MiCMRC Educational Webinar

Caring for the Diabetic Patient: Understanding Diabetes

March 27, 2018



MiCMRC Educational Webinar Caring for the Diabetic Patient: Understanding Diabetes

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Disclosures

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MiCMRC Educational Webinar

Wednesday, June 7, 2017 - 2:00pm Team Based Care Related to Addressing Social Determinants of Health

WEBINAR SOCIAL DETERMINANTS OF HEALTH

Presented by

Cherry Health Care Team

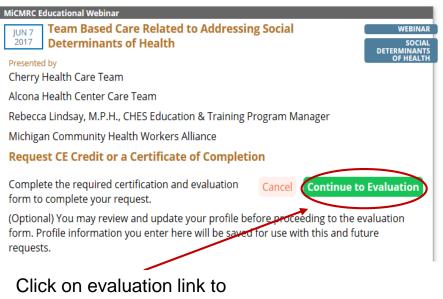
Alcona Health Center Care Team

Rebecca Lindsay, M.P.H., CHES Education & Training Program Manager

Michigan Community Health Workers Alliance

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Caring for the Diabetic Patient: Understanding Diabetes

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Objectives

- Describe the disease process and progression in the patient diagnosed with Type 1 or Type 2 Diabetes.
- 2. Discuss the effect of acute illness on diabetes glycemic control.
- 3. Describe care management issues in a patient with diabetes transitioning from acute to primary care.

Diabetes Mellitus

 Derived from Greek word for "fountain" and Latin word for "honey"



 From Polyuria of untreated Diabetes and the Sweet Smell/Taste of the urine

Types of Diabetes



Type 2 Diabetes

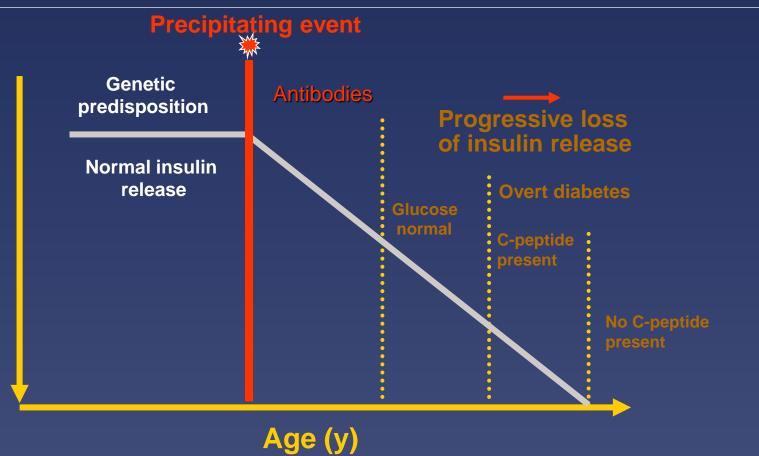
Type 1 DM

- Most commonly diagnosed among Caucasians
- Peaks at 12 yrs of age



- Characterized by immune process in genetically susceptible individuals
- Beta cell destruction and Islet Cell Antibody appearance
- Macrophages T & B lymphocytes and natural killer cells often present
- Viral infections likely cause autoimmune damage to beta cells

Progression of Type 1 Diabetes



Adapted from Atkinson. Lancet. 2002;358:221-229.

Beta-cell mass

Muscle unable to use glucose due to low insulin

Glycogen and protein breakdown, causing keto-acidosis

TYPE 1 DIABETES

Pancreas

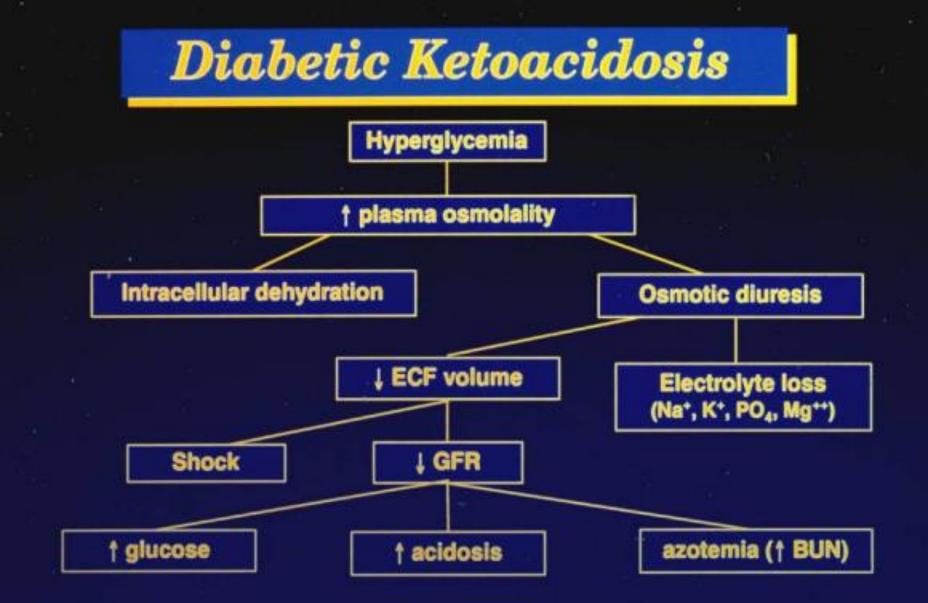
Decreased insulin in the blood vessels

Increased

glucose due

to low

insulin



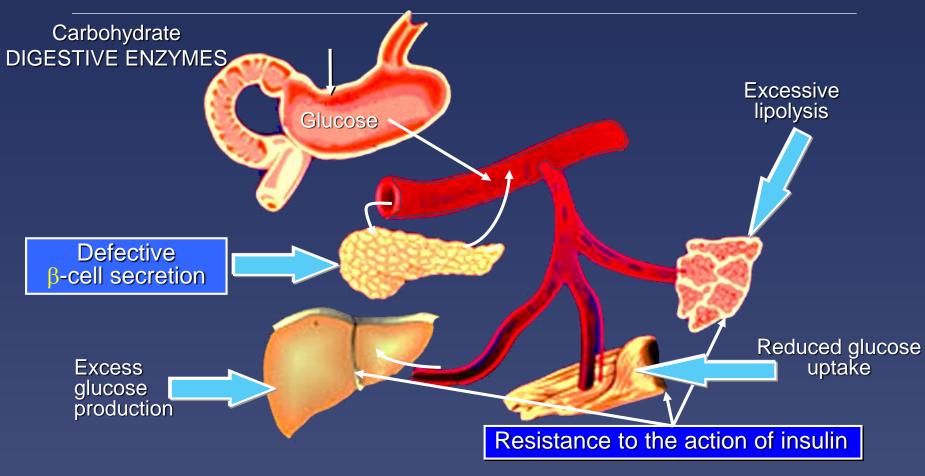
Pathophysiology of diabetic ketoacidosis

Latent Autoimmune Diabetes of Adults

- Form of Autoimmune (Type 1 DM) The presence of autoantibodies to pancreatic islet cell antigens is the element common to both type 1 diabetes and LADA
- Alternate terms: Type 1.5 or Slow Onset Type 1
- Patients often mistakenly thought to have Type 2 based on time of diagnosis
- Slowly progressive form of autoimmune diabetes mellitus characterized by older age at diagnosis, the presence of pancreatic autoantibodies, and the lack of an absolute insulin requirement at diagnosis.
- Although patients with LADA present with more preserved beta cell function than those with classic type 1 diabetes, they tend to have a rapid and progressive loss of beta cell function necessitating intensive insulin intervention
- Rapidly unresponsive to oral medications and parenteral agents, such as incretin mimetics.

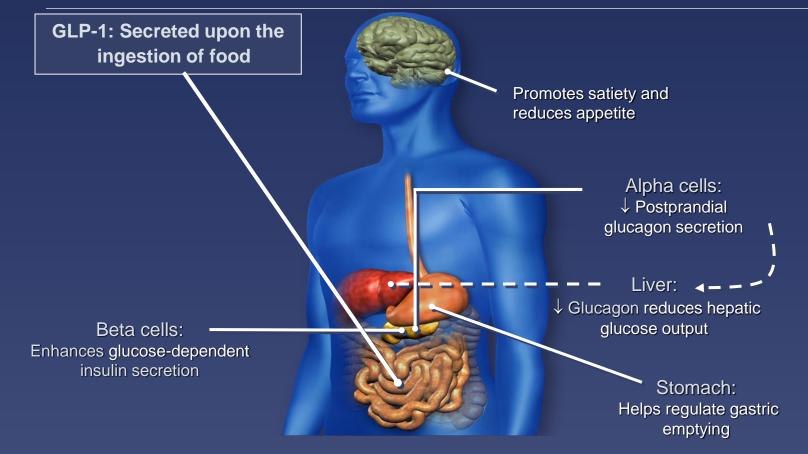


Pathophysiology of Type 2 Diabetes



Dinneen SF. Diabet Med. 1997; 14 (Suppl 3): S19-24.

GLP-1 Modulates Numerous Functions in Humans



Data from Flint A, et al. *J Clin Invest*. 1998;101:515-520; Data from Larsson H, et al. *Acta Physiol Scand*. 1997;160:413-422 Data from Nauck MA, et al. *Diabetologia*. 1996;39:1546-1553; Data from Drucker DJ. *Diabetes*. 1998;47:159-169



Diabetes Response in Illness

Infection causes a stress response in the body by increasing the amount of certain hormones such as cortisol and adrenaline.

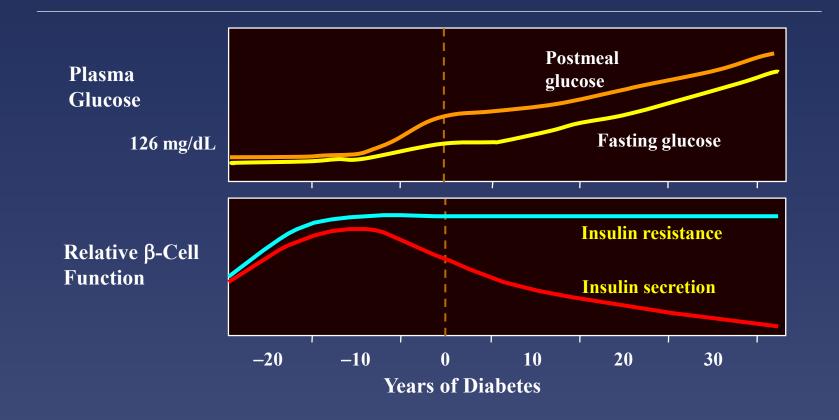
These hormones work against the action of insulin and, as a result, the body's production of glucose increases, which results in high blood sugar levels.

>When blood sugar is high, the white cells in are unable to respond to bacteria because they cannot move around at their normal speed and do not reach the infection site quickly enough to engulf and kill the bacteria.

In a person who does not have diabetes, extra insulin is produced to counter these effects, but this is not possible for someone with diabetes, and so hyperglycemia persists

Illness and Diabetes. Diabetes Wellness Foundation. Retrieved from: https://www.diabeteswellness.net/sites/default/files/Illness%20and%20Diabetes.pdf

<u>TYPE 2 DIABETES... A PROGRESSIVE DISEASE</u> Natural History of Type 2 Diabetes



Categories of Glycemia

Fas	sting (mg/dL)	2-Hour (mg/dL)	A1c
NORMAL	65-99	80-140	< 5.7%
IMPAIRED FASTING		Pre	
GLUCOSE	100-125		
IMPAIRED GLUCOSE		Diabe	etes
TOLERANCE	141-199		5.7-6.4%
DIABETES	>126 (X 2)	>200	>6.5%

American Diabetes Association (ADA) Glycemic Recommendations for Nonpregnant Adults with Diabetes

A1C	<7.0%*
Preprandial capillary plasma glucose	80–130 mg/dL*
Peak postprandial capillary plasma glucose ⁺	<180 mg/dL*

*Goals should be individualized.

 \dagger Postprandial glucose measurements should be made 1–2 h after the beginning of the meal, generally peak levels in patients with diabetes

ADA. 6. Glycemic Targets. Diabetes Care 2015;38(suppl 1):S37; Table 6.2

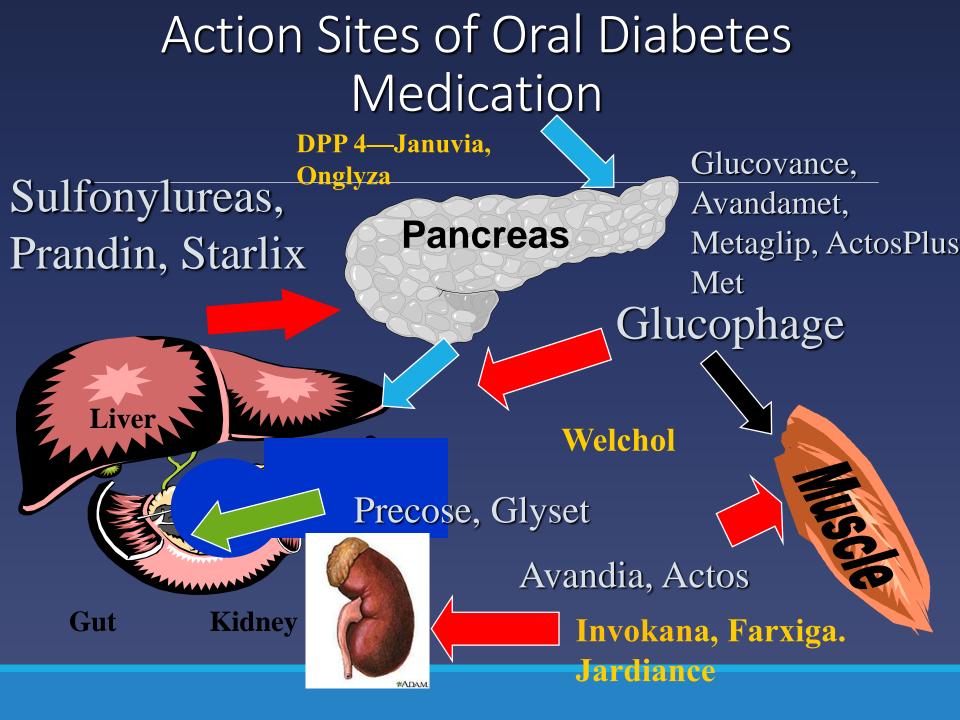
Lifestyle Therapy

RISK STRATIFICATION FOR DIABETES COMPLICATIONS



INTENSITY STRATIFIED BY BURDEN OF OBESITY AND RELATED COMPLICATIONS Maintain optimal weight Calorie restriction • Avoid trans fatty Structured (if BMI is increased) acids; limit counseling Nutrition saturated fatty Plant-based diet: Meal replacement acids high polyunsaturated and monounsaturated fatty acids 150 min/week moderate exertion Structured Medical evaluation/ (eg. walking, stair climbing) **Physical** program clearance Activity Strength training Wearable Medical supervision technologies Increase as tolerated Screen OSA About 7 hours per night Referral to sleep lab Sleep Home sleep study Basic sleep hygiene **Behavioral** Community engagement Formal behavioral Discuss mood with HCP Alcohol moderation therapy Support Nicotine Smoking Referral to No tobacco products replacement structured program Cessation therapy

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Profiles of Antidiabetic Medications



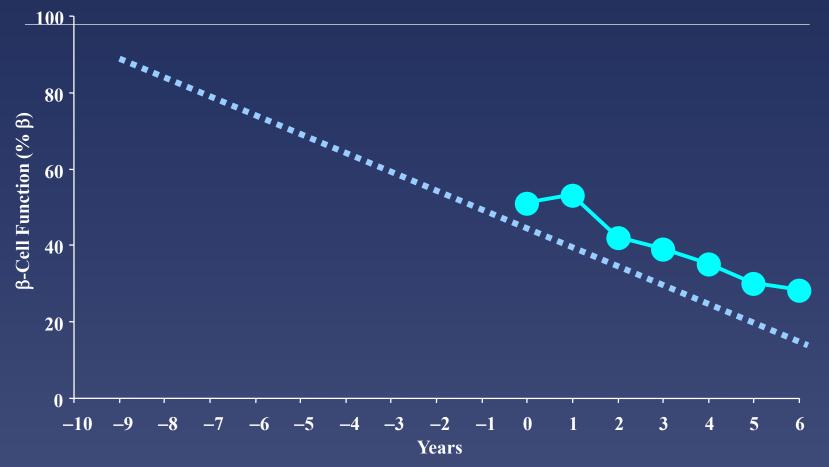
	MET	GLP-1 RA	SGLT-2i	DPP-4i	AGi	TZD (moderate dose)	SU GLN	COLSVL	BCR-QR	INSULIN	PRAML
нүро	Neutral	Neutral	Neutral	Neutral	Neutral	Neutral	Moderate/ Severe Mild	Neutral	Neutral	Moderate to Severe	Neutral
WEIGHT	Slight Loss	Loss	Loss	Neutral	Neutral	Gain	Gain	Neutral	Neutral	Gain	Loss
RENAL / GU Contra- indicated if eGFR < 30 mL/min/ 1.73 m ²	Exenatide Not Indicated CrCl < 30	Not Indicated for eGFR < 45 mL/ min/1.73 m ² Genital Mycotic Infections	Dose Adjustment Necessary (Except Linagliptin) Effective in Reducing Albuminuria	Neutral	Neutral	More Hypo Risk	Neutral	Neutral	More Hypo Risk	Neutral	
	Possible Benefit of Liraglutide	Possible Benefit of Empagliflozin									
GI Sx	Moderate	Moderate	Neutral	Neutral	Moderate	Neutral	Neutral	Mild	Moderate	Neutral	Moderate
CHF					Neutral	Moderate	Neutral	Neutral	Neutral	CHF Risk	
CARDIAC ASCVD	Neutral	See #1	See #2	See #3		May Reduce Stroke Risk	Possible ASCVD Risk	Benefit	Safe	Neutral	Neutral
BONE	Neutral	Neutral	Mild Fracture Risk	Neutral	Neutral	Moderate Fracture Risk	Neutral	Neutral	Neutral	Neutral	Neutral
KETOACIDOSIS	Neutral	Neutral	DKA Can Occur in Various Stress Settings	Neutral	Neutral	Neutral	Neutral	Neutral	Neutral	Neutral	Neutral
 Few adverse events or possible benefits Likelihood of adverse effects Liraglutide—FDA approved for prevention of MACE events. Empagliflozin—FDA approved to reduce CV mortality. Canagliflozin shown to reduce MACE events. 											

3. Possible increased hospitalizations for heart failure with alogliptin and saxagliptin.

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Use with caution

<u>TYPE 2 DIABETES ... A PROGRESSIVE DISEASE</u> **Progressive Decline of \beta-Cell Function in the UKPDS**



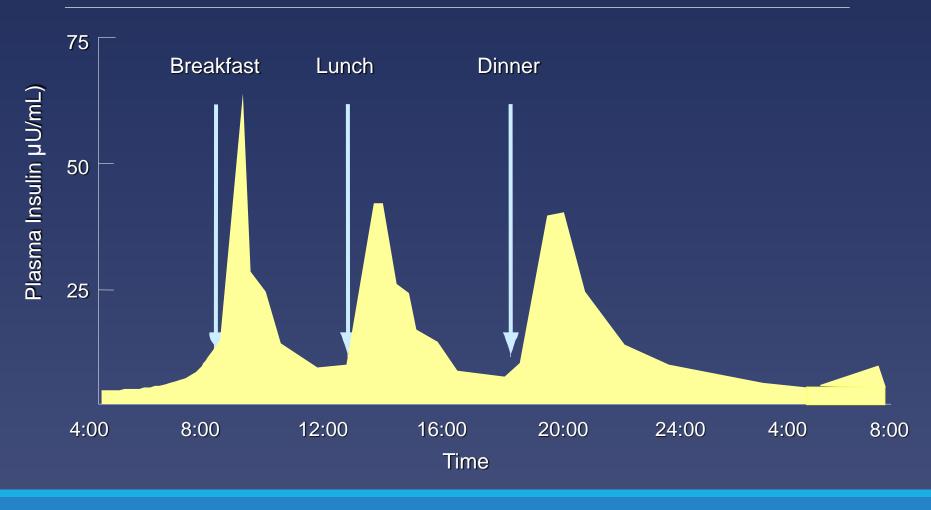
Adapted from UK Prospective Diabetes Study (UKPDS) Group. Diabetes. 1995; 44:1249-1258.

TYPE 2 DIABETES . . . A PROGRESSIVE DISEASE

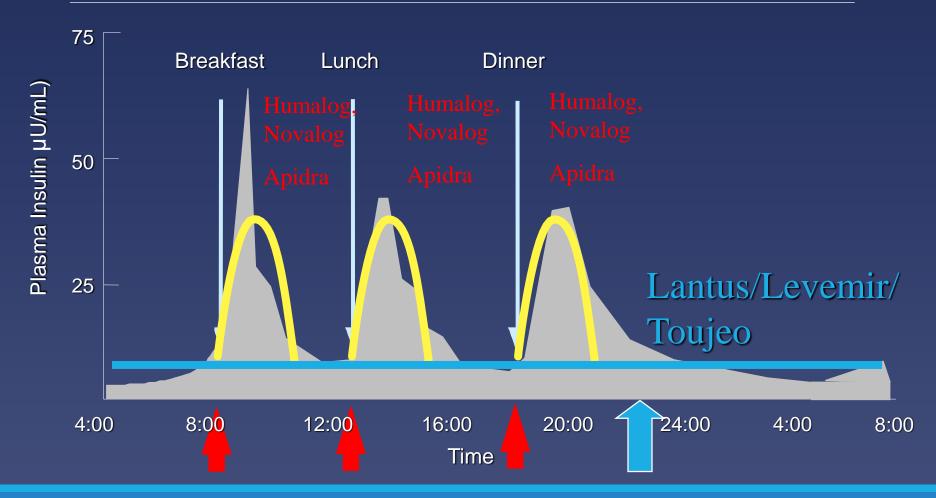
Over time, most patients will need insulin to control glucose

Mimicking Nature The Basal/Bolus Insulin Concept

Physiological Serum Insulin Secretion Profile



Combining Rapid Acting and Basal Insulin Basal/Bolus Insulin



Complications of Diabetes

Macrovascular

Brain

Cerebrovascular Disease

- Transient ischemic attack
- Cerebrovascular accident
- Cognitive impairment

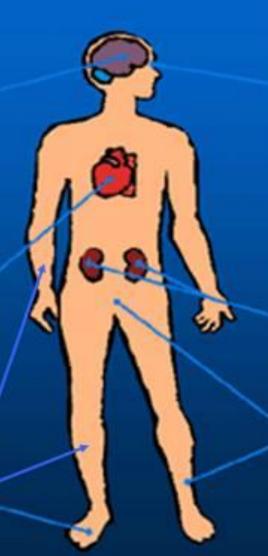
Heart Coronary Artery Disease

- Coronary syndrome
- Myocardial infarction
- Congestive heart failure

Extremities

Peripheral Vascular Disease

- Ulceration
- Gangrene
- Amputation



Microvascular

Eyes

- Retinopathy
- Cataracts
- Glaucoma
- Blindness

Kidneys

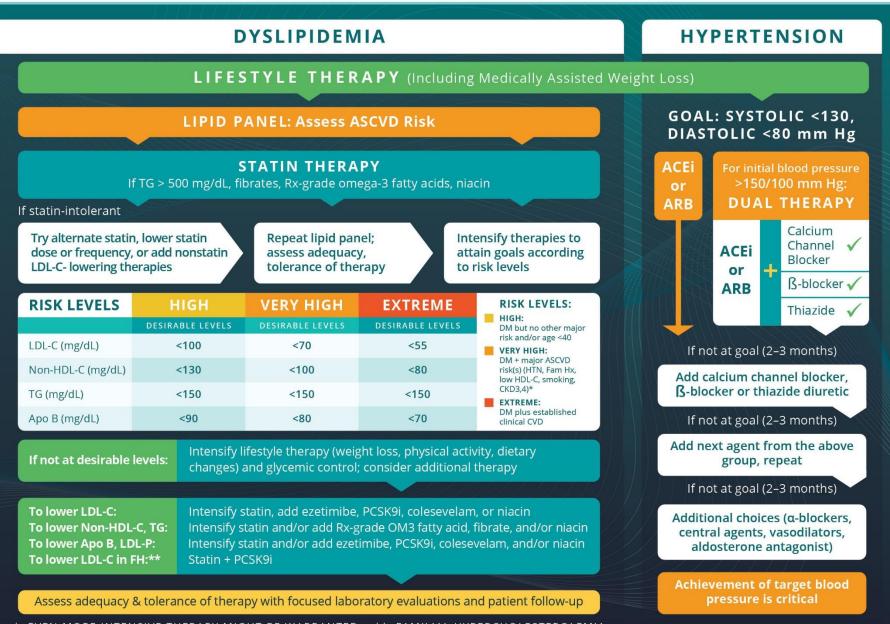
Nephropathy

- Microalbuminuria
- Gross albuminuria
- Kidney failure

Neuropathy • Peripheral • Autonomic

ASCVD Risk Factor Modifications Algorithm





EVEN MORE INTENSIVE THERAPY MIGHT BE WARRANTED ** FAMILIAL HYPERCHOLESTEROLEMIA

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Case Study-Type 2 DM Transition of Care



Dan is a 66 y/o male, DX with Diabetes 7 years ago. His BMI is 32, A1c has been 6.8%. He is a former smoker with 20 pack years of smoking before he quit 2 years ago. He is discharged today after an acute episode of pneumonia and bronchitis and meds at home include antibiotics and tapering oral Prednisone. Before admission his med list included: Metformin 1000 mg bid, Januvia 100 mg daily, Lisinopril 10 mg daily, Simvastatin 40 mg daily. He received Novolog Insulin sub-q while in the hospital when his blood sugars were running consistently over 200. He is being discharged on his prior meds plus Novolog insulin, medium sliding scale, per insulin pen.

What is the reason for Dan's high blood sugars while in the hospital?

What do you as care coordinator, need to provide on his discharge?

What is the rationale for insulin upon his discharge, when he was in good control prior?

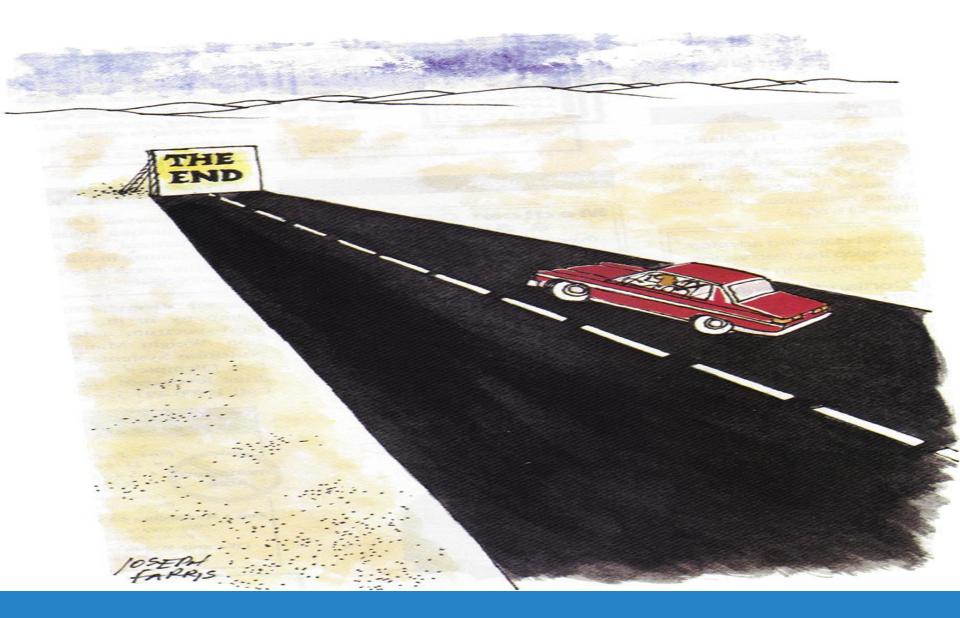
Case Study Questions

What is the reason for Dan's high blood sugars while in the hospital?

What do you as care coordinator, need to provide on his discharge?

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DRAWING TO A CLOSE



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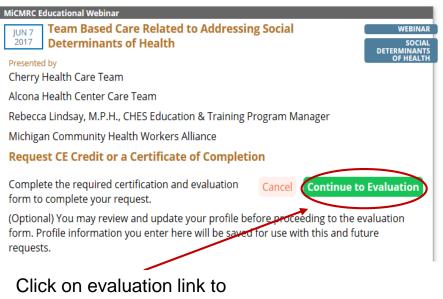
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