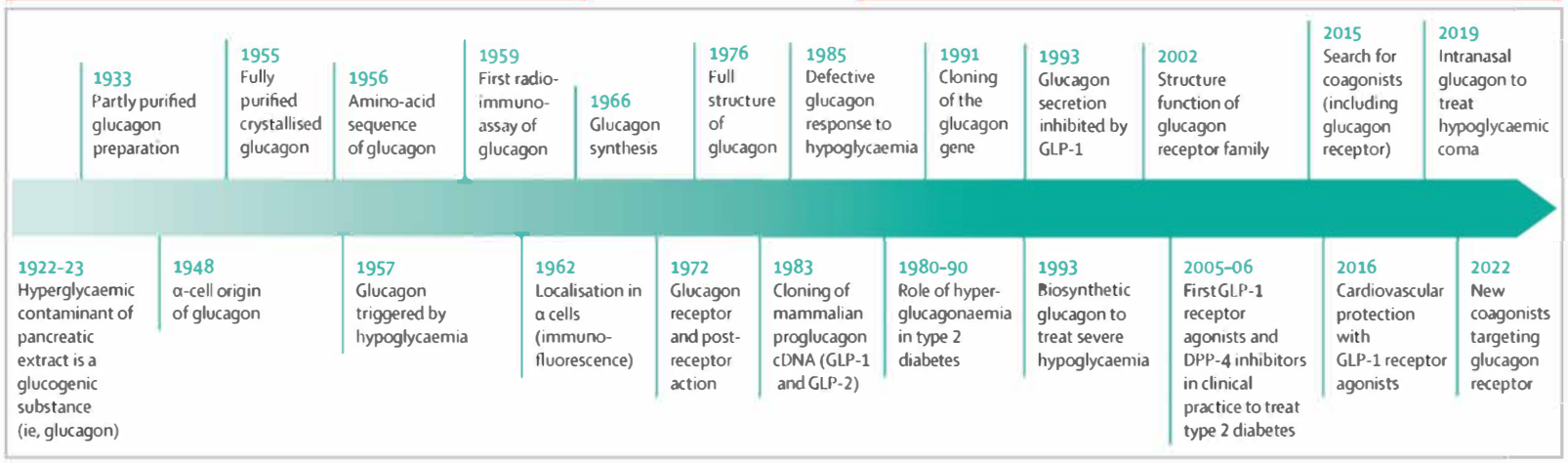
NCD Newsletter

Nov 2023 - Issue 08

Since its discovery 100 years ago in 1922, glucagon has made a significant impact in diabetes care management. 2022, marks the commemoration of the 100th anniversary of the groundbreaking discovery of glucagon, a hormone that has played a pivotal role in the realm of diabetes research and treatment.

The oral capsules could potentially be designed to allow dosing over specific time periods, similar to injection delivery. There is a need to investigate this further, develop a way of doing so and undergo rigorous testing as part of future human trials.

CLINICAL TRIAL SCENE



Timeline of the major milestones in the glucagon story

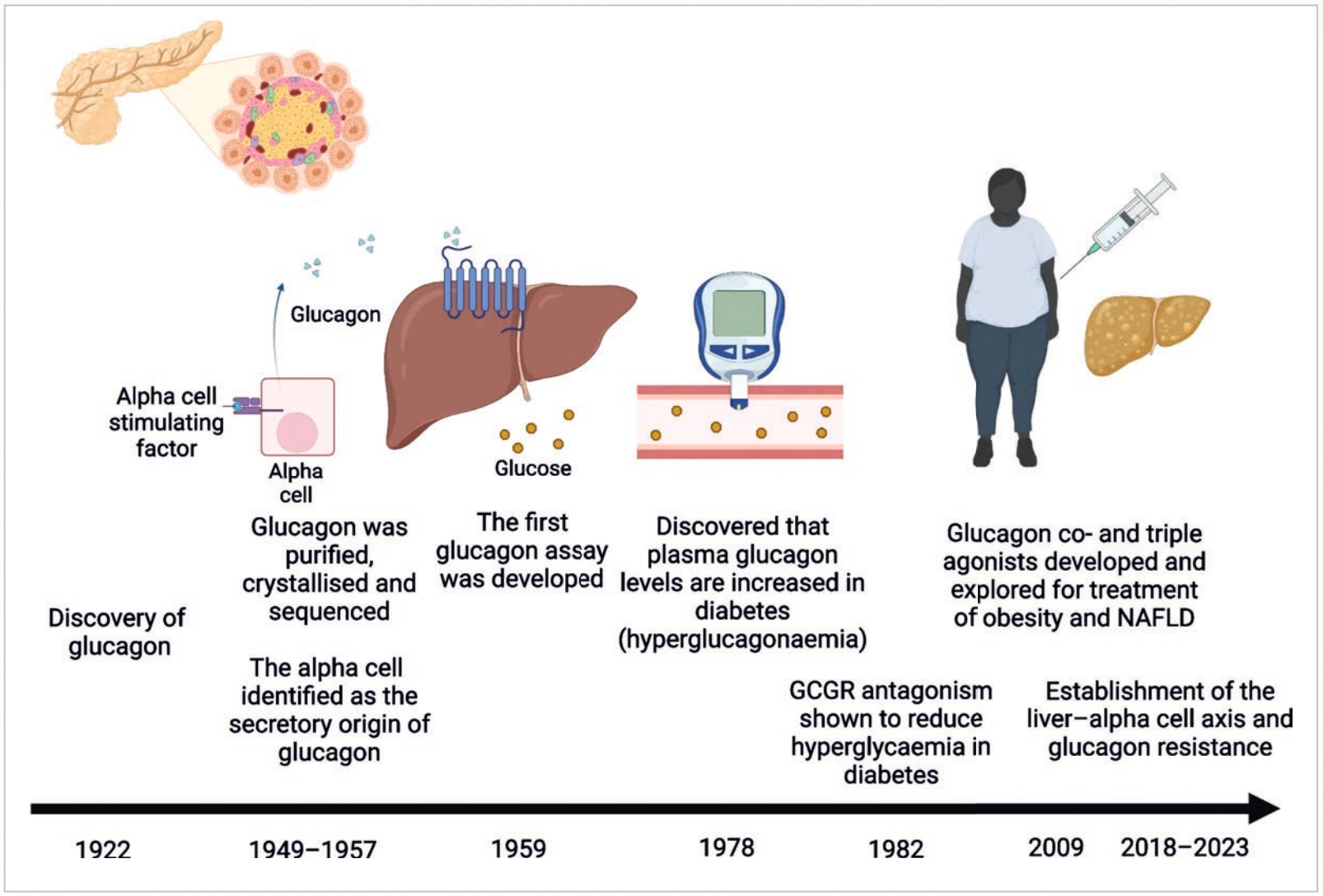
The story of glucagon begins with its early historical findings in basic research. Pioneering work, including the discovery, purification, structural elucidation, and identification of its origin in α-cells, laid the foundation for future investigations. Notably, this research engaged the efforts of three future Nobel Prize laureates, underscoring the significance of this hormone.

Three illustrious Nobel Prize laureates in Physiology and Medicine have left an indelible mark on the realm of glucagon research. Earl Wilbur Sutherland Jr., a laureate in 1971, used glucagon in his pioneering experiments, a pivotal work that led to the discovery of cyclic AMP (cAMP), a crucial signaling molecule in cell biology. Christian de Duve, who received the Nobel Prize in 1974 for his groundbreaking work on lysosomes, ventured into characterizing the hyperglycemic and glycogenolytic factor of the pancreas. Martin Rodbell, honored with the Nobel Prize in 1994 alongside Alfred Gilman, made significant contributions to the understanding of G proteins and their pivotal role in cell signaling. Rodbell's early research was dedicated to unraveling the intricate regulation of glucagon's actions at its receptors. These Nobel laureates have left an enduring legacy in the field of glucagon research, contributing to our understanding of this critical hormone's role in physiology and disease.

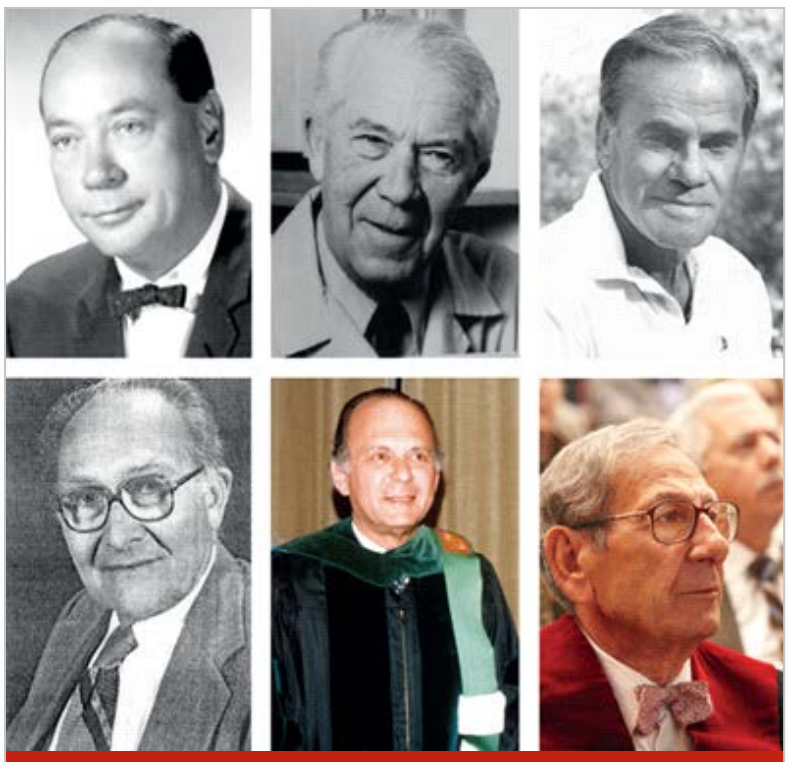
Initially regarded as an "anti-insulin" hormone, glucagon quickly found its place in the clinical arena, particularly in the management of insulin-induced hypoglycemic coma episodes among individuals with type 1diabetes. However, a pivotal turning point emerged when researchers uncovered the physiological and pathophysiological roles of glucagon and α-cells in type 2 diabetes, introducing the concept of "paracrinopathy." This paradigm shift sparked the development of various therapeutic strategies targeting glucagon.

One of the most significant breakthroughs in this narrative was the advent of GLP-1 receptor (GLP1R) agonists, which not only helped regulate glucagon but also stimulated insulin secretion in a glucose-dependent manner. These advancements offered new avenues for treating diabetes by simultaneously inhibiting glucagon secretion and promoting insulin release.

Glucagon-like Peptides (GLPs): GLPs have taken center stage in the realm of diabetes research and treatment. GLP-1(7-37), a 31-amino acid peptide secreted by intestinal L cells in response to food intake, plays a pivotal role in regulating blood sugar. It not only exerts a significant insulinotropic effect but



also acts as a robust inhibitor of glucagon secretion by α-cells, all in a glucose-dependent manner. Beyond its role in pancreatic islet regulation, GLP-1 influences peripheral and central processes, slowing gastric emptying, promoting satiety, and even improving vascular function. However, the short half-life of GLP-1 due to rapid degradation by DPP-4 limits its direct therapeutic use.



Notable people in the glucagon story Top row, from left to right: Earl Wilbur Sutherland Jr, Christian de Duve, and Martin Rodbell. Bottom row, from left to right: Piero Foà, Roger Unger, and Lelio Orci.

GLP-2 and Its Role in Intestinal Health: Another member of the GLP family, GLP-2, is secreted by intestinal L cells in response to nutrient intake. GLP-2 is primarily recognized for its role in regulating intestinal growth and function. Emerging research suggests its therapeutic potential in treating various intestinal diseases, notably short-bowel syndrome. Additionally, GLP-2 can stimulate glucagon release from islet α-cells and modulate islet adaptation to metabolic stress, underlining its intricate role in glucose homeostasis.

Glucagon's Dual Role in Diabetes: Glucagon, often overshadowed by insulin, plays a pivotal role in both hypoglycemia and hyperglycemia in individuals with diabetes. The contribution of glucagon varies based on the type of diabetes:

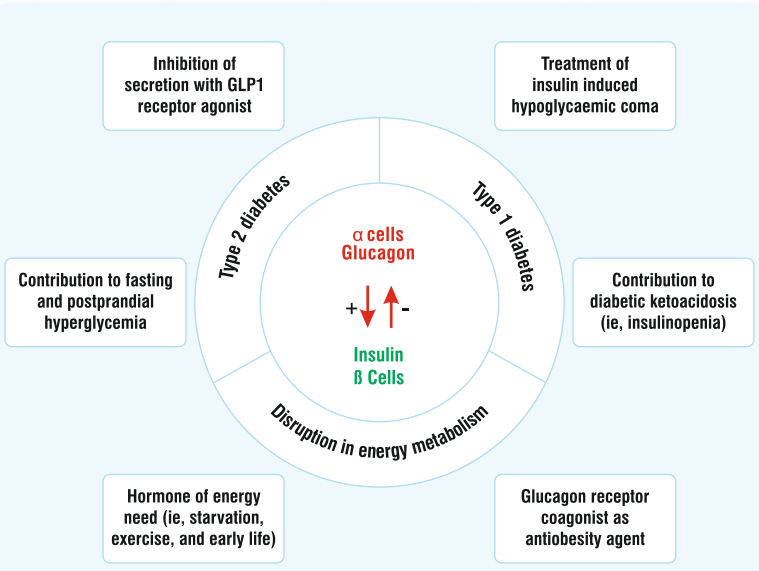
Secondary Diabetes: In cases where diabetes arises following pancreatectomy or severe pancreatic diseases, the insulin requirement is generally lower than in type 1 diabetes. This highlights the physiological role of glucagon in the hyperglycemia observed in diabetes.

Type 1 Diabetes: Individuals with type 1 diabetes exhibit insufficient glucagon responses to hypoglycemia, suggesting a pancreatic α-cells defect. While glucagon can prevent severe hypoglycemia by counteracting insulin's effects, glucagon is also responsible for diabetic ketoacidosis (DKA) when insulin is absent, emphasizing its role in this life-threatening condition.

Type 2 Diabetes: Glucagon's contribution to fasting and postprandial hyperglycemia is significant in type 2 diabetes. In the context of insulin deficiency, an excess of glucagon plays a substantial role in fasting hyperglycemia. Additionally, glucagon is implicated in post-meal hyperglycemia, as its secretion is not well inhibited after a glucose load. In people with type 2 diabetes, simultaneous insulin deficiency and glucagon excess are common, making glucagon inhibition a potential therapeutic target.

Glucagonomas and Weight Loss: Neuroendocrine tumors known as glucagonomas secrete excessive amounts of glucagon, leading to a condition known as glucagonoma syndrome. While diabetes is a common feature, it is typically mild due to preserved α-cells function compensating for glucagon's metabolic disturbances. One striking aspect of glucagonoma syndrome is severe weight loss, accompanied by disruptions in protein metabolism, making glucagon a potential candidate for promoting weight loss.

These findings illuminate the intricate role of glucagon and GLPs in diabetes,



Glucagon at the centre of important homeostatic processes and metabolic diseases

offering insights into potential therapeutic interventions and underscoring their significance in the multifaceted landscape of diabetes management.

Beyond diabetes, glucagon's ability to increase energy metabolism has led to innovative strategies for weight management, utilizing receptor coagonists to promote weight loss in individuals with obesity. The dichotomy of glucagon as both a problem and a solution has spurred research in opposing directions, seeking to either inhibit or enhance its activities, depending on the clinical context.

As we mark a century since the discovery of glucagon, our understanding of this second pancreatic hormone continues to deepen. The story of glucagon is far from over, offering exciting prospects for the future of metabolic disease research and treatment.

MEDICAL DEVICES

PerClot Polysaccharide Hemostatic System

PerClot Absorbable Hemostatic Powder (PerClot) is a powder made of absorbable granules of a substance called a polysaccharide. It is used to stop bleeding and promote blood clotting during surgery.

The PerClot System includes the following components:

- PerClot (granules/powder)
- PerClot Delivery System
- PerClot Accessory Tips (optional)

PerClot granules have a molecular structure that rapidly absorbs water, forming a sticky gel. The gel creates a physical barrier to stop bleeding. The barrier helps platelets, red blood cells, and clotting proteins (coagulation proteins such as thrombin and fibrinogen) accumulate and form a natural clot.

After the clot has formed, PerClot granules are broken down and absorbed by the body. In preclinical studies, absorption occurs within 96 hours but depends on the amount that is applied and where it was used.

It is indicated in surgical procedures (except neurological and ophthalmic) to help control bleeding when pressure, tying of blood vessels (ligature), or other typical methods do not work or are not practical.

During surgery (general, urologic, or cardiac), PerClot demonstrated the ability to stop bleeding at a rate similar to a control. The effectiveness of PerClot and the control were measured using a validated 6-point scale.

This should not be injected into arteries or veins as blockages may form (embolization) that can lead to serious injury or death.

FDA APPROVALS

FDA Approves First Oral Treatment for Postpartum Depression

Aug 4, 2023

The new drug, Zurzuvae (zuranolone), is a once-daily oral pill taken for just two weeks and works much faster than other antidepressants. This treatment is also less invasive than Zulresso, an extremely expensive 60-hour IV drip that was approved in 2019.

Health experts and advocacy groups welcomed the approval of the drug, given the lack of treatment options for postpartum depression, a life-threatening condition that can occur in the first year after childbirth and may even begin during pregnancy.

Zurzuvae showed promising results in its ability to lessen severe depression symptoms in as little as three days, but there's no data on its long-term effectiveness or safety yet.

GUIDELINE UPDATE

American Heart Association®

2023 AHA/ACC/ACCP/ASPC/NLA/PCNA-GUIDELINE FOR THE MANAGEMENT OF PATIENTS WITH CHRONIC CORONARY DISEASE

KEY TAKEAWAYS

1. Emphasis on team-based, patient-centred care in risk assessment, testing, and treatment considering social determinants and cost.
2. Nonpharmacologic therapies, recommended for all patients with chronic coronary disease (CCD).
3. Patients with CCD are encouraged to participate in habitual physical activity to reduce sitting time and increase aerobic and resistance exercise.
4. Use of SGLT2i & GLP-1 RA are recommended for select groups of patients with CCD, including groups without diabetes.
5. Long-term beta-blocker therapy is not recommended to improve outcomes in patients with CCD in the absence of MI and LVEF ≤50% & CCB or beta blocker is recommended as first-line antianginal therapy.
6. Statins remain first line therapy for lipid lowering in patients with CCD along with several adjunctive therapies (eg. Ezetimibe, PCSK9, Inclisiran, Bempedoic acid).
7. Shorter durations of DAPT therapy are safe and effective when the risk of bleeding is high and the ischemic risk is low to moderate.
8. The use of nonprescription or dietary supplements, including fish oil and omega-3 fatty acids or vitamins, is not recommended in patients with CCD.
9. Routine periodic anatomic or ischemic testing without a change in clinical or functional status is not recommended.
10. E-cigarettes are not recommended as first-line therapy for smoking cessation.

Reference: 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients with Chronic Coronary Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines, Circulation. 2023;148:e00–e00.

EUROPEAN SOCIETY OF CARDIOLOGY®

2023 ESC GUIDELINES FOR THE MANAGEMENT OF CARDIOVASCULAR DISEASE IN PATIENTS WITH DIABETES

KEY POINTS

Recommendations for screening

- In all individuals with CVD, screening for diabetes (using fasting glucose and/or HbA1c) is recommended (Class I, A)
- In patients with T2D, it is recommended to assess medical history and the presence of symptoms suggestive of ASCVD (Class I, B)
- In all patients with T2D, a systematic survey for HF symptoms and/or signs of HF at each clinical encounter is recommended (Class I, C)
- It is recommended to routinely screen patients with T2D for kidney disease by assessing eGFR and UACR (Class I, B)

Recommendations for glucose-lowering treatment

- Prioritize the use of glucose-lowering agents with proven CV benefits followed by agents with proven CV safety over agents without proven CV benefit or proven CV safety (Class I, C)
- Switch glucose-lowering treatment from agents without proven CV benefit of proven CV safety to agents with proven CV benefit (Class I, C)

Treatment recommendations

- In patients with T2D and ASCVD, GLP-1RA and SGLT2-inhibitors are recommended to reduce CV risk independent of glucose control (Class I)
- In all patients with T2D and HF (HFrEF, HFmrEF, HFpEF), SGLT2 inhibitors are recommended to reduce HF hospitalization (Class I)
- The guidelines now recommend use of empagliflozin or dapagliflozin for patients with HFmrEF and HFpEF to reduce the risk of HF hospitalization or CV death
- In patients with T2D and CKD, SGLT2 inhibitors and finerenone are recommended to reduce CV and kidney failure risk (Class I)

CV risk stratification

- The novel T2D-specific risk score SCORE2-Diabetes is introduced to assess the CV risk in patients with T2D without ASCVD or severe target-organ damage (TOD)

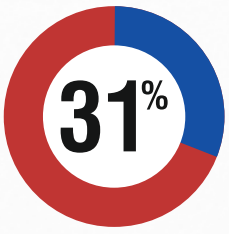
Reference: European Heart Journal, 2023;, ehad192

TRIAL UPDATE

1. SGLT2 inhibitors may reduce recurrent gout flares in patients with type 2 diabetes:

Sodium-glucose cotransporter-2 inhibitors (SGLT2is) can prevent recurrent gout flares, gout hospitalizations, as well as cardiovascular events in patients with type 2 diabetes and prevalent gout, according to research published in Annals of Internal Medicine.

Among Patients with gout, SGT2 inhibitor initiation was linked to a:



relative risk reduction for myocardial infarction

The global burden of gout - the most common inflammatory arthritis- continues to increase. Suboptimal gout care and the "modern gout epidemic" have both contributed to high rates of recurrent flares.

"These gout flares have also been associated with transiently higher cardiovascular risk, and many cardiometabolic comorbidities accompany gout, including type 2 diabetes, metabolic syndrome, chronic kidney disease, and cardiovascular disease."

Researchers conducted a general population cohort study to compare cardiovascular events and gout flares in 8,150 patients with type 2 diabetes and gout who were taking SGLT2 inhibitors vs. dipeptidyl peptidase 4 (DPP-4) inhibitors - another glucose-lowering drug not associated with cardiovascular disease or serum urate levels.

McCormick and colleagues reported that the flare rate was lower in patients taking SGLT2 inhibitors compared with those taking DPP-4 inhibitors at 52.4 vs. 79.7 events per 1,000 person-years (RR = 0.66; 95% CI, 0.57-0.75). The rate difference (RD) was -27.4 per 1,000 person-years (95% CI, –36 to –18.7). Taking SGLT2 inhibitors was linked to a 34% lower rate of recurrent gout flare - "the central goal of clinical gout care.

The researchers also noted that the corresponding HR for stroke was 0.81 (95% CI, 0.62-1.05) and the RD and HR for myocardial infarction were -7.6 (95% CI, –12.4 to –2.8) and 0.69 (95% CI, 0.54-0.88) per 1,000 person-years. SGLT2 inhibitor initiation was linked to a relative risk reduction in myocardial infarction of 31%.

Ref: McCormick N, et al. Ann Intern Med. 2023

2. Comparative cardiovascular outcomes in type 2 diabetes patients taking dapagliflozin versus empagliflozin: a nationwide population-based cohort study:

New study compared the cardiovascular outcomes between new users of dapagliflozin and empagliflozin in a broad range of patients with type 2 diabetes mellitus using a nationwide population-based real-world cohort from Korea.

Results: A total of 366,031 new users of dapagliflozin or empagliflozin were identified. 72,752 individuals (mean age approximately 56 years, 42% women) from each group were included in the final analysis, with a follow-up of 150,000 person-years. Approximately 40% of the patients included in the study had type 2 diabetes mellitus as their sole cardiovascular risk factor, with no other risk factors. The risk of the primary outcome was not significantly different between dapagliflozin and empagliflozin users (hazard ratio [HR] 0.93, 95% confidence interval [CI] 0.855-1.006). The risks of secondary outcomes were also similar, with the exception of the risks of HF-related events (HR 0.84, 95% CI 0.714-0.989) and cardiovascular death (HR 0.76, 95% CI 0.618-0.921), which were significantly lower in the dapagliflozin users.

Conclusions: This large-scale nationwide population-based real-world cohort study revealed no significant difference in composite cardiovascular outcomes between new users of dapagliflozin and empagliflozin. However, dapagliflozin might be associated with lower risks of hospitalization or death due to HF and cardiovascular death than empagliflozin in Asian patients with type 2 diabetes mellitus.

Reference: Cardiovascular Diabetology volume 22, Article number: 188 (2023)

3. SGLT2i reduces CV outcomes in HF regardless of deterioration of eGFR below 25 ml/min/1.73m²

AUG. 25, 2023

Effects of Dapagliflozin in Patients with Heart Failure and Deterioration of Kidney Function Below eGFR 25 ml/min/1.73m²: A Participant Level Pooled Analysis of the DAPA-HF and DELIVER Trials

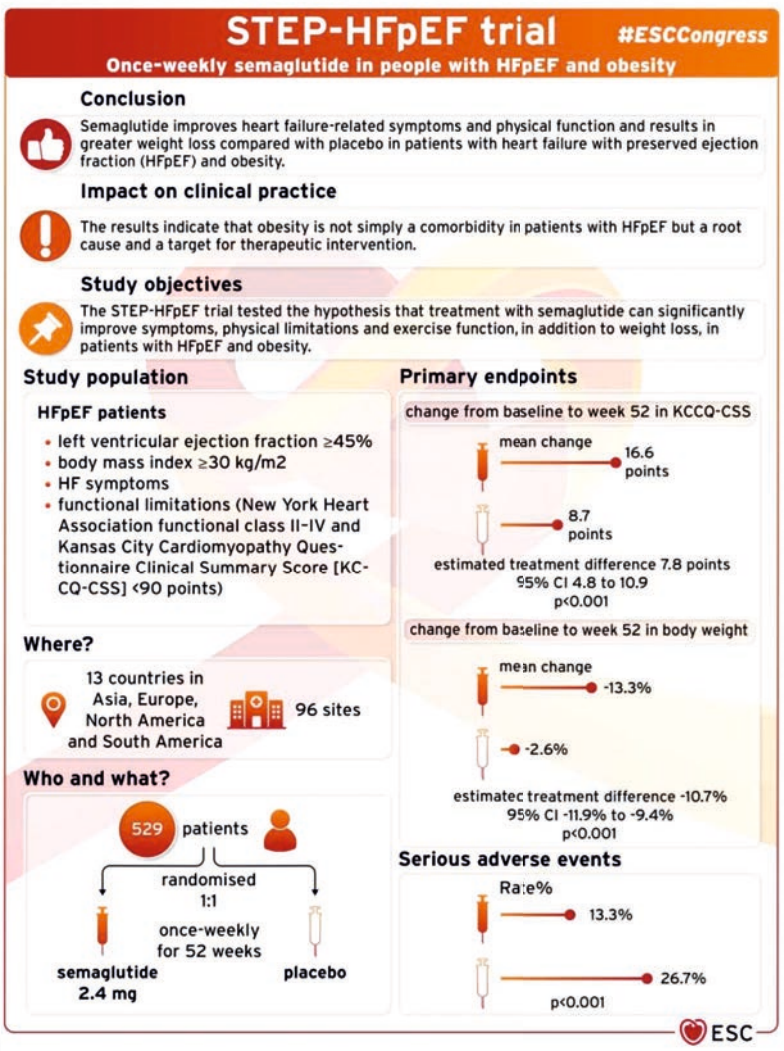
At the European Society of Cardiology 2023 Congress in Amsterdam, investigators presented late-breaking data on the benefit-to-risk ratio of continuing treatment with dapagliflozin in patients with heart failure and advancing chronic kidney disease (CKD): deterioration in kidney function to an estimated glomerular filtration rate (eGFR) less than 25 mL/min/1.73m2.

Investigators pooled data from the DELIVER and DAPA-HF trials involving

11,007 patients with heart failure and preserved, mildly reduced, or reduced ejection fraction. Of these, 347 patients (3.2%) had an eGFR that declined to less than 25 mL/min/1.73m2 during the trials. The time to deterioration of kidney function was similar for patients randomly assigned to dapagliflozin or placebo: 120 vs 121 days.

The risk for the primary composite outcome of cardiovascular death or worsening heart failure was 87% higher for patients with vs without renal decline to less than 25 mL/min/1.73m2, further reinforcing the adverse connection between heart failure and kidney disease, Scott D. Solomon, MD, of Harvard Medical School in Boston, Massachusetts, reported on behalf of his research team. The primary outcome occurred in 18.6 vs 10.2 per 100 person-years of the groups, respectively.

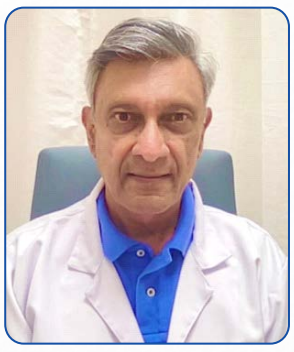
4. Semaglutide improves heart failure-related symptoms and physical function and results in greater weight loss compared with placebo in patients with HFpEF and Obesity



CASE STUDY - 1

Complete atrioventricular block complicated by QT prolongation triggering repeated torsades de pointes polymorphic ventricular tachycardia

DR DEEPAK NATARAJAN
MBBS, MD (Med) DM (Cardiology)
Senior Consultant Interventional Cardiologist
Fortis Hospital, Greater Noida



Introduction

Patients with complete atrioventricular block (CAVB) may suffer from episodes of syncope when the heart rate drops below a sustainable physiological level. There are, however, some patients with complete atrioventricular block who also have prolongation in the corrected QT interval (QTc) at rest, with increases propensity for malignant tachyarrhythmia such as a TdP polymorphic ventricular tachycardia. The first report described by Dessertenne was that of CAVB leading to TdP. Torsades de pointes polymorphic ventricular tachycardia refers to a ventricular tachycardia that twists around the isoelectric line that is triggered by delayed ventricular repolarization manifested by an abnormally increased QT and QTc intervals on the surface ECG.

Congenital long QT syndrome (LQTS) is an inherited channelopathy with prolonged QTc resulting in heightened susceptibility to poly-morphic ventricular tacyarrhythmias and sudden cardiac death (SCD). More than 500 mutations have been described in 12 LQTS susceptibility genes.

Abnormal QTc prolongation on ECG is the clinical identification of LQTS. Decrease in repolarizing outward potassium currents or increased late entry of sodium into the heart cell induces QT prolongation. This case report describes the management of a female patient presenting with the uncommon combination of CAVB and prolongation of QT in-terval triggering multiple episodes of TdP polymorphic ventricular tachycardia.

Case report

A 30 years old married woman was admitted in the Emergency for multiple episodes of unconsciousness in the previous 3 days. In the Emergency, a Torsades de pointes (TdP) polymorphic ventricular tachycardia was observed on the cardiac monitor that terminated

spontaneously into complete AV dissociation with bizarre large T waves. Both QT and QTc were prolonged more than 600 ms (Figs. 1–2; and video).

There was no prior medical history or an episode of a fainting spell in the past. The patient had no history of fever, breathlessness, loose mo- tion or drug intake previously. There was no history of syncope in her immediate family.

On examination, heart rate was 38 per minute while blood pressure was 106/70 mm Hg. Physical examination was unremarkable.

The complete blood count, biochemistry, and troponin were normal. The patient was not hypothyroid nor did she have any electrolyte disturbance. A colour Doppler 2 D echocardiogram revealed a struc- turally normal heart.

In view of the complete AV block, increased QTc interval, accom- panied by large upright and inverted T waves (Fig. 3) the patient un- derwent temporary pacing followed by insertion of a permanent dual chamber pacemaker the next day (Fig. 4). The pacemaker was pro- grammed to a minimum pacing rate of 80 per minute initially. Subse- quent to permanent pacing there were no further episodes of tachycardia or syncope. The patient was discharged on a beta blocker.

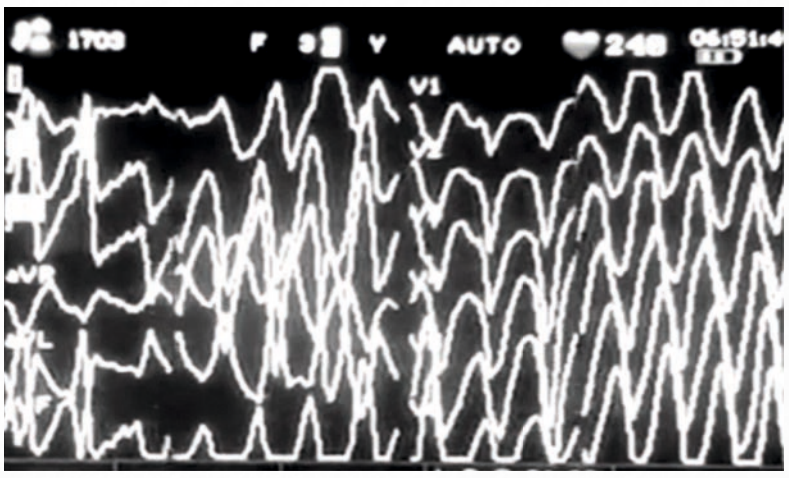


Fig. 1. Cardiac monitor showing Torsade de pointes polymorphic ventricular tachycardia.

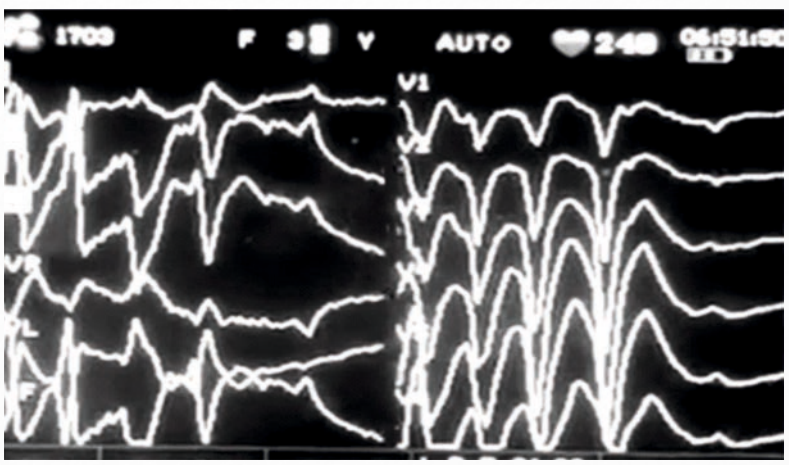


Fig. 2. Cardiac monitor showing |TdP polymorphic ventricular tachycardia spontaneously resolving to complete atrioventricular block.

Discussion

Long QT syndrome (LQTS) is abnormally delayed repolarization of myocardial cells that is reflected on the surface ECG as prolonged QT interval (QTc >480 ms the 50th percentile among LQTS cohorts) and associated with pre syncope, syncope, seizures, cardiac arrest, and sudden death in an apparently healthy person. The majority of patients carry mutations in the genes KCNQ1, KCNH2, and SCN5A. There are loss of function mutations in the KCNQ1 (Long QT Syndrome Type 1) and KCNH2 genes (Long QT Syndrome Type 2), or gain of function mutations in the SCN5A gene (Long QT Type 3).

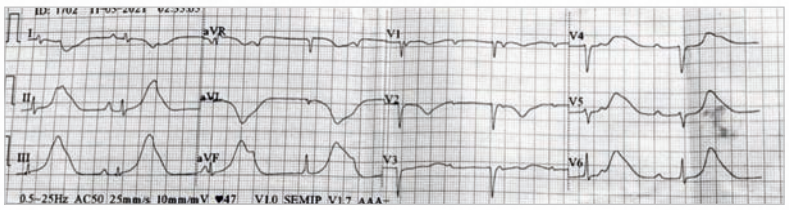


Fig. 3. 12 lead ECG demonstrating complete AV block with QTc of 657 ms accompanied by large giant upright and inverted T waves and superimposed P waves.

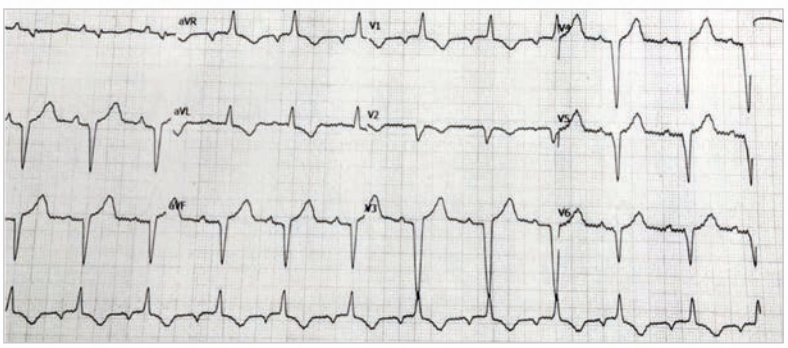


Fig. 4. 12 lead ECG showing normally functioning permanent dual chamber pacemaker with reduction in QTc to 520 ms.

The triggering factor for development of TdP in LQTS1 is physical or mental stress, swimming, or diving. IN LQTS2 the triggering event may be mental /physical stress, or a loud noise. Events occurring in sleep are associated with LQT3 (gain of function in SCN5A gene). Beta blockers are effective in LQTS1, but less in LQTS 2 and LQTS 3.

T waves in LQTS1 are broad based, while in LQTS2 they are of low amplitude, notched or biphasic. LQTS3 is associated with a long iso- electric segment leading to a narrow based T wave.

Usually LQTS is passed on in an autosomal dominant manner but there are rare cases that are inherited recessively accompanied by hearing loss. Incidence of LQTS may be as high as 1 in 2000 people. However, in clinical practice, cases of acquired LQTS predominate usually due to drugs or electrolyte imbalance.

LQTS1 is the most common congenital form, and sudden death (ac- cording to the International LQTS Registry), occurs in only 4% affected persons, while 50% of persons with mutations associated with this syndrome remain asymptomatic. A person with LQTS1 could suffer sudden death without any prior symptoms, but there also could be aLQTS1 genotype who has 200 episodes of syncope but be alive at 45 years Another conundrum is the presence of prolonged QT interval in as- sociation with atrioventricular block (AV) conduction block. A sizeable proportion of patients with complete atrioventricular block have pro- longation in the QT interval also, these patients are susceptible to TdP polymorphic ventricular tachycardia.

T wave changes as seen in LQTS2 are predictive of TdP during ac- quired AV block. Thirty cases of AV block complicated by torsades de pointes were compared with 114 cases of uncomplicated AV block. By multivariate logistic regression analysis, increased QT interval and presence of LQTS2 like T wave morphology (bifid or notched T waves) were independent and additive predictors of torsades de pointes. A QT more than 510 ms and LQTS2 like morphology almost surely predicted occurrence of TdP.

Studies show that 14%–18% of subjects with atrio-ventricular block- related QT-interval prolongation were carriers of a genetic mutation in genes coding for potassium channels, and also that there are genetic mutations involving KCNH2 and SCN5A in 36% of patients with com- plete atrioventricular block and TdP. In fact, the most commonly encountered mutation in this setting is in KCNH2 coding for potassium channel IKr.

Female patients with CAVB are more susceptible to suffering TdP, with delay in instituting a physiological heart rate resulting in further prolongation in QT and greater vulnerability to TdP. Notching of T wave is an additional risk factor for development of TdP.

Patients with CAVB who develop TdP have significantly longer QTintervals than those patients with CAVB who do not. Abnormal pro- longation of QT is more likely with heart rate 60 beats per minute or below .The largest study, however, reported that it was not the heart rate but QT interval or T wave morphology that best predicted TdP.5 Animal studies have shown that long standing bradycardia re- duces potassium current channels activity, lengthening the QT interval, and increasing susceptibility to TdP polymorphic ventricular tachycardia.

Congenital complete heart block accompanied by QT prolongation has been considered as a special type of arrhythmia. Prolonged QT in- terval is significantly higher than controls, even at higher pacing rates when corrected QT actually shortens. Treatment is permanent pace- maker implantation and beta blocker therapy.

An European LQTs-ICD Registry with 233 LQTS patients receiving an implantable cardioverter defibrillator (ICD) recorded that in 7 years follow up no appropriate shock was delivered in a patient above the age of 20 years, no previous cardiac arrest, no cardiac event despite optimal therapy, and QTc <500 milliseconds. But 70% received a shock if the reverse was true and all 4 conditions were met. This registry included, then, the largest number of LQTS who had received an ICD The latest European guidelines recommend implantation of an ICD in addition to beta-blockers in LQTS patients with cardiac arrest (Class 1). An ICD is also recommended in symptomatic LQTS patients on beta- blockers and genotype specific treatment. An ICD may be considered in an asymptomatic LQTS patient (Class II B) with a high risk profile according to the latest European guidelines on ventricular arrhythmias and sudden cardiac death. The risk profile for primary implantation of an ICD, however, is based upon inherited LQTS. There is no mention of prolonged QT triggering polymorphic ventricular tachycardia in the setting of complete atrioventricular dissociation in this predated risk profile. The risk profile is primarily based on QTc interval and the ge-notype, with the threshold for ICD implantation being 5% sudden death at 5 years.

The Rochester LQTS Registry data suggests that an ICD reduces mortality in a) patients with previous non fatal cardiac arrest, patients with syncope on beta blocker, and patients with syncope while off beta blocker and a QTc 500 milliseconds or more. The Rochester Registry, a part of the International QTS Registry, includes only patients with inherited LQTS.

Importantly neither the International nor the European LQTS Reg- istries include patients with complete AV block having prolonged QT interval, such as the patient described in this case report. There is no mention of such patients in the latest European guidelines on sudden cardiac death, despite having an elaborate section on LQTS. An ICD was not considered in this case because after insertion of a temporary ven- tricular pacemaker there was immediate cession of ventricular tachy- cardia that persisted till discharge, following permanent pacemaker was implantation.

Ideally the patient should have consented to genetic testing and counselling for genotype specific treatment, but did not. Some patients with complete atrioventricular block accompanied by prolonged QT interval may be prone to episodes of TdP resulting in syncope, cardiac arrest, or death due to ventricular fibrillation. Early diagnosis with implantation of a pacemaker may be life saving. A dual chamber ICD may be considered in the event of break through TDP despite a per- manent pacemaker. There is, however, sparse or no data on the optimal management of patients with complete AV block associated with long QT interval suffering TdP polymorphic ventricular tachycardia.

Conclusions

This case report underlines the importance of implant- ing a permanent dual chamber pacemaker in patients with CAVB accompanied by QT prolongation triggering TDP ventricular tachy- cardia. The minimal pacing rate may be kept at 80 beats per minute initially, and could be brought down to 60 or 70 per minute with nor- malisation of QT interval. Patients with complete atrioventricular block accompanied by prolonged QT interval are vulnerable to episodes of TdP resulting in syncope, cardiac arrest, and death due to ventricular fibrillation. Early diagnosis with implantation of a pacemaker can be life saving.

CASE STUDY - 2

A Study of Taste among Type 2 Diabetes Mellitus Patients with Autonomic Neuropathy at a Tertiary Care Centre in North India

DR J S KHUSHWAHA
MBBS., MD (Medicine)
Professor of Medicine
GSVM Medical College, Kanpur

Introduction : Type 2 diabetes mellitus (T2DM) is a multifactorial, complex disease associated with chronic hyperglycemia, resulting from the interplay of genetic, environmental, and epigenetic factors. Diabetic autonomic neuropathy (DAN) is one of the common complications in diabetes. The taste threshold is affected by various factors such as age, ethnic backgrounds, drugs, local and systemic diseases, consumption of alcohol, smoking, and tobacco chewing . The present study is undertaken with the objectives to compare the alteration in taste threshold for four primary sensations in Type 2 DM with autonomic neuropathy.

Material and methods: Out of a total of 75 T2DM patients recruited, only 60 patients met the selection criteria and were enrolled for the study. Four solutions of each of the four basic tastes (sweet, sour, salty, bitter) were used. subjects were asked to Rinse mouth twice with water and spit it out in the cup provided. After that 5ml of sample was provided whose nature was kept unknown to the subject and asked to hold it there for 5 seconds before spitting the solution into the cup.

Result: Out of 60 patients 26 (43.3%) were having taste dysfunction for sweet compared to 12 (20%) out of 60 healthy controls with p value 0.006. Taste dysfunction for sweet was significant in T2DM with uncontrolled hyperglycemia i.e mean HbA1c 10.83+/- 3.17 whereas in T2DM patients whose taste were preserved had mean HbA1c 8.05+/-1.87. The taste dysfunction in T2DM patients was not related to gender, disease duration, and type of treatment taken.

Conclusion: The study found a significant correlation between taste dysfunction and HbA1C level and blood sugar fasting level in type 2 diabetes mellitus patients. The taste dysfunction was mainly for sweet. Sour, and bitter did not show any difference in case groups compared to controls.

CASE STUDY - 3

Pseudohypercreatininemia after surgery for aortic dissection: a case report

Ayako Tasaki1*, Makoto Fukuda1, Yuki Ikeda1, Masatora Yamasaki1, Ikko Yamaguchi2, Shinichi Aishima3 and Motoaki Miyazono1*

Tasaki et al. BMC Nephrology (2023) 24:220

Background

Creatinine may be measured using the Jaffe method and enzymatic methods . The latter has high specificity and is used by many medical institutions but is affected by paraproteins and some drugs. There are reports of pseudohypercreatininemia caused by immunoglobulin (Ig)M but to the best of our knowledge, there are no reports of pseudohypercreatininemia caused by IgG. We report a case of pseudohypercreatininemia caused by polyclonal IgG interference in an enzymatic test routinely performed for creatinine measurement.

Case presentation

A 54-year-old woman underwent surgery for acute aor-tic dissection, Stanford type A, 4 years previously. She also underwent stent graft insertion for a dissecting abdominal aortic aneurysm 3 years previously. At the current admission, she had developed another aortic dis-section in the descending aorta and underwent graft replacement on X date. She had multiple aortic dissections at a young age, but had no findings or fam-ily history of Marfan syndrome or Ehlers-Danlos syndrome. Because of severe intraoperative bleeding, she was transfused with 10 units of red blood cells, 4 units of fresh-frozen plasma, and 10 units of platelets. She remained in the intensive care unit for 6 days postop-eratively, and her creatinine concentration was approxi-mately 1 mg/dl. However, on postoperative day 9, her creatinine concentration increased to 5.78 mg/dl. Uri- nalysis showed occult blood and proteinuria (Table 1); therefore, we considered performing a renal biopsy.

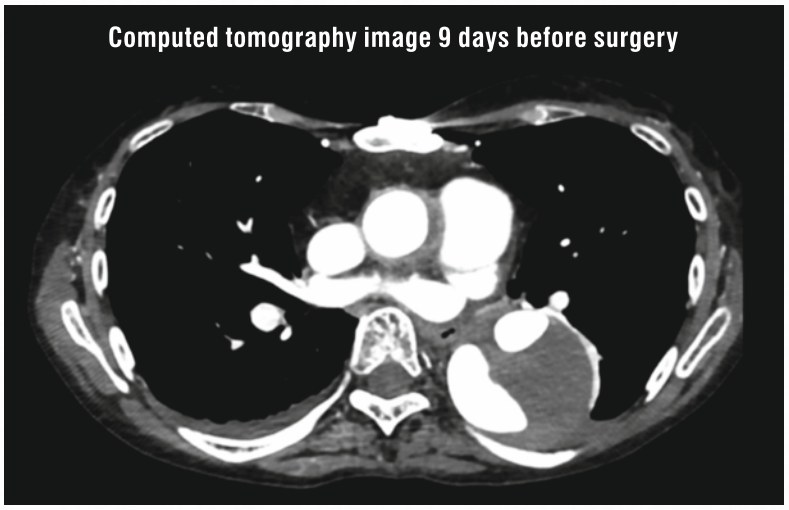


Table 1 Urine and blood test results on postoperative day 13

Urine tests	Value	Normal Value	Units
Protein	0.89	<0.15	g per 24 h
Erythrocytes	27.7	0–4	/HPF
Leukocytes	35.3	0–4	/HPF
Nitrites	positive	negative	
Blood tests			
Leukocytes	8900	3,000–7,800	/µl
Erythrocyte	3,050,000	3,530,000–4660000	/µl
Hemoglobin	9.4	10.6–14.4	g/dl
Platelets	2,500,000	138,000–309000	/µl
Total protein	6.8	6.5–8.0	g/dl
Albumin	2.7	4.0–5.2	g/dl
Blood urea nitrogen	10.2	7–24	mg/dl
Creatinine	7.68	0.47–0.7	mg/dl
Cystatin C	1.49	0.56–0.8	mg/l
eGFR	4.9	≧60	ml/min/1.73 m^2
eGFRcys	43.6	≧60	ml/min/1.73 m^2
Sodium	135	136–145	mmol/l
Potassium	4.5	3.3–4.8	mmol/l
Chloride	97	98–110	mmol/l
C-reactive protein	2.72	0–0.3	mg/dl
Immunoglobulin G	3165	870–1700	mg/dl
Immunoglobulin A	350	110–410	mg/dl
Immunoglobulin M	65	46–260	mg/dl
ANA	negative	negative	
Cryoglobulin	negative	negative	
PR3-ANCA	negative	negative	
MPO-ANCA	negative	negative	
GBM-Ab	negative	negative	

eGFR Estimated glomerular filtration rate, *eGFRcys* eGFR with cystatin, *C ANA* Antinuclear antibody, *PR3-ANCA* Proteinase 3-antineutrophil cytoplasmic antibody, *MPO-ANCA* Myeloperoxidase-antineutrophil cytoplasmic antibody, *GBM-Ab* glomerular basement membrane antibody

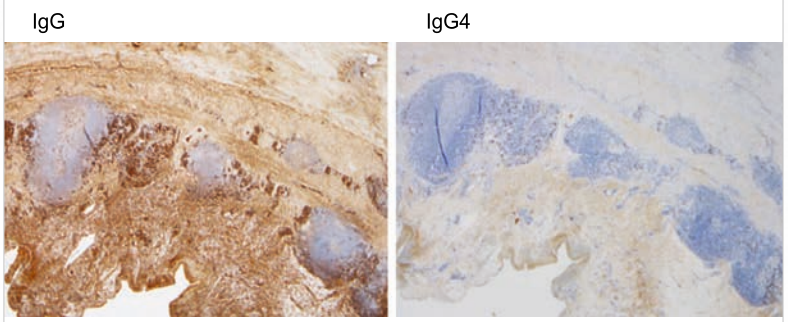
However, because there was no evidence of azotemia or hyperkalemia, pseudohypercreatininemia was consid-ered as a differential diagnosis. She had no uremic symptoms, such as general malaise or decreased appetite, and her urinary status was good. Her cystatin C concentration measured with creatinine was 1.56 mg/l (normal range, 0.56–0.8 mg/l), which supported the presence of pseudohypercreatininemia. Creatinine was measured by a different enzymatic method and the result was 0.84 mg/dl. Inulin clearance was 41 ml/min, which was consistent with pseudohypercreatininemia (Table 2). The patient had an abnormal total protein/albuminratio, suggesting the

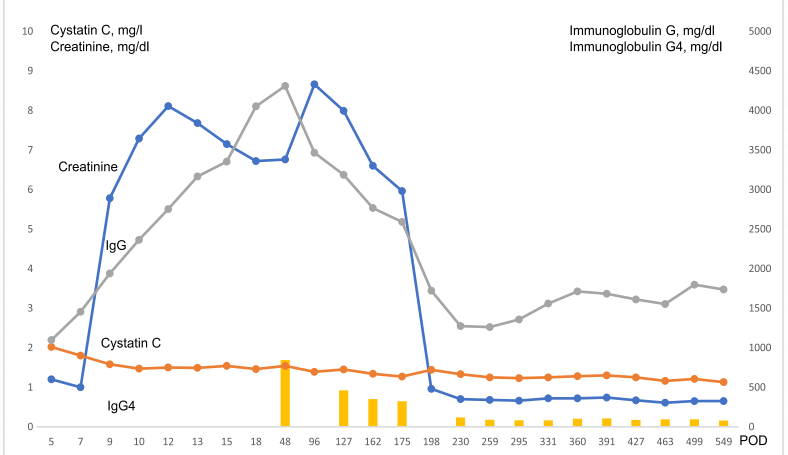
presence of abnormal proteins. Electrophoresis did not reveal any distinct M-proteins; however, IgG was markedly elevated, concurrent with the elevated creatinine concentration, and the IgG concentration fluctuated over time in parallel with the creatinine concentration. Therefore, paraproteins were considered a factor influencing the creatinine reagents. We measured IgG4 and found a very high concentration at 844 mg/dl, suggesting IgG4-related disease. However, computed tomography and gallium scintigraphy showed no obvious mass lesions other than pancreatic cysts. Histopathology of the resected aorta showed lymphatic follicles and plasma cell infiltration from the aortic adventitia to the adipose tissue, and IgG4-positive cell infiltration was observed in some areas. However, the IgG4/IgG ratio was approximately 15%, which did not fulfill the diagnostic criteria for IgG4-related inflammatory abdominal aneurysm or IgG4-related periarteritis. IgG4 and creatinine concentrations changed in tandem and were considered to be the cause of the pseudohypercreatininemia Therefore, after obtaining informed consent from the patient, we started oral administration of steroids at a dose of 20 mg, every 24 h. Thereafter, IgG and IgG4 concentrations decreased rapidly, and the creatinine concentration measured by the enzymatic method, which is routinely performed in our hospital, also improved. We are now searching for the cause of the rapid postoperative increase in IgG4, while reducing the steroid dosage.

Table 2 Postoperative cystatin C concentrations, and creatinine concentrations using different measurement methods

	Normal value	POD 7	POD 9	POD 13	POD 83
Creatinine (mg/dl)	0.47-0.7				
Enzymatic method (*Shigunasuoto*)		1	5.78	7.68	6.6
Enzymatic method (*Detamina-L*)		0.95	0.84	0.75	1.34
High-performance liquid chromatography			0.71		0.54
Cystatin C (mg/dl)	0.56-0.8	1.8	1.56	1.49	1.34

POD Postoperative day





The patient's clinical course. IgG, immunoglobulin G; POD, postoperative day

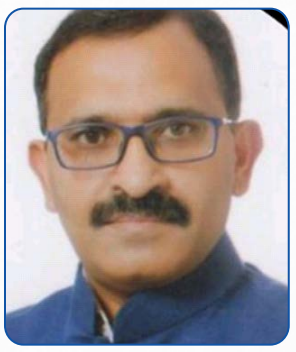
Discussion and conclusions

There are two methods for measuring creatinine concentrations the Jaffe method, which measures the active methylene group, and the enzymatic method using the Trinder reaction. The latter method is the primary method because of its sensitivity and simplicity and is the method used by many medical institutions and laboratories. Our hospital also uses the enzymatic method. However, drugs and abnormal serum proteins can cause errors in the creatinine measurement results. Hummel et al. and Storsley et al. reported that monoclonal IgM causes hypercreatininemia. All reports of hypercreatininemia indicate IgM as a cause and not IgA or IgG. There are only three known cases of IgG paraproteins causing false lowering of serum creatinine values using the Jaffe method. Our case is considered the first report of pseudohypercreatininemia caused by polyclonal IgG. Hummel et al. diagnosed pseudohypercreatininemia by high-performance liquid chromatography and the Jaffe method. Although high-performance liquid chromatography could not be performed immediately at our hospital, the presence of pseudohypercreatininemia was quickly inferred from the cystatin C concentration, and combined with inulin clearance, we confirmed normal renal function by measuring creatinine using a differentenzymatic method. The final diagnosis was made using high-performance liquid chromatography. Shigunasuoto and Detamina-L are similar enzymatic methods that differ by the buffer solution used. When the creatinine measurement was reproduced manually using Shigunasuoto, white turbidity appeared when the first reagent, the buffer solution, was mixed with the reagent. This turbidity might have increased the absorbance, resulting in a false high creatinine value. This phenomenon did not occur with Detamina-L. Generally, the M protein may become cloudy depending on the pH of the reagent and the concentration of the buffer solution, and this can occur in any enzymatic method. Reagents used in the Jaffe method may react with sugars, ketones, and cephalosporins in addition to serum creatinine. Both the Jaffe method and the enzymatic method may cause pseudohypercreatininemia; therefore, it is important to confirm the creatinine test method when hypercreatininemia is present without a typical clinical disease course. Our hospital uses the Shigunasuoto enzymatic method because, its positive aspects, including accuracy, compatibility, and cost. Accuracy is limited in any enzymatic method because of potential interference with the M protein. The strength of this case report is that although it is an event that could occur in any hospital, pseudo-hypercreatininemia caused by polyclonal IgG has not been reported before; therefore, this is the first report of its kind. A limitation is that there are approximately 30 competing reagents for measuring creatinine. We used Shigunasuoto and Detamina L, which have the largest market share; however, we were unable to validate our findings using other reagents. In addition, the pathological findings of the aorta in this case did not allow for a definitive diagnosis of IgG4-related inflammatory abdominal aneurysm or IgG4-related periarteritis. It is difficult to confirm whether IgG4 is associated with recurrent aortic dissection in this case, and further accumulation of cases is desired. In conclusion, commonly used enzymatic methods of creatinine measurement can lead to pseudohypercreatininemia. We think that pseudohypercreatininemia should be suspected when hypercreatininemia is present in the absence of common renal insufficiency symptoms, such as hyperkalemia, azotemia, and oliguria. When this disease is suspected, we consider it necessary to perform tests to determine the presence of drugs or abnormal proteins that may have caused this disease. We found it useful to measure cystatin C and inulin clearance, as well as using an enzymatic assay kit to diagnose pseudohypercreatininemia.

An assessment of serum magnesium levels in critically ill patients: A prospective observational study

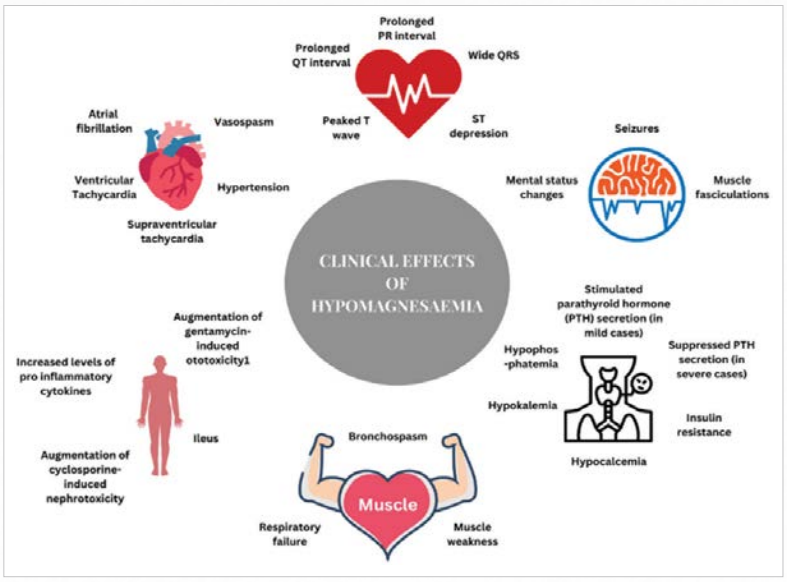
Deepak S. Laddhad, Vinayak Hingane, Tushar Ramrao Patil, Dhruv Deepak Laddhad1, Aishwarya Dhruv Laddhad1, Shantanu Deepak Laddhad2

DR DEEPAK S. LADDHAD
Laddhad Hospital, Buldhana



Background: Magnesium is an important element in the human body that is involved in forming adenosine triphosphate (ATP). It is also the second most abundant intracellular element after potassium. The total magnesium level of an average built adult is 24 g or 1000 mmol. Magnesium depletion or hypomagnesemia has been described as the most underdiagnosed yet critical abnormality in clinical practice.

A specific magnesium level is essential to be maintained to ensure appropriate neuromuscular excitability and cardiac function; an increase or decrease in its levels usually leads to critical abnormality. Hypomagnesemia in critically ill patients has many potential ramifications and is found to be an important factor in hindering their recovery. Thus, the study aimed to assess the serum magnesium levels in critically ill participants and explore its effect on their condition.



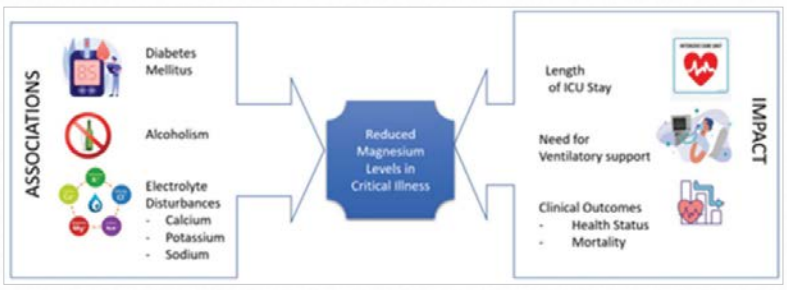
Clinical implications of magnesium deficiency

Methods: A prospective observational study was conducted for 21 months, from February 2019 to October 2020, among all critically ill participants admitted to the medical intensive care unit (ICU) of a tertiary care hospital. The Acute Physiology and Chronic Health Evaluation II score questionnaire was used to determine the severity of their condition and blood samples were collected within 24 h of their ICU admission for analysis.

Results: One hundred participants were enrolled, of which 40% were between the age group of 46 and 65 years and 71% were males. Among all participants with hypomagnesemia, 52% were diabetic, 19% had a history of alcohol use disorder and 27% had normal calcium and potassium levels. Hypomagnesemia significantly correlated with a longer duration of ICU stay among participants.

Discussion: In various similar studies conducted on critically ill patients worldwide, the average prevalence of hypomagnesemia was between 14% and 70%. Few studies have measured red blood cell (RBC) magnesium instead of serum magnesium as it would be a better index of intracellular magnesium than serum magnesium.

Hypocalcemia is another common electrolyte abnormality associated with hypomagnesemia, where the mechanism involves defects in the synthesis and release of parathyroid hormone (PTH) as well as the end organ resistance to PTH. Our study also showed the prevalence of hypocalcemia to be 20% in hypomagnesemic patients.



Association of low-magnesium levels in critical illness

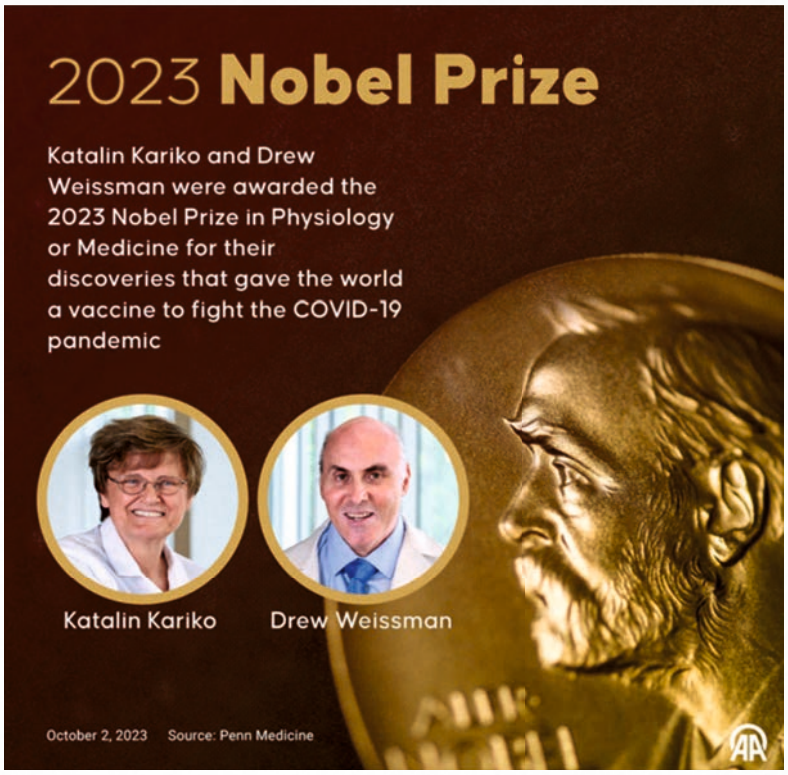
Conclusion: A significant correlation was observed between hypomagnesemia and increased ICU length of stay and mortality but not the duration of mechanical ventilation. Monitoring and appropriate supplementation of serum magnesium is recommended to limit further comorbidity and mortality in the critical care setting.

Laddhad, et al.: Magnesium levels in critically ill patient

NOBEL PRIZE AWARDED TO COVID VACCINE PIONEERS

The technology was experimental before the pandemic, but has now been given to millions of people around the world to protect them against serious Covid-19.

The same mRNA technology is now being researched for other diseases, including cancer.



The Nobel Prize committee said: The laureates contributed to the unprecedented rate of vaccine development during one of the greatest threats to human health in modern times.

Vaccines train the immune system to recognise and fight threats such as viruses or bacteria.

Traditional vaccine technology has been based on dead or weakened versions of the original virus or bacterium - or by using fragments of the infectious agent.

In contrast, messenger ribonucleic acid (mRNA) vaccines use a completely differently approach.

During the Covid pandemic, the Moderna and Pfizer/BioNTech vaccines were both based on mRNA technology.

Professor Kariko and Professor Weissman met in the early 1990s when they were working at the University of Pennsylvania, in the United States, when their interest in mRNA was seen as a scientific backwater.

I would go to meetings and present what I was working on, and people would look at me and say: Well, that's very nice, but why don't you do something worthwhile with your time mRNA will never work. But Katie and I kept pushing, said Professor Wiseman.

Asked about how the pair first reacted to hearing the news that they had won the prize, Professor Kaliko said she thought it was "just a joke" initially.

In a similar vein, Professor Weissman said: I was you know, sort of overjoyed and then disbelief, and a little bit suspecting that there was some anti-vaxxer playing a prank on us.

But when we saw the announcement, we knew it was real and there was just a fantastic feeling.

An mRNA Covid vaccine contains the genetic instructions for building one component - a protein - from the coronavirus.

When this is injected into the body, our cells start producing lots of the viral protein.

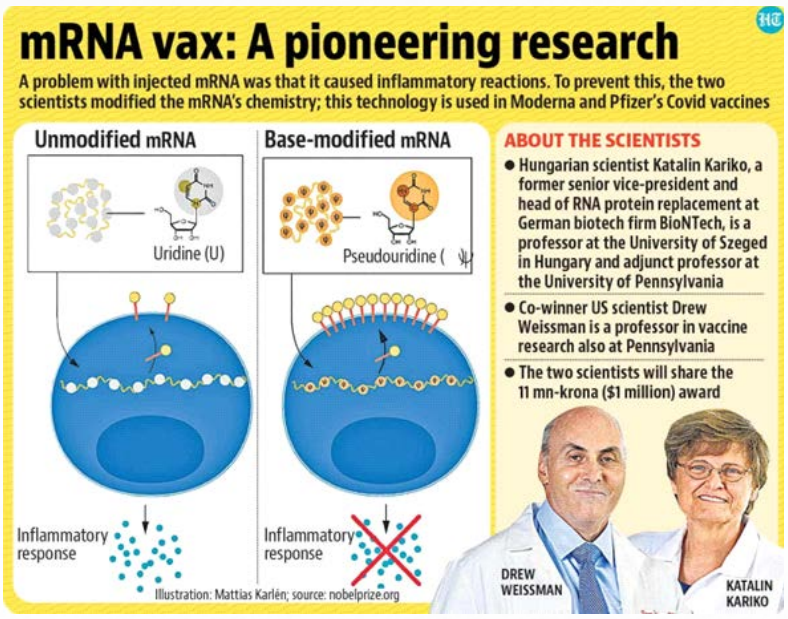
The immune system recognises these as foreign so it attacks and has learned how to fight the virus and therefore has a head start when future infections occur.

The big idea behind the technology is that you can rapidly develop a vaccine against almost any-thing-as long as you know the right genetic instructions to use.

This makes it far faster and more flexible than traditional approaches to vaccine development.

There are even experimental approaches using the technology that are teaching patients' bodies how to fight their own cancers.

Scientists analyse a patient's tumour, look for abnormal proteins being produced by the cancer that are not in healthy tissue and develop a vaccine to target those and inject that into the patient.



Profs Kariko and Weissman made the crucial breakthroughs that made mRNA vaccines happen.

The principle taps into normal human biology. RNA's role in our body is to convert the instructions that are locked away in our genetic code, or DNA, into the proteins that our body is built from.

However, there were challenges. But by refining the technology, the researchers were able to pro-duce large amounts of the intended protein without causing dangerous levels of inflammation that had been seen in animal experiments.

This paved the way for developing the vaccine technology for use in people.

HEALTHY LIVING

KNOW DIABETES NO DIABETES: Dispelling Myths and Embracing a Healthy Lifestyle

DR SUNIL BANSAL
MD, FFIACM, FICP
Physician Diabetologist
Director Cardio-Diabetes Centre, Agra

Elliott P. Joslin, the first recognized Diabetologist once said that "The person who knows more about his Disease lives more".

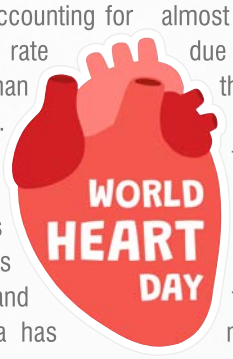
Presently the Tsunami of diabetes is hitting the whole world and the intensity is likely to intensify in coming decades. Also, the major population at target, is the low and Lower middle class and the younger generation. The cost of treatment is escalating and distorting the budgets of the Countries. One of the reasons of this pandemic is the unawareness in the society and the search for magic cure and remedies. This has given rise to lots of myths and beliefs which are not supported by the modern science and leads to delay in diagnosis and control of the disease and ultimately to complications.

- Half of the patients who develop diabetes do not have symptoms initially, therefore if you are at a high risk get your blood sugars done annually otherwise once in three years after the age of 30.
- Losing weight and decreasing energy level despite good appetite are important early features in diabetes.
- Fungal infection (white patches) around glans penis and repeated vaginal discharge and itching in an otherwise healthy adult may be the first presenting feature in diabetes.
- Traditionally described symptoms of increase urination and thirst are later on appeared when blood sugar has risen to very high levels
- Though family history of diabetes is present in half of the cases its absence does not rule out your possibility of getting diabetes.
- Eating lot of sweets and sugars are not necessary for you to develop diabetes. It is a multi factorial disease and excess of sweets only accelerate the age of onset of the disease.
- Obesity is the mother of all diseases and obese persons are at a very high risk of getting diabetes specially if you are also having a sedentary life.
- A child born to a Diabetic mother will not be diabetic since birth, though he may develop obesity and diabetes once he grows adult, which is preventable by healthy life style.
- Alcohol and tobacco do not cause diabetes by themselves but in diabetics they definitely increase the chances of complications such as heart disease, stroke and foot amputation.
- Diabetics can lead a normal marriage life and have children but try to complete your family early because with advancing age the complications of diabetes may interfere.
- Diet, exercise and healthy lifestyle measures are essential for treatment but only small percentage of cases of diabetes can be controlled exclusively by these measures, that to in the initial months. Most of the patients will ultimately require drugs.
- Oral medications do not cause kidney damage in the long run but is the long-standing uncontrolled diabetes which causes heart and kidney damage.
- Dietary substitutes such as Karela (Bitter gourd), Neem, Methi (Fenugreek) etc. are advantageous but are only adjuvants to medication and should be taken as food supplements similar to other such food such as as vegetables, whole grains, fruits and fat free dairy products.
- Fruits, rice and potatoes can be taken in moderation and are not forbidden specially if your diabetes is under control.
- There are side effects of all medicines but most of them are mild and are conveyed to the patients and at times some of the medication has to be replaced.
- Insulin is the natural hormone which is deficient in diabetes and may have to administered in some patients. It is neither addictive nor is the last resort as is highly prevalent in the society.
- Complications of diabetes do occur with time and their early detection needs regular investigations Even if your sugar has been normal you still can develop these complications.
- Quitting tobacco will help even if you been smoking for long period, similarly reducing alcohol will always benefit you and your diabetes.
- Erectile problems are very common in Diabetes. Do not hesitate to discuss with your Physician.
- Remember 80 % of Diabetes is a preventable by Healthy Lifestyle.

BREAKING THE SILENCE
Empowering Women Against Heart Disease in India

It is a common misconception that heart disease is a "man's problem". The focus of research, prevention, and treatment efforts also has primarily been on men. Studies and data from recent years have revealed that women in India are also at significant risk of heart disease. Heart disease is increasingly becoming a leading cause of death among women in India. So, it is time to shed light on this silent threat.

In India, heart disease is responsible for approximately 25% of all deaths, and women account for a significant portion of these deaths. According to the Global Burden of Disease Study, heart disease is the leading cause of death among women in India, accounting for almost 18% of all female deaths. Shockingly, the mortality rate due to heart disease among Indian women is higher than that of breast cancer and other cancers combined. According to studies published in the Journal of the American College of Cardiology, the prevalence of coronary artery disease in Indian women ranges from 3% to 13% depending on the age group and has increased by almost 300% over the past two decades and the prevalence of heart failure in women in India has more than doubled from



1.1% in 2000 to 2.6% in 2015. The mean age of heart attack in Indian women is 59 years, which is much lower than the average age of heart attack in women in developed countries. These data highlight the urgent need to raise awareness about heart disease in women and address the unique risk factors that affect them.

The prevalence of diabetes in particular, appears to be higher in Indian women compared to women in other countries (approximately 12% versus the global average of 9% according to the Demographic and Health Surveys Program and International Diabetes Federation), which is one of the high prevalence risk factor together with hypertension and dyslipidemia.

Additionally, hormonal changes during pregnancy and menopause can also impact a woman's cardiovascular health. Conditions like diabetes during pregnancy (gestational diabetes), and hypertensive disorders during pregnancy (preeclampsia and gestational hypertension) can increase the risk of heart disease. Several studies have reported that compared to women from other countries, Indian women have a higher prevalence of gestational diabetes (17% versus 6-9% in the United States), preeclampsia (6-7% versus 4-5%), and gestational hypertension (10-11% versus 5-6%). The risk of heart disease in women increases significantly after menopause, and menopause at a young age is an additional risk factor for heart disease.

Another challenge in addressing heart disease in women in India is the lack of awareness and knowledge about the condition. Heart disease symptoms in women can differ from those in men, and women often experience subtle or atypical symptoms, such as tiredness, dizziness, nausea, acidity, stomach upset, or shortness of breath, which may be easily overlooked or attributed to other causes. This can lead to delayed diagnosis and treatment, resulting in poorer outcomes. Moreover, societal norms and cultural beliefs may discourage women from seeking timely medical attention or discussing their health concerns openly, leading to underdiagnosis and undertreatment of heart disease in women. Women are often the caregiver for their families, and ignore their own health concerns in order to take care of others. Studies have shown that women are more likely to have delayed presentation of heart attacks, experience more delay in receiving the correct diagnosis, receive less guideline-recommended treatment, and consequently have poorer outcomes following a heart attack.

To address the growing issue of heart disease among women in India, a multi-faceted approach is needed.First and foremost, there is a need to raise awareness about heart disease and its risk factors among women, their families, and healthcare providers. Women should be educated about the importance of maintaining a healthy lifestyle, including regular physical activity, healthy eating habits, avoiding tobacco use and exposure to secondhand smoke, and management of stress. Access to healthcare services can be achieved through initiatives such as mobile health clinics, community health programs, and insurance schemes. Screening and aggressive management of risk factors such as high blood pressure, high cholesterol, diabetes, and obesity are needed, particularly in those with a family history of heart disease. Collecting gender-specific data on heart disease and its risk factors among women in India is crucial to understand the burden, trends, and challenges associated with heart disease in women, which can guide evidence-based interventions and policies.

CLINICAL ECG CONUNDRUMS

Are You Up for the Challenge?

Dear Colleagues,

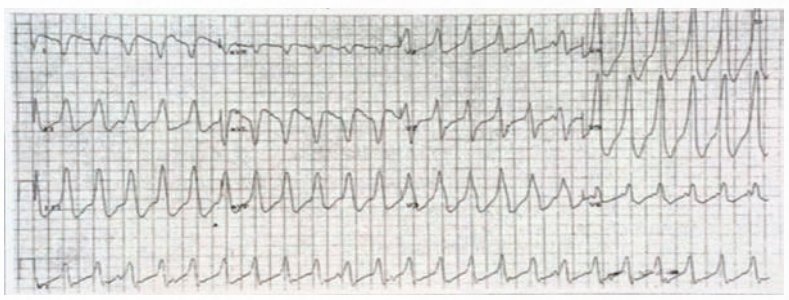
Despite a Vast increase in the availability of sophisticated and costly medical instruments, ECG still plays an important role in diagnosis, management and follow-up of many Patients. Here I will be presenting for your interest and interaction, some fascinating and at times brain coaxing ECGs.

Answers to these ECGs will be found somewhere in this issue of the Newsletter itself.

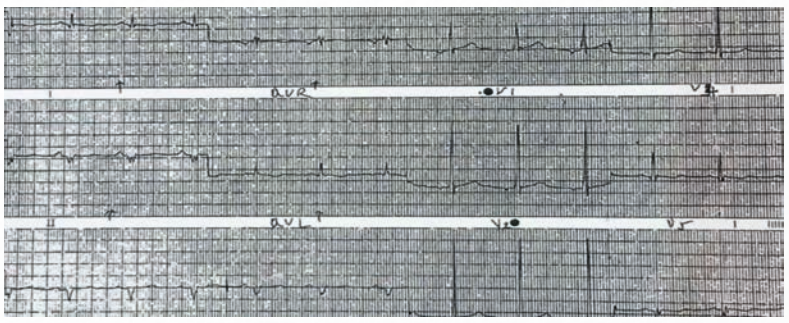
Wishing you a happy and fruitful interaction with these ECGs.

PROF. DR R S SHARMA
M. D. (Medicine)
DM. Cardiology (AIIMS Delhi)
Consultant Cardiologist
S S Medical College Jabalpur, MP

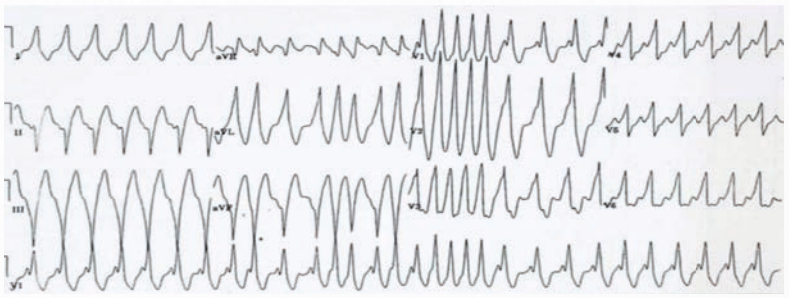
ECG NO. 1



ECG NO. 2



ECG NO. 3



Answers to the ECGs

1. ECG shows a rapid wide QRS Complex rhythm - An RS pattern in V1, is Consistent with ventricular Tachycardia. AV dissociation is obvious- confirming the diagnosis of ventricular Tachycardia. Needed Synchronised Cardioversion.
2. Same patient after Cardioversion - old infero posterior myocardial infarction with Q in Inferior leads and prominent R and upright T in V1.
3. EGG shows a very rapid and irregular, wide QRS Complex rhythm.

This triad of findings is associated with Atrial Fibrillation developing in a case of WPW syndrome. Accessory Pathway allows large number of atrial impulses to go to ventricle directly bypassing AV node which has a much limited ability to transmit impulses, hence this patient has a serious Problem because in AF the impulse reaching in large numbers from atrium to ventricle via accessing pathway can precipitate ventricular fibrillation. This patient needs Na+ channel blocker to slow conduction in the accessory pathway. Later ablation procedure should be used to ablate accessory pathway

CPR Training for Students: Empowering the Next Generation

In a bid to empower students with life-saving skills, **Aprica Healthcare Ltd** took a commendable initiative by conducting CPR and AED training sessions at Anand Niketan School, Ahmedabad. This invaluable training equipped students with the knowledge and skills necessary to respond effectively in emergency situations, potentially saving lives.

Hands-On Experience: Our training incorporates practical learning using manikins, ensuring that students gain hands-on experience and confidence in CPR and AED procedures.

Educational Videos: We use informative and engaging videos to complement practical training, making the learning experience enjoyable and memorable.

Teamwork: We encourage teamwork and communication among students by organizing group activities, reinforcing their skills and fostering a sense of responsibility.

The initiative underscores the importance of providing comprehensive education that extends beyond traditional classroom boundaries, preparing students to be responsible and proactive members of society.

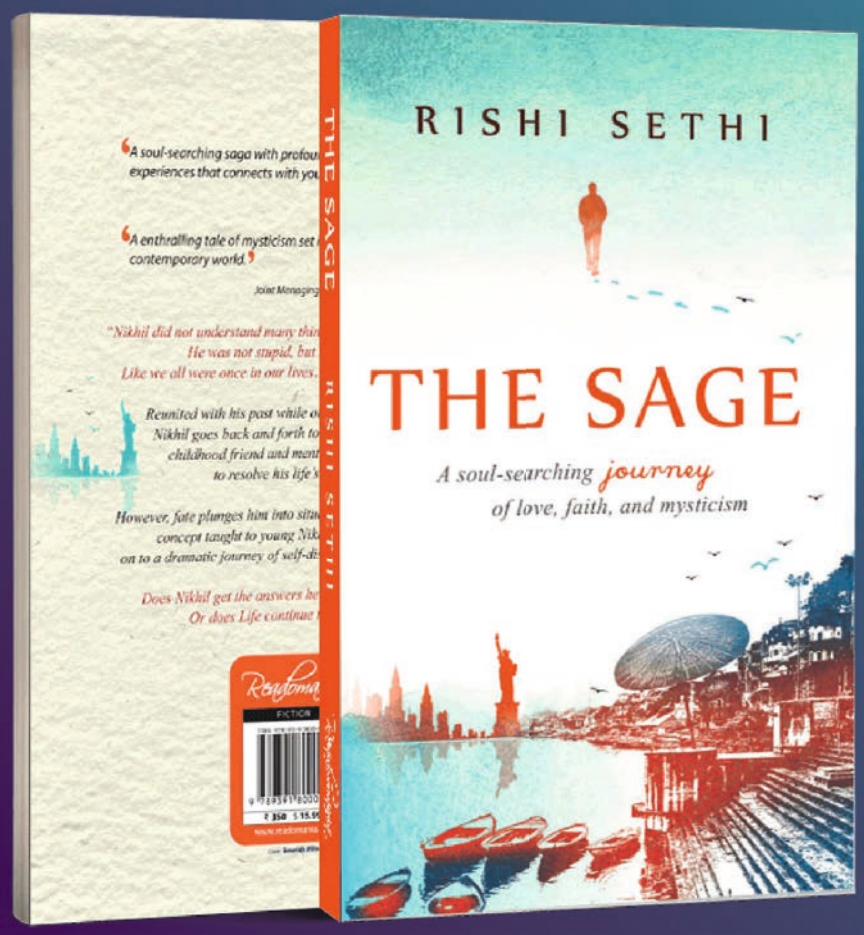
Are you also interested in providing CPR training to school and college students?

Then contact Aprica Healthcare Ltd today at **(079) 4008 4035** or mail us at **info@aprican.com**

We're dedicated to creating a generation of responsible individuals equipped to respond effectively in emergency situations. Join us in making a positive impact on your community.

About the Author

Dr Rishi Sethi is an acclaimed Interventional Cardiologist and Professor of Cardiology at King George's Medical University, Lucknow. He also has a passion for Bio-Medical Innovations and is a nominated member of the Senate at IIT Kanpur. He takes his profession very seriously, but when without his apron, he is generally a dreamer.



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