- Chetoacidosi diabetica
- Sindrome iperosmolare

DKA

Cheto-acidosi Diabetica

Insufficienza Insulinica (relativa !)

corpi chetonici

Acidosi metabolica

Disidratazione (grave)

shock

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Metabolismo epatico

- **LIPIDI**
- Corpi chetonici → acidi grassi non esterificati
- → βoxid-mitocondriale-ATP dipendente , ACETIL-coA → accumulo epatico → acetoacetil-coA

- Nei non-diabetici **acetill-CoA** e il **piruvato** entrano nel ciclo di Krebs per formare ATP.
- Nei diabetici il glucosio non entra nelle cell in quantità adeguata e il piruvato prodotto della glicolisi diminuisce
- L'attività del ciclo dell'ac. Citrico di conseguenza diminuisce con accumulo di acetil-coA

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Insulina / glucagone

- La sintesi di acetil-coA è facilitata da
- deficit di insulina
- Amento della concentrazione di glucagone
- Effetto ANABOLICO dell'insulina → deposito di acidi grassi nel t.to adiposo
- Effetto CATABOLICO di glucagone → lipolisi

Corpi Chetonici

- B-idrossi butirrato (principale nel cane e nel gatto)
- Acetoacetato

- Acetone

B-idorssi e acetoacetato si comportano da strong acids (azione media), l'accumulo porta ad ACIDOSI METABOLICA →

può essere aggravata dalla presenza di vomito, disidratazione e ipoperfusione renale

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- CHETOSI
- ACIDOSI METABOLICA
- Squilibri ELETTROLITICI

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HHS

Sindrome Iperosmolare Iperglicemica

(Coma Iperosmolare, diabete non chetosico iperosmolare)

- Rara (!?)
- IPERGLICEMIA GRAVE
- DISIDRATAZIONE GRAVE
- Tradizionalmente (Med Umana) DKA → patogenesi acuta, (in 24 ore nei soggetti givani affetti da DM tipo I, HHS → sviluppo in circa 10 giorni, pazienti anziani affetti da DM tipo II
- Recentemente : DKA e HHS possono decorrere simultaneamente, stadio "misto" DKA/HHS in 30 % dei DM in emergenza (Med Umana) inclusi giovani e pediatrici
- Indifferentemente tipo I o tipo II DM

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DKA/HHS

- *Segnalamento (cane)*
- Età media circa 8 anni (range, 8 mesi fino a 16 anni !!!!).
- Nessuna predisposizione di razza o sesso

- Segni Clinici (cane)
- Dm cronico o non-trattato
- Presenza di malattie concomitanti (70% DKA)

(Pancreatite acuta, infezioni (urinario), iperadrenocorticismo)

- DKA insorgenza acuta

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gatto

- *Segnalamento*
- Età media circa 10 anni (range, 5 - 14 anni).
- Nessuna reale predisposizione di razza o sesso

- Segni Clinici (cane)
- Dm non-diagnosticato/ non-trattato 8assenza sintomi riconoscibili)
- Presenza di malattie concomitanti

(malattia renale cronica, FLUDT, cardiomiopatie, infezioni, neoplasia)

- DKA insorgenza acuta

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Segni clinici (cane)

- Poliuria/Polidipsia
- letargia
- Inappetenza - anoressia,
- vomito
- Perdita di peso
- → Esame clinico
- BCS. Dimagrimento/ sovrappeso
- Disidratazione
- Organo-megalia (settore craniale)
- Dolorabilità alla palpazione addominale
- Soffio cardiaco
- Depressione del sensorio
- Alterazioni dermatologiche
- Tosse/ murmure vescicolare respiratorio
- cataratta

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gatto

- letargia
- Inappetenza - anoressia,
- vomito
- Dimagrimento, perdita di peso

- → Esame clinico
- BCS. Dimagrimento
- Organomegalia (settore craniale)
- Disidratazione
- Dolorabilità alla palpazione addominale
- Soffio cardiaco
- Depressione del sensorio → decubito permanente→ stato comatoso
- debolezza
- Dispnea /tachipnea
- Alito acetonico
- Ittero
- Atteggiamento plantigrado

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Diagnosi DKA

- pH < 7.35
- Beta-idrossi-but > 2 mmol/L (4)
- Iperglicemia range 180 – 900 mg/dL ****
  In media 450 mg/dL
- HHS
- Iperglicemia > 600 mg/dl
- pH > 7.30 (no chetoni)
- Osmolarità effettiva > 320
- Alterazione dello stato mentale

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Ematologia

- 50% Anemia non-rigenerativa
  (non associata a ipofofatemia)
- Neutrofilia (left shift)
- trombocitosi

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Es. biochimico

- Iperglicemia persistente
- ALT
- AST
- SAP
- Colesterolo
- Iperbilirubinemia (gatto)

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Alterazioni elettrolitiche

- Ipokalemia/Iperkalemia-
- → riduzione del potassio corporeo
- Iperkalemia : iperglicemia, ipoinsulinemia , acidosi metabolica
- Ipokalemia: corpi ketonici (binding), vomito, anoressia. Incipit insulino-terapia !

- Debolezza muscolare, dispnea → paralisi respiratoria

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ipofosfatemia

- Ipofosfatemia-normofosfatemia, alcuni iperfosfatemia
- → riduzione del fosfato corporeo
- Iperfosfatemia: iperglicemia, ipoinsulinemia, acidosi
- Ipofosfatemia: diuresi, fluido terapia/insulino-terapia
- → crisi convulsive (?- case report)
- → crisi emolitica

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- Magnesio
- Med Umana ≠ cane
- → nessuna aprezzabile diminuzione del Mg++

- Iponatriemia, ipocloremia,
- Diminuzione del Ca ionico

50% DKA cani

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Journal of Veterinary Emergency and Critical Care

Original Study

Journal of Veterinary Emergency and Critical Care 22(2) 2012, pp 211–218
doi: 10.1111/j.1476-4431.2012.00719.x

Use of lispro insulin for treatment of diabetic ketoacidosis in dogs

Kirk W. Sears, DVM; Kenneth J. Drobatz, DVM, MSCE, DACVIM, DACVECC and Rebecka S. Hess, DVM, DACVIM

Abstract

Objectives – To characterize the use of lispro insulin in dogs with diabetes ketoacidosis (DKA) and to compare the length of time required for resolution of hyperglycemia, ketosis, and acidosis, respectively, in dogs with DKA treated with lispro or with regular insulin.
Design – Randomized prospective clinical trial performed between November 2006 and May 2009.
Setting – University teaching hospital.
Animals – Client-owned dogs with naturally occurring DKA. Dogs with a blood glucose (BG) > 13.9 mmol/L (>250 mg/dL), blood pH between 7.0 and 7.35, and a blood beta-hydroxybutyrate (BOHB) concentration >2.0 mmol/L were eligible to be enrolled into the study and were randomly assigned to receive an IV continuous rate infusion (CRI) of either lispro or regular insulin.
Interventions – Lispro or regular insulin was administered as an IV CRI at an initial dose of 0.09 U/kg/h. The dose was adjusted according to a previously published protocol.
Measurements and Main Results – Twelve dogs were enrolled into the study. The time to biochemical resolution of DKA was defined as the time interval from when the IV CRI of insulin began until marked hyperglycemia (BG > 13.9 mmol/L [>250 mg/dL]), acidosis (venous pH < 7.35), and ketosis (BOHB concentration >2.0 mmol/L) resolved. The median time to biochemical resolution of DKA in dogs treated with lispro insulin was significantly shorter (26 h; range 26–50 h) than in dogs treated with regular insulin (61 h; range, 38–80 h, $P = 0.02$). Median admission blood glucose concentration of all 12 dogs (24 mmol/L [432 mg/dL; range, 17.8–38.9 mmol/L [321–700 mg/dL]) decreased significantly with fluid resuscitation and prior to insulin therapy (20.5 mmol/L [369 mg/dL; range, 14.5–33.3 mmol/L [261–600 mg/dL], $P = 0.0085$). No adverse effects were observed in association with IV lispro insulin administration.
Conclusions – Treatment of DKA in dogs with IV CRI lispro insulin is safe, and as effective as treatment with regular insulin.

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Keywords: acidosis, canine, diabetes mellitus, ketosis

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Journal of Veterinary Emergency and Critical Care

Original Study

Journal of Veterinary Emergency and Critical Care **23**(3) 2013, pp 286–290
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Intramuscular glargine with or without concurrent subcutaneous administration for treatment of feline diabetic ketoacidosis

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Abstract

Objective – To describe treatment response and outcome in 15 cats with diabetic ketoacidosis (DKA) initially stabilized with glargine administered intramuscularly (IM) with or without subcutaneous (SC) glargine.

Materials and Methods – Fifteen cats diagnosed with DKA were initially administered IM glargine (1–2 U) and in most cats (12/15 cats) this was combined with SC glargine (1–3 U). This was followed by intermittent IM glargine as required at intervals of 2 or more hours (range 2–22 h) and SC glargine (1–2 U) every 12 hours.

Key Findings – All 15 cats survived and were discharged from hospital (median 4 d; range 2–5 d) and one-third (5/15) of cats subsequently achieved remission (median time 20 d; range 15–29 d). Complications included hypokalemia and hypophosphatemia, which were likely the result of DKA therapy rather than glargine treatment specifically.

Significance – This study demonstrates that glargine administered IM is an effective treatment for DKA in cats, and may provide an alternative to regular insulin. The same vial used for initial treatment of DKA can then be used for subsequent management with SC glargine injections. Future prospective randomized controlled trials evaluating clinical outcomes in cats with DKA using different types and routes of administration of insulin are warranted. A prospective randomized controlled trial is required to compare outcomes for IM and IV administration of glargine and regular insulin in DKA cats with or without SC glargine.

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Keywords: cats, diabetes, insulin, treatment

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