Critical Assessment of Diabetes and Complications: Honing your Detective Skills

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Objectives:
1. Identify common yet often under diagnosed complications associated w/ type 1 and type 2 diabetes.
2. State strategies to identify previously undiscovered diabetes complications during patient assessments.
3. Demonstrate steps involved in lower extremity assessment.

Patient is Gaining Weight
- 68 yr old female complains of 4 lb wt gain a week for past month.
  Wt 140lbs, BMI 27. BG levels 200-300s. B/P 142/96
- Reported daily meds include:
  - glyburide 10mg ac breakfast
  - Actos 30mg ac breakfast
  - Glargine 30units at night
  - Lispro sliding scale with meals
  - Synthroid (not sure of dose)
  - Lasix 20mg a day
  - Zyprexa 10mg a day
**Fluid Weight Gain**

- People with diabetes at greater risk for Congestive Heart Failure (CHF) due to increased CVD risk factors.
- Actos and Avandia, (TZD's), can cause fluid wt gain and worsen CHF.
- Metformin used cautiously in pt's w/ CHF due to increased risk of renal impairment.

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**Thyroid Disease and Diabetes**

- 27 mil Americans have over or under active thyroid glands, but more than half remain undiagnosed.
- More than 8 out of 10 pts w/ thyroid disease women.
- 15 to 30% of people w/ diabetes & their siblings or parents are likely to develop thyroid disease (compared to 4.5 percent of the general population).
- Check TSH on Type 1 & 2 annually or if indicated.

_AACE Website_

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**Thyroid & TSH* Levels**

*Thyroid Stimulating Hormone - secreted by pituitary gland
- controls thyroid hormone thyroxine production
- first and best test
- TSH Norm = up to 4.5 mIU/mL
- Treatment based on TSH plus symptoms.
  - 4.5 – 10 based on risk, s/s
  - 10 or more = treat
- Lower = hyperthyroidism
- Higher = hypothyroidism

_AACE 2012 Guidelines_
Hypothyroidism
- Hashimoto’s thyroiditis – autoimmune thyroid
  - most common cause of hypothyroidism w/ dm
- Type 1 and type 2 at greater risk
- Screen annually for thyroid disease in diabetes
- Clinical features: fatigue, wt gain, dry skin, cold intolerance, depression, constipation, dyslipidemia
  - Higher risk of CVD – monitor risk
- Dx: high TSH, then test for free T4, autoantibodies, and thyroid scans as needed
- Tx: replacement with levothyroxine (75-125 ug)

AACE Thyroid Guidelines

Diabetes: 30% Depressed
12% of those, major depression
70% don’t receive treatment

Treatment includes:
- referral to mental health professional
- Medications

Novel / Atypical Antipsychotics
Linked to Hyperglycemia
- Severe cases of hyperglycemia – even death reported
- Monitor BG regularly for DM patients started on this class of med
- If pt at risk for DM, determine fasting glucose before initiating therapy and monitor closely during treatment
- Weight gain may require increased dosing of diabetes therapies.

Summary of FDA warning statement for atypical antipsychotics, 2004
**Novel/Atypical Antipsychotics Linked to Hyperglycemia**

- Zyprexa – olanzapine
- Geodon – ziprasidone
- Seroquel – quetiapine
- Risperdal – risperadone
- Clozaril – clozapine
- Abilify – aripiprazole
- Latuda – lurasidone

*Consensus Development Conference on Antipsychotic Drugs and Diabetes 2004*

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**New Insulin Start – No orders**

- 71 year old woman, type 2 for 8 years
- Weight 90 kg
- DM Meds -
  - Metformin 2000mg day
  - Actos 15 mg (just started)
  - Admits to taking am meds ~ 4 xs a week, but always takes pm meds
- A1c 10.3% Checks BG ~ 5 xs wk in am (200-250) C/O of Many hyperglycemia SE

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**What Would You Start her on?**

- Intensive insulin therapy based on her wt?
  - 90kg x 0.5 = 45 units a day
  - 7 units bolus each meal, ~ 20 units basal at hs?
- Start w/ 10 units Basal at HS?
- What factors would influence your decision?
What Would You Start her on?

- My insulin suggestion
  - Pre Breakfast - 20 units 70/30 insulin
    - 14 units basal / 6 units bolus
  - Pre dinner - 10 units 70/30 insulin
    - 7 units basal / 3 units bolus
- BGM suggestion
  - 2 x's a day
  - Before breakfast, 2 hrs after dinner

Bev’s Rationale

- Pt not very connected to diabetes
- Does not have a scheduled life
- Limited record keeping skills
- Overwhelmed with all her the medications she is already taking
- Start slow, gradually intensify
- Start where they are at...
- Safe and feasible short and long term?

Patient is Losing Weight

SR, 49 yr old woman w/ lean “type 2" 7 yrs.
Monitors BG 1 x daily
A1c 13.9%
Insulin: 14 u Lantus at hs (uses pens)
Humalog if BG > 200 (says too expensive)
Also on Metformin 500mg BID
At 5’7, her usual wt is 120, but now 106 lbs
C/O of nausea, fullness, fatigue
No health insurance
Physical Assessment and Referrals
- What do you include in physical exam?
- What referrals?

Comprehensive Diabetes Evaluation – Physical Exam
- Height, wt, BMI
- B/P – orthostatic hypo, hypertension
- Fundoscopic Evaluation (referral may be needed)
- Thyroid palpation
- Skin exam
- Comprehensive Foot exam (pulses, inspection, sensation, vibration)

ADA Clinical Practice Recommendations

Diabetes Detective
- What other comorbidities are you suspecting?
- Any labs you would like to check?
- What type of diabetes?
- Social situation?
- Consider her lack of insurance and low income level during your discussion.
- Medication changes?
Suggested changes

- Regular insulin 3 times a day – 3 units if don't check BG (eat 45 gms of carb)
- If check BG, add 1 unit for each 50 pts above 150
- Try and eat 3 times a day – use liquid calories as needed, low fiber
- Check BG at least once a day
- Weekly phone call check in

Hyperthyroidism

- Graves Disease (most common)
- 0.5 – 2.0% risk in type 1
- Autoimmune disorder:
  - Symptoms: wt loss, hypermetabolism, tremor, exophthalmus, palpitations, tachycardia, heat intolerance, nervousness, hyperglycemia
  - Diagnosis: Dx: low TSH, then check T3 & T4, autoantibodies, and thyroid scans
  - Treatment: antithyroid drugs, surgery, radioactive iodine. After treatment, may need thyroid replacement therapy.

Celiac Disease

- Type 1 – Affects 1-16%
- Immune reaction to gluten - affects function of villi in intestine, decreasing nutrient absorption
- S/S: bloating, malabsorption, wt loss, fatty stools, diarrhea, muscle tenderness, failure to thrive
- Diagnosis: measure either anti-endomysial antibodies (EMA) titers or tissue transglutaminase.
- If positive, refer to GI specialist for endoscopy and biopsy of small intestine to confirm diagnosis.
Gastroparesis

- Gastroparesis: affects 20 – 30% of pt's w/ longstanding dm
- Delayed emptying of stomach contents due to nerve damage
- S/S include early satiety, fullness, postprandial hypo, vomiting
- Diagnosis: gastric emptying studies, post-prandial hypoglycemia
- Tx: improve BG, small, low fat & fiber meals meds: reglan, erythromycin

SR struggling w/ eating

- Gained 20 lbs
- Low blood sugar after meals
- Doesn't feel very hungry
- Doesn't want to check BG
- A1c 9.7%

Strategies?

Worries?

Addison’s Disease

- 1 in 250 w/ type 1 (thyroid dx = > risk)
- Autoimmune destruction adrenal glands
- Cortisol deficiency
  - decreases hypoglycemia awareness
  - decreases glycogenolysis
- S/S weakness, wt loss, hypoglycemia, dehydration, hyperpigmentation, muscle weakness, salt craving, hyponatremia, hyperkalemia
- Diagnosis: test Anti-21- hydroxylase autoantibody, adrenocorticotropic hormone cortisol stimulation test
- Treatment: oral hydrocortisone replacement
NonAlcoholic Fatty Liver Disease (NAFLD)

- Increasing worldwide prevalence
  - 25% of adults
  - 75% of people w/ DM or obese
  - Up to 50% of obese children

NAFLD = greater than 5.5% fat in liver that can't be attributed to other cause.

Due to Insulin Resistance and Obesity

The Metabolically Benign & Malignant Fatty Liver - 2011

DM & Fatty Liver

- Fatty Liver and hepatic inflammation is associated with insulin resistance and measures of visceral adiposity
- It also predicts:
  - Incidence of type 2 diabetes
  - Heart disease
- Fatty liver disease is directly involved in the pathogenesis of these diseases. Maybe a cause?

Finding Liver Disease

- No markers are accurate for diagnosing NASH – only biopsy
- Obese pts or those with metabolic syndrome should be evaluated
- Signs of advanced disease include:
  - Portal hypertension, spider angiomas, reddening of palms, declining platelet counts an family hx
Treating NAFLD

Since there is no approved treatment for NAFLD and almost every patient with NAFLD will have to change their lifestyle – lose weight, exercise, and eat a healthy diet – it is not necessary to biopsy routinely.” NIH Clinical Center, Dr. Yaron Rotman

Wt loss of 7-10% linked with a 50% drop in liver fat
Clinical Endocrinology News 12/12

Natural History of NAFLD to NASH

The Spectrum of NAFLD

- Fatty Liver
- NASH
- Cirrhosis

Fat accumulates in the liver
Fat plus inflammation and scarring
Scar tissue replaces liver cells

Natural History of NAFLD -

- Over 3.5 - 11 year period
- "Benign" Group
  - 60% remain stable
  - 13% have improvement
- "Malignant" Group
  - 28% progress to liver damage

The Metabolically Benign & Malignant Fatty Liver - 2011
Diabetes + Obesity = Progression to NASH

- 50% progress from “Benign” fatty liver to Steatohepatitis.
- 2-4 fold risk of developing advanced liver disease compared to those without diabetes.
- About 15% develop cirrhosis and are at increased risk for liver cancer

NASH

- Represents the hepatic manifestation of metabolic syndrome:
  - Abdominal obesity
  - Hypertension
  - Diabetes
  - Dyslipidemia

25 million Americans will develop NASH by 2025 with 20% progressing to cirrhosis, cancer or both

Over Time Leads to NASH or SteatoHepatitis ...

- Fibrosis and Cirrhosis
- Liver Cancer
- Liver Failure

Future epidemic of liver transplants??
Liver Disease & Glucose
- Hepatitis-C > 40, 3x's rate of diabetes
  - Increased risk if familial history
- Cirrhosis: 80% of pts have glucose intolerance
- Hepatic failure: associated w/ hypoglycemia due to destruction of hepatocytes, increased insulin production, inadequate storage of glucose
- Hemochromatosis – up to 75% have diabetes
  - Condition characterized by excessive production and accumulation of iron in liver & other tissues, “bronze diabetes”

Important Stuff to Remember
- Always start with where the patient is at!
- Consider the entire milieu
- Listen
- Keep it simple
- Check in often
- Open lines of communication with medical team

Lets take a look at his Lower Extremities and Assess
Lower Extremity Complications
- Combination of vascular, neurological, and musculoskeletal dysfunction
- After Lower Extremity Amputation (LEA), people have higher mortality rates and subsequent amputation

Lower Extremity Amputations Dropping over past 10yrs
- 60% of amputations in 7% of pop
- Higher in men, elderly, minorities, Chronic Kidney Disease (CKD)
- Lower extremity complications represent 20% of hospitalizations for elderly
- Amputations cost $40,000
- Amputation associated w/ earlier death compared to revascularization
- 10 yr survival after LEA

Diabetes and Lower Extremity Ulcers
- Up to 15% of DM patients have ulcers in their lifetime
- Mortality with foot ulcers is twice usual
Risk factors for Foot Ulcers/Amputation

- Previous amputation
- Past foot ulcer history
- Peripheral neuropathy
- Foot deformity
- Peripheral vascular disease
- Visual impairment
- Diabetic nephropathy (especially patients on dialysis)
- Poor glycemic control
- Cigarette smoking

ADA Task Force - 2008

Pathway to Amputation –
Pecoraro, Frykberg

Minor Trauma (environmental) +
Faulty Healing (intercurrent pathophysiology: circulation, WBC/platelet function) +
Ulceration

Predicts 72% of amp

What Leads to Ulcers

- 86% single precipitating event leading to ulcer
  1. Tight shoe

  3 classes
  1. Neuropathic
  2. Ischemic (hard to heal)
  3. neuro-ischemic (worst)
“I didn’t notice”
- Needle in foot
- Pebble in shoe
- Stepped on a nail
- Cut too deep
- Shoes were rubbing
- Others?

Pressure Area Breakdown

Walking Cast for Neuropathic Ulcers

Emotional aspects
Impact on BG
Neuropathy Leads to Lower Extremity Complications

Neuropathies
  - Sensory
    - loss of sensation, painless trauma, repetitive low grade stress
  - Motor
    - muscle atrophy, unbalanced tendon pulling, bone/gait changes, deformities, claw foot
  - Motor + Sensory changes = ulcerations
  - Autonomic
    - decreased perspiration, fissures, Charcot's foot

Stairway to Amputation

- Neuro + Peripheral Arterial Disease
- Injury or callus
- Wound
- Infected
- Cellulitis
- Gangrene
- Amputation

Neuropathic Diabetes Foot Ulcers
Foot Motor/Nerve Deformities

Circulation Issues lead to Lower Extremity Problems
- Peripheral Arterial Disease
- Vascular Disease
- Smoking
Peripheral Arterial Disease Assessment

Pitting Edema

Venous Ulceration
Diabetes and Charcot Foot

- Damaged nerves
- Blocked blood vessels
- Shifting bones
- Collapsed arch joints

Charcot’s

- **Neurotraumatic theory:**
  bony destruction due to loss of pain sensation and proprioception + repetitive and mechanical trauma to foot.

- **Neurovascular theory:**
  joint destruction secondary to autonomically stimulated hyperemia and periarticular osteopenia associated with trauma.

45 yr old, type 2 on orals, Random BG 201mg/dl

Tx during acute phase = Casting for 3-6 mo’s then custom footwear

Comprehensive Foot Exam
1st Step – Watch Pt Walk

Foot Exam – Patient History
- Previous foot ulceration
- Previous amputation
- Diabetes > 10 years
- A1c \( \geq \) 7%
- Impaired Vision
- Neuropathic Symptoms
- Claudication

Foot Exam – Dermatologic Exam
- Dry Skin
- Absence of hair
- Ingrown nail edges, long or sharp nails
- Interspace maceration
- Ulceration
- Cleanliness
Flexibility Assessment
Stiff joint syndromes

Visual Inspection/Palpation
- Breaks in the skin
- Erythema
- Trauma
- Pallor on elevation
- Dependent rubor
- Changes in the size or shape of the foot
- Nail deformities
- Extensive callus
- Tinea pedis
- Pitting edema

VA Guidelines 2004

Foot Exam – Screening for Neuropathy

**Test**
- Semmes-Weinstein monofilament 10g
- Vibration perception threshold testing
- Tuning Fork 128 Hz

**Significant Finding**
- Lack of perception at one or > sites
- Vibration perception threshold >24 volts
- Abnormal vibration perception
Loss of Protective Sensation

- Monofilament Testing
  - 5.07 touched to plantar surface and top of foot
  - C shape delivers 10 gms pressure
  - Test four sites
    - Plantar surfaces of
      - Each great toe
      - 1st, 3rd and 5th metatarsal head

Monofilament Testing

Monofilament (MF) Procedure (Int Consensus Grp)

- Demonstrate procedure on pts forearm or hand
- Have pt close their eyes
- Test four sites in random sequence
  - (if callus or ulcer, test adjacent surface)
- Bow the MF and ask, "Do you feel it touch you, yes or no?"
- Randomly test at each site 3 times (one of which is a "sham" application – MF not applied)
5.07 monofilament = 10gms linear pressure

Tuning Fork to Detect Polyneuropathy

- 128 tuning fork
- Plantar halax
- Compare sensation to that of examiner

Back to Basics in Diagnosing Diabetic Polyneuropathy with the Tuning Fork! Meijer, et al.
Diabetes Care, Vol 28, #9 Sept 2005

Tuning Fork (TF) Procedure

- Demonstrate sensation to pt on wrist or elbow w/ and without vibration
- Ask pt to close eyes
- Apply TF perpendicularly with constant pressure to dorsum of hallux (1st great toe) just proximal to nail bed. Place your index finger of the hand beneath the pt's toe to feel vibration and verify.
Tuning Fork Procedure

- Use initial sham test and apply non-vibrating TF to be sure pt does not mistake pressure for vibration and ask.
  Is the TF vibrating? (No is right answer)
- Use “on-off” method to score.
- Conduct testing 2xs on each great toe

Tuning Fork Procedure

- On each test:
  - Ask pt to ID beginning of vibration
  - “Is it vibrating”?
  - Ask pt to ID cessation by dampening TF.
  - “Tell me when the vibrating stops”
  - The number of correct responses = 0-8
  - At least 5 incorrect responses = peripheral neuropathy

Foot Exam – Vascular Exam

- Test
  - Palpation of pulses
    - dorsalis pedis
    - tibial
  - Ankle – Brachial Index (ABI)

- Significant Finding
  - Absent pulses
  - ABI <0.90, consistent w/ peripheral arterial disease
Vascular Status Assessment
- Posterior tibial pulse
- Dorsalis pedis pulse
- Temperature
- Appearance

Dorsalis Pedis Pulse

Taking the DP Pulse
Posterior Tibial Pulse

Taking the Posterior Tibial Pulse

ABI Procedure
### Ankle Brachial Index

**Technique**
- Measure highest systolic reading in both arms
  - Record first doppler sound as cuff is deflated
  - Record at the radial pulse
  - Use highest of the two arm pressures
- Measure systolic readings in both legs
  - Cuff applied to calf
  - Record first doppler sound as cuff is deflated
  - Use doppler ultrasound device
    - Record dorsalis pedis pressure
  - Use highest ankle pressure (DP or PT) for each leg
- Calculate ratio of each ankle to brachial pressure
  - Divide each ankle by highest brachial pressure

### The Ankle-Brachial Index

\[
\text{ABI} = \frac{\text{Lower extremity systolic pressure}}{\text{Brachial artery systolic pressure}}
\]

**Should be as Close to 1 as possible = normal circulation**
- The ankle-brachial index is 95% sensitive and 99% specific for PAD
- Establishes the PAD diagnosis
- Identifies a population at high risk of CV ischemic events
- The “population at risk” can be clinically and epidemiologically defined:

### Using the ABI: An Example

<table>
<thead>
<tr>
<th>Right ABI</th>
<th>Left ABI</th>
<th>ABI</th>
</tr>
</thead>
<tbody>
<tr>
<td>80/160=0.50</td>
<td>120/160=0.75</td>
<td>(Normal &gt;0.99)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Brachial SBP 150 mm Hg</th>
<th>Brachial SBP 160 mm Hg</th>
<th>Highest brachial SBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>PT SBP 40 mm Hg</td>
<td>DP SBP 80 mm Hg</td>
<td>Highest of PT or DP SBP</td>
</tr>
</tbody>
</table>

*ABI = Ankle-Brachial Index; DP = dorsalis pedis; PT = posterior tibial.*
### Interpreting the Ankle-Brachial Index

<table>
<thead>
<tr>
<th>ABI</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.00–1.29</td>
<td>Normal</td>
</tr>
<tr>
<td>0.91–0.99</td>
<td>Borderline</td>
</tr>
<tr>
<td>0.41–0.90</td>
<td>Mild-to-moderate disease</td>
</tr>
<tr>
<td>≤0.40</td>
<td>Severe disease</td>
</tr>
<tr>
<td>≥1.30</td>
<td>Noncompressible</td>
</tr>
</tbody>
</table>

Adapted from Hirsch AT, et al. J Am Coll Cardiol. 2006;47:e1-e192. Figure 6.

### Ankle Brachial Index

- **False Negative Test: Diabetes Mellitus**
- Vessels in diabetics are poorly compressible
- Results in falsely elevated ankle pressure

**Management**

- **Segmental Arterial Pressure** indicated for ratio < 0.9
- Consider angiography or Magnetic resonance angiography

### Biomechanical Foot Assessment –

<table>
<thead>
<tr>
<th>Test</th>
<th>Significant Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plantarflexion &amp; Dorsiflexion of ankles, great toes</td>
<td>Diminished joint mobility</td>
</tr>
<tr>
<td>Watch pt ambulate</td>
<td>Decreased vision, gait imbalance, need for assistive devices</td>
</tr>
<tr>
<td>Inspect Shoes</td>
<td>Ability to see/ reach feet</td>
</tr>
<tr>
<td>Inspect for deformity</td>
<td>Corn, calluses, bunions, prominent metatarsal heads, hammertoes, claw toes</td>
</tr>
</tbody>
</table>
Risk Stratification: Population Approaches

- Reality that we cannot give maximum resources to all
- Screening: Appropriate for all, baseline
- Patient education: All need to know risks and self care
- Monitoring condition: Varies with degree of pathology: risk stratification

Risk Classification and Referral / Follow-UP

<table>
<thead>
<tr>
<th>Cat</th>
<th>Definition</th>
<th>Action</th>
<th>Re Assess</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No LOPS</td>
<td>Prevention Ed</td>
<td>Yearly</td>
</tr>
<tr>
<td></td>
<td>No PAD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>LOPS ± Deformity</td>
<td>Special foot wear</td>
<td>3-6 mos</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Consider prophylactic surg if deformity can’t be safely accommodated in shoe. Pt. Ed</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>PAD ± LOPS</td>
<td>Consider prescriptive footwear. (by specialist) Vascular consult.</td>
<td>2-3 mos</td>
</tr>
<tr>
<td>3</td>
<td>Hx of amp ulcer</td>
<td>Same as Categ 1 Vascular consult prn (by specialist)</td>
<td>1-2 mos</td>
</tr>
</tbody>
</table>

Lower Extremity Assessment – High Risk

- If one or more high risk conditions
  - Evaluate more frequently, refer to specialist
  - Neuropathy- examine each visit
  - Multidisciplinary care important
    - Vascular specialist
    - Podiatrist
    - Orthotist
    - Certified Wound Ostomy Continence Nurse
    - Podorthist
    - Neurologist
    - Pain specialist
    - Endocrinologist
    - Advanced Practice Diabetes Specialists

ADA – Stds of Care 2008
**Onychomycosis**
- Chronic Infection 50% of nail problems
- We treat on skin but reluctant in nails
- Mean duration of > 10 years
- Rarely resolves spontaneously
- Spreads to other nails, skin, other people
- May be source of more serious infections
- Affects quality of life
- Vicks Vapor Rub?

**Patient Education**
- Proper footwear – no going barefoot, even indoors
- Daily foot inspection – look between toes and on sole of foot
- Prompt reporting of any foot lesions, discolorations or swelling

**Thank You**