

Maturity-Onset Diabetes of the Young (MODY): A Review of Its Genetic Basis, Clinical Features, Diagnosis, Management and Nutritional Considerations

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Abstract

Maturity-Onset Diabetes of the Young (MODY) is a group of inherited forms of diabetes caused by changes in a single gene that affects pancreatic beta-cell function or insulin secretion. Unlike the more common forms of diabetes, MODY often develops at a younger age and may occur in several generations of the same family. Because its clinical presentation can overlap with type 1 or type 2 diabetes, many individuals remain incorrectly classified for years. Correct identification of MODY is important because different genetic forms have different patterns of blood glucose elevation, complications and responses to treatment. This review summarizes the genetic basis, major clinical characteristics, diagnosis, management and nutritional considerations of MODY. Published literature was reviewed using scientific databases and major diabetes guidelines. Particular attention was given to commonly reported forms involving the GCK, HNF1A, HNF4A and HNF1B genes, together with evidence from Indian populations. The reviewed literature indicates that genetic testing has an important role in confirming the diagnosis and selecting appropriate treatment. Some forms, such as GCK-MODY, may require little or no glucose-lowering treatment outside specific situations, whereas HNF1A-MODY and HNF4A-MODY commonly show good responses to sulfonylureas. Nutritional management remains an important part of overall diabetes care, although dietary advice should be individualized according to the specific MODY subtype, treatment and metabolic status. Greater awareness among healthcare professionals and improved access to genetic testing may help reduce misdiagnosis and support more appropriate long-term management.

Key words: Maturity-Onset Diabetes of the Young, MODY, Monogenic Diabetes, Genetic Diabetes, Young-Onset Diabetes, Hereditary Diabetes, Beta-Cell Dysfunction, Insulin Secretion, Glucokinase, GCK-MODY, HNF1A-MODY, HNF4A-MODY, HNF1B-MODY, Genetic Testing, Molecular Diagnosis, Precision Medicine, Diabetes Management, Medical Nutrition Therapy, Family History, Autosomal-Dominant Inheritance.

1. INTRODUCTION

Diabetes mellitus is a heterogeneous group of metabolic disorders characterized mainly by persistent elevation of blood glucose. Type 1 and type 2 diabetes are the most frequently recognized forms, but diabetes can also result from single-gene defects, pancreatic diseases, endocrine disorders and other specific causes. Monogenic diabetes represents a group of diabetes conditions caused by alterations in a single gene. Maturity-Onset Diabetes of the Young (MODY) is one of the best recognized forms of monogenic diabetes and is primarily associated with impaired pancreatic beta-cell function and insulin secretion [1][2].

MODY was initially recognized because some children, adolescents and young adults developed relatively mild diabetes that did not resemble the usual pattern of juvenile diabetes. Subsequent research established that these conditions could be inherited in an autosomal-dominant pattern and were associated with specific genetic abnormalities. Advances in molecular genetics have subsequently identified several genes responsible for MODY-like forms of diabetes [3].

MODY is important clinically because its presentation can overlap with both type 1 and type 2 diabetes. A young person with hyperglycemia may be diagnosed with type 1 diabetes and started on insulin, or may be considered to have type 2 diabetes when features such as obesity or insulin resistance are present. However, a strong family history, preserved endogenous insulin secretion, absence of typical autoimmune markers and the pattern of hyperglycemia may raise suspicion of monogenic diabetes [1][4].

The current understanding of MODY has also changed with advances in genetic testing. The term MODY continues to be widely used, but modern literature increasingly emphasizes the responsible gene rather than relying only on numerical MODY subtypes. This approach is useful because different genetic forms have different clinical courses and treatment requirements [5].

The purpose of this review is to discuss the genetic basis, clinical features, diagnosis, major forms, treatment and nutritional considerations of MODY, with particular attention to evidence relevant to Indian populations.

2. METHODOLOGY

This review was prepared by collecting and analyzing published scientific literature related to Maturity-Onset Diabetes of the Young and monogenic diabetes. The methodology was designed as a literature-based secondary research study and no primary patient data were collected.

A search of relevant scientific literature was carried out using databases and authoritative sources including PubMed, PubMed Central, major peer-reviewed journals and diabetes practice guidelines. Publications related to MODY genetics, clinical presentation, diagnosis, genetic testing, treatment, complications and nutritional management were considered.

Search terms included “Maturity-Onset Diabetes of the Young,” “MODY,” “monogenic diabetes,” “MODY genetics,” “MODY diagnosis,” “MODY treatment,” “MODY nutrition,” “HNF1A diabetes,” “HNF4A diabetes,” “GCK diabetes,” “HNF1B diabetes,” and “MODY India.”

Published review articles, systematic reviews, clinical studies, genetic studies and recognized clinical guidelines were considered when they were directly relevant to the objectives of the review. Articles dealing mainly with unrelated forms of diabetes or lacking sufficient relevance to MODY were excluded. Duplicate publications were also avoided.

The selected literature was reviewed for information regarding genetic causes, inheritance patterns, clinical characteristics, diagnostic approaches, treatment responses, complications and nutritional implications. Particular attention was given to studies describing Indian patients because MODY remains under-recognized in many Asian populations.

The findings were organized and synthesized under major themes rather than statistically pooled. Since the available studies differ considerably in design, population, genetic subtype and clinical characteristics, a narrative synthesis was considered more appropriate than a meta-analysis.

3. REVIEW OF LITERATURE

Jang (2020): Maturity-onset diabetes of the young—update and perspectives on diagnosis and treatment

Jang reviewed the current understanding of MODY with particular attention to diagnosis, genetic testing and treatment. The article described MODY as a clinically heterogeneous group of monogenic disorders involving beta-cell dysfunction. It also discussed the difficulty of distinguishing MODY from type 1 and type 2 diabetes using clinical findings alone [3].

The review highlighted the importance of genetic testing in confirming the diagnosis and described several major MODY genes. It also emphasized that the distribution of MODY-related genetic variants may differ between Asian and Western populations. This observation is relevant to countries such as India, where more population-specific genetic information is still needed.

Nkonge et al. (2020): The epidemiology, molecular pathogenesis, diagnosis, and treatment of MODY

Nkonge and colleagues reviewed literature published over several decades to describe the epidemiology, molecular mechanisms, diagnosis and treatment of MODY. The authors emphasized that MODY is clinically and genetically heterogeneous and is generally associated with young-onset hyperglycemia, preserved pancreatic function and an autosomal-dominant pattern of inheritance [6].

The review also discussed the possibility of misdiagnosis and the importance of identifying appropriate individuals for genetic testing. The authors noted that increasing awareness and improved genetic testing could contribute to better recognition of monogenic diabetes.

Delvecchio, Pastore and Giordano (2020): Treatment Options for MODY Patients

Delvecchio and colleagues conducted a systematic review of published literature concerning treatment of genetically confirmed MODY. Their work demonstrated that treatment cannot be considered identical across all MODY forms because the response to glucose-lowering therapy depends on the underlying genetic defect [7].

The review particularly highlighted GCK-MODY, where glucose-lowering treatment is generally unnecessary outside particular circumstances such as some pregnancies. In contrast, patients with HNF1A-

MODY and HNF4A-MODY commonly respond well to sulfonylureas. The article therefore supports the idea that molecular diagnosis can directly influence treatment decisions.

Younis et al. (2022): MODY—Mutations, Physiological Consequences, and Treatment Options

Younis and colleagues reviewed the genetic mutations associated with MODY and discussed how these mutations influence pancreatic beta-cell function and insulin secretion. The review described the clinical heterogeneity of MODY and the difficulties created by overlap with more common forms of diabetes [8]. The authors also discussed the major genetic subtypes and their treatment implications. Their work supports the use of molecular diagnosis as an important component of precision diabetes care.

Tosur and Philipson (2022): Precision Diabetes—Lessons Learned from MODY

Tosur and Philipson examined MODY from the perspective of precision medicine. They described how advances in molecular diagnosis have improved understanding of the relationship between a specific genetic defect, clinical presentation and therapeutic response [9].

The review emphasized that GCK-MODY, HNF1A-MODY and HNF4A-MODY represent the most commonly encountered forms and that treatment responses vary substantially between subtypes. The authors also noted that many people with MODY may be incorrectly diagnosed with type 1 or type 2 diabetes.

Aarthy et al. (2023): MODY in South India

Aarthy and colleagues conducted an important study at a tertiary diabetes centre in South India. Among 530 individuals clinically suspected of having MODY, 58 were genetically confirmed, giving a pickup rate of 10.9% among those selected for testing [10].

HNF1A-MODY was the most common subtype in the study, followed by HNF4A-MODY, ABCC8-MODY, GCK-MODY and HNF1B-MODY. The study provides useful evidence from the Indian population and demonstrates that genetic testing can identify clinically relevant MODY subtypes among people initially suspected of having the condition.

The study is particularly important for the present review because it shows that MODY is not only a theoretical genetic condition but is also being identified in Indian clinical practice. The findings support greater clinical awareness and appropriate selection of patients for genetic testing.

Sharma et al. (2025): Monogenic Diabetes and Therapeutic Management of MODY Subtypes

Sharma and colleagues provided a recent comprehensive review of monogenic diabetes and therapeutic management of MODY subtypes. The review brought together information on the genetic basis, clinical characteristics and treatment of different forms of MODY [11].

The article further supports subtype-specific management rather than treating all MODY cases as one uniform disorder. Its recent publication also reflects the continued development of precision approaches to monogenic diabetes.

4. GENETIC BASIS AND CLASSIFICATION OF MODY

MODY is caused by pathogenic changes in a single gene that interfere with normal pancreatic beta-cell function, insulin secretion or related metabolic pathways. Most established forms follow an autosomal-dominant pattern, meaning that an affected individual can transmit the altered gene to offspring. However, the absence of an obvious family history does not completely exclude a genetic diagnosis because new mutations and incomplete recognition of affected relatives can occur [1][6].

The genetic classification of MODY has changed over time. Earlier literature commonly used numerical labels such as MODY1, MODY2 and MODY3. Current practice increasingly uses the responsible gene because this directly communicates the biological basis of the disease. For example, GCK-MODY is used for glucokinase-related diabetes and HNF1A-MODY for diabetes caused by HNF1A variants [5].

Table 1. Major forms of MODY and their general characteristics

MODY form	Main gene	Important clinical characteristics	General treatment consideration
GCK-MODY	GCK	Mild, stable fasting hyperglycemia	Usually no glucose-lowering treatment outside specific situations
HNF1A-MODY	HNF1A	Progressive beta-cell dysfunction; young-onset diabetes; glucosuria may occur	Often highly responsive to sulfonylureas
HNF4A-MODY	HNF4A	Progressive insulin secretory defect; may be associated with macrosomia and transient neonatal hypoglycemia	Often responsive to sulfonylureas
HNF1B-MODY	HNF1B	Diabetes with renal, genitourinary or pancreatic abnormalities	Management depends on pancreatic and extra-pancreatic features

The above forms are among the best characterized MODY subtypes. Other rare genetic forms have also been described, demonstrating the considerable genetic diversity of monogenic diabetes [1][8].

5. CLINICAL FEATURES OF MODY

MODY commonly presents with diabetes or hyperglycemia at a younger age than typical type 2 diabetes. Traditionally, onset before 25 years was emphasized, although diagnosis may occur considerably later because affected individuals may remain undiagnosed for years [1].

A strong family history is an important clue, especially when diabetes appears in successive generations. This pattern can suggest autosomal-dominant inheritance. However, family history should not be used as the only criterion because incomplete family information, misclassification of diabetes and new genetic variants may obscure the inheritance pattern.

Other features that may increase suspicion include:

- Diabetes diagnosed at a young age.
- Diabetes in several successive generations.
- Absence of typical features of type 1 diabetes.

- Negative diabetes-associated autoantibodies in an appropriate clinical context.
- Preserved endogenous insulin secretion.
- Mild and stable fasting hyperglycemia.
- Lack of marked insulin resistance in many affected individuals.
- Clinical features characteristic of a particular genetic subtype.
- Diabetes accompanied by renal or genitourinary abnormalities in suspected HNF1B-MODY.

The 2026 American Diabetes Association Standards of Care recommends considering MODY genetic testing in children and young adults who do not have typical characteristics of type 1 or type 2 diabetes and who have diabetes in successive generations suggestive of autosomal-dominant inheritance [1].

6. DIAGNOSIS OF MODY

Diagnosis of MODY requires a combination of clinical assessment, biochemical investigation and, when indicated, genetic testing. No single clinical feature can establish the diagnosis.

Initially, the patient's age at diagnosis, family history, body composition, clinical characteristics and diabetes progression should be evaluated. Autoantibody testing may help distinguish autoimmune type 1 diabetes from other forms when clinically appropriate. Assessment of endogenous insulin secretion, including C-peptide in selected patients, may also provide useful information.

A genetic test is required to confirm the specific monogenic cause. Depending on the clinical situation, testing may involve targeted analysis of a suspected gene or a broader monogenic diabetes gene panel. Next-generation sequencing has increased the ability to examine multiple genes in a single testing strategy [1][9].

Correct diagnosis is important because a person with MODY may otherwise remain on treatment designed for type 1 or type 2 diabetes despite having a genetic subtype with a different natural history and therapeutic response.

Genetic diagnosis can also have implications for family members. Because many forms of MODY are inherited in an autosomal-dominant manner, identifying one affected person may lead to recognition of diabetes or genetic risk in relatives. Genetic counselling is therefore an important part of appropriate management.

7. MAJOR TYPES OF MODY

GCK-MODY

GCK-MODY results from pathogenic variants in the glucokinase gene. Glucokinase functions as a glucose sensor in pancreatic beta cells and helps regulate glucose-stimulated insulin secretion. Alteration of this pathway results in a higher glucose threshold for insulin release.

Patients generally have mild, stable and non-progressive fasting hyperglycemia. Because the hyperglycemia is relatively stable and the risk of complications is generally low, glucose-lowering medication is usually not required in nonpregnant individuals [1][7].

Pregnancy requires special consideration because maternal GCK-MODY can have different implications depending on the fetal genotype and fetal growth. Management should therefore be supervised by an appropriate diabetes and obstetric team.

HNF1A-MODY

HNF1A-MODY is caused by variants in the hepatocyte nuclear factor 1-alpha gene. It is associated with progressive impairment of insulin secretion and commonly becomes clinically apparent during adolescence or early adulthood.

One characteristic feature is an increased tendency toward urinary glucose loss because of a lower renal threshold for glucose. Individuals with HNF1A-MODY are often particularly sensitive to sulfonylureas, which is one reason why identifying this subtype can substantially alter treatment [1][7].

HNF4A-MODY

HNF4A-MODY is caused by pathogenic variants in the HNF4A gene and produces progressive beta-cell dysfunction. Some affected individuals have a history of increased birth weight or transient neonatal hypoglycemia.

Like HNF1A-MODY, HNF4A-MODY commonly responds well to sulfonylureas. However, treatment should be individualized according to the clinical situation and monitored by a qualified healthcare professional [1][7].

HNF1B-MODY

HNF1B-MODY differs from several other common forms because diabetes may occur together with abnormalities outside the pancreas. Renal cysts, renal developmental abnormalities, genitourinary abnormalities, pancreatic abnormalities, hyperuricemia and gout may occur.

The presence of diabetes together with characteristic renal or genitourinary findings should increase suspicion of HNF1B-related disease. Management may therefore require attention to both diabetes and associated organ involvement [1].

8. MANAGEMENT AND TREATMENT OF MODY

Management of MODY should be based on the genetic subtype, degree of hyperglycemia, age, pregnancy status, complications and overall clinical condition. There is no single treatment suitable for every form of MODY.

1) GCK-MODY

GCK-MODY generally produces mild, stable hyperglycemia. Most nonpregnant individuals do not require glucose-lowering medication. Unnecessary treatment may expose patients to medication-related risks without providing a clear benefit [1][7].

2) HNF1A-MODY

Patients with HNF1A-MODY often show a strong response to low doses of sulfonylureas. Treatment therefore differs from the insulin-centered approach commonly used for type 1 diabetes [7].

3) HNF4A-MODY

HNF4A-MODY is also commonly responsive to sulfonylureas. However, the treatment plan should take into account disease progression, glucose control and individual clinical circumstances [7].

4) HNF1B-MODY

Management of HNF1B-MODY is more complex because diabetes may occur alongside renal and other abnormalities. Treatment therefore needs to address both glycemic control and associated organ problems [1].

5) Genetic counselling

Genetic counselling should be considered when a pathogenic variant is identified. It helps affected individuals understand the inheritance pattern, implications for family members and the importance of appropriate clinical follow-up.

9. NUTRITIONAL CONSIDERATIONS IN MODY

Nutrition remains an important component of diabetes care, but dietary management of MODY should not simply be copied from standard type 2 diabetes recommendations. MODY is genetically heterogeneous, and the metabolic characteristics vary according to the subtype.

For individuals with MODY who require dietary management, a balanced eating pattern should provide adequate carbohydrate, protein, fat, vitamins, minerals and dietary fibre according to age, activity level and individual nutritional requirements.

Important nutritional considerations include:**1. Balanced meal pattern:**

Meals should be planned to provide adequate nutrients while supporting appropriate blood glucose management.

2. Carbohydrate quality:

Preference can be given to nutrient-rich carbohydrate sources such as whole grains, pulses, vegetables and whole fruits. The amount and distribution of carbohydrate should be individualized according to the person's treatment and glycemic response.

3. Dietary fibre:

Adequate fibre intake from vegetables, fruits, whole grains, pulses and other plant foods can support overall metabolic health.

4. Limitation of excess added sugars:

Frequent intake of sugar-sweetened beverages and highly processed foods can make overall glycemic management more difficult and should generally be limited.

5. Healthy fat sources:

Unsaturated fat sources such as nuts, seeds and appropriate vegetable oils can be incorporated into a balanced diet while excessive saturated and trans fat intake is avoided.

6. Adequate protein:

Protein requirements should be met according to age, body size, activity and any associated renal or other medical condition.

7. Individualized dietary counselling:

Dietary advice should consider the MODY subtype, medication, pregnancy status, nutritional needs, lifestyle and associated complications.

It is important to recognize that MODY-specific nutritional research is much more limited than research on nutrition in type 1 and type 2 diabetes. Therefore, nutrition recommendations should be integrated with the broader evidence base for healthy diabetes eating patterns rather than presenting a separate universal “MODY diet.”

10. MODY AND INDIAN POPULATION

Evidence from India is particularly important because genetic patterns and clinical presentation may differ between populations. Earlier reviews have pointed out that the distribution of MODY-related genetic variants may vary between Asian and Western populations [3].

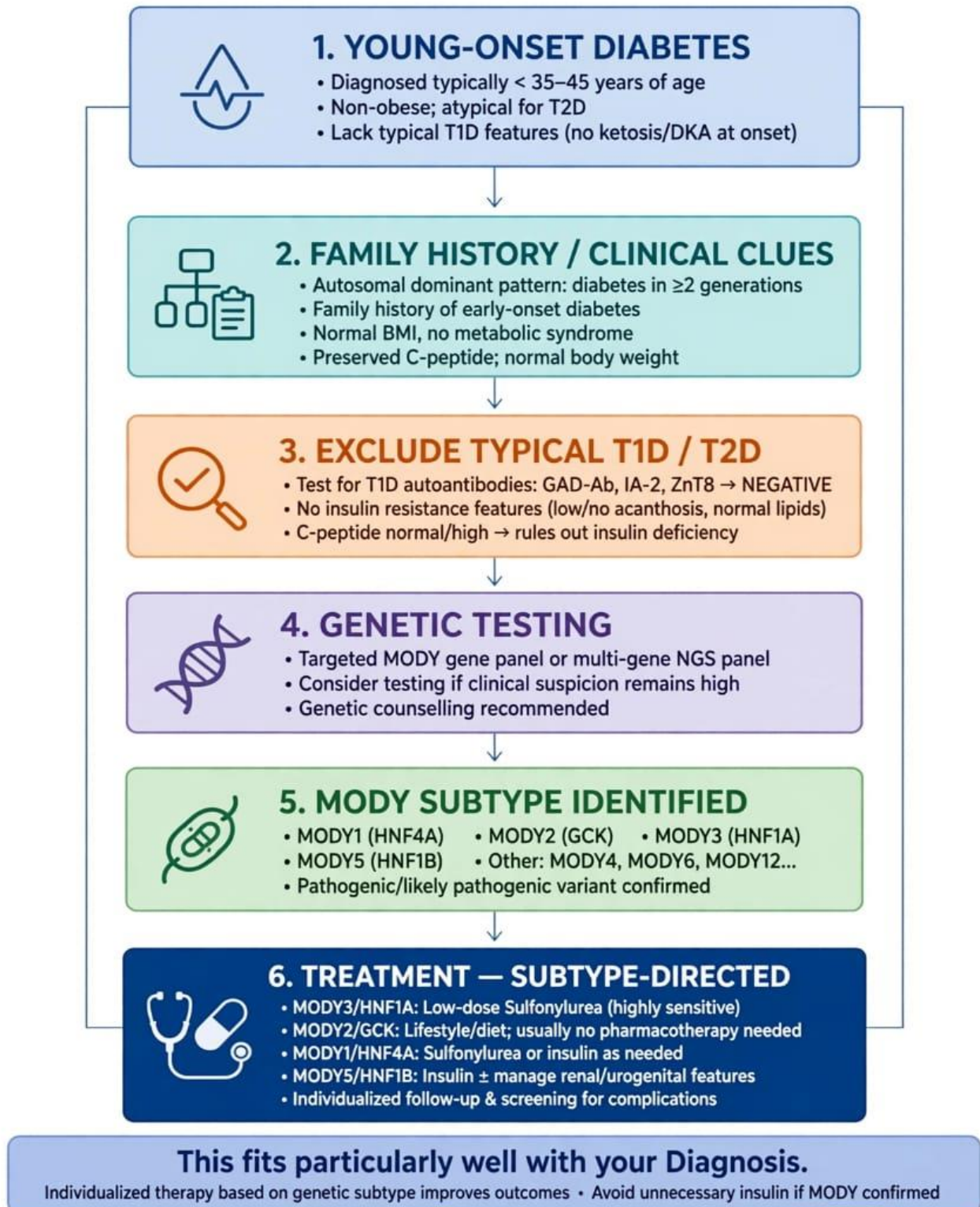
Aarthy et al. provided useful evidence from a tertiary diabetes centre in South India. In their study, 530 individuals considered clinically possible MODY underwent genetic testing and 58 were confirmed genetically, corresponding to a 10.9% pickup rate in the selected clinically suspected group [10].

HNF1A-MODY was the most frequently identified subtype, followed by HNF4A-MODY, ABCC8-MODY, GCK-MODY and HNF1B-MODY. The findings demonstrate that clinically selected patients in India can have several different monogenic causes and that genetic testing may help distinguish these forms from more common types of diabetes [10].

The study also reported diabetic complications among genetically confirmed patients, emphasizing that although some MODY forms can have a relatively mild course, MODY should not automatically be considered free from long-term complications. Appropriate diagnosis, monitoring and glycemic management remain important.

DIAGNOSTIC FLOWCHART: YOUNG-ONSET DIABETES → MODY

Evaluation & Genetic Testing Pathway for Maturity-Onset Diabetes of the Young



Clinical Note: MODY accounts for ~1–5% of diabetes cases. Consider referral to genetics / diabetes specialist.

• Key: Test before empiric treatment when young-onset is suspected • Designed / clinical education only — not a substitute for professional medical advice

9. ANALYSIS AND DISCUSSION

The literature reviewed in this paper demonstrates that MODY is not a single uniform disease. It represents a collection of genetically distinct disorders with differences in age of presentation, severity of hyperglycemia, associated clinical findings and treatment response.

One of the most important findings across the reviewed literature is the role of genetic testing. Clinical features can raise suspicion, but genetic analysis is needed to establish the specific molecular diagnosis. This is particularly relevant because the treatment of one subtype may differ substantially from another [1][7].

The reviewed studies also demonstrate why MODY is frequently missed. Young age at diagnosis may initially suggest type 1 diabetes, whereas the absence of insulin dependence or the presence of mild hyperglycemia may lead to a diagnosis of type 2 diabetes. In addition, access to genetic testing remains limited in many settings.

Another important observation is the relevance of family history. Diabetes appearing across several generations can provide a useful clue to an autosomal-dominant monogenic disorder. However, family history should be interpreted alongside biochemical and clinical information rather than being used as an isolated diagnostic test.

The Indian evidence adds another important dimension. The South Indian study by Aarthy et al. demonstrated that several MODY subtypes can be identified among clinically suspected cases and that HNF1A-MODY was common in the study population [10]. More studies from different Indian regions would be useful for understanding the actual burden and genetic distribution of MODY in the country.

Treatment findings also show the importance of subtype identification. GCK-MODY generally does not require routine glucose-lowering treatment in nonpregnant individuals, whereas HNF1A-MODY and HNF4A-MODY often respond to sulfonylureas [1][7]. Therefore, a molecular diagnosis may change both the medication plan and the need for long-term treatment.

From a nutritional perspective, there is a need for caution when applying general diabetes dietary advice to all MODY patients. Nutritional care should be individualized rather than based on the assumption that every person with MODY has the same metabolic abnormality. Further research specifically examining dietary patterns, nutritional status and long-term outcomes in different MODY subtypes would strengthen the evidence base.

11. SUMMARY

Maturity-Onset Diabetes of the Young is a group of monogenic diabetes disorders caused by single-gene abnormalities that affect pancreatic beta-cell function or insulin secretion. The condition usually presents at a young age and commonly follows an autosomal-dominant inheritance pattern, although the age of diagnosis may vary.

MODY can resemble type 1 or type 2 diabetes and is therefore frequently misclassified. Important clues include young-onset diabetes, diabetes in successive generations, preserved endogenous insulin secretion, absence of typical autoimmune features and subtype-specific clinical findings.

Genetic testing is central to confirmation of the diagnosis. The major clinically recognized forms include GCK-MODY, HNF1A-MODY, HNF4A-MODY and HNF1B-MODY, with several additional rare forms also known.

Treatment varies considerably according to the genetic subtype. GCK-MODY generally requires little or no glucose-lowering therapy outside specific circumstances, whereas HNF1A-MODY and HNF4A-MODY often respond well to sulfonylureas. HNF1B-MODY requires consideration of associated renal and other abnormalities.

Indian evidence suggests that genetically confirmed MODY can be identified among clinically selected diabetes patients, highlighting the importance of greater awareness and appropriate genetic testing. Nutritional management remains part of comprehensive diabetes care, but specific dietary recommendations should be individualized and should recognize the limited amount of MODY-specific nutritional research.

12. CONCLUSION

Maturity-Onset Diabetes of the Young represents an important but frequently overlooked group of monogenic diabetes disorders. Its significance lies not only in its genetic origin but also in the differences in clinical course and treatment response among individual subtypes. A patient diagnosed with diabetes at a young age should therefore not automatically be classified as having type 1 or type 2 diabetes when the clinical presentation is atypical.

The increasing availability of genetic testing has created an opportunity to improve diagnosis and provide more individualized diabetes care. Identification of the responsible gene can guide treatment, assist in monitoring for associated complications and help recognize affected family members. Evidence from India further suggests that MODY deserves greater attention within diabetes services.

Nutritional management should form part of comprehensive care, but it should be individualized according to the genetic subtype, medication, pregnancy status, metabolic condition and nutritional requirements of the patient. Further research in Indian and other underrepresented populations, particularly research combining genetic, clinical and nutritional information, may improve understanding and management of MODY in the future.

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