

Everyday Practice

Approach to a person recently diagnosed with diabetes

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The first physician to treat a patient of diabetes is a master of his or her destiny.

—Burns (1949)

INTRODUCTION

True to this aphorism, the physician with whom a newly diagnosed patient of diabetes comes into contact has a responsibility to (i) confirm the diagnosis of diabetes mellitus, (ii) categorize the type of diabetes, (iii) identify precipitating factors and complications, (iv) perform appropriate investigations, (v) chart a therapeutic plan and future course, and (vi) initiate diabetes education in a graded manner.

CONFIRMATION OF DIAGNOSIS

If unequivocal symptoms of hyperglycaemia such as polyuria, polydipsia and unexplained weight loss are present, a single casual (or random) plasma glucose level of ≥ 200 mg/dl is enough to confirm the diagnosis. Otherwise, a fasting plasma glucose level of ≥ 126 mg/dl on 2 occasions is needed to make the diagnosis (Table I). It is important to not base the diagnosis on a single value. Glycosuria alone should not be used to make the diagnosis and should be followed by plasma glucose estimation. The oral glucose tolerance test (OGTT) is not routinely used for diagnosis except in pregnancy or in subjects with a strong clinical suspicion of diabetes with equivocal fasting and random plasma glucose values. A raised glycosylated haemoglobin level will identify previously undiagnosed diabetes.

TABLE I. Criteria for diagnosis of diabetes*

- Symptoms of diabetes and a casual plasma glucose ≥ 200 mg/dl (11.1 mmol/L). Casual is defined as any time of the day without regard to time since the last meal. The classical symptoms of diabetes include polyuria, polydipsia and unexplained weight loss.
- OR
- Fasting plasma glucose level ≥ 126 mg/dl (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hours prior to the estimation.
- OR
- 2-hour plasma glucose level ≥ 200 mg/dl (11.1 mmol/L) during an oral glucose tolerance test. The test should be performed as described by WHO using a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water.
- *Pre-diabetes*
Impaired fasting glucose (IFG): Fasting plasma glucose level >100 mg/dl but <126 mg/dl
Impaired glucose tolerance (IGT): 2-hour plasma glucose level >140 mg/dl but <200 mg/dl during an oral glucose tolerance test

* American Diabetes Association 2007¹

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It is important to identify the setting in which hyperglycaemia is detected such as during stress—stress hyperglycaemia. Patients who are diagnosed with diabetes in the setting of infection, diabetogenic drug therapy (e.g. steroids) or hospitalization for an acute coronary event stroke or major surgery should be re-tested 3 months after the stress is over or the offending drug is withdrawn. Though a small proportion of such patients will have normal plasma glucose values during the non-stress period, a larger proportion continue to show impaired glucose tolerance or diabetes, especially those with coronary artery disease.

AETIOLOGICAL TYPE OF DIABETES

Identifying the aetiological type of diabetes (Table II) is important to plan appropriate therapy. However, this may not always be possible at the first presentation. Though type 2 diabetes is the most common, identifying type 1 diabetes is important as insulin treatment can then be started from the outset. Even though many of the different types of secondary diabetes and monogenic forms of inherited diabetes are uncommon, it is important to identify these for optimal treatment. Hence, a detailed history and physical examination is a must.

HISTORY AND PHYSICAL EXAMINATION

Age at diagnosis, presence or absence of family history, weight, rapidity of onset, severity of symptoms and presence of

TABLE II. Aetiological classification of diabetes mellitus

1. Type 1 (b cell destruction—absolute insulin deficiency)
2. Type 2 (varying degrees of insulin resistance and relative insulin deficiency)
3. Other specific types
 - a. Genetic defects of b-cell function: Maturity-onset diabetes of the young (MODY) 1-6, mitochondrial DNA mutation-related diabetes mellitus
 - b. Genetic defects in insulin action: Leprechaunism, lipotrophic diabetes, etc.
 - c. Diseases of the exocrine pancreas: Chronic pancreatitis, carcinoma, fibrocalculus pancreatopathy, haemochromatosis, cystic fibrosis
 - d. Endocrinopathies: Acromegaly, Cushing syndrome, pheochromocytoma, glucagonoma, somatostatinoma
 - e. Drug-induced: Glucocorticoids, b-blockers, phenytoin, thiazides, antiretroviral drugs, atypical antipsychotics
 - f. Uncommon immune-mediated: Stiffman syndrome
 - g. Other genetic syndromes: Down syndrome, Klinefelter syndrome, DIDMOAD (diabetes insipidus, diabetes mellitus, optic atrophy, deafness), Prader-Willi syndrome, myotonic dystrophy, etc.
4. Gestational

hyperglycaemia and documented ketonuria/ ketoacidosis in the absence of major stress have all been used to differentiate type 1 from type 2 diabetes (Table III). Of these, ketosis is the only unequivocal pointer. Age alone is not a criterion since type 1 diabetes can occur in the elderly and there is a rising incidence of type 2 diabetes in children and adolescents. Features of insulin deficiency such as severe hyperglycaemia (>300 mg/dl), osmotic symptoms and rapid weight loss irrespective of the baseline weight suggest type 1 diabetes. Yet, unlike children and adolescents, adults with type 1 diabetes can present with an indolent form of slowly progressive disease called latent autoimmune diabetes in adults (LADA), which can masquerade initially as type 2 diabetes and is suspected and diagnosed only on prolonged follow up.

A strong family history and presence of markers of the metabolic syndrome such as generalized or abdominal obesity, acanthosis nigricans, hypertension and pre-existing cardiovascular disease point to type 2 diabetes. Likewise, the presence of microvascular (retinopathy, nephropathy) or neuropathic complications at the time of diagnosis strongly point to type 2 diabetes because of its indolent, long and asymptomatic course.

A detailed drug history is important to exclude drug-induced diabetes. Traditionally, glucocorticoids, thiazides, and β -adrenergic antagonists have been recognized as the common offending drugs. Recent additions to this list are the newer atypical antipsychotics such as olanzepine and antiretroviral drugs. Specific monogenic forms of diabetes such as different subtypes of maturity-onset diabetes in the young (MODY) or mitochondrial DNA mutation-related diabetes are suspected based on young age at onset, strong family history and non-obese habitus in the former condition, and maternal inheritance and associated deafness in the latter. Clues to secondary diabetes from the history and physical examination are summarized in Table IV.

IDENTIFYING PRECIPITATING FACTORS AND COMPLICATING ILLNESSES

It is important to identify the precipitating factors that brought the patient to attention or unmasked the diabetes, because of the following reasons:

1. Treatment of the precipitating factor(s) may be equally or more important than hyperglycaemia *per se*, e.g. active infection, acute coronary event.
2. They indicate the need for insulin treatment irrespective of the type of diabetes.

TABLE III. History and physical examination

History	Physical examination
• Symptoms of hyperglycaemia	• Weight
• Duration	• Body mass index
• Precipitating factors	• Waist circumference, waist-hip ratio
• Symptoms of complications, if any	• Acanthosis nigricans
• Hypertension, pre-existing cardiovascular disease	• Blood pressure
• Drug history	• Peripheral pulses
• Diet	• Feet
• Physical activity	• Fundi
• Family history—	• Cardiovascular system
— Diabetes and complications	• Peripheral nervous system
— Age at onset	• Thyroid
— Cardiovascular disease	• Physical stigmata of secondary diabetes

3. They indicate the need for hospitalization.
4. Their management can influence diabetes treatment and vice versa.

For instance, glucocorticoids used for treating inflammatory diseases and gatifloxacin for infections have the potential to aggravate hyperglycaemia. There is clear consensus now in favour of intensive glycaemic control with insulin in hospitalized patients especially in the setting of acute coronary syndromes and critically ill postoperative and post-trauma patients.

Identification of macrovascular or microvascular complications at presentation in type 2 diabetes is important since it will influence the treatment plan in favour of early insulin therapy rather than the step-wise treatment with oral hypoglycaemic agents used in uncomplicated type 2 diabetes.

INVESTIGATIONS

A rational approach to investigations in a newly diagnosed patient should be targeted to

1. Assess the metabolic status
2. Ascertain the aetiology of diabetes
3. Detect complications at presentation, and
4. Identify associated co-morbid conditions. (Table V)

The physician should shun a glucocentric approach and view the larger perspective of cardiovascular risk in type 2 diabetes and hence investigations should be in tune with this holistic approach. Testing routinely for the presence of autoimmune markers such as antibodies to glutamic acid decarboxylase (GAD) or markers of β -cell reserve (C peptide estimation) is not cost-effective and should be guided by clinical judgement. Since C peptide levels can be low in uncontrolled diabetes due to glucotoxicity, its estimation should be deferred until good glycaemic control is achieved. When in doubt, it is prudent to treat the patient with insulin and use these laboratory markers

TABLE IV. Clues to specific subtypes of diabetes

History and clinical findings	Diabetes subtype
Strong family history, young age (<30 years), non-obese	Maturity-onset diabetes of the young (MODY)
Maternal inheritance, deafness	Mitochondrial diabetes
Severe acanthosis nigricans, lipoatrophy, genital hypertrophy	Insulin receptor defects
Deafness, optic atrophy, diabetes insipidus	DIDMOAD (diabetes insipidus, diabetes mellitus, optic atrophy, deafness)
Abdominal pain, steatorrhoea	Chronic pancreatitis
Striae, ecchymoses, proximal muscle weakness	Cushing syndrome
Coarse features, acral enlargement	Acromegaly

TABLE V. Investigations in a newly diagnosed person with diabetes

For metabolic status	For associated co-morbid conditions
Fasting plasma glucose	Thyroid function tests
Postprandial plasma glucose	Coeliac disease work up (tissue transglutaminase/anti-endomysial antibodies)
Glycosylated haemoglobin (HbA _{1c})	
Urine ketones	
Lipid profile	
For complications	For aetiology
Urea, creatinine, liver function tests	C peptide
Urine microalbumin	GAD antibodies
Electrocardiogram	Ultrasound abdomen for pancreas
Ankle-brachial index	Endocrinopathies: Dexamethasone suppression test for Cushing syndrome; growth hormone suppression and IGF-1 for acromegaly

during follow up to decide whether the patient can be switched over to oral hypoglycaemic drugs.

TREATMENT PLAN

Irrespective of the type of diabetes, advice on lifestyle modification in the form of optimum physical activity and nutrition should be imparted through a dietician and diabetes educator wherever possible. A detailed discussion on the choice of oral hypoglycaemic agents in type 2 diabetes is beyond the scope of this article and will be discussed elsewhere in this series. Broadly, the traditional management of type 2 diabetes entails lifestyle modification along with monotherapy with sulphonylureas or metformin, depending on whether the patient is non-obese or obese, followed by a subsequent switch-over to combination therapy if the desired targets are not met. In this schema insulin therapy was reserved for the stage when double- or triple-drug combinations failed to achieve the targeted glycaemic control. With the current understanding of the pathophysiology of type 2 diabetes and availability of newer drugs such as the glitazones, alternative approaches such as the use of combinations right from diagnosis, early use of glitazones and early use of insulin are emerging. The latter approach is theoretically sound as a b-cell preservation strategy in type 2 diabetes, though there is no consensus on its routine use in all patients with type 2 diabetes.

WHEN SHOULD INSULIN BE USED AS PRIMARY THERAPY?

In clinically clear-cut type 1 diabetes as evidenced by ketonuria or frank ketoacidosis at presentation, insulin treatment is absolutely indicated and not doing so will amount to poor care. Even in the absence of ketonuria, the presence of clinical pointers to insulin deficiency and type 1 diabetes will dictate the need for starting insulin; these include a rapid onset, severe hyperglycaemia (>300 mg/dl), severe symptoms and rapid weight loss irrespective of the baseline weight, especially in patients without a family history and presence of other autoimmune diseases. When in doubt, it is prudent to treat the patient with insulin and use laboratory markers such as C peptide and GAD antibodies during follow up to decide whether the patient can be switched over to oral hypoglycaemic drugs.

WHEN IS HOSPITALIZATION REQUIRED?

Other than conditions of acute metabolic decompensation such as diabetic ketoacidosis and hyperosmolar coma, precipitating factors and complicating illnesses usually require hospitalization on their own merit. Otherwise, a patient with uncomplicated diabetes usually does not require hospitalization even if insulin therapy has to be initiated. This can be done even in children in the familiar and less intimidating home environment, if frequent follow up and constant telephonic contact with the healthcare team can be assured.

CHARTING A FUTURE PLAN AND FOLLOW UP

The frequency of initial follow up visits after diagnosis depends on whether the patient is on insulin or oral drugs. Patients on insulin should contact the physician once-weekly during the first few weeks. Subsequently, monthly follow up is advised until stabilization of control and optimization of diabetes self-management education are achieved. Once these goals are achieved, 3-monthly follow up is needed. Patients with type 2 diabetes on lifestyle modification alone or on oral drugs need

less frequent follow up—monthly for the first few months and then every 3–4 months. Glycaemic control is monitored using a combination of clinical and biochemical criteria (Table VI). Self-monitoring of blood glucose is especially useful in patients on insulin to achieve treatment goals by tailoring the dose of insulin and diet to meet both fasting and post-prandial targets. Glycosylated haemoglobin is a good yardstick of glycaemic control. Initially, it can be performed with a standardized assay once in 3 months and, once the targets are met, once in 6 months. A comprehensive approach to monitoring other targets for control such as blood pressure and lipids is important to reduce the risk of macrovascular and microvascular complications (Table VII). The goals have to be individualized according to age, co-morbid conditions, socioeconomic milieu and diabetes self-management skills of the patient. Periodic surveillance is required to monitor the onset of these complications (Table VIII).

TABLE VI. Monitoring glycaemic control

Clinical	Biochemical
Symptoms	Urine analysis
Weight	Fasting and postprandial glucose from laboratory
Growth and development in children	Self monitoring of blood glucose Glycosylated haemoglobin

TABLE VII. Goals for comprehensive control*

<i>Glycaemic control</i>	
HbA _{1c} : <7.0%	
Preprandial capillary plasma glucose: 90–130 mg/dl	
Peak postprandial capillary plasma glucose: <180 mg/dl	
<i>Blood pressure</i>	<130/80 mmHg
<i>Lipids</i>	
Low density lipoprotein: <100 mg/dl	
Triglycerides: <150 mg/dl	
High density lipoprotein: >40 mg/dl in men; >50 mg/dl in women	

*American Diabetes Association Guidelines 2007¹

TABLE VIII. Schema for surveillance of complications and co-morbid conditions

Parameter/Test	Testing frequency
Blood pressure	Every visit
Feet examination	Every visit
Electrocardiogram	Annual
Fasting lipids	Annual; more often if outside goals
Examination of ocular fundi	Annual
	Type 1: 5 years after onset
	Type 2: From onset
Microalbuminuria	More frequent if abnormal
	Annual
	Type 1: 5 years after onset Type 2: From onset
Creatinine clearance	Annual
Peripheral nerve assessment (monofilament sensory testing)	Annual

INITIATING DIABETES EDUCATION

The newly diagnosed person with diabetes and her/his family goes through an emotional turmoil with a combination of depression, denial, resentment and apprehension about the future. This is more pronounced if the patient, especially a child or adolescent, has to be started on insulin. The onus is on the physician to help them tide over this crisis and initiate diabetes self-management education. This process has to be gradual to avoid bombarding the patient with too much information too soon. Counselling has to be reassuring but realistic, informative and not intimidating. A busy clinician should take help from dieticians and trained diabetes educators. Education imparted using this team approach is more rewarding. A graded approach to diabetes education is outlined in Table IX.

SUMMARY

The physician who sees a newly diagnosed patient with diabetes can adequately fulfil his professional responsibilities by following a systematic approach to clinical diagnosis, investigations, appropriate treatment planning and involvement of the patient in his or her treatment through self-management education. Correct steps taken in the crucial initial period set the pace for optimum future management of this chronic disease.

TABLE IX. Graded diabetes education

Initial visits	Follow up visits
<i>General</i>	
What is diabetes?	Importance of glycaemic control
Why does it occur?	Prevention of complications
Lifestyle measures: Diet, exercise	Carbohydrate counting— self-adjustment of insulin doses
<i>Type 1 diabetes</i>	
Insulin use, injection technique	Foot care
Self-monitoring of blood glucose	Marriage counselling
Identify and treat hypoglycaemia	Preconceptional counselling
<i>Type 2 diabetes</i>	
Detailed lifestyle advice	Newer modalities of treatment
Use of oral drugs	
To look beyond glucose:	
Blood pressure, lipids	

REFERENCES

- 1 American Diabetes Association. Standards of medical care in diabetes—2007. *Diabetes Care* 2007;**30** (Suppl 1):S4–S41.
- 2 American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2007;**30** (Suppl 1):S42–S47.
- 3 Tattersall RB, Gale EAM (ed). *Diabetes: Clinical management*. Edinburgh: Churchill