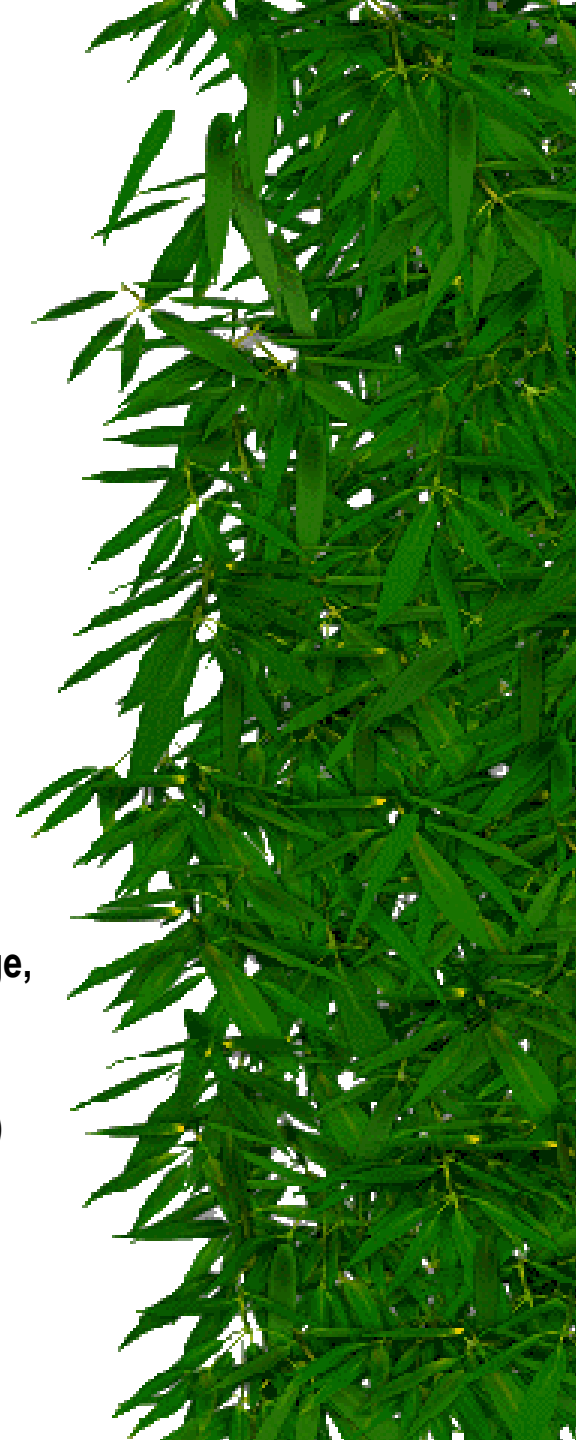
Fibrocartilaginous Embolic Myelopathy (FCEM) in animals



Dr. Sagiv Ben-Yakir

B.Sc. (in Biology, Tel Aviv University, Tel Aviv, Israel)

Doctor of Veterinary Medicine (D.V.M *in honor* from Ontario Veterinary College, University of Guelph, Guelph, Ontario, Canada)

Member of The Royal College of Veterinary Surgeons (M.R.C.V.S)

Certified Veterinary Acupuncturist (CVA by IVAS, San Diego, California, USA)

The Academic College at Wingate Institute, Israel

benyakir@netvision.net.il

FCEM is an occlusion of a few branches of spinal cord blood vessels by fragments of fibrocartilage, believed to arise from an intervertebral disk material. The occlusion results in an ischemia & infarction of the spinal cord.

FCEM occurs most frequently in dogs, but also in cats, horses, pigs, sheep & humans.

FCEM affect any spinal cord segments, but it occurs most frequently in the animal general population at the cd. lumbar area. In cats & sm' dog breeds it occurs more frequently in the cervical spinal cord.

FCEM occurs both in sm'& lg' breeds, more in Miniature Schnauzers, GSD, Labradors, Golden Retrievers & Irish Wolfhounds - young to middle age both female:male 1:1. It occurs more in nonchondrodystrophic breeds, regardless the fact that chondrodystrophic breeds are more affected by Hansen type 1 disk disease.

FCEM has a brief progressive course (a few hrs.) & then it becomes a non-progressive condition.

Although emboli to the CNS can develop from variety of sources such as endocarditis, sepsis and fat – but – the most common form that is causing spinal cord infarction is fibrocartilaginous material that histochemically stains in a manner similar to intervertebral disk's nucleus pulposus. FC material found in arterioles & veins of the meninges & spinal cord results in an ischemic necrotizing myelopathy. Exactly how this material is distributed into the circulation of the spinal cord in not known, but several theories have been proposed. Most of these are based on the belief that FCE originate from the intervertebral disks. The most probable mechanism is herniation of the nucleus pulposus into the body of the vertebrae, followed by entrance into the internal vertabral venous plexus & then into the arteriovenous anastomosis. The material then enters the spinal cord in arteries, veins, or both

Clinical signs(C.S)

The key clinical features of FCEM are an acute onset, a non-progressive course (except the first few hrs.), and a non painful dramatic asymmetric paresis/paralysis without involvement of the head. The c.s appear acutely & progress rapidly within 1-2 hrs. from an initial pain to an unilateral or in some cases a bilateral paralysis. A spinal hyperesthesia may be present at the onset of signs, but it absents after the c.s are stabilized. Trauma is not in the Hx, but dogs might be reported to be exercising at the time of the onset. Asymmetry is not found in every case but is a valuable sign when present

Clinical signs(C.S)

- The asymmetry is explained by the frequency of unilateral branches of the central branch of the vt. spinal artery. Lateralization of signs is very suggestive for FCEM because spinal cord compression generally cause bilateral signs (e.g., IVD herniation).
- Absence of spinal cord hyperesthesia distinguishes FCEM from lesions w/compression such as Hansen type 1 IVDD, neoplasia, or vertebral fractures & disease processes in which there is inflammation such as meningomyelitis

Diagnosis

Based on Hx, C.S & exclusion of other causes.
No definitive antemortem Dx procedure exists for FCAM.

Dx is supported by evidence that rules out the presence of spinal cord compression, fractures and alike.

In FCEM - survey radiography & myelography findings are within normal limits with some slight swelling of spinal cord at the very first hrs. CSF – might show some slight increase in protein in the 1st day or so. In MRI – focal, intramedullary, hyperintense lesions with varying degrees of contrast enhancement may be seen in a segment of the spinal cord overlying an IVD in which the nucleus pulposus has undergone degenerative changes resulting in a loss of signal intensity. Also, – thyroid and hypertension should be evaluated as it might predispose to CNS vascular occlusion & infarction

Treatment

- Corticosteroids might reduce spinal cord edema & inflammation – however there is no a significant association w/cortico' administration and the outcome. The issue is a controversial one.
- Rest and physical rehabilitation

Prognosis

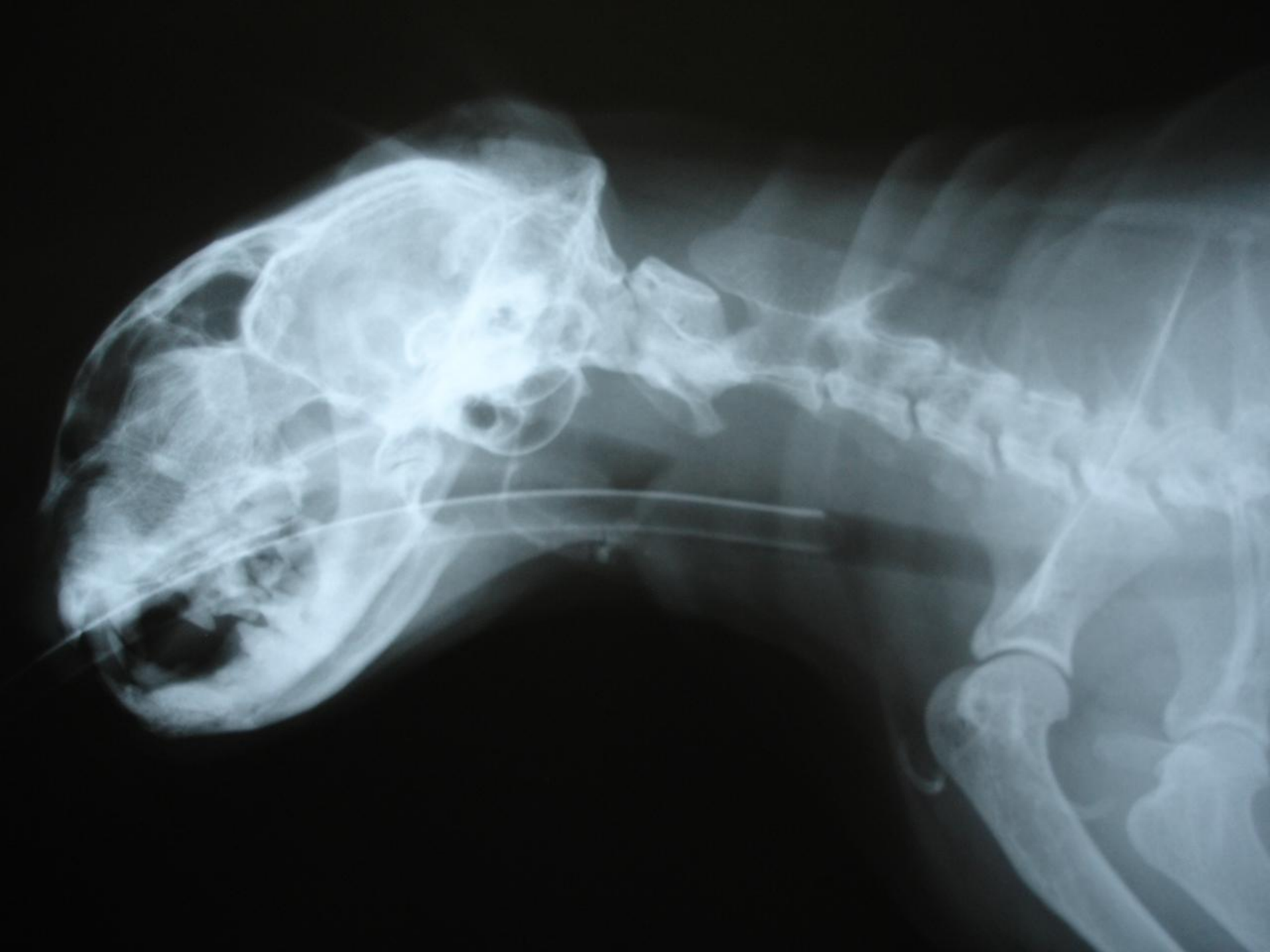
Poor prognosis.

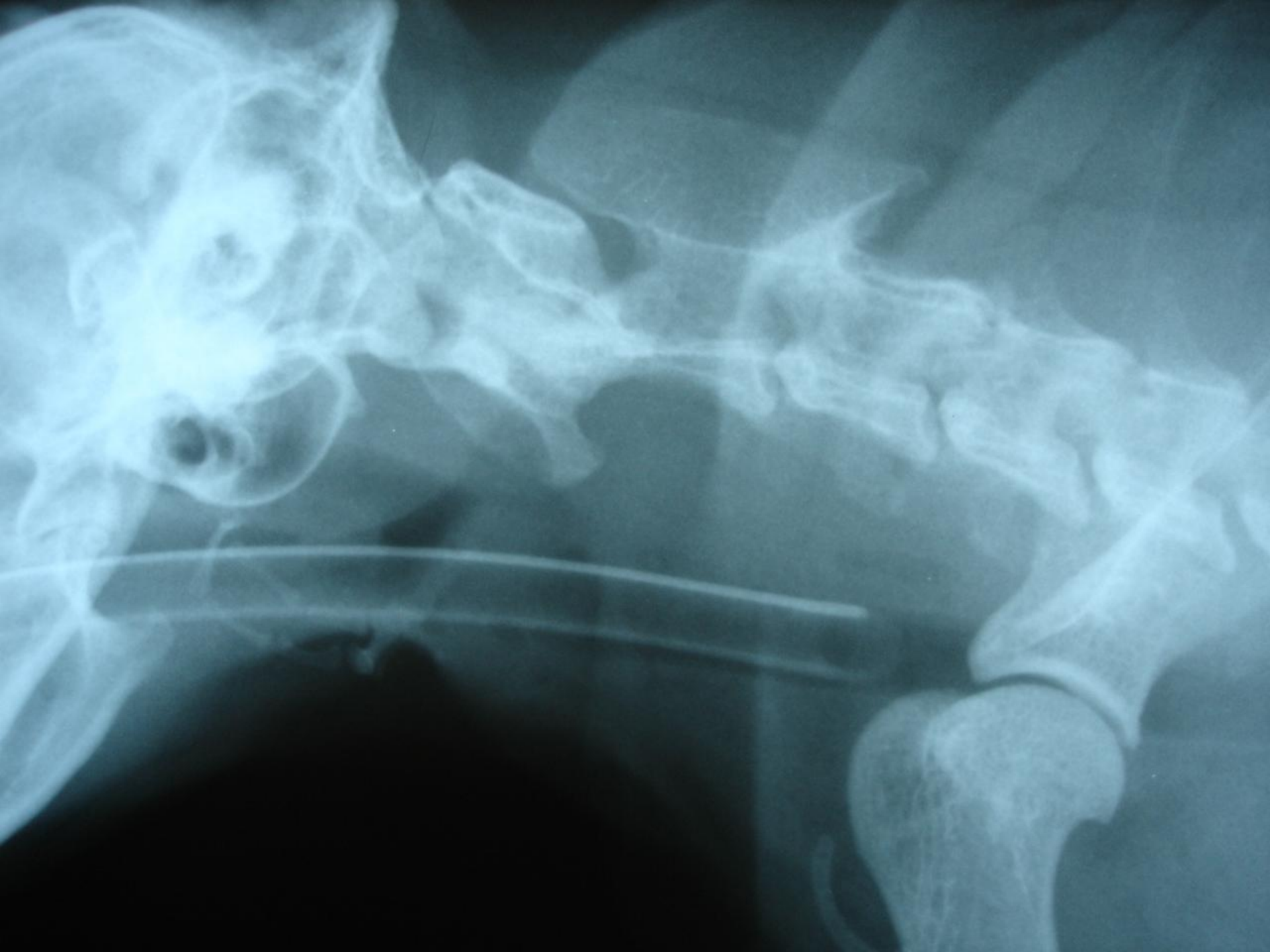
Functional recovery if happens requires months

A feline FCEM case

Hx: 9 yrs. old castrated DSH tabby&white cat was presented to our clinic with no ability to walk neither support its wt. for the last few hrs. (at least 6 hrs.)

Px: 5.8 kg. a very nervous cat with hemi-paralysis of rt side. LMN of rt. thoracic leg, UMN of rt. pelvic leg with rt. Horner's syndrome (Horner's syndrome = miosis, ptosis, enophthalmos w/elevation of nictating membrane/3rd eyelid)





HOD-HASHARON VET CLI

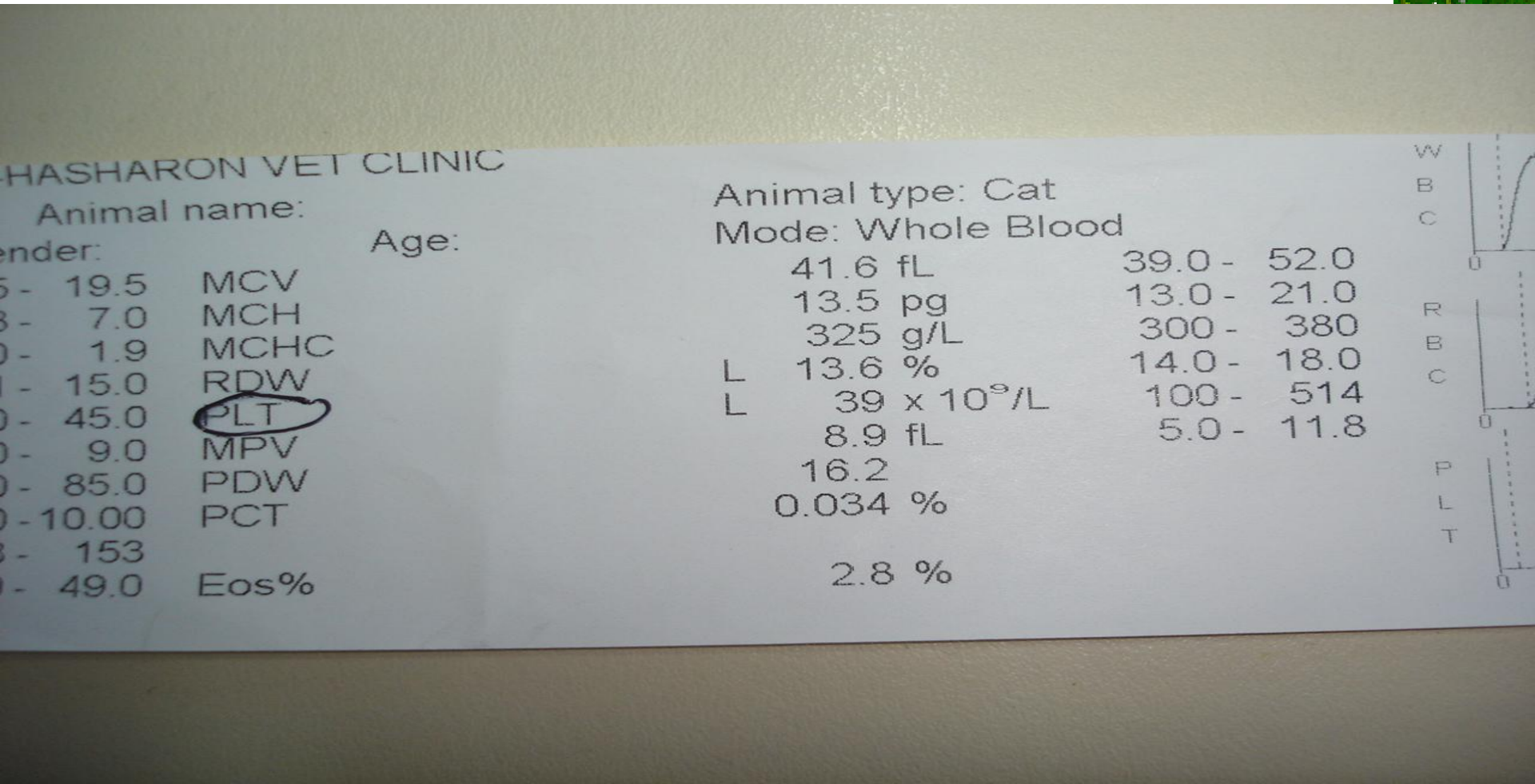
Owner's name: Animal name:

ID: 12345678 Time: 01-31-2013 00:32 Gender: Age

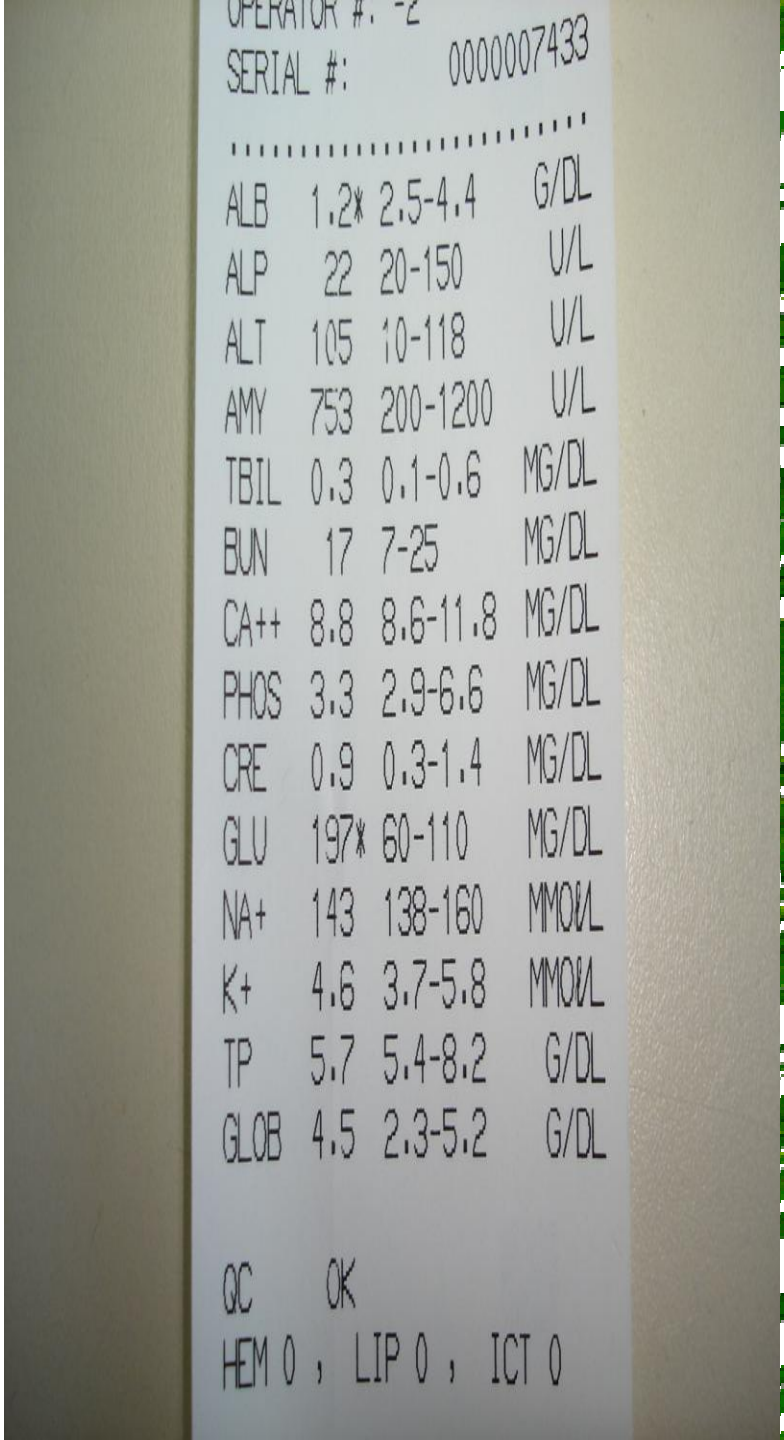
WBC		8.1 x 10^9/L	5.5 - 19.5	MCV
Lymph#		3.4 x 10^9/L	0.8 - 7.0	MCH
Mon#		0.4 x 10^9/L	0.0 - 1.9	MCHC
Gran#		4.3 x 10^9/L	2.1 - 15.0	RDW
Lymph%		42.2 %	12.0 - 45.0	PLT
Mon%		5.4 %	2.0 - 9.0	MPV
Gran%		52.4 %	35.0 - 85.0	PDW
RBC	L	2.96 x 10^{12}/L	4.60 - 10.00	PCT
HGB	L	40 g/L	93 - 153	
HCT	L	12.3 %	28.0 - 49.0	Eos%

Bld smear - Hemobartonella

T4 - normal



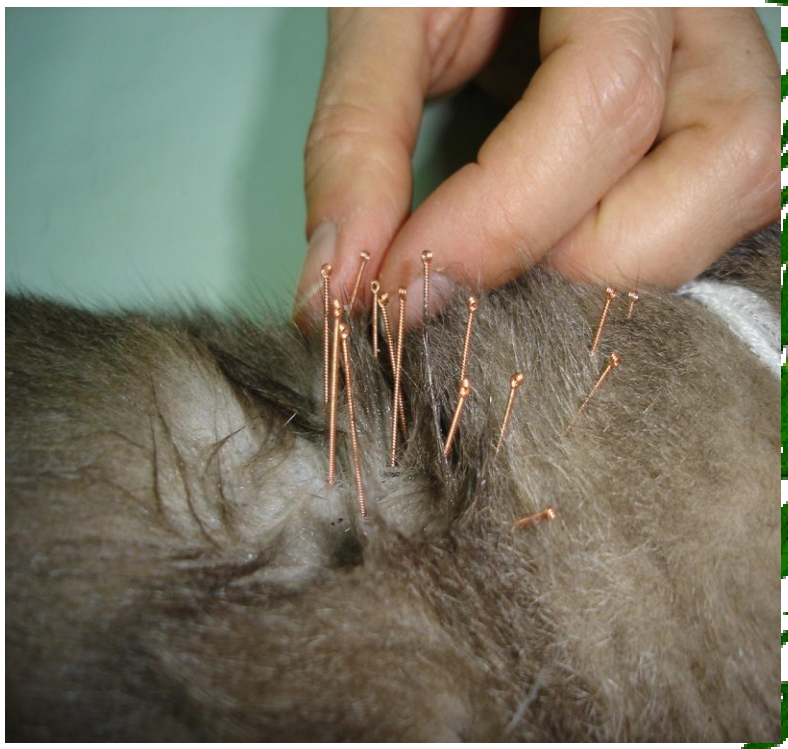
We begun Rx with doxycycline i.v followed by p.o

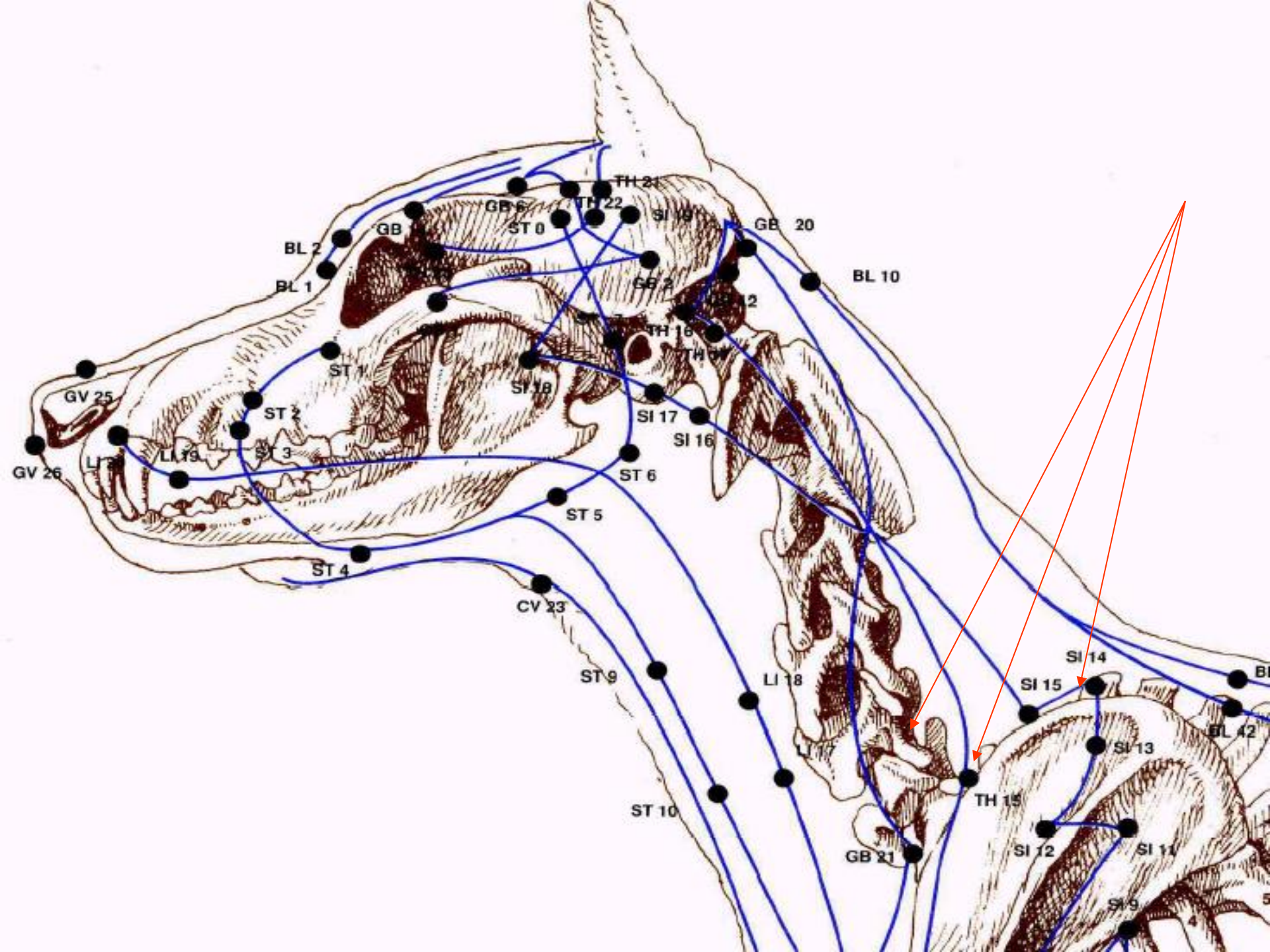


And acupuncture -

Due to the total picture of c.s the lesion was determined to be between C-07 to T-02, = extended **BL-11**

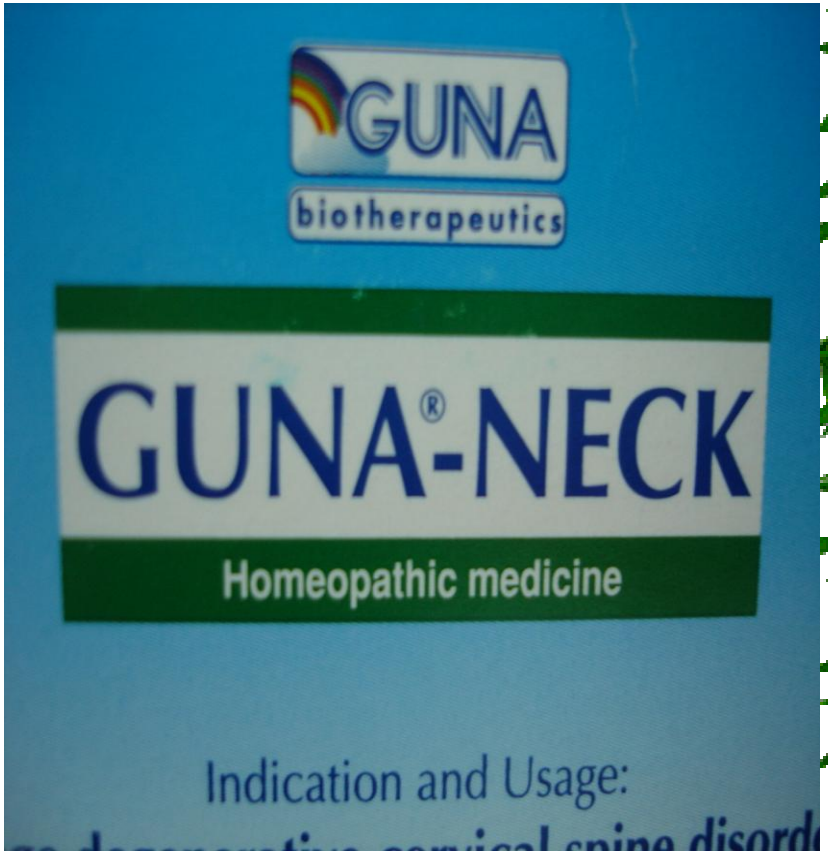
Chinese needles inserted locally till hit the bone (25 mm w/dia 0.22 mm) at the area of BL-11




TH 21
GB 6
TH 22
SI 19
GB 20
GB
ST 0
BL 2
BL 1
BL 10
GB 2
TH 16
ST 1
SI 18
GV 25
ST 2
SI 17
SI 16
GV 26
LI
LI 19
ST 3
ST 6
ST 5
ST 4
CV 23
ST 9
LI 18
SI 14
SI 15
BL 42
SI 13
LI
TH 15
ST 10
GB 21
SI 12
SI 11
SI 9

Followed by injection of -

"GUNA NECK" –
A Sterile Homeopathic Remedy diluted in 0.9 % Saline to treat neck maladies



Another pt for Rx

Owner is doing tid extension & flexion of all jts at rt fore&hind leg

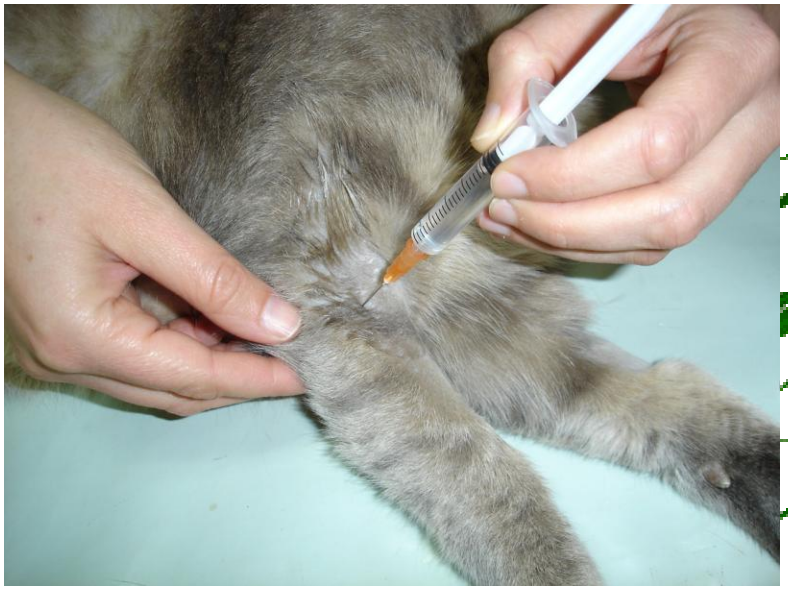
4 days later

Since LMN – modification of Rx – performing acupuncture at pts. of rt. foreleg

Followed by injection of -

“GUNA HANDFOOT”

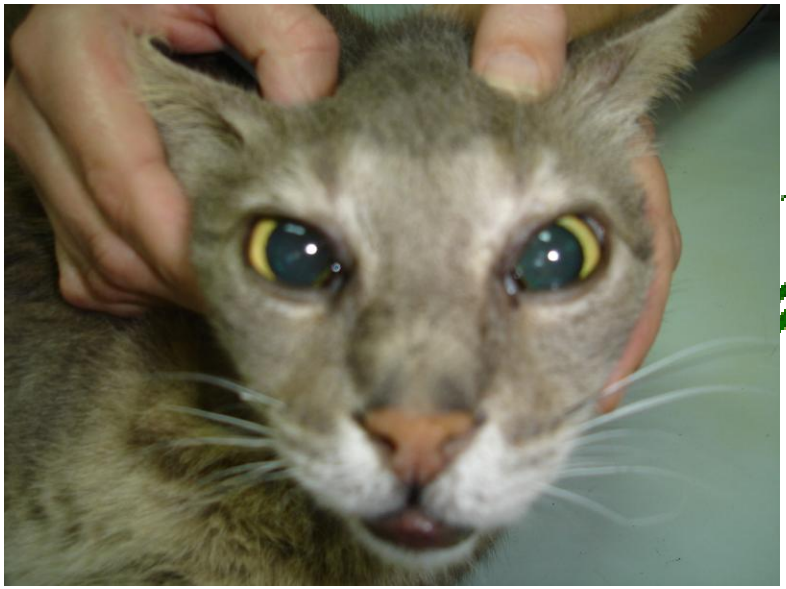
A Sterile Homeopathic Remedy diluted in 0.9 % Saline built to Rx hand/foot maladies



And a few days more -

More -

- Complete control on urine & feces
- No Hemobartonella parasites in bld smear
- No more Horner’s syndrome



Over the last 6 yrs. -

We had five different cases of FCEM –

we applied the same protocol with the same clinical result – return to a complete function