What is Diabetes?
- A group of metabolic diseases in which a person has high blood sugar, either because:
  1) the body does not produce enough insulin
  ... or because...
  2) the cells of the body do not respond to the insulin that is produced

What Types of Diabetes are there, and in general, how are they managed?
- Type I
  - "Juvenile Onset" or Insulin Dependent (IDDM)
- Type II
  - "Adult Onset" or Non-Insulin Dependent (NIDDM)
- Gestational
  - Develops during 2 to 10% of pregnancies
  - At increased risk for developing Type II later on
  - 35-60% in next 10-20 years
- Other
  - Result of genetic conditions, surgery, medications, infections, pancreatic disease, and other illnesses

What is Hemoglobin A1c?

What are the possible ocular complications associated with Diabetes?
- Front to back of the eye approach
  - Emphasis on non-proliferative and proliferative retinopathy as well as macular edema
  - Images of complications
    - Photos, fluorescein angiography, OCT, Visual Fields
- How can these complications be treated/managed?

Interesting facts
- Affecting about 26 million people in US
  - 8.3% of population
- Estimated costs of Diabetes in US in 2007
  - $174 BILLION!
- Diabetes is 7th leading cause of death in US
- Diabetes is leading cause of kidney failure, nontraumatic lower-limb amputations, and new cases of blindness among adults
- Diabetes is a major cause of heart disease and stroke

Type I Diabetes
- Body fails to produce enough insulin
- Immune system attacks/kills beta cells in the pancreas
- Develops suddenly – kids in hospital
  - auto-immune, viral, genetics
- Accounts for about 5% of cases of diabetes
- Requires insulin treatment from the beginning
Type I Diabetes

Treatment Options
- Insulin – injections or pump
  - Rapid-acting: starts working within a few minutes and lasts for a couple of hours
  - Regular- or short-acting: takes about 30 minutes to work fully and lasts for 3 to 6 hours
  - Intermediate-acting: takes 2 to 4 hours to work fully, and its effects can last for up to 18 hours
  - Long-acting: takes 6 to 10 hours to reach peak levels in the bloodstream, but it can keep working for an entire day
- Pancreas/Islet Cell Transplant

Type II Diabetes

Usually begins with insulin resistance when cells fail to use insulin properly

Need for insulin rises but body fails to produce enough = insulin insufficiency

Associated with
- Older age, obesity, family history of diabetes, history of gestational diabetes, impaired glucose metabolism, physical inactivity, and race/ethnicity

Develops gradually
- Approximately 90% of cases of diabetes in US

Type II Diabetes

3 core defects
1. Genetic:
   - Pancreas cell are deficient and do produce enough insulin
2. Obesity:
   - Increased resistance to insulin in the tissues
3. Liver sugar storage issues:
   - Problems with hormone “glucagon” and it tells liver to release more sugar out into blood stream than it should

Type II Diabetes

Treatment Options
- Lifestyle intervention – weight loss, nutrition, meal planning
- Oral or Injectable Medications to:
  - Reduces insulin resistance
  - Increase insulin secretion
  - Reduce glucose secreted by liver
  - Reduce appetite, help with weight loss
- Insulin

Diabetes Treatment

Education, Understanding, Participation

Well-managed blood sugars

Addressing other risks:
- Smoking
- Elevated cholesterol – (LDL < 100)
- High blood pressure – (< 130/80)
- Obesity
- Lack of exercise

Diabetes Challenges

Blood glucose variability
- In a healthy patient – pancreas adjusts minute to minute to keep blood sugar between 70 and 140
- In a diabetic patient – no way for us to monitor glucose this frequently and to get medication in to the body to adjust it that quickly
Glucose Monitoring

- **Serum glucose** = immediate "snapshot" of blood sugar
  - Can be measured “post-prandial” (after a meal) or “fasting” (after 8 or 12 hours of not eating)

- **Hemoglobin A1c** = 3 month average blood sugar
  - Looks at how much sugar has stuck onto a RBC

**Diabetes Control and Complications Trial (DCCT)**

- **Type I patients**
- Compared Intensive treatment vs Conventional treatment and their rate of complications

- **Intensive Treatment HbA1c**: 7.2
- **Conventional Treatment HbA1c**: 9.1

- **Overall risk reduction**: 70%
United Kingdom Prospective Diabetes Study

- Newly diagnosed Type II
- Compared Intensive treatment vs Conventional treatment and their rate of complications

- Intensive treatment HbA1c 7.0
- Conventional treatment HbA1c 7.9

- Risk reduction of 35% per 1% decrease in hemoglobin A1c

Diabetes Complications

- Heart Disease
- Stroke
- Kidney Disease
- Impaired sensation/pain of hands and feet
- Skin sores, amputation
- Erectile Dysfunction
- Gastrointestinal Disease
- Genitourinary Disease
- Eye Disease

Mechanism of eye damage

- Poor blood sugar control = dilation/constriction of blood vessels
- Damaged lining = leak blood out into tissue
- Oxygen carried by blood does not get where it is supposed to
- Lack of oxygen = “hypoxia” or “ischemia”
- Hypoxia causes swelling of retina and loss of function
- Hypoxia causes release of hormone (VEG-F) to try to grow new blood vessels

Neovascularization

- New blood vessels grow without a lining and do not grow in the retina but out in vitreous and in front of eye as well
- Leak blood constantly, act as tension cords, and can clog drain of eye
- New blood vessel growth can occur on optic nerve, retina, iris or in the drain in the front of eye

Ocular Complications

- Extraocular Muscle Palsy
- Dry Ocular Surface
- Neovascularization of iris and drain of eye
- Shift in refractive error (glasses Rx change)
- Vitreous gel hemorrhage
- Non–Proliferative Retinopathy
- Proliferative Retinopathy
- Macular Swelling
- Retinal Non-Perfusion
- Tractional Retinal Detachment
- Neovascular Glaucoma
- Optic neuropathy

Cranial Nerve/Muscle Palsy

- Symptoms:
  - Sudden onset of double vision
  - May be worse in one position of gaze
  - May be able to turn head to compensate
  - Resolves when shut one eye
  - Image may be side by side, up and down, or at a diagonal
- Usually gets better within 3 months
- 3rd, 4th or 6th nerve palsy
- If does not resolve, may put prisms in glasses to realign images
6th Nerve Palsy R eye

Symptoms:
- Burning, itchy, gritty, watering, red, foreign body sensation, tired, light sensitive, have to blink frequently, intermittently blurred vision

Lens of eye helps to focus light rays onto retina
Typically lens prevents sugar from entering it
When sugar breaches 200+ in blood stream, it can seep into lens
To neutralize the sugar, water will be drawn in and lens swells
Swollen lens bends light differently

2 forms:
1) Non-Proliferative (NPDR)
2) Proliferative (PDR)

Macular edema (CSME)
- Fluid seeping out in the most sensitive part of the retina and therefore usually affects visual acuity
- May occur on its own or with NPDR or PDR

** all of these forms start out painless, without any initial symptoms

Microaneurysms (MAs)
- Tiny bulges off of retinal arterioles

Intraretinal hemorrhages
- "dot and blot"

Cotton Wool Spots (CWS)
- Fuzzy white spots in retina due to infarct of retinal arteriole and lack of oxygen causing retina to swell

Exudates
- Proteins seeping out of leaky blood vessels
- Well defined white or yellow deposits
- Usually form in a ring around central leaky vessel

Retinal thickening/swelling

Venous Beading
- "box-carrying" or "sausage-linking" of retinal veins

IntraRetinal Microvascular Anomalies (RMA)
- Anomalous fine caliber blood vessels that appear in pre-neovascular stage

Retinal Non-Perfusion
- Drop-out of blood vessels in retina

Neovascularization of Disc (NVD)
- New blood vessels growing off of optic disc

Neovascularization Elsewhere (NVE)
- New blood vessels growing any where else in retina
Non-Proliferative Retinopathy

Exudates in NPDR

Exudates
**Fluorescein Angiography (FA)**

- Used to see where leakage is occurring and to see where blood is not getting to as well
- Inject yellow dye into vein in hand/arm and take pictures as it reaches the eyes
- Blood in retina blocks view of dye so appears black
- Leaky areas of serum appears bright white
- Highlights new blood vessels growing
Staging of NPDR

- **Mild, Moderate, Severe**
- **4–2–1 Rule**
  - 4 quadrants of retinal hemes
  - 2 quadrants of venous beading
  - 1 quadrant of IRMA
  - **SEVERE**

- Dictates how urgently patient needs to return for follow up or be referred to retinal specialist for treatment
Significant Neovascularization

Hemorrhage in front of the Retina and in Vitreous gel due to Neovascularization

Traction due to Proliferative Retinopathy
Diabetic Macular Edema (DME)

Clinically Significant Macular Edema (CSME):
- Retinal edema within 1/3 DD of the foveal center
- Exudates within 1/3 DD of fovea with adjacent retinal edema
- Area of retinal edema that is 1 DD in size as long as a part of it is within 1 DD of fovea
Ischemic Maculopathy or Non-Perfusion =
Macula not really swollen because of leaking vessels, but literally all blood vessels in macula have died off and now no oxygen getting to it = poor vision

For every diabetic – minimum of dilated exam once a year !!
- Remember – often no symptoms

Mild to Moderate Non-Proliferative Disease
- Monitor closely
  - Dilated exams every 6 months
  - Work with endocrinologist or PCP to get better blood sugar control
  - Diet modifications, change meds, etc

Diabetic Macular Edema
- Followed every 2–3 months by ophthalmologist
  - VA, DFE, OCT, possible FA
- First Line of Treatment:
  - Anti VEG-F injections: Avastin, Lucentis, Eyelea
  - “Focal” or “Grid” laser treatment to burn/seal leaky vessels if not in exact center of macula

Pre-Proliferative and Proliferative Retinopathy
- Use laser to kill off part of retina so reduces oxygen demand
  - “Pan-Retinal Photocoagulation” – “PRP”
  - May do in sectors or throughout whole retina but will not do in the area of the macula
  - Will treat over many visits

Pan Retinal Photocoagulation (PRP)
Severe Proliferative Retinopathy with Pre-Retinal Hemorrhage in and eye that had previous PRP

Proliferative Retinopathy following PanRetinal Photocoagulation

Visual field in patient who had too much laser too close to fovea

Treatment Options

- **Severe Proliferative Retinopathy**
  - Injecting Anti VEG-F medications to reduce blood vessel growth (Avastin, Lucentis, Eyelea)
  - Vitrectomy – surgical procedure to remove vitreous gel from the inside of the eye and replace it with saline solution
    - Helps to eliminate VEG-F hormone from the gel
    - Helps to clear out in blood in the gel thereby getting better vision
    - Can get more PRP in while inside the eye

Iris/drain neovascularization

- Hormone (VEG-F) causes new vessel growth at margin of pupil first, then back along iris to the drain
- Can see the vessels on the iris with the microscope but need a special mirror to look for vessels in the drain
- New vessel growth clogs the drain, preventing fluid from getting out and pressure eye can build quickly

Neovascularization of the Iris
Sudden increase in eye pressure causes eye to become red, painful, even feel nauseous.

Spike in pressure is so high that will cause damage to optic nerve quickly and this causes blindness.

Treatment – prevention, PRP, vitrectomy, eye drops to lower pressure, surgery to make new drain for fluid to flow out of.

Bad visual outcome – may end up with enucleation.

Causes sudden painless but severe loss of vision in one eye.

Due to vein being compressed by hardened overlying artery.

Poor visual outcome.

May also develop neovascular disease and its complications.

Swollen Optic Nerve (Neuropathy) due to Ischemia

Sudden painless loss of vision in one eye.

Color vision also reduced.

APD.

Usually 20/100–200.

No effective treatment – vision may improve on its own, may not.

Occlusion of Retinal Vein

Causes sudden painless but severe loss of vision in one eye.

Due to vein being compressed by hardened overlying artery.

Poor visual outcome.

May also develop neovascular disease and its complications.

“Drive the bus, or it will run over you!”

Thank you! Any questions?