator [5] does not support this view. The sequence analysis report from Madras Diabetic Research Foundation conclusively proved it is not MODY .The present case could be type 2 diabetes at an early age.

## DISCUSSION

Type 2 DM is considered as diseases of older adults where as type I DM is a diseases of the children. Type I is an autoimmune disorder with evidence of autoimmune antibodies. Immune dysfunction which is due to pancreas can be confirmed by autoantibody to Glutamic acid decarboxylase (GADA).In addition there is decreased cell function .There will be decreased C-peptide [6] i.e. (<0.8 ng/ml) on the other hand, in this case C-peptide is 1.18 slightly above normal Anti IA-2 normal. Islet Cell antibody negative which rule out the possibility of type I DM. Cardio c-reactive protein is within normal limits <0.3ng/L. C-peptide 1.16 (Reference range 0.81- 3.85ng/dL) and HDL- cholesterol 47 mg/dl. No mutation at *HNF1 $\alpha$*  and *HNF4 $\alpha$*  genes- MODY 1 or MODY 3 mutation absent not found at entire coding regions and exon - introns boundaries of *HNF4A* and *HNF1A* [4].

The only possibility is that it can be type 2 DM which is due to insulin resistance and diminished  $\beta$ -cells function. Wilmot E and Idris I [6] stated that patient with early onset of type 2 DM should be cared for in a multidisciplinary specialist diabetic clinic with access to dietary and structured health education is a cost effective acceptable way of empowering those patients self management skills. Moreover management

of obesity through diet planning and physical activity should be fulfilled. Proper health education and patients compliance to medication, diet and physical exercise are essential for successful management of early onset type 2 DM. Also primary care physician should be well aware to differentiate between Type I, MODY and type 2 DM for the proper diagnosis program and treatment.

## CONCLUSION

DM is a metabolic disorder characterized by hyperglycemia. It can be type I, MODY, type 2 variety. Type I can be identified by autoimmune antibodies, MODY by sequence analysis of the codon for *HNF1 $\alpha$* , *HNF4 $\alpha$*  and Glucokinase and also by physical factor like age, obesity, family history of DM and BMI. The present case based on the above characteristics could have been misdiagnosed as MODY but biochemical markers and genetic analysis conclusively prove it as a case of early onset of type 2 DM.

Primary care physicians need to be aware that type 2 DM is not necessarily adult on set .They should be educated by counseling, CME and seminars as the proper diagnosis of the type of Diabetes for proper treatment schedule.

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