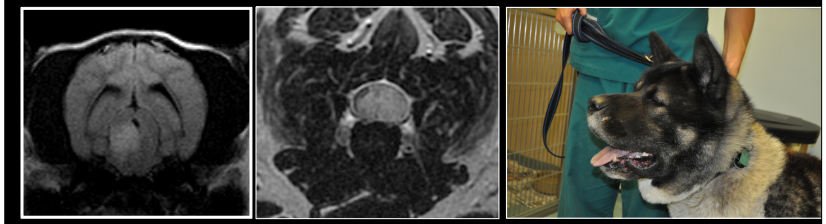
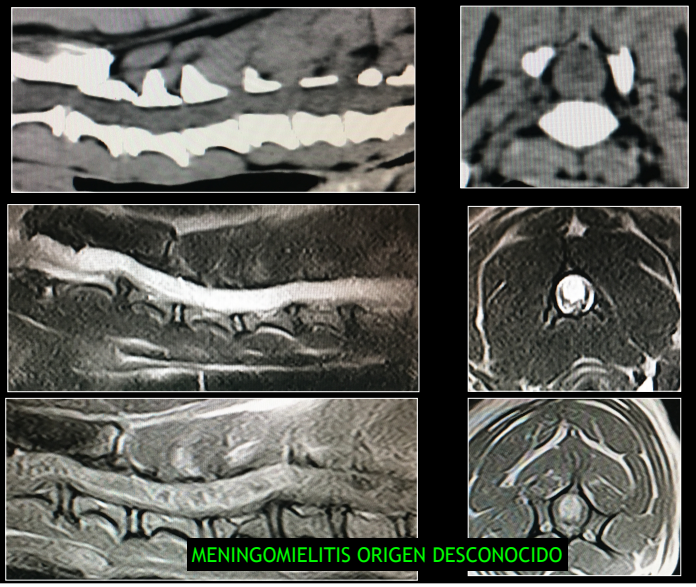
CORTICOIDES USO EN NEUROLOGIA



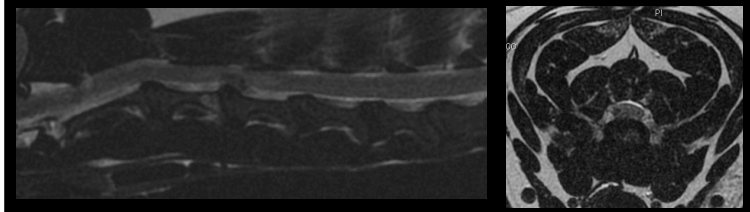
Sergio Rodenas, DVM, MRCVS, Dip ECVN
Especialista europeo reconocido en Neurologia Veterinaria

Servicio Neurologia/Neurocirugia Bluecare Parteners
Facebook; Vetneurologia neurocirugía



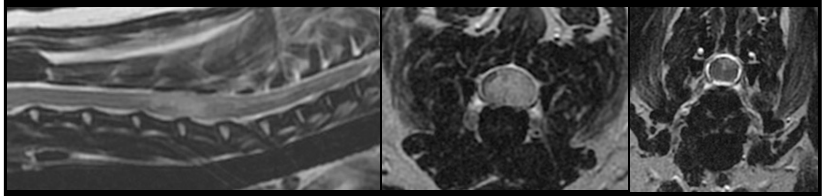
MENINGOMIELITIS ORIGEN DESCONOCIDO

Teckel de 5 años tetraparesia no ambulatoria aguda



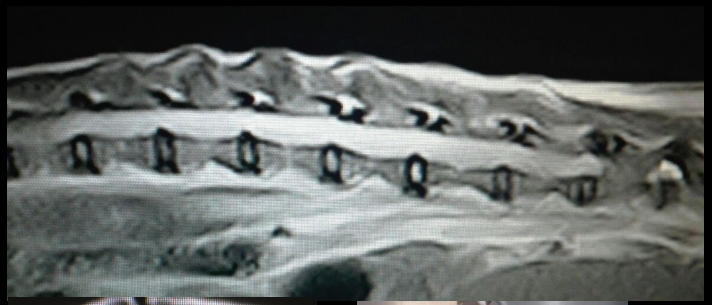
HERNIA DISCAL HANSEN I CIRUGIA

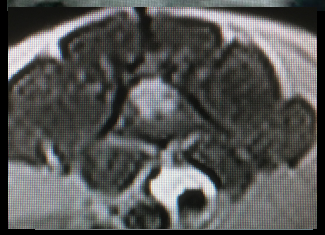
Pomerania de 5 años tetraparesia no ambulatoria aguda

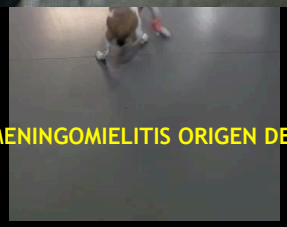


EMBOLISMO FIBROCARGILAGINOSO

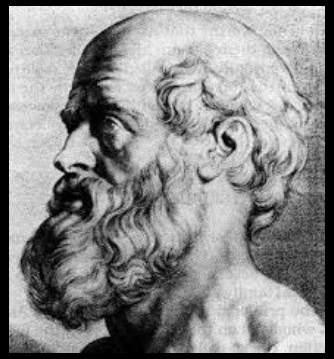
CHIHUAHUA 3 AÑOS INICO AGUDO DE PARAPARESIS







MENINGOMIELITIS ORIGEN DESCONOCIDO

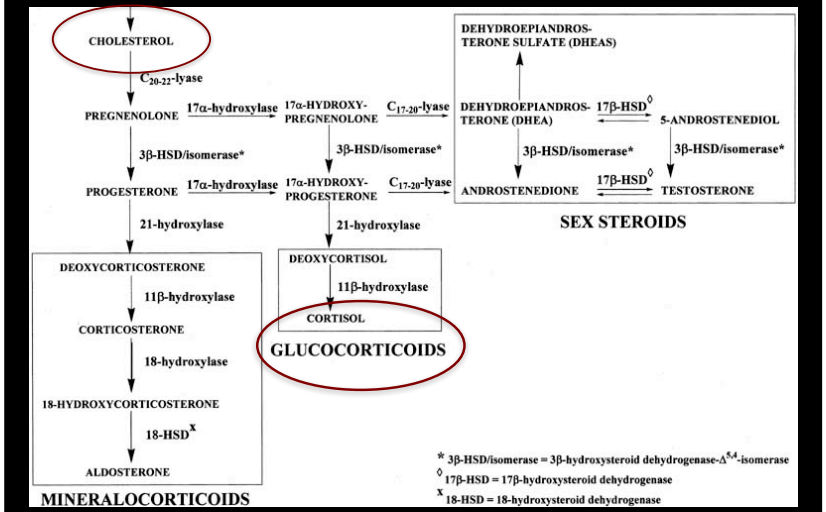


PRIMUM NON NOCERUM (FIRST DO NOT HARM)

AS TO DISEASES, MAKE A HABIT OF TWO THINGS, TO HELP OR AT LEAST NOT TO HARM

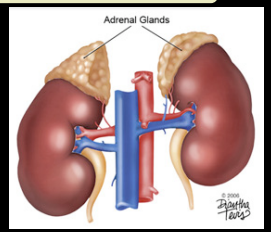
HIPOCRATES 460-357 AC

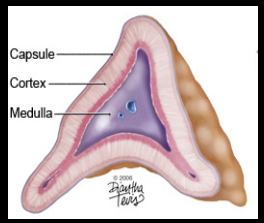
ESTEROIDES

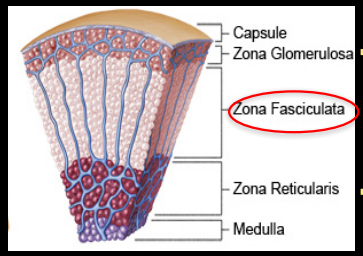


Corticosteroides

ADRENAL GLANDS



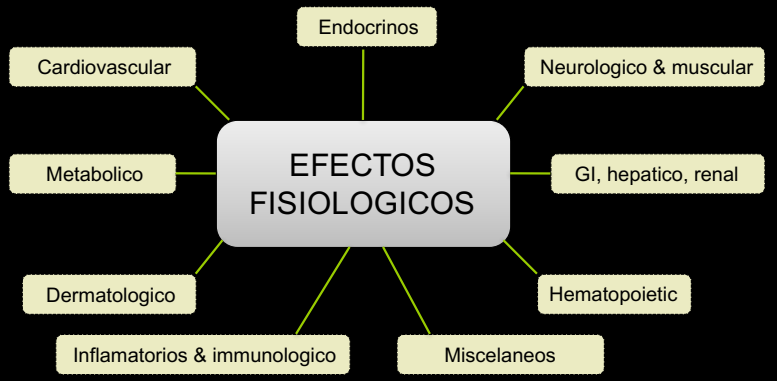




Mineralocorticoides

Glucocorticoides

Glucorticoides



Efectos fisiologicos

- **Efectos metabólicos**
  - Aumenta la gluconeogenesis
  - Aumento del catabolismo proteico
  - Antagónica la insulina y promueve lipolisis.
- **Efectos cardiovasculares**
  - Efectos ionotrópicos
  - Optimiza el numero de receptores de catecolamina
  - Vasoconstriccion
- **Efectos endocrinos**
  - Disminución producción ACTH
  - Supresión TSH y concentraciones T3/T4

Efectos fisiologicos

- **Efectos renales**
  - Aumento tasa de filtración glomerular.
  - Inhibición respuesta ADH túbulos renales
  - Promueve retención de agua, sodio y cloro.
  - Promueve secreción de potasio y calcio
- **Efectos hematopoyéticos**
  - ↑ eritrocitos circulantes, monocitos, plaquetas y neutrófilos maduros
  - ↓ eosinófilos circulantes, linfocitos.
  - Apóptosis de linfocitos activados y neoplásicos.
- **Efectos gastrointestinales y hepáticos**
  - Inducción F Alcalina (solo perros)
  - ↓ absorcion calcio y hierro.
  - ↑ secreccion de hormonas digestivas
  - Promueve deposición de glucógeno y grasa hepática

Efectos fisiologicos

- **Efectos neurológicos y musculares**
  - Atrofia y fatiga muscular
  - Euforia y cambios de comportamiento (diferencias en receptores)
  - Aumenta el numero o sensibilidad de receptores adrenérgicos
- **Efectos inflamatorios e inmunológicos**
  - ↓ formacion de prostaglandinas y leucotrienos
  - Disminucion o aumento de produccion de citoquinas
  - Deprime inmunidad celular directamente
  - Disminuye la inmunidad humoral (efecto secundario)
- **Misceláneos**
  - *Estimula apetito*
  - *Estabiliza membranas lisosomales*
  - *Actividad antioxidante*
  - Acelera reabsorción ósea

Corticoides sintéticos mas comúnmente usados

Agente	Actividad Antiinflamatoria	Mineralocortic	Dosis equivalente mg	Tiempo biológico horas
Cortisona	0.8	0.8	25	8-12
Prednisona/ Prednisolona	4	0,25	5	12-36
Betametasona	25	0	0,6-0,8	>48
Metilprednisolona	5	0	4	12-36
Dexametasona	30	0	0.75	36-54
Hidrocortisona	1	1	20	8-12

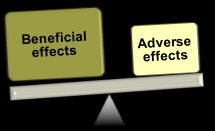
INDICACIONES TERAPEUTICAS

- ENFERMEDAD DE ADDISON
- PROPIEDADES ANTI-INFLAMATORIAS
- INMUNOSUPRESION
- EFECTOS ANTI EDEMA
- CITOTOXICIDAD VS LINFOCITOS NEOPLASICOS (Protocolo linfoma)

INDICACIONES TERAPEUTICAS

- PROPIEDADES ANTI-INFLAMATORIAS
- Regula expresión en genes (20% en leucocitos)
- Aumenta proteinas anti-inflamatorias (annexina,SLP)
- Disminuye enzimas inflamatorias (leucotrienos, prostaglandinas, PLA2)
- Disminuye IL, TNF, etc
- Disminuye fagocitosis mononuclear
- INMUNOSUPRESION
- Inmunidad sobre todo celular
- Reduce células CD4 T y citoquinas T

Glucorticoides



ADVERSE EFFECTS

- Abortion
- Alopecia
- Calcinosis cutis
- Colonic perforation
- Delayed wound healing
- Diabetes mellitus
- Gastrointestinal ulceration
- Growth suppression
- Hypercoagulable state
- Hyperlipidemia
- Iatrogenic hyperadrenocorticism
- Immunosuppression

- Insulin resistance
- Ligament and tendon rupture
- Muscle atrophy, wasting
- Myotonia/myopathy
- Obesity
- Osteoporosis
- Panting
- Polyphagia/polyuria/polydipsia
- Proteinuria
- Psychosis, behavioral changes
- Seizure threshold lowered
- Skin thinning
- Vacuolar hepatopathy

Efectos secundarios adversos

- **Gastrointestinales.** Pancreatitis, gastroenteritis hemorragica, ulceración, perforación
- **Músculo-esquelético.** Debilidad muscular y atrofia
- **Cushing iatrogénico**
- **SNC.** Ansiedad, insomnio, depresión en humanos.

ENMASCARA DIAGNOSTICO, DIAGNOSTICO TARDIO, ENMASCARA SIGNOS CLINICOS, TRATAMIENTO DESPUES MAS COMPLICADO

PREGUNTAR PROPIETARIO ANTES SI TEST DIAGNOSTICOS

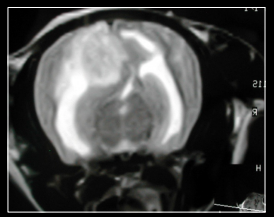
TRATAMIENTO DE CONDICIONES ESPECIFICAS CON CORTICOESTEROIDES EN NEUROLOGIA

EDEMA CEREBRAL

Edema vasógenico

- Daño membrana endotelial
- Disrupcion BHE
- Acumulación de mediadores inflamatorias
- Transporte activo vesicular

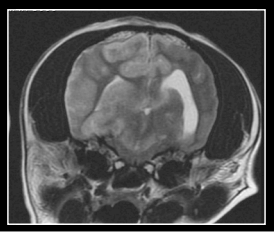
RESPONDE A TRATAMIENTO CON GLUCOCORTICOIDES



Edema citotóxico

Hinchazón de células neuronales gliales y endoteliales a expensas del fluido extracelular en cerebro

Secundario a hipoxia e isquemia



Trauma craneoencefálico

- **Asociado a alta mortalidad en humanos y animales.**

Etiología

Accidentes de trafico, caídas, disparo, mordedura…..

Fisiopatología

Daño primario. Inmediato
-Concusión
-Contusión
-Daño axonal difuso
-Hemorragia, hematoma, edema vasogénico.

La mayoría de veces instantáneo e irreversible

TRAUMA CRANEOENCEFALICO

INMEDIATO → DAÑO PRIMARIO
- Lesion directa parenquima
- Daño neuroaxonal
- Hemorragia
- Fracturas

RETRASADO → DAÑO SECUNDARIO
- Isquemia
- Hemorragia
- Edema

- Na^+, Ca^+
- radicales libres
- Citoquinas
- Glutamato
- ATP

?

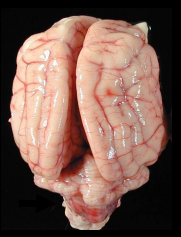
Aumento de PIC
Disminucion flujo sanguineo

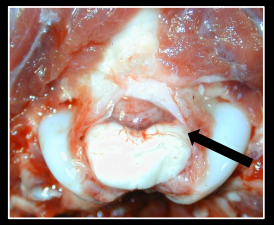
Trauma craneoencefálico

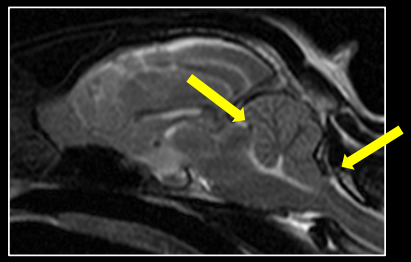
Herniación cerebral

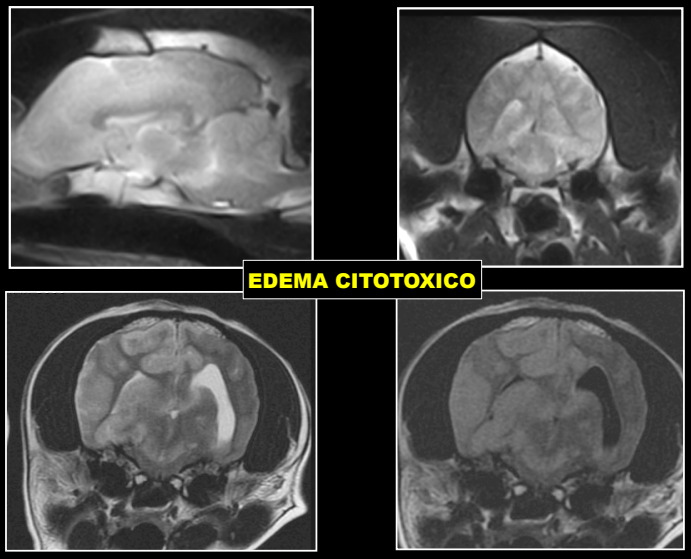
↑ V → ↑ PIC → Hernia

- Ventral al tentorio óseo (herniación tentorial)
- A través del foramen mágnum (herniación tonsilar)
- Ventral a la hoz cerebral.





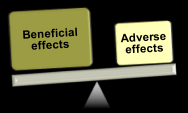




EDEMA CITOTOXICO

Beneficio de corticoesteroides en trauma craneal

- ➢ Inhibición de la peroxidacion lipídica
- ➢ Reducción del edema cerebral.
- ➢ Respuesta antiinflamatoria (inhibición lipocortina)
- ➢ Disminución de la produccion LCR



Efectos adversos en trauma craneal

- ➢ Potencian el daño neuronal en presencia de isquemia
- ➢ Inhiben remielinizacion de neuronas dañadas
- ➢ El tiempo de utilizacion es importante, la isquemia progresiva, la peroxidación lipidica y el edema resultante a menudo irreversible tras 30 minutos
- ➢ Hiperglucemia, asociada con alta mortalidad en trauma craneal
- ➢ Aumento de riesgo de infección.

➢ The Brain Trauma Foundation and American Association of Neurological Surgeons. (1995, 2000).

El uso de corticoides no indicado en trauma craneal.

Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head injury (MRC CRASH trial): randomised placebo-controlled trial

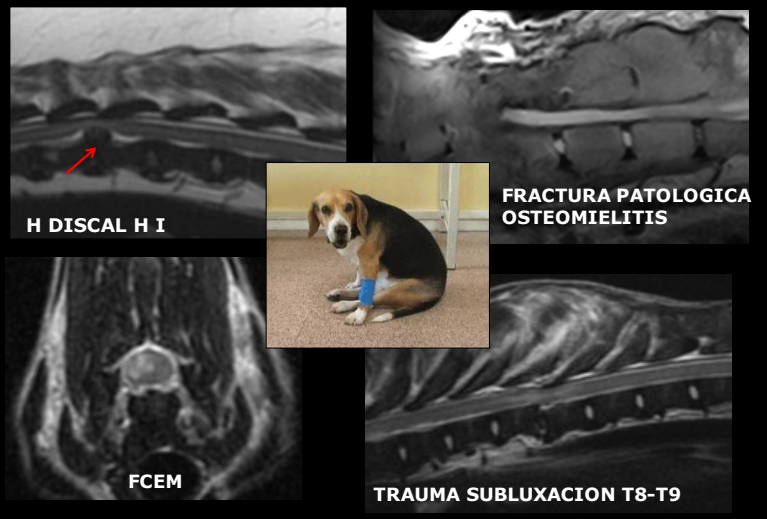
(Lancet 2004;364:1321)

MPSS:

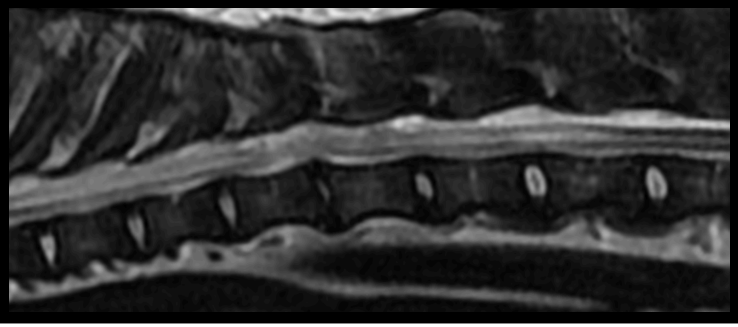
~~GLUCOCORTICOIDES~~

Mínimos beneficios si alguno en comparación con las desventajas

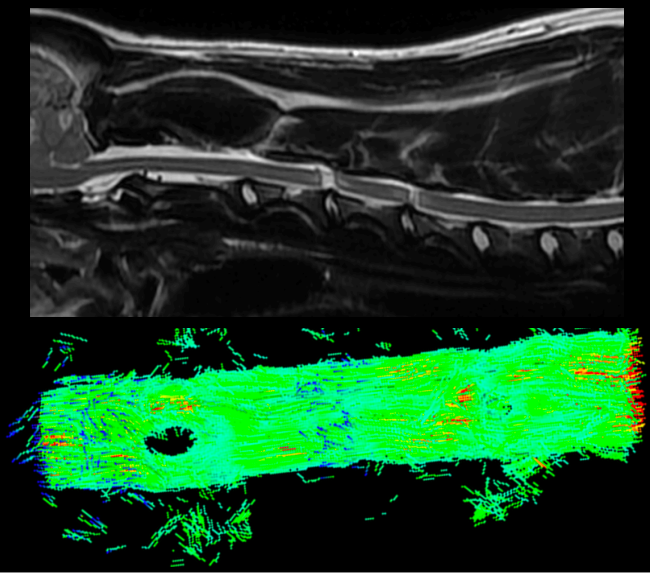
Trauma espinal



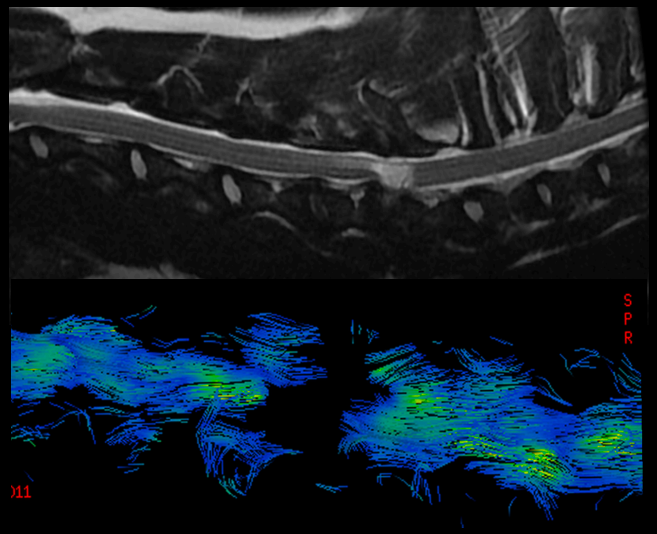
HERNIA DISCAL NO COMPRESIVA NUCLEO PULPOSO



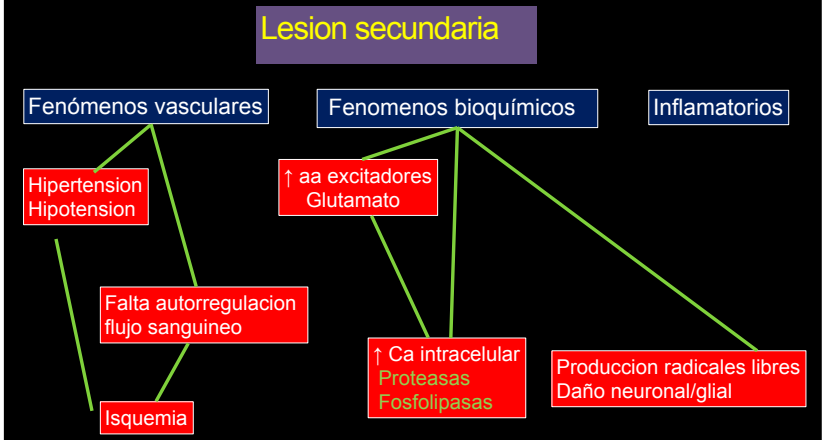
PRONOSTICO TRACTOGRAFIA



PRONOSTICO TRACTOGRAFIA



Fisiopatología



Apoptosis/muerte neuronal

Trauma espinal

- **Uso de corticoesteroides**

-Uso sugerido en base a las propiedades de luchar contra los radicales libres. **MPSS** altas dosis.

Efecto neuroprotectivo en base a las propiedades de quelar radicales libres mas que a propiedades antiinflamatorias (no efecto en concentraciones postrauma fosfolipasa A)

Humana (NASCIS II)

- 30 mg/kg IV
- 5.4 mg/kg/h IV durante 24h

Antes de 8 horas, pequeños beneficios
Más de 8 horas, empeora pronostico
Bracken et al 1990

- Antes de 3 horas

Régimen infusión 24 h

- 3 – 8 horas

Régimen infusión 48 h

CONTROVERTIDO !!!

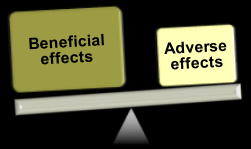
Trauma espinal

- **Uso de corticoesteroides**

Humana (NASCIS II)

- 30 mg/kg IV MPS 3-8 horas
- 5.4 mg/kg/h IV durante 24h
- REANALISIS DE LOS DATOS; CONTROVERSIA EN LA ESTADISTICA
- MPS PUEDE SER UNA OPCION PERO VALORAR RIESGOS BENEFICIOS

(Neurosurgery 2002;51:855)



High dose methylprednisolone in the management of acute spinal cord injury – a systematic review from a clinical perspective

DJ Short*[,1], WS El Masry[1,3] and PW Jones[2,4]

[1]*Midlands Centre for Spinal Injuries, Robert Jones & Agnes Hunt Orthopaedic & District Hospital NHS Trust, Oswestry, Shropshire, SY10 9DP, UK;* [2]*Department of Mathematics, Keele University, Staffordshire, ST5 5BG, UK*

Study design: Systematic literature review for primary data using predefined inclusion, exclusion and validity criteria. Primary outcome measure was standardised neurological examination or neurological function. Secondary outcomes; acute mortality, early morbidity.
Objectives: To access the literature available to clinicians systematically and evaluate the evidence for an effect of high dose methylprednisolone (MPSS) on neurological improvement following acute spinal cord injury (ACSI).
Methods: Information retrieval was based on Medline search (1966 through December 1999) using the strategy 'spinal cord injury' and 'methylprednisolone' (or 'dexamethasone') with no other restrictions. Primary data publications using high dose steroids given within 12 h following spinal cord injury and reporting outcome measures separately for steroid and non-steroid treated groups were selected. Evaluation followed the guides of Guyatt *et al*[7] (for the Evidence Based Working Group in Canada). Studies with questionable validity were excluded. Level of evidence and treatment recommendation utilised the Canadian Task Force on the Periodic Health Examination criteria.[6] Experimental spinal cord injury studies on larger animals were included; small mammal experiments were considered beyond evaluation.
Results: Three clinical trials and six cohort study publications were found to satisfy the review criteria. The evidence they provide supports 'the recommendation that the manoeuvre (high dose methylpredisolone) be excluded from consideration as an intervention for the condition'[10] (acute spinal cord injury). Twelve larger animal publications were detailed. Validity and the functional significance of results was of concern in many. The weight of evidence lay with those studies demonstrating no definite effect of MPSS on functional outcome. In cat experiments with higher level cord damage, deaths in the MPSS treated groups were notable.
Conclusion: The evidence produced by this systematic review does not support the use of high dose methylprednisolone in acute spinal cord injury to improve neurological recovery. A deleterious effect on early mortality and morbidity cannot be excluded by this evidence.
Spinal Cord (2000) **38,** 273 – 286

Keywords: methyl

NO EVIDENCIA DE MEJORIA

Trauma espinal

Perros

- 30 mg/kg IV MPSS
- 15 mg/kg IV a 2 y 6 horas
- 15 mg/kg/8h durante 48 h

- Extrapolado de estudios en humana
- Beneficio no probado. Experimental.
- Evaluar riesgos con beneficios.

Efectos secundarios

- **Hipotension**
- **Vómitos**
- **Infecciones (ej, neumonía)**
- **Perforacion gastrointestinal**
- **Hemorragia GI**

Trauma espinal

- En un estudio en perros tras una cirugía espinal, recibieron una dosis 30 mg/kg MPSS seguido de la mitad a la dosis completa 2-4 horas mas tarde.
  90% hemorragia gastrointestinal

A Placebo-Controlled, Prospective, Randomized Clinical Trial of Polyethylene Glycol and Methylprednisolone Sodium Succinate in Dogs with Intervertebral Disk Herniation

N.J. Olby, A.C. Muguet-Chanoit, J.-H. Lim, M. Davidian, C.L. Mariani, A.C. Freeman, S.R. Platt, J. Humphrey, M. Kent, C. Giovanella, R. Longshore, P.J. Early, and K.R. Muñana

~~GLUCOCORTICOIDES~~

Results: Sixty-three dogs were recruited and 47.6% recovered ambulation. 17.5% developed progressive myelomalacia but there was no association with group. There was no difference in OFS among groups. Although full study power was not reached, conditional power analyses indicated the futility of continued case recruitment.
Conclusions: This clinical trial did not show a benefit of either MPSS or PEG in the treatment of acute, severe thoracolumbar IVDH when used as adjunctive medical treatment administered to dogs presenting within 24 hours of onset of paralysis.
Key words: Neuroprotection; Paraplegia; Secondary injury; Spinal cord injury.

Enfermedad discal aguda moderada

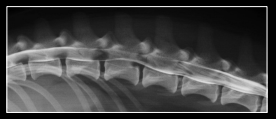
- **H discal Hansen I**

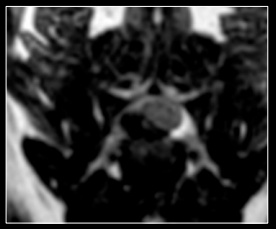
- Beneficio anecdótico no probado de AIES

- Tratamiento confinamiento estricto 3 semanas

- El uso de corticoides puede mejorar momentáneamente el cuadro favoreciendo el movimiento del animal

- Si uso de AINES, u otros medicamentos. Confinamiento





Enfermedad discal aguda moderada

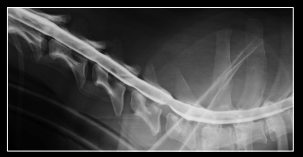
- **H discal Hansen I**

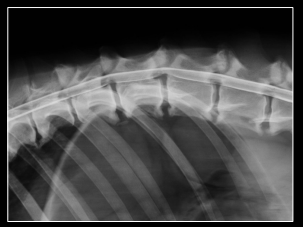
Adverse effects and outcome associated with dexamethasone administration in dogs with acute thoracolumbar intervertebral disk herniation: 161 cases (2000-2006). JAVMAM 2008
Levine JM[1], Levine GJ, Boozer L, Schatzberg SJ, Platt SR, Kent M, Kerwin SC, Fosgate GT.

Results indicated that treatment with dexamethasone before surgery is associated with more adverse effects, compared with treatment with glucocorticoids other than dexamethasone or no treatment with glucocorticoids, in dogs with thoracolumbar intervertebral disk herniation. In this study population, no difference in outcome was found among groups. These findings suggest that the value of dexamethasone administration before surgery in dogs with thoracolumbar disk herniation should be reconsidered

Enfermedad discal crónica y otras mielopatías compresivas

- **HD Hansen II**
- **Espondilomielopatia cervical caudal**
- **Diverticulo aracnoideo**
- **Otros**



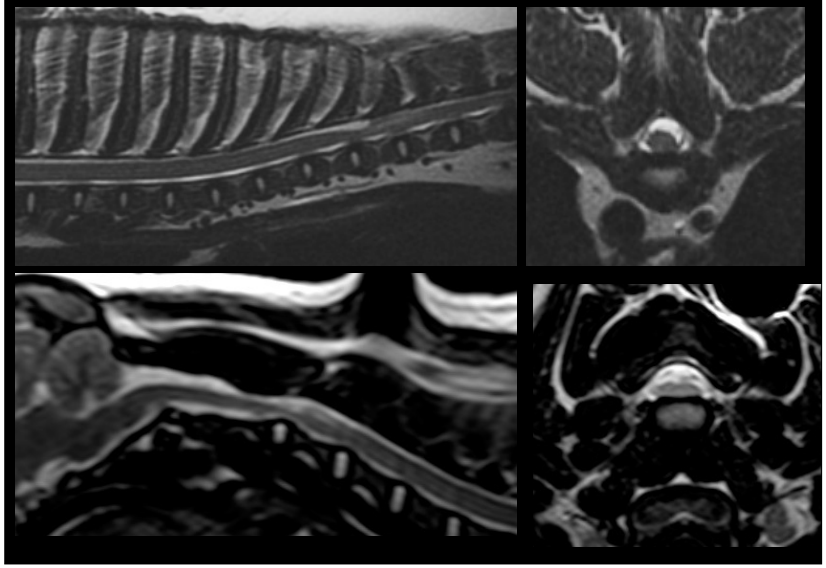


Utilizado como antiinflamatorio, mejorar flujo espinal y el edema

Prednisona 0,5 mg/kg/12h

Ir bajando paulatinamente o dosis sin efectos secundarios

DIVERTICULOS ARACNOIDEOS



CONGENITO/IDIOPATICO/MISCELANEOS

DIVERTICULOS ARACNOIDEOS

Spinal Arachnoid Diverticula: Outcome in 96 Medically or Surgically Treated Dogs

D.A. Mauler, S. De Decker, L. De Risio, H.A. Volk, R. Dennis, I. Gielen, E. Van der Vekens, K. Goethals, and L. Van Ham

Background: Little is reported about the role of medical management in the treatment of spinal arachnoid diverticula (SAD) in dogs.

Objectives: To describe the outcome of 96 dogs treated medically or surgically for SAD.

Animals: Ninety-six dogs with SAD.

Methods: Retrospective case series. Medical records were searched for spinal arachnoid diverticula and all dogs with information on treatment were included. Outcome was assessed with a standardized questionnaire.

Results: Fifty dogs were managed medically and 46 dogs were treated surgically. Dogs that underwent surgery were significantly younger than dogs that received medical management. No other variables, related to clinical presentation, were significantly different between both groups of dogs. The median follow-up time was 16 months (1–90 months) in the medically treated and 23 months (1–94 months) in the surgically treated group. Of the 38 dogs treated surgically with available long-term follow-up, 82% (n = 31) improved, 3% (n = 1) remained stable and 16% (n = 6) deteriorated after surgery. Of the 37 dogs treated medically with available long-term follow-up, 30% (n = 11) improved, 30% (n = 11) remained stable, and 40% (n = 15) deteriorated. Surgical treatment was more often associated with clinical improvement compared to medical management (P = .0002).

Conclusions and Clinical Importance: The results of this study suggest that surgical treatment might be superior to medical treatment in the management of SAD in dogs. Further studies with standardized patient care are warranted.

Key words: Arachnoid cyst; Spinal cord; subarachnoid cyst.

Enfermedades inflamatorias

Meningoencephalomielitis infecciosas

Bacterianas
Rikettsias erlichia, borrelia
Viricas moquillo, PIF, herpesvirus
Fúngicas criptococosis
Protozoarias Toxoplasma, neospora
Parasitos cuterebra, D inmitis

Propiedades inmunosupresivas, pueden agravar La enfermedad.

Propiedades antiinflamatorias pueden ser de gran ayuda sobre los efectos del daño infeccioso en el SNC. (cortos periodos de tiempo)

Meningoencephalomielitis infecciosas

Bacterianas

Dexametasona 0.15mg/kg/4 días antes del tratamiento antibiótico Ha mostrado disminuir la presión IC, la inflamación SNC y las secuelas neurológicas. (literatura humana)

Trials en humana han mostrado un efecto beneficioso del uso de Dexametasona asociado a la antibioterapia.

No estudios ni trial en pequeños animales de la eficacia de terapia con esteroides en meningitis bacterianas.

Extrapolación de estudios de humana

Apropiado???

Meningoencephalomielitis infecciosas

Virales

Moquillo

Pronostico grave en forma neurológica
En algunos casos remision o disminucion de signos temporal con tratamientos cortos de AIES o inyección simple dedexametasona

PIF Altas dosis, Prednisolona 2-4 mg/kg/dia

Rikettsias

La administración de AIES prolonga la duración de rikettsemia
La severidad de signos clínicos no aumenta en perros infectados experimentalmente. ***(Greene 1998)***

Evaluar riesgos/beneficios según estado neurológico

Meningoencephalomielitis de origen desconocido (granulomatosa)

Meningoencefalitis no supurativas

No histopatología

Signos clínicos multifocales

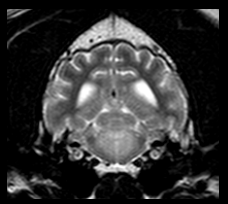
Lesiones RMN/TAC multifocales/focales

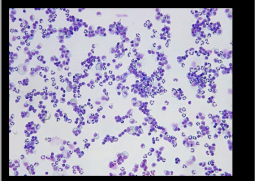
LCR inflamatorio (>> pleocitosis mononuclear)

Serologías/PCR enfermedades infecciosas negativas

Immunomediada (primaria, virus????)

Tratamiento immunosupresión





Meningoencephalomielitis de origen desconocido (granulomatosa)

-1) Forma ocular (rara)

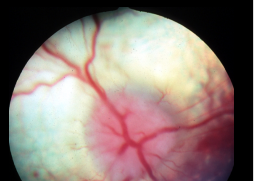
-Inicio agudo de problemas visuales
ceguera parcial/total

- Normalmente midriasis sin respuesta a la luz
Unilateral/bilateral (neuritis óptica)

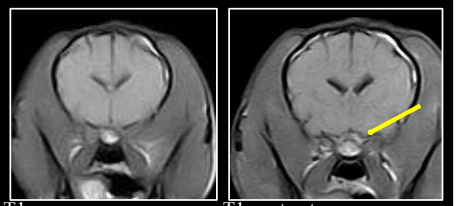
-Fondo ojo
edema, inflamacion disco óptico
Coriorretinitis, hemorragias

- RMN/LCR

- Posible progresión signos SNC



Cortesía Dra Marta Leiva (UAB)



T1 T1 contraste

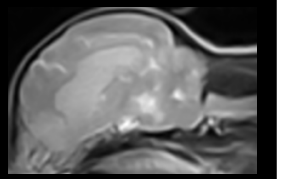
Meningoencephalomielitis de origen desconocido (granulomatosa)

-2) Forma focal

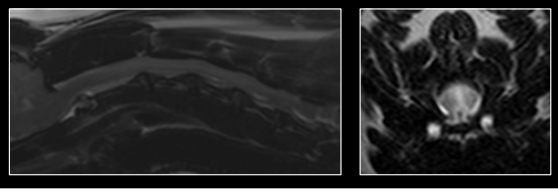
Signos neurológicos segun localización

Signos agudos o progressivos (pueden mimetizar tumores)

- encéfalo
cerebro, tronco de encéfalo, cerebelo



- médula espinal



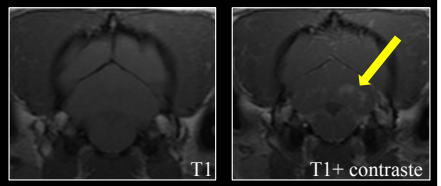
Meningoencephalomielitis de origen desconocido (granulomatosa)

-3) Forma diseminada o multifocal

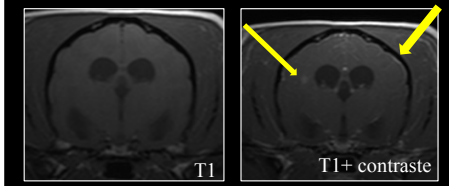
N óptico, encéfalo, médula espinal

- Signos multifocales

- Agudo, rapida progresión

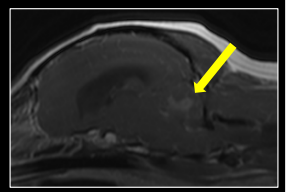


T1 T1+ contraste



T1 T1+ contraste

Hemisferio cerebral



Cerebelo T1+ contraste

Meningoencephalomielitis de origen desconocido (granulomatosa)

AIES

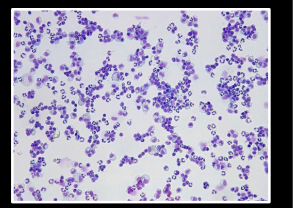
-Inyección unica dexametasona 0.25mg/kg
-Prednisonolona 1-2 mg/kg BID
-Reducir gradualmente. Dosis efectiva

Prednisona 1- a 1.5mg/kg/12 horas/3-4 semanas (casos más graves)

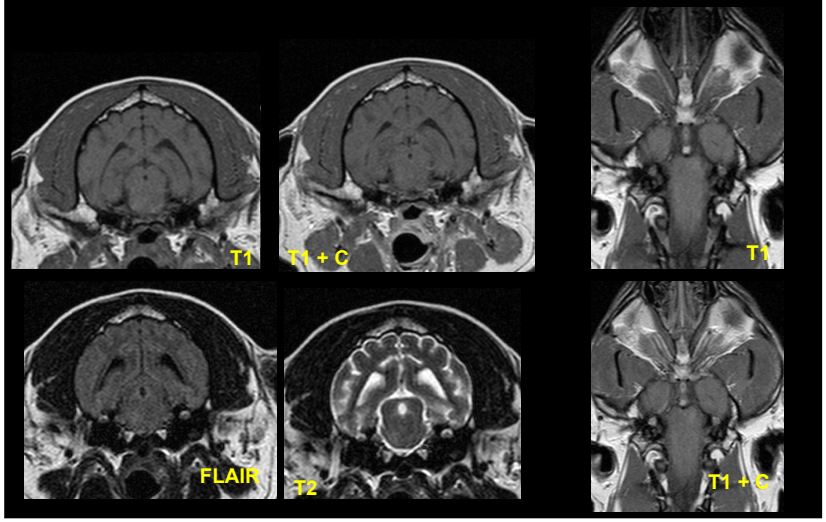
1mg/kg/12 horas/4-6 semanas
0.5 mg/kg/12 horas/3-4 semanas
0.5 mg/kg/24 horas/2-4 semanas
0.5 mg/kg/48 horas/2-4 semanas

Tratamientos alternativos

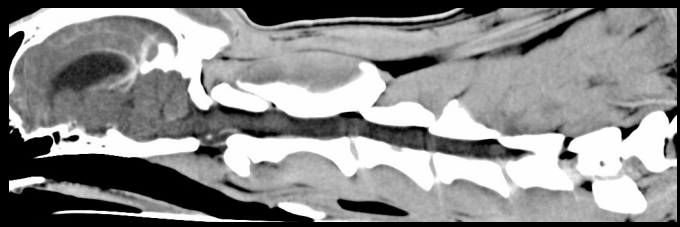
- ✓Citosine- arabinoside
- ✓Procarbazine
- ✓Ciclosporina A
- ✓Leflunomida
- ✓Cirugía y Radioterapia



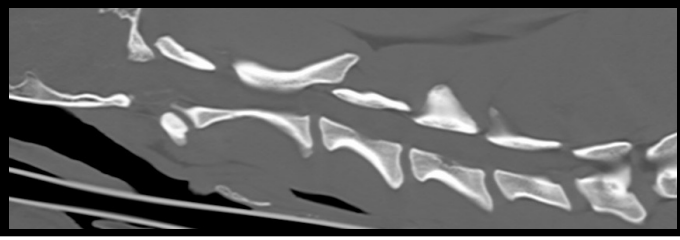
Meningoencephalomielitis de origen desconocido (granulomatosa)



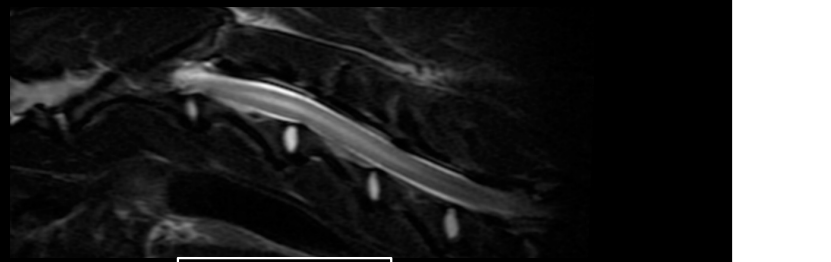
- MENINGOMIELTIS INMUNOMEDIADA



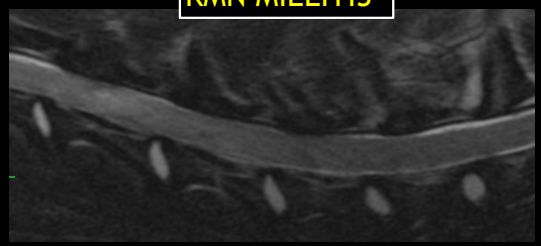
TAC NO LESION VISIBLE



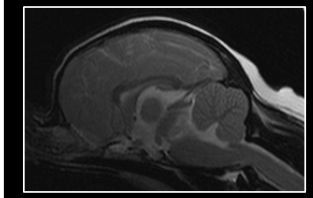
- MENINGOMIELTIS INMUNOMEDIADA

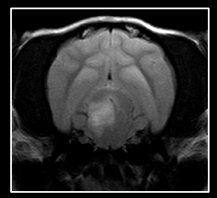


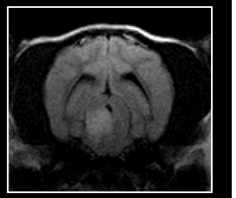
RMN MIELITIS

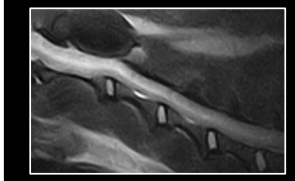


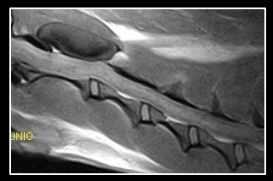
Meningoencephalomielitis de origen desconocido (granulomatosa)

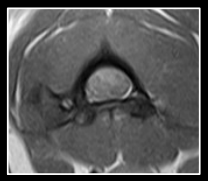

T2


T2


FLAIR

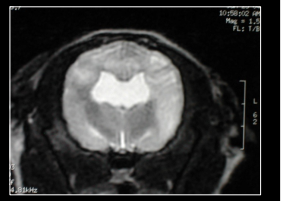

T2


T1+C


T1+C

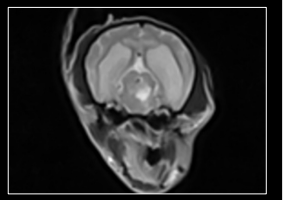
Meningoencefalitis necrotizante

Carlino, maltese. yorkshire, chihuahua
Causa desconocida
LCR pleocitosis mononuclear
Histología: lesiones necróticas con
Meningitis diseminada, coroiditis y
encefalitis



Leucoencefalitis necrotizante

Yorkshire y chihuahua



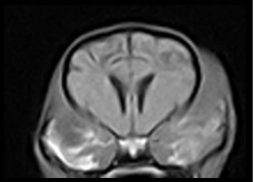
Tratamiento como la MOD

Meningoencefalitis necrotizante

- Tratamiento

N=46. Tratamiento. Media supervivencia 100d (1-680d)
N=6. No tratamiento. Media 7.4d (3-18)

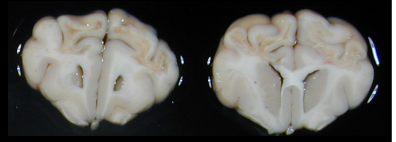
Anticonvulsivos (Fenobarbital)
Prednisona
Otros



- Pronóstico

Grave a fatal
N=60. Media supervivencia 93d (1-680d)

Levine et al, JVIMK 2008

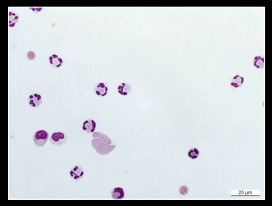


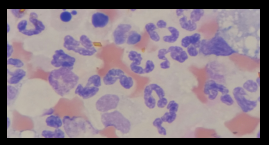
- Histopatología

- Inflamación asociada a areas de necrosis

Meningitis-arteritis SRMA

Perros raza grande jovenes < 2 años
LCR: Pleocitosis neutrofílica
IgA en LCR y suero
Poliartritis inmunomediada asociada
Causa inmunológica?
Moderada vasculitis?
BOXER, BOYERO, PERRO AGUA, BEAGLE

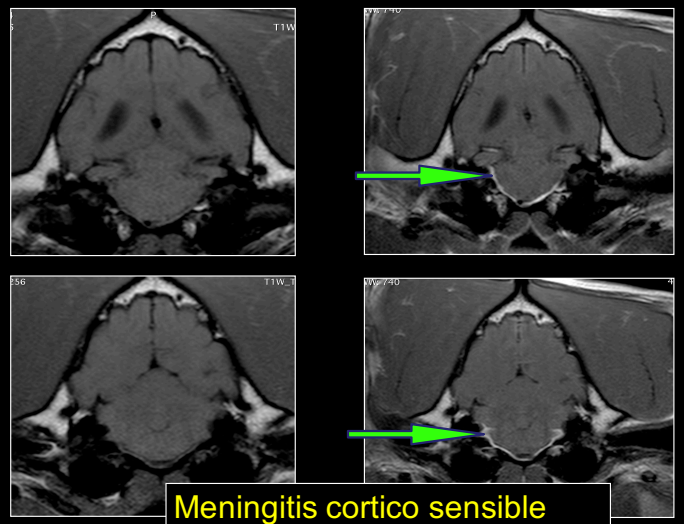




o Prednisona
-4mg/kg/24h durante 2 días
-2 mg/kg/24h 2-3 semanas
-Bajar gradualmente.
- Control PCR

Pronostico resolución signos excelente
En 50% casos. 2 años remisión

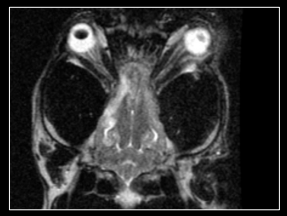
Si recidiva o no mejoria, Ciclosporina, citarabina, azatioprina



Meningitis cortico sensible

Enfermedades neoplásicas

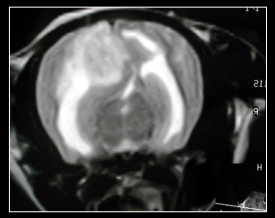
Controlar hidrocefalia secundaria
Edema peritumoral
Reducir presión IC



Dosis antiinflamatorias reducen
Produccion LCR, edema vasógenico
Así como flujo sanguíneo al tumor en 24 h

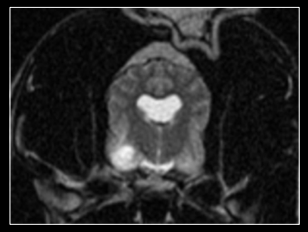
Reducción del edema al disminuir la patológicamente Incrementada permeabilidad capilar de la BHE

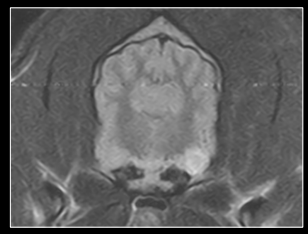
Actuan sobre cel endoteliales ↓ la permeabilidad y ↓ la Presión IC

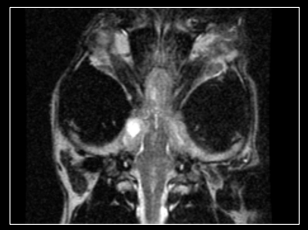


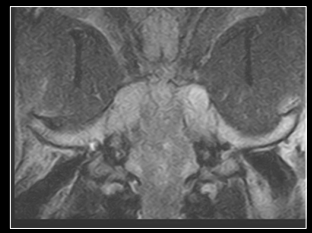
PREDNISONA A 0,5 MG/KG/12 HORAS

Enfermedades neoplásicas









Glioma lob piriforme

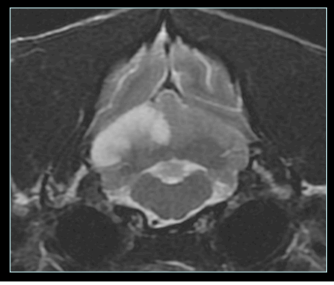
4 meses tras AIES y lomustina

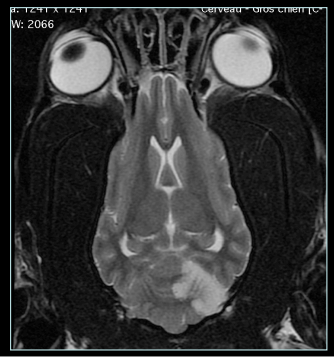
Enfermedadesvasculares

ENFERMEDAD CEREBROVASCULAR

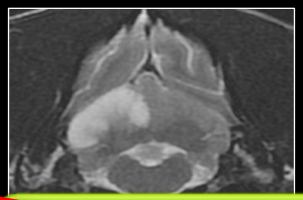
Progresión edema 24-72 horas en Isquemias.
Hemorragias pueden ser más progresivas

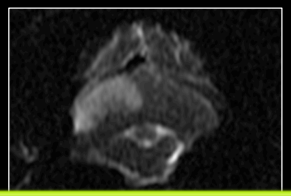
La mayoría del daño cerebral por edema citotóxico.
Disfunción en membrana celular



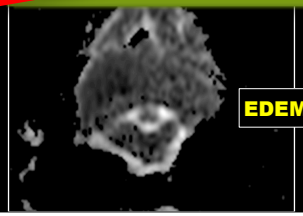


Enfermedadesvasculares

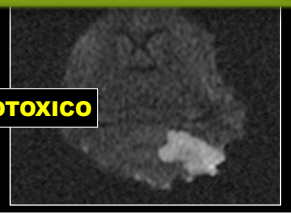




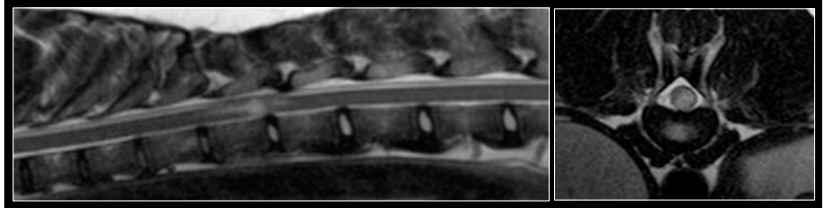
~~GLUCOCORTICOIDES~~



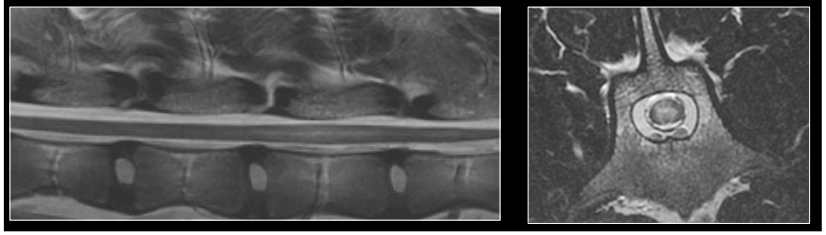
EDEMA CITOTOXICO

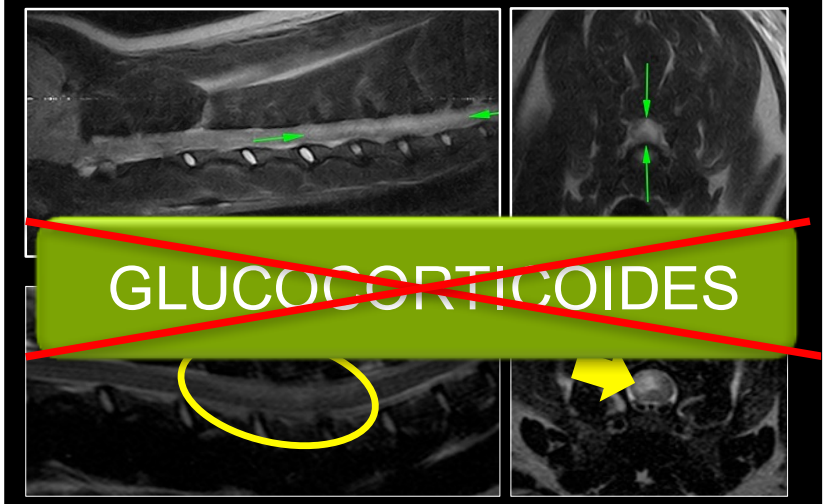


MIELOPATIA ISQUEMICA (EFC)



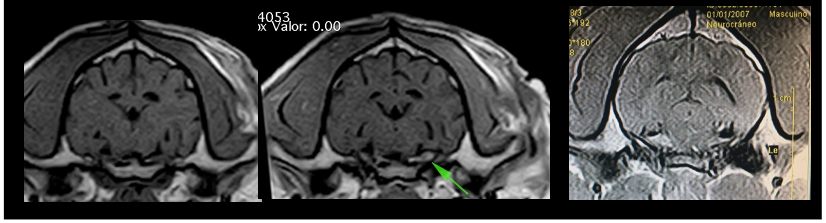
MIELOPATIA ISQUEMICA (EFC)



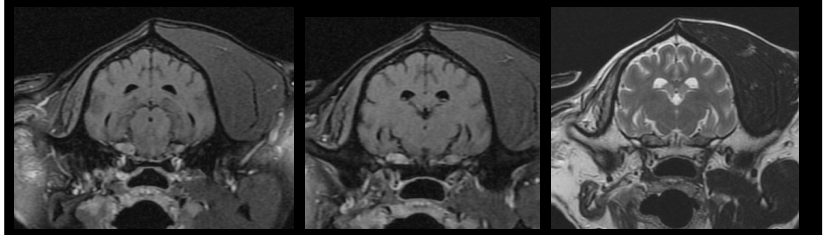


EDEMA CITOTOXICO

NEURITIS DEL NERVIO TRIGEMINO



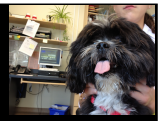
NEOPLASIA VERSUS NEURITIS NC V



TRATAMIENTO INMUNOSUPRESOR

- **CORTICOESTEROIDES a 1 mg/kg/12 horas (ojo atrofia muscular)**
- **CICLOSPORINA, CITARABINA, OTROS**
- **GABAPENTINA NEURALGIA DEL TRIGEMINO**

NC V (TRIGEMINO)



NEUROPATIA DEL NERVIO TRIGEMINO IDIOPATICA

Trigeminal neuropathy in dogs: a retrospective study of 29 cases (1991-2000).
Mayhew PD, Bush WW, Glass EN. J Am Anim Hosp Assoc. 2002

The medical records of 29 dogs unable to close their mouths due to flaccid paralysis or paresis of the muscles innervated by the mandibular branch of the trigeminal nerve, were reviewed. **Idiopathic trigeminal neuropathy was diagnosed in 26 dogs based on complete resolution of clinical signs and lack of any long-term neurological disease.** Of these dogs, golden retrievers were overrepresented. No age, sex, or seasonal predispositions were identified. Trigeminal sensory innervation deficits were observed in 35% (9/26), facial nerve deficits were observed in 8% (2/26), and **Horner's syndrome was observed in 8% (2/26) of dogs.** Electromyographic examination of the muscles of mastication revealed abnormalities in seven of nine dogs. Results of cerebrospinal fluid analysis were abnormal in seven of eight dogs. Corticosteroid therapy did not affect the clinical course of the disease. Mean time to recovery was 22 days. Lymphosarcoma, Neospora caninum infection, and severe polyneuritis of unknown origin were diagnosed in three of 29 dogs at necropsy.

NEUROPATIA DEL NERVIO TRIGEMINO IDIOPATICA

~~GLUCOCORTICOIDES~~

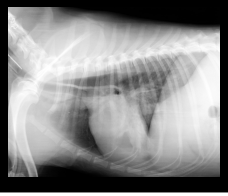
MIASTENIA GRAVE

- CORTICOIDES?

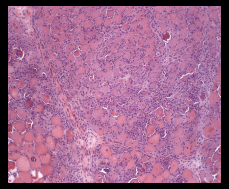
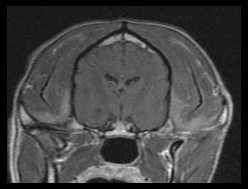
- Si no respuesta optima con anticolinesterásicos
- Aumento de debilidad al comienzo de tratamiento (hospitalización, problemas respiratorios)

- Perros comenzar a bajas dosis 0.5 mg/ día e ir Incrementando.
- Valorar efectos secundarios

Intentar no usar, la mayoría de los casos controlados con anticolinesterásicos



POLIMIOSITIS, MMM, EXTRAOCULAR


Perros	Gatos
Prednisona a:	Prednisona a:
2 mg /kg/12 horas/2 semanas 1 mg/kg/12 horas/3 semanas 1 mg/kg/24 horas/3 semanas 0,5 m/kg/24 horas/3 semanas 0,5 mg/kg/48 horas 2 semanas	3 mg/kg/12 horas/2 semanas 2 mg/kg/12/3 semanas 1 mg/kg/12 horas/3 semanas 0,5 mg/kg 12 horas/3 semanas 0,5 mg/kg/24 horas/2 semanas 0,5 mg/kg/48 horas/2 semanas

Conclusiones

- Aunque muchos pacientes pueden beneficiarse de la terapia con AIES no evidencia clinica ni estudios en pequeños animales en muchas patologías
- Mayoría de protocolos en enfermedades neurológicas son

  extrapoladas de medicina humana. No trial en medicina veterinaria
- No usar si posible antes de establecer un diagnóstico
- **Conocer efectos adversos, evaluar riesgos/ beneficios**

PRIMUM NON NOCERUM (FIRST DO NOT HARM)

AS TO DISEASES, MAKE A HABIT OF TWO THINGS, TO HELP OR AT LEAST NOT TO HARM

GRACIAS

PREGUNTAS?