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Indian Diabetes

EDUCATOR JOURNAL

Theme of the Month

Diabetes and Associated Health Conditions

To keep Members Diabetes Care team abreast about
DSME /DSMS - (Diabetes Self management Education / Support) Concepts

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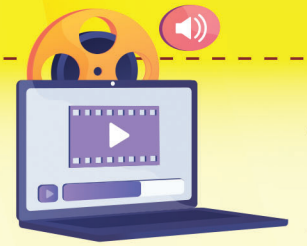

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FOREWORD

Research Society for the Study of Diabetes in India (RSSDI) founded by Prof MMS Ahuja in the year 1972 is the biggest scientific association of healthcare professionals involved in promoting diabetes education and research in India. RSSDI is happy to collaborate with USV to support their endeavour to make India the 'Diabetes care capital of the world'. Through this collaboration, RSSDI would like to strengthen the cadre of diabetes educators by empowering them with recent updates in diabetes management helping bridge the gap between the physician and the patient. Today, the rule of 50% is prevailing in terms of awareness, detection, treatment and control in T2DM.Our aspiration is to achieve 90-90-90-90 i.e.90% of people with diabetes should be made aware, 90% should be detected, 90% of those detected should be treated, and 90% of those treated should reach their goals.

Indian Diabetes Educator Journal (IDEJ) is the first of its kind in India, and the longest running monthly diabetes educator journal since April 2015 & continues its endeavour to spread awareness, knowledge and enable healthcare teams to manage individuals with diabetes and empower them for self-care. RSSDI IDEJ will continue to keep the members of diabetes care team abreast with concepts of Diabetes Self-Management Education/Support (DSME/S) with a reach of 44000 doctors and diabetes educators digitally.

Apart from the macro and microvascular complications of diabetes, there are several health conditions that are caused due to or manifest themselves along with diabetes or may lead to or increase the risk of diabetes. This month's IDEJ aims to propagate information on some of the health conditions associated with diabetes and the latest evidence-based recommendations for their management. We hope this journal will enable the diabetes educators to spread awareness about the prevention and management of various health issues associated with diabetes.

We sincerely thank our contributors for making this issue delightful reading for our readers. We dedicate this journal to all the healthcare professionals who are working relentlessly towards making "India–The Diabetes Care Capital of the World."

Sincere Regards,

Dr. Sanjay Agarwal
RSSDI Secretary

*DSME: Diabetes Self-Management Education, DSMS: Diabetes Self-Management Support

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RSSDI Indian Diabetes EDUCATOR JOURNAL

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Cover Story: Types of Diabetes

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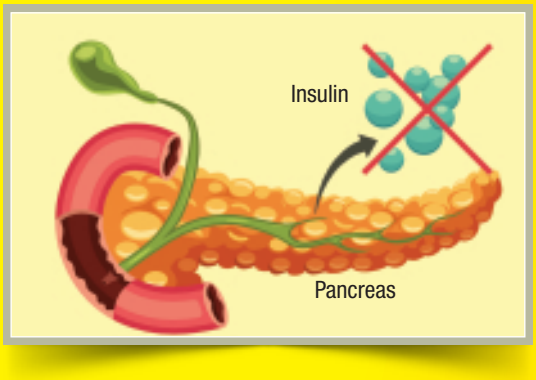
Together with cardiovascular diseases (CVD), respiratory disease, and cancer, diabetes is one of the biggest global health emergencies of the twenty-first century. Globally and in emerging nations like India, the burden of diabetes is large and rising, primarily due to rising rates of overweight/obesity and unhealthy lifestyles. China, India, and the United States are the top three nations with the highest prevalence of diabetes, and according to the International Diabetes Federation (IDF) 2021, India houses around 74 million people with diabetes.

Diabetes mellitus (DM), commonly referred to as diabetes, is a set of metabolic conditions characterized by persistently elevated blood glucose levels. The development of diabetes involves several pathogenic processes. These range from autoimmune destruction of the β-cells of the pancreas with resulting insulin deficiency to abnormalities that cause resistance to insulin action. Based on the cause and characteristics of the condition, diabetes is classified as:

Type 1 Diabetes Mellitus (T1DM)

Most cases of diabetes fall into two broad categories- Type 1 and Type 2 diabetes. Type 1 diabetes is an autoimmune condition leading to β-cell destruction, usually leading to absolute insulin deficiency. Previously, this condition was referred to as "juvenile diabetes" or "insulin-dependent diabetes mellitus" (IDDM). There is no known cause for the onset of T1DM. Several genes, including specific HLA genotypes known to affect diabetes risk, are partially inherited with T1DM. When around 80% of β-cells have been damaged, insulin deficiency leads to hyperglycemia and the risk of ketosis. People with T1DM need external insulin injections for survival.



Type 2 Diabetes Mellitus (T2DM)

The most prevalent form of diabetes is Type 2 diabetes. It is caused by a combination of resistance to insulin action and an inadequate compensatory insulin secretory response overtime. It was known as NIDDM (non-insulin-dependent diabetes mellitus) or "adult-onset diabetes" earlier but nowadays T2DM is also commonly seen in children or adolescents with excess body fat. The main causes of T2DM are genetics and lifestyle choices including obesity, unhealthy food, stress, inactivity, and urbanization. The risk of microvascular complications is similar in Type 1 diabetes and Type 2 diabetes, with diabetes

duration and blood glucose control playing major roles in their development. Both types of diabetes increase the risk of macrovascular atherosclerotic complications.

Gestational Diabetes Mellitus (GDM)

The third kind of diabetes, known as GDM, affects pregnant women who experience high blood glucose levels first time during pregnancy. In several ways, GDM and T2DM share a combination of insulin responsiveness issues and secretion issues that is generally insufficient. It happens in 2-10% of pregnancies, and it usually disappears after delivery but increases future risk of diabetes Type 2 in both mother as well as the child. Although completely manageable, GDM needs close medical monitoring. Dietary modifications, blood glucose monitoring, and in rare situations the use of insulin are all possible forms of management.

Other Specific Types

This includes specific types of diabetes due to other reasons such as monogenic diabetes syndromes (e.g. neonatal diabetes and maturity-onset diabetes of the young), exocrine pancreas diseases (e.g. cystic fibrosis and pancreatitis), and diabetes induced due to certain drug or chemical use (e.g. glucocorticoid use, diabetes post organ transplant, or caused due to certain treatments like that of HIV/AIDS).

Monogenic diabetes, an inherited kind of diabetes is caused by mutations or alterations in a single gene. The majority of monogenic diabetes mutations decrease the body's capacity to make insulin. Most cases of monogenic diabetes are misdiagnosed. DNA is obtained from the blood or saliva sample for genetic testing for monogenic diabetes. While certain types of monogenic diabetes can be managed with oral medications, others require insulin injections. The two primary types of monogenic diabetes are maturity-onset diabetes of the young (MODY) and neonatal diabetes mellitus (NDM).

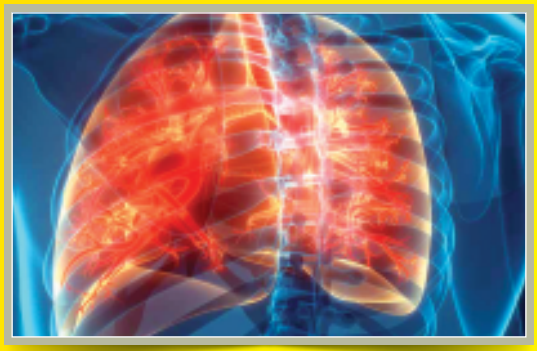
- **Maturity-onset diabetes of the young (MODY)**

MODY, a monogenic form of diabetes is characterized by the onset of hyperglycemia at an early age, mostly before the age of 25 years, although diagnosis may occur at older ages too. MODY has been linked to a variety of gene mutations, all of which reduce the pancreas' capacity to generate insulin thus elevating blood glucose levels. The individual's gene mutations determine the clinical characteristics of MODY. People who carry specific mutations may have mildly elevated blood glucose levels that remain steady throughout the course of their lives with minimal or no signs of diabetes, and no long-term consequences. Only standard blood testing may reveal their elevated blood glucose levels. Other mutations, however, demand specialized care, either with sulfonylurea or insulin therapy. Testing for MODY should be considered in people who have atypical diabetes and many family members with diabetes not characteristic of Type 1 or Type 2 diabetes. Genetic testing is available to identify the type of MODY.

- **Neonatal diabetes mellitus (NDM)**

Neonatal or congenital diabetes occurs under 6 months of age and about 80–85% of cases usually have a monogenic cause. Infants with NDM do not produce enough insulin & is sometimes confused for T1DM. However, neonatal diabetes rarely occurs after 6 months of age, whereas autoimmune Type 1 diabetes rarely occurs before 6 months of age. Neonatal diabetes can either be transient or permanent. Frequent urination, fast breathing, and dehydration are common symptoms, and elevated glucose levels in the blood or urine are a sign of NDM. A potentially fatal condition known as diabetic ketoacidosis may develop because of the body producing ketones due to a lack of insulin. Genetic testing helps to confirm neonatal diabetes. Neonatal diabetes is usually treated with sulfonylurea-glibenclamide or with insulin.

- **Cystic fibrosis-related diabetes (CFRD)**



Nowadays, between 40 and 50% of adults with cystic fibrosis survive long enough to develop CFRD. It is advised that screening for CFRD start at age 10. The build-up of sticky mucus which happens in cystic fibrosis can lead to inflammation and damage to the pancreas, leading to CFRD. The ideal test is a yearly oral glucose tolerance test (OGTT), with a 2-hour postprandial glucose level of 200 mg/dL or higher being considered diagnostic of diabetes. CFRD is associated with poor nutritional status, severe inflammatory lung disease, and greater mortality. Insulin insufficiency is the main defect in CFRD and is usually treated with insulin therapy.

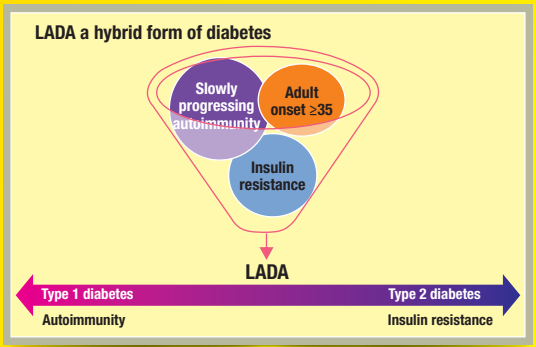
- **Pancreatic diabetes**

Type 3c diabetes refers to hyperglycemia brought on by overall pancreatic dysfunction, and pancreoprivic diabetes, which describes diabetes in the context of exocrine pancreas pathology. Pancreatic diabetes is the preferred umbrella term due to the wide range of etiologies, which include pancreatitis (acute and chronic), trauma or pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy, rare genetic disorders, and idiopathic forms. Even a single episode of pancreatitis might result in post-pancreatitis diabetes mellitus (PPDM). Concurrent pancreatic exocrine insufficiency, abnormal pancreatic imaging, and absence of T1DM-associated autoimmunity are distinguishing characteristics. Loss of glucagon and insulin secretion occurs, and insulin needs are frequently higher than anticipated.

▶ Post-transplant diabetes mellitus

Screening for hyperglycemia should be done after organ transplantation. A formal diagnosis of post-transplantation diabetes is made once the individual is stable on an immunosuppressive regimen and in the absence of an acute infection. One term used to characterize people who acquire new-onset diabetes after transplant is "new-onset diabetes after transplantation" (NODAT). Patients with undetected pre-transplant diabetes and post-transplant hyperglycemia that goes away at the time of discharge are not included in NODAT. Another term for diabetes in the post-transplant situation is "post-transplantation diabetes mellitus" (PTDM), regardless of when the condition first manifested itself.

▶ Latent autoimmune diabetes in adults (LADA)



Latent autoimmune diabetes in adults (LADA) is a slow-progressing type of autoimmune diabetes. LADA has similar clinical and metabolic characteristics with both Type 1 and Type 2 diabetes and so is also called type 1.5 diabetes by some. The three key criteria for diagnosing LADA, include adult age of onset (>30 years); the existence of any auto-antibodies against islet cells; and the absence of insulin demand for at least six months following diagnosis.

There can be many other causes of diabetes including endocrinopathies, certain drugs leading to diabetes, infections, and certain genetic syndromes like Down's syndrome, Wolfram syndrome, Turner syndrome, etc. Any form of diabetes may require insulin therapy but that itself does not classify the type of diabetes. Proper diagnosis of the type of diabetes often helps determine appropriate therapy.

Classification of Diabetes based on Etiology

I. Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   - A. Immune mediated
   - B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)

III. Other specific types
   - A. Genetic defects of β-cell function
     1. MODY 3 (Chromosome 12, HNF-1α)
     2. MODY 1 (Chromosome 20, HNF-4α)
     3. MODY 2 (Chromosome 7, glucokinase)
     4. Other very rare forms of MODY (e.g., MODY 4: Chromosome 13, insulin promoter factor-1; MODY 6: Chromosome 2, *NeuroD1*; MODY 7: Chromosome 9, carboxyl ester lipase)
     5. Transient neonatal diabetes (most commonly ZAC/HYAMI imprinting defect on 6q24)
     6. Permanent neonatal diabetes (most commonly KCNJ11 gene encoding Kir6.2 subunit of β-cell KATP channel)

Classification of Diabetes based on Etiology (Table contd...)		
7. Mitochondrial DNA	8. Others	
B. Genetic defects in insulin action		
1. Type A insulin resistance	2. Leprechaunism	3. Rabson-Mendenhall syndrome
4. Lipoatrophic diabetes	5. Others	
C. Diseases of the exocrine pancreas		
1. Pancreatitis	2. Trauma/pancreatectomy	3. Neoplasia
4. Cystic fibrosis	5. Hemochromatosis	6. Fibrocalculous pancreatopathy
7. Others		
D. Endocrinopathies		
1. Acromegaly	2. Cushing's syndrome	3. Glucagonoma
4. Pheochromocytoma	5. Hyperthyroidism	6. Somatostatinoma
7. Aldosteronoma	8. Others	
E. Drug or chemical induced		
1. Vacor	2. Pentamidine	3. Nicotinic acid
4. Glucocorticoids	5. Thyroid hormone	6. Diazoxide
7. β-Adrenergic agonists	8. Thiazides	9. Dilantin
10. γ-Interferon	11. Others	
F. Infections		
1. Congenital rubella	2. Cytomegalovirus	3. Others
G. Uncommon forms of immune-mediated diabetes		
1. Stiff-man syndrome	2. Anti-insulin receptor antibodies	3. Others
H. Other genetic syndromes sometimes associated with diabetes		
1. Down syndrome	2. Klinefelter syndrome	3. Turner syndrome
4. Wolfram syndrome	5. Friedreich ataxia	6. Huntington chorea
7. Laurence-Moon-Biedl syndrome	8. Myotonic dystrophy	9. Porphyria
10. Prader-Willi syndrome	11. Others	
IV. Gestational diabetes mellitus		

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Frequently Asked Questions

1. I am a 37-year-old man. In the past few months, I started experiencing symptoms such as excessive thirst and hunger, frequent urination, drastic weight loss, muscle loss, night sweats, fatigue, and tiredness. After consulting a doctor and completing the suggested blood tests, I was diagnosed with a type of diabetes that requires insulin treatment. Isn't insulin-dependent type 1 diabetes seen in children and teenagers?

Ans. The condition that you are stating is similar to Type 1 and Type 2 diabetes mellitus, but is actually latent autoimmune diabetes in adults (LADA). It is a condition that evolves at a much slower pace, as compared to classic Type 1 diabetes. Like Type 1 diabetes, LADA develops when your pancreas stops making enough insulin, most likely as a result of some sort of "injury" that gradually harms the insulin-producing cells in the pancreas. Many experts think LADA, also known as type 1.5 diabetes, as a subset of Type 1 diabetes. Those with LADA are often older than 30 years of age. People with LADA are usually misdiagnosed as having Type 2 diabetes because they are older when symptoms first appear as compared to Type 1 diabetes. Also in LADA, your pancreas still has the capacity to generate a significant amount of insulin in the first 6 months of diagnosis, so LADA can initially be controlled with dietary changes, weight loss if necessary, exercise, and perhaps oral medicines. However, you will eventually require insulin injections as your body gradually loses the capacity to make insulin.

2. I am a 15-year-old girl; I have recently been diagnosed with PCOS. My doctor has informed me that for my height my weight is on the higher side. I have been put on medication for PCOS, and have been suggested to follow a Diabetic Diet. According to my reports my fasting blood sugar is normal, so why do I have to follow a diabetic diet?

Ans. Polycystic Ovary Syndrome (PCOS) is a hormonal condition that affects women and results in a specific set of symptoms, including irregular periods, weight gain, excess facial hair, acne and even infertility. Acanthosis Nigricans (Black patches on the armpits, groin, and back of the neck) is a common symptom in PCOS, which develops due to 'Insulin Resistance.' A majority of overweight women with PCOS have insulin resistance, wherein the body is unable to use the insulin effectively which then leads to rise in blood glucose levels and eventually increases the risk of developing diabetes. Since the risk of developing diabetes is high in PCOS and especially PCOS with overweight or obesity, you are advised to follow a diet for diabetes. There is nothing like a diabetic diet, it just means eating healthy balanced meals which have complex carbohydrates and fiber, adequate protein, and low fat. It includes avoiding refined and processed foods with high glycemic index. This kind of diet will help in weight loss as well as in better blood glucose control and will prevent the progression to diabetes.

3. My aunty is 75 years old and has had diabetes for the past 15 years. Lately, she has been diagnosed with Alzheimer's. Is there any specific diet that needs to be followed to prevent further degeneration?

Ans. Alzheimer's is irreversible. Research has stated that deranged blood glucose levels are capable of further worsening the condition. Thus in order to prevent further degeneration, it is important for her to regulate her blood glucose levels, by following a healthy lifestyle- healthy eating, meditation, and physical activity. With respect to Alzheimer's, there are certain foods that help in preventing and lessening the symptoms such as Omega-3 fatty acids (walnuts, flaxseeds, fatty fish, chia seeds), Resveratrol (grapes, blueberries, strawberries, cherries, cranberries, etc.), Vitamin E (pumpkin seeds, sesame seeds, sunflower seeds, almonds), Vitamin C (citrus fruits, guava, amla), Protein (non-vegetarian foods, milk & milk products, pulses, nuts, soy). She should avoid foods high in saturated and trans-fats, such as red meat, butter, ice cream, commercially baked goods, and some margarine that include partially hydrogenated oils. The MIND (Mediterranean-DASH Intervention for Neurodegenerative Delay) Diet which focuses on the intake of plant-based foods, and limiting the intake of animal products that are high in saturated fat is seen to be beneficial in Alzheimer's disease.

4. My 10-year-old son has Type 1 diabetes. Recently, he has been diagnosed with Celiac disease and is on a gluten-free diet. However, he has been complaining of fatigue, and tiredness, he is unable to concentrate on his studies and has low energy levels. Even after following the suggested meal plan and fairly good blood glucose levels, what could be the reason for his tiredness?

Ans. The symptoms mentioned are that of an iron deficiency, which could be a result of Celiac disease. Consumption of gluten damages the small intestine and causes malabsorption of nutrients leading to their deficiency in the body. It is best to consult a doctor for the same, in case he needs supplementation for iron. You can also include iron-rich foods in his diet like garden cress seeds, green leafy vegetables, ragi, horsegram, legumes, etc. Now that you have started the gluten-free diet, the absorption of all nutrients will improve as the small intestine heals and he will feel better once the iron levels go up. Visit a doctor and get the required blood tests done to confirm the cause of these symptoms.

Celiac Disease and Diabetes

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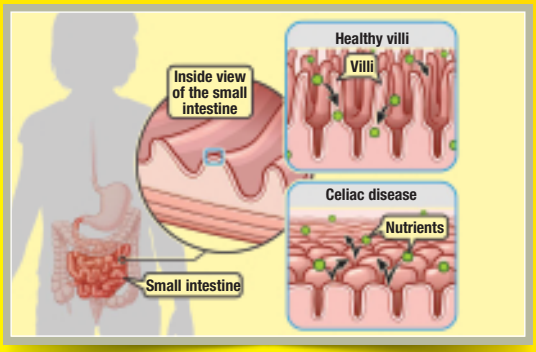
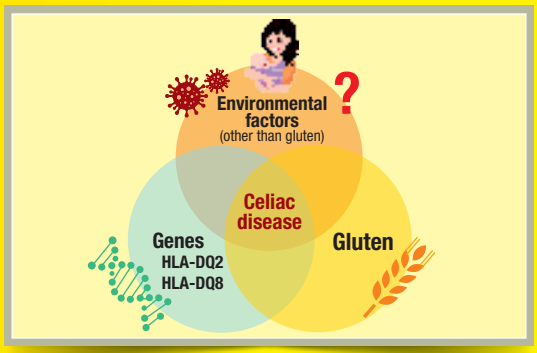
For those who are genetically predisposed, celiac disease (CD) is a chronic immune-mediated condition marked by inflammation brought on by gluten and structural damage to the small intestinal mucosa. CD is more commonly seen with Type 1 diabetes (T1D), as it shares a genetic tendency, and both are autoimmune disorders. Estimates of the prevalence of CD, that affect T1D patients, range from 3-16%, with a typical prevalence of 8%. Type 2 diabetes, which is not autoimmune-mediated, is not commonly associated with CD. Both conditions have rising incidences around the globe, indicating that environmental influences are just as essential in disease pathogenesis as hereditary ones. The gut microbiome and infectious diseases, among other things, influence innate and adaptive immunity to raise the risk of both CD and T1D, according to recent research.



Risk factors



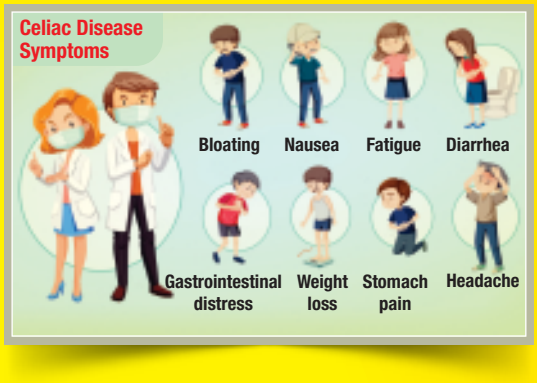
Among genetic risk factors, the strongest link is with the HLA class II DQ region; however, at least 39 non-HLA loci are associated with CD. Although HLA is a very obvious common risk factor, it is likely that other genetic, immunological dysregulation, and environmental variables also play significant roles in the development of CD. Similar environmental risk variables, like infantile feeding methods, breastfeeding, and viral infection exposure, have been found in studies on T1D and CD. Regarding infant feeding habits, it has been discovered that either an early (before 3 months) or a late (after 7 months) introduction of cereal is related with autoimmune seropositivity in new-borns who are genetically predisposed for both T1D and CD. Despite conflicting evidence from studies, breastfeeding seems to be protective against both disorders. Viruses seem to raise the risk of T1D and CD. There is evidence linking enteroviruses to increased risk for T1D. Childhood rotavirus infection has been linked to CD as a risk factor.

Screening

It is advised to screen T1D patients for CD upon diagnosis and then once every two years for the subsequent six years because of the higher prevalence of CD in T1D and the lack of symptoms. Deaminated gliadin peptide (DGP) IgA and IgG antibodies, tissue transglutaminase (tTG) IgA, and endomysial (EMA) IgA, are the sensitive and specific serologies used for screening. Villous atrophy and an increase in intraepithelial lymphocytes are indicators of positive serologies on duodenal biopsies. At least five small intestinal biopsies taken from the duodenum offer the maximum diagnostic yield, which continues to be the gold standard for the confirmation of CD. One important factor while screening is that the patient should be on a gluten-containing diet to avoid false negative results.

Symptoms

Signs and symptoms of CD may become distinct at any age through adulthood.



- Classical symptoms in CD (with or without concomitant T1D) include diarrhea, bloating, weight loss, and growth failure (in children).
- Non-classical (intestinal & extra-intestinal) symptoms include constipation, heartburn, neuropathy, mouth ulcers, and ataxia among others.
- Clinical signs include malabsorption, vitamin deficiencies, iron deficiency anemia, malnutrition or low bone density with or without concomitant symptoms, failure to thrive, short stature, diarrhea, anorexia, constipation, vomiting, and abdominal distension.
- Subclinical (silent CD) diseases are seropositive patients with no gastrointestinal or extra-intestinal manifestations.

Dietary tips

The standard therapy for CD is a gluten-free diet (GFD). Gluten is a type of storage protein found in cereals such as wheat, barley, and rye. People with CD should consult a qualified dietician for GFD instructions and must follow this diet for their entire life. Multivitamin supplements can be considered if the requirements cannot be met via diet alone.

- GFD requires the complete removal of gluten-containing foods including wheat, barley, and rye, and products made from them like bread, biscuits, vermicelli, rye bread, malted beverages, etc.
- Gluten-free cereals like rice, bajra, jowar, and other millets can be consumed safely.
- The threat of cross-contamination is a daily issue for individuals on GFD. Sharing cupboards, countertops, and kitchen appliances with individuals who do not follow a GFD presents possible contamination

opportunities that impair the diet's success. For increased safety, meals should be prepared in separate utensils and stored away from non-gluten-free foods.

- Spices like asafoetida (hing) may contain wheat and are therefore not free from gluten so care should be taken while selecting the right spices which do not contain wheat at all.
- Oats may be contaminated with gluten if processed in a factory that also processes wheat.
- While buying packaged foods, care must be taken to check for gluten-free labels and one must read the ingredient list carefully to be sure that the product does not contain wheat barley, rye, or any derivative of these.

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Did You Know?

Prader-Willi Syndrome can lead to Type 2 Diabetes Mellitus

Prader-Willi Syndrome (PWS) is a genetic disorder usually caused by the deletion of paternally inherited imprinted genes at chromosome 15q11-q13. In 60–65% of cases, the cause is a paternal 15q11-q13 deletion whereas, in remaining cases, the cause is maternal uniparental disomy of chromosome 15 or rarely an imprinting defect or translocation. This condition is linked to hypothalamic dysfunction. People with PWS have learning impairments and mild to moderate intellectual impairment. Common behavioral problems frequently observed in children include temper outbursts, stubbornness, and compulsive behavior like picking at the skin. Sleep abnormalities can also occur. Short stature, small hands and feet, and distinguishing facial characteristics such as a narrow forehead, almond-shaped eyes, and a triangular mouth are further symptoms of this condition. Genitals are undeveloped in both affected males and females. Puberty is delayed or incomplete, which can lead to infertility and most affected individuals are unable to have children. A new-born with PWS typically has a lower birth weight than average, weak muscles (hypotonia), and trouble sucking. They develop excessive weight gain, hyperphagia, and liking towards food that leads to severe early obesity in the absence of strict regulation of food access and nurturing environment. Up to nine months of age, people with PWS show poor eating but after that point, due to hyperphagia and a lack of satiety caused by hypothalamic-pituitary dysregulation, they tend to become obese. This can lead to severe obesity in childhood, which often progressively develops into Type-2 diabetes mellitus (T2DM), which is eventually associated with increased morbidity and mortality in PWS. T2DM affects around 20% of people with PWS. It is more common in people with PWS who have severe obesity, psychiatric and metabolic disorders, and a family history of overweight and T2DM. The treatment recommendations are the same as that for the general population with T2DM. There is a need for weight management and regular screening to prevent developing T2DM in people with PWS.

Resources:

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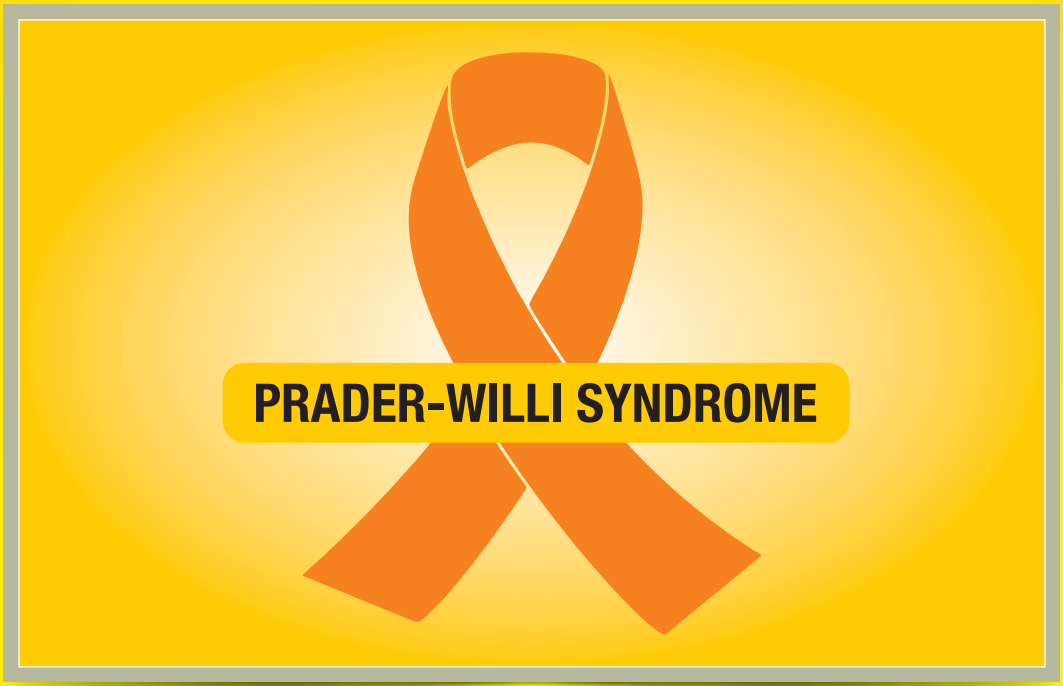
2. Yang A, Kim J, Cho SY, Jin DK. Prevalence and risk factors for type 2 diabetes mellitus with Prader-Willi syndrome: a single center experience. *Orphanet J Rare Dis.* 2017;12(1):146. Published 2017 Aug 30. doi:10.1186/s13023-017-0702-5

Facts and Figures

1. The incidence of Prader Willi Syndrome (PWS) from world literature is one in 15,000 live births.
2. The prevalence of PWS is highest in Caucasians.
3. Despite of a higher prevalence, very few case studies of PWS are published among the Indian population.
4. In a recent study on PWS children and adolescents in India, 50% of those with PWS had diabetes.

Resources:

- Sridhar S, Nazirudeen R, Ramasamy S, Natarajan V, Thiagarajan K, Karthika LN. Clinical Profile and Molecular Genetic Analysis of Prader - Willi Syndrome: A Single Center Experience. *Indian J Endocrinol Metab.* 2022;26(4):384-388. doi:10.4103/ijem.ijem_122_22



What's Trending? Is Diabetes a Disability?

Dr. Bhavesh Patel

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Diabetes is regarded as a severe health risk because of its rising incidence, the numerous microvascular, and macrovascular health issues that it causes, and its effect on the quality of life. According to the IDF Atlas, 2021, 10th Edition, India has the highest number of children and adolescents with Type 1 diabetes (T1DM) in the world with approximately 229,442 individuals in the age group of 0-19 years diagnosed with T1DM. An estimated 24,000 newly diagnosed cases in the age group of 0-19 years emerge each year.

Apart from other health conditions, diabetes also increases the risk of various disability domains by 50–90%, including mobility loss, a reduction in instrumental or basic daily living activities, and employment disability. According to various studies, diabetes-related impairment may also be mediated by particular physiological variables, such as inflammation, insulin resistance, hyperglycemia, and sarcopenia.

Understanding the relationship between diabetes and disability is important from several distinct perspectives. Loss of physical function may be more alarming and detrimental to the quality of life for people with diabetes than the identification of classic clinical problems like retinopathy and neuropathy.

Unfortunately, diabetes has not been included in the list of disabilities in India, which if included would aid to ensure that all people with diabetes can get free access to insulin, medications, continuous glucose monitoring, and insulin pumps. This would help to provide a conducive environment at school, in their families, and at the workplace.

Type 1 Diabetes is specifically categorized as a disability in several countries, including the US and the UK. People with diabetes are given all the accommodations and rights they need to live their lives under the Americans with Disabilities Act (ADA).

Children with T1DM are able to test their blood glucose levels in class, receive extra time in examinations, if they experience low blood glucose levels in the middle of them, or receive reasonable accommodations at work like a space to check their blood glucose levels or a few extra minutes to treat a hypoglycemic episode. Employees are protected by the ADA, and employers are not allowed to inquire about or treat a T1DM employee differently because of their diabetes.

In the UK, if a person has either Type 1 or Type 2 diabetes, they will be eligible for certain benefits, depending on the extent to which their condition affects their life. Every person with diabetes in the UK is entitled to free prescriptions for diabetes medication as well as free eye check-ups starting at age 12. These services also include yearly screenings for diabetic retinopathy.

There are additional benefits available to those with diabetes-related disabilities, in case they need help or if they're unable to work. Parents can also claim on behalf of children with diabetes. Due to the free availability of sensors for continuous glucose monitoring and insulin pumps, children in advanced countries are free from multiple injections and finger pricks every day. Citizens with impairments also receive a variety of other benefits and allowances.

In India, the government should prioritize providing free access to insulin, glucose monitoring equipment, medications, and an environment that will allow them to nurture productive and effective citizens. They should develop affordable insurance plans for people with diabetes to ensure that all the needs of these people are met and that they can live their lives with respect and without difficulty.

Resources:

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3. Declare Type 1 Diabetes a disability in India. Available at: https://www.change.org/p/prime-ministers-office-declare-type-1-diabetes-a-disability-in-india
4. American Diabetes Association. Is Diabetes a Disability? Available at: https://diabetes.org/tools-support/know-your-rights/discrimination/is-diabetes-a-disability#:~:text=The%20short%20answer%20is%20%22Yes,diabetes%20are%20protected%20as%20disabilities.

Diabetes and Wolfram Syndrome

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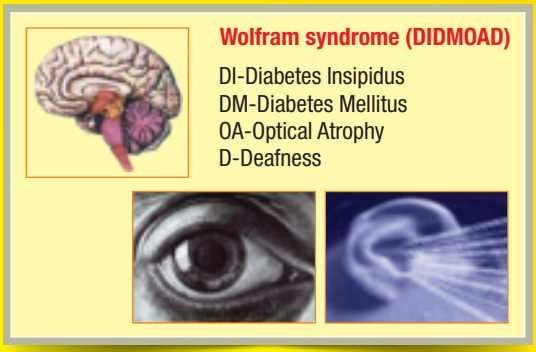
Wolfram syndrome (WS) is a rare genetic disorder, also known as DIDMOAD, marked by the four most common features - **D**iabetes **I**nsipidus, **D**iabetes **M**ellitus (type1), **O**ptic **A**trophy, and **D**eafness. The diagnosis is suspected in instances of childhood-onset diabetes mellitus along with optic atrophy, and this visual impairment is not caused due to diabetes. This syndrome was first explained by Wolfram and Wagener in 1938. WS has two main types: Type 1 (WS1) and Type 2 (WS2), primarily distinct from one another due to their underlying genetic causes. Wolfram syndrome is thought to affect 1 in 770,000 people worldwide. Diabetes mellitus is generally the first sign of WS followed by optic atrophy that occurs later. Unlike Type 1 diabetes mellitus, it is non-auto-immune, but insulin dependent. Also, microvascular complications like retinopathy (which can cause blindness) or nephropathy (which can cause kidney failure) are not observed, and a lower insulin requirement with lower levels of HbA1c can be seen. In one European Study, it was noted that patients with WS experienced severe hypoglycemia more frequently than those with Type 1 diabetes. This was the first study describing WS-related diabetes in a significant European population. Diabetes insipidus affects about half of people with WS being more common in WS1. This is when the body can't concentrate urine due to insufficient production of the hormone vasopressin by the posterior pituitary gland. Polydipsia and polyuria are its classical features.



Other symptoms in WS include sensory neuronal hearing loss, urinary tract problems, bowel dysfunction, ataxia (uncontrolled gait), dysphagia, dysarthria, dementia, and other endocrine disorders like hypogonadism, hypothyroidism, and growth retardation. In some cases, psychological symptoms like anxiety and depression have also been reported. A genetic screening confirms the diagnosis of WS. The symptoms may vary from person to person and can range from mild to severe. WS has a negative impact on the quality of life and participation in many daily activities of those who are affected. Currently, the first step in WS treatment involves managing clinical symptoms, especially diabetes. However, occupational therapy, especially in children, fostering social interaction and identifying sports that are appropriate for any visual, auditory, or neurological difficulties are also focused upon for its treatment. Wolfram syndrome impacts different organs and systems in the body. Thus, multidisciplinary treatment provided by medical doctors and other healthcare workers from various fields may be required for efficient patient management.

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Diabetes and Gastroparesis

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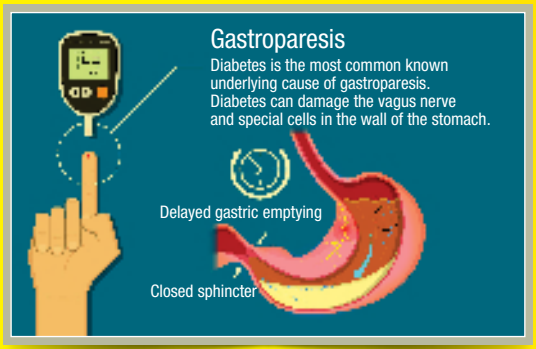
Uncontrolled diabetes affects almost every organ system in the body. The period and severity of the condition may directly impact specific organs. The link between gastric dysfunction and diabetes is well-known for the past 70 years.

Gastrointestinal (GI) complications are common in individuals with long-standing diabetes. Esophageal dysmotility, gastro-esophageal reflux disease (GERD), gastroparesis, etc. are some GI-related common disorders in people with diabetes.

What is Gastroparesis?

Gastroparesis is a condition characterized by delayed gastric emptying without any evidence of mechanical obstruction. It happens due to uncontrolled diabetes and is a form of autonomic neuropathy. It occurs when there is damage to a vagus nerve which delays food from moving into the intestines from the stomach. It is commonly seen in people who have had diabetes for more than 10 years and those with microvascular complications.



It was observed that Diabetic Gastroparesis is frequently seen in individuals with Type 1 diabetes (5.2%), as compared to people with Type 2 diabetes (1%). Studies also report that females have higher chances of developing gastroparesis as compared to males.

The most typical gastroparesis symptom is nausea. Other typical symptoms include vomiting, post-meal fullness, early satiety, bloating, and upper abdominal pain.

Pathophysiology

Diabetic gastroparesis has a complex pathophysiology that is still poorly understood. The development of diabetic gastroparesis is known to be predisposed by elevated glycated hemoglobin levels. Research suggests that acutely elevated serum glucose levels may contribute to delayed gastric emptying by reducing stomach motility. This phenomenon can be triggered by several causes, including the death of enteric neurons, which may happen in hyperglycemic situations (although it has only been seen in rodents) and then result in delayed stomach emptying and rapid intestinal transit.

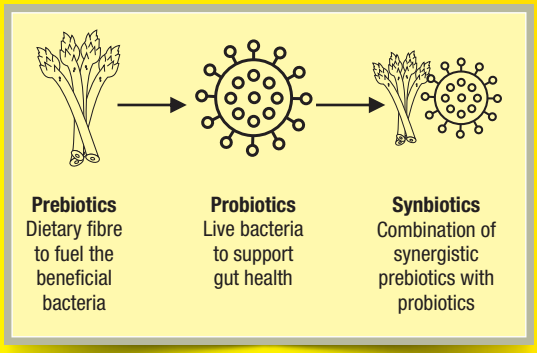
The pathophysiology of gastroparesis has also been linked to oxidative stress. According to studies, the gastric macrophages' ability to up-regulate the enzyme heme oxygenase-1 (HO-1) shields the stomach from oxidative stress. Loss of HO-1 in people with diabetes leads to a subsequent loss of receptor tyrosine kinase (c-Kit) expression, which is crucial for maintaining normal stomach function.

Other elements that contribute to the pathophysiology include defective inhibitory nitric oxide-containing neurons, missing or aberrant interstitial Cajal cells (the electrical pacemakers responsible for promoting muscular activity and neurotransmission in the stomach), smooth muscle fibrosis, and inappropriate immunological infiltrates with macrophages.

Treatment

General approaches in the management of gastroparesis include good hydration, correction of electrolyte imbalances, targeting glycemic control, and symptom reduction with pharmaco-therapeutic agents.

Dietary tips to manage Gastroparesis



- Include probiotics, prebiotics & synbiotics:

  Probiotics: Yogurt, sauerkraut, tempeh, kimchi, kombucha, and traditional buttermilk are all sources of probiotics in food.

  Prebiotics: Garlic, chicory, onion, wheat, raw banana, barley, tomato, soybean, human and animal milk, peas, beans, seaweeds, and microalgae.

  Synbiotic: Kefir is a symbiotic food source.
- Include low-fat, low-fiber foods in your diet: Fat and fiber delay gastric emptying further and so can be limited in the diet.
- Include liquid-based meals as the rate of liquid emptying from the stomach is not affected as much.
- Consume an average of 2-3 liters of water in a day.
- Ensure there is a minimum of 30-45 minutes of physical activity done daily.
- Consume small, frequent meals instead of large calorie dense meals.
- Cessation of smoking & alcohol consumption: Smoking and alcohol both slow down stomach emptying, so they should be avoided.

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1. Start trial- Gnanaraja TV et al., Indian J Endocrinol Metab. 2017 Sep-Oct; 21(5): 745–750.MLA. 2. Diabetes Obes Metab. 2017; 19:1189-1192. 3. Data on file.

Information
Metformin hydrochloride ((as prolonged release) and glimepiride tablets
Glycomet-GP 0.5/Glycomet-GP 0.5 Forte/ Glycomet-GP 1/ Glycomet-GP 1 RED/ Glycomet-GP 2/ Glycomet-GP 2 RED/ Glycomet-GP 3/ Glycomet-GP 3 RED/ Glycomet-GP 4/ Glycomet-GP 4 RED/ Glycomet-GP 1 Forte/ Glycomet-GP 2 Forte/ Glycomet-GP 3 Forte/ Glycomet-GP 4 Forte.

Abridged Prescribing Information

Composition: • Glycomet GP 0.5 mg: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 0.5 mg. • Glycomet GP 0.5 Forte: Each uncoated tablet contains: metformin hydrochloride IP (as prolonged release form) 1000 mg and glimepiride IP 0.5 mg. • Glycomet GP 1: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 1 mg. • Glycomet GP 1 RED: Each uncoated tablet contains: metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 1 mg. • Glycomet GP 2: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 2 mg. • Glycomet GP 2 RED: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 2 mg. • Glycomet GP 3: Each uncoated tablet contains: metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 3 mg. • Glycomet GP 3 RED: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 3 mg. • Glycomet GP 4: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 4 mg. • Glycomet GP 4 RED: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 4 mg. • Glycomet GP 1 Forte: Each uncoated tablet contains: metformin hydrochloride IP (as prolonged release form) 1000 mg and glimepiride IP 1mg. • Glycomet GP 2 Forte: Each uncoated tablet contains: metformin hydrochloride IP (as prolonged release form) 1000 mg and glimepiride IP 2mg. • Glycomet GP 3 Forte: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 1000 mg and glimepiride IP 3mg. • Glycomet GP 4 Forte:Each uncoated tablet contains:metformin hydrochloride IP (as prolonged release form) 1000 mg and glimepiride IP mg. **Indications:** Glycomet GP is indicated for the management of patients with type 2 diabetes mellitus (T2DM) when diet, exercise and single agent (metformin hydrochloride or glimepiride alone) do not result in adequate glycemic control. **Dosage and Administration:** Dosage of Glycomet GP should be individualized on the basis of effectiveness and tolerability while not exceeding the maximum recommended daily dose of glimepiride 8mg and metformin 2000 mg. Initial dose: 1 tablet of Glycomet GP should be administered once daily during breakfast or with the first main meal. Do not crush or chew the tablet. In several cases the tablet may remain intact during transit through the gastrointestinal (GI) tract and will be eliminated in feces as hydrated mass (ghost matrix). Patients should be advised that this is normal as all drug components have already been released during GI transit. **Contraindications:** In patients hypersensitive to glimepiride, other sulfonylureas, other sulfonamides, metformin or any of the excipients of Glycomet GP; pregnancy and lactation; diabetic ketoacidosis, diabetic pre-coma, in patients with eGFR<30 ml/min/ 1.73 m², acute conditions with the potential to alter renal function (dehydration, severe infection, shock, intravascular administration of iodinated contrast agents), acute or chronic disease which may cause tissue hypoxia (myocardial infarction, shock, cardiac/respiratory failure) hepatic insufficiency, acute alcohol intoxication, alcoholism. **Warnings:** Keep out of reach of children. Patient should be advised to report promptly exceptional stress situations (e.g. trauma, surgery, febrile infections). Blood glucose regulation may deteriorate and a temporary change to insulin may be necessary to maintain good metabolic control. In case of lactic acidosis, patient should be hospitalized immediately. **Precautions:** In the initial weeks of treatment, the risk of hypoglycemia may be increased and necessitates especially careful monitoring. Serum creatinine levels should be determined before initiating treatment and regularly thereafter: at least annually in patients with normal renal function. Intravascular contrast studies with iodinated materials can lead to acute alteration of renal function. In patients in whom such study is planned, Glycomet GP should be temporarily discontinued at the time of or prior to the procedure, and withheld for 48 hours subsequent to the procedure and reinstituted only after renal function has been re-evaluated and found to be normal. Use of Glycomet GP should be discontinued 48 hours before any surgical procedure. **Adverse reactions:** For glimepiride – hypoglycaemia; temporary visual impairment; GI symptoms like nausea, vomiting, abdominal pain, diarrhoea may occur; increased liver enzymes, cholestasis and jaundice may occur; allergic reactions may occur occasionally. For metformin – GI symptoms like nausea, vomiting, abdominal pain or discomfort may occur.

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High Dose Metformin improves Insulin sensitivity Vs Other OADs

Source: 1. JAPI 2020 68.51.-55 2. Data on File, 3. Cureus 2020; 12(9): e10.7759/cureus.1070 4. Diabetes Technology & Therapeutics 2019..2,79-84 5. Kalra, et al.: Sulfonylurea and combinations: International Task Force Indian J Endocr Metab 2018;22:132-57.

Prescribing information

Information: Metformin hydrochloride (as prolonged release) and glimepiride tablets. 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Alzheimer's Disease as Type 3 Diabetes

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MD Medicine, DM Endocrinology

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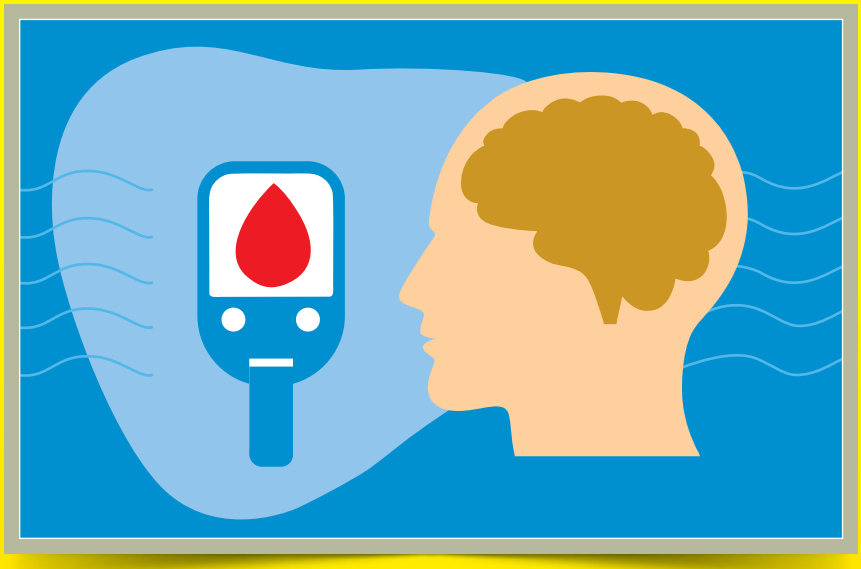
The two most common forms of diabetes include Type 1 (T1DM) and Type 2 diabetes mellitus (T2DM). However, there is a recently identified type of diabetes called Type 3 diabetes (T3DM) which is a brain-specific type of diabetes. It is known as Alzheimer's disease (AD). It is a metabolic syndrome that may cause anomalies connected to advancing brain insulin resistance, resulting in impaired central insulin signaling mechanisms, the build-up of neurotoxins, neuronal stress, and ultimately leading to a path of neurodegeneration.

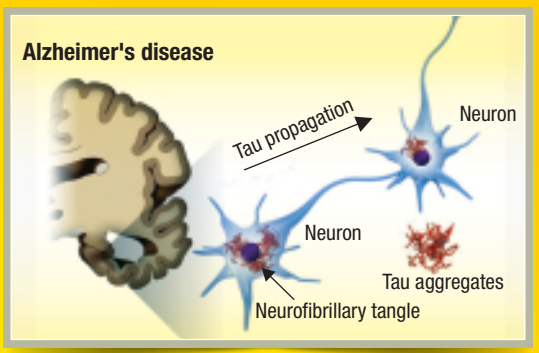
Along with diabetes and cardiovascular diseases, the possibility of having neurodegenerative diseases is also increasing. Cognitive impairment and dementia, particularly vascular dementia and AD, are more common in people with diabetes. T2DM has been linked to an elevated risk of dementia and AD by 45–90%. By 2030 and 2050, the number of dementia sufferers is predicted to increase by 1.6 and 2.8 times, respectively.

Alzheimer's disease (AD) is the most prevalent form of dementia and a chronic neurodegenerative illness that is characterized by altered behavior and personality as well as memory loss and cognitive decline. Brain lesions that occur in AD are accompanied by synaptic dysfunction, neuronal disorders, and neurodegeneration while the disease is characterized by extracellular plaques of insoluble β-amyloid protein and intracellular neurofibrillary tangles (NFTs) of hyperphosphorylated tau protein.

The severity of cognitive impairment depends on the type of diabetes, age of onset, and other comorbidities. There is accumulating evidence demonstrating that hyperglycemia may increase the risk of mild cognitive impairment (MCI) or AD. There has been an increase in interest in the involvement of β-amyloid and tau protein in the peripheral nervous system and its organs as well as in causing insulin resistance in recent years due to the mounting evidence that suggests AD may represent diabetes-related dementia as Type 3 diabetes.



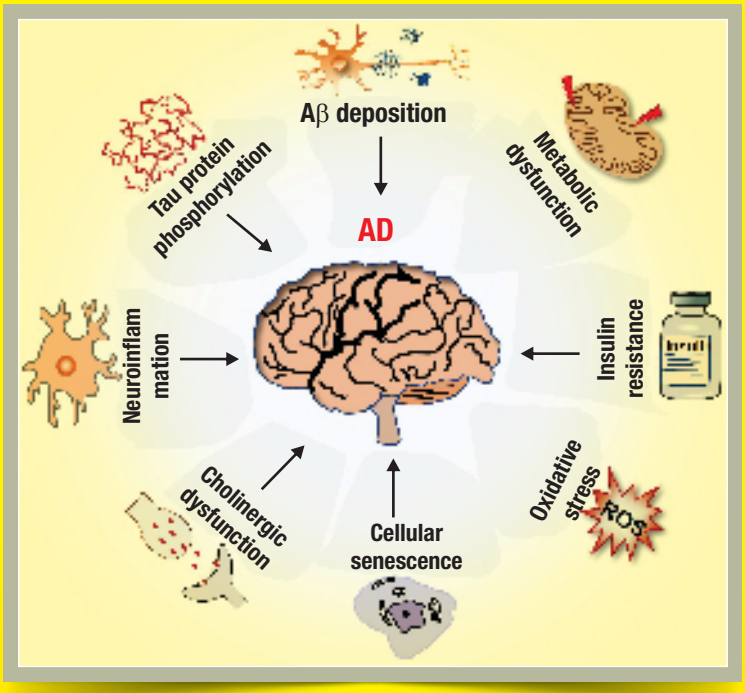
Alzheimer's disease and T2DM, share many common pathophysiological characteristics and signaling pathways, such as neuroinflammation, oxidative stress, advanced glycosylation end products, mitochondrial dysfunction, and metabolic syndrome. Significant involvement of β-amyloid, tau protein, and amylin in both diseases is also indicated. There is involvement of β-amyloid in brain insulin resistance, thus contributing to the pathological vicious cycle that forms between oxidative stress and neuroinflammation.



Diabetes, obesity, and AD may all have a common trait called insulin resistance. In patients with T2DM insulin resistance has been highly correlated with the reduced rate of glucose metabolism in specific areas of the brain such as the frontal, temporal, and parietal cortex areas. In addition, in the white and grey matter of patients with T2DM who also suffer from mild cognitive impairment (MCI), a total cerebral decrease in 18-fluoro-deoxyglucose (FDG) intake has been observed.

Since insulin may not be completely necessary for neuronal glucose uptake, the concept of insulin resistance in the brain is more closely tied to compromised insulin signaling pathways. Overall, poor insulin signaling is linked to altered brain metabolism that could result in brain dysfunction, offering potential explanations for the relationship between diabetes, obesity, and AD.

When it comes to screening and treatment, not many people with diabetes are routinely evaluated for cognitive outcomes and they are rarely treated for cognitive impairment. Similarly, people with AD are not regularly screened for high levels of insulin or for T2DM. The evident involvement of T2DM in the pathology of AD requires a holistic and modern therapeutic approach based on current data regarding this neurological disease.



Nutritional intervention for AD

Nutrition is considered an important element that can alter the risk of dementia. Nutrients reach the brain via the blood-brain barrier (BBB). It is generally believed that an anti-diabetic strategy can protect against the development of dementia caused by diabetes. However, few other strategies, have been shown to be protective. Resveratrol, which has been found to be helpful in lowering the incidence of dementia linked to diabetes, is the most successful nutritional component. So far, it has been established that polyphenols, phospholipids, omega-3 fatty acids, and antioxidants are all good for brain health.

There is evidence to show that one kind of diet called the 'MIND' diet which stands for "Mediterranean-DASH Intervention for Neurodegenerative Delay" helps to reduce the risk of AD by about 53%. This diet is a combination of the DASH diet and Mediterranean Diet with the main focus being the protection of the aging brain. The MIND diet stresses on intake of fruits, mainly berries, green leafy vegetables, nuts, olive oil, whole grains, beans, fish, and poultry. The MIND diet also limits the intake of butter, cheese, red meat, fried foods, and sweets. Although the aim of the MIND diet is good brain health, it may also benefit heart health, diabetes, and some cancers because it includes components of the Mediterranean and DASH diets, which have been proven to lower the risk of these diseases. In conclusion, both diabetes mellitus and Alzheimer's disease are common in the elderly population and are becoming more prevalent globally. There is enough evidence to show that both the conditions are linked to each other, and so current and future research is warranted within each of these conditions which can help in development of new treatment options and a better understanding of the pathogenesis of each condition.

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Diabetes Educator Tip of the Month

Contributed by
Name: Saili Randive

PG Dip Dietetics, CDE

Dietary Tips for Hyperuricemia in Diabetes Mellitus

Hyperuricemia occurs when there is an excessive amount of uric acid in the blood. High uric acid levels can lead to a painful type of arthritis called gout. Hyperuricemia often accompanies diabetes as a metabolic syndrome.

Dietary tips for Hyperuricemia

1. One of the key elements that raise the risk for elevated uric acid levels is being overweight. As a result, losing weight gradually is advisable.
2. Vitamin C-rich foods like citrus fruits, guava, and gooseberry (amla) should be included in the diet since they can help to reduce uric acid levels.
3. Dehydration is one of the reasons for elevated uric acid levels. Drinking adequate fluid is crucial to promote uric acid clearance.
4. Intake of fructose-containing foods must be limited. Soda, soft drinks, and juices sweetened with high-fructose corn syrup (HFCS) should be avoided. Food labels must be read carefully to see if a product contains HFCS.
5. Liver breaks down purine from food and produces uric acid. Therefore, purine-rich food intake must be limited.
6. Regular physical activity helps in overall well-being preventing high uric acid levels.
7. An alkaline diet with adequate hydration is recommended along with a reduction in alcohol intake.

Food group	Foods to be avoided
Cereal, millet, and their products	Yeast and yeast-containing products like bread, naan
Meat and meat products	Organ meats such as liver, kidney, heart, and tongue red meat, shrimps, mackerel, sardines, fish eggs (roe) sausages
Vegetables	Peas, spinach, cauliflower, brinjal, mushroom
Fruits	Sapota (chickoo), custard apple
Miscellaneous	Aerated drinks, alcohol especially beer, sugar-sweetened beverages, sugary foods, foods containing high-frucotse corn syrup

Resources:

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2. Kakutani-Hatayama M, Kadoya M, Okazaki H, *et al.* Nonpharmacological Management of Gout and Hyperuricemia: Hints for Better Lifestyle. *Am J Lifestyle Med.* 2015;11(4):321-329. Published 2015 Sep 2. doi:10.1177/1559827615601973
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Superfood: Guava

The Myrtaceae family member guava *(Psidium guajava)* is nutrient-rich and has potent therapeutic qualities. It is frequently grown in Asian nations. Both the fruit and the leaves have excellent medicinal properties. The pulp is whitish or pinkish and contains many seeds.

Guava has anti-inflammatory, anti-diabetic, anti-bacterial, anti-diarrheal, anti-hypertensive, and antipyretic qualities. It is also rich in vitamins and minerals.

Nutritional benefits

- Rich in Vitamin A and Vitamin C.
- Antioxidant and anti-inflammatory properties.
- Low in glycemic index and glycemic load.
- Contains bioactive substances such as pinene, menthol, β-sitosterol, and quercetin which have health benefits.

Health benefits

Anti-diabetic

Guava pulp contains a good amount of pectin, a soluble dietary fiber that helps in blood glucose control.

Pedunculagin, Isostrictinin, and Strictinin are only a few of the flavonoid glycosides that are abundant in pulpy guava and are known to promote insulin sensitivity. Tannic acid and gallic acid, two polyphenols, present in the fruit pulp have anti-glycation properties.

Anti-hypertensive and anti-hyperlipidaemic:

Being high in soluble fiber, guava helps in lowering LDL cholesterol, triglycerides, and total cholesterol. Guava contains polyphenols and flavonoids that prevent lipid peroxidation.

Guava's high potassium and fiber content aids in decreasing cholesterol levels and blood pressure.

Anti-oxidant

The antioxidant activity of guava is due to the fruit's high Vitamin C content. Gallic acid, polyphenols, and flavonoids also contribute to this feature.

How to consume?

Guava is a fruit with a pulpy texture. Semi-ripe guava is the best for people with diabetes. It can be consumed as a mid-meal snack. However, the common practice of adding salt over guava while consuming it should be avoided to keep the sodium intake within safe limits.

Recommended intake

For people with diabetes, a medium-sized guava with a weight of 60 gm delivers around 20 kilocalories, 3 g of carbohydrates, 0.8 g of protein, and 0.2 g of fat, making it a perfect mid-meal snack.

Resources:

1. Kafle, Arjun, Mohapatra , *et. al.* A review on medicinal properties of Psidium guajava. *Journal of Medicinal Plants Studies* 2018; 6(4): 44-47
2. Sanzia & Uzzaman, Shakib. A short review on a Nutritional Fruit : Guava. Toxicology Research. 2018 1. 1-8.
3. Shivani Sharma and Dr. Anjan Borah, Bioactive compounds present in different parts of Guava and their significance: A review, *The Pharma Innovation Journal* 2021; 10(5): 163-171
4. Kumari S, Rakavi R, Mangaraj M. Effect of Guava in Blood Glucose and Lipid Profile in Healthy Human Subjects: A Randomized Controlled Study. *J Clin Diagn Res.* 2016;10(9):BC04-BC07. doi:10.7860/JCDR/2016/21291.8425
5. Souri, M.; Oktavia, S.; ifora, I. Potential Anti-Inflammatory Effects of Psidium Guajava L.: A Review. Asian Journal of Pharmaceutical Research and Development 2021, 9, 47-52

Recipe: Guava Granita

Serves: 2

Ingredients	Amount
Ripe Guavas (pink or white)	3 nos after removing the peel
Lemon juice	1 tbsp
Salt	A pinch
Water	½ cup

*1 cup: 250 mL; 1 tablespoon: 15 mL; 1 teaspoon: 5 mL

Method

1. To make the guava nectar cut the fruit in half and scoop out all the pulp.
2. Using a sieve and a large cooking spoon work the seed pulp with the back of the spoon against the sieve until you have all the juice extracted into a bowl.
3. Ensure that you remove all the small white seeds.
4. Combine the guava nectar, salt, lime juice, and water in a shallow tray or baking dish.
5. Cover with plastic wrap and place in the freezer.
6. Stir every 30 minutes, breaking up the larger frozen chunks.
7. Repeat this process until slushy, about 3 hours.
8. Scrape out the granita with a fork, scoop and serve chilled.

Jumbled Words

Find the health conditions associated with diabetes from the jumbled words below:

1. E C C I L A
2. O L R F A M W
3. P G S S A R T O I E R A S
4. U O T G
5. P T Y O I D O H R H Y
6. A Z E M I L S R E H
7. S B E I T O Y

1. Celiac
2. Wolfram
3. Gastroparesis
4. Gout
5. Hypothyroid
6. Alzheimers
7. Obesity

Patient Speaks

I am Manoj Patil, a 43-year-old man, living with diabetes for the last 7 years. Last year my company sponsored an annual body check-up, in which I got to know that I had hypercholesterolemia. My LDL levels were 200 mg/dL. My triglycerides were also high. I visited my doctor and he started me on medication for my deranged lipid profile. I was very surprised on seeing these readings as I did not have any symptoms. I got worried and decided to visit my DE.

My DE checked my reports and started asking me about my lifestyle, daily diet, and exercise routine. Being a marketing professional, I used to travel a lot. I would end up eating most meals outside and alcohol consumption was almost every weekend. My DE advised me to restrict alcohol intake for the time being to reduce my triglyceride level. She explained to me about moderate drinking and how I should stick to a certain safe amount of alcohol intake only once my levels were back in range. She also explained to me that food from outside is usually made in low-quality oil and reused oil which has trans-fat which is the reason for my high LDL levels. She explained to me how much oil to use in a month and even spoke to my cook and explained to her the same along with some healthy cooking methods. I was given ample healthy snack options by her which I could carry with me so that I did not depend on outside fried snacks when I am traveling. I was asked to increase the fiber in my meals. She advised me to include salad in all meals. I was advised to include high-soluble fiber foods like barley, and oats in my meals. I followed her advice diligently and also took my medications as prescribed by the doctor. I also started brisk walking as she recommended for 30 mins a day. After 3 months when I re-checked my lipid profile, it was much better and all my levels were near normal. What also excited me was that my blood sugar was also better controlled with these lifestyle changes that I made to bring my cholesterol level in control. Overall, I felt much better and energetic too. I thanked my DE and doctor and continue to follow their advice.

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Win Life

For screening people with High & Moderate Risk of Diabetes

Indian Diabetes Risk Score

Share this simple test with your family & Friends

Users can share it with their family & friends

Video on Suggestive Lifestyle Interventions to minimise risks - for each category

12 Languages

An awareness initiative by USV

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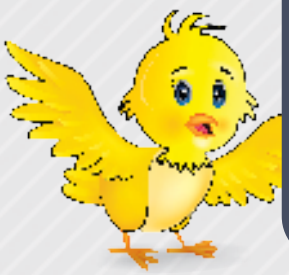

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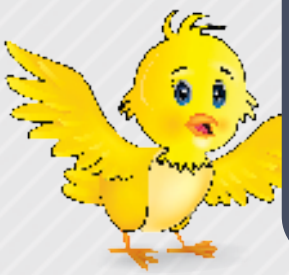

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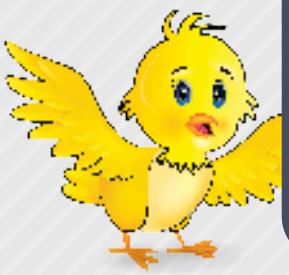

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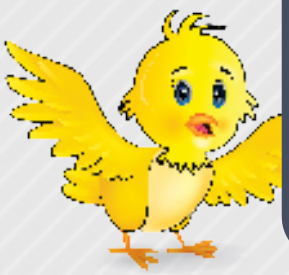

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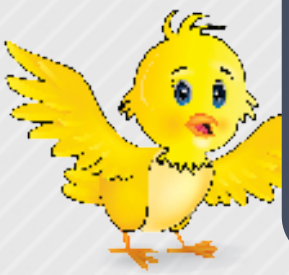

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In T2DM Across Continum,

Start with

Glycomet®-GP 1/2

Metformin Hydrochloride 500 mg SR + Glimepiride 1/2 mg

Source: 1. JAPI 2020 68,51.-55 2. Data on File, 3. Cureus 2020; 12(9): e10.7759/cureus.1070 4. Diabetes Technology & Therapeutics 2019,.2,79-84 5. Kalra, et al.: Sulfonylurea and combinations: International Task Force Indian J Endocr Metab 2018;22:132-57.

Priscribing information

Information: Metformin hydrochloride (as prolonged release) and glimepiride tablets. Glycomet-GP 0.5/Glycomet-GP 0.5 Forte/ Glycomet-GP 1/ Glycomet-GP 1/850/ Glycomet-GP 2/ Glycomet-GP 2/850/ Glycomet-GP 3/ Glycomet-GP 3/850/ Glycomet-GP 4/ Glycomet-GP 4/850/ Glycomet-GP 1 Forte/ Glycomet-GP 2 Forte/ Glycomet-GP 3 Forte/ Glycomet-GP 4 Forte Abridged Prescribing Information **Composition:** Glycomet GP 0.5mg: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500mg and glimepiride IP 0.5mg.• Glycomet GP 0.5 Forte: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 1000mg and glimepiride IP 0.5mg.• Glycomet GP 1: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 1 mg. • Glycomet GP 1/850: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 1 mg. • Glycomet GP 2: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 2 mg. • Glycomet GP 2/850: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 2 mg.• Glycomet GP 3: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 3 mg. • Glycomet GP 3/850: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 3 mg. • Glycomet GP 4: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 500 mg and glimepiride IP 4 mg. • Glycomet GP 4/850: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 850 mg and glimepiride IP 4 mg. • Glycomet GP 1 Forte: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 1000mg and glimepiride IP 1mg. • Glycomet GP 2 Forte: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 1000mg and glimepiride IP 2mg. • Glycomet GP 3 Forte: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 1000mg and glimepiride IP 3mg. • Glycomet GP 4 Forte: Each uncoated tablet contains metformin hydrochloride IP (as prolonged release form) 1000mg and glimepiride IP mg. **Indications:** Glycomet GP is indicated for the management of patients with type 2 diabetes mellitus (T2DM) when diet, exercise and single agent (metformin hydrochloride or glimepiride alone) do not result in adequate glycemic control. **Dosage and Administration:** Dosage of Glycomet GP should be individualized on the basis of effectiveness and tolerability while not exceeding the maximum recommended daily dose of glimepiride 8mg and metformin 2000 mg. **Initial dose:** 1 tablet of Glycomet GP should be administered once daily during breakfast or with the first main meal. Do not crush or chew the tablet. In several cases the tablet may remain intact during transit through the gastrointestinal (GI) tract and will be eliminated in feces as hydrated mass (ghost matrix). Patients should be advised that this is normal as all drug components have already been released during GI transit. **Contraindications:** In patients hypersensitive to glimepiride, other sulfonylureas, other sulfonamides, metformin or any of the excipients of Glycomet GP; pregnancy and lactation; diabetic ketoacidosis, diabetic pre-coma, in patients with eGFR<30 ml/min/ 1.73 m2, acute conditions with the potential to alter renal function (dehydration, severe infection, shock, intravascular administration of iodinated contrast agents), acute or chronic disease which may cause tissue hypoxia (myocardial infarction, shock, cardiac/respiratory failure) hepatic insufficiency, acute alcohol intoxication, alcoholism. **Warnings:** Keep out of reach of children. Patient should be advised to report promptly exceptional stress situations (e.g. trauma, surgery, febrile infections). Blood glucose regulation may deteriorate and a temporary change to insulin may be necessary to maintain good metabolic control. In case of lactic acidosis, patient should be hospitalized immediately. **Precautions:** In the initial weeks of treatment, the risk of hypoglycemia may be increased and necessitates especially careful monitoring. Serum creatinine levels should be determined before initiating treatment and regularly thereafter: at least annually in patients with normal renal function. Intravascular contrast studies with iodinated materials can lead to acute alteration of renal function. In patients in whom such study is planned, Glycomet GP should be temporarily discontinued at the time of or prior to the procedure, and withheld for 48 hours subsequent to the procedure and reinstituted only after renal function has been re-evaluated and found to be normal. Use of Glycomet GP should be discontinued 48 hours before any surgical procedure. **Adverse reactions:** For glimepiride - hypoglycaemia; temporary visual impairment; GI symptoms like nausea, vomiting, abdominal pain, diarrhoea may occur; increased liver enzymes, cholestasis and jaundice may occur; allergic reactions may occur occasionally. For metformin – GI symptoms like nausea, vomiting, abdominal pain or discomfort may occur.

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