Ten Dogs, An Elevated HbA1c & Severe Hypoglycemia: An Autoimmune Endocrine Story

Pediatric Endocrinology Nursing Society National Conference
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The Children's Hospital of Philadelphia
- 516 beds, endocrine patients admitted to a medical floor
- Large city-based hospital

The Diabetes Center for Children
- Manages 2500 patients with diabetes, 90% have T1D

HPI: 2003
- 5 yo Caucasian boy presents to the ED in 2003
- Admitted with hyperglycemia, ketonuria, dehydration
  - Lives with mom, dad, older brother
  - Education completed

Labs during admission 5/2003

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin Antibodies</td>
<td>137 (H)</td>
</tr>
<tr>
<td>GAD 65 Antibodies</td>
<td>0.4 (H)</td>
</tr>
<tr>
<td>ICA512 Antibodies</td>
<td>23 (H)</td>
</tr>
<tr>
<td>Thyroxine (4.5-11.0 ug/dL)</td>
<td>3.8 (L)</td>
</tr>
<tr>
<td>TFT (0.3-4.5 u/mL)</td>
<td>1.977</td>
</tr>
<tr>
<td>Hemoglobin A1c (3.8-5.9%)</td>
<td>11.2 (H)</td>
</tr>
</tbody>
</table>
2003-2010: Diabetes Management

- Started initially TID NPH insulin
- After diagnosis, tries pump
- Trouble with control begins
- Hates having labs drawn

<table>
<thead>
<tr>
<th>Date</th>
<th>HbA1c</th>
</tr>
</thead>
<tbody>
<tr>
<td>9/2003</td>
<td>7.1</td>
</tr>
<tr>
<td>11/2003</td>
<td>7.5</td>
</tr>
<tr>
<td>12/2003</td>
<td>8.4</td>
</tr>
<tr>
<td>1/2004</td>
<td>8.1</td>
</tr>
<tr>
<td>10/2004</td>
<td>8.0</td>
</tr>
<tr>
<td>1/2005</td>
<td>8.7</td>
</tr>
<tr>
<td>4/2005</td>
<td>8.8</td>
</tr>
<tr>
<td>6/2005</td>
<td>7.6</td>
</tr>
<tr>
<td>11/2005</td>
<td>9.5</td>
</tr>
<tr>
<td>2/2006</td>
<td>8.4</td>
</tr>
<tr>
<td>5/2007</td>
<td>10.6</td>
</tr>
<tr>
<td>10/2007</td>
<td>9.4</td>
</tr>
<tr>
<td>2/2010</td>
<td>9.8</td>
</tr>
</tbody>
</table>

Psycho-social history

- Several calls from the School RN
- Not enough supplies
- Not testing consistently
- Poor school performance
- Child Protective Services involvement
- Diabetes control continues to be poor with HbA1cs in the 9-10% range
- Many “insurance issue” phone calls
- Difficulty with adjustment

Admission 4/2011, age 13

- Presents to the ED
- Several episodes of severe hypoglycemia over the past few weeks
- 5-7 syncope or near-syncope events reported
- Lantus dose decreased from 29 to 10 units with continued lows
- Dose given for BG of 250 on day of admission for lunch: 2 units; BG decreased to 50
Admission 2011, age 13

• PE: pale, weak, vomiting
• BP 90s/30s
• HR increased
• Afebrile

<table>
<thead>
<tr>
<th>Admission labs 4/2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin A1c</td>
</tr>
<tr>
<td>Blood glucose</td>
</tr>
<tr>
<td>Sodium (136-145)</td>
</tr>
<tr>
<td>Potassium (3.8-5.4)</td>
</tr>
</tbody>
</table>

Admission 4/2011, age 13

ACTH series:
(time 0, +15, +30, +60 & +90) showed persistently elevated ACTH (>1250) and low cortisol levels (1.54-1.86)

<table>
<thead>
<tr>
<th>4/2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adderalline (4-48)</td>
</tr>
<tr>
<td>Cortic, Panel TTG (0-20)</td>
</tr>
<tr>
<td>Celiac, Panel EMA</td>
</tr>
<tr>
<td>T4 (4.7-9.9)</td>
</tr>
<tr>
<td>TSH (0.5-3.8)</td>
</tr>
<tr>
<td>21-hydroxylase antibody (0.0-1.0)</td>
</tr>
<tr>
<td>AM Cortisol (7-72)</td>
</tr>
<tr>
<td>ACTH (7-63)</td>
</tr>
</tbody>
</table>

Discharge 4/2011: Addison’s Disease

• Hydrocortisone 5mg three times per day
• Florinef 0.1mg daily
• Stress dosing (for use with febrile illness, vomiting, trauma):
  • Hydrocortisone 15 mg three times daily
  • Solu-Cortef (100mg/2mL)–injectable
Discharge 4/2011: Diabetes Management

- Increase Lantus dose slowly to keep blood sugars in range
- Use 50% bolus doses initially, then increase ratios to keep blood sugars in range.
- Similar plan to managing blood sugars when a patient starts prednisone.

T1D and Cortisol Deficiency

- Incidence: 1.2% in patients with T1D
- T1D present in 10-18% of those with Addison's Disease
- Autoimmune condition resulting in insufficient production of cortisol and often aldosterone
- Clinical indications for testing:
  - Changes in skin color
  - Hypoglycemia (15-20% decrease in TDD)
  - Weight loss
  - Decline in normal growth velocity
  - GI upset
  - Postural hypotension

History 2011-2014, age 13-16

- Continued poor diabetes control: HbA1c 9.0-13.2% range
- Infrequent follow up
- Patient continued to refuse labs; POC HbA1c done
- 3/2013-4/2013 two ED visits and one admission for hypoglycemia
Admission 11/2014: DKA

- Age 16
- BG >500; large ketones
- pH 7.07
- ACTH not measured
- Lengthened admission for behavioral health issues
- CPS consult; patient sent home with paternal grandparents

Hyperglycemia

Ketosis

Acidosis

Outpatient follow up after 11/2014

- More comfortable living situation and consistent follow up!
- HbA1c 8.5-9.3-%
- Normal TFTs
- ACTH 487->2000
- Tested for other endocrine disorders (parathyroid)—normal

12/2014

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>AM Cortisol (8.2-19.4)</td>
<td>27.8 (H)</td>
</tr>
<tr>
<td>ACTH (7.2-63.3)</td>
<td>1799.0 (H)</td>
</tr>
<tr>
<td>Thyroid Peroxidase Antibody (0-26)</td>
<td>14</td>
</tr>
<tr>
<td>Thyrogblobulin Antibody (0.0-0.9)</td>
<td>0.2 (H)</td>
</tr>
<tr>
<td>T4</td>
<td>8.4</td>
</tr>
<tr>
<td>TSH</td>
<td>6.012 (H)</td>
</tr>
<tr>
<td>Celiac Panel</td>
<td>negative only</td>
</tr>
<tr>
<td>HbA1c</td>
<td>5.6%</td>
</tr>
</tbody>
</table>

Admission 3/2016: DKA & Adrenal Crisis

- Age 18
- Kicked out of grandparents’ home
- Living with mom again
- Food insecurity
- Angry and depressed
- HbA1c 10.2%
Follow up: 4/2016

- BP 100/52; HR 74
- Skin color changes
- Angry and frustrated with diabetes and adrenal management
  - Taking hydrocortisone 1-2 times per day (sometimes total dose)
  - Taking Novolog once (9am-2pm); not eating or testing otherwise
  - Claims to not miss Lantus (30 units)
- Not eating much at all due to food insecurity

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<tbody>
<tr>
<td>HbA1c</td>
</tr>
<tr>
<td>T4</td>
</tr>
<tr>
<td>TSH</td>
</tr>
</tbody>
</table>

| Blood Panel | Normal |

- Glomerular Filtration Rate | 80 |
- Plasma Renin | 6 |
- Vitamin B12 (199-732) | 606 |
- Celiac Panel | negative |
- ACTH | 154 |

Follow up: 4/16/2016

- Changed steroid management to once daily:
  - Prednisone 5mg daily
  - Stress Dose 15 mg daily
  - Solu-Cortef (200mg/mL) - injectable
- Encouraged daily BG testing, Lantus & Novolog for all food and BG correction
- Initiation of psycho social support.
Take Home Points

- Suspect adrenal insufficiency with hypoglycemia
- Pay attention to your physical exam
- Routine lab work for evaluation of other endocrine disease
- Managing diabetes with adrenal insufficiency isn’t easy
  - Option: once daily dosing with prednisone when growth is complete
- Managing diabetes with complex social situations is harder!
CASE PRESENTATION:
Frightening the Dermatologist!
A CLOSER LOOK AT XANTHOMA

Shari Liesch APNP, CDE
PENS 2016
Denver Colorado

CASE STUDY: HISTORY

15 yo female diagnosed with type 1 diabetes (T1DM), BMI over 85th centile with weight gain
History of hyperlipidemia
Vitamin D deficiency.
T1DM was diagnosed at 11 years of age.
  Strong family history of T1DM: father as youth, mother as adult
  Younger sister with autoimmune hyperthyroid & + diabetes
AB: also with type 1 needing insulin
Familial hyperlipidemia (mom and mat. grandmother).
Dad on dialysis, waiting on pancreas/kidney transplant

Presented for routine follow up diabetes visit.
A1c machine read Error: unable to process, specimen lipid.
Patient also complains of a rash noted a month ago

NEW RASH

- Rash “to upper arm, lower back and legs (inner thigh)”.  
- Rash is red with yellow lumps & clusters of raised yellow lesions.  
- It is very noticeable and she wants it “gone”.  
  - scheduled with dermatology  
- For diabetes: she complains of being low, especially if the meal is delayed.
- She is the oldest of 6 children (now 17)
MORE FAMILY INFO

- Mom recently got a job at a local gas station;
  - Dad is in charge of more meals:
    - Food more processed, higher carb (pizza, mac & cheese).
    - Tried to incorporate age, knowledge & ability to help create healthy choices (add veggies) → (eye roll)
- Other factors: on large insulin dose (food ratio 1:2).
- Not interested in following activity plan—
  - doesn’t really want to.
  - Is not taking fish oil or vitamin D as “they were “out”

A FEW DAYS LATER RECEIVED FASTING LABS

- After visit, received labs:
  - Cholesterol 860, HDL 22, Triglycerides >2100.
  - Vitamin D <12.8.
  - A1c 12.8%?
  - Amylase 43, lipase 33 (normal):
    - Sed rate 45, Hg 14, liver normal
- Goals: Discuss pathophysiology of diabetes and the impact of sugar control on blood lipids.
- Describe Xanthoma: appearance, symptoms, diagnosis, and treatment.
- Explore strategies to improve diabetes plan: beyond insulin, the importance choices.

XANTHOMAS
**TYPE 1 DIABETES REVIEW**

- Loss of insulin production from islet cells of pancreas (auto-immune)
- Associated with ketone production and diabetic keto-acidosis (DKA)
- Defined: look at clinical picture
  - Random sugar over 200
  - Fasting over 126
  - A1c >6.5%** (do 2-hour glucose tolerance)
  - Presence of ketones (+/-)
  - Presence of ICA 512, GAD 65, insulin AB, triangle (to Barbara Davis)

**AUTO-IMMUNE**

- Body has antibodies that destroy beta cells: unable to make insulin
- Triggers:
  - Genetic predisposition (HLA type)
  - Environmental triggers: viral, food, toxins, stress
- 5-10% of total population with diabetes
  - 85% pediatric diabetes is type 1 or About 1 in 300-400 kids
  - 85% are overweight at diagnosis
  - Most common auto-immune condition in children

Other screening labs:
- Thyroid antibodies: free T4, TSH, (17-30%)
- Celiac (TG1A, IGA) (1-16%),
  - Also look at growth, weight loss, diarrhea, abdominal pain, red absorption
- Unexplained lows and deteriorating A1c, erratic blood sugars, fatigue may need to re-screen
- Random cholesterol
- If question the type of diabetes: IC 512 & Gud 65 AB, Insulin AB, Triangle test zinc transport

**INSULIN ACTION TIMES**

- Basal: Lantus = 24 hours, Levemir
- Bolus: insulin analogs
  - Humalog, Novolog, Apidra
    - Start in 5-10 min., peak in 00-90 min., lasts 120-180 minutes: some up 6 hours
  - Regular
    - the only insulin used IV
    - IV absorbed immediately, drip turned off effect gone in short time
    - Only in 15-20 min., lasts 6 hours
  - NPH:
    - starts in 1-2 hours, peaks in 6-8 hrs., gone in 12 hours
    - (rarely used, helps with dawn effect)
- Multiple daily is Safer
  - Allows for flexibility: amount, timing, the frequency one eats
  - allows adjustment for activity
  - Reduces wide fluctuations in BGs
COMPLICATIONS OF DIABETES
- Microvascular
- Macrovascular
- Neuropathy
- Nephropathy
- Heart disease
- Blindness
- Depression
- Mood
- Autoimmune co-morbidities
- Xanthoma

Energy metabolism, untreated diabetes

In a high glucose environment, the reuptake is dysregulated:
Uptake of triglycerides to the liver (by HDL) does not occur.
Triglycerides/cholesterol need to go somewhere so is stored in the mid abdomen, skin, etc.
If you add high simple carb & high fat diet it worsens.

LIPID METABOLISM
- Lipids insoluble in water: transported by complex lipoproteins & apo proteins
- Proteins facilitate trans membrane transport & regulate enzyme activity
- Lipids classified by density
  - VLDL very low density lipoproteins
  - IDL intermediate
  - LDL low density
  - HDL high density
LIPID METABOLISM, ETC.

- Metabolic path of lipoproteins
- Exogenous: metabolism of intestinal lipoprotein, triglyceride (TG)-rich chylomicrons, formed in response to dairy fat
- Endogenous: Lipo & apo- are non-intestinal, primarily liver
- Liver: secretes TG rich VLDL that contains apoproteins B-100, C-II & E into circulation

METABOLISM OF LIPIDS, CONT

- The role of HDL is to accept cholesterol to transport back to liver
- Lipoprotein Lp[a] =
  - Is LDL with apo B + side glycosylated protein
  - Lp[a] has role in atherogenesis and thrombogenesis

DISORDERS IN LIPID METABOLISM

- Altered lipoprotein result from genetic mutation
- Defective apoliproteins (primary) hyperlipoproteinemia
- Or from other systematic disorders:
  - diabetes,
  - hypothyroidism
  - nephrotic syndrome
XANTHOMA

- Xanth from Greek “xanthos” = yellow & oma or “swelling”
- An irregular yellow patch or nodules on the skin,
  - caused by deposition of lipids (harmless growth of tissues)
  - characterized by accumulation of lipid-laden macrophages.
- Xanthomas can develop in the setting of altered systemic lipid metabolism or as a result of local cell dysfunction.
- Under microscope look...

SKIN CHANGES

- Change to skin & tendons
- Foamy macrophages
  - Filled with lipid droplets
- Lipid-laden macrophages in superficial dermis
- May contain fibrosis and cholesterol clefts

FOAMY MACROPHAGES

- When macrophages take up massive amounts of cholesterol they form “foam cells,” characterized by multiple lipid droplets (stained red).
**XANTHOMA**

Common lipid disorder

Exanthelasmas = 6% eyelid tumors

Mortality:
- Mostly cosmetic,
- Presence suggest disorder of lipid metabolism

- Morbidity & mortality R/T atherosclerosis & pancreatitis
  - Male = female
  - Any age

- Xanthelasmas usually in over 50 yrs

- Xanthoma disseminatum
  - often pre age 25 yrs (2/3 of the time)

**HISTORY**

- Family Hx:
  - Some have Xanthomas,
  - MI, aortic regurgitation, atherosclerosis, pancreatitis
  - May be seen in some leukemia, non-Langerhans disease,

- Seen in POEMS:
  - Polyneuropathy,
  - Organomegally,
  - Endocrinopathy,
  - M-protein skin change

- Cutaneous eruptions may precede dx of hyperlipidemia

**OTHER XANTHOMA**

- Eruptive xanthoma: common to buttocks, shoulder, extensor surface
  - Rare to oral mucous or face

- Erupt in crops of small, red-yellow papules on erythematous base,

- May have pruritus, tender

- Our case had itching

- Lesions to arms, back

- Inner thigh
CUTANEOUS XANTHOMA ASSOCIATED WITH HYPERLIPIDEMIA

- Xanthelasma palpebrarum:
  - Most common
  - Lesions asymmetric, soft, velvety, yellow flat polygonadal papules
  - Most common to upper eye lids, inner canthus
  - Giant xanthoma involves all 4 lids

Tuberosus xanthoma: firm, painless red yellow nodules
- Develop in pressure area
- Extensor of knee, elbows, buttocks

Tendinous xanthoma
- Slow appearing
- Subcutaneous nodule to tendons or ligaments
- Gout-tophi-like over the great toe
- Extensor tendons of hands feet Achilles
- Often related to trauma

SYMPTOMS

- Xanthoma disseminatum may present with
- Dysphasia, dyspnea & obstructive blindness
- May also have GI, CNS, Musculoskeletal, intracranial symptoms
  - May persist, and progress
  - Or be self limiting
DIFFERENTIAL DX
- Necrobiosis lipoidica
  - Hard to get rid of
  - **Sugar control**
  - Hctz cream/systemic Injections
  - Baby ASA
- Molluscum contagiosum (eruptive xanthoma)
- Sarcoïdosis complications
- Amyloidosis
- Cerebrotendinosus
- Dermatofibroma
- Erythema elevatum diutinum
- Erythema nodosum
- Gout
- Haemochromatosis
- Juvenile xanthogranuloma
- Lymphangioma
- Maffucci syndrome
- Mesothelioma
- Osteosarcoma
- Pancoast Tumor
- Histiocytosis X
- Erdheim-Chester disease
- Erythema elevatum diutinum
- Lichen planus
- Lupus
- Leukemia
- Skin tumors

LAB
- Primary hyperlipidemia is diagnosis of exclusion
- Lipoprotein panel used to assess cardio risk
- 12 hour fasting lipid panel
- LDL = total cholesterol - (TG/5 + HDL)
- VLDL = TG level / 5
  - If TG over 700: /10
  - Cardiac risk determined by total cholesterol, LDL, HDL

TREATMENT
- Treat underlying hyperlipidemia
  - Decreases size of xanthomas
  - Prevent atherosclerosis
  - Prevent pancreatitis
- Diet & lipid lowering agents
- Statins, fibrates, bile acid-binding resins, probucol, nicotinic acid
- Eruptive xanthoma resolve within week of treatment
- Tendinous xanthoma may take years
- **Sugar control**
- Hctz cream/systemic Injections
- Baby ASA
TREATMENT

- Explore strategies to improve diabetes plan: beyond insulin, the importance of choices.
- Dietary measures should include:
  - Prepare most meals from vegetables, salads, cereals and fish
  - Minimize saturated fats (found in meat, butter, other dairy produce, coconut oil, palm oil)
  - Minimize intake of simple, refined sugars found in fizzy drinks, sweets, biscuits and cakes
- If obese or overweight, aim to slowly reduce weight by reducing caloric intake and increasing exercise.

THE RESULTS OVER TIME

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<tbody>
<tr>
<td>Cholesterol</td>
<td>278</td>
<td>560</td>
<td>246</td>
<td>445</td>
<td>285</td>
</tr>
<tr>
<td>HDL</td>
<td>low</td>
<td>24</td>
<td>22</td>
<td>HDL 21</td>
<td></td>
</tr>
<tr>
<td>TG &gt;2100</td>
<td>&gt;2100</td>
<td>1115</td>
<td>&gt;2100</td>
<td>TG 1645</td>
<td></td>
</tr>
<tr>
<td>LDL &gt;400</td>
<td>&gt;400</td>
<td>LDL</td>
<td>11.8</td>
<td>8.9</td>
<td></td>
</tr>
<tr>
<td>1:2014 A1c</td>
<td>9.5</td>
<td>Metformin added</td>
<td>Amylase, lipase, liver normal</td>
<td>TSH normal</td>
<td>Celiac, urine micro NL</td>
</tr>
<tr>
<td>Fenofibrate 54 mg (generic)</td>
<td>Fish oil 3000 mg (EPA)</td>
<td>Vitamin D 5000 IU daily for 8 weeks then 2000 IU</td>
<td>Later: Job at local gas station—candy, Ho-Ho's, pizza abundant &amp; has cash!</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

LIPID MANAGEMENT IN PEDS

- Identify youth at risk through screening
- Trial meal plan, activity first
- When elevated LDL-C elevated over 6 months, age over 10 years

<table>
<thead>
<tr>
<th>Total cholesterol</th>
<th>Goal &lt;170 mg/dl</th>
<th>Borderline 170-190 mg/dl</th>
<th>Abnormal &gt;200 mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides (10-10)</td>
<td>&lt;90</td>
<td>90-120</td>
<td>&gt;130</td>
</tr>
<tr>
<td>HDL-C</td>
<td>&gt;40</td>
<td>40-45</td>
<td>&lt;40</td>
</tr>
<tr>
<td>LDL-C</td>
<td>&lt;110</td>
<td>110-120</td>
<td>2130</td>
</tr>
<tr>
<td>Non HDL-C</td>
<td>&lt;120</td>
<td>120-144</td>
<td>2145</td>
</tr>
</tbody>
</table>
RISK FACTORS

- STATUS LDL-C cut point
- No other > 190 mg/dl
- Other risks >160
  - Obesity
  - HTN
  - Smoker
  - Family hx of premature CVD
- Diabetes ≥ 130

If abNL, need more aggressive lifestyle choices
- Genetic dyslipidemias
- Familial hypercholesterolemia
  - manifests w/ elevated LDL-C
  - LDL-C is quite high & other lipid lipoproteins are normal
- Combined
  - high LDL-C & triglycerides,
  - low HDL-C
- Atherogenic: asso. w obesity
  - High triglycerides, low HDL-C
  - Found w/ insulin resistance, metabolic syndrome, abd. Girth
  - LDL-C may be mild to mod. Elevated, may not need to treat

TREATMENT

- Diet:
  - Total fat <30% total cal.
  - Sat. fat <7% total cal
  - Trans. Fat <1% total cal
  - Cholesterol <200 mg/day
  - Increased soluble fiber
  - Pt age +5
  - Over 9 yrs = up to 14 gm fiber
  - Supplement: age 2-12 = 6 grams
  - Over 12 yrs = 12 grams
  - (up to date)

Elevated TG
- N-3 omega fatty acids
  - High intake N3 omegas may lower atherosclerosis
- Physical ex.
  - Vigorous increases HDL-C
- Weight mgmt: decreased consumption, increase vigorous exercise
- Weight most important for tx elevated TG, HDL-D
  - 5-10% weight change has important benefit from CVD

HYPERTRIGLYCERIDES

- Diet, fish oil, ex, weight loss
- Primary Hyper TG
  - TG > 500 or single over 1000
    - Children with TG 200-499 plus non-HDL > 145
- Omega 3 fish oil ethyl esters
  - Begin 1G, max 4 G (adult)
  - Gemfibrozil 1200 mg, initial dose or
  - Fenofibrate 40 mg, initial max. 130-200 adult

Fibric acid derivatives:
  - (gemfibrozil, fenofibrate)
  - These raise HDL-C & lower TG levels
  - TG improved with fibric acid derivatives, not statins
  - Fibric acids tolerated in monotherapy
  - Most useful for over 1000 TG
  - Often for older teens
  - Use w statins increase risk myopathy, rhabdomyolysis esp. in renal
TREATMENT AIM: LOWER LDL-C

- LDL-C 3 classes
- Bile acid sequestering agent
  - Binds with cholesterol rich bile acids so they are excreted in stool
  - GI, constipation, bloating, cramps
  - www.co-cardiology.com
- HMG-CoA reductase inhibitors (statins)
  - Blocks cholesterol synthetic pathway and induces LDL receptors in the liver
  - Myopathy, rare rhabdomyolysis
- Cholesterol absorption inhibitors
  - Blocks absorption of cholesterol in GI track
  - GI, bloat, cramp, myopathy

Shari Liesch, MSN, APNP, CDE
Children’s Hospital of WI- Fox Valley
sliesch@chw.org

REFERENCES
- ADA guidelines: Cardiovascular disease & risk management. Diabetes Care 2016; 39 (S60-S71).
  - www.co-cardiology.com
FAMILIAL HYPERCHOLESTEROLEMIA

- Combination therapy
- Surgical repair: disfiguring, impair function
- Xanthoma disseminatum - may respond to 2-chlorodeoxyadenosine, simvastatin
- Partial resolution of verruciform xanthoma - chloroxylenol surgical scrub
- Surgery
- Laser
- Lesions may recur
Taking the Leap: A Patient’s Journey to Adult Care
Mandi Cafasso DNP, RN, CNP
Cincinnati Children’s Hospital Medical Center
May 14, 2016

What Do We Know?
Emerging Adulthood (18-25 years of age)
• Period of considerable life changes:
  • College or employment
  • Moving away from the family home
  • Increasing financial independence
  • Developing new relationships
• Sense of invulnerability
  • Discount risks to future health
• Challenge: Integrate the increasing demands of diabetes self-management with competing educational, social and economic priorities

Arnett JJ Am Psychology 2000; Weissberg-Benchell Diabetes Care 2007

What Do We Know?
Transition to Adult Diabetes Care
• Specific barriers (patient perspective):
  • Lack of specific adult provider name (47%)
  • Competing life priorities (43%)
  • Difficulty getting an appointment (41%)
  • Feeling upset about leaving pediatrics (24%)
  • Insurance problems (10%)

What do we assume?

- Visit frequency changes
- Glycemic control worsens
- Comorbidities increase

Transfer vs. Transition

- How many of you have a process of transitioning patient?
- How long is the process?
- How many of you have mainly unanticipated transfer of care?

The Patient

- RM 23 year old non Hispanic Male
- Private insurance
- Resides in a large metropolitan area
- College student at local university
- Lives with mother
- Engaged to be married summer 2016
- Paternal half sister with Type 1 DM
The History

- Type 1 DM dx 2008 (16 yo)
  - Hgb A1c 10.1%
- Regular follow-ups (between 2-6 months intervals since diagnosis)
- BBT
- Avg. Hgb A1c ~8%
- TOC discussion initiated by SW in February 2014

The Evaluation

What are indicators of readiness to transfer to adult care?

- Attends clinic alone
- Hgb A1c 8%
- Frequent BG checks
- Makes insulin adjustments comfortably
- Initiates discussion
- BUT...

The Evaluation

- READY
  - Readiness for Emerging Adults with Diabetes Diagnosed in Youth

<table>
<thead>
<tr>
<th>Readiness for Emerging Adults with Diabetes Diagnosed in Youth</th>
<th>Ready</th>
<th>Partial</th>
<th>Not Ready</th>
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</thead>
<tbody>
<tr>
<td>Provides diabetes education on diabetes</td>
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<tr>
<td>Helps with recognition of diabetes</td>
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<tr>
<td>Assists with monitoring of diabetes</td>
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<td>Tracks progress and goals of patient</td>
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<td>Self-care independence</td>
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</table>
Interventions

- December 2015 - TOC assessment was repeated (READdY)
- Dec. 2014 - Dec. 2015 - RM was seen by education staff
  - RN educator and SW
- September 2015 - RM met with SW to choose adult provider.

Longitudinal Diabetes Transition Planning:

Diagnosis: There is a future with diabetes

Goal: Recognition that diabetes extends beyond childhood and there is a future with diabetes:
- Age 12: Shared responsibility of care
- Age 16: Development of independence
- Age 18-24: Planning Transfer to Adult Care

Goal: READdY increases participation in disease management with caregiver and provider

Goal Further development of diabetes self-management and health navigation skills

Goal: Individual plan for transition from pediatric to adult care

Goals:
- Confidence and competence in self-management
- Established care with adult provider
- Improved quality of life and clinical outcomes

Providers:
- Education Team:
  - National Diabetes Prevention Program (NDPP) diabetes educators
  - Certified Diabetes Educator (CDE)
  - Certified Diabetes Nurse (CDN)
  - Registered Dietitian Nutritionist (RDN)

Transition Coordinator:
- Collaborative interdisciplinary team including primary and secondary care practitioners
- Team includes risk assessment for transitioning to adult diabetes care
Approximately 1,800 patients with diabetes at CCHMC

- 519 patients 16-18 years of age with diabetes
- 440 patients > 19 years of age with diabetes
- 58% of patients 16-18 have a documented transition plan (n=301)
- 86% of patients >19 have a documented transition plan (n=378)

Program Development: Room to Grow
What about RM?

- September 2015 - SW finished referral to the adult provider in January
- January 2016 - RM had not scheduled a follow-up with chosen provider. (He was waiting on a call from adult transition coordinator)

Rationale for Diabetes Transition Program

- During transition between pediatric and adult care, young adults with diabetes are at risk for:
  - Gaps in care
  - Sub-optimal glycemic control
  - Inadequate prevention and screening
  - Increased morbidity and mortality
  - Risk decreased through enhanced preparation and care coordination