Diabetes Mellitus in Small Animals

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Diabetes Mellitus

“Persistent hyperglycemia caused by a relative or absolute insulin deficiency”

Not a single disease but a syndrome that results in hyperglycemia

- Defects in insulin secretion
- Defects in insulin sensitivity
Insulin

• Produced by β-cells of the islets of Langerhans in the pancreas
• Associated with energy abundance
• Binds to insulin receptor on it’s target cell
  • Enzyme linked receptor
    • Binds to alpha subunits outside the cell
    • Beta subunits become autophosphorylated >> activates local tyrosine kinase >> phosphorylation of insulin-receptor substrates [IRS]
    • Different IRS are expressed in different tissues
  • Increases uptake of glucose
    • Muscle cells
    • Adipose tissue
    • NOT neurons in the brain
Insulin

• Glucose transport – within seconds
  • Translocation of multiple intracellular vesicles which contain multiple molecules of glucose transport proteins to the cell membrane
  • Glucose transport proteins bind to cell membrane and facilitate uptake of glucose into the cells
  • When insulin is no longer available vesicles separate from the cell membrane within 3-5 minutes >> move intracellularly and are reused

• Cell membrane permeable to amino acids, potassium and phosphate ions
  • Move intracellularly

• Slower effects
  • In 10-15 minutes, changes activity of intracellular metabolism via phosphorylation of enzymes
  • Over hours to days, changes translation of mRNA in ribosomes and transcription of DNA in the nucleus of the cells >> molds cellular enzymes activity to achieve metabolic effects
Carbohydrate Metabolism

• Cause rapid secretion of insulin once in the blood stream
• Muscles
  • During rest, use fatty acids for energy
  • During moderate to heavy exercise, muscle contraction increased translocation of GLUT 4 from intracellular stores >> facilitates diffusion of glucose into the muscle cell
  • Few hours after meals, high insulin secretion causes rapid transport of glucose into muscle cells >> can be stored as glycogen in the muscle cell and used later for energy
Carbohydrate Metabolism

• Liver

  • Insulin causes storage of glucose as glycogen
    • Inactivates liver phosphorylation >> prevent break down of glycogen
    • Enhances uptake of glucose by hepatocytes
      • Increased activity of glucokinase [phosphorylates glucose>> taps it in the hepatocyte]
    • Increases activity of enzymes that promote glycogen synthesis
      • Glycogen synthase

  • Decreased insulin
    • Reverses effect >> decreases glycogen synthesis and prevents further uptake of glucose by hepatocytes
    • Increases splitting of glycogen into glucose [activates phosphorylase and glucose phosphatase enzymes]

  • Insulin promotes conversion of excess glucose into fatty acids
    • LVDL triglyceride production >> transported to adipose tissue
Carbohydrate Metabolism

• Most brain cells are permeable to glucose and can use glucose without the intermediation of insulin

• When blood glucose is very low >> hypoglycemic shock
  • Progressive nervous irritability
  • Fainting
  • Seizures
  • Coma
Fat Metabolism

• Increased transport of glucose into hepatocytes
  • After glycogen concentration in the hepatocytes reaches 5-6%, further glycogen synthesis is inhibited
    • Fatty acids synthesized >> transported in VLDL as triglycerides

• Insulin activates lipoprotein lipase in capillary walls
  • Splits triglycerides back into fatty acids >> absorbed into adipocytes >> converted to triglycerides and stored as fat

• Insulin inhibits lipase
  • Release of fatty acids from fat stores is inhibited

• Insulin promotes glucose transport into adipocytes
  • Ultimately increases glycerol >> combines with fatty acids to form triglyceride
Fat Metabolism

• Insulin deficiency
  • Lipase is strongly activated in the absence of insulin
    • Hydrolysis of stored triglycerides >> release of fatty acids
    • Fatty acids used for energy
  • Increased conversation of fatty acids into cholesterol and phospholipids
    • High cholesterol promotes development of atherosclerosis in people with DM
  • Causes excess acetoacetic acid formation in the liver >> blood >> intracellularly >> energy [acetyl-CoA]
    • Lack of insulin also depresses utilization of acetoacetic acid in peripheral tissue >> acidosis
      • Some acetoacetic acid gets converted to ketones bodies [β-hydroxybutyric acid and acetone] >> ketosis
Protein Metabolism

• Insulin
  • Stimulates transport of amino acids into cells
  • Increases translation of mRNA >> Promotes new protein synthesis
  • Increases transcription of select DNA genetic sequences>> Promotes new protein synthesis
  • Inhibits catabolism of proteins
    • Diminishes normal degradation of protein in cellular lysosomes
    • In hepatocytes, depresses the rate of gluconeogenesis [from amino acids]

• Insulin deficiency
  • Increased protein degradation
    • Amino acids used for energy
    • Increased urea secretion by the kidneys
  • Protein synthesis stops
  • Weakness
Diabetes Mellitus

• Caused by
  • Lack of insulin secretion
  • Decreased sensitivity of the tissues to insulin

• Syndrome of impaired carbohydrate, fat and protein metabolism
  • Insulin deficiency or insulin resistance
    • Results in increased blood glucose concentration
    • Decreased cellular utilization of glucose
    • Increased utilization of fat and protein
Diabetes Mellitus

• Blood glucose rises >> spillage of excessive glucose into the urine
  • Polyuria [osmotic diuresis]

• High blood glucose >> cellular dehydration [osmotic pressure]
  • Polydipsia

• Increased utilization of fats for energy
  • Release of keto acids >> metabolic acidosis
    • Rapid deep breathing pattern [Kussmal breathing]

• Depletion of body proteins
  • Weight loss
  • Asthenia [lack of energy]
  • Polyphagia
Diagnosis

• Appropriate clinical signs
  • PU/PD/PP
  • Weight loss

• Persistent fasting hyperglycemia

• Glucosuria
Diabetes Mellitus

• **Type 1**
  • Deficiency of insulin production by pancreatic islet β-cells – usually absolute deficiency
    • Viral infection
    • Autoimmune – cell mediated
    • Hereditary
  • Typically occurs abruptly
    • Over days to weeks

• **Type 2**
  • Usually relative insulin deficiency
  • Resistance to metabolic effects of insulin
    • Hyperinsulinemia due to target cell resistance
  • Gradual onset
    • Obesity
    • Excessive glucocorticoids
    • Excessive growth hormone [acromegaly]
    • Pregnancy
    • Autoantibodies/mutations to insulin receptor
Canine

• Type 1 most common
  • Have been associations found between three specific haplotypes and DLA [MHC] genotypes in diabetes-prone breeds of dogs

• Pancreatitis

• Insulin resistance
  • Hyperadrenocorticism
  • Diabetogenic drugs – pre-existing β-cell defect, islet pathology
    • Steroids – glucocorticoids, progestin
  • Obesity – does not play a significant role although does produce insulin resistance

• Gestational/diestrus induced diabetes mellitus
  • Progesterone induced growth hormone secretion >> insulin resistance, carbohydrate intolerance
  • Diabetic remission possible after resolution of diestrus
Feline

- Secretory failure
  - Damaged pancreatic islet β-cells
    - Amyloid deposition >> cytotoxicity > apoptosis
  - Glucose toxicity
    - Oxidative stress and inflammatory cytokines??
    - Dependent on degree of hyperglycemia and duration of hyperglycemic state
  - Lipid toxicity
    - Increased fatty acids >> cell death

- Commonly Type 2
  - Insulin resistance cause by
    - Obesity
      - Decreased expression of GLUT4 transporter in muscle and fat
      - Decreased adiponectin >> decreased insulin sensitivity, decreased anti-inflammatory
      - Leptin modulates insulin sensitivity – obese cats are leptin resistant
  - Acromegaly*
  - Diabetogenic drugs* - pre-existing β-cell defect, islet pathology
    - Steroids – glucocorticoids, progestin

*Type 3 in the American Diabetes Association scheme
Complications of uncontrolled DM

• Canine
  • Cataracts
    • Long term complication – days months or years
    • Irreversible
    • Lens induced uveitis
  • Diabetic retinopathy
    • Uncommon
  • Diabetic nephropathy
    • Occasionally reported
  • Hypertension
    • Incidence 46% (Struble, et al 1998)

• Feline
  • Peripheral neuropathy
Prognosis

Canine

• ~ 3 years average survival time
• High mortality rate un the first 6 months
  • DKA
  • Concurrent illness
  • Owner unwillingness to treat
• If survive the first 6 months, average survival time is ~ 5 years

Feline

• Variable
• If survive the first 6 months, average survival time is ~ 5 years
Treatment

INDIVIDUALIZED THERAPY

• Goal – good quality of life
  • Reduce blood glucose to control PU/PD/PP and weight loss
  • Prevent ketosis
  • Avoid hypoglycemia
Treatment

Canine

• Remission is not a goal
• Near euglycemia is not necessary
  • Increases risk of hypoglycemia and its consequences
Treatment - Canine

• Vetsulin

• NPH

• Detemir – very potent!!!!
  • 0.1 unit/kg
  • Very difficult to use in a small breed dog
    • High risk for hypoglycemia
    • Lack of ability to fine tune the dose
  • Can get a specific diluent from manufacturer

• Glargine, PZI – overall poor response
  • Maybe useful in an individual patient
Diet - Canine

Many specific diabetic diets available
  • Majority are high fiber
• Remains to be proven that these diets offer a clinically significant advantage
• Choose a diet based on
  • Comorbidity
  • Palatability to individual patient
    • Reliable intake of a steady amount of calories at regular intervals
Treatment - Feline

• Insulin
  • Longer acting insulins: Glargine, PZI, Detemir
    • Longer average duration of action >> improved glycemic control >> reduce glucotoxicity and lipotoxicity >> greater remission potential

• Oral hypoglycemic - not optimal if diabetic remission is a goal
  • Glipizide
  • Acarbose

• Addressing obesity if applicable
Diet - Feline

Goals

• Improve metabolism
• Correct obesity
• Remove persistent hyperglycemia
  • High protein
  • Low carbohydrate

Many specific diabetic diets available
Insulin Therapy
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**Long-Acting**

| Insulin glargine (Basaglar, Lantus, Toujeo) | 1-1 1/2 hours | No peak time, Insulin is delivered at a steady level. | 20-24 hours | Long-acting insulin covers insulin needs for about one full day. This type is often combined, when needed, with rapid- or short-acting insulin. |
| Insulin detemir (Levemir) | 1-2 hours | 6-8 hours | Up to 24 hours |
| Insulin degludec (Tresiba) | 30-90 min | No peak time | 42 hours |

**Pre-Mixed***

| Humulin 70/30 | 30 min. | 2-4 hours | 14-24 hours | These products are generally taken two or three times a day before mealtime. |
| Novolin 70/30 | 30 min. | 2-12 hours | Up to 24 hours |
| Novolog 70/30 | 10-20 min | 1-4 hours | Up to 24 hours |
| Humulin 50/50 | 30 min. | 2-5 hours | 18-24 hours |
| Humalog mix 75/25 | 15 min. | 30 min. - 2 1/2 hours | 16-20 hours |

*Premixed insulins combine specific amounts of intermediate-acting and short-acting insulin in one bottle or insulin pen. (The numbers following the brand name indicate the percentage of each type of insulin.)

from wedmd
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Insulin Cost

• NPH insulin
  • Novolin N   GoodRx: $104-154 for pet Rx [$24-28]
  • Humulin N   GoodRx: $160-62 for pet Rx [$100]

• Glargine   Good Rx: $262-285 for pet Rx [$184]

• Vetsulin   Online pharmacies $36-43

• ProZinc   Online pharmacies $96-126
Monitoring

Various tools available

• Clinical picture
  • Level of PU/PD/PP
  • Weight - loss or gain

• Spot check blood glucose
  • Beware of stress hyperglycemia
  • Good for revealing hypoglycemia, remission relapses
  • Not recommended to use to make daily or inter-day dosage changes to insulin

• Glucose curves
  • Glucose nadir
  • Time to onset of action
  • Duration of action of insulin
Monitoring

Home urine glucose monitoring

- Negative urine glucose should trigger checking a blood glucose check
- Stress hyperglycemia can cause glucosuria
- Checking for remission relapses
Glucose curves

Home blood glucose monitoring

- Increasingly popular
  - Stress free environment
  - More economical
  - Aids in preventing hypoglycemia and promoting diabetic remission in cats
- Owner AND pet must be willing/able

In clinic

- For patient who will not tolerate or owners that are uncomfortable/unable at home monitoring
  - Stress hyperglycemia

Continuous glucose monitors

- Can limit stress
- Cost – device, probe/curve
- Requires calibrations with peripheral blood glucose 1-3 times/day
Rapid metabolism of insulin

![Rapid metabolism of insulin graph](http://www.vetsulin.com/vet/images/Cats-Rapid-metabolism_small_rev.gif)

Somogyi

![Somogyi effect graph](http://www.vetsulin.com/vet/images/Cats-Somogyi-effect_small_rev.gif)
Insulin administered at time=0

Blood Glucose Concentration (mg/dL)

Time in Hours

Monitoring
Glycated proteins
- Fructosamine
  - Sampling artifacts that lower Fructosamine
    - Storage
    - Lipemia
    - Azotemia
    - Hypoproteinemia
  - Hypoglycemia-induced hyperglycemia will increase Fructosamine
  - Concurrent disease that effect protein catabolism can lower Fructosamine
    - Hyperthyroidism
- Glycosylated hemoglobin
  - Ac1
Roary
4 year old castrated male DSH

Lethargic
Decreased appetite

PE: normal vitals, normal rectal temperature

CBC – normal

Biochemical profile
  • Hyperglycemia 357
  • Hyponatremia 142
  • Hypercholesterolemia 490

UA
  • 3+ glucosuria
  • 3+ ketonuria
  • WBC, RBC
Does this cat have Diabetes mellitus?
What treatment should we use?
What insulin should we use?

Glargine OR PZI [ProZinc] insulin
Roary

• Treatments
  • IVF, Cefazolin
  • Glargine insulin
    • 1 unit SQ q 24 hrs
  • MD diet

Day 1
• 8am  BG 106
• 11 am  BG 198

Day 2
• 8 am  BG 517
• 12 pm  BG 266

Sent home on
  ▪ Glargine 2 u SQ q 12 hrs
  ▪ Amoxicillin for UTI
Roary

5 days after diagnosis
lethargic again
- BG 419
- Ketone 4.9

6 days after diagnosis at ER clinic
- BG 633

Treated with an insulin CRI
- Regular insulin

On day 8 after diagnosis
- Transition back to Glargine 2 u SQ
  - 8 pm BG 544
  - 11 pm BG 364

Day 9
- 2 am BG 300
- 5 am BG 469
- 8 am BG 428 – Glargine 3 u SQ
- 11 am BG 295
- 2 pm BG 150
- 5 pm BG 237
- 8 pm BG 583 – Glargine 3 u SQ

Day 10
- 2 am BG 85, 77
- 4 am BG 88
- 5 am BG 71
- 6 am BG 130
- 9 am BG 521
  - Glargine 3 u SQ
- 12 pm BG 391
- 3 pm BG 56
- 6 pm BG 205
- 9 pm BG 328
  - Glargine 1 u SQ
Roary

Day 11
• 12 am BG 390
• 3 am BG 249
• 6 am BG 598

Used combination of Glargine and Regular insulin

Discharged on Glargine 3 u SQ q12 hrs

Day 23 after diagnosis
Owner did “mini” curve at home
• 9am BG 583 – Glargine 3 u
• 2 pm BG 369
• 5 pm BG 367
• 7 pm BG 483
• 9 pm BG 488 – Glargine 3 u
• 7 am BG 269 – Glargine 3 u
Roary

Changed insulin dosing to

Glargine 4 u SQ in am and 3 u SQ in pm
Feline

• Diabetic remission?
  • Improve β-cell function
    • Treating hyperglycemia in a timely manner
    • Optimize glycemic control
  • Cats that develop DM later in life
  • Newly diagnosed DM that is treated effectively
  • Cats with diabetic neuropathy less likely to achieve remission
  • Reduced carbohydrate, high protein diet may help
    • Consistent diet type and calorie intake most important for a more predictable insulin requirement
Summary

• Persistent hyperglycemia causes by relative or absolute insulin deficiency
• Insulin effects carbohydrate, protein and fat metabolism
• Dogs generally have absolute insulin deficiency
• Cats generally have a relative insulin deficiency

• Treatment must be individualized
• Different treatment goals for dogs vs cats
• Monitoring must be individualized
• Technicians play a vital role in helping manage a diabetic patient