Guidelines on neuro FIP

Prepared by V.Vives DVM

OVERVIEW & PURPOSE

Treatment of urgent Neuro cases according to our experience. Many aspects to be reviewed to produce some guidelines on best practice.

WhAT happens to the brain?

Cerebral damage▸compensatory mechanisms▸decompensation▸increase in IC pressure▸herniation▸death

FIP Related lesions:

- **Ventriculomegaly**
- **Interstitial edema**
- **Foramen magnum herniation (cerebellum)**
- **Cerebellar compression by dilated 4th ventricle**
- **Compression of the interthalamic adhesion**
- **Dorsal compression of brainstem by dilated 4th ventricle**
- **Caudal transtentorial herniation**

Neuro Related symptoms

- **Incoordination, ataxia,difficulty jumping, tetraparesis**
- **Fever**
- **Lethargy**
- **Tremors**
- **Circling**
- **Personality change**
- **Head tilt**
- **Dysphagia, poor appetite, anorexia**
- **Nystagmus**

- **Loss of bladder function**
- **Altered behavior or mental state**
- **Uveitis,Blindness**
- **Seizures**
- **Changes in respiratory pattern**

EMERGENCY PLAN

1. **AIRWAYS**: intubate if needed. Aim for:
    - PO2>=90 mm Hg
    - PaCO2=35-45 mmHg
    - spO2>=95%

2. **BREATHING**: assess respiratory pattern
    - Cheyne-stokes: hyperventilation followed by apnea. Prosencephalic/diencephalic lesion
    - Central neurogenic hyperventilation: regular and mantained hyperventilation as a result of cerebral acidosis, hypoxia, mesencephalic lesions or transtentorial herniation. Midbrain diseased
    - Ataxic respiration: irregular pattern.Brain stem lesion. Poor prognosis
    - Pain/hyperthermia: can produce tachycardia

3. **CIRCULATION**: monitor HR ,BP and check temperature

4. **ASSESS POSTURAL changes**
    - Decerebrate rigidity: opisthotonus, hyperextension of all limbs,mental status: stupor, coma Altered pupillary reflexes 🆘 severe cerebral damage
    - Descerebellate rigidity:: flexion/ extension in hindlimbs +rigidity in forelimbs. Normal mental status +normal pup reflexes

5. **OBTAIN A VENOUS LINE** for fluids+blood sample(PCv, GLUCOSE, TP, BUN, CREA, electrolytes, Blood gases)
    - **Cristaloids(NaCl 0,9%)**
        i. 20 ml/kg bolus (max 60 ml/kg
        ii. They last for 1 h- switch to..
    - **Colloids**
        i. 2-4 ml/kg ev 5-10 min(max 20 ml/kg)
    - **Hypertonic (Nacl 7,5%)**
        i. Use cristaloids first
        ii. 2-4 ml/kg ev 5-10
        iii. Check for dehydration
    - AIM for MAP=80-100 mmHG
    - Keep head elevated @30 ^

6. **CHECK FOR SIGNS Of ELEVATED IC PRESSURE**
    - Caused by cerebral edema or intracranial hemorrhage
    - Leads to herniation
    - Vasogenic— responds to mannitol/hypertonic saline.
      cytotoxic—- responds to **glucocorticoids at O,5 mg/kg**
    - Look for **Cushing's reflex**: bradycardia + systemic hypertension
    - Cerebral edema ➡ miosis ➡mydriasis (unresponsive)
    - **MAKE SURE CAT IS HYDRATED!!**
    - **FRUSEMIDE 0,7 mg/kg before mannitol**
    - **MANNITOL 0,5-1 g/kg bolus given over 20 min. Max 2 g/kg in 24 h**
    - **Can also use HYPERTONIC SALINE if no mannitol available. Very effective to ⬇ ICP**

7. **PERFORM FULL NEURO EXAM**

- **Repeat it every 30-60 min**
- Use the modified Glasgow coma scale for animals

	6	5	4	3	2	1
Motor	Normal gait, normal spinal reflexes	Hemiparesis, tetraparesis or decerebrate activity	Recumbent, intermittent extensor rigidity	Recumbent, constant extensor rigidity	Recumbent, constant extensor rigidity with opisthotonus	Recumbent, hypotonia of muscles, depressed or absent spinal reflexes
Brain stem reflexes	Normal papillary light reflexes (PLRs) and oculocephalic reflexes	Slow PLRs and normal to reduced oculocephalic reflexes	Bilateral unresponsive myosis with normal to reduced oculocephalic reflexes	Pinpoint pupils with reduced to absent oculocephalic reflexes	Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	Bilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes
Level of consciousness	Occasional periods of alertness and responsive to the environment	Depression or delirium, capable of responding, but response may be inappropriate	Semi-comatose, responsive to visual stimuli	Semi-comatose, responsive to auditory stimuli	Semi-comatose, responsive only to repeat noxious stimuli	Comatose, unresponsive to repeated noxious stimuli

A modified coma scale for use in animals

8.**MRI/TC ??????Pros/cons**

ADDITIONAL THERAPIES

- SEIZURES:
  i. Status epilepticus : **DIAZEPAM 0,5-1 mg/kg iv/rectal OR MIDAZOLAM 0,2-0,4 mg/kg im or CRI 0,2-0,4 mg/kg/kr**
  NOTE that Propofol CRI can be used for the first 12-24 h in the cat at 0,1-0,2 mg/kg/min but no longer
  ii. Maintenance: **PHENOBARBITONE start at 2-3 mg/kg bid(Some vets recommend 7,5 mg po bid and then increase by 7,5 mg once daily increments). +-LEVETIRACETAM 20 mg/kg po q 8h**

- **ANTIVIRALS ➡ Antiviral treatment using the adenosine nucleoside analogue GS441524 in cats with clinically diagnosed neurological feline infectious peritonitis Peter J. Dickinson Journal of veterinary Internal Medicine 2020**
  Dr.Pedersen states Gs can reverse signs, if used at higher dose ,within 2 weeks but it is not always curative

INTENSIVE CARE MONITOR

- iF CAT UNABLE TO EAT place a naso-esophagic tube
- Be careful with hypo/hyperthermia
- Express bladder manually
- Control Glucemia(supl. With dextrose if necessary)
- Elevate head 30 degrees
- Aim for SBP 80-120 mmHG
  i. Adjust fluids
  ii. Dopamine 2-10 microg/kg/min
  iii. Amlodipine 0,625-1,25 mg/cat sid
- Treat PAIN
  i. Buprenorphine, Methadone, butorphanol, pethidine

DiFFERENTIALS

Diseases associated with seizures in the cat:

Intra-cranial disorders:

- Neoplasia (e.g. meningioma, lymphoma, metastatic ttumors pituitary tumors, glial tumors, choroid plexus tumors)
- Hippocampal necrosis (although it is unclear whether this is a cause or result of seizure activity)
- Infectious/inflammatory (e.g. FIP, *Cryptococcus,* FIV, FeLV, *Toxoplasmosis, Cuterebra* migration, rabies virus, idiopathic meningoencephalitis, brain abscessation)
- Vascular (e.g. hypertension, idiopathic ischemic encephalopathy, cardiogenic/infectious/tumor emboli, polycythemia)
- Developmental/degenerative (e.g. storage diseases, hydrocephalus)
- Trauma
- Idiopathic/primary epilepsy
- Secondary epilepsy (e.g. post-traumatic, post-ischemic, post-encephalitic)

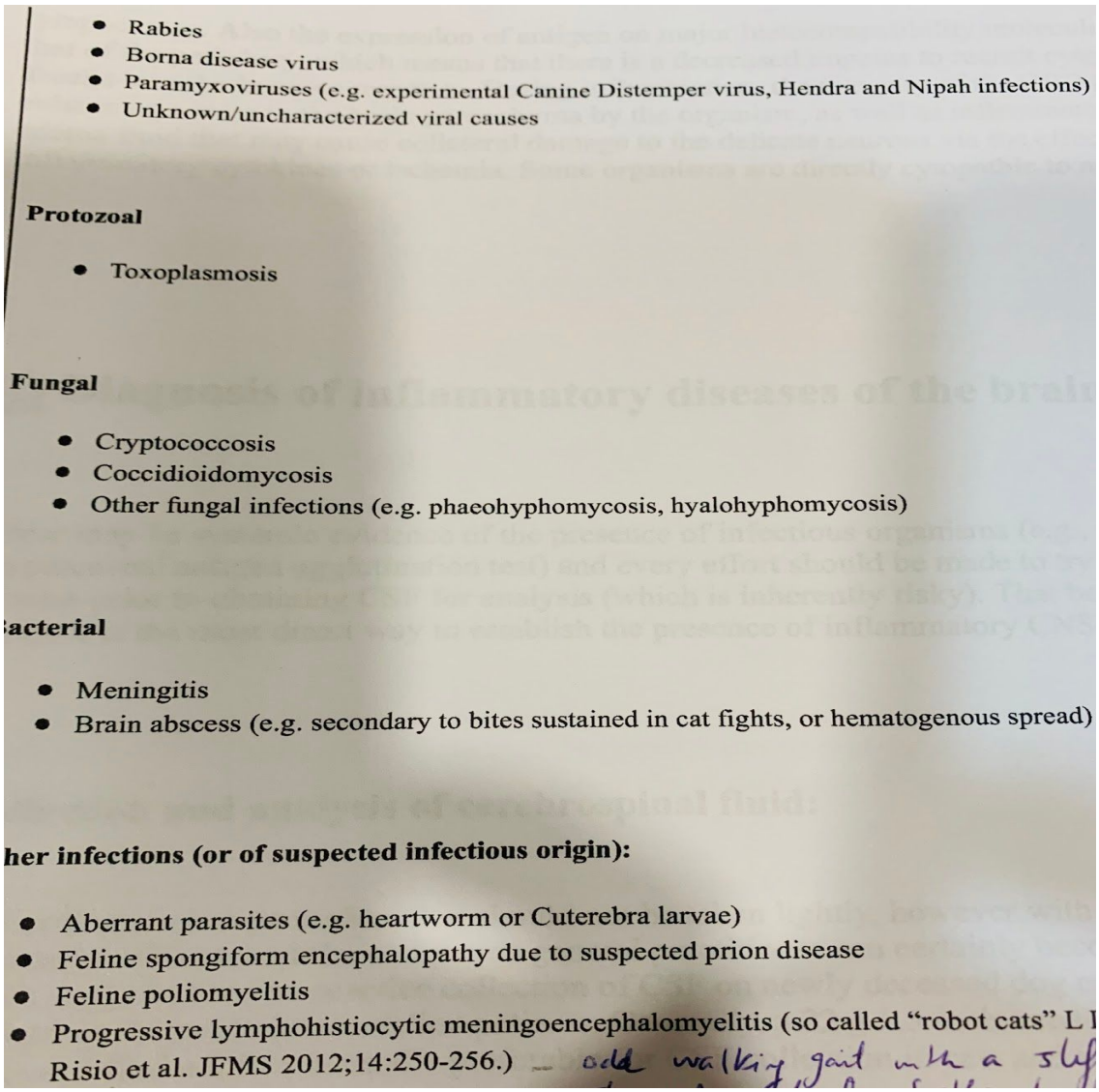
Extra-cranial disorders:

- Hepatic encephalopathy (e.g. due to hepatic lipidosis, portosystemic shunts, liver cirrhosis, neoplasia, cholangiohepatitis)
- Hypoglycemia (e.g. insulin overdose, insulinoma)
- Diabetic ketoacidosis
- Hyperosmolarity
- Hypoxia (e.g. secondary to severe anaemia, pulmonary edema etc.)
- Uremia
- Toxicity (e.g. lead, organophosphates, ethylene glycol,)
- Nutritional (e.g. thiamine or cobalamin deficiency)
- Hyperthyroidism

Infectious/inflammatory causes of cerebral disease in cats:

Viral

- Feline infectious peritonitis (FIP)
- FeLV/FIV



- Rabies
- Borna disease virus
- Paramyxoviruses (e.g. experimental Canine Distemper virus, Hendra and Nipah infections)
- Unknown/uncharacterized viral causes

Protozoal

- Toxoplasmosis

Fungal

- Cryptococcosis
- Coccidioidomycosis
- Other fungal infections (e.g. phaeohyphomycosis, hyalohyphomycosis)

acterial

- Meningitis
- Brain abscess (e.g. secondary to bites sustained in cat fights, or hematogenous spread)

her infections (or of suspected infectious origin):

- Aberrant parasites (e.g. heartworm or Cuterebra larvae)
- Feline spongiform encephalopathy due to suspected prion disease
- Feline poliomyelitis
- Progressive lymphohistiocytic meningoencephalomyelitis (so called "robot cats" L
  Risio et al. JFMS 2012;14:250-256.)

TABLE 1 Neurological differential diagnoses for blindness grouped using the 'Vitamin D' mnemonic

Disease process	Central blindness (ie, forebrain localisation)	Peripheral blindness (ie, retinal, optic nerve and optic chiasm localisation)
Vascular	Brain infarct Brain haemorrhage Feline ischaemic encephalopathy	Ischaemic necrosis of the optic chiasm (secondary to feline ischaemic encephalopathy) Retinal detachment and/or haemorrhage secondary to arterial hypertension
Inflammatory	Infectious encephalitis (viral, protozoal, fungal, bacterial)	Retinitis/chorioretinitis Infectious/non-infectious optic neuritis Osteomyelitis of the sphenoid bone Retrobulbar abscess/cellulitis
Traumatic	Head trauma	Trauma to the globes or orbits
Toxic	Lead poisoning Metronidazole toxicity Global brain ischaemia (post-anaesthetic)	Enrofloxacin toxicity (causing retinal degeneration)
Anomalous	Intracranial arachnoid cyst Porencephaly/hydranencephaly Hydrocephalus	Optic nerve hypoplasia Progressive retinal atrophy
Metabolic	Hypoxia/ischaemia/excitotoxicity (eg, post-ictal) Hepatic encephalopathy Osmotic abnormalities (sodium imbalance) Hypoglycaemia Ketoacidosis Thiamine deficiency	
Neoplastic	Primary or secondary brain tumour	Tumour of, or in vicinity of, optic chiasm (eg, meningioma, pituitary macroadenoma) Neoplasia of the optic nerve or compressing the optic nerve
Degenerative	Feline neuroaxonal dystrophy	

TABLE 2 Some of the more common intracranial causes of blindness and/or behavioural changes in cats

Disease	Onset of clinical signs	MRI appearance and CSF changes	Therapy	Prognosis
Vascular Cerebrovascular accidents	Peracute/acute*	*Infarct:* Intra-axial areas, with minimal mass effect, confined to a specific vascular territory, hyperintense on T2WI, delayed mild CE	Treat the underlying cause; otherwise supportive therapy	Good if no underlying cause identified
		Haemorrhage: Intra- and/or extra-axial with mass effect, variable signal intensity and CE depending on the timing	Medical treatment; surgical therapy in the case of superficial compressive haematoma or failure of medical treatment	Fair to guarded, dependent on severity of clinical signs and response to therapy
Neoplastic Meningioma	Chronic	Intra-axial space-occupying lesion, hyperintense on T2WI, with homogeneous CE	Surgical removal and/or chemotherapy or radiotherapy	Fair
Lymphoma	Variable	Intra/extra-axial ill-defined lesions, with variable mass effect and signal intensity; CSF tap can reveal neoplastic cells	Chemotherapy; radiotherapy possible for a single lesion	Guarded/poor
Inflammatory/ infectious	Subacute/acute	Meningeal/intra-axial single or multiple lesions, with variable mass effect and signal intensity; CSF usually confirms inflammatory aetiology	Immunosuppressive in case of sterile form of unknown aetiology	Guarded/fair
Traumatic Brain trauma	Peracute/acute*	Skull fracture, brain damage (contusion, laceration, etc)	Conservative (anti-oedema, anti-epileptic, to decrease ICP) or surgical	Related to severity of presenting signs
Anomalous eg, Hydrocephalus	First weeks/months to first years of age	Hydrocephalus (dilation of ventricular system)	Conservative (decrease CSF production) or surgical (VPS)	Guarded/fair
Metabolic Thiamine deficiency	Acute/subacute	T2 hyperintensity of caudal colliculi, vestibular nuclei	Vitamin B_1 supplementation	Good
Global brain ischaemia	Strictly related to anaesthesia	Bilateral and symmetrical T2 hyperintense lesions in the cerebral cortex	Supportive treatment	Related to severity of presenting signs
Hepatic encephalopathy	Subacute	Widened sulci, hyperintensity of lentiform nuclei on T1WI	Surgical correction of underlying hepatic disease when possible. Otherwise medical treatment (dietary management, lactulose, etc)	Guarded/fair

have a belated onset. CE = contrast enhancement, ICP = intracranial pressure, VPS = ventriculoperitor