The Weight Loss Revolution

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Weight Loss Drugs and How to Use Them

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with
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JUGGERNAUT BOOKS
C-I-128, First Floor, Sangam Vihar, Near Holi Chowk,
New Delhi 110080, India

First published by Juggernaut Books 2025

10 9 8 7 6 5 4 3 2 1

P-ISBN: 9789353456979
E-ISBN: 9789353453039

Typeset in Adobe Caslon Pro by R. Ajith Kumar, Noida

Printed at Thomson Press India Ltd

To the brave souls who've survived countless diet plans

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Introduction

Obesity is a word that conjures up all kinds of thoughts and images, from the corpulent uncle who is the butt of family jokes, to Santa Claus ho-ho-ho-ing his way to our homes on Christmas, to the comedian Hardy of *Laurel and Hardy* fame. All of these are seemingly happy, jolly people. Scratch the surface, however, and you will often find an unhappy person longing to be thin.

How we perceive 'fatness' or obesity depends on prevailing social norms. In essence, **obesity means the presence of excess body fat that can impair health.** The word 'obesity' is derived from the French *obésité*, which comes from the Latin *obesitas*, which means 'fatness', and *obesus*, which means 'something that's eaten itself fat'. The roots are '*ob*', which means 'over', and '*edere*', which means 'to eat'.

Obesity has existed for thousands of years, but it was rare in ancient times. A famous 25,000-year-old sculpture, the Venus of Willendorf, housed in Vienna, shows the torso of a morbidly obese woman. The ancient Greek

physician Hippocrates mentioned that sudden death was more common in those who were fat rather than lean. Other Greek physicians noted the association of obesity with infertility and irregular menses. Ancient Indian physicians Charaka and Sushruta astutely observed the tendency of sedentary, overweight individuals to develop diabetes and proposed exercise as a solution. (Wherever we say diabetes in this book, we mean type 2, unless otherwise mentioned.)

In the Middle Ages, food was scarce, and obesity was a status symbol of wealth and health. Throughout most of the nineteenth century, carrying an extra 10–20 kg of fat was considered healthy, with the hope that it would help a person cope with an extended illness. Being thin was not considered healthy and attributed to anxiety or disease. Instead of reducing caloric intake, the emphasis was on how to gain weight.

Only in the latter half of the nineteenth century did being fat begin to be regarded as aesthetically unpleasant. Its association with increased mortality was recognized only in the twentieth century. In other words, before it was realized that fat was bad for health, it was considered ugly. This continues to be the case in society today.

In his 1892 textbook *The Principles and Practice of Medicine*, the legendary Sir William Osler attributed obesity to 'overeating, a *vice* which is more prevalent and only a little behind overdrinking in its disastrous effects'.

In recent decades, obesity has become a global epidemic due to shifts in food consumption patterns and is recognized as a chronic disease with numerous adverse health consequences.

While the current understanding of obesity as a disease is accepted by much of the medical fraternity, it is still regarded as a predominantly cosmetic problem by the general community. It surprises me that many of my patients don't realize the association between obesity and diabetes or heart disease. Patients often ask me how they can 'reverse' or get rid of their diabetes. When I tell them that the way to get rid of diabetes is to lose weight, most find it hard to believe. That, for me, is one of the biggest challenges in managing obesity: making people understand that obesity is a health problem and the reason for their diabetes, breathlessness, knee pain, high blood pressure, high bad cholesterol, and so on. Another challenge is explaining that integral to their treatment is lifestyle modification, which is daunting for most and impossible for many.

Over the years, the problem with clinically managing obesity has been the lack of tools. The history of anti-obesity drugs is full of failures. None of the anti-obesity medicines developed before semaglutide and tirzepatide (Ozempic and Mounjaro) have stood the test of time. The older drugs were either not effective enough, or their

side effects were unacceptable. These new drugs are based on glucagon-like peptide-1 (GLP-1), a natural hormone produced in the body in response to food. The drugs mimic the hormone.

Until recently, the only option for the severely obese patient was bariatric surgery. The procedure entails making the stomach smaller or bypassing it altogether. While the benefits of bariatric surgery are well documented and proven, most of my patients do not agree to undergo it for fear of side effects or just the fear of surgery per se.

The extent of weight loss that can be achieved by surgery far exceeds that of any other method – sometimes to the tune of 40–50 kg. I have been a strong advocate of bariatric surgery for appropriately selected patients: those with a body mass index (BMI) over 40 (in some cases, with a BMI over 35). Unfortunately, many patients regain weight even after losing it through bariatric surgery. This happens because the stomach pouch expands again. After bariatric surgery, some patients think they no longer have to care about diet and exercise. That is not the case. If your doctor has told you to drink a lot of fluids after a bariatric surgery, it can't be sugar soda.

Our understanding of how obesity develops has also been limited. We have not been able to move beyond the fundamental axiom that it reflects the imbalance between what goes in (energy consumed through food) and what

comes out (energy spent through physical activity). We haven't made much progress in understanding why, with apparently similar diets, one individual puts on weight while the other does not. Or, when two friends join a slimming programme, why one loses 10 kg, while the other struggles to lose even 2 kg. Or, why some of our 'foodie' friends think and dream about food all the time and cannot curtail their food cravings. It has been said that the decision to lose weight has to be made by the brain, not by the stomach. It is now understood that this may have something to do with the way the brain is wired and how the hormone–brain axis works differently for some people. The success of GLP-1 drugs is helping us understand these mechanisms better.

Before the modern crop of GLP-1 drugs was discovered, we had no way to address this brain defect. **Currently available GLP-1 drugs produce weight loss between 10 and 20 per cent of total body weight, which is immensely beneficial for health. Future drugs from the same stable may cause even greater weight loss.** These drugs fill a huge void that existed in the field of obesity management.

These days, I often get patients who have regained weight after bariatric surgery and now want GLP-1 drugs. There is no harm in using GLP-1 drugs for such patients. A patient who needs to lose 60 kg may first lose some of it through GLP-1 drugs, and the rest through bariatric

surgery. This shall also make the surgery easier. A big advantage of GLP-1 drugs over bariatric surgery is the greater surety that the lost weight won't return *until the drug is continued*.

The best way to tackle obesity is to prevent it from developing in the first place. This can only be done by a judicious combination of a healthy diet, regular exercise, adequate sleep and low stress. In India, about half of the population is overweight or obese, despite a high simultaneous prevalence of malnutrition – a double whammy. As economic growth continues, food becomes more abundant, and fast or junk food permeates every nook and corner of India; obesity rates can only rocket skywards.

A study published in *The Lancet* estimates that a third of India's population will be overweight or obese by 2050. So much so that, in February of 2025, Prime Minister Narendra Modi highlighted the issue of rising obesity in India and suggested that people reduce their consumption of edible oil.

The cost of unhealthy 'junk' food is always lower than healthy food. Sweetened beverages cost less than milk; candy costs less than fruits. This drives people further towards unhealthy choices. To expect that medication can be a major method to combat the *public health* problem of obesity and diabetes in India is unrealistic. What these drugs do is provide a *medical* means to treat the disease,

something that did not exist so far. They can provide relief to millions who are suffering the ill effects of obesity and prevent many from developing them.

When a drug captures the imagination of the public, like GLP-1 drugs definitely have, it moves from medical conferences to daily news headlines, with everyone forming their own opinion. Misinformation and half-baked information abound in the media. I have not read so much about any medical issue, other than Covid-19, in the media. Social media takes this to another level. There are some who believe that GLP-1 drugs are the panacea for all chronic diseases and that everybody should be taking them. Then there are others who feel that they should not be taken by anyone, even those suffering from obesity and its comorbidities. They say these drugs are very 'new', we do not have enough experience and they have not been studied well enough yet. Some others are terrified of side effects, both known and unknown.

The exaggeration of side effects is a big challenge. There is a risk of side effects with every medication we use. To say that a drug *can* have side effects is very different from saying that it *will*. This is true of most decisions in life. When we buy a new car, we check all the safety measures, yet it does not guarantee that we will not have an accident. We need to know how common a side effect is, and which patients are more likely to experience it.

Using any drug is always a benefit versus risk calculation. We forget that a commonly used non-prescription drug like aspirin can cause bleeding, which can even be fatal. Even low-dose aspirin, used commonly to reduce the risk of heart attacks, can cause brain haemorrhages in two out of every 1,000 people who take it. It is a known hazard, and it doesn't make newspaper headlines. The benefits of aspirin far outweigh the risks for people who have had a heart attack or are at high risk of getting one. Yet, the risks may exceed the benefits in those who are over seventy-five or are healthy. All individuals are not the same.

Choosing the right candidate, the right dose and appropriate medical supervision are essential. This will ensure maximal efficacy and minimize the risk of adverse effects. I am of the belief that we should repose our faith in our doctor. Yes, we should ask them all about side effects before embarking on a new treatment, but ultimately, it is the doctor who will have the knowledge, experience and perspective to guide you.

This book is meant to help the general public understand GLP-1 drugs from the right perspective, drawing on the latest research and clinical outcomes. It is not intended as individual medical advice.

With GLP-1 drugs, minor side effects are common and manageable, but major side effects are exceedingly rare. Those who worry excessively about the side effects

need to keep in mind the risks of obesity and uncontrolled diabetes, which are powerfully and consistently reduced by these drugs. Novel and sometimes unexpected benefits of GLP-1 drugs are being reported every day. It is, however, important for physicians to keep their antennae up and report all minor and major side effects. This process, known as pharmacovigilance, is a public health responsibility.

Undoubtedly, GLP-1 medications are the biggest drug discovery in recent times. They could considerably improve the prevention and management of several non-communicable diseases. Their discovery has unlocked new pathways and approaches to tackle disease. The ability of some newer GLP-1 drugs to act via additional pathways, like glucose-dependent insulinotropic polypeptide (GIP) and glucagon, opens up exciting possibilities. In the future, we may see greater efficacy, specific organ-targeted effects, reduction of unpleasant side effects and easier oral administration schedules. The speed of research in this field is astounding. New drugs are expected almost every year for the next few years. We are living in the GLP-1 decade!

One of the biggest challenges to the widespread adoption of GLP-1 medications is their high cost, which puts them out of reach for most, especially in India. The success of these drugs has significantly impacted the economy of the countries where they originated – Denmark and the USA. The good news is that the cost is expected

to decline significantly when new molecules emerge and the patents on some of the earlier ones, like semaglutide, expire. I expect that in 2026, the price of GLP-1 drugs will crash in India.

Another issue with these drugs has been a shortage of supplies. The innovator companies were possibly caught off guard by the enormous demand for these drugs, and there was a global shortage for an extended period of time. I understand that it has been largely resolved. Going off-patent will allow generic manufacturers to enter the fray, and I hope drug shortages will be a thing of the past.

In this book, I will try to answer many of the questions that have been bothering you. Many friends are taking these drugs, should I try them too? Can I use them to lose just 2 kg for my daughter's wedding so that I can fit into my old dress again? Am I putting myself at risk by using these new drugs to control my diabetes? Will they damage my organs? The questions that I get asked in the clinic and on the phone on a daily basis are all answered here.

1

It Started with a Lizard

I remember giving talks in the early 2000s about the dream of every endocrinologist: an anti-diabetes drug that would control blood sugar without lowering it below normal levels and cause weight loss instead of weight gain.

For persons with type 2 diabetes, medicines are needed to bring down their blood sugar levels, but the drugs can sometimes cause them to plummet, a condition called hypoglycaemia. Symptoms of hypoglycaemia include trembling, sweating, sudden hunger, fatigue, anxiety, confusion, dizziness and irritability. If not addressed immediately with food, it can be dangerous.

Some diabetes drugs cause weight gain. The increasing weight worsens the diabetes, requiring more medication. This becomes a vicious cycle.

The dream of having a drug that caused weight loss and no hypoglycaemia came true one day. That day wasn't when

Ozempic was approved for treating diabetes. It was not the first 'weight-loss' drug, and it is not the last. No doubt very effective, it has the distinction of becoming a household name, a by-word for all such medications we call GLP-1 drugs. However, the first such drug was approved in 2005. It was called exenatide, sold under the brand name Byetta.

The story begins with a lizard. This is no ordinary lizard. It is one of a few lizard varieties that are venomous. It can be up to two feet long and weigh about 2 kg. Found mostly in south-western United States (US), it is a slow-moving, colourful desert reptile that spends most of its time hidden in burrows. This lizard eats often only in spring, and can eat meals as large as one-third of its body weight. Then it stores fat in its tail and doesn't need to eat for months. It is called the Gila monster.

In the 1970s, Dr John Eng was studying the venom of the Gila monster. He found that it had a peptide (chain of amino acids) called exendin-4, which was similar to the hormone GLP-1 found in human intestines. Both hormones were produced in response to food and helped release insulin, but the difference was that the Gila monster's exendin-4 lasted much longer in the blood than the human GLP-1.

All of us produce GLP-1 as a response to eating food, but it disappears in no time. It has a 'half-life' of just two minutes, meaning that half of GLP-1 produced in our

bodies in response to food is degraded within two minutes. This is because of an enzyme found throughout the human body called DPP-4, which breaks down GLP-1.

The earliest trials gave a continuous infusion of GLP-1 to test if and how it was affecting sugar levels and appetite. That, obviously, was not practically feasible. Hence, the challenge in making a GLP-1-like drug was to prolong its action in the body.

If scientists could use what they learnt from the Gila monster's venomous saliva to create a drug that could stay longer than the natural GLP-1 in the human body, they could make the pancreas of patients with diabetes produce more natural insulin, which would manage their blood sugar levels without increasing weight.

In 1987, Amylin Pharmaceuticals and Eli Lilly and Company teamed up to try and make such a drug possible. After years of research and development, they came up with exenatide – a synthetic version of exendin-4, the peptide found in the Gila monster. It was only in 2005 that exenatide was approved for public use by the US Food and Drug Administration (US FDA), and sold under the brand name Byetta, becoming the first GLP-1 drug. By 2007, I was prescribing it to my patients in India.

Exenatide enhances natural insulin production only when blood sugar is high, thus removing the risk of hypoglycaemia. It suppresses glucagon, a hormone that

raises blood sugar. It slows gastric emptying, making food move slowly in the stomach and thus letting the patient feel full for longer. It also reduces appetite, thus helping with weight loss. It has to be injected subcutaneously (under the skin) twice a day, an hour before breakfast and dinner each.

Over the years, my colleagues and I have published many research papers on GLP-1 drugs by measuring their impact on our patients in India. In a paper published in 2013, we observed that our patients with diabetes lost 4–5 kg weight after taking exenatide (Byetta) for six months. It also helped in sugar control. However, Byetta's gastrointestinal side effects like nausea and vomiting were quite harsh, and it had to be injected twice a day, limiting its adoption.

Sadly, the Gila monster is at risk of extinction, affected by climate change and drought.

While Byetta was a product of learning from its venom, scientists were also working on a parallel track to make a drug that would entirely mimic the human GLP-1 and stay longer in the bloodstream.

Five years later, in 2010, came liraglutide, first sold under the brand name Victoza, which had to be injected only once a day. This was an important landmark since it was the first GLP-1 drug to be entirely based on the human hormone. It produced good results in controlling

sugar levels. In 2015, liraglutide became the first GLP-1 drug in the US to be approved specifically for obesity, under the brand name Saxenda. Some were able to lose 8–10 per cent of their total body weight with this drug.

In 2014, there was albiglutide (Tanzeum), followed by dulaglutide (Trulicity). Trulicity had to be taken only once a week, marking a major advance in enhancing the convenience of GLP-1 treatment. This was possible because scientists figured out how to make the drug last longer in the bloodstream, extending its half-life. The drug rapidly gained widespread popularity because of its ease of use. Meanwhile, scientists had also succeeded in making a long-acting (weekly) version of Byetta.

Most of these older drugs are still in use because they either serve a specific purpose for patients with diabetes, such as when combined with insulin in the same injection pen, or are cheap and easily available now that their patents have expired.

In December 2017 came semaglutide, sold under the brand name Ozempic. This was a major turning point since it demonstrated an average weight loss of 15 per cent of total body weight for patients, and up to 20 per cent in some lucky ones.

Converting the injectable drug into an oral pill was not easy. In 2019, Novo Nordisk got approval for semaglutide in pill form, under the brand name Rybelsus. This is the

first and – so far – only GLP-1 pill. It has been approved and available in India since 2022.

In 2022, tirzepatide was approved by the US FDA under the brand name Mounjaro. It was launched in India in March 2025. Tirzepatide mimics the action of not one but two hormones: GLP-1 and GIP. Patients can lose up to 22.5 per cent of their total body weight with this drug.

To understand the world of weight-loss drugs, it is important to appreciate this history. It has been twenty years now since the first one, Byetta, hit the market.

The Discovery of GLP-1

Hormones are messengers in the body, carrying a specific message to the brain and other organs. Most people reading this book would have heard of some of the well-known hormones such as testosterone, oestrogen, cortisol, insulin and melatonin. There are a large number of hormones in the human body, and many more are still being discovered. When I was a postgraduate student of endocrinology at the All India Institute of Medical Sciences (AIIMS) in Delhi in the 1980s, it used to take a month for a hormone blood test to come in, if at all. Today, patients are angsty if they don't get it the same day. The study of hormones is called endocrinology. A doctor trained in endocrinology is an endocrinologist.

In 1964, scientists researching diabetes found something counterintuitive: glucose given orally raised insulin levels in the human body more than glucose given intravenously. This meant that something in the stomach makes the pancreas produce more insulin and helps break down food. They concluded that there are some hormones found in the gut that increase insulin levels. It was not known what these hormones were or how many in number. They were named incretin hormones – incretin means intestinal hormones that increase insulin secretion.

The first incretin hormone to be identified was GIP by John E. Brown in 1970. However, it was not found to be very effective when used as a drug for diabetes. GIP initially stood for gastric inhibitory polypeptide but was later renamed glucose-dependent insulinotropic polypeptide.

I was in training as an endocrinologist, when, in 1986, the discovery of GLP-1 was announced. It was found to be a key hormone responsible for regulating blood sugar in humans. I remember participating in a departmental discussion on the findings in the journal club. The importance of this new discovery was hotly debated.

The discovery took place at the Massachusetts General Hospital in Boston. Yugoslavia-born Svetlana Mojsov, a molecular biologist, began to delve into the mysteries of GLP-1 after looking at glucagon, the sugar-raising hormone. In a different lab in the same research hospital,

endocrinologist Dr Joel Francis Habener and his team had also been looking at glucagon and found a stretch of amino acids that resembled glucagon but was different. Due to the structural similarity, they named it glucagon-like peptide-1 or GLP-1.

Mojsov hypothesized that a particular string of amino acids, 7-31, was GLP-1. She then began to test if it was present in the intestines as suspected. Until then, there was only one known incretin, GIP, and it had failed to stimulate much insulin in trials. Mojsov isolated GLP-1 in glass vials and injected its antibodies into a rabbit to test the response.

Dr Habener began to study the biology of GLP-1 with a new colleague, Dr Daniel J. Drucker. They approached Dr Mojsov to collaborate on GLP-1. Soon, the trio was able to establish its presence in rat intestines in a landmark paper. Then they went for the next step, looking to see if increasing GLP-1 levels corresponded with increase in insulin. 'It was a beautiful experiment,' she would later say about how the rat pancreas responded with a proportionate increase in insulin to the GLP-1 injections. The team then went on to successfully do the experiment in humans.

It often happens in science that two different teams make the same discovery or invention around the same time in different research laboratories. Dr Jens Juul Holst and his team in Copenhagen published the same research on

GLP-1 and its insulin-stimulating powers roughly during the same period (mid-1980s) as did the trio in Boston.

However, the patents were registered solely in Dr Habener's name. As GLP-1 drugs took off, the fame and patent royalties excluded Dr Svetlana Mojsov, who had to hire lawyers to have her name included in the patents.

Dr Daniel Drucker now works as an obesity researcher at the Lunenfeld-Tanenbaum Research Institute, Mount Sinai Hospital, in Toronto. Forty years after he was part of the team that identified and named GLP-1, he continues to research GLP-1 drugs, conduct experiments and publish findings. He is today the most influential scientist in the field.

How GLP-1 Works

After we eat food, GLP-1 tells the pancreas to produce insulin. Insulin lowers blood sugar. Simultaneously, GLP-1 reduces the release of glucagon, a hormone that increases blood sugar. GLP-1 also delays how quickly food leaves the stomach, making you feel full. It also tells the brain to reduce appetite. Thus, GLP-1 is a key spoke in the wheel of metabolism.

GLP-1 'receptors' are found all over the body, including in the brain, heart, gut, small intestine, liver, pancreas, kidneys, lungs and even in the blood vessels and fat tissues.

Think of these receptors as switches. The pathway of the drug begins with reaching the switches and turning them on, which activates a chain of reactions and processes, leading to biological effects.

GLP-1 drugs are 'agonists', synthetically produced molecules that mimic the natural hormone, travelling to GLP-1 receptors and activating them. GLP-1, both the hormone and the drugs that mimic it, stabilizes blood sugar and reduces weight through four actions: by making the person feel full for longer; by making the pancreas produce more natural insulin but only in response to high blood sugar; by reducing the sugar-raising hormone glucagon; and by telling the brain to make the person eat less. This quadruple action produces remarkable results and makes these drugs ideal for people with or without diabetes who have excessive body fat.

It is important to appreciate here that GLP-1 lowers blood sugar only within the normal range, never below it, and only in response to high sugar levels. This glucose-dependent insulin stimulation ensures that the patient is not at risk of hypoglycaemia.

GLP-1 has a sibling, GIP, which mainly helps boost insulin release and does not have any effect on stomach emptying. Scientists found that GIP alone was not producing significant results in trials, and it seemed that GLP-1 was the hormone to target.

What if we targeted both GLP-1 and GIP? Not many scientists shared the hope that adding GIP agonists to GLP-1 would lead to better results. German scientist Matthias Tschöp and his colleagues persisted with the idea that doing so would lead to better results. They were eventually proved right, and that is how we got tirzepatide (Mounjaro), which produces greater weight loss than other GLP-1 drugs.

Patients taking GLP-1 drugs often find they don't feel like eating – their appetite is reduced – and don't think about food all day like they used to. They also find that they feel full very quickly once they start eating – improvement in satiety. Many say that they are now more inclined to eat healthier foods, and some complain they no longer find eating pleasurable. All of this seems to suggest that GLP-1 drugs are acting powerfully on the brain, apart from slowing gastric emptying and stimulating insulin.

It is their actions on the brain that make semaglutide (Ozempic) and tirzepatide (Mounjaro) path-breaking. This has opened up a world of possibilities that is being explored in trials. They could even play a role in alleviating Parkinson's and Alzheimer's disease.

A growing number of trials are showing us that GLP-1 and GIP hormones are beneficial for much more than metabolism. There is increasing evidence that they improve cardiovascular health and reduce the risk of heart attacks

and brain stroke. Many patients taking these drugs say that their desire to drink alcohol has gone down significantly, which is leading to research on the use of GLP-1s for addiction treatment. Large trials are underway to study if they can help wean people off smoking and drugs.

The beneficial effects of GLP-1 drugs on kidney health and fatty liver have now been seen across the world. They have already been approved in the US for some conditions, especially for patients of obesity and diabetes, including sleep apnoea, heart disease and chronic kidney disease. Many of these benefits are, of course, a direct result of weight loss. But there may be more to it. They work directly on the brain and possibly on other organs as well.

A Holistic Approach

When I started as an endocrinologist forty years ago, there were only three tools to address diabetes: insulin, metformin and sulphonylureas. All three were focused on controlling blood sugar. It may surprise you that the concept of tight glucose control with intensive treatment was not established at that time.

In the mid-1990s, a landmark study called the UKPDS, or United Kingdom Prospective Diabetes Study, demonstrated that good glucose control resulted in reduced diabetes complications. The focus moved to better control.

I remember saying often in my talks in the early 2000s that the only thing that matters is controlling blood sugar. How you do it does not matter. That is no longer true.

Today, we have been able to move beyond mere glucose management. With newer drugs like GLP-1 receptor agonists and SGLT2 inhibitors (which throw out excess glucose through urine) we are able to address underlying issues, such as obesity and metabolic health, and thus look at diabetes holistically. These molecules also reduce the risk of long-term complications, such as those involving the heart, kidneys and liver, independent of their actions on blood glucose.

Let's take the example of a patient I saw recently. He was fifty years old, on forty units of insulin a day along with oral medication, yet his HbA1c, a measure of average blood sugar levels in the last two–three months, was 8.5 per cent (the target is to keep it under 7 per cent). He was obese, weighing 90 kg, and his BMI was 31. I prescribed him Rybelsus, the oral form of semaglutide.

The first thing I noticed was the decline in his insulin dosage. I reduced the amount of prescribed insulin, and yet he continued to get attacks of hypoglycaemia (sugar levels below 70 mg/dL). The dosage of oral medications (sulphonylureas) was then reduced. As mentioned earlier, sulphonylureas are popular glucose-lowering drugs that can cause hypoglycaemia. His insulin dosage was

halved in just four weeks. In three months, he was only on metformin and semaglutide. His HbA1c now is 6.5 per cent. He does not get hypoglycaemia any more, and so long as he continues to be on these two medications, there is no risk of it in the future too. The absence of hypoglycaemia means that better control is possible with less risk. Besides this, semaglutide reduces his risk of heart attacks, the progression of his kidney complications, and squeezes out fat from his liver. And he has lost 4 kg too! In other words, this patient is in a much better place now than three months ago.

We have now started prescribing GLP-1 drugs to patients early in the course of diabetes, helping them lose weight and thus increasing the possibility of inducing remission. Metformin is still the frontline drug given to people when they're first diagnosed with diabetes, but I foresee that changing in favour of GLP-1 drugs in the near future. I have many patients who do not need to take metformin any more – just a GLP-1 drug is enough to maintain both their sugar and weight.

Type 2 diabetes is only one of many diseases caused by the meta-disease of obesity. For people who are overweight or obese and are unable to lose weight through lifestyle modification, these drugs offer a way out.

For overweight and obese patients who have pre-diabetes or have other obesity-related health issues, GLP-1

drugs may be useful in not only preventing future diabetes but also reducing their weight. Excess body fat is the driver of most non-communicable diseases that can be prevented with weight loss.

Diabetes reduces the average lifespan by seven to ten years, and excessive body fat accelerates ageing. By making people healthier, GLP-1 drugs could add many years to their lives.

Takeaway

GLP-1 and GIP are natural hormones found in the body that help regulate hunger, satiety, stomach movement and insulin secretion. Drugs that mimic these hormones have been around since 2005. Ozempic and Mounjaro are only the latest drugs in the series. Since the action of natural GLP-1 lasts only a few minutes, the development of GLP-1 drugs has sought to extend the duration of action, and to preferentially enhance weight-loss properties.

PATIENT STORY

AKSHAT RATHEE, 44

Both sets of my grandparents had diabetes and hypertension, so there was always a genetic predisposition for diabetes. I gained weight with age, work stress and a lot of travel.

I was 101 kg. My HbA1c was a worrying 11. Dr Mithal had put me on a total of 11 medicines. I would often forget to take them or not take them when I was supposed to. My compliance rate with these medicines was about 40 per cent. Some of them were giving me terrible side effects. So I went back to Dr Mithal and said that this was not going to work. I travel twenty days a month and have a stressful work life. He asked me to try a GLP-1 drug. Now I take just two pills a day, a daily metformin along with a statin for cholesterol. My sugar and weight are both coming down with a weekly Mounjaro injection. My current weight is 88 kg. I am targeting 82 kg.

I started with Ozempic 0.25 mg and it had no effect on me – no side effects, no weight loss, no sugar control. I increased it to 0.5 mg, which had some impact on my sugar levels but no impact on my weight. Then came 1 mg, which was good for both sugar and weight. Ozempic

2 mg produced further weight loss but had no incremental impact on my blood sugar. It became difficult to get the 2 mg pen, so I switched to Mounjaro.

However, there was a month in between when I was on neither of them. In this one month alone, I gained back 5 kg of the 12 kg I had lost. I started with Mounjaro 2.5 mg, on which I gained another 1 kg weight in a month. The next dose was 5 mg, which gave me some weight loss but very slowly. Dr Mithal then made me jump straight to 10 mg, which has been perfect for me. I'm now losing 1 kg a week on 10 mg. My HbA1c is down to 6.1.

Ozempic made me gassy and irritable; it parched the sides of my tongue and I felt thirstier. With Mounjaro, I face a lot of burping and flatulence only for one day, the day after I take the injection.

From three heavy meals, I am down to eating two light meals, breakfast at 9.30 a.m. and dinner at 7 p.m., and nothing in between. From eating three parathas, I am in a situation where I can barely finish one. I have higher energy levels and my cholesterol levels have also improved.

I have friends who needed to lose weight, and I suggested they take one of these drugs. Some tried but gave up – they wanted the joy of eating three parathas. I want the joy of being healthy for my newborn child.