RSSDI NEWS

1998 • 7 • September

DIABETES RESEARCH TRUST, RSSDI

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DIABETES RESEARCH FORUM
by **Prof. M.M.S. Ahuja,** 1929-1998

On the 25th anniversary of the Society, Prof. M.M.S. Ahuja has decided to take steps for formation of Diabetes Research Forum with a seed money donation of Rs.One lakh

These research objectives by Prof. M.M.S. Ahuja will be met through a Research Committee to be set up by Dr. C.S. Yagnik, Pune in association with Dr. Ajay Sood, New Delhi; Dr. Eesh Bhatia, Lucknow; Prof. K.M. Prasannakumar, Bangalore; and Prof. R.V. Jayakumar, Kottayam.

RSSDI Executive and Secretariat will raise a corpus fund of Rs.10 lakhs to be named after Prof. M.M.S. Ahuja as a research trust fund of the RSSDI for giving research grants of Rs.10,000 each under the supervision of the Research Committee of RSSDI. It is proposed that the corpus funds of the Research Trust will be managed by Prof. K. Kannan, Madurai for RSSDI.

Research Committee will invite applications and meet annually to award the grants. Criteria for award will include scientific merit, design of study and competence of investigator.

26th Annual Scientific Meeting of the Research Society for the Study of Diabetes in India
on December 18, 19 and 20 at Ahmedabad

RSSDI annual scientific meeting in 1998 will start with a Commemoration Lecture as a homage to Prof. M.M.S. Ahuja

RSSDI theme symposium will be named as AHUJA SYMPOSIUM

Submit abstracts before September 30 to
Prof. H.B. Chandalia, Chairman,Scientific Committee
18 Kala Bhavan, 3 Mathew Road, Mumbai 400 004
tel 022 363 3695, 363 4320, 287 1613 fax 022 493 8322

Registration is Rs.1000, Rs. 750 for life members and Rs.500 for accompanying persons by draft favoring RSSDI 98 to be sent before September 30 to the Organizing Secretary, Dr. Mayur R. Patel, Yash Diabetes Clinic, 304 Supath, Nr Vijay Char Rasta, Navrangpura, Ahmedabad 380 009, Gujarat,
tel 079 755 7888, 46 0607, 40 1030 fax 589 3235
e-mail : mayur-patel-98@yahoo.com

INTERNATIONAL JOURNAL OF DIABETES IN DEVELOPING COUNTRIES

We request medical teachers, researchers and scientists to send us a few articles a year for publication. These could be in the form of original articles, comprehensive reviews or case reports. If your busy schedule does not permit you to submit materials to us we would appreciate it if you could encourage other junior doctors to work under your guidance and to submit their work for publication.

RSSDI LIFE MEMBERSHIP

Life Membership payment is Rs.1500, and Corporate Membership payment is Rs.100,000 by draft favoring RSSDI to be sent to the Secretariat.

APPEAL

For a major project at St. John's Medical College, Bangalore in association with Center for Disease Control, Georgia, USA and World Bank, all Indian diabetes research work unpublished or published in unindexed Journals is solicited for storing in a data bank.

Dr. PREM PAIS
Professor of Medicine
St. John's Medical College
Bangalore 560 034
Tel 553 0724
Fax 552 0777, 552 1485, 553 1786
Email ppais@stjohn.dabang.ernet.in

ELECTIONS FOR RSSDI EXECUTIVE COMMITTEE, 1999

Nominations received for Office Bearers:

President

Prof. B.K. Sahay
Hyderabad

Vice Presidents

Prof. B.N. Srivastava
Jabalpur

Dr. Sidhartha Das
Cuttack

Honorary Secretary

Prof. P.V. Rao
Hyderabad

Honorary Joint Secretary

Dr. G.R. Sridhar
Visakhapatnam

Executive Committee Members

Prof. V. Seshiah
Chennai

Prof. K.C. Samal
Cuttack

Prof. A.K. Das
Pondicherry

Prof. Muralidhar S. Rao
Gulbarga

Dr. C. Munichoodappa
Bangalore

Dr. Sharad Pendsey
Nagpur

rssdi 98 themes

QUALITY OF DIABETES CARE AND ITS EVALUATION

This topic is selected as the main theme of our Annual Scientific Meeting to be held in Ahmedabad this year. This will give us an opportunity to

1. Assess the quality of diabetes care being provided at different levels (primary care physician, tertiary care centre, private clinics, medical colleges etc.).

   Some of the questions we need to address are :

   a. What is the quality of care being provided at different levels of health care?
   b. If inadequate, what remedial measures should we consider?
   c. What is the cost of diabetes care? What other inputs are required? Can we prioritise amongst different aspects of diabetes care which are more important than others?
   d. Whom should we entrust different aspects of diabetes care?

2. Define and elaborate upon the methods we should use to evaluate the quality of diabetes care.

   Some of the questions we need to address are :

   a. What is good diabetes care? What are its components?
   b. What is the relative importance of each component? Can we design a screening system to quantitate the quality of diabetes care?

DOES FAST FOOD KILL FAST?

Fast Food : It is difficult to define fast food by convention, it is the food that can be prepared and served rapidly to suit modern fast pace of life.

The typical examples of American fast foods are : French fries, Hamburger, Pizza, Ice-cream, Hot Dog etc. The few typical Indian fast foods are : Pav Bhaji, Bhel Puri, Idli, Dosai, Pakoras etc. The fast foods may be nutritious but are likely to suffer from following drawbacks :

1. They are usually calorie packed or calorically dense.
2. They usually are high in carbohydrate and fat but somewhat low in protein.
3. They usually have poor contents of vegetable or fruits and hence are deficient in micronutrients which are present in vegetables and fruits. They may have poor content of antioxidants, vitamins, fiber and other micronutrients.

Diabetes and Cardiovascular Complications

at Agartala, Tripura
on September 11 and 12, 1998
approved for 10 RSSDI credit hours
register by sending draft for Rs.200 favoring **All Tripura Diabetic Forum** to
Dr. P.K. Bhattacharya
Sarat Sarani, Durga Chowmuhani
Agartala 799 001 TR
tel 0381 225444 224555 228159
fax 225001 223201

COMPLICATIONS OF DIABETES MELLITUS AND MANAGEMENT

at Jaipur on September 27, 1998
approved for 5 RSSDI credit hours
Registration enquiries to
Dr. Arvind Gupta
D-88 Krishna Marg
Siwar Area, Bapu Nagar
Jaipur 302 015 RJ
tel 0141 517459

CME in DIABETES

at Khajuraho
on October 3 and 4, 1998
by **Prof. B.N. Srivastava**
approved for 10 RSSDI credit hours
Register by sending Rs.750 by draft favoring **RSSDI-CME (MP) KHAJURAHO** payable at **JABALPUR** to
Dr. Parimal Swamy
601/6 West Nillar Ganj
Jabalpur 482 002 MP
tel 0761 314832 25374 23688
fax 424973

9th RSSDI Course in Diabetology

by Dr. Pradeep Y.R. Mulay at Government Medical College, Aurangabad
on October 9, 10 and 11, 1998
approved for 20 RSSDI credit hours
register by sending draft for Rs.1000 favoring **Diabetes Forum** to
Dr. Sanjeev A. Indurkar
Behind MSFC - Station Rd
Rachanakar Colony
Aurangabad 431 005 MS
tel 0240 333124 335030 332772 320310

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* RSSDI theme symposium will be named as AHUJA SYMPOSIUM.

CME in Diabetes at Khajuraho

Register by sending Rs.750 by draft favoring RSSDI-CME (MP) KHAJURAHO
payable at JABALPUR.

9th RSSDI Course in Diabetology by Dr. PRADEEP Y.R. MULAY
at Government Medical College, Aurangabad
on September 25, 26 and 27, 1998
register by sending Rs. 1000 by draft favoring DIABETES FORUM to
Dr. SANJEEV A. INDURKAR, BEHIND MSFC - STATION Rd,
RACHANAKAR COLONY, AURANGABAD 431 005 MS
tel 0240 333124 335030 332772 320310

DIABETES and CARDIOVASCULAR COMPLICATIONS
September 11 and 12, Agartala,
Register by sending a draft for
Rs. 200 in favor of All Tripura Diabetic Forum to
Dr. P.K. BHATTACHARYA, DIABETIC CARE CLINIC, SARAT SARANI
DURGA CHOWMUHANI, AGARTALA 799 001, TR
tel 0381 225444 228159 224555 fax 225001 223201

COMPLICATIONS OF DIABETES MELLITUS AND MANAGEMENT
on September 26 and 27 at Jaipur
Registration enquiries to
Dr. ARVIND GUPTA, D-88 KRISHNA MARG
SIWAR AREA, BAPU NAGAR, JAIPUR 302 015 RJ, tel 0141 517459

COMPLICATIONS OF DIABETES

Introduction

Good glucose control prevents or minimizes serious diabetes complications (Figure 1). Treatment goal should encompass euglycemia, normalize blood lipids, regulate blood pressure, and individualize diet and-exercise plan. Life style goals that help minimize complications include giving up smoking and alcohol, low saturated fat, low sugar, high fibre diet, and four or five daily servings of fruit and vegetables.

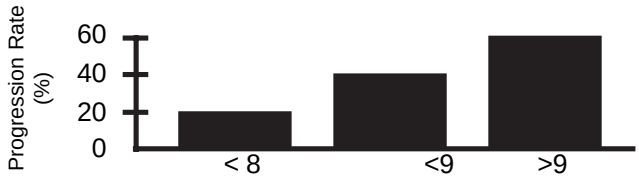

Figure 1: Rate of progression of Diabetic Retinopathy as a Function of HbA1C.

Diabetic Ketoacidosis (DKA)

When insulin is inadequate, or insulin injection is suddenly stopped, glucose is not readily available for energy. The body then uses fat for energy. Fat breakdown produces ketone bodies leading to ketonemia and ketonuria. Eventually, patient may develop DKA with the potential risk of coma and death (Figure 2).

INSULIN DEFICIENCY

Glucose Uptake by Tissues Hepatic Glucose Production Lipolysis

Hyperglycfmia Gluconeogenesis Ketogenesis

Ketonuria Ketonemia

Osmotic Diuresis ELECTROLYTE DEPLETION

Hypotonic Losses DEHYDRATION

ACIDOSIS

Figure 2: Pathophysiology of Diabetic Ketoacidosis

Generally, an acute illness, like infection, induces insulin resistance and causes DKA (Figure 2), notably in type 1 diabetes. It is an avoidable condition. The clinical features relate to the biochemical profile (Table 1).

Table 1: DKA Clinical Features

Profile	Symptoms & Signs	Profile	Symptoms & Signs
Hyperglycemia	Polyuria, Blurred Vision	Electrolyte Depletion	Weakness, Headache, Myalgia,
Dehydration	Polydipsia, Intravascular Volume Depletion Supine and Orthostatic Hypotension		Hypotonia, Stupor Uncoordinated Eye Movements Fixed Dilated Pupils, Coma
Ketosis	Acetone Breath Anorexia, Nausea, Vomiting	Acidosis	Nausea, Vomiting, Abdominal Pain Hyperventilation

Monitored fluid, electrolyte, and insulin infusion are best carried out in a hospital. Glucose level must decline 75 to 100 mg/dL per hour, ketosis in 12 to 24 hours. Acidosis and dehydration persist longer.

Hyperosmolar Non Ketotic Syndrome (HNKS)

Hyperglycemia in type 2 diabetes may cause hyperosmolar non-ketotic syndrome (Table 2), particularly in presence of a severe illness. Diazoxide, phenytoin, calcium channel blockers, chlorpromazine, cimetidine, and glucocorticoids may precipitate HNKS. Fluid and insulin infusion over several days in a hospital are recommended.

Table 2: Clinical Features of HNKS

Profile	Symptoms & Signs	Profile	Symptoms & Signs
Hyperglycemia	Blood Glucose of 500 to 1000 mg/dL. Polyuria, Blurred Vision	Electrolyte Depletion	Weakness, Headache,Myalgia Hypotonia Stupor
Dehydration	Polydipsia Intravascular Volume Depletion Supine and Orthostatic Hypotension		Uncoordinated Eye Movements Fixed Dilated Pupils, Coma

Hypoglycemia

Factors that significantly increase circulating insulin or intensify glucose utilization result in hypoglycemia (Table 3), defined as a blood glucose value (60 mg/dL ≤ 3.3 mmol/L). Missed meal, dieting, intake of extra antidiabetic dose, or alcohol, or strenuous exercise may also precipitate hypoglycemia.

Table 3: Signs and Symptoms of Hypoglycemia

Due to Autonomic Disturbances caused by Epinephrine Release	Due to Neuroglucopenic Disorder caused by Low Glucose Level in Brain
Anxiety, Nervousness, Irritability Tremor and Weakness Hunger, Nausea and Vomiting Sweating, Tingling of the mouth or fingers Palpitations and Tachycardia	Confusion, Mental dullness, Amnesia Feeling cold, Hypothermia Vision disturbances Seizure, Coma Headache

Pregnancy decreases urinary glucose threshold. Thus, urine test gives false positive result for sugar. Hence, increasing insulin based on urine test during pregnancy can cause hypoglycemia.

The reduced insulin demand in nephropathy needs a

downward dose titration. If mental changes are seen following hypoglycemia, treatment in a hospital over several days may be necessary. Diabetics should always carry with them some sugar to be consumed at the first unusual symptom.

Table 4: Step-Care Approach in the Management of Hypoglycemia

Step	Conscious Patient	Unconscious Patient
Step 1: Immediately	Oral 10g glucose, 4 teaspoon sugar, jaggery, or honey, or 1 cup orange juice. Only glucose or milk for patient on acarbose.	1 mg glucagon to induce liver to produce glucose.
Step 2: After 10 minutes	Repeat the above	i.v. Glucose 25 to 50%. Turn patient's head to one side to avoid choking on vomitus.
Step 3: As symptoms resolve, or consciousness returns	Balanced snack or meal to prevent hypoglycemia recurrence.	

Sick Day Rules

An acute illness induces insulin resistance and consequent hyperglycemia. Since they feel unwell, patients decrease their food intake. Correspondingly they tend to lower the dose of their medication and enhance their risk for further deterioration in glucose control.

Sick Day Rules for Type 1 Diabetes

1. Patients must continue their usual insulin dose, even if food intake is decreased. Some may require additional insulin based on results of urine and blood glucose monitoring (Table 5).

Table 5: Computing Additional Insulin Dose

Urine Test		Blood Glucose	Additional Regular Insulin Dose (U)	
Glucose (%)	Ketone Bodies	(mg/dL)	Non-obese	Obese
1 or 2	Negative, trace, small	> 300	4	8
up to 2	Moderate, large	> 300	8	12

2. Check blood and urine sugar, at least four times a day, namely, before each meal and at bed time. Also, check urine ketone levels, respiratory and gastrointestinal signs and symptoms.
3. Added fluid intake and enough calories from a mix of protein, fat, carbohydrate, salt, and minerals are important. Fluid balance can become critically negative in presence of gastrointestinal symptoms.
4. Monitor changes in symptoms, urine ketone levels, blood sugar values, onset of fruity breath, or diarrhea, nausea and vomiting, or breathing difficulty.

Sick Day Rules for Type 2 Diabetes

1. Patients must continue their usual dose. The sulfonylurea dose can be increased, if warranted by symptoms and blood glucose level. Reduced food intake may offset the worsening of control.
2. Patients on additional insulin may be able to lower insulin dose if food intake is markedly reduced.
3. Monitor blood glucose before each meal and at bed time. Self-monitoring is recommended.
4. Continue normal diet, or take several small meals, and drink extra fluid, one glass every hour.
5. Dehydrated patients and those with mental confusion are best treated in the hospital.

Macrovascular Complications

Type 2 diabetics are more prone to macrovascular complications. Nearly 25% of stroke victims are diabetic. The combination of diabetes, hypertension and/or coronary artery disease (CAD) creates a high-risk profile for stroke. It is possible to eliminate smoking and obesity and significantly regulate other risks like hyperlipidaemia, hypertension, atherosclerosis and hyperglycemia. Incidence of heart disease increases 30% for each 40 mg/dL rise in total blood cholesterol. One should achieve target levels for LDL cholesterol and triglyceride (Table 6), initially with diet (Table 7) and exercise. Regular exercise decreases LDL oxidation and the risk of fatty build-up on arterial walls. Patients should also receive antioxidants to overcome oxidative stress.

Table 6: Target Levels for LDL Cholesterol & Triglycerides

CAD	Number of Risks	LDL Cholesterol	Triglyceride
Present	Irrespective of number of risks	100 mg/dL	200 mg/dL
Absent	One or more	130 mg/dL	400 mg/dL
Absent	None	160 mg/dL	400 mg/dL

Table 7: Diet Principles

Constituent	Daily Level	Constituent	Daily Level
Total Fat	< 30 % of calories	Carbohydrate	55 to 60 % of calories
Saturated Fat	< 10 % of calories	Fibre	25 g/1000 calorie diet
Polyunsaturated Fatty Acid (PUFA)	< 8 % of calories	Protein	10 to 20 % of calories
Monounsaturated Fatty Acid (MUFA)	10 to 15 % of calories	Dietary Cholesterol	< 300 mg

Saturated fat has two times greater cholesterol elevating potential than unsaturated fats. Figure 3 depicts the fat profile of some common dietary fat sources. The body is unable to synthesize two essential fatty acids, linoleic (omega-6 = (ω6) and alpha-linolenic (ω3). Fish, shell fish, common beans and soybeans contain ? -3, which benefits cases with hypertriglyceridemia. The average Indian diet is ω -3 deficient. So, one can use a preparation containing ? -3 marine fatty acids.

When triglyceride level exceeds 400 mg/dL, after a month of diet and exercise, add gemfibrozil 600 mg twice daily. When triglyceride is less than 400 mg/dL and LDL cholesterol is within target, gemfibrozil alone may be used. However, when LDL is higher than target, gemfibrozil is replaced with a statin (HMG-CoA-reductase inhibitors, like lovastatin or pravastatin or simvastatin). After another month, if both triglyceride and LDL values are within target levels, the statin is continued. However, if the triglyceride increases, add gemfibrozil to the statin dose.

Figure 3: Fatty Acid Composition of Common Dietary Fat Sources

Obesity

Diabetics receiving intensive treatment with insulin can become obese, increasing their risk of developing heart disease. Thus, diet and exercise aspects of diabetes management assume great significance. Monitor obesity by calculating the waist-to-hip ratio. A healthy ratio for females is ≤ 0.8, and for males ≤ 1.0) Higher values increase the risk of complications. To find the ratio, divide the waist circumference by the hip circumference.

Hypertension

Generally, goal blood pressure suggested for hypertensive diabetic is 160/90 mm Hg, or less. In presence of microalbuminuria or clinical proteinuria, the target blood pressure should be 130/85 mm Hg, or less. Recent studies showed that there is 51% reduction in major cardiovascular events in diabetics achieving diastolic blood pressure of 80 mm Hg, vis-a-vis 90 mm Hg. Aspirin reduced major cardiovascular events by 15% and all myocardial infarctions by 36%.

Angiotensin converting enzyme (ACE) inhibitors lower blood pressure and total peripheral vascular resistance. They improve the body's insulin sensitivity, halt or reverse microalbuminuria and clinical proteinuria, and improve renal function. Alternate antihypertensive drugs include diuretics, β-adrenoceptor blockers, or calcium channel blockers.

Peripheral Vascular Disease (PVD)

Chronic hyperglycemia induces structural protein changes and jeopardizes the flow properties of blood. The platelet and coagulation systems also behave differently in diabetics This further disturbs hemorheology.

PVD produces intermittent claudication, nocturnal or rest pain. Eventually, patchy or extensive gangrene of the foot and toes occur. It is responsible for more than 50% of all non-traumatic lower limb amputations. Anecdotal reports suggest that giving thyroid improves glucose control, and may prevent or delay PVD.

Table 8: Ambulatory Management of Peripheral Vascular Disease

Near-normal Glycemia	Achieve Target Blood Pressure	Achieve Target Lipid Levels
Antibiotics for Infection	Improve Haemorheology, (Aspirin, Dipyridamole, Pentoxifylline)	
Personal Hygiene	Aggressive Foot Care	Refer to Vascular Surgeon

Microvascular Complications

Sustained hyperglycamia enhances the risk for microvascular complications, especially in type 1 diabetics. Reducing HbA1c to 6.5 to 7.0% halts the progression and development of microvascular complications (Figure 1). ACE inhibitors decrease proteinuria and delay the progression of renal disease.

Diabetic Retinopathy

Retinopathy changes may occur in almost all diabetics after more than 20 years. It is possible to prevent blindness and retard the rate of progression (Table 9). Cataract formation tends to occur at a younger age. Eye care in a diabetic reflects a partnership between the primary care physician and the ophthalmologist.

Table 9: Factors that Arrest Progression of Retinopathy

HbA1C level ≤ 6.5%	Fasting blood glucose ≤ 110 mg/dL 2-hour post-prandial blood glucose ≤ 180 mg/dL
Control blood pressure, best with ACE inhibitor	ACE inhibitor therapy delays progression of retinopathy

Diabetic Nephropathy

Diabetic nephropathy plus hypertension is a frequent cause of chronic renal failure and end-stage renal disease (ESRD). Microalbuminuria and proteinuria carry a high

cardiovascular mortality risk in diabetics. Family physicians treat diabetes and hypertension, which are the two leading causes of ESRD. Thus, they can help (Table 10) reduce the incidence of this complication. Initially, the patient develops microalbuminuria, defined as urinary albumin excretion rate of 20 to 200 μg/min or 30 to 300 mg in 24 h. The simplest test for microalbuminuria is to determine the albumin-to-creatinine ratio. A ratio of 30 μg/mg (3.5 μg/ μmol) or higher is diagnostic.

Table 10: The Four Fundamentals in the Treatment of Microalbuminuria

Control Pressure	: Aggressive antihypertensive therapy is necessary. Studies have shown that lowering the blood pressure effectively reduces albumin excretion rate.
Control Hyperglycemia	: Long term control of hyperglycemia prevents reno-vascular damage. To halt or reverse progression of nephropathy euglycemia goals are: HbA1C level down to at least 7%; fasting plasma glucose < 110 mg/dL; 2h post prandial blood glucose < 180 mg/dL.
ACE Inhibition	: ACE inhibitors regulate BP, reduce intra-glomerular pressure, improve GFR and renal function, and prevent progression to ESRD in normotensive and hypertensive diabetics.
Low Protein Diet	: High protein diet increases intra-glomerular pressure. Therefore, protein restriction may help. It is not safe to reduce dietary protein drastically, particularly in children and adolescents. Moderate decrease in those with clinical proteinuria is advisable. Substituting vegetable protein for animal protein by itself decreases albuminuria.

There is strong evidence that ACE inhibitors are preferred first line agents in diabetics with hypertension and/or renal insufficiency. However, the debate continues on the value of prophylactic ACE inhibitors in normotensive diabetics without evidence of renal dysfunction. Some suggest that ACE inhibitor therapy should begin when hypertension or proteinuria begins. Others assert that ACE inhibitor therapy should begin early because incipient microalbuminuria may be missed. Besides, it is the phase of impaired glucose tolerance that sets the stage for subsequent hyperinsulinemia and macrovascular and microvascular disease.

Neurological Complications

Prolonged hyperglycemia causes ischaemic changes in nerves. These impair transmission of nerve signals. A decade after diagnosis, close to 30% of diabetics develop neuropathy. Progressively, the incidence rises to nearly 50%. It is estimated that about 20% of type 2 diabetic patients have peripheral neuropathy at the time of diagnosis of their diabetes, reflecting the preceding many years of asymptomatic hyperglycemia.

Diabetic Peripheral Neuropathy

It is a sensorimotor polyneuropathy affecting legs earlier than the hands. The initial sensory symptoms are tingling, burning, pricking, or cramps, often worse at night. There is early loss of tendon reflexes, reduced sense of vibration and touch. Gradually, as numbness begins, the patient may not even be aware of injury. Wounds become infected and due to prolonged hyperglycemia and poor circulation. Often, these wounds are difficult to treat.

Diabetes Foot and Foot Care

Nearly three quarters of all amputations caused by neuropathy can be prevented with regular and careful foot care management and near euglycemic control. Regular examination of the feet, personal hygiene, early and adequate treatment for injuries, use of well-fitting footwear, avoiding use of toe-grip sandals, regular exercise, cutting back alcohol consumption, and stop-smoking efforts is needed.

Diabetic Autonomic Neuropathy

Autonomic neuropathy appears long after peripheral neuropathy is established. The resultant delayed stomach emptying can pose a serious threat for insulin requiring patients. This because, patients on insulin need a predictable response to meals to cover the action of the injected insulin. Blunting of normal hypoglycemia response poses a serious threat to diabetics.

Table 11: Signs & Symptoms of Autonomic Neuropathy

Difficult bladder control, Anorexia, nausea and vomiting Frequent diarrhea	Sexual dysfunction Abdominal bloating Swallowing difficulty	Delayed stomach emptying Constipation Postural hypotension

Treatment in Diabetic Neuropathy

Tight glycemic control and good tissue perfusion are essentials of medical management. Management of neuropathy pain can be very frustrating. Analgesics should be tried initially. It is best not to use non-steroidal anti-inflammatory drugs because of their potential to reduce renal function. Narcotic analgesics are not very effective. Many patients may find the pain less intolerable with analgesics combined with an adjuvant medication (Table 12).

Table 12: Adjuvant Medication for Pain Relief in Diabetic Neuropathy

Drug	Dose	Drug	Dose
Imipramine	Up to 150 mg bed time	Amitriptyline	Up to 150 mg bed time

Carbamazepine	400 mg to 1200 mg/day	Mexiletine	75 mg to 200 mg tid
Clonidine	75 mg to 100 mg at bed time	Baclofen	5 mg to 15 mg/day

Quinine sulphate, 200 to 300 mg at bed time helps in night cramps. Patients with delayed gastric emptying should receive their insulin just before or immediately after the meal. Additionally, 15 to 60 minutes before each meal and at bed time, they may receive, metoclopramide 5 mg to 20 mg, or cisapride 10 mg. Cisapride should be used with caution in susceptible patients, because of its potential for cardiac arrhythmias. Erythromycin 125 mg to 500 mg three to four times daily, stimulates gastrointestinal motor activity and speeds up gastric emptying. Loperamide, diphenoxylate and empirical antibiotics help control diabetic diarrhoea . Bulk or osmotic laxatives are useful in constipation. For erectile dysfunction, referral to a specialist for evaluation and therapy is suggested.

Infection

Diabetics are prone to infection, bacterial, fungal, or viral. Organ systems often involved include the skin, urogenital system, lower respiratory tract, septicemia. They have five times the risk of wound infection than non-diabetics. The risk is even higher in obese diabetics. Presence of infection, particularly a severe infection, alters insulin sensitivity and further aggravates hyperglycemia. Thus, the onset of infection sets in motion a vicious cycle.

Endocrine Problems

Women with type 1 diabetes may experience premature menopause, by an average of about nine years. This places them at high risk for cardiovascular disease and early mortality. Type 1 diabetic women also have a higher incidence of non-carcinogenic breast lump (mastopathy). Furthermore, an interaction between oestrogen and insulin may encourage growth and proliferation of breast cells, a finding that may explain why diabetes is more common in women with breast cancer.

Iatrogenic Complications

Drug usage statistics show that diabetic patients receive more prescriptions, particularly of antibiotics, central nervous system drugs, cardiovascular drugs, gastrointestinal agents, analgesics, and anti-inflammatory agents. Most of these drugs are used two to three times more often by diabetics. Thus, they have an increased possibility of adverse drug reactions that may require additional drug treatment.