Keynote Forum
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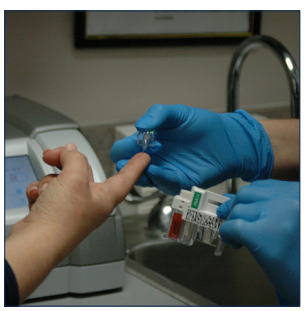
Diabetes Congress 2019







27th International Conference on

Diabetes and Endocrinology

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Udaya M Kabadi[1,2]

[1]Broadlawns Medical Center, USA

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Diabetes Mellitus: Disorder of cellular dysfunction due to lack of entry into cell of glucose; the most efficient fuel for cellular function

Traditionally, diabetes Mellitus has been deemed to be a chronic hyperglycemic disorder secondary to altered glucose metabolism. Alternatively, hyperglycemia may be one of several manifestations in subjects with type 1 and type 2 diabetes Mellitus. Almost all tissues require insulin for entry of glucose, the possible exceptions being red blood cells, renal medulla as well as central and peripheral nervous systems. Hyperglycemia in intravascular compartment and other extra cellular milieu may be attributed to impaired glucose entry into endothelial cells of the vessel wall and almost all other cells including hepatocytes, myocytes of all varieties, adipocytes and individual cells in most other organs respectively due to absence of insulin in type 1 and both the insulin resistance as well as the decline in both phases of insulin secretion in type 2 diabetes. Albeit, the decline in both phases of insulin secretion are induced by lack of glucose entry into pancreatic beta cells. Finally, hyperglycemia is perpetuated by increased hepatic glucose production caused by into sustained circulating hyperglucagonemia secondary to lack of glucose entry into the pancreatic alpha cells. Alternatively, both the decline in insulin secretion by the beta cells and the rise in glucagon release by the alpha cells are enhanced by fall in GLP1 and GIP caused by dysfunction of L cells and K cells respectively secondary to lack of glucose entry in both type 1 and type 2 diabetes. Similarly, increased prevalence of infections and thromboembolic micro and macrovascular events may be attributed to dysfunction of leukocytes and platelets respectively due to impaired glucose entry. Finally, alterations in several other metabolemics including serum concentrations of Adiponectin (Adipose cells), TNF alpha, Plasminogen inhibitor factor 1, Homocysteine, CRP, Lipids etc. (Hepatocytes) as well as dysfunction of several organs (liver, heart, kidney, adrenal, pituitary, lungs etc.) in both type 1 and type 2 diabetes may also be attributed to the lack of glucose entry into these specific cells. This hypothesis is validated by improvement in metabolemics and organ function on facilitation of glucose entry into cells by insulin administration and/or improvement in insulin sensitivity. Therefore, in conclusion, diabetes mellitus is a disorder manifesting dysfunction involving almost all organs and cells induced by lack of entry of glucose, the most efficient substrate for cellular function.

Speaker Biography

Udaya M Kabadi is a graduate of Seth G.S. Medical College, the University of Bombay in Bombay, India. He completed his internal medicine residency at KEM Hospital Parel in Bombay and a medicine residency at Jewish Memorial Hospital and Beth Israel Medical Center in New York, He also completed a fellowship in endocrinology and metabolism at VA Medical Center and Beth Israel Medical Center in New York. He is board certified in internal medicine, endocrinology and metabolism and geriatric medicine by the American Board of Internal Medicine. He is a fellow of the Royal College of Physicians of Canada, the American College of Physicians and the American College of Endocrinology. He has been a chief editor, associate editor and member of editorial boards of several medical journals. He is currently an adjunct professor of Medicine at the University of Iowa College of Medicine, Iowa City as well as Des Moines University, Des Moines, Iowa. He has over 200 publications in peer-reviewed journals. He has presentations to his credit, at regional, national, and international arenas. He has been selected as 'Teacher of the Year' many times by students, residents, and fellows in training. He has been involved in research in the area of carbohydrate metabolism and diabetes, thyroid disorders and osteoporosis as well as in clinical practice and education for several years.

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Rekha Annie Prasad

Sir Charles Gardiner Hospital, Australia

Epidemiology, pathophysiology, investigations, treatment for exocrine dysfunction of diabetes

The exocrine dysfunctions in diabetes is an unrecognised problem in the community. There are no adequate data available to quote the exact number of cases. It is estimated to be in 50% of diabetic patients in retrospective historical observational studies. The exocrine pancreas secretes enzymes that digest carbohydrates, proteins, fats and bicarbonates for neutralisation of acidic chyme from the stomach. Exocrine pancreas consists of 80% to 85 % of the pancreas rest of the gland constitutes the endocrine portion. Functional unit of the exocrine pancreas: Acinus. Pancreatic exocrine insufficiency is the condition where there is inadequate secretion of enzymes in response to the food to maintain normal digestion. The main reasons areas follow, inadequate secretions of pancreatic enzymes, reduced production, reduced secretion, insufficient stimulation, inadequate acid mediated inactivation and obstruction to pancreatic duct. The main clinical consequence of the pancreatic exocrine insufficiency is fat malabsorption and digestion leading to steatorrhea's studies have shown that in almost 50 percent of diabetics have pancreatic dysfunction. In type 1 diabetes up to 51 % and in type 2 diabetes it is up to 32%. There are few theories of pathophysiology of the exocrine dysfunction in types 1 and 2 which are as follow pancreatic islet cells hormones regulatory functions may be impairment, diabetic neuropathy, diabetic angiopathy causing impaired blood flow leading to fibrosis and atrophy, elevated hormones and peptide concentration example somatostatin and glucagon may suppress exocrine function, concurrent damage done by viral infections, genetics autoimmune changes. The reclassification study talks about type 1 presence of autoantibodies early onset, early requirement, type 2 absence of autoantibodies, no (late) insulin requirement, insulin resistance, type 3 absence of autoantibodies, exocrine pancreatic insufficiency, typical morphological findings. The investigations for the diagnosis Pancreatic exocrine dysfunction are done by faecal elastase concentration (most commonly available) with the formed stool, coefficient of fat absorption (gold standard), carbon 13 (13C) mixed triglyceride breath test (not widely available). The main clinical consequences of Pancreatic exocrine dysfunction are steatorrhea, and this is evident when almost 90% of pancreatic function is lost however experts suggests that early recognition, screening of pancreatic exocrine dysfunction is valuable as their thoughts are it could happen at earlier stage of the disease. Clinical signs are abdominal pain, flatulence, weight loss and steatorrhea? Glycaemic control (yet to be explored). The treatment with Pancreatin improves HbA1C, improvement in clinical sign, improves in increatin effects, fewer hypos. Do we routinely ask for Gastrointestinal side effects in our diabetic patients, most often no and is put down to medication (metformin) and autonomic dysfunction rather that Pancreatic exocrine dysfunction? I think it is time to rethink the way we look at diabetes and pancreatic insufficiency. Early recognition can treat the pancreatic exocrine dyfuncyion and in fact improve HbA1C and reduce the risk of the complications associated with diabetes.

Speaker Biography

Rekha Annie Prasad is a consultant physician at a tertiary hospital and works at other sites. She has been a clinician in Australia for the past 18 years with varied experience, remote and in urban areas. She has her speciality interests as acute medicine, pre and peri operative medicine, obstetric medicine. She is passionate about diabetes management and chronic disease management especially in aboriginal population. She is involved in teaching under graduate students for Notre Dame University and University of Western Australia. She is also a mentor for post graduate students taking their fellowship exams. She is on the safety and medication committee of Sir Charles Gardiner Hospital. She is also a panel member of undiagnosed disease panel in Western Australia. She also practices tele-medicine for remote communities dealing with chronic diseases especially diabetes.

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Uthara Upadhrashta

SRM Institutes for Medical Science, India

Handling the prediabetes burden: The way forward

India's fastest growing disease, diabetes, was recorded in 72 million cases in the year 2017, making India account for 49% of the world's diabetes burden. Diabetes prevalence has increased by 64% across India in the last 25 years, reports the Indian Council of Medical Research ICMR), November 2017. The prevalence of Prediabetes or Impaired Glucose Tolerance (IGT) is 1.4 times higher than the diabetes prevalence of 7.3%, reported by the ICMR INDIA B study of 57,117 adults over 20 years from 14 states and the Union Territory of Chandigarh. It was observed that low diabetes prevalent states (4.3%) such as Bihar, had a higher prevalence of Prediabetes (10%) indicating an expected rise in diabetes in the next decade. There is sufficient evidence supporting the "Asian Phenotype" in diabetes, which is characterized by early onset, higher risk even at low BMI, higher abdominal adiposity, higher CVD in South Asia and stroke in East Asia. Researchers have shown that lifestyle interventions may decrease the risk of prediabetes progressing to diabetes for as long as 10 years. The benefits of long-term diet and exercise intervention to prevent diabetes was shown in the Chinese Da Quing study of more than 500 IGT subjects. The Finnish Diabetes Prevention Study also demonstrated a 58% reduction in the progression from IGT to diabetes with intense lifestyle changes. During Preventive health screening, routinely done in many hospitals across India we find many cases of IGT/ Prediabetes. These patients are counselled about diet as well as therapeutic lifestyle changes and weight management that could prevent diabetes. It is often observed that there is lack of follow up in this segment. A better follow-up, constant motivation and monitoring not only helps in establishing the lifestyle changes, but also in sustaining these on a long-term basis. This presentation would throw light on how therapeutic lifestyle changes, if closely followed up by the dietitians/ wellness consultants, can prevent IGT patients from becoming diabetics. We have an ongoing study where we follow-up with the Prediabetic patients in our hospital for a span of three months with interventional intensive therapeutic lifestyle changes, the results of which will be discussed in this presentation. This study gives scope for further research on the duration of follow-up required to sustain the change and the barriers faced in implementing these lifestyle changes in the community. As compared to a standard lifestyle advice, an intensive therapeutic lifestyle change, with periodic follow-up would help in the prevention of diabetes.

Speaker Biography

Uthara Upadhrashta is a registered dietitian in India. She graduated in food science and nutrition from Sri Sathya Sai Institute of Higher Learning, Anantapur. She holds a post graduate certificate in practical diabetology conducted by Steno Diabetes Research centre, Denmark. She is also a certified diabetes educator from the Christian Medical College, Vellore- under project HOPE, recognized by the International Diabetes Federation. She is a clinical dietitian and has completed 21 years of clinical nutrition practice in various multi-specialty hospitals in India. She has actively participated in national and international conferences and currently pursuing research on lifestyle risk factors and coronary artery disease, she works with a passion to advocate nutrition therapy in various clinical conditions, both in the hospital as well as in the community. She is the chief dietitian at SIMS Hospital (SRM Institutes for Medical Science), India.

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Euthyroid Sick Syndrome: Role of glucagon

Euthyroid sick syndrome is a manifestation of transient hypothalamic-pituitary dysfunction along with altered thyroid hormone metabolism. It is not prudent to rely solely on a single thyroid test in the evaluation of thyroid function of patients with critical illness, and a careful assessment of multiple tests may be needed. It is reasonable to delay the final diagnosis for several days to weeks, or after recovery from the acute illness, to determine the appropriate thyroid status. Thyroid hormones have been used in the setting of NTI in various settings with T4 and T3 replacement and remain controversial in the absence of prospective studies to demonstrate benefit. Assessing thyroid function in patients with severe illness such as those in the ICU is difficult. Many of them have low serum concentrations of thyroxine (T4), free T4, and triiodothyronine (T3), free T3, and their serum thyrotropin (TSH) concentrations also are frequently low. Thyroid function tests need not be assessed in seriously ill patients unless there is a strong suspicion of thyroid dysfunction. Also, measurement of serum TSH alone is inadequate for the evaluation of thyroid function and, in this scenario, free T4 and free T3 along with TSH are recommended. However, these tests frequently fail to differentiate between euthyroid sick syndrome and central hypothyroidism. Determination of serum reverse T3(RT3) may be helpful since RT3 is almost always elevated in euthyroid sick syndrome while being low in central hypothyroidism. Treating patients with critical illness with low serum T3 and/or low T4 concentrations with no other clinical signs of hypothyroidism is not commonly recommended. Patients may receive thyroid hormone replacement if there is additional evidence to suggest a diagnosis of hypothyroidism (such as a TSH over 20 mU/L with low free T4 and/or history, symptoms, and signs of hypothyroidism), in which case cautious administration of thyroid hormone is appropriate. Therefore, thyroid functions should not be assessed in critically ill patients in the absence of a suspicion of thyroid dysfunction as these abnormalities are not a true reflection of actual hormonal activity at the cellular level and treatment of these patients with thyroid hormones is of little benefit and sometimes may be detrimental. In this presentation, several clinical disorders manifesting altered thyroid hormone levels noted with euthyroid syndrome are described. Moreover, presence of hyperglucagonemia as well as its relationship with thyroid hormone metabolism and hypothalamic pituitary thyroid axis in these disorders is documented. Finally, role of glucagon is established in both altered thyroid hormone metabolism and altered hypothalamic pituitary thyroid axis documented in euthyroid sick syndrome by determination of serum thyroid hormones and TSH concentrations in response to glucagon administration in dogs, normal human subjects as well as subjects with clinical disorders. Moreover, influence of TSH in conversion of T4 into T3 in nonthyroidal peripheral tissues is also demonstrated in athyreotic dogs and human subjects.

Speaker Biography

Udaya M Kabadi is a graduate of Seth G.S. Medical College, the University of Bombay in Bombay, India. He completed his internal medicine residency at KEM Hospital Parel in Bombay and a medicine residency at Jewish Memorial Hospital and Beth Israel Medical Center in New York, He also completed a fellowship in endocrinology and metabolism at VA Medical Center and Beth Israel Medical Center in New York, New York. He is board certified in internal medicine, endocrinology and metabolism and geriatric medicine by the American Board of Internal Medicine. He is a fellow of the Royal College of Physicians of Canada, the American College of Physicians and the American College of Endocrinology. He has been a chief editor, associate editor and member of editorial boards of several medical journals. He is currently an adjunct professor of Medicine at the University of Iowa College of Medicine, Iowa City as well as Des Moines University, Des Moines, Iowa. He has over 200 publications in peer-reviewed journals. He has presentations to his credit, at regional, national, and international arenas. He has been selected as 'Teacher of the Year' many times by students, residents, and fellows in training. He has been involved in research in the area of carbohydrate metabolism and diabetes, thyroid disorders and osteoporosis as well as in clinical practice and education for several years.

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Royce Vincent

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Remission of type 2 diabetes mellitus: Bile acid signalling and incretins

There is growing evidence of long-term remission of type 2 diabetes after metabolic surgery (bariatric surgery) however; the pathophysiology of improved glucose metabolism after surgery remains poorly understood. Bile acids are the main component of human bile and have traditionally been considered mediators of lipid absorption and cholesterol metabolism, facilitated by their amphipathic nature. In recent years bile acids have been identified as metabolic molecules which regulate glycaemic control amongst other processes via activating the nuclear receptor, farnesoid X receptor (FXR) and the G protein-coupled membrane receptor (TGR5). Furthermore the interplay between bile acids and incretin hormones such as glucagon like peptite-1 (GLP-1) has given us new insight into their collective contribution in improving glycaemic control. Bile acid pool and composition are altered following certain metabolic surgeries such as Roux-en-Y gastric bypass (RYGB) and the post-prandial GLP-1 responses are enhanced after RYGB. This session will review our current understanding of these metabolic regulators and the potential role they play in the remission of type 2 diabetes mellitus after metabolic surgery.

Speaker Biography

Royce Vincent is a consultant chemical pathologist at King's College Hospital NHS Foundation Trust and an Honorary Senior Lecturer at King's College London, UK. He has a special interest in nutrition and endocrinology and is the clinical lead for biochemistry and parenteral nutrition services. He obtained his MD (Res) at Imperial College London and his research interests are in obesity, endocrinology and clinical nutrition. He has published multiple original and review articles and is serving as an international editorial board member for Translational Metabolic Syndrome Research.

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