Diabetes Mellitus: Diagnosis and Management Guidelines

SV Madhu[1], Saurabh Srivastava[2]

[1]Department of Medicine, Division of Endocrinology and Metabolism University College of Medical Sciences, Guru Teg Bahadur Hospital, Delhi; [2]Department of Medicine, School of Medical Sciences and Research, Sharda University, Greater Noida, Uttar Pradesh, India

Abstract*: Diabetes mellitus is a metabolic disorder of carbohydrate metabolism, with under utilization of glucose, leading to hyperglycaemia. Type 2 diabetes is in epidemic proportions and achieving specific glycemic goals can substantially reduce morbidity. Effective treatment of hyperglycemia is the center stage in the treatment of diabetes. Therapies directed at other coincident features, such as dyslipidemia, hypertension, hypercoagulability, obesity, and insulin resistance, have also been a major focus of research and therapy. The present article focuses on the diagnosis and management strategies of this complex metabolic disorder.*

INTRODUCTION

Diabetes mellitus is a group of metabolic disorders of carbohydrate metabolism in which glucose is underutilized and overproduced, causing hyperglycemia. Diabetes mellitus is a complex, chronic illness which requires continuous medical care. There are multifactorial risk reduction strategies which act beyond glycaemic control. Prevention of acute complications and reduction of the risk of long-term complications can be done by patient self-management education and support. It is a well recognized fact that the type 2 diabetes is in epidemic proportions and achieving specific glycemic goals can substantially reduce morbidity. Thus effective treatment of hyperglycemia has attained a top priority [1–3]. Center stage in the treatment of diabetes, is management of hyperglycemia, the hallmark metabolic abnormality associated with type 2 diabetes, therapies directed at other coincident features, such as dyslipidemia, hypertension, hypercoagulability, obesity, and insulin resistance, have also been a major focus of research and therapy. Maintaining glycemic levels as close to the nondiabetic range as possible has been demonstrated to have a powerful beneficial effect on diabetes-specific microvascular complications, including retinopathy, nephropathy, and neuropathy, in the setting of type 1 diabetes[4,5].

DIAGNOSIS OF DIABETES

Diabetes is diagnosed based on plasma glucose criteria, either the fasting plasma glucose (FPG) or the 2-hour plasma glucose (2-h PG) value after a 75-g oral glucose tolerance test (OGTT) [4,6]. Recently, an International Expert Committee, which comprised members appointed by the American Diabetic Association (ADA), the European Association for the Study of Diabetes, and the IDF, added the A1C (threshold > 6.5%) as a third option to diagnose diabetes[7].

The World Health Organization (WHO), American Diabetic Association (ADA) and the International Diabetes Federation (IDF) have recommended an FPGvalue ≥ 7.0mmol/L (126 mg/dL); a 2-h postload glucose concentration ≥ 11.1 mmol/L (200 mg/dL) during an OGTT; or symptoms of diabetes and a casual (i.e., regardless of the time of the preceding meal) plasma glucose concentration ≥ 11.1 mmol/L (200 mg/dL). If any one of these criteria is met, confirmation by repeat testing on a subsequent day is necessary to establish the diagnosis [note that repeat testing is not required for patients who have unequivocal hyperglycemia, i.e., ≥ 11.1 mmol/L (200 mg/dL) with symptoms consistent with hyperglycemia][8]. (Table 1)

PREDIABETES

In 1997 and 2003, a group of individuals whose glucose levels did not meet the criteria for diabetes, but were too high to be considered normal, were recognized by the Expert Committee on Diagnosis and Classification of Diabetes Mellitus[9,10]. These persons were diagnosed to be having prediabetes. Prediabetes was defined as impaired fasting glucose (IFG) (FPG levels 100–125mg/dL [5.6–6.9 mmol/L]), or impaired glucose tolerance (IGT) (2-h PG OGTT values of 140–199 mg/dL [7.8–11.0 mmol/L]). (Table 2)

Correspondence: ***Prof. Saurabh Srivastava*** *,Department of Medicine, School of Medical Sciences and Research, Sharda University, Greater Noida (U.P), India* ***e-mail****: saurabhsrivas@gmail.com*

Table 1*: Criteria for the diagnosis of diabetes*

Criteria for the diagnosis of diabetes
HbA1c ≥ 6.5% (48 mmol/mol)
or
FPG ≥7.0 mmol/L (126 mg/dL)
or
2-h Plasma glucose ≥11.1 mmol/L (200 mg/dL) during an OGTT
or
Symptoms of hyperglycemia and casual plasma glucose ≥11.1 mmol/L (200 mg/dL)
• In the absence of unequivocal hyperglycemia, these criteria should be confirmed by repeat testing • HbA1c should be performed in a laboratory that is NGSP certified and standardized to the DCCT assay • Fasting is defined as no caloric intake for at least 8 h • The OGTT should be performed as described by the WHO, with a glucose load containing the equivalent of 75 g of anhydrous glucose dissolved in water • "Casual" is defined as any time of day without regard to time since previous meal • The classic symptoms of hyperglycemia include polyuria, polydipsia, and unexplained weight loss.

Table 2: *Prediabetes*

Prediabetes
FPG 100 mg/dL (5.6mmol/L) to 125 mg/dL (6.9 mmol/L) (IFG)
Or
2-h PG in the 75-g OGTT 140 mg/dL (7.8 mmol/L) to 199 mg/dL (11.0 mmol/L) (IGT)
Or
A1C 5.7–6.4%
*For all three tests, risk is continuous, extending below the lower limit of the range and becoming disproportionately greater at higher ends of the range.

Criteria for testing for diabetes in asymptomatic adult individuals
1. Testing should be considered in all adults who are overweight (BMI>25 kg/m2) and have additional risk factors: • physical inactivity • first-degree relative with diabetes • high-risk race/ethnicity (e.g., African American, Latino, Native American, Asian American, Pacific Islander) • women who delivered a baby weighing > 9 lb or were diagnosed with GDM • hypertension (≥ 140/90 mmHg or on therapy for hypertension) • HDL cholesterol level < 35 mg/dL (0.90 mmol/L) and/or a triglyceride level >250 mg/dL (2.82 mmol/L) • women with polycystic ovarian syndrome • A1C ≥ 5.7%, IGT, or IFG on previous testing • Other clinical conditions associated with insulin resistance (e.g., severe obesity, acanthosis nigricans) • History of CVD
2. In the absence of the above criteria, testing for diabetes should begin at age 45 years
3. If results are normal, testing should be repeated at least at 3-year intervals, with consideration of more frequent testing depending on initial results (e.g., those with prediabetes should be tested yearly) and risk status.

SCREENING OF DIABETES

Screening of diabetes is done in an asymptomatic individual if there is an increased risk of development of Diabetes or after the age of 45 years. The individuals at an increased risk of diabetes are shown in table 3[8].

MANAGEMENT OF DIABETES MELLITUS

The goals of therapy for type 1 or type 2 DM are:

(1) Eliminate symptoms related to hyperglycemia,
(2) Reduce or eliminate the long-term microvascular and macrovascular complications of DM

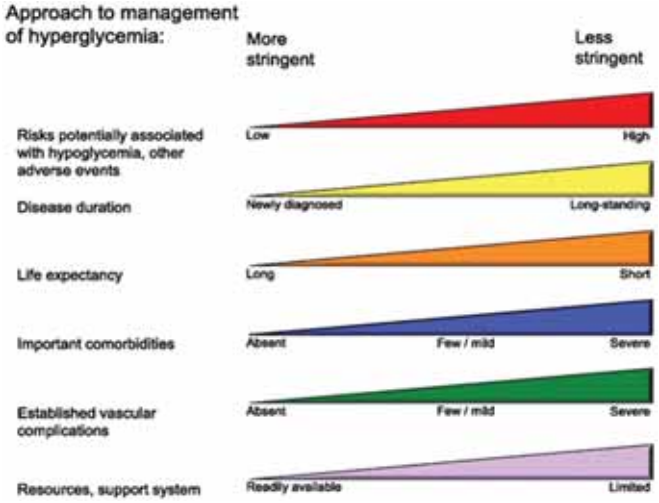

Figure 1: *Approach to Diabetic patient – Adopted from American Diabetic Association*

(3) Allow the patient to achieve as normal a lifestyle as possible

GLYCAEMIC GOALS FOR DM

The glycaemic goals for patients with Diabetes are[11]

1. A1C < 7.0%
2. Preprandial capillary plasma glucose 70–130 mg/dL (3.9–7.2 mmol/L)
3. Peak postprandial capillary plasma glucose < 180 mg/dL (10.0 mmol/L)

Goals should be individualized based on: duration of diabetes, age/life expectancy, co-morbid conditions, known cardiovascular disease (CVD) or advanced microvascular complications, hypoglycemia unawareness, individual patient considerations, more or less stringent glycaemic goals may be appropriate for individual patients[12]. (Figure 1)

More stringent HbA1c targets (e.g., 6.0– 6.5%) might be considered in selected patients (with short disease duration, long life expectancy, no significant CVD) if this can be achieved without significant hypoglycemia or other adverse effects of treatment[12,13]. Conversely, less stringent HbA1c goals e.g., 7.5–8.0% or even slightly higher are appropriate for patients with a history of severe hypoglycemia, limited life expectancy, advanced complications, extensive co-morbid conditions[14].

BLOOD PRESSURE AND LIPID TARGETS IN PATIENTS WITH DM

Blood pressure target in these patient is <130/80 mm Hg. The low-density lipoprotein cholesterol should be <2.6 mmol/L (100 mg/dL), High-density lipoprotein cholesterol should be >1 mmol/L (40 mg/dL) in men >1.3 mmol/L (50 mg/dL) in women and triglycerides should be <1.7 mmol/L (150 mg/dL).

APPROACH TO DIABETIC PATIENT

Now the management of diabetes is *'comprehensive diabetes care'* to emphasize the fact that optimal diabetes therapy involves more than plasma glucose management. Though glycaemic control is central to optimal diabetes therapy, comprehensive diabetes care of both type 1 and type 2 DM should also detect and manage DM-specific complications and modify risk factors for DM-associated diseases. The management is a multidisciplinary approach with a team comprising of a diabetologist, a certified diabetes educator, a nutritionist and as and when required subspecialists (including neurologists, nephrologists, vascular surgeons, cardiologists, ophthalmologists, and podiatrists) with experience in DM-related complications. Central to the success of this team are the patient's participation, input, and enthusiasm i.e. the therapy is now individualized and patient centered.

MEDICAL NUTRITION THERAPY (MNT)

Nutrition therapy is recommended for all people with type 1 and type 2 diabetes as an effective component of the overall treatment plan by ADA. All individuals who have diabetes should receive individualized MNT as needed to achieve treatment goals. For overweight or obese adults with type 2 diabetes, reducing energy intake while maintaining a healthful eating pattern is recommended to promote weight loss which translates in clinical benefit in form of improved glycaemia, blood pressure, and/or lipids.

The amount of carbohydrates and available insulin are the most important. Carbohydrate intake should be monitored either by carbohydrate counting or experience-based estimation. Substituting low–glycaemic load foods for higher–glycaemic load foods may modestly improve glycaemic control.

The patients with no evidence of diabetic kidney disease, the goals should be individualized as there is no recommendation for ideal protein intake, however in patients with kidney disease reducing the amount of dietary protein below usual intake is not recommended because it does not alter glycaemic measures, cardiovascular risk measures, or the course of Glomerular Filteration Rate (GFR) decline.

The quality of fat is more important than quantity so trans fat should be reduced in diet. The goal of MNT in the individual with type 1 DM is to coordinate and match the caloric intake. MNT for type 2 DM should emphasize modest caloric reduction (low-carbohydrate or low-fat), reduced fat intake, and increased physical activity[15].

EXERCISE

For individuals with type 1 or type 2 DM, exercise is also useful for lowering plasma glucose (during and following exercise) and increasing insulin sensitivity. In patients with diabetes, moderate aerobic physical activity of 150 min/week (distributed over at least 3 days) is recommended by ADA. The exercise regimen should also include resistance training[16].

PHARMACOLOGIC THERAPY

Pharmacological therapy is aimed at maintaining the glycaemia and reducing the long term complications of Diabetes. Drug classes used for the treatment of type 2 diabetes include the following: (Table 4)

(1) **Insulin sensitizers:** (a) Biguanides; (b)Thiazolidinediones (TZDs); (2) **Insulin secretagogues:** (a) Sulfonylureas; (b) Meglitinide derivatives; (3) **Alpha-glucosidase inhibitors;** (4) **Glucagonlike peptide–1 (GLP-1) agonists;** (5) **Dipeptidyl peptidase IV (DPP-4) inhibitors;** (6) **Selective sodium-glucose transporter-2 (SGLT-2) inhibitors** (7) **Insulin;** (8) **Amylinomimetics**

INSULIN SENSITIZERS

1. Biguanides

Biguanides decreases hepatic gluconeogenesis. It also decreases intestinal absorption of glucose and improves insulin sensitivity by increasing peripheral glucose uptake and utilization. It is contraindicated in patients with Congestive Heart Failure (CHF), renal or hepatic dysfunction, or binge alcoholism. They should be held shortly before surgical procedures and before radiologic studies involving intravenous contrast. The initial starting dose of metformin is 500 mg once or twice a day can be increased to 1000 mg bid[17–19].

2. Thiazolidinediones (TZDs)

Thiazolidinediones reduce insulin resistance by binding to the Peroxisome proliferator activatedreceptor (PPAR) gamma. The therapeutic range for pioglitazone is 15–45 mg/d. These agents are contraindicated in patients with liver disease or CHF (class III or IV). These agents are associated

Table 4: Various drug classes used in treatment of Diabetes

Class	Mechanism	Action	Disadvantages
Biguanides	Activates AMP-kinase	Decreased hepatic glucose production	* GI side effects * Lactic acidosis * Vitamin B12 deficiency
Sulphonylureas	Closes K ATP channels on beta cells	Increased insulin secretion	* hypoglycaemia * weight gain *? Blunts myocardial preconditioning
Meglitinides	Closes K ATP channels on beta cells other than sulphonylures	Increased insulin secretion But short acting so helpful in postprandial hyperglycaemia	* hypoglycaemia * weight gain *? Blunts myocardial preconditioning
Thiazolidinediones	Activates the nuclear transcription factor PPAR-gamma	↑ Insulin sensitivity	* Weight gain *Edema/heart failure *Bone fractures *↑ LDL-C *? ↑ MI *? ↑ Bladder cancer
Alpha -Glucosidase inhibitors	Inhibits intestinal alpha-glucosidase	Slows intestinal carbohydrate digestion/absorption	Gastrointestinal side effects (flatulence, diarrhea)
DPP-4 inhibitors	Inhibits DPP-4 activity, increasing postprandial active incretin (GLP-1, GIP) concentrations	* ↑ Insulin secretion * ↓ Glucagon secretion (both glucose-dependent) * No hypoglycaemia	* Urticaria/angioedema *? Pancreatitis
Bile acid sequestrants	Binds bile acids in intestinal tract, increasing hepatic bile acid production; ? activation of farnesoid X receptor (FXR)	* Unknown *? ↓ Hepatic glucose production * ? ↑ Incretin levels	* Constipation * ↑ Triglycerides * May ↓ absorption of other medications
Dopamine-2 agonists	Activates dopaminergic receptors	* Modulates hypothalamic regulation of metabolism * ↑ Insulin sensitivity	* Dizziness/syncope * Nausea * Fatigue * Rhinitis
GLP-1 receptor agonists	Activates GLP-1 receptors	* ↑ Insulin secretion (glucose-dependent) * ↓ Glucagon secretion (glucose-dependent) * Slows gastric emptying * ↑ Satiety	* Gastrointestinal side effects (nausea/vomiting) * ? Acute pancreatitis * C-cell hyperplasia/medullary thyroid tumors in animals * Injectable * Training requirements
Amylin mimetics	Activates amylin receptors	* ↓ Glucagon secretion * Slows gastric emptying * ↑ Satiety	* Gastrointestinal side effects (nausea/vomiting) * Hypoglycemia unless insulin dose is simultaneously reduced * Injectable * Frequent dosing schedule
Insulins	Activates insulin receptors	* ↑ Glucose disposal * ↓ Hepatic glucose production	* Hypoglycemia * Weight gain * ? Mitogenic effects * Injectable * Training requirements * "Stigma" (for patients)
SGLT 2inhibitors	block the reabsorption of glucose in the kidney	increase glucose excretion	Vaginal yeast infections urinary tract infections contraindicated in - ESRD - Ketone in urine

with increased risk of fractures, and rarely may experience a worsening of diabetic macular edema. The safety of thiazolidinediones in pregnancy is not established[17–19].

Insulin secretagogues: Insulin secretagogues stimulate insulin secretion by interacting with the ATP-sensitive potassium channel on the beta cell[17–20]

1. Sulfonylureas

They reduce both fasting and postprandial glucose and should be initiated at low doses and increased at 1- to 2-week intervals based on self monitoring of blood glucose (SMBG) commonly used drugs are glimeperide (dose range: 1-8 mg), gliclazide (dose range: 40-240 mg) and glipizide (dose range: 5-20 mg).

2. Meglitinide derivatives

Repaglinide (dose range: 0.5-16 mg) and nateglinide (dose range: 180-360 mg) are not sulfonylureas but also interact with the ATP-sensitive potassium channel. Because of their short half-life, these agents are given with each meal or immediately before to reduce meal-related glucose excursions.

Alpha-glucosidase inhibitors

Alpha-glucosidase inhibitors acarbose (dose range: 25-100 mg with each meal), miglitol (dose range: 25-50 mg with each meal) and voglibose (dose range: 0.1- 0.3 mg with each meal), reduce postprandial hyperglycemia by delaying glucose absorption. These agents should not be used in individuals with inflammatory bowel disease, gastroparesis, or a serum creatinine >177 mol/L (2 mg/dL) [17–20].

Glucagon like peptide–1 (GLP-1) agonists

GLP-1 is produced in the L cells of small intestine and stimulates insulin secretion and inhibits glucagon secretion and hepatic glucose production in a glucose-dependent manner. Dosing is twice daily by subcutaneous injection. Starting dose of exanatide is 5 µg. If this dose is tolerated, titrate after 1 month to 10 µg. Due to its delaying effects on gastric emptying, the major side effect is GI complaints such as nausea, vomiting, and diarrhea[17–20].

Dipeptidyl peptidase IV (DPP-4) inhibitors

Dipeptidyl peptidase 4 (DPP 4) is a cell membrane protein that rapidly degrades GLP-1 and glucose-dependent insulinotropic polypeptide. Suppression of DPP 4 leads to higher levels of insulin secretion and suppression of glucagon secretion in a glucose-dependent manner.

The gliptins in common use are- sitagliptin, vildagliptin, saxagliptin and linagliptin.

Dosing of sitagliptin is 100 mg orally once daily, saxagliptin is 2.5 or 5 mg orally once daily, linagliptin is 5 mg orally once daily and vildagliptin is 50 mg twice daily. Linagliptin does not require dose adjustments with renal failure[17–19].

Insulin

Insulin is the oldest therapy available for diabetes. It was discovered in 1921, and clinical testing in humans started in 1922.To this date it remains the most effective method of reducing hyperglycemia. There is no upper limit in dosing for therapeutic effect. Hypoglycaemia is the major side effect. Various forms of insulin are shown in Table 5[16–19].

Table 5: *Insulin Preparations*

Insulin Preparations			
	Time of Action		
Preparation	Onset, h	Peak, h	Effective Duration, h
Short-acting			
Aspart	<0.25	0.5–1.5	3–4
Glulisine	<0.25	0.5–1.5	3–4
Lispro	<0.25	0.5–1.5	3–4
Regular	0.5–1.0	2–3	4–6
Long-acting			
Detemir	1–4	—	Up to 24
Glargine	1–4	—	Up to 24
NPH	1–4	6–10	10–16
Insulin combinations			
75/25–75% protamine lispro, 25% lispro	<0.25	1.5 h	Up to 10–16
70/30–70% protamine aspart, 30% aspart	<0.25	1.5 h	Up to 10–16
50/50–50% protamine lispro, 50% lispro	<0.25	1.5 h	Up to 10–16
70/30–70% NPH, 30% regular	0.5–1	Dual[b]	10–16

APPROACH TO GLYCAEMIA MANAGEMENT

The algorithm for glycaemia management recommend an HbA1c of 6.5% or lower for healthy patients without concurrent illness and at low risk for hypoglycemia but individualized target HbA1c values greater than 6.5% for patients with concurrent illness and those who are at risk for hypoglycemia. Lifestyle modification, including weight loss, is a component of all treatments. Metformin is the preferred initial agent for monotherapy and is a standard part of combination treatments.

Dual drug therapy

If the glycaemic goal is not achieved or sustained within 2-3 months, another medication should be added. The choice of addition of second drug is also individualized as per the patient characteristic and involvement (eg, a DPP-4 inhibitor if both postprandial and fasting glucose levels are elevated; a GLP-1 agonist if postprandial glucose levels are strongly elevated; a TZD if the patient has metabolic syndrome and/or nonalcoholic fatty liver disease). Before adding a second agent for a patient who is taking an insulin secretagogue, the clinician should warn the patient about the possibility of hypoglycemia.

Triple drug therapy

If 2 drugs prove unsuccessful after 2-3 months, the next step is triple therapy. The third drug may be an oral agent from a third class of antidiabetic drugs or basal insulin (typically at bedtime).

Insulin therapy

In case of type 1 DM the only available therapy is insulin however in type 2 DM the patients who are not able to achieve glycaemic targets by oral agents, the insulin therapy should be instituted. Sulphonyureas should preferably be omitted from the treatment and patient should be subjected to insulin therapy. All insulin injections should preferably be administered in the abdomen, although they can also be given in the thigh, hip, or buttock regions.

Multiple daily dosing

Multiple daily dosing of insulin gives the patient the greatest flexibility. Long-acting insulin (eg, glargine, detemir or NPH) is generally given once daily as the basal insulin and rapid-acting insulin (eg, aspart, glulisine, lispro or regular) is administered just before each meal.

Twice daily premixed insulin

Twice daily injections of premix insulin are given before breakfast and before dinner. This regimen is suitable for those patients who are reluctant for multiple injections.

Continuous subcutaneous insulin infusion (CSII) or insulin pump therapy

The ideal CSII candidate is a patient with type 1 diabetes mellitus or intensively managed type 2 diabetes mellitus who is currently performing 4 or more insulin injections and 4 or more self-monitored blood glucose measurements daily; is motivated to achieve optima blood glucose control; is willing and able to carry out the tasks that are required to use this complex and time-consuming therapy safely and effectively; and is willing to maintain frequent contact with their health care team.

EMERGING THERAPIES

Whole pancreas transplantation (performed concomitantly with a renal transplant) may normalize glucose tolerance and is an important therapeutic option in type 1 DM with ESRD, Pancreatic islet transplantation had a limitation of pancreatic islet supply and graft survival and remains an area of clinical investigation. Other newer therapies under investigation are outlined in table 6

Bariatric surgery for markedly obese individuals with type 2 diabetes has shown considerable promise. The ADA clinical guidelines state that bariatric surgery should be considered in individuals with DM and a BMI >35 kg/m^2.

Table 6: *Novel Compounds Currently in Clinical Development for the Treatment of Hyperglycemia in Patients with T2DM*

Drug Category	Mechanism of Action	Potential Advantages
Ranolazine	Unknown; modulation of sodium channels in pancreatic β cells may enhance glucose-stimulated insulin secretion	Improves diastolic function and cardiac microvascular flow
Cannabinoid-1 receptor antagonists	*Blockade of the cannabinoid-1 receptor in the central nervous system *significantly affects appetite regulation	Favorable effects on weight, insulin resistance and dyslipidemia; low potential for hypoglycemia
Dual[2] or pan-PPAR agonists	Variable activation of the nuclear transcription factor PPAR-γ in addition to PPAR-α and/or PPAR-δ	Improves triglycerides and HDL cholesterol (PPAR-α) increased fat catabolism and weight reduction (PPAR-δ)
Selective PPAR-γ modulators	Partial and selective activation of the nuclear transcription factor PPAR-γ	less adipogenesis and weight gain and less plasma volume expansion, with possibly reduced HF risk, vs TZDs
Fructose 1, 6 bisphosphatase inhibitors	Inhibition of the hepatic enzyme of the gluconeogenic pathway	Low potential for hypoglycemia
Glucokinase activators	Stimulation of a key enzyme in liver to increase hepatic glucose metabolism and in pancreatic β cells to increase insulin secretion	Complementary mechanisms of action
11ß-Hydroxysteroid dehydrogenase 1 inhibitors	Inhibition of the enzyme that regenerates cortisol from inactive cortisone in liver and adipose tissue, thereby improving insulin sensitivity	Reduces insulin resistance; possible lipid lowering and weight reduction;
Protein tyrosine phosphatase 1B inhibitors	Inhibition of a protein in muscle and liver that down-regulates insulin signaling, thereby improving insulin sensitivity	Reduces insulin resistance
Acetyl-CoA carboxylase-1 and -2 inhibitors	Reduction in malonyl-CoA production, with subsequent increase in fatty acid oxidation in liver and adipose tissue	Reduces insulin resistance, body fat content, and body weight
Glucagon receptor antagonists	Blockade of the effect of glucagon in liver to stimulate hepatic glucose production	Low potential for hypoglycemia

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TELEMEDICINE

Telemedicine is an innovative, currently used to strengthen continued healthcare in the rural communities.
Lack of computer savvy personnel, non-availability of advance equipment and uniform guidelines are some grey areas.
Telemedicine is the use of electronic information and communication technologies to provide and support healthcare in remote areas.

For more than 3 decades clinicians, health service researchers and others have been investigating that use of advanced telecommunication and information technologies (IT) for improving healthcare.

Telemedicine has variety of application in patient care, health education research, administration and public health. Commonest of all is use of emergency number call by ordinary telephones. Other applications like telesurgery, home monitoring of patients are yet to get attention and routine application in day-to-day practice

Early application of Telemedicine was often focused on remote populations scattered across mountainous areas, islands, open planes and arctic regions where doctors were not easily reached, but recently as the cost of communication and IT has dropped there is an wave of interest propelled upon in the field of telemedicine.

In the era of superspecialisation, telemedicine offers a mechanism for centralising specialist and supporting primary care physician at remote places.

Proper use of telemedicine will emphasis doctor to doctor communication and fill up the gap of non-availability of superspecialist in remote unaccessible areas. It will improve advanced emergency help to the patients and will provide more room for evidence-based medical practice. However, some medicolegal and ethical issues will prop up and will require a plausible solutions.