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A QUARTERLY PUBLICATION OF THE POTTER-RANDALL COUNTY MEDICAL SOCIETY

FALL 2022 | VOL 32 | NO.4

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President's Message: As Fall Approaches...

by Evelyn Sbar, MD, FFAFP, AQH

COVID 3.0...monkeypox....and now polio once again rearing its head. The last few years have not been what I imagined when starting my medical career. I still remember the bright-eyed medical student in awe of technological advances with each passing year, believing that I would see a day where my profession wasn't needed. I truly had hopes that we would conquer cancer and diabetes, dementia and osteoporosis, and that the life expectancy of the average human would be in the hundreds. I also thought we would be living in sky high towers, traveling through transit tubes, and flying autonomous vehicles a la *The Jetsons*. Nature just laughed at these visions and mutated a microbe that sent us into a tailspin.

What I do know is that our role has

changed. It is one thing to face the challenge of a new infectious disease or damage from the American fast food diet, but it is quite another to spend 30 minutes on hold, finally connecting with a non-medical attendant on the other end who wants to challenge my recommendation for hospital admission. We spend a ridiculous amount of time arguing with insurance companies, trying to get our patients the care they deserve. We have been forced to spend more time in front of computers or in meetings than in front of patients. I actually feel that it is harder to practice medicine today than it was 20 years ago. Medical students and resident physicians get less face-to-face time, not only with patients but with their teachers. There are more lectures about quality metrics, RVUs, and contracting and fewer about end-of-

life decision making, community resources and empathy.

I find this new world of medicine challenging and exhausting to navigate. I am thankful, however, that TMA has my back. The concerns that drive many of us towards burnout are on their list, with some of our smartest physicians and support staff leading the charge. Please support the **Texas Medical Association** with whatever resources you can spare – be it your time, your money, or your expertise. As my golden years become visible on the horizon, I sincerely hope that those taking care of me will get the opportunity to see medicine as I remember it, leveraging all the benefits of innovative technology and once again, **patient focused**.

PANHANDLE HEALTH

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Editorial Policy and Information for Authors

Purpose *Panhandle Health* strives to promote the health and welfare of the residents of Amarillo and the Texas Panhandle through the publication of practical informative papers on topics of general interest to most physicians while maintaining editorial integrity and newsworthiness.

Spectrum *The Journal* seeks a wide range of review articles and original observations addressing clinical and non-clinical, social and public health, aspects as they relate to the advancement of the state of health in the Texas Panhandle. Pertinent letters to the editor, news submissions, and obituaries listings are accepted pending editorial review. The Editorial Board accepts or rejects submissions based on merit, appropriateness, and space availability.

Submission process Material should be e-mailed to the editor at prcms@suddenlinkmail.com or mail a hard copy to Cindy Barnard, PRCMS, 1721 Hagy, Amarillo, TX 79106. A recent photograph of the author (optional) and a curriculum vitae or a biographical summary are also to be submitted.

Conflict of Interest Authors must disclose any conflict of interest that may exist in relation to their submissions.

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Obituaries Listings of deceased members of PRCMS with highlights of their contributions are published when adequate information is available.

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Executive Director's Message

by *Cindy Barnard, Executive Director*

Diabetes, “also known as diabetes mellitus, is a group of diseases resulting in too much blood sugar (high blood glucose).” The most common types are Type 2 diabetes, Type 1 diabetes, pre-diabetes, and gestational diabetes.

You'll never guess who has diabetes: yes, your favorite Medical Society Director and author of our magazine's "Message"! Yes, me! I am fairly typical in that I didn't have warning signs, or if I did, they were too mild to be noticeable. The diabetic literature says this is quite typical of Type 2 diabetes (which is what I have). Many diabetic people are unaware of the disease and don't find out they are diabetic until they have problems caused

by the disease. With Type 1, people are more likely to notice the symptoms because they are more severe and occur in as little as a few days or weeks. Early signs of diabetes are hunger, fatigue, and thirst, more frequent urination, dry mouth, itchy skin, and blurred vision. Type 1 diabetes may present with significant weight loss and nausea and vomiting. Symptoms of Type 2 diabetes become more obvious after your glucose has been high for a long time. Yeast infections are common in both men and women as well as cuts that are slow to heal and pain in one's feet. Symptoms of gestational diabetes are practically none during the pregnancy. Hyperglycemia, or high blood sugar, has many symptoms like hypoglycaemia, but

the blood sugar is over 180 milligrams per decilitre (mg/dl). Finally, a diabetic coma is a serious complication in both Type 1 and 2 although far more common in Type 2. Blood sugar becomes too high, and severe dehydration occurs when blood sugar is over 600 mg/dl. Additionally, the patient has a high fever, dry mouth but extreme thirst, confusion, hallucinations, and/or seizures. This complication can lead to death. On that frightening note, let me tell you that it is important to get tested if you are over 45. My own diabetes was discovered with a simple blood test. Hopefully, with early detection, I have avoided nerve damage, heart trouble, and more. The same can be true for you if you spot the condition early. Good luck!



Message from the Potter-Randall County Medical Alliance

by *Tricia Schniederjan, President*

On July 17th, the Medical Society Alliance welcomed our new medical residents to town. We enjoyed a beautiful evening in the coterie suites at the Sod Poodles game. If you weren't able to join us, we look forward to a really fun fall social. Save the date for Thursday evening on October 20th. Any new physicians and spouses in your practice should come too! Invitations will come in the mail. Make sure we have your address. If you haven't received anything from us in a while, please email me and I will make sure you're included. (tschnied@gmail.com)

More information to come soon; I look forward to seeing you all this fall!



Finally, I'm really excited to announce that the Regional Alliance meeting will be held here in Amarillo! Alliance members from all over the state will be in town for this meeting on October 13th and 14th.



Guest Editor's Message:

by William C. Biggs, MD, FACE

When we consider the most impactful achievements of modern medicine, the discovery of insulin in 1921 ranks high on the list. Instantly everyone with type 1 diabetes was spared from a death sentence, and patients with type 2 diabetes had a better life.

And yet, one hundred years after the discovery of insulin, the treatment of diabetes remains imperfect. Patients with diabetes still risk the development of long-term complications such as neuropathy, kidney disease, loss of vision, and premature cardiovascular disease. Living with diabetes is still complicated by the need for watching a diet, exercising, and taking meds on time.

Diabetes treatments are expensive. Insulin was \$8 for a 1000-unit vial when I was a fellow at Joslin Diabetes Center, and now it can run over \$500 a vial. Newer injectable treatments can run \$1200 a month for a single drug. Nationwide, the cost of diabetes in 2017 was \$327 billion. In Texas, where 30% of our citizens are uninsured, diabetes can be both a medical and financial catastrophe.

Providing effective diabetes care that is available and affordable to everyone

requires all hands on deck. The incidence of diabetes has exploded, with 37.3 million Americans afflicted, or 11.3% of the population. There is an insufficient supply of endocrinologists nationwide, and the primary management of diabetes will need to be from family physicians, pediatricians, and internal medicine physicians, assisted by strong support from dietitians, pharmacists, and nurse practitioners.

This issue is devoted to helping everyone understand where contemporary diabetes care stands and providing you with ideas on how to improve the care of your diabetic patients.

The patient experience in Amarillo for diabetes has been significantly better than other areas of the state. Based on data that the Amarillo Legacy Medical ACO gains from participating providers, and data from CMS and commercial insurers, patients in Amarillo have better A1Cs and fewer complications than average. Diabetes related amputations in our area are less than half the incidence of the Rio Grande Valley area. An exception to this trend is for the reported incidence of retinal disease, and there is some evidence that this is from over-reporting rather

than actual disease. We also have worse outcomes in women and children from gestational diabetes.

Amarillo has several assets that most communities don't. The J O Wyatt Clinic, operated by Northwest Texas Healthcare System, has been a shining star for diabetes care for years, providing care for Amarillo residents who otherwise cannot afford care. Heal the City provides amazing diabetes care to people who are unable to access any other source. Texas Tech has traditionally provided great diabetes care from infancy on.

The high cost of diabetes care continues to haunt everyone with diabetes, and, as a prescriber, I spend all day dealing with it. A law firm called me in 2017, asking about online comments I made in 2002 regarding Lantus insulin. I described how Lantus would precipitate if left in a syringe more than a day or two, unlike NPH or Regular. The manufacturer of Lantus claimed that they discovered this precipitation in 2003. This became the basis of a patent extension for Lantus, a \$20 billion per year product, for several more years. I was asked to provide a deposition about my earlier discovery, and it was made a key component of chal-

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lence to the patent. After enduring several depositions, my role was enlarged to become the medical expert witness at trial, and I spent a week in Federal Court in Newark, NJ. The court ruled that new competitors to Lantus could begin selling their products, causing the price of glargine insulin to fall almost 60% overnight. This proves, that whatever you say, online never really goes away.

Given the continued increase in diabetes, and the shrinking endocrinologist workforce, we may need to rethink how diabetes care is provided. Even though Amarillo has done better than average, there is still a lot of work to do. UPMC in Pittsburgh has tested a primary care-based model, where all diabetes care is provided by their primary care physicians. Endocrinologists are assigned to work side by side with groups of PCPs, coaching them on managing their difficult cases. This leverages the impact of the endocrinologists, improves the diabetes care repertoire of the PCPs by improving their skills beyond what is taught in residency, and increases access to high level diabetes care for more patients.

How healthcare is financed influences

how we do our work. It is important for Medicare and commercial payors to consider new ways to better compensate physicians for the cognitive work and the time spent to adequately train patients with chronic disease and to begin payment for collaborative care. I met with Medicare Director Meena Seshamani MD PhD in Baltimore last April, and we discussed health issues specific to Amarillo and well as the challenges delivering diabetes care to a widely dispersed area such as the Panhandle.

I appreciate the work that all the authors have done for this issue, and I want to thank all of them for their contributions.

Reddy Biggs, MD

William C. Biggs, MD FACE has practiced in Amarillo since 1986. He attended UT Southwestern in Dallas, interned at the University of California, San Diego, and completed a residency at New England Deaconess Hospital and Harvard Medical School in Boston, followed by fellowship at Joslin Diabetes Center and Harvard Medical School. He is a past president of the Texas Chapter of the American Association of Clinical Endocrinologists

and is an advisor to the AMA CPT Editorial panel. He is the managing partner of Amarillo Medical Specialists, LLP, and CEO/Medical Director of the Amarillo Legacy Medical Accountable Care Organization. He is married to Emily Archer, MD, and they have four children. Sons Richard and William are attorneys in Amarillo. Sarah Biggs MD practices Allergy and Immunology in Amarillo, and Grace Biggs Steel MD is an Ob/Gyn resident at Baylor Scott & White.

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Austin, TX



Intensive Insulin Treatment

by Susan Wingo, MD

INTRODUCTION

Diabetes mellitus is an enormous problem in the United States, with heavy economic, social, and personal costs. The Diabetes Control and Complications Trial (DCCT), published in 1993, conclusively established a strong correlation between glucose control and the development and progression of microvascular complications – retinopathy, nephropathy, and neuropathy – for patients with Type 1 Diabetes (1). The UK Prospective Diabetes Study (UKPDS), published in 1998, established this correlation for patients with Type 2 Diabetes (2). Even before these studies were published, physicians had been working with patients with diabetes to help them achieve better blood glucose control while minimizing hypoglycemia. This led to the need for more individualized and flexible strategies for dosing insulin, an approach often called Intensive Insulin Therapy (IIT). Actually, this is probably a bit of a misnomer; “Intensive Multifaceted Co-Management of Insulinopenic Diabetes” would better describe what endocrinologists and other physicians and diabetes treatment teams do. But old terms die hard, especially when newer terms are longer and not easily reduced to 3-letter abbreviations. I will continue to use “Intensive insulin Therapy” (IIT) throughout this article to discuss a comprehensive system for an individualized treatment plan with flexible insulin dosing designed to allow patients to adjust insulin for current blood sugars, meal composition, and activity. Components of IIT include education, glucose monitoring, patient-adjusted insulin dosing, meal planning, activity planning, sick day management, frequent contact with staff, and psychological support (3).

MONITORING

One key element of IIT is knowing what one’s blood sugar is. This may be done either with traditional Finger Stick

Blood Glucose checks (FSBG), or with a Continuous Glucose Monitor (CGM). For IIT, FSBGs are typically done at least 3 times a day, before meals, and may be done 7 or even more times a day to check after meals and at bedtime. This is probably the one aspect of diabetes management that patients like least. Fortunately, for many, CGM is available and can eliminate the need for most FSBGs. CGM can show what’s happening in between the dots on a graph from FSBGs, and can also send alerts to patients (or caregivers) when blood glucose levels are outside of a specified range, or are likely to go outside that range in the next 30 minutes. This in turn allows patients to feel more confident about striving for lower blood glucose levels with the assurance that, if they overshoot and cause their glucose to become too low, they will be alerted to this and able to manage the episode while they are still able to do so.

DELIVERY MODELS

Normally, the pancreas releases insulin in two different modes. There is a constant release of a small amount of insulin to allow all the cells of the body to take in glucose to meet their own metabolic requirements, known as basal insulin secretion. This mode of insulin secretion is critical for avoiding Diabetic Ketoacidosis (DKA). The second mode is bolus insulin secretion, in which larger amounts of insulin are released into the bloodstream over a fairly short period of time during and shortly after meals. These boluses drive excess glucose from the blood into liver and muscle cells, where it can be stored as glycogen. Later, liver and muscle cells can break down glycogen, releasing glucose into the bloodstream when needed for muscle activity or to maintain glucose levels in the normal range.

For the purposes of this article, we will focus on patients who make little

to no insulin. This includes all patients with Type 1 Diabetes, some patients with longstanding Type 2 Diabetes, and some patients with histories of chronic pancreatitis or pancreatic surgery. These patients will require insulin dosing to provide both basal and bolus patterns of insulin secretion. In order to achieve glucose targets while minimizing hypoglycemia, most patients will have to take insulin as Multiple Daily Injections (MDI) of two types of insulin with different pharmacokinetics, or with an insulin pump to deliver Continuous Subcutaneous Insulin Infusion (CSII). While other patients who maintain some ability to produce insulin may be able to control glucose levels with once daily injections of a long-acting insulin, or twice daily injections of short and intermediate-acting insulins, neither of these options provides sufficient flexibility or adjustability to meet the needs of patients who do not produce insulin.

MDI generally includes injections of an intermediate- or long-acting insulin 1-2 times a day as well as injections of rapid- or short-acting insulin before each meal, typically 2-3 times a day, for a total of 3-5 daily injections. While intermediate- or long-acting doses are generally the same day-to-day, mealtime doses, or boluses, can be adjusted based on current glucose level, carbohydrate content of the meal, overall meal size, activity shortly before or after the meal, or illness.

CSII provides a pre-programmed amount of rapid-acting insulin continuously to mimic the pancreas’s basal insulin secretion, and boluses of insulin calculated using individualized ratios to provide coverage for meals. CSII allows for different amounts of basal insulin to be delivered at different times of day. One example where this is helpful is “dawn effect” in which many patients need higher rates of insulin for several hours around their usual wake-up time

due to increased levels of the counter-regulatory hormones cortisol, epinephrine, and growth hormone, which decrease the body's response to insulin (or increase resistance to insulin). So, a patient who requires 24 units of basal in a day might receive 1.5 units/hour for 4 hours from 5-9AM (6 units) and 0.9 units/hour for the remaining 20 hours of the day (18 units). Pumps can also temporarily reduce basal insulin during exercise, or increase basal insulin after a steroid shot.

2017 saw the first of a new generation of insulin pumps which use information from CGM and algorithms to adjust the amount of insulin, trying to keep blood sugars at a preset level. These automated systems help many more patients achieve the goal of maintaining glucose levels in the target range of 70-180 over 70% of the time.

INSULINS

Over the last 40 years, many new insulins have become available. In 1983, Humulin, the first human insulin made with recombinant DNA technology, was approved, followed shortly thereafter by Novolin, allowing human insulin to replace bovine and porcine insulins. In the next advance, recombinant DNA technology was used to create a human insulin analogue in which two amino acids were swapped (a lysine and a proline) creating lispro (Humalog), followed a few years later by aspart (NovoLog) (swapping an aspartate with a glutamate). These changes decrease the formation of insulin dimers and hexamers in the subcutaneous tissue commonly seen with regular insulins, allowing the body to absorb and clear the insulin more rapidly. Later, insulin glargine was developed using other amino acid substitutions that cause the insulin to form microcrystals in the subcutaneous tissue, providing a new once-a-day alternative to NPH, Lente, and Ultralente insulins. The chart on this page illustrates the wide range of insulins commonly used today.

LIFESTYLE FACTORS

Any discussion of diabetes management must address the role of diet on blood glucose levels and insulin requirements. Most patients do benefit from con-

Class	Insulin	Brand(s)	Onset	Peak	Duration
Rapid	lispro-aabc	Lyumjev	15 Min	1 Hr	2-4 Hr (1)
	lispro	Humalog, Admelog	10-15 Min	1-3 Hr	3-5 Hr (2)
	aspart	Fiasp	15 Min	1 Hr	2-4 Hr (1)
	aspart	Novolog, ReliOn	10-15 Min	1-3 Hr	3-5 Hr (2)
	glulisine	Apidra	5-15 Min	1-3 Hr	3-5 Hr (2)
	Inhaled R	Afrezza	10-20 Min	12-15 Min	3 Hr (3)
Short	Regular(R)	Humulin	30-60 Min	1-2 Hr	5-8 Hr (2)
	Regular(R)	Novolin, ReliOn	30-60 Min	1-2 Hr	5-8 Hr (2)
Intermediate	NPH(N)	Humulin	1-2 Hr	4-12 Hr	14-24 Hr (2)
	NPH(N)	Novolin, ReliOn	1-2 Hr	4-12 Hr	14-24 Hr (2)
Long	glargine	Lantus, Basilar	3-4 Hr.	None	Up to 24 Hr (2)
		Toupee	6 Hr	None	Up to 36 (2)
	detemir	Levemir	1 Hr	3-14 Hr	Up to 24 (2)
	degludec	Tresiba	1 Hr	None	Up to 42 (2)
Pre-Mixed	70/30	Humulin			
	70/30	Novolin, ReliOn			
	75/25, 50/50	Humalog			
	70/30	Novolog			

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sultation with a dietitian. I have found that having patients bring in an honest food diary for the preceding 3-7 days increases the likelihood of coming out with a plan that is closer to what they're used to eating, and that will be easier for them to adopt. Two different approaches can be used. In one approach, the patient counts the carbohydrate content of their planned meal, and calculates an insulin dose to cover this. A second approach is to ask patients to keep the carbohydrate content for breakfast, lunch, and supper consistent on a daily basis, and to use a set insulin dose to cover this. Patient preference should figure very strongly in deciding which approach to use.

Carbohydrate counting can be done either in grams or exchanges. 1 exchange is equivalent to a 15 g serving of carbohydrates, such as one standard slice of bread, 1 small piece of fruit, ½ cup of mashed potatoes or ⅓ cup of cooked rice or pasta. Counting exchanges can be simpler for patients to understand and adopt. Counting grams is more accurate and is easier to use with foods providing small amounts of carbohydrates. A wide range of smart phone applications are available which allow patients to easily look up carbohydrate grams in many different foods, as well as for many foods offered by chain restaurants.

| continued on page 12

Another consideration is deciding how much carbohydrate the patient should consume daily. My experience is that a low or low-ish carb diet is more helpful for patients who have insulin resistance, defined as a daily insulin requirement exceeding 1 unit of insulin/kilogram, while accurate carbohydrate counting is more important for patients who are more sensitive to insulin. Meals that are high in fat content will result in higher levels of chylomicrons and triglycerides in the bloodstream. These cause cells to become more insulin-resistant and lead to higher insulin doses. Additionally, high fat content foods delay absorption of glucose from a meal, so that a patient may have lower glucose levels during and shortly after a meal, and higher glucose levels several hours later. All carbohydrates are not created equal. Sugars, white flour, and highly processed starches can be quickly digested and absorbed leading to glucose spikes immediately after meals, while more complex and fiber-rich carbohydrates (fresh fruit, whole grains, starchy vegetables, and beans or lentils) are digested and absorbed more slowly, leading to less spiking.

Applying an appropriate Insulin: Carb Ratio (I:C Ratio) should be a starting point for determining a mealtime insulin dose. A common starting point is 1 unit of insulin per 10 grams of carbohydrate in the meal, but the ratio can vary considerably from one person to the other, based on their degree of insulin resistance, and the final ratio will have to be individualized. Other factors that may increase or decrease the insulin dose include recent or planned activity, fat content, and current blood glucose level.

Exercise, or activity, is another factor which can significantly affect the insulin requirement for a given meal. Patients should either strive for a consistent day-to-day activity pattern, or receive education on how to adjust insulin doses for activity. It is important to recognize that, during exercise, muscle and liver cells are breaking down glycogen and releasing glucose into the bloodstream, resulting in temporarily high glucose levels for up to 30 minutes after exercise. For patients

who are insulin resistant, muscle-building exercises can increase muscle mass, creating additional insulin-sensitive tissue, increased capacity to clear glucose from the blood after a meal, and additional glycogen stores to prevent hypoglycemia hours after a meal.

Illness can also affect blood glucose levels and insulin requirements. Often patients are unable to eat as much as they usually do, and when they do eat, select foods with more simple carbohydrates, and less protein or fat. Many illnesses are associated with inflammation, which can increase insulin resistance. These factors can vary significantly from one illness to another, and even day-to-day during a single illness. Therefore, frequent blood sugar checks and flexible guidelines for adjusting insulin are the key to maintaining reasonable blood glucose levels during intercurrent illnesses.

ADVANTAGES AND DISADVANTAGES

Advantages to using IIT include the ability to decrease overall blood sugars while lowering the risk of hypoglycemia. This in turn reduces the risk of developing microvascular complications of diabetes affecting the eyes, the kidneys, and the nerves, as well as reducing the risk for macrovascular disease due to atherosclerosis including heart attack and stroke. An additional benefit is that patients feel empowered when they have the tools to control their diabetes, or as one patient told me, “I feel like I have diabetes now, instead of diabetes having me.”

One disadvantage to using IIT is complexity. Many patients feel heavily burdened by the more frequent blood sugar monitoring, carbohydrate counting, and calculations needed before each dose of insulin. IIT can raise costs; analogue insulins such as Lantus and Humalog cost considerably more than NPH and Regular insulins. Additionally, increased reliance on technology including pumps and continuous sensors can also increase costs. And, while the use of technology and insulins with more physiologic pharmacokinetics can mitigate the risk of hypoglycemia, striving to achieve

lower glucose targets and A1C's will generally still cause occasional episodes of hypoglycemia.

CONCLUSIONS

Intensive Insulin Therapy, or Intensive Multifaceted Co-Management of Insulinopenic Diabetes affords providers and patients with a set of tools to help achieve better glycemic control with a relatively lower risk of hypoglycemia. Implementing IIT requires a significant investment of time in understanding patients' own goals for their diabetes control, the management rationale for improving glucose control, and education. It need not be an all-or-nothing plan, and can be implemented in phases, with priority given to what patients feel they are ready to do. Helping patients achieve their goals and feel like they are controlling their diabetes more effectively is one of the most rewarding aspects of working with patients with diabetes.

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Newest Treatments for Diabetes: 2022

by Cesar Arias, MD

By 2021, one hundred years had passed since the discovery of insulin. It is important to realize that, prior to the discovery of insulin, the average life expectancy for a type 1 diabetic ranged from 18 months to a few years. Insulin transformed this condition from a terminal disease into a chronic one. Life expectancy has been extended and quality of life has been improved for millions of people living with the condition.

The treatment of diabetes has changed profoundly over these hundred years. First, longer-acting insulins (Lantus, NPH) were developed to supplement short-acting regular insulin. Then, oral antidiabetic agents – first the sulfonylureas, then biguanides like metformin, thiazolidinediones like the glitazones, and DPP-4 inhibitors like the gliptins – were added to the mix. Insulin analogs, SGLT2 inhibitors – the list goes on. Now physicians are confronted with a dizzying array of treatment choices for both type 1 and type 2 diabetes. And it seems as though a new drug is being approved every week. So, let’s go over the newest drugs available to treat both type 1 and type 2 diabetes. These new drugs now allow patients to achieve better metabolic control with relatively low risk of hypoglycemia.

NEW INJECTABLES

1. New forms of insulin

Tresiba: approved in 2015. This is a brand-type version of the insulin called degludec. Tresiba is used to treat both type I and type 2 diabetes. It can last up to 42 hours – longer than commonly used insulins. It is injected once daily.

Basaglar and Toujeo: 2 new forms of insulin glargine (previously marketed as Lantus). Both are injected daily and both can used to treat both types of diabetes. Approved in 2015. These **biosimilar insulins** are very similar to the brand-name forms, with no clinically meaningful difference in purity, potency, and safety (7).

Icodec: a once-a-week insulin which is superior to once-daily degludec (A1C decrease of 0.91 % over 26 weeks compared to 0.71% with degludec). Icodec was also superior to glargine over a 52-week period, with an A1C reduction of 1.55% compared to 1.35% with glargine. Rates of low blood sugar (hypoglycemia) were the same with both drugs.

2. **GLP1 (glucagon-like peptide 1)-agonists:** these drugs help your pancreas release more insulin when glucose levels are high. They also slow down glucose absorption during digestion.

Ozempic (semaglutide): approved late 2017 and only used to treat type 2 diabetes. A1C reduction with Ozempic is modest (0.8%), but it has cardiovascular benefits and has the added benefit of promoting weight loss.

Trulicity (dulaglutide): similar to Ozempic, used once a week.

Byetta (exenatide): used twice a day.

Bydureon BCise (exenatide extended-release): used once a week.

Victoza, Saxenda (liraglutide): used once a day. Saxenda is approved for weight loss as well.

Adlyxin (lixisenatide): Once daily.

All these drugs can cause nausea and vomiting as common side-effects. It is crucial to inform patients they need to stop eating when they feel “full” – that can reduce the frequency of side-effects. These drugs curb the appetite and slow the movement of food from the stomach into the small intestine. Patients may feel full faster and longer, so they eat less (7). Pancreatitis is a potential complication of the use of all GLP-1 agonists.

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3. Combined injectables.

Xultophy. Approved in 2016. This is a combination of insulin degludec and liraglutide, a GLP-1 agonist (7) and is only used to treat type 2 diabetes. It is injected once a day.

Soliqua. Approved in 2016 for treatment of type 2 diabetes. It is a combination of insulin glargine and lixisenatide, a GLP-1 agonist (7).

Ryzodeg. Approved in 2016 but still not available. This combination of insulin degludec with insulin aspart was designed to treat both type 1 and type 2 diabetes. It is meant to be injected twice a day (7).

NEW ORAL AGENTS

1. Oral GLP-1 agonists:

Rybelsus (semaglutide). Taken every day, first thing in the morning on an empty stomach. This drug stops the liver from making too much sugar; in other words, it mimics glucagon-like peptide in that respect. It also helps the pancreas to make more insulin (7).

Mounjaro (tirzepatide): the most exciting drug in the class of GLP-1 agonists. It is a dual incretin agonist drug because it is also a GIP (glucose-dependent insulinotropic polypeptide) agonist drug. In the SURPASS-4 trial, this drug was associated with very impressive A1C reduction and remarkable weight loss. The results are better than any drug on the market right now. Those benefits were maintained up to 2 years without excess cardiovascular risk, something the FDA has been emphasizing the last few years. A 2% reduction in HbA1C was seen with

the highest dose. In clinical trials, 75% of the participants on the lowest dose of the drug achieved an A1C of less than 7%, as did 83% of those in the 10-mg dose group and 85% of those on the 15 mg dose.

And what about the weight loss? Outstanding results, better than those seen before with any antidiabetic drug:

- 90% patients lost weight, with a total body weight loss of 21% in the highest dose (15 mg) group.
- Patients on the highest dose lost 52 pounds on average at the end of the trial.
- By comparison, semaglutide only causes a 15% weight loss.
- Cost is a major issue: this drug will cost about \$1000/month (10).

2. Other new oral agents/oral combinations

Steglatro (ertugliflozin): belongs to the new class of drugs called SGLT2 inhibitors. They decrease blood glucose levels and improve A1C by preventing some of the glucose in the system from reentering blood through kidneys, i.e., they increase the elimination of glucose into the urine. SGLT2 inhibitors are not approved for treatment of type 1 diabetes because of the risk of ketoacidosis (7).

Xigduo XR: Approved for use in 2014. This agent combines a biguanide, metformin, with dapagliflozin, a SGLT2 inhibitor. It comes as a 24-hour extended-release oral tablet (7).

Synjardy: Approved in 2015. This combination pill includes metformin and empagliflozin, an SGLT2 inhibitor,

and comes in an oral pill. Empagliflozin's brand name is Jardiance. Jardiance has been shown to decrease risk of cardiovascular death by 38%, besides lowering blood sugar, blood pressure and having a beneficial effect on weight loss (7).

Glyxambi: Approved in 2015. This oral tablet combines the DPP4 inhibitor linagliptin with the SGLT2 inhibitor empagliflozin. Like all DPP4 inhibitors, linagliptin helps the pancreas to release insulin (7).

Steglujan: approved in late 2017. This oral tablet combines ertugliflozin and sitagliptin (7).

Segluromet: was approved in late 2017. This oral tablet combines the SGLT2 inhibitor ertugliflozin with metformin (7).

I must honestly say that I do not use most of these combo drugs. I prefer to use SGLT2 inhibitors alone and to combine them with other, less expensive drugs. Access and cost are issues we deal with on a daily basis. It is of paramount importance to find combinations that will allow the patient to afford their medications while providing the best care possible with minimal side-effects.

3. Diabetes Medications in Development:

In addition to the medications above mentioned, several diabetes drugs are currently in development. These drugs include:

Oral-Lyn: designed to treat both type 1 and 2 diabetes. It provides insulin as a fast-acting oral spray.

Dance 501: An aerosol device that contains a liquid insulin that is intended to be inhaled at mealtime (3). It is designed to treat both types of diabetes (7).

DIABETIC RESEARCH: LOOKING DOWN THE ROAD

Diabetic research has evolved tremendously over the last few years, and I am excited that there is a very strong possibility that there will be a cure for type 1 diabetes sooner than we expected. In

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Germany, researchers have discovered a novel insulin inhibitor receptor called inceptor. It is a remarkable finding. The blocking of inceptor function leads to an increase sensitization of the insulin signaling pathway in pancreatic beta cells. Result? This may allow protection and regeneration of beta cells, perhaps leading to diabetes remission.

Teplizumab: The potential to delay or prevent type 1 diabetes is real with this drug. It has been described as a possible “game changer”(4). It has shown encouraging clinical trial results, with evidence that teplizumab can delay the onset of type 1 diabetes for almost three years. (32.5 months) in high-risk patients. Also quite exciting was the fact that, more than five years after just a 14-day treatment with the drug, 18% of treated patients still did not have clinical diabetes, compared with only 6% in the placebo group (5).

VX-880: investigational stem cell-derived, fully differentiated pancreatic islet cell replacement therapy for type 1 diabetes. Combined with immunosuppressive therapy, VX-880 produced a robust restoration of islet cell function after 90 days in the first patient in a phase ½ clinical trial. The first person in the trial saw a 91% decrease in his insulin needs (2).

Gene transfer (6,8). At the University of Texas at San Antonio, investigators have introduced selected genes into the pancreas, using a viral vector. These genes can then cause other cell types to make insulin. It is noteworthy to point out that the investigators believe that it is not necessary to replicate “all the insulin-making function of beta cells. Only 20% of this capacity is sufficient for a cure of type 1 diabetes.” This strategy has cured diabetes in mice.

Artificial Pancreas: a device that can continuously monitor a patient’s blood sugar and then continuously release insulin as appropriate for the measured glucose level.

Oral Insulins have not been successful so far because the GI tract does absorb large molecules well. But there are 3 possibilities:

1. Oral nanoparticles. These are especially designed pills that can resist breakdown in the GI tract.
2. Oral delivery systems. A pill that is really a little device that lands in the GI tract and delivers insulin through a microscopic needle injection.
3. Insulin pill. Currently studied in clinical trials by the company **Oramed**. It is too early to tell if any of these options will be successful, but I am inclined to believe that the insulin pill has the best chance.

Smart Insulin: an insulin that responds to glucose levels. It would work when blood sugar levels are high and would turn off when they fall. It is still years away from making its way into clinical trials, but researchers are being helped by computer simulation models. There is always a chance.

Verapamil. The use of this well-known drug is showing benefit in patients with type 1 diabetes. Treated patients have required less daily insulin two years after being diagnosed with the disease (1). Ana Shalev, at the University of Alabama in Birmingham, showed that verapamil can completely reverse diabetes in animal models. These animals also show increased beta cells survival. She is planning to test the drug in a human clinical trial. Verapamil seems to allow patients to produce higher levels of their own insulin, thus limiting their need for injected insulin to regulate their blood sugar levels.

Researchers are really pushing to find ways to prevent or cure type I diabetes. At the Icahn School of Medicine at Mount Sinai in New York City, a research team is investigating ways to revitalize beta cells. They are exploring at least five different approaches:

1. Regeneration of existing beta cells.
2. Restoration of beta cell function using stem cells.
3. Reprogramming beta cells with differentiated cell types.
4. Replacing beta cells with cells from nonhuman donors.
5. Replacing beta cells with beta cells from deceased donors.

These discoveries and current research are extremely promising. We may see the day when, finally, we can be able to get rid of diabetes. More than 100 years after the discovery of insulin (realizing that insulin is not a cure for diabetes but only a treatment of the symptoms) we need to find a real cure. Let’s hope that one day diabetes will become a disease of the past. That will be quite a day, and we ALL are looking forward to it.

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Technology and the Management of Diabetes

by Ken Brantley, MD

The role of technology in the management of diabetes has greatly expanded in the last few years. Technology has long had some role in the management of diabetes, but the role was much more limited in the past. In this article, I will review the current state of the art in glucose sensors and insulin delivery systems and will provide a brief glimpse into technological advances that may help us in the future in the management of diabetes.

Early sugar measurement technologies used chemical test strips to measure urinary sugar. This would tell the patient

that their blood sugar was high but little else. Eventually, technologies for directly measuring blood sugar became available. Early devices to measure blood sugar took several minutes to arrive at the result, and some were literally suitcase-sized. Over time, these technologies improved in multiple ways. The meters themselves gradually became much smaller. The testing technologies improved so that the readings appeared within 15 seconds or less, and the amount of blood required decreased dramatically. Meters also had memory added to them, and many would record the date and time along with the

sugar value. This information could be downloaded to a computer for analysis with appropriate software. The only problem was that these technologies were still dependent on fingersticks. This means that the amount of data collected was directly related to the number of fingersticks that were done by the patient. The need for fingersticks is the most common complaint of diabetic patients. Most patients find fingersticks inconvenient and annoying, and that means that they don't get done nearly as often as they should.

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We are grateful for the support of these organizations and anticipate another great year of serving the needs of our members. The purpose for Circle of Friends is to provide a valuable base of

resources to assist the physician in the business of medicine so their practice of medicine can improve.

This program has proven to be a valuable resource of services such as liability insurance, accounting, banking and much more. This year, we hope to expand the Circle to include services the physician may use in his or her personal life. Through this program, we can invite businesses serving physicians to support the Society and increase their visibility among its members. Corporate support contributes to the Society's ability to advocate and care for physicians and patients in Potter and Randall Counties.

The Medical Society thanks all of its supporters as it offers new opportunities to its membership. If your business is interested in being a part of our Circle of Friends, please contact Cindy Barnard at 355-6854 or e-mail prcms@suddenlinkmail.com.

Glucose Sensors

This state of affairs led to efforts to develop sensor systems that could provide sugar information to a patient without a fingerstick. Early systems such as the Glucowatch did work but had significant drawbacks. The system used an electrode that had to remain in the same spot on the skin, required a significant warmup period and very frequently caused skin irritation. Later systems began to use electrodes that were inserted into the skin to measure interstitial fluid glucose. This decreased the delays between plasma glucose and the sensor readings, and it allowed the development of sensor systems that could remain in the skin for increasing periods of time. Early sensors also explicitly recommended that a fingerstick be done to verify the sugar prior to treating any sugar reading with insulin. Most also required multiple calibrations per day using fingersticks in order to maintain an acceptable degree of accuracy.

There are now several glucose sensor systems available that represent substantial improvements over the older technologies. The systems are typically easy to insert, and the start-up periods are significantly reduced. Some of these systems now provide updated readings every 5 minutes or less. This significantly improves the odds of catching low blood sugars even if a rapid descent is happening. The current systems are now much easier to use, and most of them require a little or no calibration on a routine basis. Collection of data at intervals of every five minutes or less allows the accumulation of much better information about the real patterns of glucose in an individual.

Most sensors use a small electrode that is inserted in the skin. The electrodes are usually inserted either into the abdomen or into the back of the arm depending on the device. The **Libre** sensor (<https://www.freestyle.abbott/us-en/products/freestyle-libre-3.html>) uses a single integrated transmitter and electrode that are inserted and replaced as a unit. The **Dexcom** (www.dexcom.com) and **Medtronic** (www.Medtronic.com)

devices use a separate electrode and transmitter. Dexcom and Libre sensors typically require no calibration fingersticks. The Medtronic sensor typically requires at least two calibration fingersticks per day and may require more at times.

One slightly different design is the **Eversense** (www.ascensiadiabetes.com) glucose sensor system. This device uses an implantable RFID-like chip to measure glucose. This chip requires a minor surgical procedure for implantation and has to be replaced about every six months. A small external reader is placed on the skin above the chip and sends the data to the patient's phone.

Most of these sensors use Bluetooth and a phone app to handle much of the communication. This unfortunately means that the sensors may not be usable in an area that completely bans any sort of radio transmitters.

Currently available sensors also have some way to share sugar information with friends or family. This includes sending text messages or other types of alerts to other individuals if the sugar is either high or low. There are a number of case reports of this capability allowing friends or family to save patients during a severe low sugar event.

Using glucose sensors

Sensor systems are extremely effective at helping improve blood sugars in most patients. The devices provide a lot of information. If the patient will use that information, then significant improvement of blood sugar usually occur. Diet almost always improves, primarily because the patient is now aware of the effect of each meal on the sugar – it is no longer possible to pretend that ice cream doesn't raise the blood sugar! Insulin doses can be much more effectively tailored to meals. By watching their sugars, patients can now tell if the dose is correct or if they will need to adjust the doses. This may greatly improve the patient's comfort level with

lower sugar levels, since they have less worry of hypoglycemic events. The ability to identify low sugars is extremely helpful, especially in those patients who do not perceive low sugars well. This is especially useful during the overnight time frame, when low sugars may escape notice unless they are especially severe.

The current sensor systems all have some form of predictive alert. That is, the system extrapolates the recent sugar trend line out for some period of time (The time period varies depending on the sensor). If this trend predicts a value outside the high or low parameters, then the user gets a warning of a predicted high or low. This can be extremely helpful since it gives patients time to deal with low sugars before they become symptomatic.

Early sensor systems often had fairly complex insertion schemes, but the more recent systems are much easier to install. Skin irritation with the sensor systems is relatively uncommon but does occur. Patients who have very physical jobs and sweat significantly may have problems keeping the sensor on for the full 10 to 14 days.

Many factors influence the choice of sensor for an individual patient. Often the biggest one is the question of which one their insurance will cover. Quite often, insurance plans have very specific preferences and provide little or no coverage for other sensor types. And patient preference can play a big role as well. Some patients may find a specific sensor system much easier to use than others. Some patients need a sensor system with a receiver because they may have a job that prohibits carrying a cell phone and they need a dedicated blood sugar device. A patient who is using an insulin pump will need to use the sensor system that works with their pump.

The issue right now is that patients typically need to be on insulin in order to qualify to use the sensors with most insurance companies. The exact requirements are variable, but many insurance plans require multiple insulin shots per

day before they will cover the sensor systems. Medicare typically requires that the patients take multiple insulin doses per day and check the sugars at least four times per day.

Insulin delivery technologies

The **InPen** (<https://www.medtronic-diabetes.com/products/inpen-smart-insulin-pen-system>) is a Bluetooth-enabled insulin pen that transmits insulin dose data to a phone app. The app records the time, date and amount of all insulin doses. Sugar levels from the Dexcom are also recorded. In addition, the app can generate reminders for the insulin doses. This helps improve compliance with the planned dose regimen. Missed insulin doses are frequently a cause of high blood sugars. Integrating sugar data from the sensor with insulin dosage and timing information can allow more informed adjustments of the insulin dosing, and typically results in improved sugars over time. The data is stored in a cloud service and can be downloaded by the patient's physician. This system combines the lower complexity of a pen insulin delivery system with the data collection capabilities of an insulin pump.

Insulin pumps work by slowly infusing a rapid-acting insulin into the patient. The slow infusion mimics a basal long-acting insulin. Unlike long-acting insulin, which essentially provides a single rate for the day, the infusion of a short acting insulin means that the dose can be adjusted at any given time during the day to account for activity or other issues. The patient can also give larger doses of short-acting insulin to mimic a mealtime insulin dose. A small infusion set, with either a short needle or a short plastic catheter, is used to infuse the insulin under the skin.

There are two basic design philosophies with insulin pumps. One type of pump has a separate reservoir pump system with tubing connecting it to an infusion site that is inserted in the skin. The other type is the pod pump. With the pod, everything except the user interface is typically integrated into a single small device.

Typical wear time for infusion sets or pods is three days. The infusion set or pod is then changed. Most commonly, the infusion sites are located on the abdomen or on the back of the arm. It is possible

to use other locations, but in most cases there are significant practical difficulties with their use.

There are three major pump systems available in the United States: the tandem **T:slim X2** (<https://www.tandemdiabetes.com>), the **Medtronic 770G** (<https://www.medtronicdiabetes.com>) and **Omnipod 5** (<https://www.omnipod.com>). Each of these pumps now has the ability to use a glucose sensor and adjust the insulin dosage based on current glucose levels. Each device works slightly differently, but improved sugars usually result. If the sugar is higher, then the patient's insulin dose is increased by giving small bonuses of insulin; if the sugar is beginning to get low, then the insulin dose is reduced in an effort to minimize the risk of hypoglycemia. This can produce extremely good control, particularly in the overnight periods. Between-meal control is also usually significantly improved. Each system uses different algorithms, but so far all seem to be able to achieve satisfactory results in many patients. Pump systems make it much easier to give insulin and significantly increase the convenience of giving meal time boluses. Instead of pulling out a pen or a syringe, patients merely have to use an app on their phone or the pump to give the meal time dose. The increased convenience typically means fewer missed doses. Adjustments in the various settings on the pump can be made to account for exercise, unusual schedules or significant dietary changes.

Many pump users also count carbohydrates and then use the insulin to carbohydrate ratio to calculate the insulin dose for any particular meal. This provides insulin doses that are better tailored to specific meals and is effective in compensating for unusual meals. Use of a sensor system allows effective tracking of the accuracy of these ratios. The pumps or phone apps typically have some sort of calculator available, and the ratios can simply be changed in the calculator. This allows the



Figure 1. The larger object on the right is the transmitter for the Libre 2 glucose sensor, the middle one is the transmitter for the Libre 3.

patient to calculate a correct meal time dose easily.

Long term prospects

The manufacturers of both glucose sensor systems and insulin pumps are maintaining intense research and development efforts to try to improve this technology in the long term. Glucose sensor systems will gradually become more accurate with significant improvements in usability. Sensors will become easier to use with smaller transmitters and electrodes that cause less irritation and last longer. A good example of this trend is the recently developed **Libre 3** transmitter which is significantly smaller than the Libre 2 (see Figure 1). This trend will likely continue with other sensor systems in the long-term. Improved wear time will be extremely helpful since it means patients won't have to insert new sensors as often.

Insulin pumps and sensors will become better integrated with glucose sensor systems and with smartphones. Rapid-acting

insulin and better sensor systems will provide pumps with the ability to generate a more effective response to both high and low sugars. Work is also being done to improve wear time for insulin infusion sets.

Eventually it is very likely that there will be systems that use extremely rapid-acting insulins and good sensor technology to produce sugar control that may be nearly as good as the natural pancreas could do.

Implantable insulin pumps have been evaluated in the past. Direct infusion of the insulin into the peritoneal cavity produces an extremely rapid insulin response. Improvements in external infusion pumps and glucose sensors have significantly reduced interest in this technology at the moment.

Frequently reports appear in the media describing new or interesting technology. In many cases, the reports do not make clear the stage of development of

the technology. Often the technology is still in a very early stage, and it will likely take years before the technology reaches the market. To be approved for patient use, medical devices have to go through extensive approval evaluations and safety evaluations through the FDA prior to being released to the market. These evaluations can take significant amount of time. Unfortunately COVID and other problems have produced a significant increase in the FDA's workload, and this is slowing down approval for technologies.

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When You Hear Hoof Beats, Look for Horses, Not Zebras ...Unless you are a Pediatric Endocrinologist

by Alison Lunsford, MD

The ISPAD etiological classification of diabetes includes Type 1 DM, Type 2 DM, Other specific types and Gestational diabetes as the 4 broad categories (3). The third category, other specific types, incorporates all the zebras. In this article, I would like to discuss how to listen for zebra hooves amongst all the horses commonly seen here in West Texas.

CASE 1:

E.M. received his diagnosis of diabetes at 10 years of age after presenting to his PCP for concerns of inattention at school. At diagnosis, hemoglobin A1C was 6.2%. The patient was hospitalized and placed on insulin in a basal/bolus fashion with Levemir/Humalog. He eventually transitioned to a subcutaneous insulin pump. For the first several years, diabetes control always maintained Hgb A1C in the 6% range, with total daily dose of insulin less than 4-6 units per day. Upon my arrival to Amarillo, we reviewed his special case and noted that his autoantibodies were all negative at diagnosis. Furthermore, his mother received the diagnosis of Type 2 DM during a failed OGTT during pregnancy. His mother reported that four of her eight siblings, as well as her maternal grandmother, carried the diagnosis of Type 2 DM. There were no signs of insulin resistance or other dysmetabolic features in the family. None of the family members, including the grandmother, developed complications from their diabetes. Family heritage was European American. The patient was phenotypically thin and remarkably healthy otherwise.

Cue sounds of zebra hoof beats...

Maturity Onset Diabetes of the Young (MODY) genetic panel was sent and returned with positive GCK MODY (MODY 2).

Monogenic diabetes accounts for approximately 1-2 % of newly diagnosed diabetes in children and adolescents (5). MODY is caused by a single gene defect, with more than 14 genes having been associated with a distinct subtype. Each subtype differs in clinical presentation (age at onset and pattern of hyperglycemia), extra-pancreatic function, risk of complications, and response to treatment (5). You should consider this diagnosis in a child/adult with antibody negative, C-peptide positive diabetes who does not clinically fit Type 2 DM. Usually, there is a strong family history, but most of the time the family has been diagnosed with Type 2 DM (as this family had been). Differentiating the diagnosis is important as it often alters the pharmacologic treatment plan, as it did for this case. MODY 2, glucokinase (GCK) mutation, is one of the most common MODY forms (1:1000), leading to fasting hyperglycemia throughout life (5). Many women inadvertently receive the diagnosis of gestational diabetes. With normal weight maintenance and healthy diet, these persons with diabetes will maintain their Hgb A1C 6-7% without treatment, and this specific form does not have long-term sequelae. This young man was able to come off insulin pump and all subcutaneous insulin with annual follow up, maintaining Hgb A1Cs around 6.5%.

CASE 2:

B.A. was a 14 year-old, 60 kg (BMI 23) Hispanic female who arrived at an outside ER with severe abdominal pain, vomiting and respiratory distress. She reported the onset of vomiting the evening before arrival. Previous medical history was unremarkable. She was noted to be in severe DKA and was transferred to a local Pediatric ICU. Upon admission to the PICU, her abdominal pain was out of proportion to that expected

in DKA and she described a one-month history of loose, fatty stools. Further evaluation revealed pancreatitis (lipase 632). The evaluation for pancreatitis included gallbladder US, which was normal. Labs included a total cholesterol of 253 mg/dL, triglycerides > 4000 mg/dL, HDL 22 mg/dL, LDL unable to calculate. She had a strong family history of Type 2 DM on the paternal side, with complications in the father including non-alcoholic steatohepatitis and congestive heart disease. Phenotypically, neither the child nor father were classically obese but both had distal lipodystrophy with increased facial, neck and visceral adiposity.

Cue hoof beats...

This child was sent to UT Southwestern in Dallas to Dr. Abhimanyu Garg, a lipodystrophy specialist, after discharge. The family subsequently received the diagnosis of Familial Partial Lipodystrophy (FPLD) Type 1 (Kobberling-type lipodystrophy).

Lipodystrophies are a heterogeneous group of disorders characterized by selective partial or generalized loss of adipose tissue. There are congenital and acquired forms, which are further categorized into localized, partial or generalized according to the distribution of adipose tissue loss (1).

Familial Partial Lipodystrophies are inherited in an autosomal fashion and are characterized by loss of subcutaneous adipose tissue (usually noticed late in childhood or at puberty) from the upper and lower extremities as well as from the truncal region. Associated clinical features noted in Table 1 on next page (1).

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Table 1: Clinical Features of Familial Partial Lipodystrophies

<p>Metabolic abnormalities:</p> <ul style="list-style-type: none"> • Insulin resistance, acanthosis nigricans • Hyperglycemia, diabetes mellitus • Hypertriglyceridemia, eruptive xanthomas, pancreatitis • Ectopic fat deposition
<p>Liver:</p> <ul style="list-style-type: none"> • Hepatic steatosis, hepatomegaly • Non alcoholic steatohepatitis • Liver cirrhosis
<p>Heart:</p> <ul style="list-style-type: none"> • Cardiomyopathy • Atherosclerotic coronary heart disease
<p>Reproductive:</p> <ul style="list-style-type: none"> • Hyperandrogenemia, hirsutism • Oligomenorrhoea, subfertility • Polycystic ovarian syndrome
<p>Other:</p> <ul style="list-style-type: none"> • Proteinuric renal disease • Myopathy

Teasing out this diagnosis can be a bit challenging. The diagnosis of FPLD frequently requires genetic testing, but even a negative result cannot rule out lipodystrophy. Clinical, lab and physical exam findings often flush out this zebra from the herd of horses. The majority of the management options for this condition are standard therapies for other patients with NASH, PCOS, and Type 2 DM. Often times, these patients require high doses of medications and still fail to achieve control of their disease with standard therapies. Metreleptin, a recombinant analogue of human leptin, received approval for use in the United States in 2014 and can benefit those patients who are most severely affected by this disease (1).

CASE 3:

A.S. is a 12 year-old female status post liver transplant at 1 year of age. At 11 years of age, she developed acute rejection, which was treated with high dose antirejection medications including steroids. She developed steroid-induced diabetes requiring insulin. Steroids were eventually tapered and the insulin discontinued. Approximately 6 months after insulin was tapered, the patient began

having polyuria and polydipsia. Labs obtained by the transplant team revealed blood sugar >250 mg/dL with hemoglobin A1C of 9%. Her PCP contacted our team for further evaluation. Laboratory evaluations were sent to rule out Type 1 DM and all returned normal. The patient had not received any form of systemic corticosteroids for more than 6 months. Her working diagnosis: Post-Transplant Diabetes Mellitus.

Okay so maybe not a zebra, but at least an antelope...

Approximately 3-20% of pediatric solid organ transplant (SOT) recipients will develop post-transplant diabetes mellitus (PTDM), depending on the organ transplanted, age at transplantation, immunosuppressive regimen, family history, and time elapsed since transplant (2). (Of note, this is why we do not transplant the pancreas in children with Type 1 DM) The diagnosis of PTDM can be made after the patient is stable on a chronic immunosuppression regimen and free from an acute infection (2). PTDM results from both insulin resistance and insulin deficiency. The majority of cases I have seen in the pediatric

population receive the diagnosis during puberty with the rise of insulin resistance. Immunosuppressant drugs, which are the mainstay of post-transplant therapy, are a key contributing factor to PTDM in adolescents. SOT recipients are commonly treated with a combination of corticosteroids, calcineurin inhibitors, mycophenolate mofetil or azathioprine (2). Calcineurin inhibitors (tacrolimus and cyclosporin), as well as inhibitors of mammalian targets of rapamycin (sirolimus and everolimus), lead to a dose-dependent decrease in insulin synthesis and secretion. One study showed that, of 214 pediatric liver transplant patients who developed PTDM, 14.2% were on tacrolimus compared to 5.5% on cyclosporin (2). Management of PTDM will vary based on the organ transplanted and post-transplant immunosuppressive therapy drugs. Given the lack of approved T2DM medications in the pediatric population, insulin therapy is frequently employed.

CASE 4:

An endocrinology fellow was called to the NICU for a preterm infant boy with severe, sustained hyperglycemia. The infant was born after an uncomplicated pregnancy and was slightly preterm (34 weeks). The infant was found to have intrauterine growth retardation (IUGR). After birth, he developed severe diarrhea, anemia, and hyperglycemia.

A whole herd of zebras...

The prevalence of hyperglycemia in preterm infants in the NICU ranges from 25-75%; causes include TPN, sepsis, increased counter-regulatory hormones due to stress, and steroid use (4). Typically, this resolves within 3-5 days of onset. Neonatal diabetes mellitus (NDM) should be considered in infants with insulin-dependent hyperglycemia, with blood glucoses persistently greater than 250 mg/dL, without an alternative cause. Typically, if hyperglycemia lasts > 10 days, consideration should be given to a more permanent cause. Initial evaluation

should include pancreas ultrasound to establish the presence or absence of a pancreas, along with standard laboratory testing: C-peptide, insulin, glucose, and urine ketones. Pancreatic autoantibodies are not recommended due to trans-placental crossing of maternal antibodies. Genetic testing, however, is recommended immediately upon suspicion of the diagnosis. Age at presentation varies from immediately after birth to up to 1 year. There are more than 20 known monogenic causes of NDM, which may be transient or permanent (4). Treatment varies based on genetic mutation. If long-term therapy is required, subcutaneous insulin via insulin pump is the most effective therapy due to the ability to dose in small increments. The infant in the case scenario returned with a *FOXP3* mutation, known as IPEX syndrome. This is an often-lethal form of autoimmune neonatal diabetes, which also includes exfoliative dermatitis, enteropathy and autoimmune thyroid disease. These infants frequently succumb to infections due to immunodeficiency.

CASE 5:

T.C. experienced a near-syncope event at school, which landed him at his PCP office for evaluation. His EKG was normal, but the chemistry panel surprisingly showed a blood sugar of 320 mg/dL. He reported that he had just drunk a Sprite before the labs due to the syncope event. He denied polyuria or polydipsia. Hgb A1C was only 7%. He was a healthy 15 year-old Hispanic male with BMI of 19 and no past medical history other than depression. The patient was admitted to the hospital for further evaluation. He lived with his uncle who himself had severe complications of diabetes. The uncle reported he and his brother (the patient's father) were, like this patient, diagnosed with diabetes in adolescence. They lived in rural West Texas and were never seen by an endocrinologist. They were treated both with oral medications and insulin when younger. The uncle reported they were not obese as adolescents. They reported that diabetes had plagued their family for generations.

Labs were sent to help distinguish between Type 1 and Type 2 DM. The patient was sent home on low dose basal insulin with correctional insulin as needed.

At follow up, it was noted that all pancreatic autoantibodies returned negative and that C-peptide was 1.5. The patient did not have any further dysmetabolic concerns such as NASH, hyperlipidemia, or hypertension. MODY testing was obtained.

This zebra goes by the name of HNF1A autosomal dominant MODY 3.

Hepatocyte nuclear transcription factors (HNFs) regulate the expression of many genes essential for the normal development and function of the liver, gut, kidney and pancreatic islets. Worldwide, HNF1A-MODY is the most common MODY type (5). The majority of patients with this form will present post-puberty to 25 years of age. The prevalence of microvascular complications correlates to glycemic control.

I brought this subject up because of how easily we could have put this off as Type 2 DM. However, this child was thin and had no evidence of insulin resistance. HNF1A MODY responds remarkably well to sulfonylureas. If additional medication is required, GLP1 agonists or insulin can be added over time.

MODY genetic testing is available through multiple commercial and institutional laboratory settings such as Prevention Genetics, Lab Corp, GeneDx, and the University of Chicago. Many companies allow you to receive free kits sent to your office and require buccal swab only.

In summary, I hope you have found these cases interesting and may use some of the clinical clues in your practice to help weed out these unique cases to improve the lives of the children and adults in our community.

“There’s no limit to how much you’ll know, depending how far beyond zebra you go.”

Dr. Seuss

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Distinguishing Type 1 from Type 2 Diabetes Mellitus in Children and Adolescents

by Maria F. Contreras, MD

Introduction

Obesity is a chronic disease that is increasing in prevalence and is now considered to be a global epidemic. The increasing prevalence of obesity among US youth has been demonstrated across all age groups and ethnicities (1). Type 1 diabetes mellitus (T1DM) is more common among children and adolescents; however, the incidence of type 2 diabetes (T2DM) in youth is on the rise due to the high rates of obesity (2). The development of diabetes at a young age has serious repercussions on the health of affected individuals due to age-specific issues and the more aggressive nature of the disease. The management of diabetes in children and adolescents is challenging in some cases; the increased prevalence of obesity in children has minimized the value of body mass index as a distinguishing feature between T1DM and T2DM. In the past, researchers considered patients suffering from T1DM lean, whereas those with T2DM generally presented with obesity. However, recent studies indicate an increased BMI even in children with T1DM (3). The increasing prevalence of obesity in children has resulted in the growing incidence of T2DM and, more frequently, in the development of combined T1DM and T2DM.

Type 1 Diabetes

T1DM is characterized by profound hyperglycemia due to absolute insulin deficiency caused by immune-associated destruction of the insulin-producing beta cells of the pancreas; this condition depends on exogenous insulin to prevent ketosis and preserve life. Thus, it was termed insulin-dependent diabetes mellitus. The natural history of the disease indicates that there are preketotic non-insulin-dependent phases before

and after diagnosis. Although the onset is predominantly in childhood, the condition may occur at any age (4).

Type 1a diabetes is generally distinguished by its association with certain histocompatibility locus antigens (HLAs) and other genetic markers, the majority of which determine the response to self (or exogenous) antigens. It is characterized by the presence of circulating antibodies to islet cells; antibodies to insulin, antibodies to glutamic acid decarboxylase (GAD), antibodies to IA-2 (islet cell) and antibodies to the zinc transporter molecule (ZnT8); by lymphocytic infiltration of islets early in the disease; and by coexistence with other autoimmune diseases. Occasionally, antibodies are absent despite profound insulinopenia and dependence on insulin without evidence of mitochondrial or another genetic defect (4). In these cases, T1DM is considered idiopathic (Type 1b).

Type 2 Diabetes

T2DM occurs when insulin secretion is inadequate to meet the increased demand posed by insulin resistance, leading to relative insulin deficiency; it is generally associated with other metabolic abnormalities. Unlike T1DM, no identified autoimmune process leads to inadequate insulin secretion; instead, T2DM appears to result from genetic, environmental, and metabolic causes that may differ between individuals and populations. T2DM typically occurs in adolescents at midpuberty, most likely precipitated by the physiologic but transient pubertal insulin resistance aggravating the preexisting metabolic challenges of obesity (5). Persons with this subclass of diabetes may not be

permanently insulin dependent and only occasionally develop ketosis. Some may, however, need insulin to correct symptomatic hyperglycemia, and ketosis may develop during severe infections or other stress. T2DM was previously considered to be an adult disease, but we now see more cases in young children and adolescents due to a rise in obesity. Evidence suggests that T2DM in youth is different not only from T1DM but also from T2DM in adults and has unique features, such as a more rapidly progressive decline in β -cell function and accelerated development of diabetic complications.

Type 1 vs. Type 2 Diabetes: Diagnosis

T1DM and T2DM have similar symptoms; the classic presentation of diabetes in children is a history of polyuria, polydipsia, polyphagia, and weight loss. Polyuria may be heralded by the recurrence of bedwetting in a previously toilet-trained child and polydipsia by a child constantly requesting fluids to drink. Unexplained weight loss should raise suspicion of diabetes that should be confirmed or excluded by serum blood glucose (BG) and urine tests; these provide a simple and sensitive screening tool. If the BG is elevated, prompt referral to a facility treating children with diabetes is essential.

The provider should pay attention to history and characteristics at presentation. Management, though, can be challenging; it is essential to remember that any child with initial Hb A1C > 8.0 should be seen immediately by a pediatric endocrinologist.

In T1DM, nearly 90 % of individuals have the presence of one or more islet

autoantibodies such as insulin IAA, GAD, IA-2, and ZnT8A (2). In T1DM, approximately one-third present with DKA (6). A physician has seen most children who are diagnosed with T1DM within a week or so of diagnosis. Often, however, diabetes was not considered, and no appropriate tests were performed.

T1DM children at diagnosis present with a history of polyuria, polydipsia, and weight loss; the duration varies but is usually less than one month. On the other hand, patients with T2DM manifest slowly over time; this is what can cause those with T2DM not to realize they have the condition for years before diagnosis. The primary care doctor plays a vital role in this phase, screening obese/overweight patients with risk factors to help with an early diagnosis to avoid further complications for the child.

T2DM patients often have a family history of diabetes, may have acanthosis nigricans, are more commonly girls, and often display poor metabolic control that predisposes them to the earlier appearance of complications. They may present initially in DKA, suggesting T1DM, but after recovery, they may manifest a prolonged “honeymoon phase”, as documented by significant insulin and C-peptide levels not consistent with T1DM. They also lack markers of islet autoimmunity and the classic HLA associations (4).

The American Diabetes Association guidelines in children suggest that: 1) Screening for prediabetes and/or T2DM should be considered after the onset of puberty or ≥ 10 years of age, whichever occurs earlier, in youth with overweight (BMI ≥ 85 th percentile) or obesity (BMI ≥ 95 th percentile) and who have one or more additional risk factors for diabetes. 2) Children and adolescents with overweight or obesity in whom the diagnosis of T2DM is being considered should have a panel of pancreatic autoantibodies tested to exclude the possibility of autoimmune T1DM (7). The presence of islet autoantibodies has

been associated with faster progression to insulin deficiency.

Type 1 vs Type 2 Diabetes: Management

The management of diabetes in children and adolescents cannot simply be derived from care routinely provided to adults with diabetes. The epidemiology, pathophysiology, developmental considerations, and response to therapy in pediatric-onset diabetes differ from adult diabetes.

Children with T1DM will require insulin treatment, which must be continued for the rest of their lives, since their bodies cannot produce insulin. Insulin can be provided through an insulin pen, pump, or syringe. All children and adolescents with T1DM should self-monitor glucose levels multiple times daily (up to 6–10 times/day by glucose meter or continuous glucose monitoring).

FDA-approved pharmacologic treatment options for youth-onset T2DM are limited to 3 approved drug classes: insulin, metformin, and liraglutide (glucagon-like peptide 1 receptor agonists). Presentation with DKA or marked ketosis requires a

period of insulin therapy until fasting and postprandial glycemia have been restored to normal or near-normal levels. Initial treatment should also be with insulin when the distinction between T1DM and T2DM is unclear and in patients with random BG concentrations ≥ 250 mg/dL and/or A1C $\geq 8.5\%$. When insulin treatment is not required, initiation of metformin is recommended. A recent randomized clinical trial in children aged 10–17 years with T2DM demonstrated the addition of subcutaneous liraglutide to metformin (with or without basal insulin) as safe and effective to decrease A1C, although it did increase the frequency of gastrointestinal side effects (7).

Youth-onset T2DM is associated with significant microvascular and macrovascular risk burden and a substantial increase in the risk of cardiovascular morbidity and mortality at an earlier age than those individuals diagnosed later in life (9). The higher complication risk in earlier-onset type 2 diabetes is likely related to prolonged lifetime exposure to hyperglycemia and other atherogenic risk factors, including

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insulin resistance, dyslipidemia, hypertension, and chronic inflammation. These comorbidities are seen earlier in the youth with T2DM when compared with T1DM children diagnosed at same age.

In youth-onset T2DM, genetics and epigenetics represent nonmodifiable risk factors. Many major risk factors, however, are modifiable, including obesity and lifestyle habits of excess nutritional intake, low physical activity, and increased sedentary behaviors with decreased energy expenditure, resulting in the surplus of energy being stored as body fat.

Conclusion

Optimal management of diabetes in the pediatric population requires an integrated approach; it is essential to consider the overall level of functioning of the child and family. T1DM and T2DM are heterogeneous diseases in which clinical presentation and disease progression may vary considerably. Classification is important for determining therapy, but some individuals cannot be clearly classified as having T1DM or T2DM at the time of diagnosis.

The main differences between T1DM and T2DM are how fast symptoms present, the severity of the symptoms, age at onset, family history of diabetes, presence of autoantibodies, C-peptide, and insulin level, but, as mentioned earlier, some clinical features can overlap.

Unfortunately, the diagnosis of diabetes (especially T2DM) is sometimes delayed, leading to prolonged periods of uncontrolled hyperglycemia and consequent risk of acute and chronic complications, and misclassification occurs occasionally. A timely and accurate diagnosis, combined with regular follow-up and maintenance of optimal glycemic and risk factor control by judicious use of the available therapies, will ensure that these young people enjoy a long, fruitful, and complication-free life despite diabetes.

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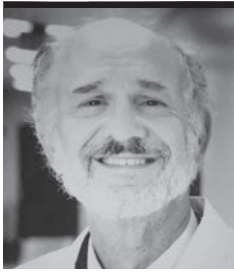
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Metabolic Complications of Uncontrolled Diabetes: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State

by Steve Urban, MD, MACP

As the incidence of DM continues to skyrocket, the complications of uncontrolled diabetes – diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) – occur more frequently than ever before. In the case of DKA, where we have the best data, the incidence has increased 30% in the past decade, leading to over 160,000 hospital admissions per year at a cost of over \$2.4 billion. Half of all deaths in diabetics under the age of 24 are due to DKA, and recurrent admissions for DKA increase the mortality rate fourfold. The statistics for HHS are harder to come by, but it too is probably increasing, since much of the increase in the incidence of DM is due to Type 2 diabetes (T2DM). HHS is less common than DKA, but the mortality rate (10-15%) is higher. In this paper, I will review the diagnosis and management of these severe complications of this increasingly common disease.

Back in the late Devonian Period (the early 1980's), when I was a resident at U.T. Southwestern, controversies swirled around the proper management of these patients – the dose of insulin, the rate of fluid and electrolyte replacement, when to give bicarbonate, whether to supplement the phosphorus, etc. Our chief of medicine was the brilliant but extremely intimidating Dr. Donald Seldin, one of the great IM chairs in the history of American medicine. Presenting the flow sheet of your complicated DKA patient to Dr. Seldin was like asking James Joyce to criticize a hastily written account of your summer vacation. Dr. Seldin never acknowledged that we only had one glucometer in the hospital (!!!!), that we had to draw our own blood samples, and that it took hours for the Parkland lab to run a simple chem panel. The best you could hope for was for Dr. Seldin to fling your flow sheet back to you without comment; most of the time you were subjected to a

level of critical scrutiny generally associated with the Spanish Inquisition.

Now, almost all controversies have been settled and the lab results come back promptly. Still, however, the management of these patients requires a solid understanding of fluid and electrolyte physiology and careful attention to detail. A well-run DKA or HHS (i.e., prompt resolution, no treatment-induced hypoglycemia or hypokalemia, no complications) should be a source of pride and celebration for the doctor, nursing staff, and the patient's loved ones, as the patient, desperately ill only a few hours before, wakes up and leaves the hospital in a day or two.

A brief review of the pathogenesis of DKA and HHS

Insulin is the hormone that builds you up after you eat (i.e., anabolism). The postprandial rise in blood sugar stimulates insulin secretion, and insulin pushes the nutrients – not only carbohydrate but also fat and protein – into the cells for later use. An hour or two later, insulin levels fall back to baseline, counterregulatory hormone levels rise, and you start to use up what you have stored (i.e., the process of catabolism). Without insulin, not only does your blood sugar rise, but breakdown of adipose tissue leads to a rise in free fatty acids (the source of the “ketone” bodies, acetoacetic acid and beta-hydroxybutyric acid) and muscle cells sacrifice their proteins to supply amino acids for gluconeogenesis. A state of insulin deficiency (in T1DM – the usual case) or relative insulin insuffi-

ciency (in the occasional T2DM patient with DKA), plus the counter-regulatory hormone storm associated with severe stress, leads to the massively catabolic state that we call diabetic ketoacidosis

If you don't have a total lack but just an insufficient supply of insulin – or if your cells are relatively resistant to the effects of insulin – (i.e., T2DM), there will be enough insulin to suppress ketone production but not enough to lower the blood sugar level. The high blood glucose level spills into the urine, pulling fluid and electrolytes with it (osmotic diuresis). If you can't keep up with these losses (let's say, you get sick from another illness and start vomiting), this osmotic diuresis will lead to dehydration. With severe dehydration, kidney function begins to fail and you no longer lose sugar in your urine. The “pop-off valve” effect of renal glucose excretion had been keeping the blood sugar around 300 or so, but now the blood sugar level skyrockets to 600 or more (my personal record is 1800 mg%). Hypertonicity of the extracellular fluid pulls water out of cells of the body (especially cells of the brain), leading to severe cellular dehydration, altered mental status, and, in about 30% of HHS patients, to coma.

Clinical manifestations and diagnosis of DKA and HHS

This table outlines the main ways of distinguishing DKA from HHS.

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Table 1	Diabetic ketoacidosis	Hyperglycemic hyperosmolar state
Glucose level	250-400 mg/dL	>600 mg/dL
Osmolarity	<310 mosm/L	>320 mosm/L
pH	<7.3	>7.3
Beta-hydroxybutyrate	>3 mmol/L	<1 mmol/L
Anion gap	usually >15	normal or slightly elevated
Setting	usually type 1 DM	usually type 2 DM

Diabetic ketoacidosis classically occurs in type 1 DM, but DKA in type 2 diabetics is being increasingly recognized (as many as 25% of DKA cases occur in T2DM in some recent series; when DKA occurs in T2DM it is usually provoked by severe physiological stress such as sepsis). The onset of DKA is often rapid – it may occur within 24 hours of discontinuing insulin, especially in patients on a pump. Most patients with DKA have nausea and vomiting, and many have abdominal pain that can mimic mild pancreatitis. Patients with moderate to severe DKA will have rapid, deep, “driven” respirations, and their breath will smell like Juicy Fruit gum. DKA patients will have a wide-anion gap metabolic acidosis (pH <7.3, sometimes <7.0). The old semiquantitative serum ketone tests (i.e., the ones reported in dilutions such as 1:2 or 1:4) would sometimes give confusing numbers because of alterations in the redox state due to hypoxia and tissue ischemia; this problem is obviated by measuring serum beta-hydroxybutyrate levels (BOHB) directly. The BOHB level (>3 mmol/L in DKA) will provide excellent discrimination between DKA and HHS. Some hospitals have point-of-care BOHB levels with the results available

in minutes; no longer to do have to treat with IV insulin “until the gap closes”. In-lab BOHB levels are available at BSA, but point-of-care BOHB levels are not available in Amarillo hospitals at this time. Occasionally, patients on SGLT2 inhibitors will have so-called euglycemic DKA (glucose level <250 mg/dL); these patients can be recognized by their clinical symptoms (nausea, vomiting, abdominal pain) and by the presence of a wide anion gap ketoacidosis (1).

Hyperglycemic hyperosmolar state classically occurs in type 2DM patients but has increasingly been reported in T1DM patients who have enough insulin (either endogenously or exogenously) to suppress ketogenesis but not enough to control the glucose level. HHS patients are often elderly and usually are thrown into HHS by an intercurrent illness, often a life threatening one (this is why the mortality rate is so high). The patient will have been sick for days to weeks, and the clinical manifestations will be dominated by dehydration and mental status changes. Abdominal pain and Kussmaul respiration will be absent. The two main laboratory features of HHS are severe hypertonicity and the absence of ketoacidosis. The hypertonicity is partly due

to extreme hyperglycemia but also to free water loss from the osmotic diuresis. Effective osmolarity (i.e., not counting the BUN) exceeds 320 mosm/L and may approach 350 mosm/L. This corresponds to a free water deficit approaching 5 to 6 liters. In addition, extracellular fluid volume deficiency is equivalent to several liters of saline; this leads to pre-renal azotemia with a corresponding rise in the BUN and creatinine levels. Although these patients may have a mild acidosis (pH usually >7.3, anion gap in the 15 range), this is due to lactic acid from tissue underperfusion and uremic acids from renal insufficiency. The urine ketones may be trace positive but the serum beta-hydroxybutyrate level will be <1 mmol/L.

Does making a precise distinction between DKA and HHS really matter? The treatment algorithms from the American Diabetes Association (ADA) are very similar (2). You don’t have to think about giving bicarbonate in HHS, but then we rarely give bicarb to DKA patients anyway. The thresholds for decreasing the rate of the insulin drip are slightly different (see below). The main difference is in mortality rate (again, 10 times higher in HHS) and in understanding that patients with type 2 DM (most HHS patients and a few DKA patients) may not require long term insulin – but almost all will need to be treated with insulin for a while after discharge. Furthermore, several groups (especially Umpierrez’s group at Emory University) have reported that 20-30% of their patients have DKA-HHS overlap, with both a wide anion gap ketoacidosis and severe hyperosmolarity (3). They report that these patients have an even higher mortality rate than patients with HHS alone.

Management of DKA and HHS

Again, many of the controversies that used to surround the management of diabetic emergencies – high dose vs low-dose insulin, when to add bicarbonate or phosphorus – have been fairly well settled, leading to the algorithmic approach recommended by the ADA (Figure 1). As you can see, the only (minor) difference between DKA and HHS is the recommendation that the rate of the insulin

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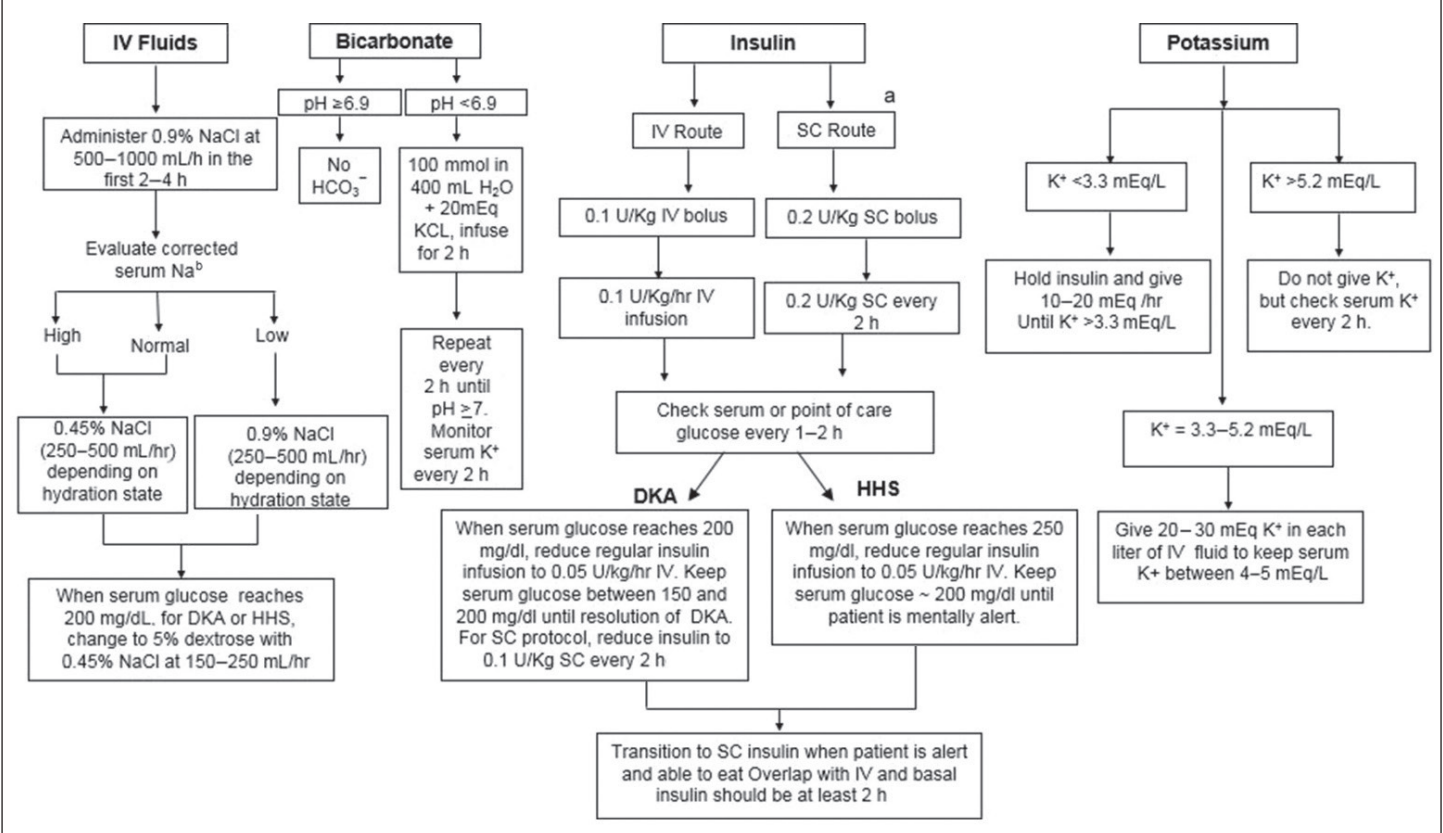
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Table 2



drip should be reduced at a glucose level of 200 in DKA and 250 in HHS; otherwise, they are exactly the same.

I am not going to plod laboriously through the algorithm, but I want to make a few general points. I believe that the most important aspect of management is careful and frequent monitoring, especially in the first few hours. Sometimes you will read, for instance, that serum chemistries should be checked every 4 hours. If you wait 4 hours before checking the first set of electrolytes in patients with moderate to severe DKA, you are asking for trouble. You are very likely to find severe hypokalemia, and you won't know when the anion gap closes. Especially in patients with severe acidosis, I recommend that the electrolytes be checked every 2 hours until the anion gap closes (this may take 6-8 hours); by then, you and your patient are usually out of the woods. In addition to laboratory monitoring, the patient should be assessed frequently during the initial stages, partly to assess hemodynamic stability, urine output, and volume status, but also to remain vigilant for the underlying precipitating cause.

Common pitfalls in the management of DKA and HHS

1. **Glucose management.** The commonest iatrogenic problem is treatment-induced hypoglycemia. The reason is that you have to continue the insulin drip even after the blood sugar level drops into the 200s; you should continue the insulin drip until “the ketosis is broken” (i.e., until the anion gap closes or the beta-hydroxybutyrate level decreases to around 1 mosm/L.). It takes several more hours to break the ketosis than it does to get the blood sugar levels down; so for several hours you will have to give IV glucose (to prevent hypoglycemia) at the same time you are giving IV insulin. This is why fingerstick glucose levels are checked hourly during the acute stage. You should switch to D5 ½ NS and halve the rate of the insulin drip when the blood sugar gets down to the 200s. If you wait too long to add glucose (or if the pharmacy and nurses take too long to hang the D5 1/2 NS that you have ordered), the patient can develop symptomatic hypoglycemia. In addition, the maximum rate of insulin-driven glucose utilization can occasionally reach 300-600 mg/min (i.e., D10 at 150-300 ml/hour); so some-

times the blood sugar will continue to fall despite adding D5 and slowing the rate of the insulin drip. In these circumstances (albeit rare), you should use D10 instead of D5 in your IV fluids. Be careful if the glucose level drops into the 100s; treatment-induced hypoglycemia increases DKA mortality twofold (3).

Very rarely (usually when there is a severe driving condition like sepsis), the usual insulin drip (0.1 U/Kg/hour – about 7 U/hour) will not be enough. You should expect to see the blood sugar level drop at the rate of 75-100 mg/dL/ hour (it will usually drop a little faster than this at first – especially in HHS – since volume replacement will lower the blood sugar all by itself, by allowing the kidneys to start excreting glucose again). If the glucose level is not falling, you should double the rate of insulin administration. I should mention here that some centers use subcutaneous short-acting insulin to treat DKA and HHS (here we use IV insulin for all except the mildest cases). Using subq insulin every 2 hours is fine as long as you remember that the dose is twice as high as for IV insulin.

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2. Potassium management. Severe hypokalemia is a serious complication. Potassium levels below 2.5 mEq/L are associated with a fourfold increase in mortality (3). This problem arises because the potassium level is almost always elevated to start with – even in HHS (this shows you that the main cause of the hyperkalemia is not acidosis but insulin deficiency and hypertonicity). You should start IV potassium whenever the potassium level drops below 5.2 mEq/L; often you have to give it faster than the usual 10 mEq/hour. If the patient is hypokalemic to begin with, you are in real trouble – that potassium could easily drop into the low 2's during treatment. In such cases, you should wait before starting the insulin drip. Instead, give saline to start the flow of urine and aggressively replace the potassium – I would start with 20-30 mEq per hour (giving K at this rate requires a central line and ICU monitoring). You may end up giving as much as 300 mEq of potassium in the first 24 hours to patients with such severe potassium deficiency. Wait to start the insulin drip until the potassium level rises to above 4 mEq/L.

3. Fluid replacement. This is usually straightforward as long as you assess the patient's free water status by estimating the corrected sodium – that is to say, what the serum sodium will be once you get the glucose level down. Remember that, for every 100 mg/dL that you lower the glucose, the serum sodium will rise by 1.6 mEq/L. Once you get the volume status up with a couple of liters of saline, most patients will need free water (i.e., 1/2NS or D5 ½ NS) to replete their total body water deficit. Since HHS patients are so hypertonic, it may take a day or two to replace free water in these patients.

4. Bicarbonate and phosphorus. This is easy nowadays. You give IV bicarbonate if the pH is below 6.9 (remember that all those ketone molecules will be metabolized to bicarbonate once you start the insulin). Phosphorus replacement is no longer felt to be so important, although I would usually check the PO₄ level after 4 hours and consider replacement if the PO₄ level drops below 1 mg/dL or if the patient shows evidence of severe muscle weakness or heart failure.

5. Being alert to the underlying cause. In about 1/3 of DKA patients – especially those with T2DM – an underlying illness will precipitate the DKA. These can include sepsis, myocardial infarction, pulmonary embolism, or pancreatitis. Most patients with HHS will have a precipitating cause; again, it is the underlying acute illness, not the HHS per se, that accounts for their higher mortality rate. If the underlying cause is not apparent at presentation, remain vigilant for a covert illness. Sometimes, the patient stopped taking their insulin for a reason – i.e., they were sick to begin with.

6. Cerebral edema. We almost never see this complication of DKA in adults, but it is a feared complication in children and carries a 20-40% mortality rate. If your patient with DKA starts to wake up as they should, but then develops mental status changes or focal findings (e.g., cranial nerve abnormalities), you should start treatment with mannitol immediately (even before the CT scan) and be prepared for a rocky course.

Outcome

Although DKA still carries a 10-20% mortality in resource-poor countries, almost all patients in the U.S. should recover – with their 1-2% mortality rate due either to an underlying illness, to the development of cerebral edema, or to hospital-acquired issues like PE or infection. Recurrent episodes of DKA, however, define a high-risk group. Whether due to socioeconomic factors (such as cost) that limit access to insulin, to lack of commitment to the hassles associated with insulin use, or to intrinsically more unstable disease, these patients have a fourfold increase in long-term mortality. As mentioned repeatedly above, patients with HHS have a 10 fold higher mortality to start with, mostly to the precipitating illness or to advanced disease with renal failure, blindness, etc.

In either case, however, both DKA and HHS, although associated with severe metabolic and hemodynamic changes at the outset, will generally respond to modern aggressive management. Successful treatment demands attention to detail and anticipation of rapid fluid and electrolyte fluctuations, but patients usually respond promptly and completely. And an understanding of why they went into DKA or HHS in the first place – e.g., discontinuation of the insulin due to cost or social factors, or a treatable underlying illness – will lessen the likelihood of recurrence of these harrowing and expensive life-threatening episodes.

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The Role of a Diabetes Educator/Dietitian

by Amanda Ast, RD

Imagine walking through a moment as a new patient. In seeking medical care, you are vulnerable. During this appointment, you are asked to share intimate details of your personal life. Not only is this appointment likely challenging, it is also only a blip in time in your day. You have a day and week full of life – a life filled with personal biases, beliefs, expectations, plans, and desires of how to live life. You have an entire repertoire of people – family, friends, coworkers and many others – who have assisted in molding these unique thoughts, feelings, and behaviors.

Now, imagine receiving the diagnosis on diabetes. Those same individual biases, beliefs, expectations, thoughts, and feelings exist within this diagnosis – all this beginning to take shape within seconds of hearing the news.

The words that the patient hears in this moment and in the years to follow create strong impressions. These words can be powerfully impactful in the context of this patient’s grief in dealing with the new diagnosis. Depending on the stage, choosing the right words can lay the foundation for lifelong understanding or acceptance of the conversations between patient and their care team.

A person with diabetes experiences the stages of grief around their diagnosis or the changes of the disease. The role of a diabetes educator/dietitian includes educating the patient on the acceptance of their diagnosis. Without acceptance, reception of education is bleak. Even with the best education, resources, and support, a patient is unable to move forward if they have not learned to accept their diagnosis.

The role of a diabetes educator has always been to teach the patient about their disease, but the goals and the motivators have changed. Dr. Joslin developed diabetes education teams in the early 1900’s. His mantra was “those who know the most, live the longest”. With very little known about diabetes at that time, education was focused on survival, as the expectation was blindness, limb loss, kidney failure, and death. Fear of death was the motivation and purpose of education. Dietary education had focused on restriction for hundreds of years prior to the implementation of insulin. In fact, it was not until 1994 that the ADA released official guidelines on a less restrictive diet, one that included carbohydrates. **From that time and to this day, the nutritional goal of carbohydrates is at least 130 grams a day.**

“I was told to cut out my carbs and sugar.” “I was told to not eat white foods.” “I was told fruit was bad.” “I was told carrots are a bad carb.” “I was told I can’t have carbs.”

Working in diabetes education for the past 20 years, I have heard these quotes continuously from patients. Historically, the foundation of diabetes education has been restriction – so the transition to accepting carbohydrates into the diet has been challenging. Additionally, there is an extensive amount of nutrition misinformation and misunderstanding in American culture and the healthcare community.

Considering the history of diabetes education, it makes sense that there has been some resistance to change. The inclusion of an intensive management approach was debated for years. In the early 1900’s, physicians picked sides between pushing patients for quality of life versus attempting to forestall complications. In the 1930’s, the Joslin Diabetes Center began practicing “Troika”. This three-horse chariot analogy of living with diabetes included diet, exercise and insulin to achieve victory over diabetes. However, it was not until the 1993, when the impactful results of the Diabetes Control and Complications Trial (DCCT) were published, that the evolution of intensive management and individualized education truly began.

Before the 1921 discovery of insulin, physicians prescribed high fat, high protein and no or low carbohydrate diets. It was not a new diet trend, even in those years; we still see this today as the “Keto” or recently the “Atkins” diets. When used to treat patients with diabetes, this restric-



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tion proved ineffective at prolonging a person's life. Carbohydrates are essential to overall body function, and a lack or inadequacy of insulin was still the problem despite chosen fuel sources.

As intensive management gained momentum, identifying the need for carbohydrates in the diet advanced. Even with evidence, the healthcare community remained cautious and restrictive. In the 1980's, we saw the transition to a nutrition treatment focused on correcting the impairment of the body's inability to use, store and retrieve carbohydrates from the diet. This education was given to patients in the form of diet plans, calorie counting, or division into starchy and simple carbohydrate foods. During this transition,

the available insulins resulted in medication-driven timing of insulin and meals.

The 1994 ADA nutritional guideline standards did not include a specific percentage of calorie distribution or a recommendation to exclude particular foods or groups of foods from the diet. The 2022 standards do state that "the recommended approach is to individualize meal plans with a macronutrient distribution that is more consistent with personal preference and usual intake to increase the likelihood for long-term maintenance." These latest standards provide guidance on the inclusion of mixed-meals, variety, and culture-based meal choices. Research shows that improved compliance and glycemic

management is not gained through restriction, but instead through adequate and varied macronutrient-dense food choices. We now educate patients on how to obtain variety and adequate portions – not to remove foods from the plate but to make room for others. Food balance is achieved with individualized approaches that take into consideration the patient's possible dietary imbalances.

America's current diet culture is grounded in caloric restriction to decrease body size. This restriction is promoted by all scopes of healthcare professionals, for overall health benefits. Many recommendations promote weight reduction as a means of preventing and reducing diabetes complications. However, this is often encouraged through restriction, which ultimately leads to a dysfunctional relationship with food and body image. This restrictive approach is not sustainable. It is critical to understand this as a healthcare provider or caregiver. Misinformation and misunderstanding of the role of food in the body manifests in the fabric of our society – from media outlets using snapshots of data to break a story to "influencers" with inadequate medical or nutritional training blaring harmful advice. Unfortunately, patients are listening, and they may lack the ability to discern right from wrong information. This harmful advice is readily available when a person is told they have diabetes. Research shows that the body needs adequate nourishment to reach diabetes management goals. And if the ultimate goal of diabetes management is to improve quality and length of life, quality needs to include how to live their life.

Commonly, a provider does not have available time to invest in individualized patient education. This opens the door to somewhat harmful diet suggestions, similar to the patient quotes I mentioned. No two people are the same, thus no two people will have the same daily routines with food, movement, or diabetes management. The role of the diabetes educator and dietitian is to create a

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patient-driven, individualized approach to diabetes self-management. Diabetes educators create personalized education with a foundation of self-management strategies. These areas are healthy coping, healthy eating, being active, medications and monitoring, and problem-solving to reduce risks in their life with diabetes.

Ultimately, the role of the diabetes educator and dietitian is to provide a translation of complex medical jargon into simple examples for the patient. This approach gives the patient an improved understanding. And, as Dr. Joslin said, “those who know the most, live the longest.”

Healthy Coping teaches a patient how to listen to their body. This step is crucial in first identifying the stage of grief and acceptance of their diagnosis. Secondly, it teaches a patient to find the connection between lifestyle choices and the daily management of diabetes.

Healthy Eating relies on a solid foundation of understanding the role and the function of food in the body. Demonstrating the journey of food in the body offers a visual for the patient to understand the impact of food, medications, and movement in their body. This foundation of nutrition knowledge allows the patient to discern misinformation.

Being Active discourages inactivity, with realistic and individualized goals. Helping a patient understand the benefit of movement connects their relationship of movement with improved quality of life. This approach is more effective than “shaming” a person into moving their body to change their body shape.

Monitoring and Medication Education is best when individualized to the patient’s needs. Simple explanations on the purpose and function of medication improve acceptance and compliance with these tools to manage diabetes. This education also exposes patients to technological advancements now available to promote their quality of life. Educating

patients about the history of why and how monitoring and medications have advanced provides a beneficial connection to their everyday choices and an improved quality of life.

Problem Solving & Reducing Risks teaches a person how to navigate daily management of diabetes with a long-term goal of preventing complications. Reminding patients of the problem-solving already in place in their daily lives can provide a connection to mindfulness as the patient moves forward with diabetes management.

An educator best provides care to patients by implementing these steps into several modes of learning. The use of tactile learning, visual aids and practice improves understanding, which increases quality of life with reduced risks. In addition to instruction, offering patients the opportunity to express their concerns allows the patient to identify where they might be in acceptance of their diagnosis.

As patients receive diabetes self-management education, they are learning how to fish versus receiving a plate of fish. This educational process promotes independence in their own care. Meeting a patient where they are on their journey of acceptance allows proper education to meet their individual needs. Explaining complex medical terms, diagnoses, and medications allows a patient to understand the importance of caring for themselves.

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Exercise in Diabetes

by Celee Spidel, PhD

What if there were a diabetes drug that could lower blood glucose levels and improve insulin sensitivity, and whose side-effects include lower blood pressure, improved lipids and enhanced feeling of well-being? Exercise may not fit the technical definition of a drug, but it is a tool that can do all of the above, with minimal to no adverse effects. And it's free. The Novo Nordisk professional cycling team, comprised entirely of athletes with type 1 diabetes, may be the best and most extreme example of what exercise can do for diabetes. Many of these athletes maintain Hgb A1Cs that rival those of non-diabetics, on less than 20 units of insulin per day! But what about the rest of us lesser mortals? Patient compliance and adherence to exercise programs is essential for

them to work. And it isn't realistic to ask the typical sedentary diabetes patient to bicycle for hours every day. What is the minimum effective dose of this exercise drug for diabetes? Many researchers are attempting to answer this very question.

One randomized cross-over study compared 30 minutes of high-intensity interval training (HIIT) to 30 minutes of moderate intensity continuous (MICT) treadmill walking in 15 volunteers with type 2 diabetes, mean age 60, on oral agents only (4). Both exercise intensities significantly lowered blood glucose levels during and for 50 minutes after exercise, with the HIIT protocol having a significantly greater effect than the MICT intervention. Adherence was 100% in both exercise groups, and the

HIIT, while higher-intensity than the moderate one, was not unrealistic for a middle-aged to older untrained adult. The HIIT session consisted of 5x (3 min at 70% HRR + 3 min at 30% HRR) compared to 30 min at 50% HRR for the MICT session, where HRR= (220-age - resting HR). The grade of the treadmill incline was raised to achieve the correct walking intensity for each subject, making this a safe exercise protocol for older adults. It's encouraging that just 30 minutes of walking can improve blood glucose levels so much.

Perhaps more encouraging is the study by Li, et al. (5) that found that just 20 minutes of moderate exercise (40% HRR) after dinner significantly reduced the 2-hour postprandial glucose spike,

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mean blood glucose level, and peak glucose level in 29 adults with uncomplicated type 2 diabetes on oral agents only. So, it seems that exercise has a dose-response effect on blood glucose levels, with higher intensity exercise being better than moderate steady-state exercise, but even 20 minutes of moderate exercise leading to significant improvements in glycemic control. Time of day may be an important factor, as well, when trying to maximize the efficacy of an exercise program for diabetes. One recent study compared morning and afternoon exercise programs with matched energy expenditure, and found that afternoon exercise is more effective than morning exercise at improving blood glucose levels in individuals with type 2 diabetes (6).

If adding a formal exercise program of any kind seems unlikely to be followed, one group of researchers found that merely increasing the number of active sitting breaks in sedentary individuals with type 2 diabetes reduced glucose levels by 36% and significantly lowered daily glucose AUC (2). Subjects in the “Sit Less” group were instructed to break up their sitting time every 30 minutes with standing and self-perceived light walking. These breaks from sitting added up to a whopping 5 hours less sitting per day, compared to the 14 hour sedentary control. The “Sit Less” group not only outperformed the sedentary control group but also lowered overall daily glucose levels more than the “Exercise” group that cycled for 1 hour daily, suggesting that sitting itself contributes to insulin resistance, and active sitting breaks throughout the day can make a bigger difference than the addition of one hour of exercise for sedentary individuals. In summary, when it comes to aerobic exercise for glycemic control, higher intensity is optimal, but 20-30 minutes of moderate intensity exercise in the afternoon, especially after meals, will lead to significant improvements, and, surprisingly, even taking breaks from sitting every 30 minutes throughout the day can have a profound impact on daily glucose levels in sedentary adults with type 2 diabetes.

Strength training, while often unappreciated and under-utilized in diabetes management, causes multiple beneficial physiological and metabolic adaptations. First of all, resistance training increases muscle mass, which in turn improves body composition, increases resting metabolic rate, and increases the muscle’s glycogen storage capacity. This becomes even more important in older patients to combat the loss of muscle mass that occurs with age. In addition, three months of strength training has been shown to improve insulin sensitivity to a similar extent as moderate-intensity aerobic training in older men and women (1). Strength training appears to increase the pool of Glut4 receptors in trained skeletal muscle and to cause a remodeling of the muscle fiber that allows Glut4 to migrate to the cell surface more easily than in untrained muscle. (3 and unpublished data). The significance of this is that, while aerobic and strength training undoubtedly have some overlapping benefits in diabetes – such as increasing energy expenditure and reducing time spent sitting – strength training improves glycemic control and insulin sensitivity in other ways that are distinct from aerobic exercise, meaning that an ideal exercise prescription for diabetes management will include both types of exercise whenever possible.

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Diabetic Foot Infections

by Scott Milton, MD, FACP

Both soft tissue infections and bony infections (osteomyelitis) have enormous economic consequences, especially in patients with diabetes. Approximately 15% of diabetics will develop ulcers during their lifetime, and around 6% will eventually require hospitalization. Hospitalizations can be long and expensive and usually require both medical and surgical treatment. Diabetic foot infections are most commonly the result of long-standing neuropathic and vascular pathology associated with hyperglycemia. Peripheral neuropathy, with the loss of protective sensation from long-standing diabetes, eventually leads to callus formation. If not carefully observed and managed, this can lead to the development of an ulcer. Diabetic ulcers are usually infected with multiple organisms and can eventually lead to osteomyelitis by contiguous spread of these pathogens and penetration of the periosteum. Vascular disease, both macrovascular and microvascular, is often present, and worsens the severity of diabetic foot infections. While microvascular disease is not amenable to surgical intervention, large vessel disease often can be addressed. Therefore, large vascular disease, specifically peripheral artery disease, should be suspected and diagnosed as soon as possible. This is best diagnosed by peripheral arterial Doppler ultrasound, which is noninvasive and readily available in most hospitals.

Initial Approach to Diabetic Foot Infections

Upon presentation, a careful physical assessment of not only the foot but the involved extremity is necessary. The dorsalis pedis and posterior tibialis arterial pulses should be palpated and assessed. If weak or not present, palpation of the popliteal pulses in the posterior knee area should be performed. The femoral artery in the groin of the affected leg should

also be palpated and assessed. Careful examination of the infected area of the foot should include the size and potential depth of the infected ulcer or wound. An ulcer greater than or equal to 2 cm² in diameter is often associated with osteomyelitis. Further, the “probe to bone test”, where a probe is passed into the wound, is also highly predictive of a bony infection. If the probe can touch bone, osteomyelitis is almost always present and should be managed accordingly.

As the goal of any treatment plan should be the preservation or restoration of a usable appendage, the clinician should keep in mind two important points. First, the most important toe of the foot is the great toe, as it is most important in maintaining balance. Second, the most important bone of the foot is the calcaneus as this bone bears the majority of weight when ambulating. Therefore, careful consideration of these two important anatomical features of the foot must be incorporated into any treatment plan, both medical and surgical. In my experience, calcaneal osteomyelitis is almost never curable by medical therapy alone. Local debridement of the calcaneus in attempt to preserve the foot is usually futile as the calcaneus, by bearing the majority of the weight, cannot heal. Further, chronic osteomyelitis causes softening and degradation of the bony tissues. This pathology in the most important bone of the foot, the calcaneus, almost always renders the foot useless. Therefore, chronic osteomyelitis of the calcaneus usually results in amputation of the foot. The patient should be counseled accordingly if this diagnosis is made. Often, patients will choose an amputation of the foot when presented with likelihood of failure of treatment plans (often long and potentially toxic) designed to preserve the foot.

Diagnostic tests that may be useful in the initial assessment include MRI scan, bone scan, and measurement of transcutaneous oxygen pressures. Plain film x-rays of the foot are usually the initial radiologic procedure, as they are inexpensive and usually readily available. While not as sensitive as MRI or bone scan in assessing for osteomyelitis, valuable information concerning the depth of the diabetic infection can often be obtained. Periosteal thickening of contiguous bone is diagnostic of osteomyelitis. This finding, in combination with a positive “probe to bone test”, is usually all that is needed for the diagnosis of osteomyelitis. MRI or bone scan are often utilized to confirm the diagnosis but usually do not alter the clinical management and therefore are often unnecessary. Furthermore, surgical exploration usually confirms whatever is found on MRI scan. As a result, a team approach, where an experienced surgeon works in tandem with clinicians knowledgeable in the use of antimicrobials and of the management of diabetes, is preferred.

Antibiotic management

As mentioned initially, most diabetic foot infections are polymicrobial, and broad-spectrum antibiotic therapy is usually required. Any surgical procedure should include obtaining deep cultures at the time of surgery – if possible, prior to initiating antibiotics. Cultures obtained from the wound at the bedside are often misleading and, by themselves, should not dictate the choice of antibiotic therapy. Multiple pathogens, both aerobic and anaerobic, are often present and should be considered when choosing a treatment regimen. A history of resistant pathogens such as MRSA or resistant gram-negative pathogens should be sought as well.

There are many antibiotic regimens that are reasonable. Piperacillin/tazobactam (Zosyn) is a broad-spectrum intravenous antibiotic that offers excellent gram-negative therapy (including *Pseudomonas*) as well as anaerobic coverage. Gram-positive pathogens such as MSSA (methicillin-sensitive *Staph aureus*), *Streptococcus* and enterococcus are often covered as well. MRSA (methicillin-resistant *Staph aureus*) is not covered by Zosyn; if MRSA is suspected, the clinician should consider linezolid, vancomycin, or another antimicrobial that is useful in treating resistant gram-positive pathogens. Each has its pros and cons, and having a clinician and or pharmacist familiar with these antimicrobials is extremely useful. I'll make a few important points here for the reader.

The length of any antibiotic regimen is usually dependent upon the ability of the surgical procedure to restore adequate blood flow and to remove nonviable infected tissue, while maintaining function. Resistant pathogens such as MRSA or highly resistant gram-negative pathogens

may extend the length of antibiotic therapy. When ischemic tissue remains, several weeks of antibiotics are required, as ischemic tissue slows recovery. Intravenous antibiotic therapy, which typically offers higher blood and tissue levels, is usually necessary. Linezolid is an exception to this rule. This antibiotic, which only treats gram-positive pathogens, is 100% bioavailable as a tablet; oral linezolid has been shown to be equivalent to intravenous vancomycin. Therefore, this antibiotic is often utilized in the treatment of diabetic foot infections. However, the prescribing clinician should be familiar with the common side effects, toxicities and drug interactions of linezolid and should monitor for these during the treatment course.

Ertapenem is a once-daily carbapenem that is also often used in diabetic foot infections. This antibiotic can only be given intravenously but has several advantages. Being a carbapenem, ertapenem offers excellent gram-negative coverage including resistant extended-spectrum beta lactamase (ESBL)-producing organisms. The

one important exception is *Pseudomonas*, and this antibiotic should not be used if *Pseudomonas* is obtained in a wound culture. Ertapenem also offers excellent anaerobic coverage as well as decent gram-positive coverage including strep and sensitive staph. Ertapenem is usually very well tolerated, but the clinician should be aware of neurologic side effects including seizures. The risk of seizures is around 1%; the elderly and those with impaired renal function are more susceptible to neurologic side effects including seizures. Therefore, ertapenem is most useful in an outpatient setting in patients who have normal renal function and are relatively young. Any patient with history of seizures, recent head trauma, or brain tumor should only receive ertapenem if other regimens are exhausted and patients can be monitored closely. When given to elderly patients for longer than two weeks, I have noticed neurologic decline and confusion more commonly than seizure activity. Once again, the clinician should consider

| continued on page 40

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consulting with a knowledgeable pharmacist or infectious disease physician in scenarios as complicated as that described above.

Fluoroquinolones should also be mentioned here as a reasonable alternative to or in addition to the above. As a class, the fluoroquinolones are highly absorbed, and oral therapy can be considered equal to intravenous therapy. The fluoroquinolones are the only class of antibiotics that offer an oral treatment of *Pseudomonas*. Levofloxacin offers better gram-positive activity than the other fluoroquinolones and has some anaerobic coverage as well. Moxifloxacin is a later generation quinolone and offers even better anaerobic coverage while preserving activity against gram-positive and gram-negative pathogens. Ciprofloxacin is considered a better gram-negative antibiotic in comparison to the other quinolones. It is also usually cheaper. The quinolones also have many side effects, which should be considered when selecting a treatment regimen. Neurologic side effects, including nightmares, confusion and seizures, have all

been well described and appear to be worse in elderly patients and those with renal insufficiency. Tendinopathy – including tendon rupture – can occur with fluoroquinolone use, and the patient should be instructed to avoid activities that involve strenuous contraction of arm and leg muscles. Some newer evidence suggests that the fluoroquinolones can induce *C. difficile* colitis more commonly than other classes. This has led to efforts to reduce the use of quinolones in the hospital. Once again, a knowledgeable pharmacist can be quite useful when prescribing this class of antibiotic.

In summary, diabetic foot infections can be a devastating complication in individuals who struggle in the management of this horrible disease – the subject of this issue of Panhandle Health. Prevention is best accomplished by strict glycemic control and smoking cessation. Every patient with diabetes should be instructed to inspect their feet every day. This should include the inspection of the soles of their feet. All diabetic patients should have their feet covered all the time with protective

footwear. House shoes should be at the foot of the bed, readily accessible for use at night. Clinicians should regularly examine their patient’s feet; this should include a neurologic assessment. Custom-made diabetic shoes should be considered in those who developed neuropathy. Referral to a skilled podiatrist is most useful in preventing severe diabetic foot infections and, in my opinion, should be offered when neuropathic diseases is diagnosed. As we all know, “an ounce of prevention is worth a pound of cure”, and diabetic foot infections and the complicated care that follows exemplify how true is this statement by Benjamin Franklin.

Dr. Scott Milton attended the University of Texas Medical School in Houston. He completed his internship and residency at the Medical College of Georgia. Dr. Milton did a Fellowship in Infectious Diseases at Vanderbilt University. He is Board Certified in Internal Medicine and Infectious Diseases and is a member of PRCMS. Dr. Milton is currently Regional Medical Director, Public Health Region 1, TDSHS.

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Heal The City Diabetes Care: Transforming One Life at a Time

by Alan Keister, MD & Maren Brady, Clinic Operations Manager – Heal the City Clinic



Cindy arrived at Heal the City by referral from a local agency complaining of thirst, frequent urination, and weight gain. She felt poorly and was certain something was wrong. As a widowed mother of two daughters, she was anxious to find out what was going on. After evaluation, Cindy was diagnosed with new onset diabetes. She underwent a comprehensive diabetes education and management assessment through the Shalom Chronic Care program at HTC. Her sugars began to improve with the addition of medicines. She also went to wellness classes that taught her how to shop for good foods at the grocery store. Soon Cindy began to get control of her diabetes. She started taking ownership of her disease and started to lose weight intentionally through diet and regular exercise classes at HTC. Cindy also saw an added benefit when she made dietary changes. Her 2 girls, who had been bullied about being fat, also started to lose weight and improved their self-image. Obviously, Cindy is a dramatic success story in both her individual response as well as the secondary benefits to her children and a chance to avoid generational chronic disease.

At Heal the City Free Clinic, we manage our patients in our chronic disease management clinic called the Shalom Clinic. Shalom is a Hebrew word that is often used as a greeting, but the word has a richer meaning. Shalom communicates the idea of wholeness, wellness, completeness, and flourishing. Our goal for diabetes care in the Shalom clinic is to provide this type of comprehensive whole-person care for our patients. This approach involves medication, education, access to food and wellness opportunities. Finally, we seek to measure and improve outcomes.

Caring for indigent patients with diabetes presents significant challenges to the

health care system. The cost of medications and insulin continues to skyrocket. Many people must make difficult decisions about managing their healthcare or taking care of their family. Some patients can only afford the cheapest diabetic meds which often do not control their sugars and may not protect them from end organ damage. So, patients and their physicians are often stuck trying to find meds that will help somewhat but do not give optimal sugar control. The pharmaceutical companies do have patient assistance programs that can help. Qualifying for these programs is onerous and requires documentation of patient finances and personal information to determine if they will qualify. Even if they do qualify or can afford the meds, they still ultimately must take them to improve their sugar control. Most patients in our clinic live in chaos mode. It is difficult for them to see the importance of tight control when they have no symptoms and often will have no diabetic complications for several years.

One of the solutions at HTC for our Shalom patients is that we can get meds for our patients through several avenues. One of our employee's sole job is to run the Medication Assistance Program or MAP. The MAP program allows the clinic to get newer, name-brand medication from the drug manufacturers. Our staff helps the patients collect all their financial records to speed the process

Jose arrived at HTC with a long history of diabetes and a strong family history of diabetic complications. He admitted that he occasionally forgot to take his diabetes meds, but he really did not want to be on insulin. He reported that he occasionally skipped meds when he did not have time to eat at his job. He noticed his vision has changed a bit, but he cannot afford to see an eye doctor. He also complained of some

burning in his feet at night that interferes with his sleep.

Along with the difficulty of navigating medication acquisition, education is another issue for patients. Many come to the clinic believing that, if they just take their medication, they will be ok. We spend time counselling them on the importance of diet and exercise in addition to meds. We spend time discussing what a hemoglobin A1C is and why it is important to understand how this lab reflects the average of their sugars over the last few months. We talk about the dangers of hypoglycemia or low blood sugar and the dangers of uncontrolled diabetes. We stress the importance of routine eye exams, and we can provide them on site (pun intended) through the generosity of local optometrists. We also talk about other warning signs like microscopic protein in the urine (microalbuminuria), and we screen for this regularly to assess any possible diabetic kidney damage. We also educate our patients on diabetic neuropathy and the importance of diabetic control to prevent this complication. What makes this process a challenge is that most of our patients speak only Spanish, and many have a limited education. Finding the best ways to communicate the nuances of managing a chronic disease with this population represents a unique challenge. Heal the City staff do an amazing job of investing in the Shalom patients to see true transformation.

David arrived at Heal the City with uncontrolled diabetes. He was eager to get on track and get his sugars under control. He went to his diabetic education classes and got his medicines from the pharmacy. When he returned to check on his progress, his labs were worse. After talking with him, he reported that he often did not take his medicine when he was uncertain if he was

going to have anything to eat that day. He also admitted that transportation was an issue and he could not always get to the pharmacy during hours it was open. He reported that he was not exercising because his apartment was small, and he did not have a safe space outdoors to walk.

As the last issue of *Panhandle Health* demonstrated, social determinants of health play a huge role in diabetes care for patients who experience poverty. One of the most important assessments done on each Shalom patient is a survey of their basic needs including questions about food insecurity. Three out of four patients in Shalom experience concerns about hunger at some point during the year. Managing diabetes in patients with limited access to food is a real challenge. Through collaborations with High Plains Christian Ministries, Snack Pack 4 Kids, High Plains Food Bank, and Hillside Church, HTC is working to address the hunger issue in our patients. When their basic needs are met, patients can focus on their diabetes issues. In addition, HTC requires Shalom patients

to participate in wellness classes. Our new FIT Center allows patients to focus on their health through exercise classes and nutrition classes.

Comprehensive care sounds good in theory, but does it make a difference? Our data show that it can. 75% of diabetics lowered their HbA1C over the prior year (HbA1C is a measure of the average blood sugar over the last 6-8 weeks). Only 16% of diabetic patients in Shalom had a HbA1C over 9. In fact, 48% of diabetic Shalomies had a HbA1C < 7, demonstrating excellent control. 80% of our diabetic patients had a diabetic eye exam in the last year. In addition, 80% of our diabetic patients have a blood pressure <140/90.

In conclusion, Heal the City is tackling the challenge of diabetes management in the indigent patient population through a comprehensive program. It begins with a welcoming environment to educate our patients and provide proper medications. It seeks to address key social determinates of health like access to food that may

affect our patients' compliance. It expands to incorporate wellness so our patients can take ownership in healthy life choices like exercise. Finally, we track our data so that we can see true changes in the health of our patients. We have the privilege of watching our patients transform before our eyes.

Maren Brady is originally from Newtown, CT and graduated from Baylor University in 2020. She currently serves as the Mission Director at Heal the City Free Clinic in Amarillo. At HTC, she enjoys working alongside a dedicated team of individuals passionate about providing high quality care to uninsured individuals in the Panhandle area.

Dr. Alan Keister attended medical school at the University of Texas Southwestern Medical Center. He completed his internship and residency at the Vanderbilt University School of Medicine. Dr. Keister is board certified in Internal Medicine. He is a member of PRCMS and is currently associated with Amarillo Medical Specialists.

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Elliott Joslin, MD and the Joslin Clinic: *A Brief History of Diabetic Monitoring in the United States*

by Rouzbeh K. Kordestani, MD, MPH

Incidence and Population Impact of Diabetes in the United States

Diabetes mellitus is an ever-increasing disease that seems to be reaching epidemic proportions in the United States. A recent census placed the number of individuals with diabetes in the US population at 34.2 million, or approximately 11%. While diabetes was more prevalent in the African American and Hispanic communities decades ago, the incidence has now risen across the spectrum. Between 1990 and 2009, there was a consistent increase (4.4%) year over year in those afflicted. More recent tabulations show that the increase in diabetes now affects all ages, sexes, races, ethnic groups and education levels--no population is unaffected or spared. In fact, the United States has the highest prevalence of diabetic individuals between the ages of 20-79 of all the developed countries on Earth. Most of the recent increase seems to be directly related to the levels of obesity and severe obesity seen in the population. As the number of patients affected has risen, the costs of treating diabetics have exploded. Cost analyses place the price tag of diabetic management at a staggering \$327 billion (2017). This crippling cost comes from direct hospital care, prescriptions and medical supplies, and doctor's visits/treatments.

In an effort to help blunt such a crippling disease, efforts have been directed for many years now to diagnosing diabetes early and also to predicting the affected populations. Efforts to understand diabetes mellitus have a multi-faceted and colorful history. That being said, however, very few efforts are more notable than that of the Joslin Clinic in Boston and, specifically, of Dr. Elliot Joslin.

The Early Years of Elliott Joslin

Elliott Joslin was born in 1869 in Massachusetts and showed early promise

in his interests in science. After graduating from Yale, he continued his scientific training by getting a masters in physiological chemistry. Early on, because of his interests in his aunt's diagnosis of diabetes, he began to research what was already known to that point about diabetes. He also began to keep a diabetes registry, a personal diary of sorts of individuals he encountered with the diagnosis of diabetes and their particular presentation(s) and their progress.

Dr. Joslin's astute focus and diligent note-taking eventually led to his first publication in 1916. "The Treatment of Diabetes Mellitus" by Dr. Joslin was the result of his early personal registry. In this first publication, Joslin carefully and painstakingly detailed the presentation, the diagnosis, the treatment and the course of diabetes and the complications witnessed in his first 1000 patients in the Boston clinics. Through his observations, he showed how impactful dietary modifications and restrictions were in regards to patients' clinical progress and development of complications.

Even before the publication of his first book, Dr. Joslin had noted and had lectured about the beneficial effects of dietary restriction in diabetics. As early as 1908, he had collaborated with fellow physiologists at Harvard Medical School to show that fasting and dietary restrictions dramatically improved the clinical progress of patients suffering from diabetes.

In 1918, towards the end of World War I, while still in Boston, Dr. Joslin published the first edition of his "Diabetes Manual for the Doctor and the Patient" (later to be called the Joslin Manual). This manual would go on to be updated and published in 13 further editions and would become the mainstay of standardized hospital-based diabetic practice in

the United States. The manual and its subsequent editions detailed to patients how best to take control of their disease. Much of this work was based on Dr. Joslin's own understanding of the disease and the extensive journals from his personal encounters and from patients admitted to Deaconess Hospital in Boston.

With Dr. Joslin's focus on dietary management and education, he and his clinic had a tremendous impact on patients and patients' survival. In 1922, when insulin became available, these doctors and nurses formed a team of educators that helped establish diabetes teaching throughout New England. The impact of Joslin's work was further evidenced by the development of the first blood glucose monitoring system in 1939. The strict monitoring system eventually led to the invention of the modern glucometer.

From his experience, Dr. Joslin very early on noted an increased incidence of diabetes in the population, especially among adolescents. He was the first person in a position of medical authority to raise concerns about this epidemic. Directly because of Dr. Joslin and his personal efforts, the Surgeon General of the United States authorized studies of the increased prevalence of diabetes in the US population at large and furthermore made diabetes a focus of the efforts of the medical core.

The Joslin Clinic

Dr. Joslin and his team of collaborators and educators had formed a unique practice setting, solely focused on one disease, diabetes mellitus. In 1954, this practice officially became the Joslin Clinic and joined the Harvard Medical School group of institutions in Boston. Even though Dr. Joslin died in 1962, the Joslin Clinic continued his work and does so even today.

The Joslin Clinic survives today as a unique entity with its sole focus on diabetes and diabetic-related maladies and problems. It has the largest staff of board-certified physicians treating diabetes in the world. It also houses and funds the world's largest diabetic scientific research staff with more than 350 researchers. Its achievements and accomplishments are numerous. The following are some examples of note. Through research completed at the Joslin Clinic, the infant mortality rate of diabetic mothers dropped significantly. In 1924, before the clinic made infant mortality a focus, the rate was 46%. Now it is less than 5%. In the 1970's, researchers at the Joslin Clinic developed and perfected the hemoglobin A1C test to assess diabetic control on a more long-term basis. In the 1980's, additional research at the Joslin Clinic led to a much better understanding of insulin resistance in type II diabetics and obese patients. In the 1990's, newer molecular research at the Joslin led to the identification of molecular pathways for diabetic resistance and helped pave the way for the development of diabetic receptor targets for new drugs.

In a more recent testament to the work completed by Dr. Joslin in his early years and his team at the Clinic, population research has shown the benefits of diet and exercise in the diabetic population. The Diabetes Prevention Program (DPP) research has confirmed that populations at-risk for type II DM can affect a more than 50% reduction in their risk for the disease and its complications with moderate weight loss and increased exercise. This was the foundation of Joslin's focus and his teachings.

Over the 50 years since Dr. Joslin's death, the Joslin Clinics have expanded to multi-disciplinary care with a singular focus on diabetes. The clinics in Boston now include some notable additions. The Beetham Eye Institute in Boston is part of the Joslin Clinic Group and is designated with the study, development and treatment of advanced ophthalmologic problems in the diabetic population. It is also tasked with the development of diagnostic tools for these same patients.

The Joslin Pediatric Clinic focuses on the care of pediatric diabetic patients. As the number of pediatric patients with diabetes has skyrocketed, the pediatric clinics have been tasked with the care of this specific population and its needs. There are also primary health and nephrology clinics associated with the Joslin Clinic in Boston. Finally, the Joslin Clinic has associations with multiple foreign clinics, extending its focus on diabetes and diabetic teaching throughout the world health community.

Conclusions

Dr. Elliott Joslin focused on the study of diabetes and, through his careful analysis and treatment, demonstrated the benefits of dietary management and exercise. He often stated – "Education is not part of the treatment of diabetes – it IS the treatment." His teachings helped create a better understanding of diabetes and did much to help treat the disease process in the growing modern population. It is because of Dr. Joslin--his research, his team members, and his fellow educators--that we have a better understanding of the prevalence, incidence, and associated co-morbidities of diabetes. Because of his efforts and his singular focus, thousands of lives have been saved and many thousands more will be saved in the future.

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Continuous Glucose Monitor (CGM) Report Primer

by William C. Biggs, MD, FACE

Patient-connected devices that continuously report physiological data to your office are now commonplace. Continuous Glucose Monitors measure the glucose in interstitial fluid under the skin. They correlate well with capillary or venous glucose measurements but have a slight time delay to take into account. Patients can view the CGM results on their watch, phone, insulin pump or a CGM reader.

Many CGM patients have a report app on their phone and can share it with you at their office visit. If you have a number of CGM patients, you can set up an online professional account at no charge, where patients can share their CGM data in real time with you. This saves an enormous amount of time at patient visits. The cloud reporting sites are available from Medtronic (CareLink), Abbott Libre (Libre View), and Dexcom (Clarity). All these companies have cooperated to provide reports with similar data elements, so you only need to learn this once.

There are two basic components of the CGM report: the Overview, and the AGP report.

The key performance indicators are:

Number of Days worn: Ideally this is every day

Percentage of time active: Minimum acceptable is 70%

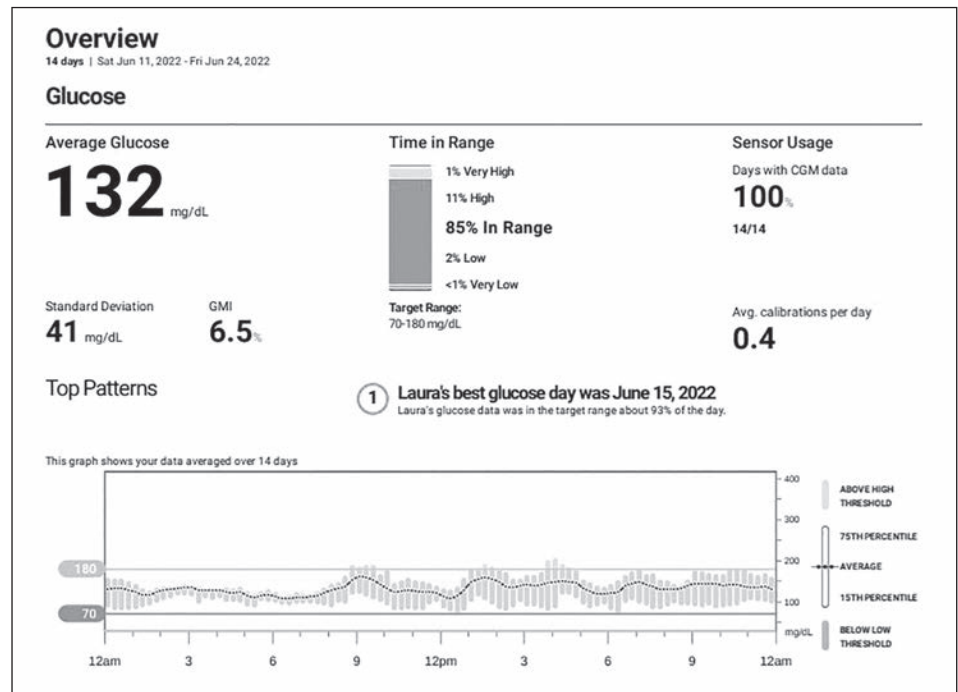
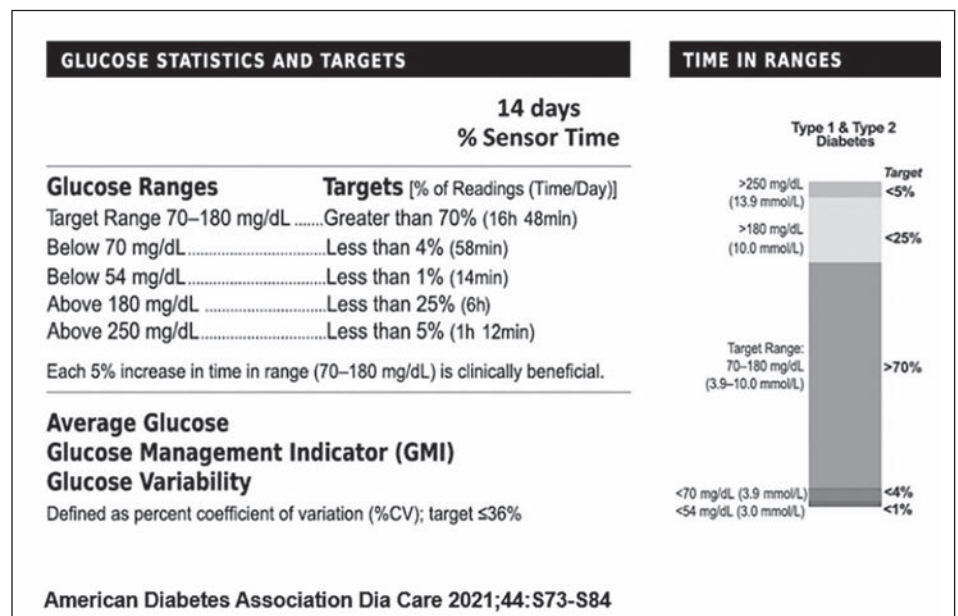
GMI – Glucose Management Indicator. This is the estimated glycohemoglobin (HbA1C) based on CGM data. Since the data is for only 14 days by default, it may deviate from the actual A1C. The interactive online reports allow you to change the 14 days to 30 or 90 days which will help improve accuracy.

“Time In Range” – TIR. TIR is rapidly replacing the HbA1C as the key metric of diabetes control. All clinical trials conducted recently use Time in Range. First, recognize that patients rarely have 100% TIR. The default glucose range is 70 – 180 for most patients. TIR can be changed in

the app or in the report software for special situations. In pregnancy, use a TIR of 65 – 140.

The Ambulatory Glucose Profile (AGP) superimposes all 14 days of data and graphs them by time of day. Each

Recommended TIR levels:



time has an average (bold burnt orange line), a blue shaded zone that includes the middle 25th to 75th (Interquartile Range), and dotted lines indicating the 10th percentiles and 90th percentiles. These help you easily identify problem times of day and filter out the noise from random events.

Coefficients of Variation: Target is < 36%. Lower targets such as 33% provide more protection against hypoglycemia for those on insulin or sulfonylureas.

Individual daily plots allow you to scan for episodes of significant hypoglycemia or hyperglycemia, and to see any gaps where data wasn't captured. It helps to have correlation from the patient on the

timing of meals, medications and activity.

A common workflow for reviewing CGM reports with patients is called DATAA.

Download or View data with the patient. (Ideally the patient has done this before arrival to your office.)

Assess Safety: Review time below range and hypoglycemia, discuss potential reasons and offer realistic solutions.

Time in Range: Review progress towards the patient's time in range goals.

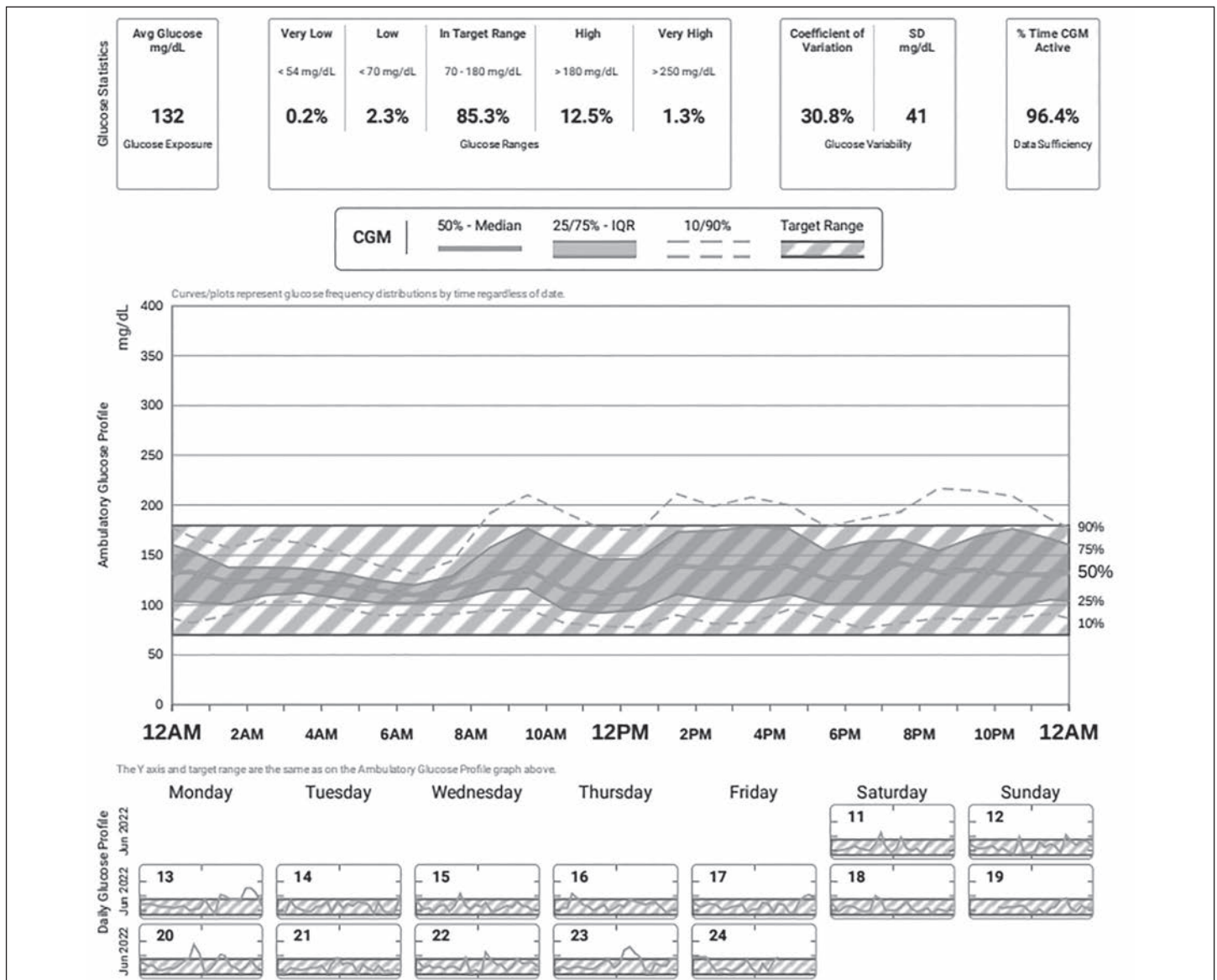
Areas to Improve: Review time above range and identify causes, solutions, and adjustments to self-management.

Action Plan: Discuss potential changes in the treatment plan.

When medically necessary, your review and documented report of a CGM interpretation can be CPT coded as 95251 every month. CGM interpretation can be provided as an in-person at an office visit or as a standalone remote patient monitoring service. Any other E&M encounters the same day of service need to have a -25 modifier.

Dr. William Biggs attended the University of Texas Southwestern Medical School in Dallas. He completed his internship at the University of California in San Diego and a residency at Harvard Medical School in Boston. Dr. Biggs is board certified in Internal Medicine. He is a member of PRCMS and is currently associated with Amarillo Medical Specialists.

Ambulatory Glucose Profile





Cannabinoids as a Cause of Coronary Vasospasm Leading to Myocardial Infarction

by M Atif Khan, MD; M Kashif Amin, MD; Rohan Anand, MS4; Jasmin Rahesh, MS4; Faiza H Khan, Waqas Rasheed, MD; Bala Mohanakrishnan, MD; Asma Zakir, and David Brabham, MD

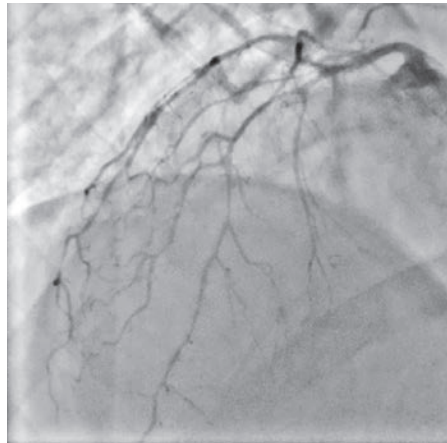
Marijuana has long been popular as an illicit substance in the United States and the rest of the world. Coronary artery disease is a particularly lethal sequela of cannabis use. We present a case of diffuse coronary vasospasm related to marijuana use, leading to myocardial infarction.

Case report

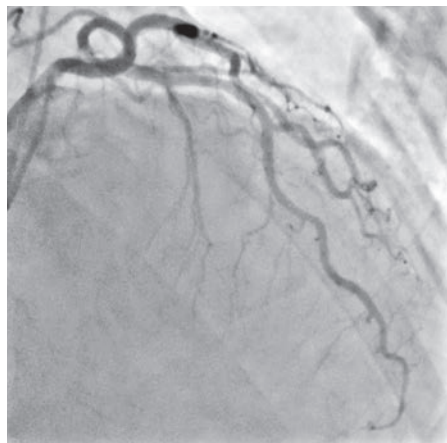
A 53-year-old male with a past medical history of hypertension, hyperlipidemia, and COPD woke up at 3 AM with severe shortness of breath unresponsive to his albuterol inhaler. Three hours later, he reported severe chest pain and became unresponsive. EMS was called and found the patient to be in ventricular fibrillation. CPR was initiated, and the patient was intubated. He was shocked a total of 5 times, and was given 4 rounds of epinephrine and one round of amiodarone. Return of spontaneous circulation was achieved after 30 minutes. In the emergency department, laboratory studies revealed serum creatinine 1.5 mg/dl, HCO₃ 13 mmol/L (normal 22-26), and lactic acidosis with pH 7.04. Troponins and B-type natriuretic peptide (BNP) were within the normal range. Admission EKG showed no ST-segment changes. Urine drug screen was positive for marijuana.

The patient underwent immediate Left Heart Catheterization (LHC) (Figure 1), which showed severe spasm involving all 3 coronary vessels, responsive to intra-coronary nitroglycerin. Otherwise, no significant obstructive coronary disease was noted. Six hours after LHC, the patient developed ST elevation in leads V₃-V₆ and a wide QRS rhythm lasting for 20 minutes associated with hypotension (Figure 2). At this point, the patient was still intubated with good oxygen saturation and stable electrolytes. EKG changes resolved

Figure 1 Initial coronary angiogram



After nitroglycerin infusion



with increasing rate of nitroglycerin drip, and hypotension responded to IV fluid bolus; he did not require pressor support. Troponin HS drawn at this time was found to be elevated at 197 ng/L. Repeat LHC was not deemed necessary, but the patient was switched from carvedilol to diltiazem. There were no further notable events. The patient was discharged 8 days after admission and counseled to abstain from marijuana.

Discussion

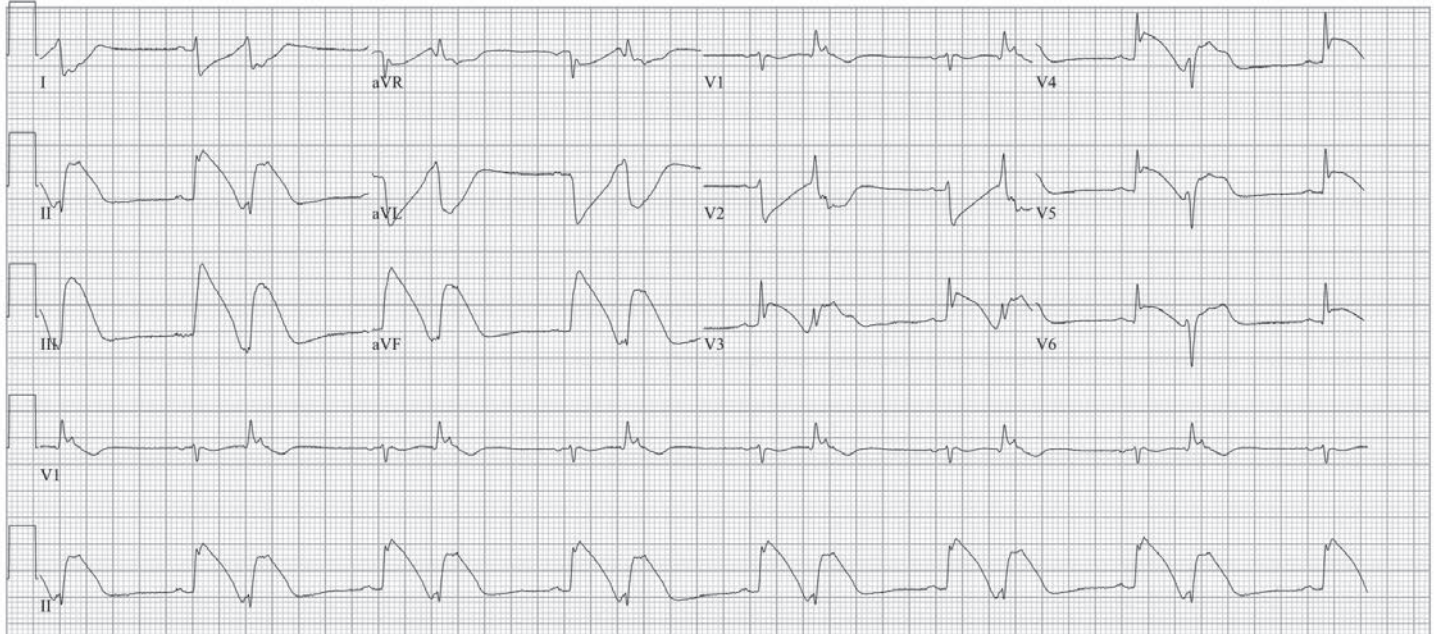
Marijuana is one of the most widely abused substances in the United States.

When smoked, THC results in a rapid, dose-dependent tachycardia by 20-100%, an increase in blood pressure, and an increase in cardiac output by > 30% (3). Anxiety, panic, impaired attention, and psychosis are sometimes experienced as well. Several EKG abnormalities have been identified, including sinus tachycardia, premature ventricular contractions, left or right bundle branch blocks, and ventricular tachycardia or fibrillation (4, 5).

Myocardial infarction (MI) is a rare complication from marijuana use. Proposed mechanisms are linked to a five-fold increase in carboxyhemoglobin, an increase in factor VII activity, and THC-induced hemodynamic stress (6, 7), resulting in a lower ischemic threshold. Such factors can contribute to the disruption of preexisting atherosclerotic plaque, resulting in coronary artery occlusion and subsequent MI, which has been reported in numerous cases in the literature. Mittleman et al. found the relative risk of MI to be 4.8 one hour after THC exposure and 1.7 after 2 hours (8). Less commonly, the symptoms of MI are found to occur in patients exposed to marijuana in whom subsequent workup reveals patent and clean coronary arteries (9, 10).

Synthetic cannabinoids, such as Spice and K2, are rapidly gaining popularity among recreational drug users. Despite being marketed as a safer alternative to conventional cannabinoids, the active ingredient, JWH-018, has been found to be four to five times as potent as THC and is associated with similar physiological effects (2, 6). Perhaps most concerning to clinicians is that these synthetic cannabinoids are undetectable on standard urine drug screen (UDS).

Figure 2



MI as a sequela of synthetic cannabinoid use is rare but has previously been reported, both in the presence and absence of atherosclerotic coronary plaque (1, 5, 6). Our patient was unusual in that his MI symptoms were due to global vasospasm of the coronary arteries, which we theorize was related to marijuana toxicity, perhaps augmented by undetected synthetic cannabinoids. Synthetic cannabinoids are not routinely tested for; indeed, Mir et al. noted that, in one patient actively tested for synthetic cannabinoids, the result was negative despite the patient affirming to use of K2 (5). It is important to note that hundreds of chemical variants are likely in use relative to the few that clinicians are aware of.

The workup for such patients, therefore, is identical to the workup for any patient suspected of acute coronary syndrome (ACS). Patients presenting with symptoms of MI, even when substance use is suspected or known, must still be evaluated according to standard protocols based on risk stratification. Several of these patients will require LHC, which may or may not reveal evidence of obstructed coronary vasculature. The diagnosis of substance-induced angina is a diagnosis of exclusion, with the particular substance identified through a careful patient his-

tory, UDS, and the clinician's awareness of current commonly abused substances.

In patients with low to absent risk for cardiovascular events, particularly pediatric and young adult patients, presenting with symptoms of MI, substance-induced MI should be suspected. The detection of synthetic cannabinoids is not a part of routine UDS, therefore the clinician must maintain a high index of suspicion when evaluating the patient for possible etiologies for their ACS.

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