



MODY Diabetes: A Hospital Series of 12 Cases

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Introduction

Maturity Onset Diabetes of the Young (MODY) is a heterogeneous group of monogenic diabetes characterized by a primary defect in insulin secretion. It is a rare form of diabetes caused by autosomal dominant mutations, usually non-autoimmune and non-ketotic [1, 2]. The diagnosis of MODY is often overlooked in resource-limited countries, notably due to the frequent unavailability of genetic testing [3].

This study reports a series of 12 suspected MODY diabetes cases managed at the Endocrinology Department of Ibn Sina University Hospital in Rabat, aiming to describe their clinical, evolutionary, and diagnostic characteristics.

Materials and Methods

This retrospective study included 12 patients hospitalized for suspected MODY diabetes between 2017 and 2022. Diagnostic criteria were: young age at diagnosis (<35 years), family history of diabetes across at least three generations, absence of autoimmunity (negative anti-GAD antibodies), normal body mass index (BMI), and clinical evolution compatible with non-insulin-dependent diabetes [4].

Clinical, biological, and therapeutic evaluations were conducted without molecular genetic testing due to its local unavailability.

Results

Twelve patients were included, with a mean age of 24 years and a sex ratio of 0.6 (female predominance). All had a family history of diabetes spanning three generations. Mean BMI was within the normal range, and all patients tested negative for type 1 diabetes autoantibodies.

- Clinically, 9 patients did not present with diabetic ketosis, while 2 cases were revealed by diabetic ketoacidosis (DKA) and 1 by mild ketosis associated with onychomycosis.
- Treatment with sulfonylureas (glibenclamide) achieved satisfactory glycemic control in 10 patients, whereas 2 patients required conversion to insulin therapy due to persistent glycemic imbalance.
- Additionally, 4 patients had a solitary kidney, suggesting possible MODY 5, a subtype associated with congenital renal anomalies [5].

MODY Subtypes Overview

MODY diabetes includes several clinically and genetically distinct subtypes. Table 1 summarizes the main characteristics of the most frequent MODY subtypes, their implicated genes, typical age at diagnosis, clinical manifestations, and recommended treatments. This classification aids diagnosis and management, especially in settings without available genetic testing like ours.

Discussion

The diagnosis of MODY relies on a combination of clinical and biological criteria, with genetic confirmation often unavailable, particularly in resource-limited countries like Morocco. Presentation in young subjects, dominant family history, and absence of obesity are key indicators [6].

Appropriate management is essential, especially the good response to sulfonylureas, which can avoid unnecessary insulin therapy [7]. Family screening is mandatory to identify other cases and optimize care [8].

The association of renal anomalies such as solitary kidney in some patients points toward MODY

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Table 1: Summary comparison of major MODY subtypes: clinical features, genetics, and therapeutic management [1, 6].

MODY Subtype	Gene Involved	Age at Diagnosis	Clinical Features	Recommended Treatment
MODY 1	HNF4A	Childhood to young adult	Possible neonatal hypoglycemia, progressive diabetes	Sulfonylureas
MODY 2	GCK	Childhood	Mild stable hyperglycemia, often asymptomatic	Often no treatment necessary
MODY 3	HNF1A	Young adult	Progressive diabetes, sensitivity to sulfonylureas	Sulfonylureas
MODY 5	HNF1B	Childhood to young adult	Diabetes with renal anomalies (solitary kidney, cysts)	Insulin therapy often required

5 subtype, linked to mutations in the HNF1B gene, highlighting the need for systematic assessment of extra-pancreatic manifestations in this pathology [9].

Conclusion

MODY diabetes is a rare entity posing significant diagnostic challenges, especially where genetic tests are unavailable. It should be suspected in young diabetic patients with normal BMI and a strong multigenerational family history. Effective management with sulfonylureas can significantly improve clinical outcomes. Early diagnosis and family screening are essential to optimize treatment and prevent complications.

References

1. Fajans SS, Bell GI. MODY: history, genetics, pathophysiology, and clinical decision making. *Diabetes Care*. 2011; 34(8): 1878-84.

2. Pearson ER. Personalized medicine in diabetes: the role of 'Maturity Onset Diabetes of the Young'. *Clin Med (Lond)*. 2019; 19(1): 39-44.

3. Shields BM, Hicks S, Shepherd MH, Colclough K, Hattersley AT, Ellard S. Maturity-onset diabetes of the young (MODY): how many cases are we missing? *Diabetologia*. 2010; 53(12): 2504-8.

4. Tattersall RB. Mild familial diabetes with dominant inheritance. *Q J Med*. 1974; 43(169): 339-57.

5. Bellanné-Chantelot C, Chauveau D, Gautier JF, et al. Clinical spectrum associated with hepatocyte nuclear factor-1beta mutations. *Ann Intern Med*. 2004; 140(7): 510-7.

6. Murphy R, Ellard S, Hattersley AT. Clinical implications of a molecular genetic classification of monogenic β -cell diabetes. *Nat Clin Pract Endocrinol Metab*. 2008; 4(4): 200-13.

7. Steele AM, Shields BM, Wensley KJ, et al. Prevalence of vascular complications among patients with glucokinase mutations and prolonged mild hyperglycemia. *JAMA*. 2014; 311(3): 279-86.

8. Fajans SS, Olson L, Mackenzie T, et al. Mutations in the hepatocyte nuclear factor-1 alpha gene in maturity-onset diabetes of the young. *N Engl J Med*. 1996; 335(16): 1151-6.

9. Horikawa Y, Iwasaki N, Hara M, et al. Mutation in hepatocyte nuclear factor-1beta gene (TCF2) associated with MODY. *Nat Genet*. 1997; 17(4): 384-5.