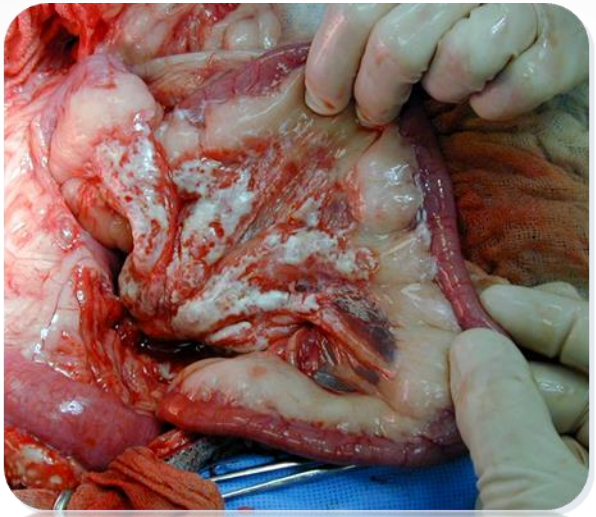


pancreatite

- Infiammazione del parenchima pancreatico
- NON settica (di solito)
- Comune nel cane e nel gatto

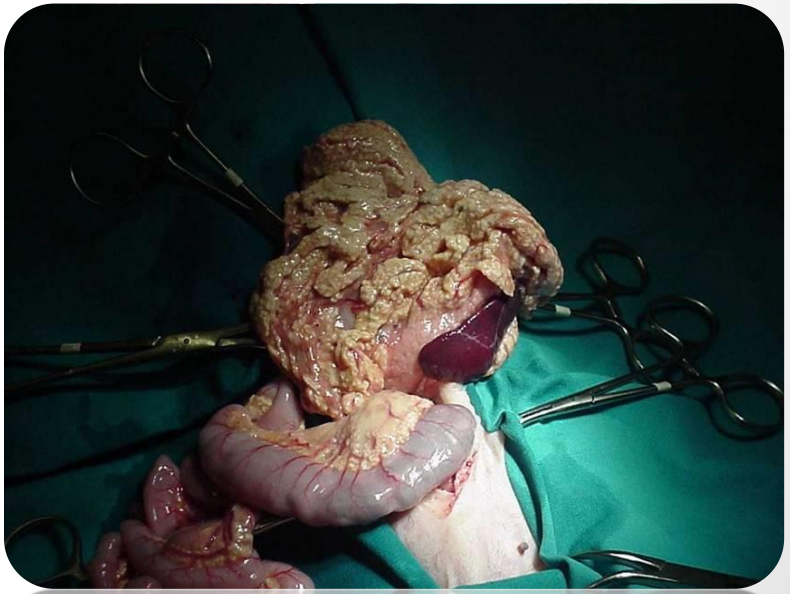
Pancreatite canina

ACUTA

non è associata a alterazioni permanenti del parenchima

CRONICA

con alterazioni permanenti del parenchima



NON sono differenziabili clinicamente
ENTRAMBE possono essere associate a infiltrati neurofilici o linfo-plasmocitari

PANCREATITE ACUTA GRAVE

Severe Acute Pancreatitis (SAP)

COMPLICAZIONI SISTEMICHE
ARDS
CID
SIRS (Systemic Inflammatory Response Syndrome)
MODS (Multiorgan Dysfunction Syndrome)

COMPLICAZIONI LOCALI
(necrosi del tessuto pancreatico, accumulo di fluido nel tessuto pancreatico)

Pancreatite cronica

Meno comunemente associata a complicazioni locali o sistemiche
è origine di una malattia lieve-moderata

Tabella I, classificazione pancreatite felina su base isto-patologica	
Pancreatite acuta necrotizzante	Aspetto predominante: necrosi del parenchima Necrosi acinosa-cellulare del parenchima, steatonecrosi peripancreatica, vari gradi di infiammazione, emorragie, mineralizzazione e fibrosi
Pancreatite acuta suppurativa	Aspetto predominante: infiammazione neutrofilica Infiltrato neutrofilico >50%, segni di necrosi
Pancreatite Cronica non-suppurativa	Aspetto predominante: infiammazione linfocitaria Possono essere presenti quadri focali con aspetti suppurativi e necrotici.
Iperplasia pancreatica nodulare	Aspetto predominante: lesioni nodulari a carico del parenchima e dei dotti Di solito NON si accompagna a necrosi, emorragia, infiammazione e fibrosi.
Neoplasia Pancreatica	Primaria: adenoma, adenocarcinoma o secondaria Quadro predominante: adenocarcinoma Prevalentemente a carico del dotto, si accompagna a necrosi, infiammazione, fibrosi, emorragia o mineralizzazione
Pseudocisti pancreatica	Complicazione locale non-infrequente Quadro predominante: struttura cava dotata di parete non-epiteliale, contenente liquido, cellule pancreatiche o enzimi localizzata nel parenchima pancreatico
Ascesso pancreatico	Aspetto predominante: collezione di materiale purulento a carico del lobo destro o sinistro con aspetti del tutto simili alla pseudocisti Sconosciuti reali incidenza e significato nel gatto
Atrofia pancreatica	Aspetto predominante: atrofia del parenchima Possibile risultato di quadri degenerativi involutivi, necrotici del parenchima. Probabile stadio finale della pancreatite cronica

epidemiologia

Razze	RATE	*P* VALUE
Miniature Schnauzer	4.52%	$p < 0.0001$
Shetland Sheepdog	2.34%	$p < 0.0001$
Yorkshire Terrier	2.10%	$p < 0.0001$
Miniature Poodle	1.69%	$p < 0.0001$
Bichon Frise	1.60%	$p < 0.0001$
Samoyed	1.56%	$p = 0.0007$
Standard Poodle	1.56%	$p < 0.0001$
Miniature Pinscher	1.40%	$p = 0.032$
Maltese	1.39%	$p = 0.0035$
Shih Tzu	1.19%	$p = 0.0014$
Pomeranian	1.18%	$p = 0.028$
Toy Poodle	1.18%	$p = 0.012$
Miniature Dachshund	1.18%	$p = 0.041$
Basset Hound	1.15%	$p = 0.045$
Dachshund	1.13%	$p = 0.005$

eziologia

- Cause sconosciute **IDIOPATICA (90%)**
- **Fattori predisponenti**
- Stati ipossici, ischemici (chirurgia-anestesia, shock, trauma)
- Dieta e fattori nutrizionali: elevato contenuto in grassi, «errori alimentari» – indiscrezioni alimentari
- Iperlipemia, ipertrigliceridemia: Schwauzer – idiopatica, secondaria a **DM**, iperadrenocorticismo, CKD (liberazione di adipochine)
- Obesità
- Ipercalcemia
- Farmaci

L-asparaginasi, azatioprina, estrogeni, diuretici (furosemide), bromuro di potassio, salicilati e FANS, sulfamidici, tetracicline, procainamide, clomipramina, organo fosforici, fenobarbitale, propofol, composti antimoniali, N-metil-glucosammina, alcaloidi della vinca

eziologia

- Cause **genetiche- ereditarie** (uomo, gene tripsinogeno cationico !)

Schnauzer nano (SPINK 1 inibitore secretorio pancreatico della tripsina)

→ **Traumatica**

→ **Ostruzione duttale, reflusso biliare, secrezione aumentata**

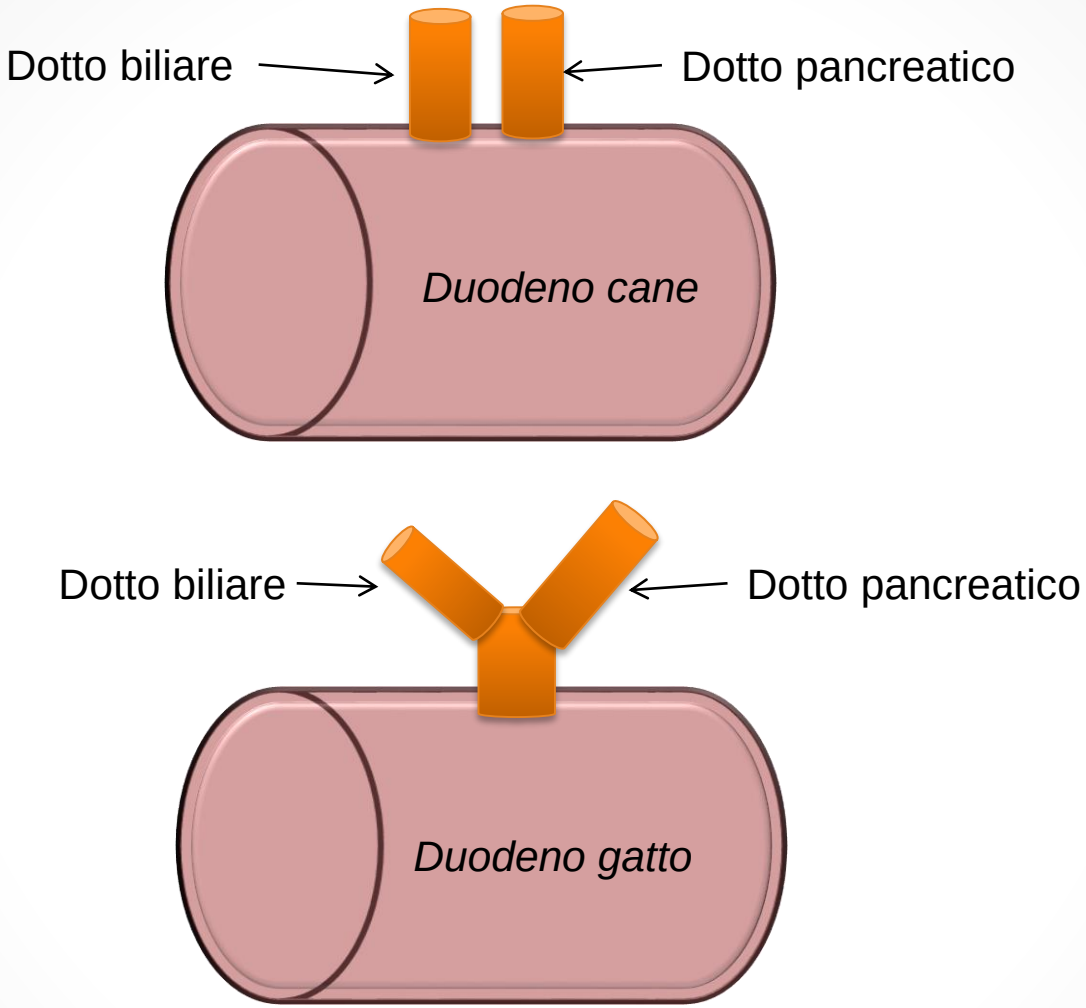
Vomito persistente, corpo estraneo, occlusione intestinale

- Cocker Spaniel (pancreatite cronica autoimmune: colangite distruttiva)
- Babesia
- Batteri ? (*Pseudomonas aeruginosa, Klebsiella pneumonia*)

eziologia: gatto

- → **Malattie concomitanti del tratto biliare**
- → **Malattie concomitanti del tratto gastro-enterico**
- → Ischemia
- → Malattie ostruttive del dotto pancreatico
- → Malattie infettive (Toxoplasma gondii, herpesvirus felino I, coronavirus, parvovirus, calicivirus, Peritonite Infettiva Felina)
- Trauma
- Ipercalcemia (Acuta)/Organofosforici
- Nutrizione
- Reazioni idiosincrasiche ai farmaci

Tabella II. Eziologia della pancreatite acuta necrotizzante felina
Associazioni riconosciute **Malattie epato-biliari** **Ischemia** **Malattie infettive** **Organofosfati** **Malattie gastro-enteriche** **Malattie ostruttive del dotto pancreatico** **Trauma** **Lipodistrofia**
Associazione probabili **Ipercalcemia** **Reazione da farmaci** **Nutrizione**


Dotto biliare
Dotto pancreatico
Duodeno cane
Dotto biliare
Dotto pancreatico
Duodeno gatto

patogenesi

- *l'attivazione **precoce ed inappropriata** dello zimogeno tripsinogeno a tripsina e di altre proteasi pancreatiche all'interno degli acini pancreatici: «**co-localizzazione**» idrosilasi + enzimi pancreatici*

- GATTO…«crinofagia» coalescenza catepsina B N-acetil-glucosaminidasi - zimogeni

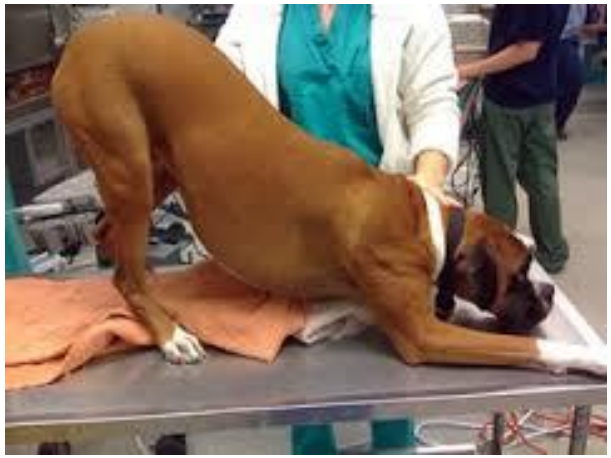
- sopraffazione delle **difese locali** tissutali (PSTI, Pancreatic Secretory Trypsin Inhibitor/ SPINK 1) e delle molecole antiproteasiche (α1 antitripsina, α2 macroglobuline) in circolo

- fenomeni stress-ossidativi o ipotensivi + diminuzione del pH all'interno dell'acino ed elevata concentrazione di calcio intra-citoplasmatico
- CCK (colecistochinina) agisce come mediatore intracellulare del rilascio delle proteasi lisosomiali.

- risposta infiammatoria locale con migrazione di neutrofili e conseguente liberazione di ROS (reactive oxygen species) e NO (ossido nitrico)
- «cytokine storm» - TNFα, IL-1-2-6, NO, ROS, PAF platelets activating factors

Segni clinici

- Dolorabilità addominale
- Vomito !
- Depressione del sensorio



- Diarrea
- Sintomi sistemici… ittero, tachipnea, tachicardia – disaritmia, ipertermia

- Patologia Clinica
- Leucocitosi, neutrofilica→ left shift
- Aumento PCV - diminuzione
- Trombocitosi – trombocitopenia
- Ipoalbuminemia
- ipocalcemia

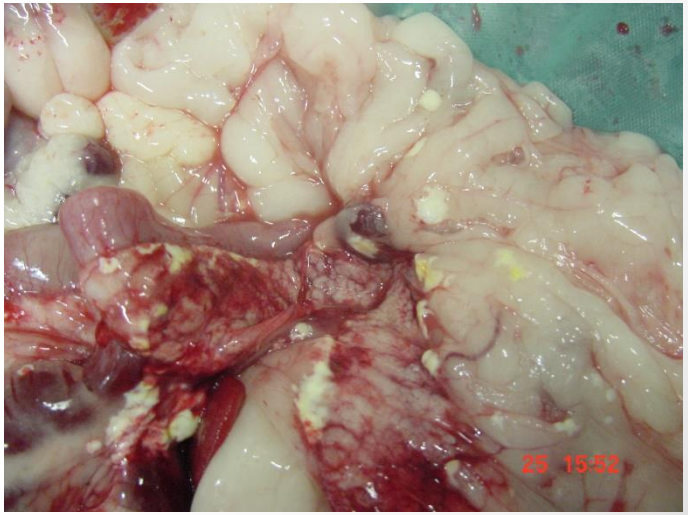


Tabella V. Principali alterazioni emato-biochimiche in gatti affetti da pancreatite acuta necrotizzante

Segno clinico-patologico	Incidenza*	
Anemia	38%	(39/103)
Emoconcentrazione	17 %	(14/32)
Leucocitosi	46%	(46/99)
Leucopenia	15%	(14/94)
Aumento AST,ALT	57%	(37/65)
Aumento ALP	49%	(32/65)
Aumento Bilirubina	58%	(38/65)
Aumento Colesterolo	72%	(28/39)
Aumento Glucosio	45%	(32/65)
Diminuzione Calcio	65%	(55/85)
Diminuzione Albuminemia	36%	(14/39)

*** in base ai dati di incidenza indicate dalle pubblicazioni[1,2,4-17] cliniche dal 1993 al 2004**

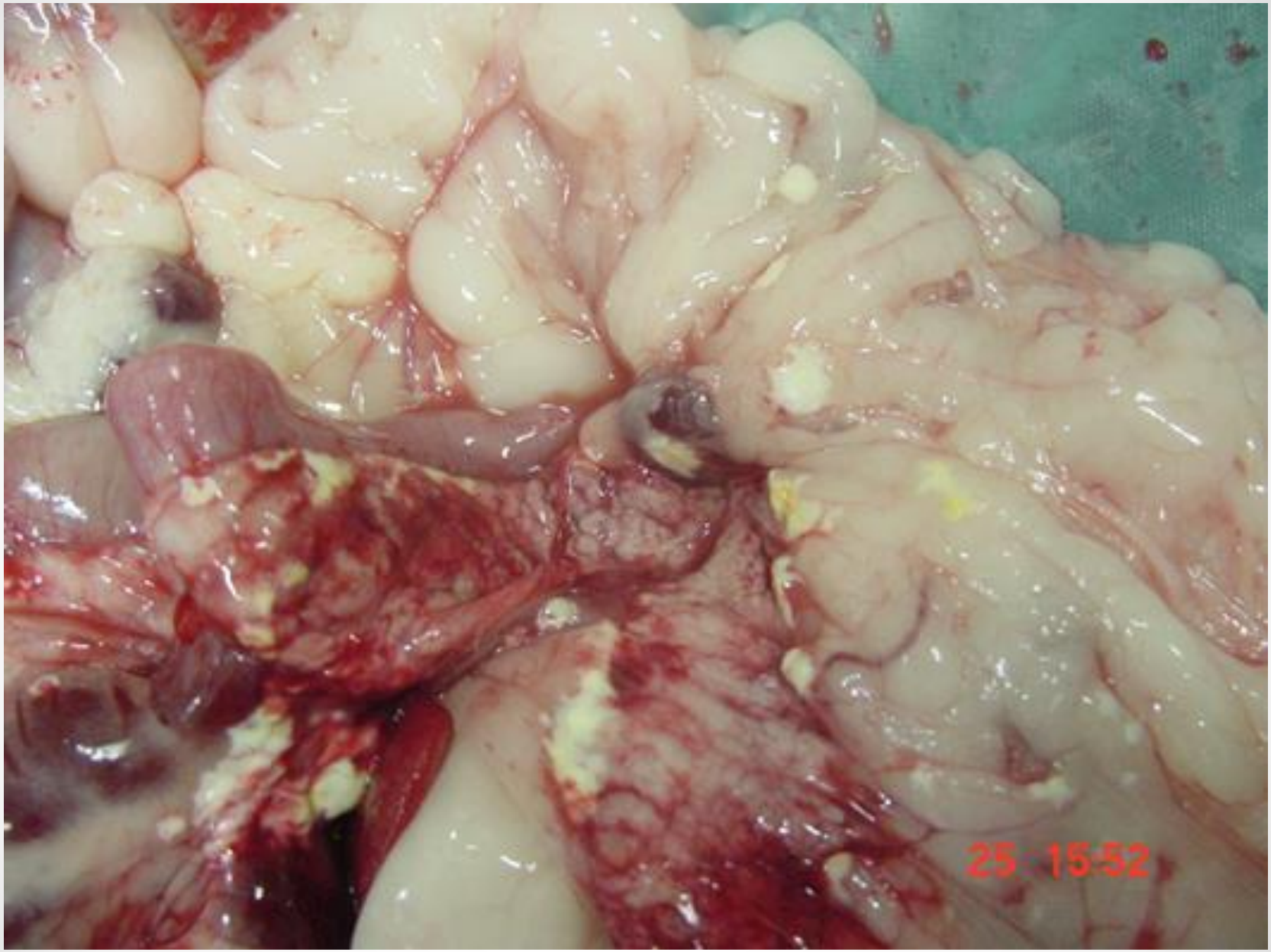

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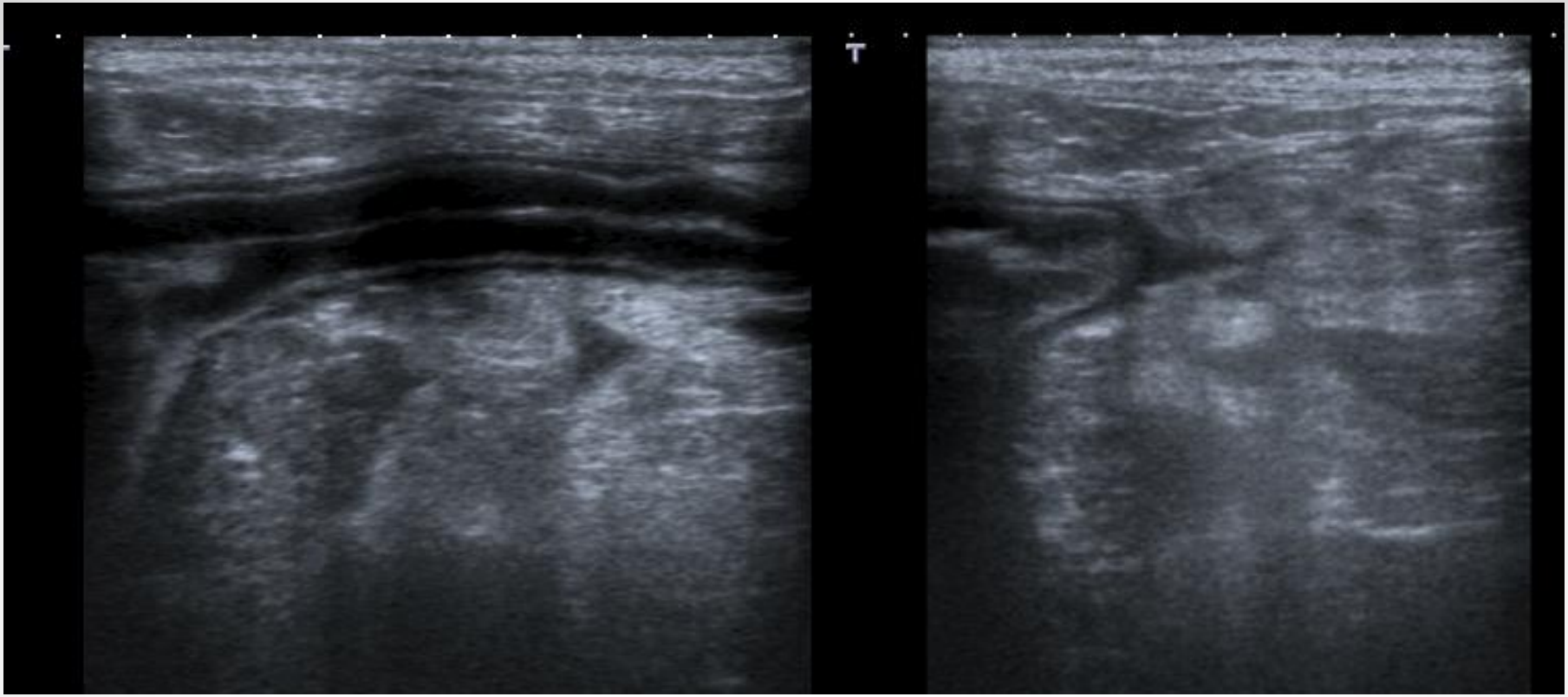
Tabella III. Sintomi riferiti in gatti affetti da pancreatite acuta necrotizzante	
Sintomo	Incidenza*
Anoressia	87% (131/150)
Letargia	81 % (129/150)
Perdita di peso	47% (75/159)
Vomito	46% (71/159)
Diarrea	12% (19/159)
*** in base ai dati di incidenza indicate dalle pubblicazioni[1,2,4-17] cliniche dal 1993 al 2004**	

Tabella IV. Segni clinici in gatti affetti da pancreatite acuta necrotizzante	
Sintomo	Incidenza*
Disidratazione	54% (50/92)
Ipotermia	46 % (23/54)
Ittero	37% (51/138)
Febbre	25% (15/62)
Dolore addominale	19% (30/159)
“Massa” addominale	11% (12/159)
*** in base ai dati di incidenza indicate dalle pubblicazioni[1,2, 4-17] cliniche dal 1993 al 2004**	

diagnosi

- Segni clinici ?
- Diagnostica per immagini
- Ecografia
- Radiologia
- TAC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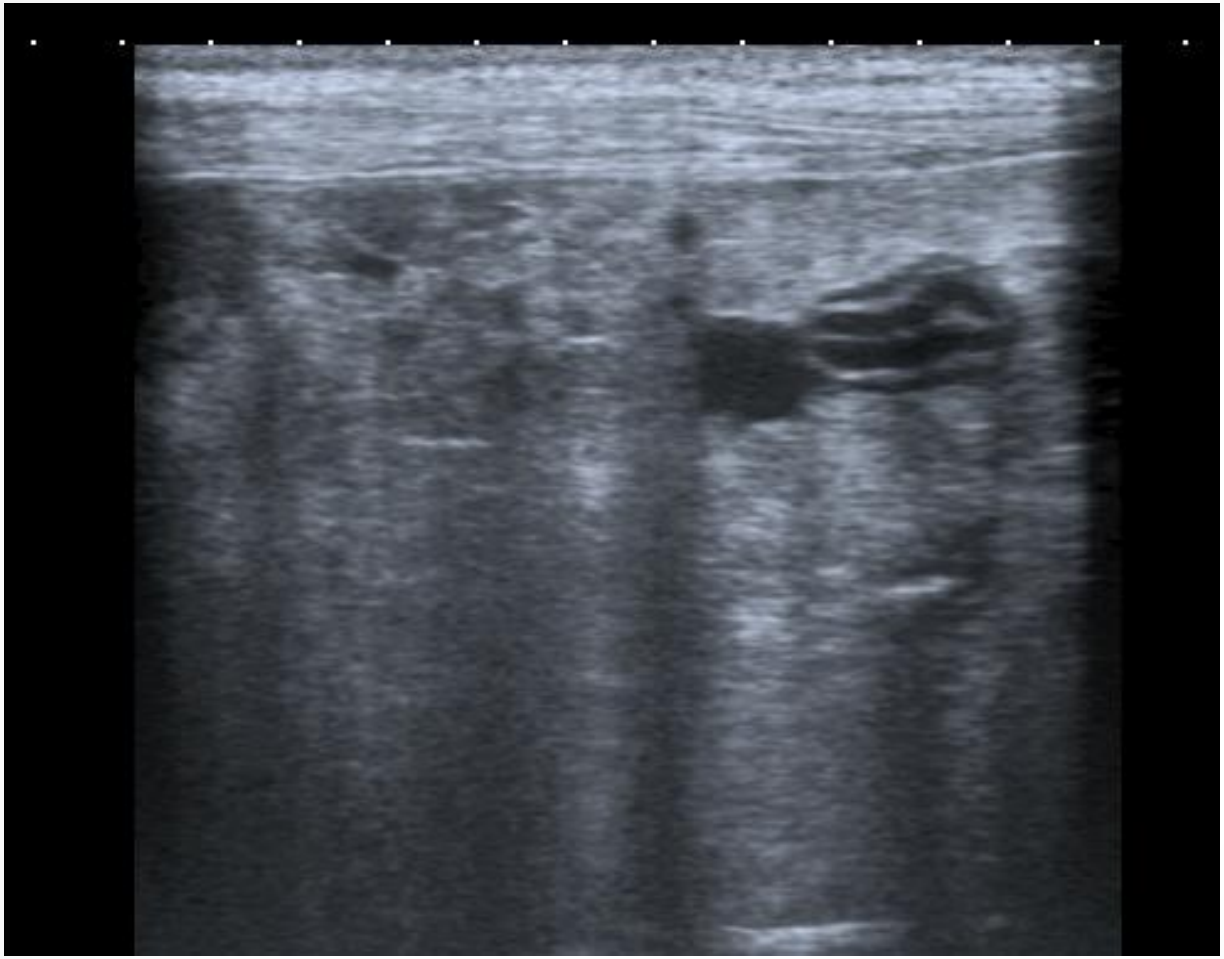

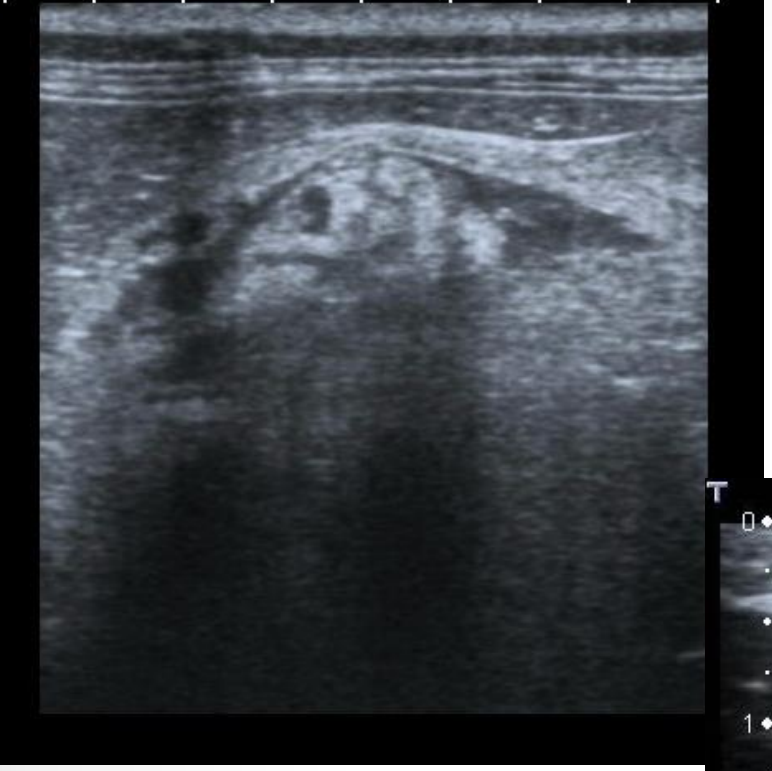

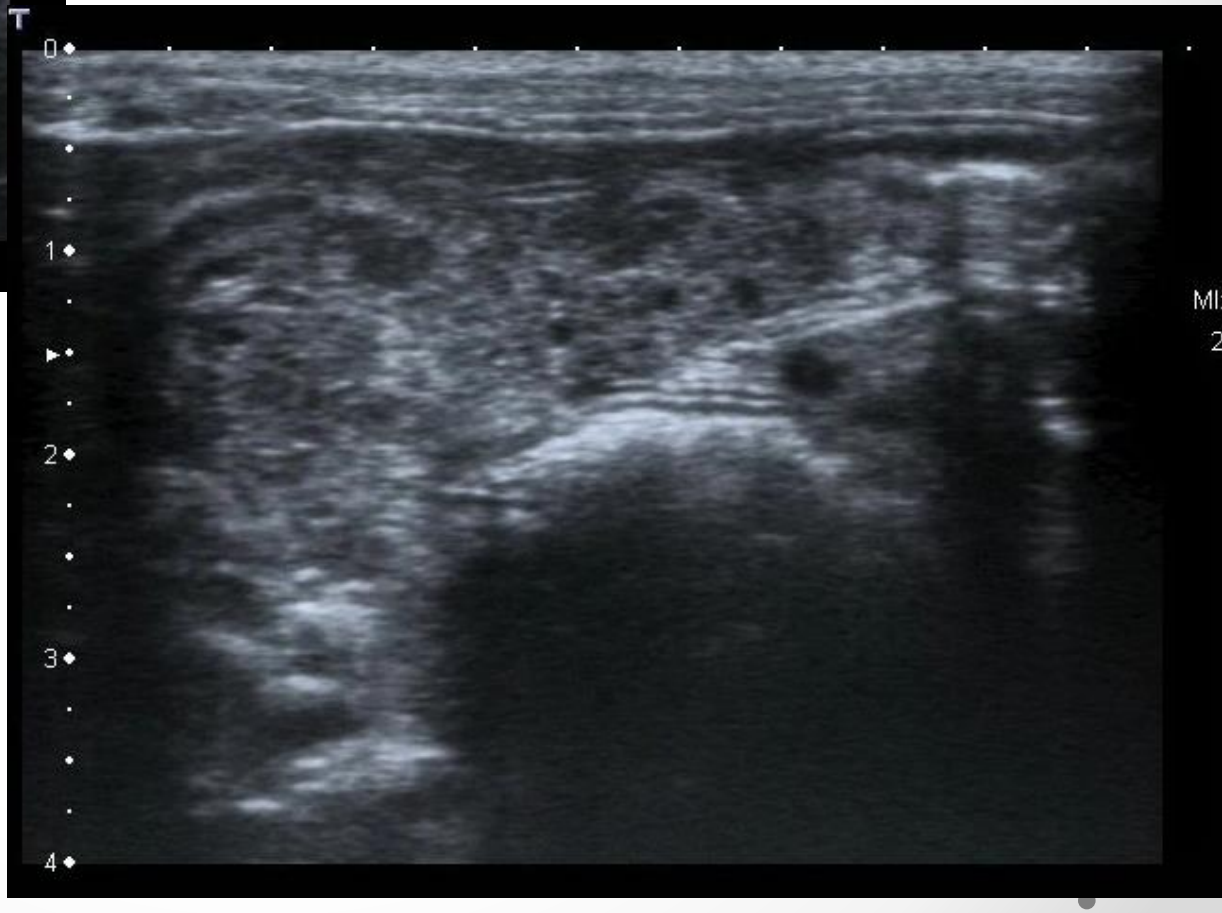
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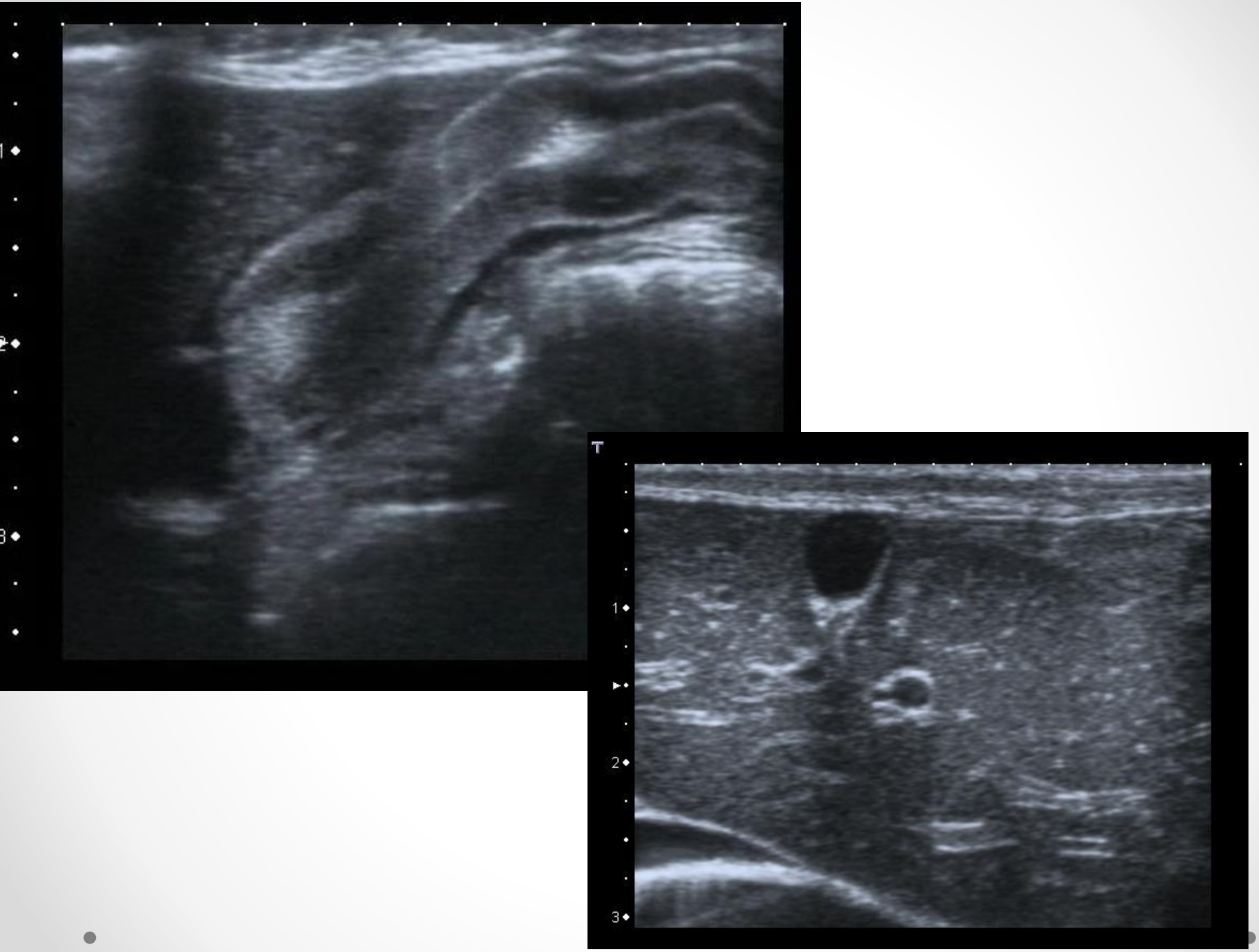
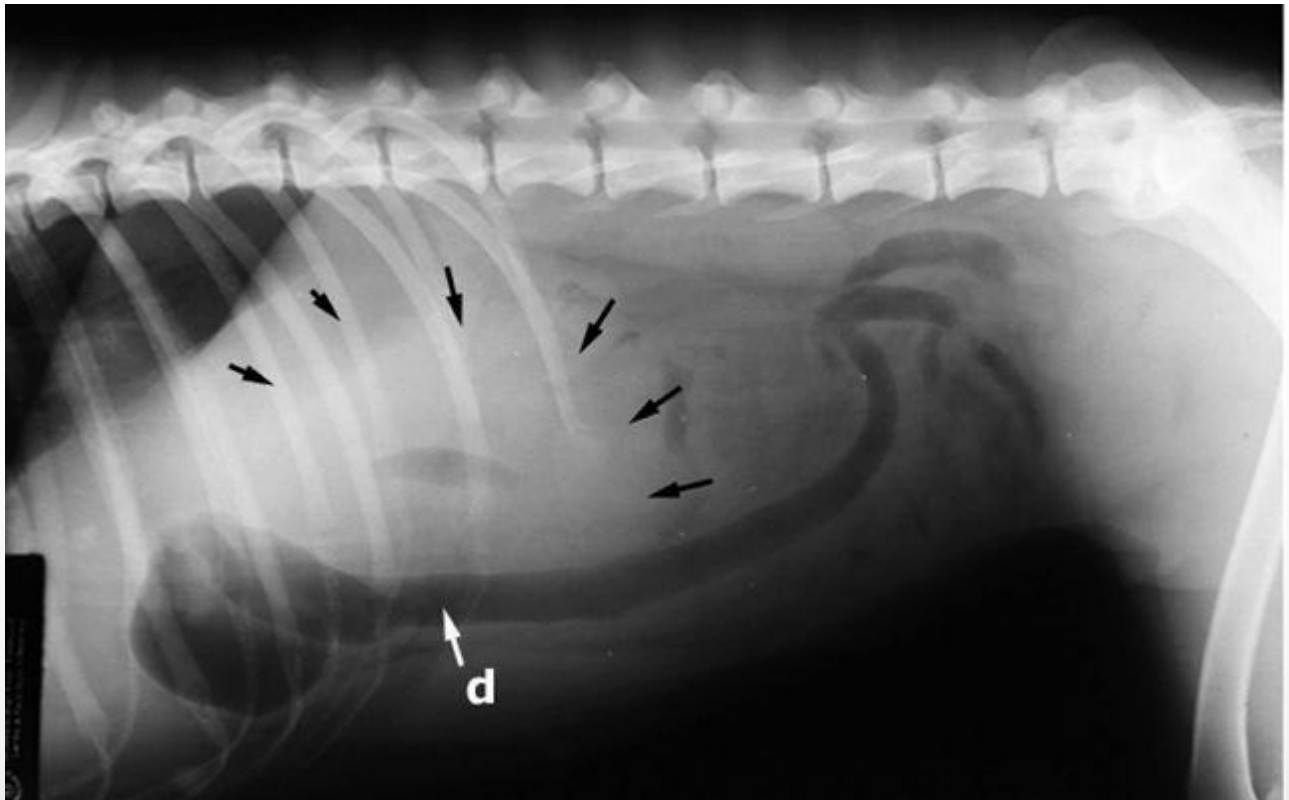


Aumento dell’opacità del tessuto soffice nel settore craniale destro dell’addome
Effetto massa caudale allo stomaco
Dilatazione duodeno discendente (gas)
Diminuzione del dettaglio – craniale destro



markers

- cTLI - fTLI
- cPLI
- fPLI
- Spec cPL
- SNAP cPL

Morbimortality Indicators in Severe Acute Pancreatitis

Tercio De Campos, Cinara Cerqueira, Laíse Kuryura, José Gustavo Parreira, Silvia Soldá, Jacqueline AG Perlingeiro, José Cesar Assef, Samir Rasslan

JOP. J Pancreas (Online) 2008; 9(6):690-697.

Table 2. Comparison of patients with severe acute pancreatitis (APACHE II score greater than 8) who survived *versus* those who died.

Variables	Did not survive (No. 3)	Survived (No. 36)	P value
Age (years)	57.3±20.0	63.4±14.2	0.490 [a]
Male gender	2 (66.7%)	22 (61.1%)	1.000 [b]
Alcoholic etiology	1 (33.3%)	12 (33.3%)	1.000 [b]
APACHE II score	12.7±1.5	11.5±3.2	0.528 [a]
SOFA score	5.0±1.0	3.0±2.0	0.098 [a]
SOFA score > 3	3 (100%)	9 (25.0%)	0.024 [b]
Marshall score	2.3±1.5	1.5±1.9	0.483 [a]
CRP (mg/dL)	3.6±2.8	11.6±9.1	0.142 [a]
Hematocrit (%)	33.1±3.8	37.4±6.7	0.284 [a]
Leukocytes (mm^{-3})	27,200±12,827	13,559±4,761	<0.001 [a]
Leukocytes > 19,000 mm^{-3}	2 (66.7%)	3 (8.3%)	0.038 [b]
Necrosis	1 (33.3%)	10 (27.8%)	1.000 [b]
Necrosis > 50%	1 (33.3%)	1 (2.8%)	0.150 [b]
Balthazar D/E	1 (33.3%)	9 (25.0%)	1.000 [b]
Length of stay (days)	3.0±2.0	10.6±7.8	0.105 [a]

Mean±SD values or frequencies are reported.
[a] Student's t test
[b] Fisher's exact test

- APACHE: Acute Phisiologic and Chronic Heltah Evaluation
- SOFA : Sequential Organ Failure Assessment

Severity Score Index

(Ruaux C, Atwell AT *Aust J Vet*, 1998)

TABLE 1: Criteria for organ systems compromise in a severity score for spontaneous canine acute pancreatitis and expected mortality rates of each score level for clinical cases under general practice conditions.

Organ System	Criteria for Compromise or Failure	Laboratory Reference Range
Hepatic	Any of Alkaline Phosphatase (ALP), Aspartate aminotransferase (AST) or Alanine aminotransferase (ALT) > 3x upper reference range.	ALP 0–140 IU/L AST 15–80 IU/L ALT 15–80 IU/L
Renal	Blood Urea (BUN) > 14 mmol/l or Creatinine > 0.3 mmol/l	BUN 2.5–9.5 mmol/L Creat. 0.06–0.18 mmol/L
Lymphoid	>10% Band neutrophils or white cell count (WCC) > 24 × 10^9/L	0.0–0.2 × 10^9/L Band neutrophils 4.5–17.0 × 10^9/L WCC
Endocrine Pancreas*	Blood glucose > 13 mmol/L and/or β-OH butyrate > 1 mmol/L	3.3–6.8 mmol/L glucose 0.0–0.6 mmol/L β-OH butyrate
Acid/Base Buffering*	Bicarbonate <13 or >26 mmol/L and/or Anion Gap <15 or >38 mmol/L	15–24 mmol/L Bicarbonate 17–35 mmol/L Anion Gap

SCORE	0	1	2	3	4
Expected Mortality (%)	0	11	20	66	100

* When hyperglycaemia and ketoacidosis co-exist, counted as one system.

CLINICAL SEVERITY INDEX

Development of a clinical severity index for dogs with acute pancreatitis

Caroline S. Mansfield, BSc, BVMS, MVM; Fleur E. James, BSc, BVMS; Ian D. Robertson, BVSc, PhD

Objective—To establish a clinical severity index that correlates severity of body system abnormalities with outcome in dogs with acute pancreatitis (AP) and determine the usefulness of serum C-reactive protein (C-RP) concentration as an objective measure of AP severity.

Design—Retrospective cohort study.

Animals—61 client-owned dogs with ultrasonographically or histologically confirmed AP.

Procedures—Medical records of AP-affected dogs were reviewed, and signalment, physical examination findings, clinicopathologic data, and outcome (death or discharge from the hospital) were evaluated. The correlation of specific abnormalities in endocrine, hepatic, renal, hematopoietic, cardiovascular, and respiratory systems; local pancreatic complications; and intestinal integrity were evaluated, and a clinical severity index was developed for AP in dogs. The severity index score was compared with outcome and, for 12 dogs, with serum C-RP concentration.

Results—The clinical severity index had a good correlation with outcome and interval from hospital admission until end point (days until outcome), but there was no difference in days until outcome between survivors and nonsurvivors. All 12 dogs evaluated had high serum C-RP concentration, but this variable was not related to outcome; however, within a 2-day period after onset of clinical signs, serum C-RP concentration in survivors and nonsurvivors differed significantly.

Conclusions and Clinical Relevance—Among AP-affected dogs, the clinical severity index may be useful for treatment comparisons and prediction of intensive management requirements. Serum C-RP concentration was best related to AP severity within a 2-day period after onset of clinical signs, but daily measurement may be more useful for monitoring progress. (*J Am Vet Med Assoc* 2008;233:936–944)

System	Finding	Point allocation
Endocrine	No abnormalities	0
	Preexisting diabetes mellitus	1
	Diabetic ketoacidosis	2
Hepatic	No abnormalities	0
	≥ 2.5-fold increase (compared with upper limit of reference range) in at least 2 of the following: serum alkaline phosphatase, alanine transferase, and aspartate aminotransferase activities	1
	≥ 5-fold increase (compared with upper limit of reference range) in at least 2 of the following: serum alkaline phosphatase, alanine transferase, and aspartate aminotransferase activities	2
	Extrahepatic bile duct obstruction	3
Renal	No abnormalities	0
	Azotemia (≤ 1.5-fold increase [compared with upper limit of reference range] in serum urea and creatinine concentration)	1
	Anuria or azotemia (≥ 1.5-fold increase [compared with upper limit of reference range] in serum urea and creatinine concentration)	2
Hematopoietic	No abnormalities	0
	WBC count ≥ 20.0 × 10^3 cells/L or ≤ 4.0 × 10^3 cells/L, with ≤ 10% band neutrophils	1
	WBC count ≥ 20.0 × 10^3 cells/L or ≤ 4.0 × 10^3 cells/L, neutrophil count ≤ 1.0 × 10^3 cells/L, or ≥ 10% band neutrophils	2
	Clinicopathologic evidence of hypercoagulability or coagulation abnormalities	3
	Clinical evidence of disseminated intravascular coagulation or bleeding diathesis	4
Local complications	No abnormalities	0
	Peritonitis extending beyond peripancreatic area	1
	Pseudocyst or other acute fluid accumulation	2
	Pancreatic abscess	3
Cardiac	No abnormalities	0
	< 60 ventricular premature complexes/24-hour period or heart rate > 180 beats/min	1
	Paroxysmal or sustained ventricular tachycardia	2
Respiratory	No abnormalities	0
	Clinical evidence of dyspnea or tachypnea (> 40 breaths/min)	1
	Clinical evidence of pneumonia or acute respiratory distress syndrome	2
Intestinal integrity	No abnormalities	0
	Intestinal sounds not detected during > 3 auscultations in 24-hour period	1
	Hematochezia, melena, or regurgitation	2
	No enteral food intake for > 3 days	3
	No enteral food intake for > 3 days and at least 2 of the following: hematochezia, melena, and regurgitation	4
Vascular forces	No abnormalities	0
	Systolic arterial blood pressure < 60 or > 180 mm Hg or serum albumin concentration < 18 g/L	1
	Systolic arterial blood pressure < 60 or > 180 mm Hg and serum albumin concentration < 18 g/L	2

Table3—Data obtained from 6 dogs with AP for which the scores derived by use of the final clinical severity index and organ scoring scheme were poorly correlated.

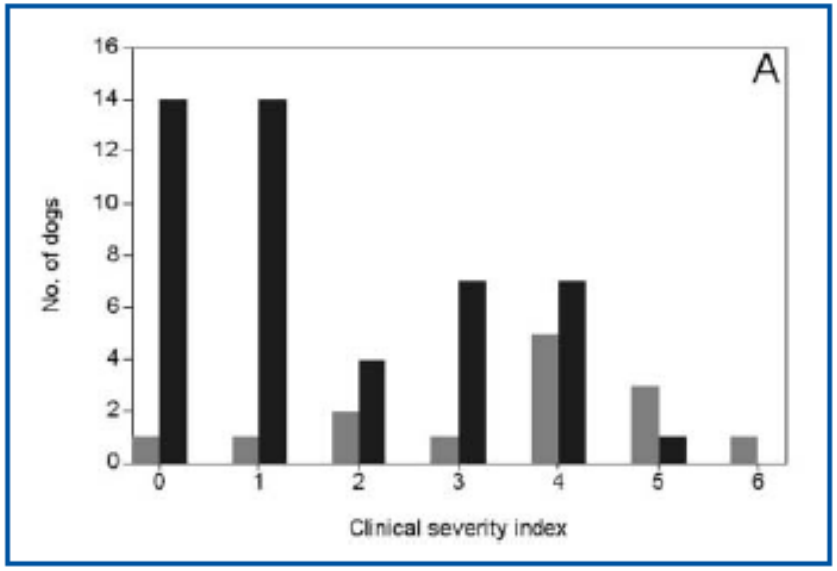
	Dog					
Variable	**1**	**2**	**3**	**4**	**5**	**6**
Signalment						
Breed	Australian Cattle Dog	Border Collie	Corgi	Australian Cattle Dog	Border Collie	Tibetan Terrier
Sex (reproductive status)	Male (N)	Female (S)	Male (N)	Male (SI)	Female (N)	Female (N)
Age (y)	9	7	12	10	6	13
Clinical severity index point allocations						
Cardiac	1	0	1	0	1	1
Respiratory	0	0	1	0	1	0
Intestinal integrity	3	3	3	4	2	3
Vascular forces	1	2	1	0	1	0
Total clinical severity index score	5	5	6	4	5	4
Laboratory organ score	1	1	0	0	2	1
Serum C-RP concentration (mg/L)	48.8	ND	ND	ND	ND	ND
Outcome	ERD	ERD	Died	Recovered	Died	ERD
Days until outcome from initial onset of signs (days until outcome from admission to hospital)	10 (6)	6 (5)	10 (8)	12 (10)	3 (1)	5 (3)
Potential underlying cause of AP	Postsurgery complication	Suspected dietary indiscretion	Unknown	Unknown	Unknown	Unknown

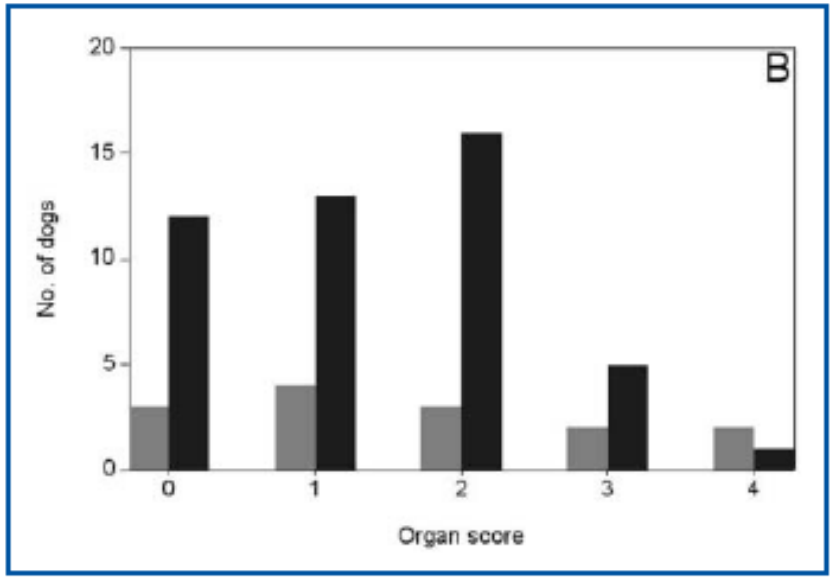
N = Neutered. S = Spayed. SI = Sexually intact. ND = Not done. ERD = Euthanatized as a result of disease.

System	Point allocation	No. of dogs (%)	No. of survivors (No. of nonsurvivors)
Endocrine	0	49 (80.3)	39 (10)
	1	4 (6.6)	3 (1)
	2	8 (13.1)	5 (3)
Hepatic	0	22 (36.3)	19 (3)
	1	14 (22.9)	10 (4)
	2	9 (14.8)	6 (3)
	3	16 (26.2)	12 (4)
Renal	0	51 (83.6)	39 (12)
	1	7 (11.5)	6 (1)
	2	3 (4.9)	1 (3)
Hematopoietic	0	17 (27.9)	15 (2)
	1	19 (31.1)	14 (5)
	2	18 (29.5)	14 (4)
	3	5 (8.2)	4 (1)
	4	2 (3.3)	1 (1)
Local complications	0	19 (31.1)	14 (5)
	1	36 (59)	29 (7)
	2	5 (8.2)	4 (1)
	3	1 (1.7)	0 (1)
Cardiac*	0	47 (77)	38 (9)
	1	14 (23)	9 (5)
	2	0	NA
Respiratory*	0	57 (93.4)	45 (12)
	1	4 (6.6)	2 (2)
	2	0	NA
Intestinal integrity*	0	22 (36.1)	21 (1)
	1	13 (21.3)	9 (4)
	2	5 (8.2)	3 (2)
	3	20 (32.7)	13 (7)
	4	1 (1.7)	1 (0)
Vascular forces*	0	45 (73.7)	41 (6)
	1	15 (24.6)	8 (7)
	2	1 (1.7)	0 (1)

*Assessed system factors were significantly ($P < 0.05$) associated with outcome.

NA = Not applicable.

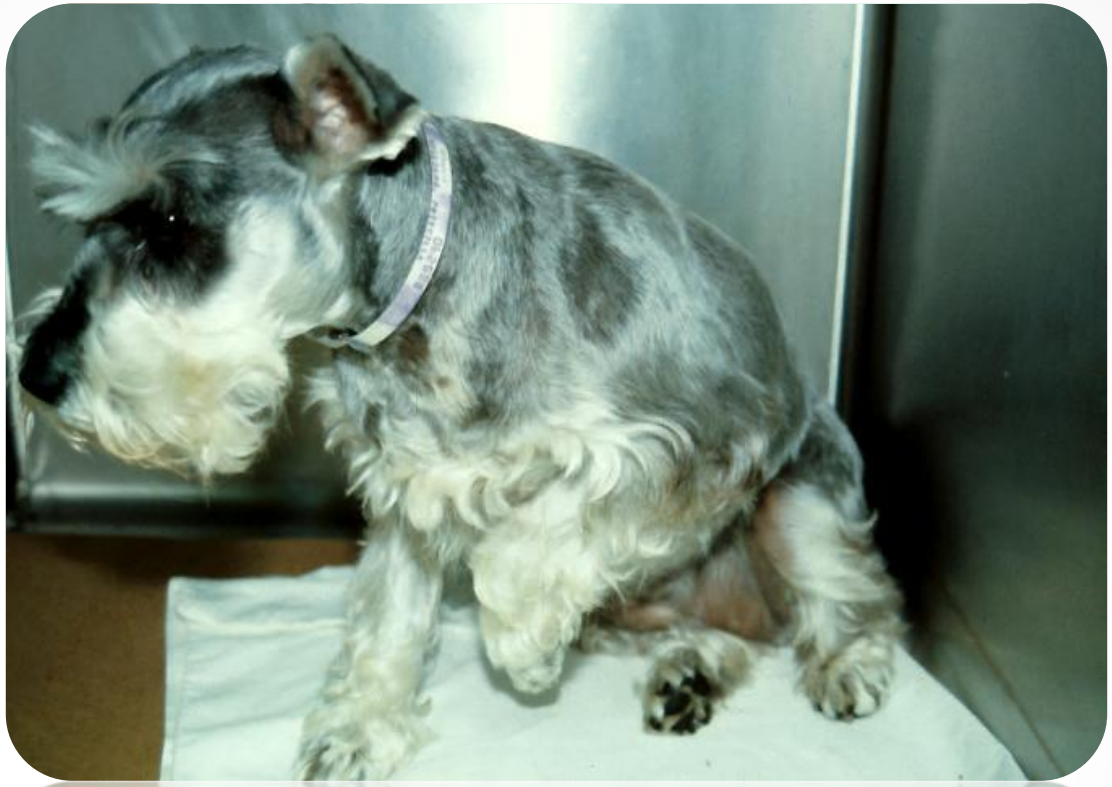

A
No. of dogs
16
14
12
10
8
6
4
2
0
0
1
2
3
4
5
6
Clinical severity index


B
No. of dogs
20
15
10
5
0
0
1
2
3
4
Organ score

Markers di Gravità

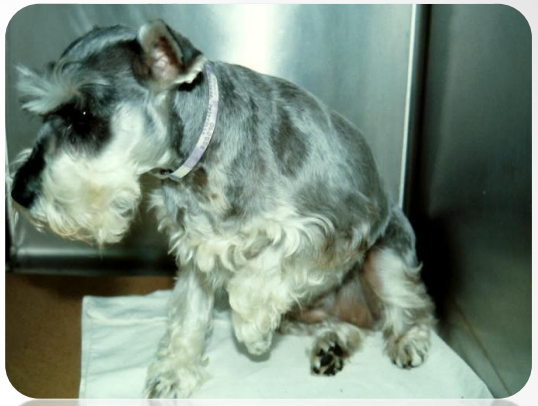
- Severity Score Index
- C-RP , monitoraggio sequenziale *(Chan D, JVIM 2006, abstr; Spillmann et al , 2002, abstr)*
- SIRS (alterazioni WBC, tachicardia, tachipnea)
- CID, complicazioni tromboemboliche (?)
- FUNZIONALITA' INTESTINALE

(ritardo nell'alimentazione enterale)



Susy, Schnauzer, 7 aa

8 Kg



- Anoressia, vomito, letargia
  da 2 giorni

- Da 2 anni in terapia per DM

- Febbre (40.7°C), grave depressione, disidratazione 10%, tachipnea, dispnea, 180 btt/min,

PCV = 47.8% (35-55)

WBC = 24.200/ul (6-14.000)

Segm = 21.400/ul (4-12.000)

Banda = 2900 (< 500)

Nessuna alterazione della Coagulazione

Creatinina = 1.3 mg/dl (< 2.0)

Glicemia= 450 mg/dl (70-105)

Calcio = 8.8 mg/dl (9.3-11.8)

Sodio = 131 mEq/L (138-148)

Potassio = 3.9 mEq/L (3.5-5.0)

TCO_2= 15.0 mmol/L (21-28)

Albumina = 2.0 g/dl (2.4-3.6)

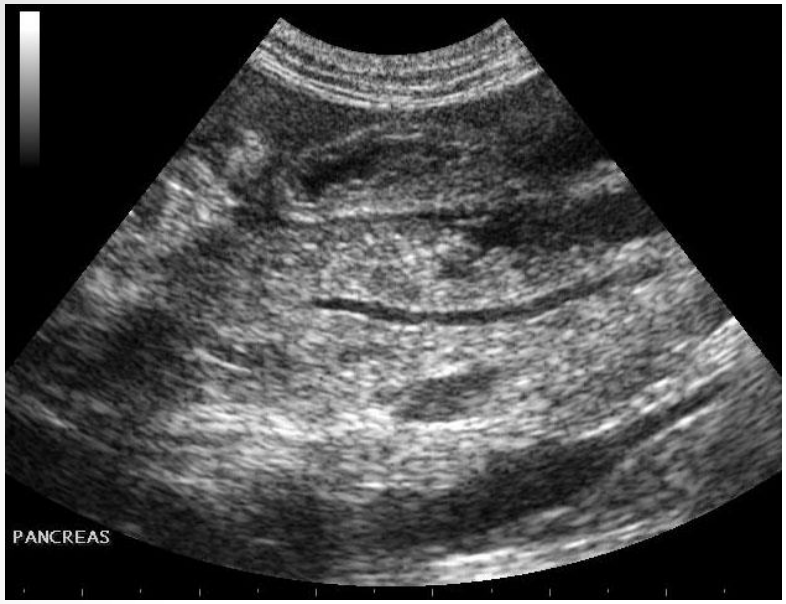
ALT = 304 IU/L (< 130)

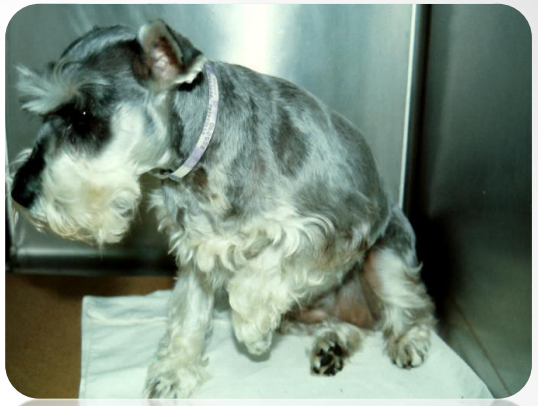
SAP = 1.368 IU/L (< 147)

Lipasi = 1.380 IU/L (< 1.750)

Amilasi = 450 IU (< 1.036)

Lipemia = 4+



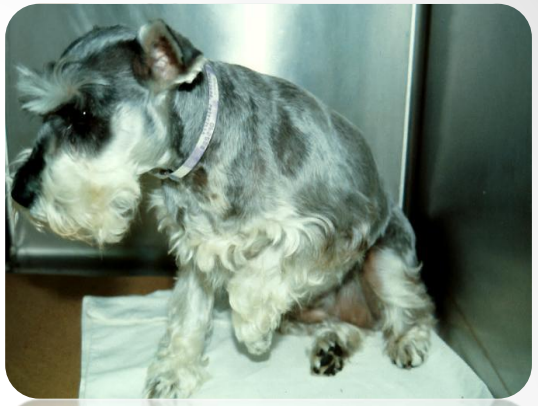


- Pancreatite acuta
- Peritonite focale peripancreatica

- Ketonuria +++
- Ketonemia (6 mmol/dL 0,0 – 0,3 mmol/dL)
- cPLI >1000 mcg/L

System	Finding	Point allocation
Endocrine	No abnormalities	0
	Preexisting diabetes mellitus	1
	Diabetic ketoacidosis	2
Hepatic	No abnormalities	0
	≥ 2.5-fold increase (compared with upper limit of reference range) in at least 2 of the following: serum alkaline phosphatase, alanine transferase, and aspartate aminotransferase activities	1
	≥ 5-fold increase (compared with upper limit of reference range) in at least 2 of the following: serum alkaline phosphatase, alanine transferase, and aspartate aminotransferase activities	2
	Extrahepatic bile duct obstruction	3
Renal	No abnormalities	0
	Azotemia (≤ 1.5-fold increase [compared with upper limit of reference range] in serum urea and creatinine concentration)	1
	Anuria or azotemia (≥ 1.5-fold increase [compared with upper limit of reference range] in serum urea and creatinine concentration)	2
Hematopoietic	No abnormalities	0
	WBC count ≥ 20.0 × 10^3 cells/L or ≤ 4.0 × 10^3 cells/L, with ≤ 10% band neutrophils	1
	WBC count ≥ 20.0 × 10^3 cells/L or ≤ 4.0 × 10^3 cells/L, neutrophil count ≤ 1.0 × 10^3 cells/L, or ≥ 10% band neutrophils	2
	Clinicopathologic evidence of hypercoagulability or coagulation abnormalities	3
	Clinical evidence of disseminated intravascular coagulation or bleeding diathesis	4
Local complications	No abnormalities	0
	Peritonitis extending beyond peripancreatic area	1
	Pseudocyst or other acute fluid accumulation	2
	Pancreatic abscess	3
Cardiac	No abnormalities	0
	< 60 ventricular premature complexes/24-hour period or heart rate > 180 beats/min	1
	Paroxysmal or sustained ventricular tachycardia	2
Respiratory	No abnormalities	0
	Clinical evidence of dyspnea or tachypnea (> 40 breaths/min)	1
	Clinical evidence of pneumonia or acute respiratory distress syndrome	2
Intestinal integrity	No abnormalities	0
	Intestinal sounds not detected during > 3 auscultations in 24-hour period	1
	Hematochezia, melena, or regurgitation	2
	No enteral food intake for > 3 days	3
	No enteral food intake for > 3 days and at least 2 of the following: hematochezia, melena, and regurgitation	4
Vascular forces	No abnormalities	0
	Systolic arterial blood pressure < 60 or > 180 mm Hg or serum albumin concentration < 18 g/L	1
	Systolic arterial blood pressure < 60 or > 180 mm Hg and serum albumin concentration < 18 g/L	2

Susy



- Chetoacidosi diabetica
- Pancreatite Acuta Grave
- Clinical Severity Index: 8

Trattamento della Pancreatite

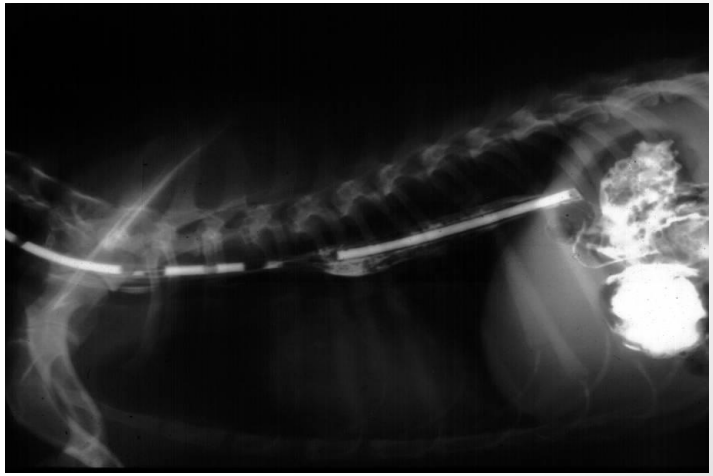
- *Rimozione della cause scatenanti*
- ***Ripristino e mantenimento del volume intravascolare e della perfusione pancreatica***
- ***Controllo del dolore***
- ***Adeguato supporto nutrizionale***
- ***Controllo delle complicazioni***
- ***Riduzione delle secrezioni pancreatiche***

“*Feed early and enterally* ”

(Nathens et al. *Crit Care Med* 2004; 32 2524-36; Heinrich et al. *Ann Surg* 2006; 243: 154-168; Kingsnorth et al. *Br Med J* 2006; 332: 1072-1076; Highes et al. *Anaest Intensive Care* 2010; 38: 167-74)

- Digiuno/ ritardo nell’alimentazione enterale:
  a) malnutrizione
  b) alterazione della barriera intestinale
  c) translocazione batterica
  d) diminuita sopravvivenza

(Mansfield,2008)



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World J Gastroenterol 2007 February 21; 13(7): 1123-1128
World Journal of Gastroenterology ISSN 1007-9327

RAPID COMMUNICATION

Effect of parenteral and early intrajejunal nutrition on pancreatic digestive enzyme synthesis, storage and discharge in dog models of acute pancreatitis

Huan-Long Qin, Zhen-Dong Su, Lei-Guang Hu, Zai-Xian Ding, Qing-Tian Lin

CONCLUSION: Early intrajejunal nutrition might be effective in dogs with acute pancreatitis.

J Vet Intern Med 2003;17:791–798

Effect of Early Enteral Nutrition on Intestinal Permeability, Intestinal Protein Loss, and Outcome in Dogs with Severe Parvoviral Enteritis

Albert J. Mohr, Andrew L. Leisewitz, Linda S. Jacobson, Jörg M. Steiner, Craig G. Ruaux, and David A. Williams

PI between groups. Thirteen NPO dogs and all EEN dogs survived ($P = .48$). The EEN group showed earlier clinical improvement and significant weight gain. The significantly decreased %L in the EEN versus NPO group might reflect improved gut barrier function, which could limit bacterial or endotoxin translocation.

J Vet Intern Med 2009;23:559–563

Relationship among Plasma Amino Acids, C-Reactive Protein, Illness Severity, and Outcome in Critically Ill Dogs

D.L. Chan, E.A. Rozanski, and L.M. Freeman

Background: Alterations in circulating amino acids have been documented in animal models and in critically ill people but have not been evaluated in dogs with spontaneously occurring disease.

Hypothesis/Objectives: To compare amino acid concentrations in critically ill dogs and healthy controls and to investigate potential relationships among amino acids, markers of inflammation, illness severity, and clinical outcome.

Animals: Forty-eight critically ill dogs and 24 healthy control dogs.

Methods: Plasma was analyzed for amino acids and C-reactive protein (CRP) was measured in serum. The Fischer ratio (the molar ratio of branched chain amino acids [BCAA] to aromatic amino acids [AAA]) and survival prediction index (SPI2) were calculated.

Results: Median CRP concentrations were significantly higher in the critically ill dogs compared with controls ($P < .001$). Critically ill dogs had significantly lower concentrations of alanine ($P = .001$), arginine ($P < .001$), citrulline ($P < .001$), glycine ($P < .001$), methionine ($P < .001$), proline ($P < .001$), and serine ($P = .001$) but significantly higher concentrations of lysine ($P = .02$) and phenylalanine ($P < .001$; Table 1). This pattern resulted in a significantly lower Fischer ratio ($P = .001$) in the critically ill group. Median SPI2 score was significantly higher in dogs that survived ($P = .03$). Concentrations of arginine ($P = .02$), isoleucine ($P = .01$), leucine ($P = .04$), serine ($P = .04$), valine ($P = .04$), total BCAA ($P = .03$), and the Fischer ratio ($P = .03$) were significantly higher in survivors compared with nonsurvivors.

Conclusions and Clinical Importance: Critically ill dogs have altered amino acid profiles and additional research to investigate potential benefits of amino acid supplementation is warranted.

Key words: Critical care nutrition; Nutritional status; Response to inflammation.

$$\begin{aligned}\text{Logit } P = {} & 0.3273 + (0.0108 \times \text{mean arterial pressure}) \\ & - (0.0102 \times \text{respiratory rate}) - (0.2183 \times \text{creatinine}) \\ & + (0.0164 \times \text{packed cell volume}) + (0.3553 \times \text{albumin}) \\ & - (0.1184 \times \text{age}) \\ & - (0.8069 \times \text{medical versus surgical status})\end{aligned}$$

where medical status = 1 and surgical status = 0.

Table 1. Comparison between critically ill and healthy control dogs and among different subgroups of diseases within the critically ill group.

	Controls	All Critically Ill	P^c-Value	Sepsis[d]	Pancreatitis[d]	Trauma[d]
n	24	48	23	23	14	11
Age	5.5 (2.0–15.0)	7.3 (1.3–15.9)	.09	9.7 (1.3–13.5)	7.8 (3.2–15.9)	5.0 (2.0–11.0)
Sex			.18			
Male	8 (8 castrated)	26 (21 castrated)		12 (8 castrated)	8 (7 castrated)	6 (6 castrated)
Female	16 (14 spayed)	22 (17 spayed)		11 female (7 spayed)	6 (6 spayed)	5 (4 spayed)
Weight (kg)	26.7 (3.7–47.7)	26.4 (3.2–66.0)	.88	34.4 (3.2–66.0)	15.5 (4.5–63.6)	18.1 (5.5–41.0)
CRP (μg/mL)	3.2 (2.5–23.1)	57.7 (2.6–98.4)	< .001	55.7 (9.4–98.4)	57.7 (40.1–69.0)	71.7 (2.6–92.6)
SPI2	—	0.62 (0.23–0.91)	—	0.58 (0.36–0.88)[a]	0.63 (0.23–0.91)[a,b]	0.72 (0.54–0.83)[b]
Alanine	480.6 (259.8–702.7)	325.0 (75.6–1,635.0)	.001	365.6 (75.6–1,635.0)	268.4 (102.5–570.0)	354.0 (102.7–704.9)
Arginine	117.7 (64.8–165.9)	64.0 (0.0–251.8)	< .001	64.3 (2.0–173.4)	68.5 (0.0–251.8)	61.6 (0.0–102.0)
Aspartic acid	5.7 (2.8–10.5)	6.2 (2.2–21.0)	.39	7.2 (2.4–21.0)	6.0 (2.5–9.4)	5.0 (2.2–15.2)
Asparagine	47.4 (32.0–89.1)	49.0 (12.6–147.0)	.82	50.9 (13.8–143.5)	61.0 (14.9–147.0)	36.2 (12.6–89.0)
Citrulline	57.6 (33.7–143.2)	22.6 (0–55.0)	< .001	19.0 (0.0–50.5)	21.5 (6.6–55.0)	33.8 (19.0–47.4)
Cysteine	6.7 (2.0–24.8)	7.4 (1.0–53.2)	.52	11.0 (1.5–53.2)	7.3 (1.9–30.0)	4.5 (1.0–15.0)
Glutamic acid	46.2 (26.5–101.3)	42.7 (19.0–149.8)	.07	44.5 (19.1–81.2)[a]	49.5 (19.5–149.8)[a]	28.5 (19.0–50.7)[b]
Glutamine	706.5 (168.8–942.3)	591.9 (132.0–1,286.0)	.13	630.0 (132.0–1,286.0)	587.1 (139.8–853.1)	527.9 (376.1–940.9)
Glycine	249.5 (175.3–606.8)	160.1 (61.1–407.0)	< .001	163.9 (61.5–407.0)	159.5 (61.1–267.0)	150.6 (101.4–261.7)
Histidine	75.2 (54.7–98.0)	72.8 (33.4–162.0)	.57	71.5 (45.5–162.0)	70.9 (37.5–97.0)	79.0 (33.4–150.3)
Isoleucine	65.3 (38.3–114.9)	74.7 (14.2–213.8)	.50	67.1 (26.0–213.8)[a]	101.0 (31.4–148.4)[b]	85.7 (14.2–127.0)[a,b]
Leucine	155.1 (97.0–245.1)	176.5 (51.0–384.1)	.46	158.1 (74.3–384.1)	201.4 (51.0–367.0)	173.5 (51.6–270.2)
Lysine	163.6 (102.3–258.0)	186.9 (48.6–500.0)	.02	168.0 (78.3–396.7)	232.5 (91.0–500.0)	202.5 (48.6–394.5)
Methionine	67.5 (39.3–131.6)	44.7 (11.5–115.2)	< .001	44.0 (11.5–115.2)	57.2 (22.1–95.0)	36.7 (23.7–80.0)
Ornithine	14.7 (5.8–30.3)	17.6 (2.7–97.8)	.24	14.4 (6.4–79.0)	17.8 (2.7–97.8)	27.3 (12.7–87.8)
Phenylalanine	56.1 (46.2–92.0)	103.2 (29.0–188.8)	< .001	98.0 (62.8–188.8)	111.3 (61.3–155.0)	107.2 (29.0–147.6)
Proline	107.7 (54.2–258.5)	45.4 (10.1–217.0)	< .001	45.5 (10.1–217.0)	51.6 (11.6–154.0)	37. (13.9–134.0)
Serine	128.9 (85.0–193.9)	88.1 (32.7–206.7)	.001	89.3 (32.7–206.7)	109.3 (36.5–169.2)	62.7 (32.8–117.4)
Taurine	104.0 (47.1–193.4)	97.5 (20.6–1,374.0)	.87	102.4 (20.6–1,374.0)	67.6 (24.5–293.0)	114.8 (49.6–270.1)
Threonine	200.0 (110.7–314.8)	209.1 (66.4–626.0)	.79	211.2 (66.4–475.8)	205.4 (77.6–626.0)	222.2 (80.7–383.0)
Tryptophan	67.5 (13.7–134.4)	37.4 (2.0–111.7)	.10	35.2 (2.0–91.5)	33.8 (13.2–103.5)	45.8 (12.2–111.7)
Tyrosine	46.7 (30.9–77.5)	42.6 (17.1–123.6)	.93	41.9 (21.5–92.1)	43.1 (28.2–99.0)	50.1 (17.1–123.6)
Valine	182.0 (123.2–324.9)	215.4 (65.2–511.0)	.15	195.0 (100.3–434.9)	246.6 (93.0–511.0)	205.9 (62.2–363.4)
BCAA	407.1(260.0–684.9)	465.2 (128.0–1,032.8)	.24	425.4 (219.9–1,032.8)	538.7 (176.0–996.0)	465.1 (128.0–760.6)
Fischer ratio	3.9 (2.4–5.8)	3.1 (1.1–5.2)	.001	2.9 (1.6–4.3)	3.6 (1.6–5.2)	3.0 (1.1–4.2)

Data are presented as median (range). Amino acid concentrations are in nmol/mL.

[c]Comparison of all critically ill dogs (n = 48) to healthy controls (n = 24).

[d]Comparison of values among different disease groups of critically ill dogs (ie, sepsis versus pancreatitis versus trauma). Data within a row with a different superscript letters are significantly different.

CRP, C-reactive protein; SPI2, survival prediction index; BCAA, branched chain amino acids; AAA, aromatic amino acids.

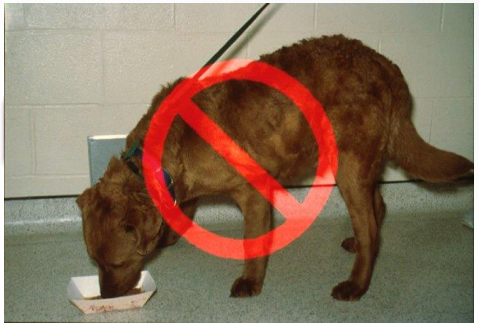
Table 2. Comparison of amino acid concentrations between critically ill dogs that survived (survivors; n = 28) and those that died or were euthanized (nonsurvivors; n = 20).

	Survivors (n = 28)	Nonsurvivors (n = 20)	*P*-Value
CRP	53.1 (2.6–98.4)	58.1 (22.3–94.3)	.29
SPI2	0.67 (0.36–0.91)	0.57 (0.23–0.89)	.03
Alanine	306.7 (75.6–1,070.0)	361.4 (80.4–1,635.0)	.43
Arginine	72.5 (0.0–251.8)	56.2 (1.6–126.8)	.02
Asparagine	50.9 (12.6–147.0)	43.4 (13.8–93.0)	.28
Aspartic acid	5.7 (2.2–14.0)	7.3 (2.4–21.0)	.18
Citrulline	22.3 (0.0–50.5)	22.6 (0.0–55.0)	.75
Cysteine	6.9 (1.0–53.2)	9.5 (1.5–29.0)	.59
Glutamic acid	41.7 (19.0–149.8)	43.2 (19.1–81.2)	.96
Glutamine	574.9 (139.8–863.5)	616.9 (132.0–1,286.0)	.66
Glycine	167.7 (61.1–268.8)	148.6 (61.5–407.0)	.29
Histidine	74.6 (33.4–118.9)	69.4 (45.5–162.0)	.82
Isoleucine	84.7 (14.2–213.8)	54.2 (26.0–127.0)	.01
Leucine	201.4 (51.6–384.1)	149.6 (51.0–270.2)	.04
Lysine	204.6 (48.6–500.0)	158.8 (78.3–394.5)	.17
Methionine	46.8 (22.1–115.2)	43.0 (11.5–95.0)	.22
Ornithine	16.0 (4.8–97.8)	19.9 (2.7–87.0)	.25
Phenylalanine	108.8 (29.0–188.8)	99.0 (69.2–150.0)	.56
Proline	45.9 (11.6–154.0)	44.8 (10.1–217.0)	.48
Serine	97.8 (32.8–206.7)	73.7 (32.7–157.4)	.04
Taurine	91.9 (24.5–289.0)	155.0 (20.6–1,374.0)	.27
Threonine	209.1 (66.4–626.0)	198.3 (94.1–336.0)	.47
Tryptophan	41.9 (12.2–111.7)	34.8 (2.0–104.2)	.31
Tyrosine	46.3 (17.1–99.0)	41.6 (21.5–123.6)	.49
Valine	237.4 (62.2–511.0)	189.4 (93.0–363.4)	.04
BCAA	522.2 (128.0–1,032.8)	406.2 (176.0–760.6)	.03
Fischer ratio	3.5 (2.0–5.2)	2.8 (1.1–4.7)	.03

Data are presented as median (range). Amino acid concentrations are in nmol/mL.

CRP, C-reactive protein; SPI2, survival prediction index; BCAA, branched chain amino acids; AAA, aromatic amino acids.

NPO (ni



- Niente acqua e cibo fino al cessare del vomito

Alimentazione per bocca

- A 12 ore dalla cessazione del vomito → CONTROLLO del VOMITO
- Se il paziente NON RIFIUTA il cibo
- In PICCOLE quantità, insieme ad acqua fresca

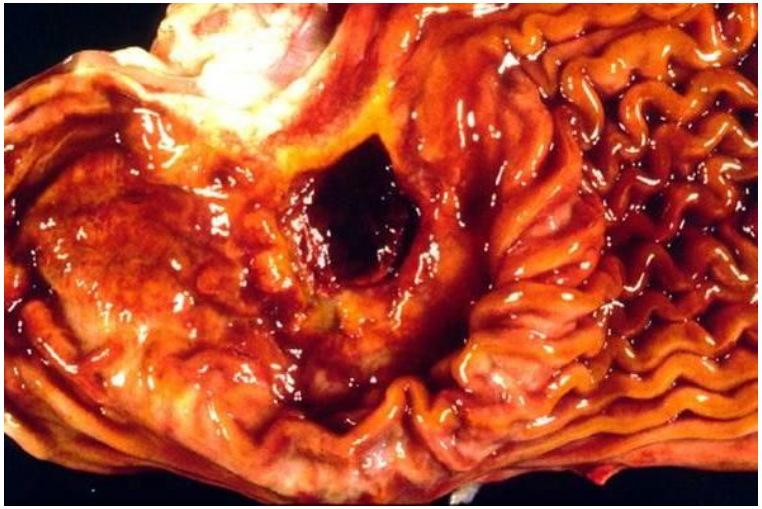
DIETA

- LOW FAT
- Iperdigeribile
- " energetica"
- Integrata (complessi vitaminici ed oligominerali, aminoacidi- arginina,citrullina, prolina, metionina, fenilalanina, glicina, prolina)

Antiemetici

- Clorpromazina:; 0.5 mg/Kg SC, IM, IV ogni 6-8 ore; 0,11- 0,44 mg/Kg IM ogni 6-8 ore
- Metoclopramide: 0,3 mg/Kg SC, IM, IV ogni 8-12 ore; 0,01-0,02 mg/Kg/ora (CRI)
- Ondansetron: 0,1-0,2 mg/Kg IV lenta, ogni 12 ore fino a 6 ore, nei gatti 0,22 mg/Kg IV ogni 12 ore
- Dolasetron: 0,3 – 0,6 IV ogni 24 ore
- Maropitant: 1 mg/Kg SC ogni 24 ore; 2 mg/ PO ogni 24 ore

Antiacidi



H_2-ANTAGONISTI

Famotidina 0,5 mg/Kg SC,IM,IV, PO ogni 12-24 ore

Ranitidina 2mg/Kg IM,SC, IV, PO ogni 8-12 ore

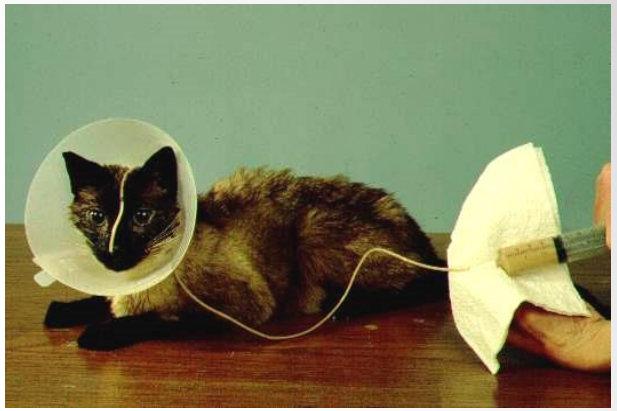
INIBITORI POMPA PROTONICA

omeprazolo 0,5 – 1 mg /Kg PO ogni 24 ore

pantoprazolo 0,7-1 mg/Kg IV ogni 24 ore

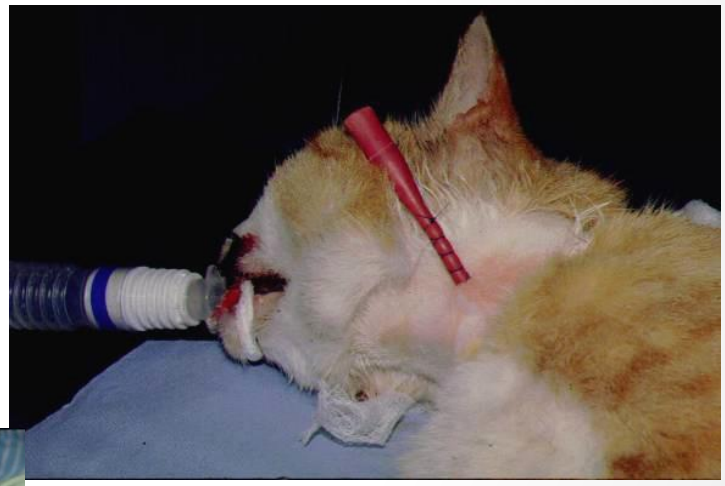
RIFIUTO del CIBO

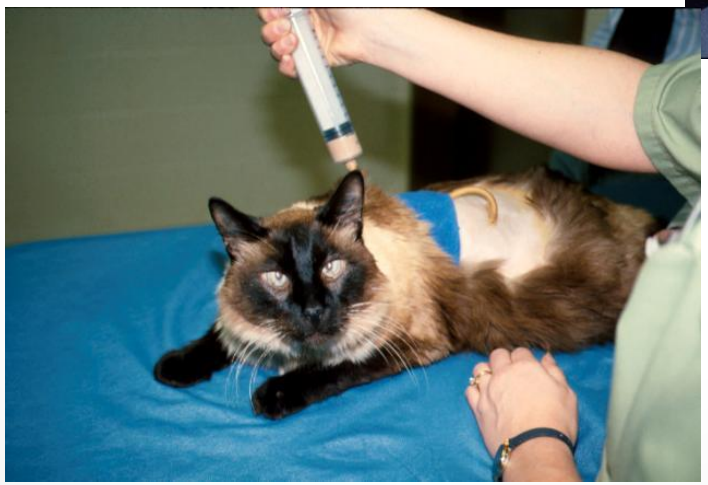
Vomito controllato



Nutrizione Enterale

- RINOEOFAGEA
- NASOGASTRICA
- ESOFAGOSTOMIA
- GASTROTOMIA (PEG-TUBE)

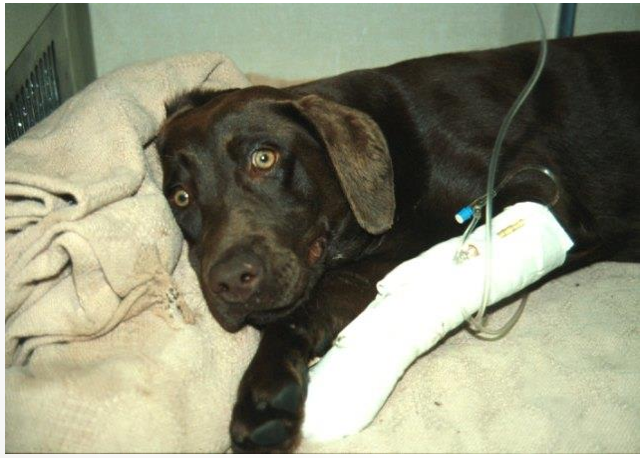




VOMITO PERSISTENTE

Nutrizione Enterale Digiunale

Nutrizione Parenterale Parziale



J Vet Intern Med 2002;16:440–445

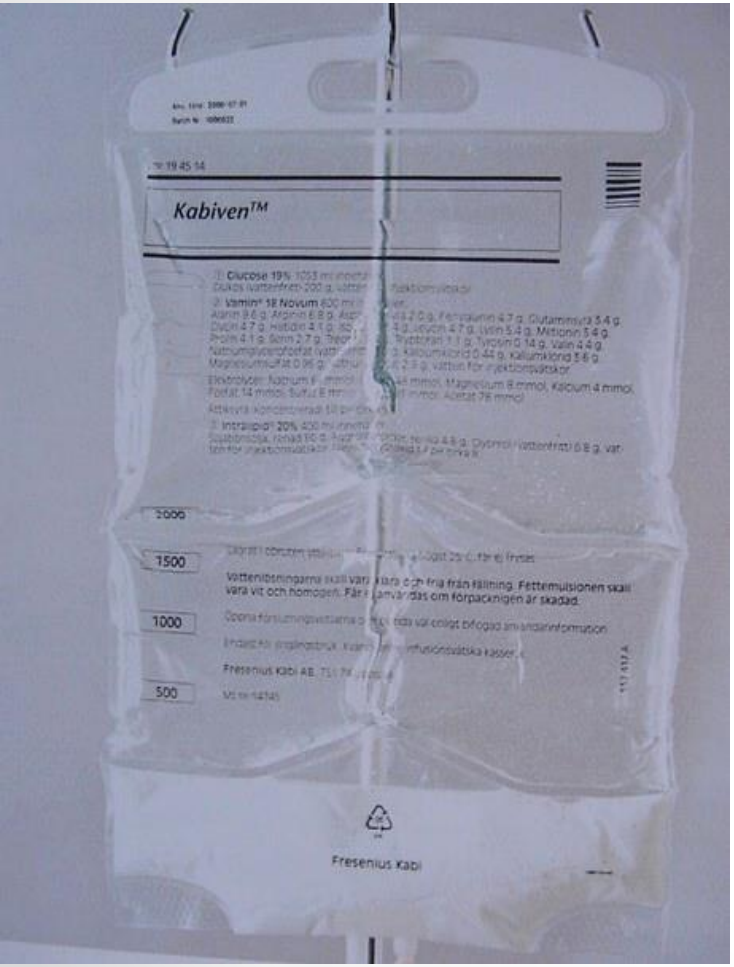
Retrospective Evaluation of Partial Parenteral Nutrition in Dogs and Cats

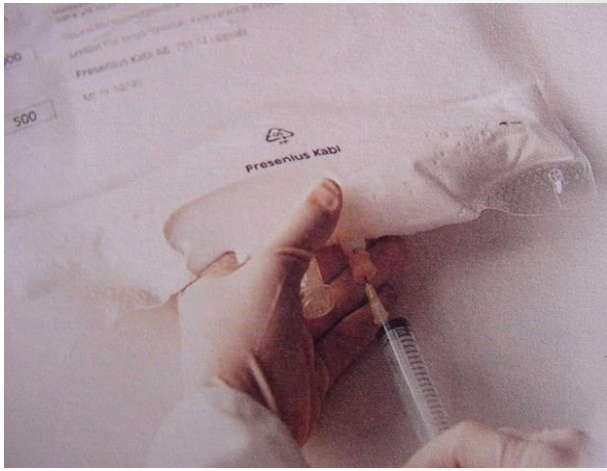
Daniel L. Chan, Lisa M. Freeman, Mary A. Labato, and John E. Rush

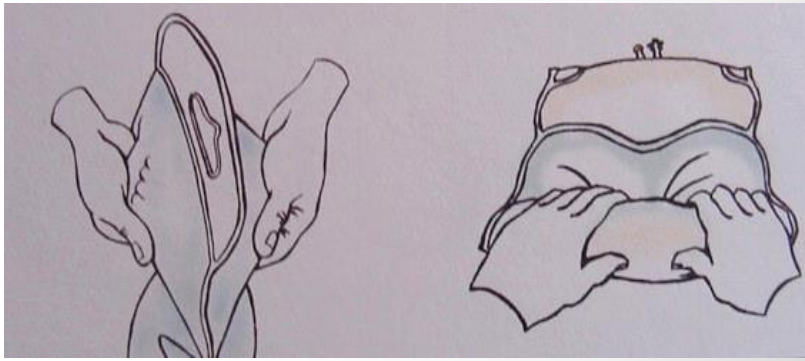
- RER (resting energy requirement)= 30 x p.v. + 70 (per 3-25 Kg)
- PER(partial energy requirement) = RER x 0.7
- PER x 0.25 (6-10 Kg) Kcal/die glucosio (0.33 cani di 11-30Kg e 0,50 >30Kg)
- PER x 0.25 (6-10 Kg) Kcal/die proteine (0.33 cani di 11-30Kg e 0,25 >30Kg)
- PER x 0.50 (6-10 Kg) Kcal/die lipidi (0.33 cani di 11-30Kg e 0,25 >30Kg)

- Glucosio 5% fornisce 0.17 Kcal/ml
- Sol. Aminoacidi 8% fornisce 0.34 Kcal/ml
- Lipidi 20% forniscono 20 Kcal/ml

Sacca a 3 scomparti per nutrizione parenterale

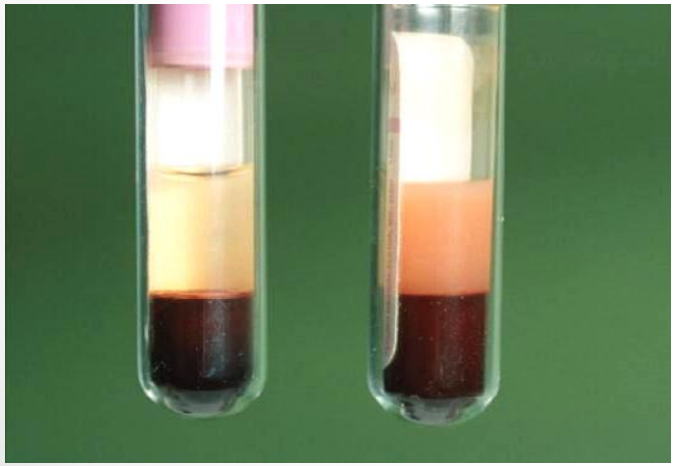






PPN

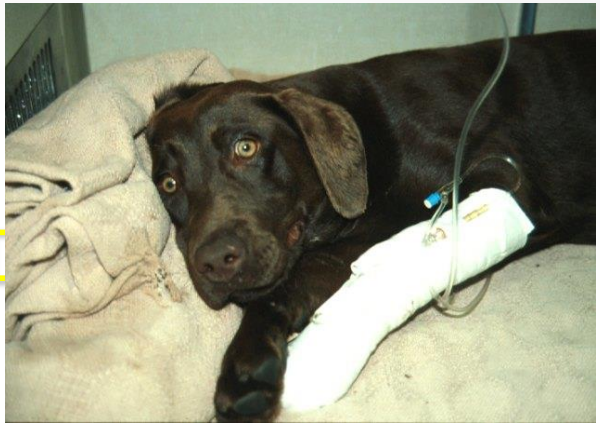
- MONITORARE IL PAZIENTE PER IPERLIPEMIA O IPERGLICEMIA
- MEGLIO SE ASSOCIATO A NUTRIZIONE ENTERALE
- DURATA MASSIMA 3-5 GIORNI



Nutrizione attraverso sonde

- Iniziare lentamente
  - **½ RER giorno 1**
  - **¾ RER giorno 2**
  - **RER giorno 3**

- Ideale 3-6 piccoli pasti al giorno



- Metadone (agonista oppioide) 0,1-0,25 mg/Kg IM,SC,IV ogni 4-6 ore
- Fentanyl (oppioide) 1- 4 mcg/Kg/ ora (CRI)
- Fentanyl Patch

Cani-gatti < 5 Kg 25-12,25 mcg/ora → 2,5-1,5 mg

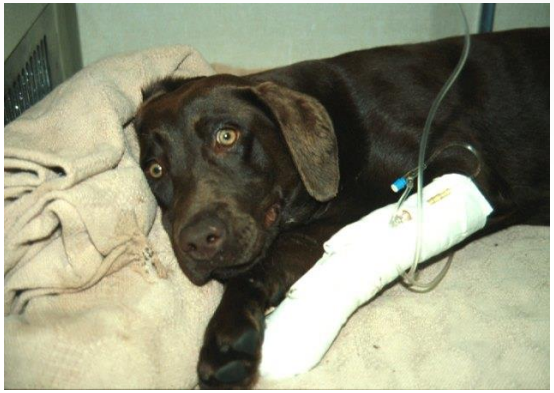
Cani-gatti 5-10 Kg 25 mcg/ora → 2,5 mg

Cani-gatti 10-20 Kg 50 mcg/ora → 5 mg

Cani-gatti 20-30Kg 75 mcg/ora → 7,5 mg

Cani-gatti > 30 Kg 100 mcg/ora → 10 mg

NON efficaci fino a 12-24 ore



- *Bupremorfina* (agonista oppioide parziale, mu) 0,01-0,015 mg/Kg IM,IV ogni 6-12 ore
- *Tramadolo* (oppioide) 1- 4 mg/Kg/ PO ogni 8 -12 ore

- *Butorfanolo* 0,2-0,4 mg/Kg ogni 2-4 ore

Ketoacidosi diabetica

CLSevIndex 8

Terapia di supporto

FLUIDI

Cristalloidi isotonici, NaCl 0,9% 20 mL/Kg/0ra

Colloidi (plasma) 2 ml/Kg/ora

Integrazione KCl (fino a 10- 20 mEq/L)

Monitoraggio, PCV, PT, glicemia, emogasanalisi

Ketoacidosi diabetica

CLSevIndex 8

Fentanyl (2mcg/Kg ora)

Dolasetron (0,04/ Kg/ 24 ore)

Famotidina (0,5 mg/Kg ogni due ore)

Insulina rapida (0,05-0,1UI/Kg/ora CRI in soluz. NaCl 0,9%)

In contemporamea alla somministrazione di soluzioni cristalloidi isotoniche al 5% e al 2,5% di glucosio.

Fino al mantenimento di una glicemia di circa 180 - 200mg/dL

metabolica

vomito episodico, rifiuta il cibo

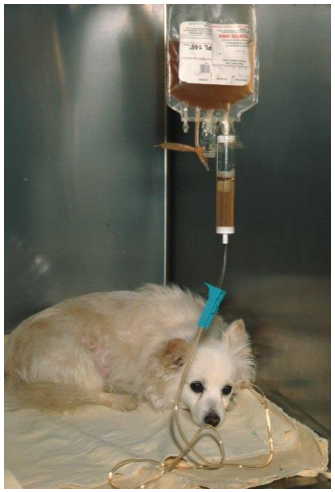
no ketonemia

Dolasetron, 0,04/ Kg/ 24 ore
Nutrizione Enterale Digiunale (via laparotomica) → dieta liquida
Vitamine e aminoacidi
Cristalloidi isotonici bilanciati 5 mL/Kg/ora
Plasma 1 ml/Kg/ora
Insulina lenta (0,5 UI/Kg ogni 12 ore)

Enrofloxacin (10 mg/Kg ogni 24 ore), metronidazolo (10mg/Kg ogni 12 ore)

COLLOIDI/PLASMA

- Ripristinano la pressione colloido-osmotica
- Migliorano il microcircolo



- Plasma: 10-40 ml/Kg/24 ore
  fattori della coagulazione
  ATIII
  albumina

Nessun beneficio significativo (Weatherton et Streeter 2009, *J Vet Emerg Crit Care*, 19:617-22

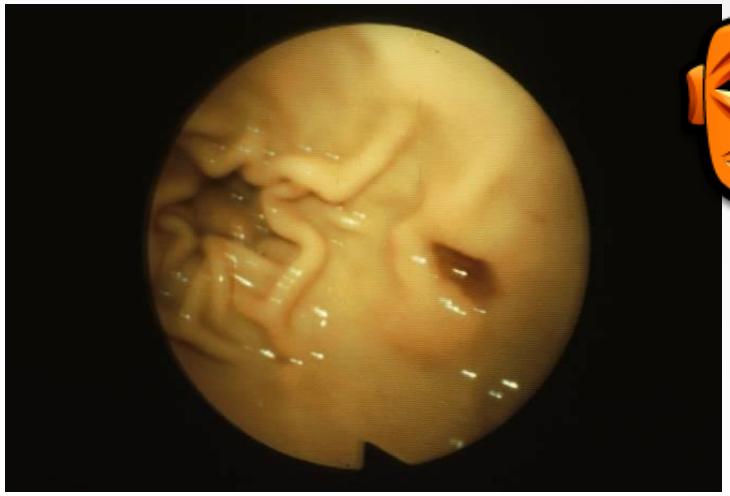
- Colloidi (Voluven): 20 ml/Kg/24 ore

ANTIBIOTICI

- In umana due recenti “consensus reports” per il trattamento delle gravi forme di pancreatite NON ne raccomandano l’uso routinario

- Utilizzo NON associato ad una prognosi migliore

FANS



- Associati a pancreatite
- Noti effetti sul tratto GI (ulcere ed erosioni)
- Nessun effetto benefico sulla flogosi in corso di pancreatite

CORTICOSTEROIDI

- Fino a qualche anno fa associati a pancreatite
- Pazienti in terapia con corticosteroidi possono avere patologie predisponenti pancreatite!

- Nell'uomo eziologia autoimmune responsabile di pancreatite cronica

- Nel cane e gatto pancreatite cronica spesso caratterizzata da infiltrazione linfociti e plasmacellule ed associata ad altre patologie quali IBD, lipidosi, epatite, colangioepatite

Case Report

Journal of Veterinary Emergency and Critical Care **19**(3) 2009, pp 262–268
doi: 10.1111/j.1476-4431.2009.00407.x

Critical illness-related corticosteroid insufficiency in a dog with septic shock

Jamie L. Peyton, DVM and Jamie M. Burkitt, DVM, DACVECC

Abstract

Objective – To describe a case of hydrocortisone-responsive hypotension and critical illness-related corticosteroid insufficiency (CIRCI) in a dog with septic shock.

Case Summary – A dog with aspiration pneumonia developed septic shock with pressor-refractory hypotension. A standard ACTH stimulation test was performed that showed a blunted cortisol response consistent with CIRCI. Reversal of shock was achieved within 2 hours of hydrocortisone administration, and complete weaning from pressors was accomplished over the subsequent 8 hours. The patient recovered and was discharged from the hospital. An ACTH stimulation test performed 1 month after hospital discharge showed normal adrenal responsiveness consistent with resolution of CIRCI.

New or Unique Information Provided – This case is the first published report of hydrocortisone-responsive hypotension and transient CIRCI associated with naturally occurring septic shock in a dog.

(J Vet Emerg Crit Care 2009; 19(3): 262–268) doi: 10.1111/j.1476-4431.2009.00407.x

REVIEW

Therapy for acute pancreatitis with platelet-activating factor receptor antagonists

Chong Chen, Shi-Hai Xia, Hong Chen, Xiao-Hong Li

Acute pancreatitis (AP) causes release of platelet-activating factor (PAF), which induces systemic effects that contribute to circulatory disturbances and multiple organ failure. PAF is a cell surface secretion of bioactive lipid, which could produce physiological and pathological effects by binding to its cell surface receptor called platelet-activating factor receptor (PAF-R). Studies showed that PAF participates in the occurrence and development of AP and administration of platelet-activating factor receptor antagonists (PAF-RAs) could significantly reduce local and systemic events after AP. PAF has also been implicated as a key mediator in the progression of severe AP, which can lead to complications and unacceptably high mortality rates. Several classes of PAF-RA show PAF-RAs significant local and systemic effects on reducing inflammatory changes. As a preventive treatment, PAF-RA could block a series of PAF-mediated inflammatory injury and thus improve the prognosis of AP. This review introduces the important role of PAF-RA in the treatment of AP.

World J Gastroenterol 2008 August 14; 14(30): 4735-4738
World Journal of Gastroenterology ISSN 1007-9327

UNITE

Cane ♂ Pastore Tedesco 3 anni, L

- Da più di un mese diarrea, appetito vorace e progressivo dimagrimento.
- Un anno fa episodio acuto di diarrea. Risoltosi dopo circa 10 giorni con dieta e antibiotici. Da allora frequenti episodi di diarrea (durata circa 7 giorni) sempre associati ad appetito conservato.
- Vaccinato regolarmente e sverminato regolarmente di cui l'ultima 1 settimana prima con Drontal plus.

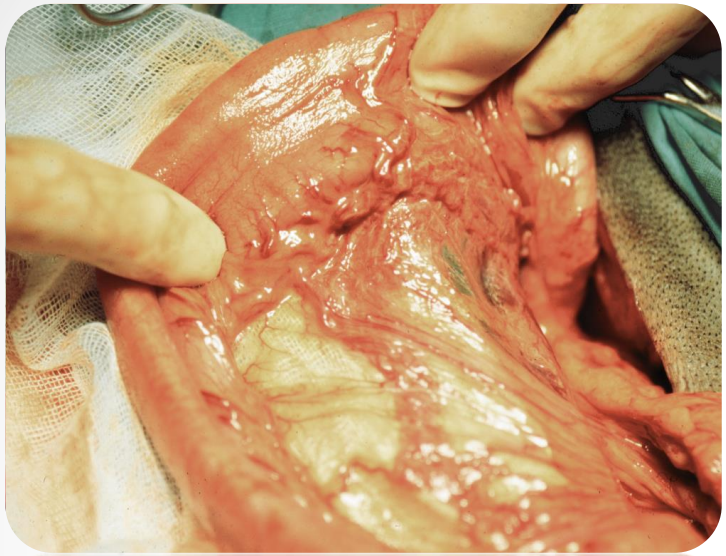
Esame fisico dire

- Scadente stato di nutrizione (BCS 3/9)

- Anse intestinali ripiene di gas e liquido e facilmente apprezzabili.

- Diarrea cronica del piccolo intestino
- Valutazione profilo ematobiochimico completo
- Emocromocotometrico: lieve eosinofilia

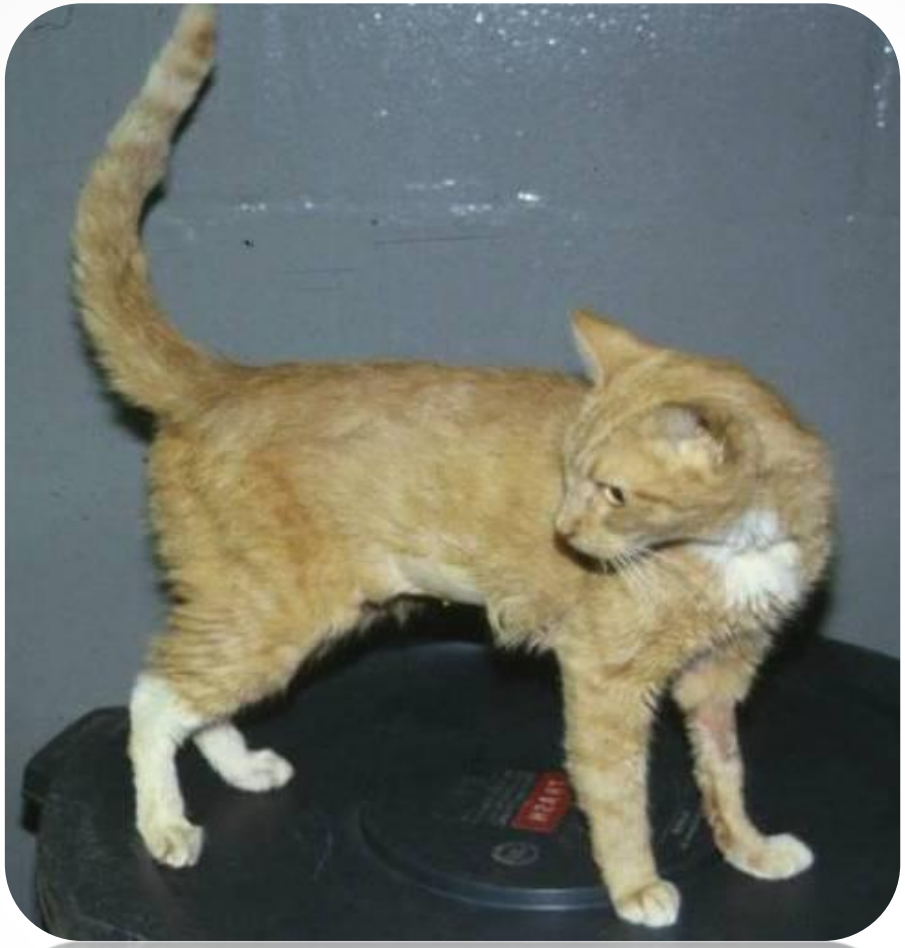
- ALT= 51 IU/L (15-50)
- Prot. Tot.= 5.9 g/dl (5.7-7.7)
- Albumine= 2.8 g/dl (2.56-3.8
- Colesterolo= 90 mg/dl (110-300)
- Amilasi = 500 (< 1036)
- Lipasi= 900 (< 1769)
- Urea= 23 mg/dl (15-45)
- Creatinina= 1.17 mg/dl (0.75-1.30)
- Glucosio= 88 mg/dl (75-103)
- Calcio= 9 mg/dl (8-12)
- Fosforo= 4.6 mg/dl (2.5-4.7)
- Sodio= 146 mEq/l (144-152)
- Potassio= 4.7 mEq/l (4.0-5.2)



TLI < 1 mcg/L (5- 35 mcg/L)

Folati 12 mcg/L (4mcg/l – 13mcg/L)

Cobalamina 213 ng/L (200 ng/L – 600 ng/L)



Gatto europeo FS, 9 anni, Fufina,

- Da 4 mesi dimagrimento
- Polifagia con occasionali episodi iporessia
- Feci voluminose
- Trattato con antielmintici

- Nessuna risposta a trail terapeutici con:

  estratti pancreatici

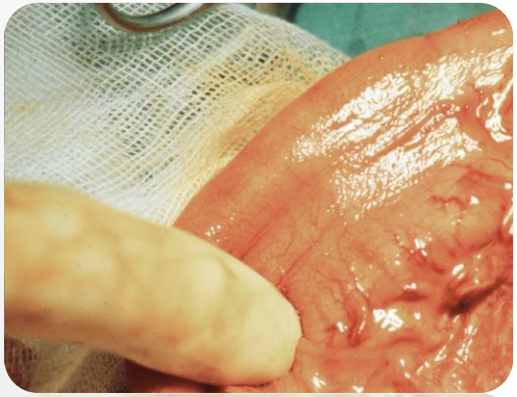
  prednisolone

  prednisolone e metronidazolo

  Dieta: ipoallergenica + idrolizzati

- Esame completo emocromo e biochimico e urine
- Tutto privo di significato
- Lieve aumento ALT
- T_4 : nei limiti della norma

Diarrea cronica del piccolo intestino



fTLI = 3 ug/L (12 - 80 ug/L

Cobalamina = <100 ng/L (300 - 1200 ng/L)

Folate= 5.2 ug/L (6.4 - 21.0 ug/L)

- Folati e cobalamina poco
  sensibili ma molto specifici

- Malattia intestinale molto probabile associata a IPE

- La carenza di cobalamina COMPLICA il quadro e RIDUCE l’efficacia della terapia.

TERAPIA IPE

- Supplementazione dietetica con enzimi pancreatici (Pancreazyme, Viokase, Tryplase)

→ Estratti essiccati di pancreas di manzo o maiale rappresentano il miglior mezzo

1 cucchiaio da tè ogni 10 Kg di peso vivo

(Wiberg et al. 1998, Westermark, 1987, Pidgeon, 1982)

L'attività degli enzimi può variare da confezione a contenitore,
→ La dose effettiva può essere leggermente aggiustata

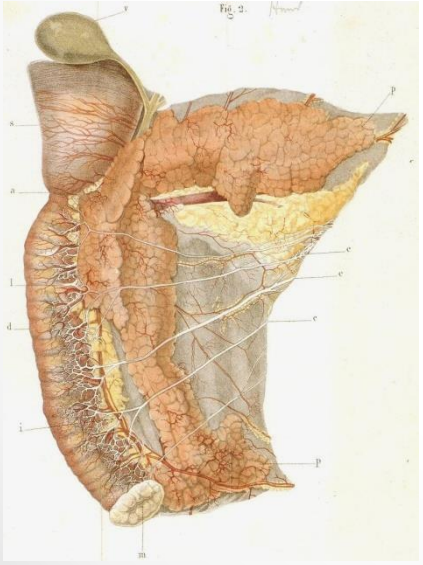
- Sanguinamento buccale

→ Profilo coagulativo (vit K)

→ diminuzione della dose di enzimi

(*Rutz, 2002; Perry, 1991*)

Pancreas crudo, tagliato a pezzi e congelato
(bovino, maiale, ovino)



30g – 90 g corrispondono a 1 cucchiaio da té

Problemi ?

BSE
Malattia di Aujesky
Echinococcosi

- DIETA

→ RESTRIZIONE dei grassi nella dieta → nessun miglioramento nella digestione dei grassi

→ Rischio di carenze vit. Liposolubili e ac. Grassi essenziali

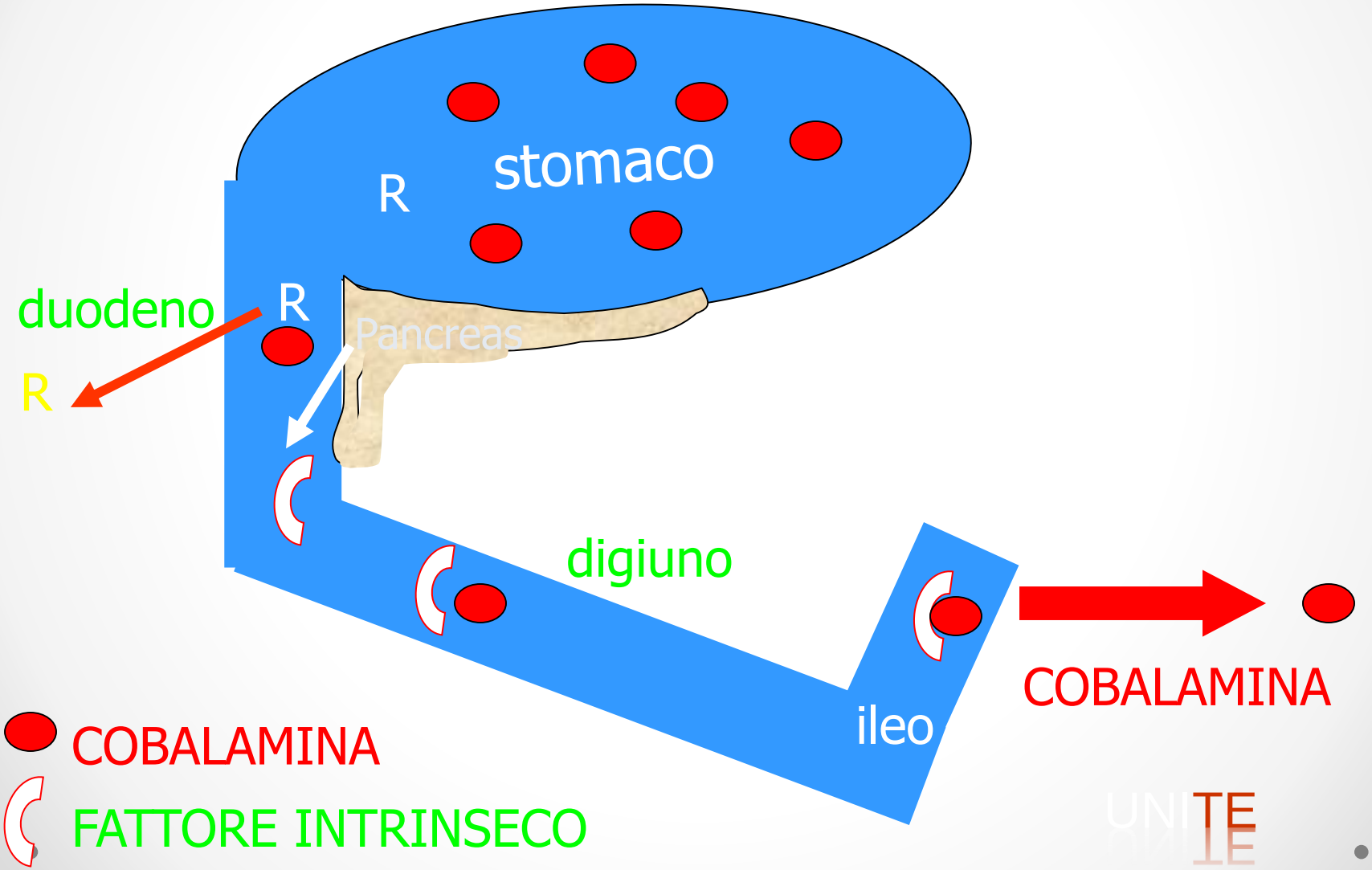
→ diminuzione della dose di enzimi (*Pidgeon, 1982, Rutz, 2004*)

Dieta di mantenimento ad alta qualità
e contenuto in fibre insolubile moderato

COBALAMINA

- Cani: 250-1200 ug
- Gatti: 150-250 ug
- Frequenza: - settimanale per 6 volte
  - ogni 2 settimane per 3 volte
  - 1 dose dopo 1 mese
  - controllo dopo 1 mese
- Via di somministrazione: sottocutanea

ASSORBIMENTO COBALAMINA



J Vet Intern Med 2007;21:54–60

Prognostic Factors in Canine Exocrine Pancreatic Insufficiency: Prolonged Survival is Likely if Clinical Remission is Achieved

Daniel J. Batchelor, Peter-John M. Noble, Rebecca H. Taylor, Peter J. Cripps, and Alexander J. German

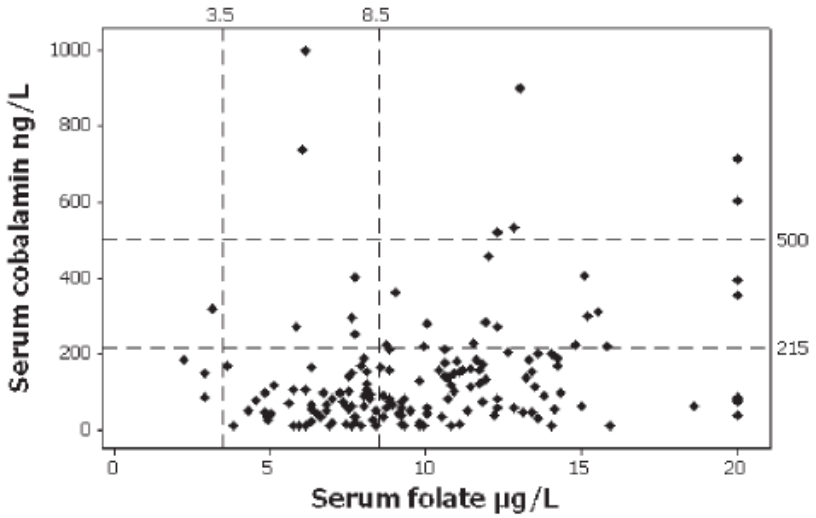


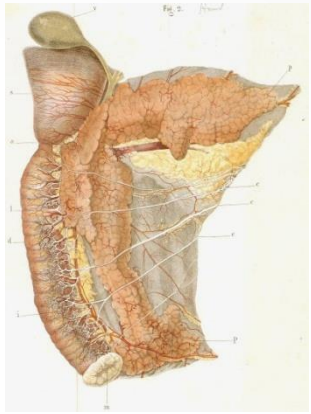
Fig 1. Serum folate and cobalamin concentrations in 163 dogs.

Serum Folate and Cobalamin Concentrations

Serum folate and cobalamin concentrations were measured at the same time as cTLI in 163 dogs, and results are presented in Figure 1. Serum folate concentration was high in 98/163 (60%) dogs, normal in 61/163 (37%), and low in 4/163 (2%). Serum cobalamin concentration was high in 7/163 (4%) dogs, normal in 21/163 (13%), and low in 135/163 (82%). The combination of high folate and low cobalamin concentrations was seen in 77/163 dogs (47%). Fifty-eight dogs (36%) had marked hypocobalaminemia (<100 ng/L) at the time of diagnosis.

Grave ipocobalaminemia associata a diminuita sopravvivenza in cani con EPI

Supplementazione cobalamina parenterale altamente consigliata in cani con EPI



- Vitamine Liposolubili

→ Diminuito l'assorbimento in cani e gatti con IPE (*Rutz, 2004*)

→ ancora non si è compreso il reale ruolo dell'integrazione delle vit liposolubili nel trattamento IPE, considerando gli effetti collaterali del sovradosaggio

Vit E 400 – 500 IU PO ogni 24 ore per 1 mese anedottico

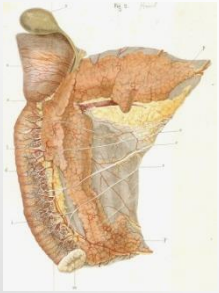
- Mancata RISPOSTA al trattamento

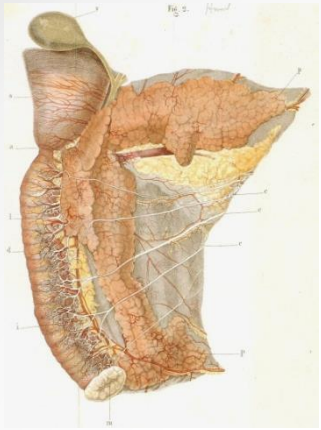
→Riconsiderare dose

→modalità di somministrazione

→Tipo di formulazione utilizzata

→ complicazioni, SIBO, IBD, Diabete mellito

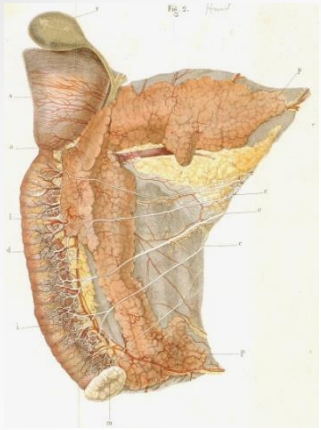




→ Tilosina, 25 mg/ Kg PO ogni 12 ore

→ Metronidazolo, 10 mg/Kg PO ogni 12 ore

→Ossitetraciclina, 25 mg/Kg PO ogni 8-12 ore



Terapia antiacida

Famotidina 0,5 mg/Kg SC,IM,IV, PO ogni 12-24 ore
Ranitidina: 2mg/Kg IM,SC, IV, PO ogni 8-12 ore

Omeprazolo 0,5 – 1 mg /Kg PO ogni 24 ore

Approccio Terapeutico

- Tryplase (2 cps nei 3 pasti giornalieri)
- Dieta “maintenance”
- Ossitetraciclina (15 mg/Kg bid) per 21 giorni
- Dopo 2 settimane nessun miglioramento significativo

- Tryplase
- Tilosina: 25 mg/Kg/bid
- Ranitidina (2 mg/Kg bid os)
- Dopo 2 settimane nessun migioramento

- Utilizzo pancreas di suino (ovino, bovino): circa 100g a pasto
- NETTO MIGLIORAMENTO
- Follow up
- Cane oggi ottime condizioni dopo 8 anni di terapia (addirittura sovrappeso): ancora in terapia con pancreas di suino

- Enzimi pancreatici
- Cobalamina (via Parenterale)
- Ac Folico (1 mg sid per 1 mese)
- Prednisolone (3 mg bid)
  - Da considerare metronidazolo / tilosina
  - Da considerare modificazione dieta

OTTIMO DECORSO