

BEPSS

BELGIAN EQUINE PRACTITIONERS SOCIETY

**40th Scientific Meeting
of the Belgian Equine Practitioners Society
Saturday, December 2nd, 2023**


Dechra *zoetis*
Veterinary Products

 **Boehringer
Ingelheim**

Auditorium BMW2
Onderwijs & Navorsing 2, Campus Gasthuisberg, Herestraat 49, B-3000 Leuven

Bureau – Bestuur

President : Dr. Emmanuelle Van Erck

Secretary: Prof. L. Vlaminck & Dr. I. Tosi

Treasurer : Dr. S. Grulke

Board Members :

Prof. F. Pille

Prof. G. Van Loon

Dr. J. Desmedt

Dr. Barbara Van Waerebeek

Dr. K. Vanschandevijl

Dr. M. Devos

Dr. P. Paindavaine

Dr. I. Caudron

Website : <http://www.beps-be.org>

<p style="text-align: center;">PROGRAMMA / PROGRAMME BEPS 2023</p>
--

09:00 – 12:00 MORNING SESSION
IN DEPTH EQUINE DERMATOLOGY - PROF. DR. DEREK KNOTTENBELT

- Official opening by the president, Dr. Emmanuelle Van Erck
- The dermatological examination
- Diagnostic tests in equine dermatology
- Update on Equine Sarcoids

13:30 – 17:00 AFTERNOON SESSION

- President's word
DR. EMMANUELLE VAN ERCK
- Update equine focus point belgium
DR. ANNICK GRYSPEERDT
- Dermatology: Pastern dermatitis – What's in a name
PROF. DR. DEREK KNOTTENBELT
- Practical guidelines to examine the equine eye
DR. ELINE VERCRUYSSSE
- What does it mean when gamma-GT is too high?
DR. EMMANUELLE VAN ERCK
- PSSM in horses: what is it?
DR. IRENE TOSI
- Update on West-Nile virus
DR. CARLA CESARINI
- Cyst-like lesions – what the heck???
DR. FILIP VANDENBERGHE / PROF. FREDERIK PILLE

CLOSING DRINK

Morning Session

Equine Dermatology

Prof. Dr. Derek Knottenbelt



1

Why is it important to ensure clinical excellence?

- ❑ Veterinarians in practice are expected to 'make a diagnosis' many times a day, so **the diagnostic process** is vital to clinical excellence
- ❑ We need to identify the factors limiting our ability to establish the **correct diagnosis and devise strategies to limit them and so limit the implications of "getting it wrong"**
- ❑ Studies have shown that uncertain diagnoses or mistakes in diagnosis are made in about **50%** of all medical primary care consultations. We are likely to be worse!
- ❑ An **incorrect diagnosis** is made in around **1 in 5 consultations**
 - ❑ This may lead to serious harm and biases treatment toward failure
 - ❑ Guesswork is responsible for significant morbidity and even mortality.
- ❑ There is a serious need to learn about the causes of incorrect diagnoses and what clinicians can do to **mitigate the risk of patient harm** from these events
- ❑ This presentation will help you to understand **how a diagnosis can be reached in almost all cases both rationally, sensibly and economically**

2



3

Clinical excellence is vital!

- ❑ For every mistake that is made by not knowing **100** are made by not trying
- ❑ Professional responsibilities are a duty
- ❑ Try not to make mistakes....
- ❑ Try hard to keep up with your knowledge....
- ❑ Admit your mistakes- they are part of our learning....
 - ❑ Try to avoid making them twice..... or three times!

EXCELLENCE IS NOT A SKILL. IT IS AN ATTITUDE.

It takes **Time** to create excellence. If it could be done quickly, more people would do it.
- John Wooden

4

CHOICES....

- ❑ Mediocrity V Excellence
- ❑ Professional V Amateur
- ❑ Caring V Uncaring / Casual
- ❑ Communicative V Silence / Mystery

You are free to make whatever choice you want, but you are not free from the consequences of the choice.

5

You can only devise and use rational treatment when you KNOW what you are dealing with....

- ❑ A meticulous clinical approach to **every case** will ...
 - ✓ Improve your reputation in the profession and the public
 - ✓ Lead to better clinical outcomes
 - ✓ Lead to better use of time and effort
 - ✓ Create more rational medical / surgical therapy
 - ✓ Provide strong job satisfaction / stimulation

6

The diagnostic ladder

- ❑ LOGIC IS THE KEY FACTOR
- ❑ IT SHOULD BE POSSIBLE TO ACHIEVE A DIAGNOSIS IN ALMOST EVERY CASE
- ❑ TIME IS ESSENTIAL
- ❑ SHORT CUTS LEAD TO ERRORS and BAD OUTCOMES
- ❑ RELIANCE ON LUCK AND GUESSWORK → DISASTER!



7

You cannot climb the mountain without a carefully planned METHOD and meticulous execution

- ❑ Use staged logic and aim to limit mistakes



8

Only when you reach the top can you REALLY know the satisfaction of high achievement

- ❑ The diagnostic process leads to a final conclusion
- 1. A definitive diagnosis
- 2. Appropriate and logical treatment
- 3. Evidence based prognosis



9

What would YOU prefer to be called to...



10

Reasons for Dermatologic Consultation

1. Overt skin disease
 - ❑ Concerned owner
2. Skin disease in systemic disease
 - ❑ owner more worried about other signs?
3. Pre-purchase examination



Different approaches required

11

Categorising Skin Disease

1. PRURITUS
2. PAIN
3. DRY DERMATOSIS (flaking / scaling)
4. WET DERMATOSIS (crusting / eczema)
5. PIGMENT CHANGES
6. ALOPECIA & HAIR QUALITY CHANGES
7. NODULES / LUMPS

GET IT RIGHT!
Combinations
Separate entities

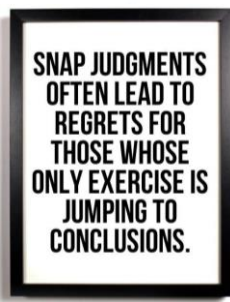
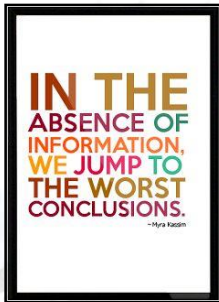


Rudyard Kipling, 1865-1936



*"I have six honest serving men,
they taught me all I knew
Their names are WHAT and WHY and WHEN
and HOW and WHERE and WHO"*

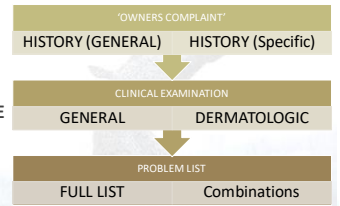
12



13

Objectives

- TO REACH A DIAGNOSIS!
- APPLY LOGIC and ROUTINE



14

It's small... it's slow... it's benign...

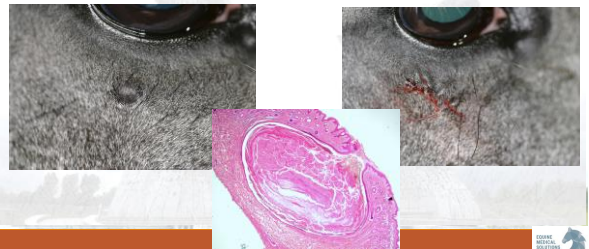
- BUT IS IT BAD?!



15

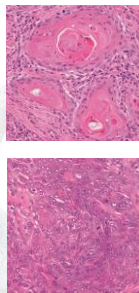
Once we know what it is....

- We can make informed decisions!



16

Or this....

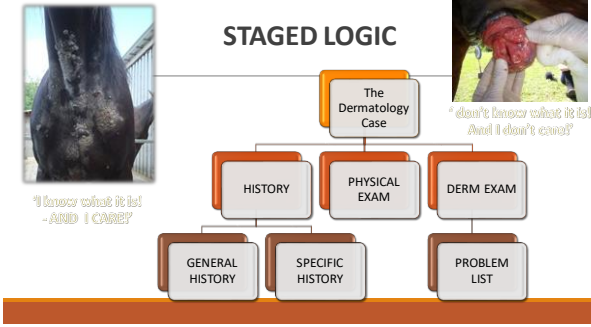


17

But then... what's this?



18



19

Signalment

- Breed
 - Breed specific diseases
- Sex
 - Sex specific disease
 - Diseases related to breeding
- Age
 - Congenital / hereditary disorders
 - May be manifest later
 - Geriatric disease
- Colour
 - Grey
 - Cream / non pigmented horses




20

Why does age matter?

- Congenital diseases**
 - Genetic / hereditary disease
 - Present at birth
 - Delayed onset
- Old age / geriatric disease**
 - Cushing's Disease
 - > 15 years
 - Melanoma
 - > 5 years





22



23



24



25

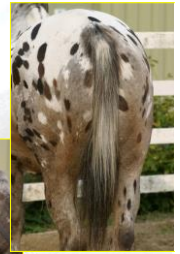


26

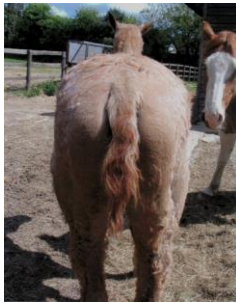
Why does breed matter?

Specific breed-related disease

- Appaloosa
 - Fading syndrome
 - Mane and Tail dystrophy
- Quarterhorse
 - HERDA SYNDROME



27



28



29



30

Sex matters too!

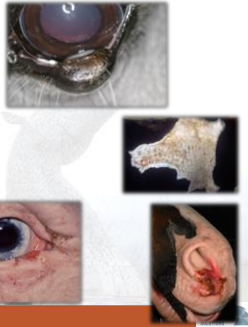
- Squamous cell carcinoma
 - Mares / geldings
 - VERY rare in entire stallions
- INFECTIONS



31

What about colour?

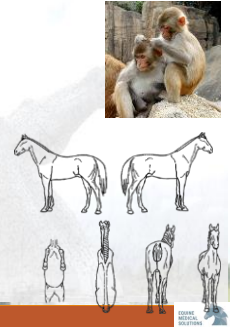
- ❑ Colour related diseases
- ❑ Colour-related predispositions
- ❑ Colour-related neoplasia



32

History & Clinical Examination

- ❑ Prepared sheet
 - ❑ History
 - ❑ Clinical examination
 - ❑ Follow up
- ❑ Nothing gets missed out!
 - ❑ Accurate record for follow-up
 - ❑ Failures/success/treatment effects
 - ❑ Accurate record for a colleague
 - ❑ Accurate record for referral centre



33

Owners Complaint History / Anamnesis

- ❑ Onset of problem
- ❑ Changes
 - ❑ Better / worse
 - ❑ Circumstances
- ❑ Treatment attempts
 - ❑ Type / duration
 - ❑ Results



34

Disease / Treatment History

- ❑ Previous disease
 - ❑ Dermatologic
 - ❑ Other system
- ❑ Therapeutic attempts
- ❑ Concurrent treatment(s)
- ❑ Routine medications
 - ❑ Wormers / Vaccines etc.



35

Management

- ❑ Stabling / Pasture?
- ❑ Feed types/quantity
- ❑ Exposure to infectious agents
- ❑ Work/harness / training



36



37

Don't forget the physical examination!

- ❑ Vital signs
 - ❑ (T/P/HR/RR/mm/CRT/WT/BC)
- ❑ All body systems
 - ❑ Alimentary / Respiratory / Cardiovascular / Neurological / Endocrine / Urinary / Genital / Musculoskeletal / Special senses
- ❑ Integumentary system (detailed)
 1. Concurrent disease
 2. Cutaneous disease with systemic implication
 3. Systemic disease with cutaneous implication



38

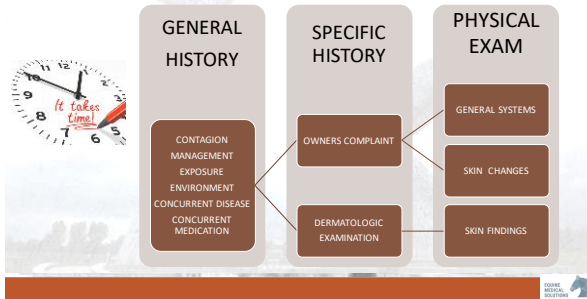
Specific Dermatological Examination

- ❑ Distribution of lesions
- ❑ Type of lesions
 - ❑ Configuration
 - ❑ Solitary / multiple
 - ❑ Confluent / grouped
 - ❑ Annular linear / serpiginous / arciform
 - ❑ Variable in some conditions
- ❑ Extent / severity of lesions
- ❑ Changes with treatment
- ❑ Establish **primary lesion / sign**

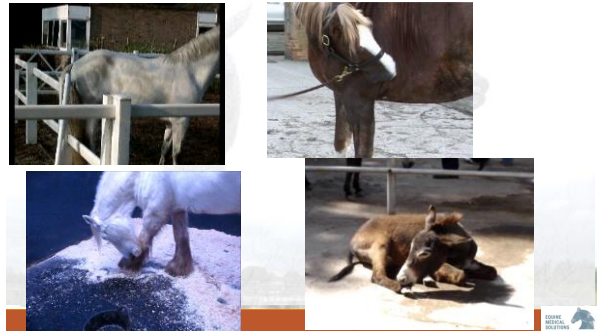


39

THE PROBLEM LIST



40



41

The problem list ...

- ❑ ALL FINDINGS *no matter how trivial*
- ❑ LIST
- ❑ TRY TO COMBINE findings → potential differentials

When I'm feeling sick, I google my symptoms and usually find out that I have cancer.

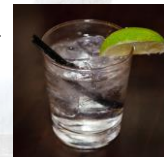


good things take time.

42

Differential Diagnosis List

- ❑ Combinations of problems
- ❑ Multiple possibilities
 - ❑ Independent
 - ❑ Related
- ❑ Use a system and get used to it!
 - ❑ IS IT INFECTIOUS?
 - ❑ Contagious? Are others affected also?
 - ❑ IS IT NON-INFECTIOUS



It will hurt.
It will take time.
It will require dedication.
It will require willpower.
You will have to keep working.
It requires sacrifice.
There will be temptation.
But I promise you, when you reach your goal, it's worth it.

43

Differential Diagnosis

- ☐ It's ALL already in your best reference source!
- ☐ Available 24/7
 - ☐ ... no electricity source required....
- ☐ Almost every dermatological problem can be solved
 - ☐ That does not mean its treatable of course!

"For every mistake that is made by not 'knowing' 99 are made by NOT looking/asking"

IS YOUR
EYE/BRAIN
SWITCH OFF
OR
ON..?



44

A system for reaching a sound Differential List

INFECTIOUS DISEASE		NON-INFECTIOUS DISEASE	
VIRAL		B	Genetic
BACTERIAL		I	Immunologic
FUNGAL		N	Nutritional
PROTOZOAN		A	Allergic
PARASITIC		N	Neurologic
<ul style="list-style-type: none"> • Ectoparasitic • Suckers! • Chewers! • Endoparasitic • Luminal <ul style="list-style-type: none"> • Gut • Airway • Tissues / blood 		D	Developmental
		T	Traumatic
		O	Oromal (!!!)
		N	Neoplastic
		I	Iatrogenic / Idiopathic
		C	Chemical / Toxic
		C	Cardiovascular

45

So let's have a poll!
This is a sarcoid!

1. TRUE
2. FALSE
3. ABSTENTION / FENCE SITTER



46

INTUITIVE SUPPOSITION

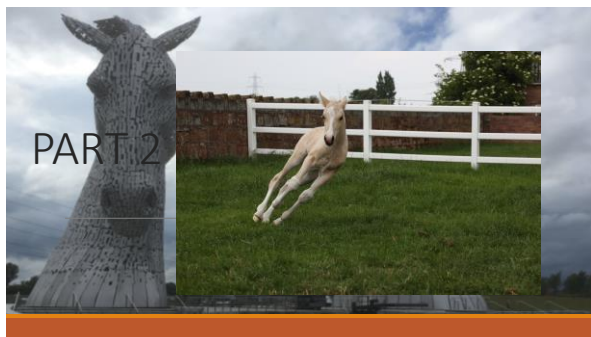
FAST
SEEN IT BEFORE
I ONLY KNOW ONE THING SO THIS IS IT FOR SURE
THEY ALL NEED THE SAME TREATMENT SO IT DOES NOT MATTER

LOGIC AND DETAIL

TIME CONSUMING
SATISFYING
FEE JUSTIFYING
REPUTATION BUILDER

THINK TWICE BEFORE YOU DO SOMETHING YOU MIGHT REGRET

47



52

Diagnostic Tests for Skin Disease

Diagnostic Tests for Skin Disease

Derek C Knottenbelt

53

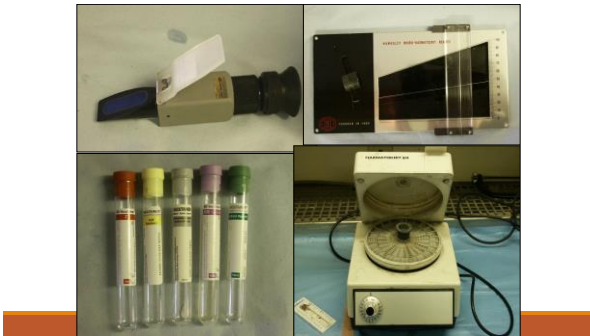


54

Sample Selection

- ☐ Samples for different diseases
 - ☐ Presumptive diagnosis
 - ☐ Diagnostic confirmation
 - ☐ Diagnostic "rule-out"
- ☐ Specimens
 - ☐ Skin tissues / scraping / groomings
 - ☐ Blood
 - ☐ Haematology / biochemistry / serology
 - ☐ Biopsy
 - ☐ Diagnostic microbiology

55



56

Skin Scrapings

Mites / other ectoparasites

- *Chorioptes equi*

Not useful in horses?

- Burrowing mites
- Dermatophytes

Hair pluckings / brushings better

Method:

- 22 scalpel blade
- Areas shaved directly into sterile containers
- Moisten liq paraffin?
- Examine directly
- DEMODEX sp. needs deep scraping
- **Blood**

57

Skin groomings

Very useful

Ectoparasites

- Lice
- Mites

Dissecting microscope useful

Method:

Use denture brush & Petri dish

Groom into pot

Examine directly

58

You may need sharp eyes...
... but you don't need much equipment!

59



60



61



62

Hair Plucking



- Easily obtained
- Edges are better
- Artery forceps
- Examine accordingly
- Dermatophytes

Method:

- Wipe with spirit?
- Hairs from edge of "young" lesions
- Place in a sterile pot
- Repeat 4 – 6 x
- Submit for tests
- PCR / CULTURE/ MICROSCOPY

63



64



65

Acetate / Adhesive Tape Preparations

Oxyuris equi mostly
Lice/ lice eggs (nits)
Occasional mites

4 cm length of acetate tape stuck onto
skin (perineal)

Clip haired areas short (not shaven)

Apply to slide directly + 1-2 drops of
liquid paraffin



66



67



68

Skin /Lesion Washings / Direct Smear & Swabs

Bacterial species detection

Stain

Fungal detection

Extraction with KOH

Stains

Parasite detection

Habronema

Strongyloides westerii

Cytological examination

Impression smears

Pathological recognition not easy?



69

Skin /Lesion Washings / Direct Smear/ Swabs

Bacterial species detection

Stain

Fungal detection

Extraction with KOH

Stains

Parasite detection

Habronema

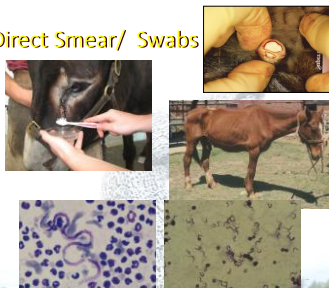
Strongyloides westerii

Cytological examination

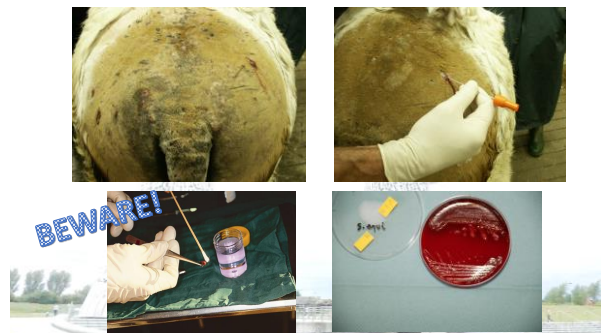
Impression smears

Capillary blood smear

Pathological recognition not easy?



70



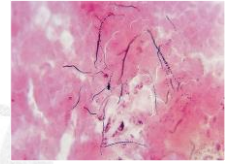
71



72

Specialized Techniques

- ❑ *Dermatophilus congolensis*
- ❑ Direct smear onto saline drop
 - ❑ Heat fix / dry and stain
- ❑ Culture needs microaerophilic conditions
 - ❑ Candle in bottle technique
 - ❑ Consult with lab first
 - ❑ PCR?

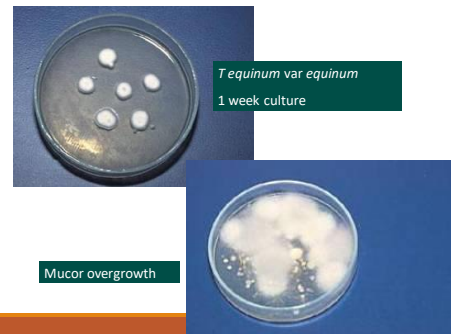


73

Specialized Techniques

- ❑ Dermatophytosis
- ❑ Samples pushed gently into Sabourauds medium (*Fungassay*)
 - ❑ Some need a specialized culture
 - ❑ Slow results (c. 10 – 14 days possible)
 - ❑ Indicator methods faster
- ❑ Potassium hydroxide 10%
 - ❑ Warm ! DO NOT BOIL!!
- ❑ Stain directly
 - ❑ Lactophenol cotton blue

74



75



76

Skin Biopsy

- ❑ Establish diagnosis
- ❑ Eliminate defined clinical conditions
- ❑ Monitor course of disease
- ❑ Confirm excision of tumours etc.
- ❑ Limitations:
 - ❑ Not good for chronic / complicated lesions (S-I-T etc.)
 - ❑ Single biopsy seldom answers all four!



77

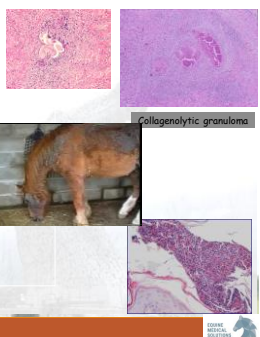


ONLY if it is going to alter what you do

78

Conditions for which biopsy is potentially useful

- ❑ Autoimmune disease (pemphigus etc)
 - ❑ Specific staining / appearance
- ❑ Granuloma (bacterial / fungal / parasites)
- ❑ Neoplasia
- ❑ Parasitic disease ? (Habronema / Onchocerca)
- ❑ Infections (section / swab for culture)
- ❑ Specific diseases (amyloid / collagen necrosis +)



79

Conditions where biopsy is useful

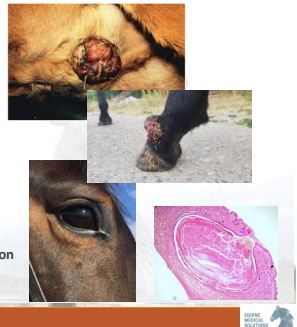
- Autoimmune disease
 - Specific staining / appearance
- Granuloma (bacterial / fungal / parasites)
- Neoplasia
- Parasitic disease ? (Habronema / Onchocerca)
- Infections (section / swab for culture)
- Specific diseases (amyloid / collagen necrosis +)



80

Types of Biopsy

- ❑ Fine needle aspiration
- ❑ Shave
 - ❑ Shave off epidermis - needs no sutures
- ❑ Punch
 - ❑ 6/8/9 mm punch: CAREFUL; no sutures
- ❑ Wedge
 - ❑ Larger lesions; 'Cake slice' (direction)
- ❑ Excisional
 - ❑ Normal / abnormal skin - whole (?) lesion
 - ❑ Very useful for bullae etc



81

Fine Needle Aspiration

- ❑ Technique matters
- ❑ Skilled cytopathologist essential



82



83

TruCut (Hollow) Needle Biopsy

- Manual operation
 - TruCut
- Semi automatic
 - Cook UK
- Automatic (gas driven) ('Biopty')



84



85



86



87



88



89



90

Biopsy

- Specimen handling
 - Use scalpel NOT scissors
 - Use needle not forceps
 - Large samples must be cut
 - Label and draw for pathologist
 - Pin flat samples to card
 - Correct orientation
- Remember to take microbiology samples before interference (formalin!)

BEWARE!

91

Biopsy

- **Fixatives**
 1. Formal saline
 - Preferably buffered
 2. Michel's medium
 3. Glutaraldehyde
- Always > 10x sample size
- Suitable container

93

Biopsy Interpretation

- PROVIDE AS MUCH INFORMATION AS YOU CAN!
- DO NOT expect the pathologist to make a diagnosis for you...
- Try to understand the limitations and tell the pathologist what you suspect – send history, clinical information and pictures!

94

- **KEY POINTS**
 1. USE BIOPSY SYSTEMS REGULARLY BUT CAREFULLY
 2. SELECT THE RIGHT TYPE OF BIOPSY
 3. SELECT THE RIGHT REGION
 4. COLLECT REPRESENTATIVE SPECIMENS
 5. CONSIDER MULTIPLE BIOPSY
 6. PREPARE APPROPRIATELY
 7. USE APPROPRIATE INSTRUMENTS

97

Other Techniques

- Immunohistochemistry
 - Specialised pathologist / lab
- Skin allergy testing / Patch Testing?
 - Intradermal allergens
 - Histamine control +ve
 - Pure sterile saline control -ve
 - Major interpretive problems
- IgE blood sampling?

98

Other diagnostic tests...?

- Ultrasonography
- Rectal examination
- Radiography / CT
- Bone marrow biopsy



99

Other diagnostic 'techniques'

- Intuitive supposition
- Detailed history
- Careful clinical investigation
- Environmental examination
- Other organs e.g. liver / gut



100

Objectives

1. ALWAYS EXAMINE THE WHOLE HORSE!
2. Establish full **PROBLEM LIST**
 - Primary condition
 - Secondary changes
3. Establish **DIFFERENTIAL DIAGNOSIS**
 - Order of likelihood!
 - **Don't completely ignore the unusual**
4. Select **FURTHER TESTS**
5. Establish **DIAGNOSIS**
6. Define **PROGNOSIS**
7. Select **TREATMENT**
8. Monitor **PROGRESS**



101

Remember!

- Diagnosis may not be easily established
- Further tests often required
 - Select carefully with full objectivity!
 - **MUST** be focussed!
 - Pathology will not help if poor clinical is done!
- Repeat examinations
- Responses to treatment



102

The Hoof

- Part of the skin
- Chestnuts / ergots also important
- Often gives historical information
 - Altered horn / keratin
 - 'Growth' bands
- Solear structures
 - Thrush
 - Canker etc.



103

Remember

- Always
 - Consider other animals
 - Consider management
 - Enquire carefully on what has been done before result it produced



104



105



106



1

Sample Selection

- Samples for different diseases
 - Presumptive diagnosis
 - Diagnostic confirmation
 - Diagnostic "rule-out"
- Specimens
 - Skin tissues / scraping / groomings
 - Blood
 - Haematology / biochemistry / serology
 - **CAN HELP DETECT SYSTEMIC DISEASE**
 - Biopsy
 - Diagnostic microbiology

2

Skin Scrapings

- Mites / other ectoparasites
 - *Chorioptes equi*
- Not useful in horses?
 - Burrowing mites
 - Dermatophytes
- Hair pluckings / brushings better

• Method:

- 22 scalpel blade
- Areas shaved directly into sterile containers
- Moistened liq paraffin ?
- Examine directly
- DEMODEX sp. need deep scraping
- Blood

3

Skin groomings

- Very useful
- ectoparasites
 - Lice
 - Mites
- Dissecting microscope useful

• Method:

- Use denture brush & Petri dish
- Groom into pot
- Examine directly

4

You may need sharp eyes!

5

6

Hair Plucking

- Easily obtained
- Edges are better
- Artery forceps
- Examine accordingly
- Dermatophytes

Method:

- Wipe with spirit?
- Hairs from edge of "young" lesions
- Place in sterile pot
- Repeat 4 – 6 x
- Submit for tests
 - Direct microscopy / KOH
 - CULTURE / PCR



7

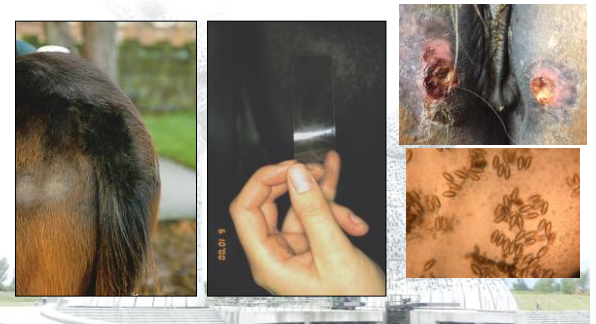


8

Acetate / Adhesive Tape Preparations

- *Oxyuris equi* mostly
- Lice/ lice eggs (nits)
- Occasional mites

- 4 cm length of sellotape stuck onto skin (perineal)
- Clip haired areas short (not shaven)
- Apply to slide directly + 1-2 drops of liquid paraffin

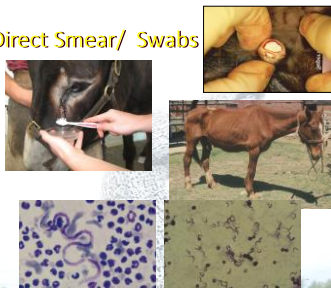


9

10

Skin /Lesion Washings / Direct Smear/ Swabs

- Bacterial species detection
 - Stain
- Fungal detection
 - Extraction with KOH
 - Stains
- Parasite detection
 - *Habronema*
 - *Strongyloides westerii*
- Cytological examination
 - Impression smears
 - Capillary blood smear
 - Pathological recognition not easy?

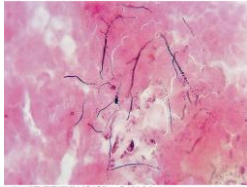


11

12

Specialized Techniques

- *Dermatophilus congolensis*
- Direct smear onto saline drop
 - Heat fix / dry and stain
- Culture needs microaerophilic conditions
 - Candle in bottle technique
 - Consult with lab first



13

Specialized Techniques

- Dermatophytosis
- Samples pushed gently into Sabourauds medium (*Fungassay*)
 - Some need specialized culture
 - Slow results (c. 10 – 14 days possible)
 - Indicator methods faster
- Potassium hydroxide: 10%
 - Warm ! DO NOT BOIL!!
- Stain directly
 - Lactophenol cotton blue
- **PCR – FAST AND EASY**



Mucor overgrowth

14

Skin Biopsy

- ✓ Establish diagnosis
- ✓ Eliminate defined clinical conditions
- ✓ Monitor course of disease
- ✓ Confirm excision of tumours etc.

Limitations:

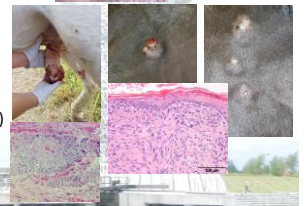
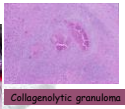
- Not good for chronic / complicated lesions (S-I-T etc.)
- Single biopsy seldom answers all four!



15

Conditions where biopsy is useful

- Autoimmune disease
 - Specific staining / appearance
- Granuloma (bacterial / fungal / parasites)
- Neoplasia
- Parasitic disease ? (*Habronema* / *Onchocerca*)
- Infections (section / swab for culture)
- Specific diseases (amyloid / collagen necrosis *)



16

Fine Needle Aspiration

- Technique matters
- Skilled cytopathologist essential



17

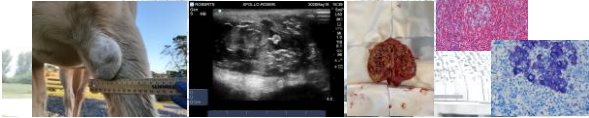


ONLY if it is going to alter what you do

19

Biopsy Interpretation

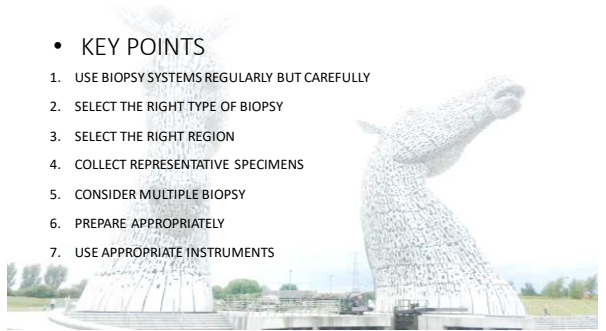
- PROVIDE AS MUCH INFORMATION AS YOU CAN!
- DO NOT expect the pathologist to make a diagnosis for you...
- Try to understand the limitations and tell the pathologist what you suspect – send history, clinical information and pictures!



22

KEY POINTS

1. USE BIOPSY SYSTEMS REGULARLY BUT CAREFULLY
2. SELECT THE RIGHT TYPE OF BIOPSY
3. SELECT THE RIGHT REGION
4. COLLECT REPRESENTATIVE SPECIMENS
5. CONSIDER MULTIPLE BIOPSY
6. PREPARE APPROPRIATELY
7. USE APPROPRIATE INSTRUMENTS



23

Other Techniques

- Immunohistochemistry
 - Specialised pathologist / lab
- Skin allergy testing / Patch Testing?
 - Intradermal allergens
 - Histamine control +ve
 - Pure sterile saline control -ve
 - Major interpretive problems
- IgE blood sampling?



24

Other diagnostic tests...?

- Ultrasonography
- Rectal examination
- Radiography / CT
- Bone marrow biopsy



25

Other diagnostic 'techniques'

- Intuitive supposition
 - Detailed history
 - Careful clinical investigation
 - Environmental examination
 - Other organs e.g. liver / gut



26

Objectives

1. ALWAYS EXAMINE THE WHOLE HORSE!
2. Establish full **PROBLEM LIST**
 - Primary condition
 - Secondary changes
3. Establish **DIFFERENTIAL DIAGNOSIS**
 - Order of likelihood!
 - Don't completely ignore the unusual
4. Select **FURTHER TESTS**
5. Establish **DIAGNOSIS**
6. Define **PROGNOSIS**
7. Select **TREATMENT**
8. Monitor **PROGRESS**



27



**'Fill what's empty, empty what's full,
and scratch where it itches.'**

Duchess of Windsor, when asked for the secret of a long and happy life



Derek C Knottenbelt

EQUINE
MEDICAL
SOLUTIONS
office@equinesarcoid.co.uk
www.equinesarcoid.co.uk

1

What is "Pruritus"



"Unpleasant sensation provoking the desire to scratch / itch"

- Common / important skin sign
- Many causes
- Single presenting sign



2

Pruritus is NOT...

- Attempts to relieve discomfort
- Response to focal or generalised pain
- "Worry" from occasional "visitors"!
- Displacement activity unrelated to skin



BUT

Sometimes very difficult to differentiate

3

Pruritus

- Severity varies
 - No common severity for defined causes
- Localised responses
 - Local causes
- Generalised responses
 - Generalised / systemic causes



4



5

I have six honest serving me,
they taught me all I knew
Their names are **WHAT** and **WHY** and **WHEN**
and **HOW** and **WHERE** and **WHO**



Rudyard Kipling, 1865-1936

6

Clinical Objectives

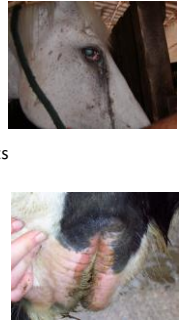
1. DIFFERENTIATE FROM SIMILAR SIGNS!
 - Eliminate other conditions such as worry / psychosis / PAIN
2. IDENTIFY THE CAUSE!
3. STOP THE PRURITUS!
 - a) Limit self trauma / damage
 - b) Prevent further secondary infection / infestation
4. PREVENT RECURRENCE



7

Logical approach

1. Take a history
2. Perform a clinical examination
3. Create a problem list
4. Establish a differential list
5. Select suitable differentiating tests
 - Rule in / Rule out
6. Define diagnosis
7. Apply treatment
8. ASSESS RESULTS CRITICALLY
 - Successful?
 - NOT SUCCESSFUL?



8

Step 1

- GET A REALLY GOOD HISTORY
 - Onset / duration
 - Circumstances
 - INSIDE / OUTSIDE
 - Other horses affected?
 - Other animals present?
- Concurrent signs
- Therapeutic measures / effects
- Peers and contacts!



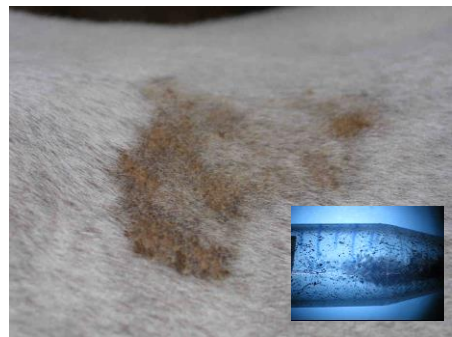
9



10



11



12



13

WHAT has been done already?

- Most owners have done something!
 - Misguided treatment (or it would have worked!)
 - Bought a 'Boett Blanket' at great expense
 - Chosen to ignore it
 - Undertaken management alterations



14



What measures has the owner tried?

- Parasiticides?
- Antibiotics?
- Antifungal?
- Steroids?
- Antihistamines?

Use the information
... don't criticise it!

15

Making the diagnosis....

"Only when you know what you are dealing with can you make sensible therapeutic decisions"

Stannard, 1991



16

Step 2
Clinical Investigation

- Identify all the signs
 - Beware concurrent systemic disease
- CONSIDER ALL DIAGNOSTIC POSSIBILITIES!
 - Eliminate by specific questions / tests
- Remember:
 - Some cases are secondary to systemic illness
 - Some cases are due to very serious disease



17

Watch the horse!

- **WHY** does it itch?
- **WHEN** does it itch?
- **WHERE** does it itch?
- **HOW** does it itch?
- **WHO / WHAT** is responsible



18

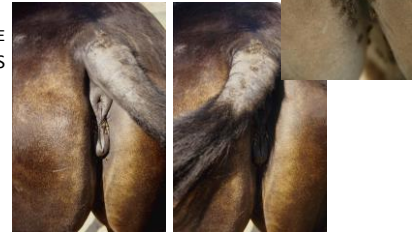


19



WHEN DOES IT ITCH?

- Circumstances when its better / worse
 - Seasonality?
 - INSIDE V OUTSIDE
- SPECIFIC FACTORS
 - FOODS
 - CLOTHING
 - WASHES
 - ENVIRONMENT
- GEOGRAPHY?

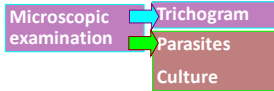


20

Step 3

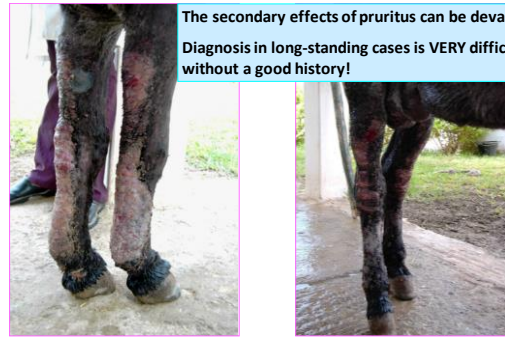
Perform further clinical investigations

- Brushings
 - Useful
- Scrapings
 - Little value
- Biopsy
 - Beware self-inflicted trauma
- Blood / Skin 'Allergy' Tests
 - Confusing? / possibly useful!
- Test therapy
 - Steroids / antihistamines



21

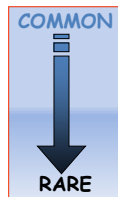
The secondary effects of pruritus can be devastating!
 Diagnosis in long-standing cases is VERY difficult without a good history!



22

Aetiology

1. Parasitic infestation
2. Allergies/Immune conditions
3. Inflammatory disease
4. Infections
5. Systemic illness

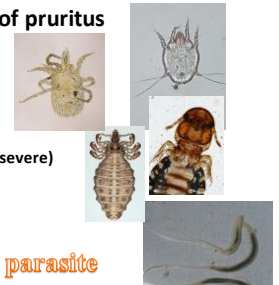


INVESTIGATE & Eliminate in sequence!

23

Parasitic infestation is commonest overall cause of pruritus

- External parasites
 - Mites (moderate – severe)
 - Lice (Mild)
 - Insect worry / bites (moderate –severe)
- Internal parasites
 - Intestinal parasites (localised)



Pruritus at the site of the parasite

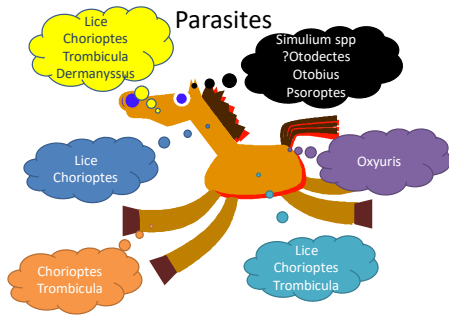
24



25



26



27



28



**... BUT SOME ARE EASIER TO IDENTIFY
- MAY NOT BE EASY TO TREAT!**

29

Generalised Pruritus

- **Persistent**
 - Paraneoplastic pruritus
 - Hepato-cutaneous syndrome
 - MsEED
- **Intermittent**
 - Seasonality
 - Insect bite hypersensitivity
 - Insect worry
 - Parasitic infestation (lice?)



30



31



32

Focal Pruritus

- One site or few but consistent / unvarying
 - Neurologic 'pruritus'
 - Spinal fracture
 - Headshaker
 - Rabies
 - Infections
 - Dermatophytosis / Deep mycosis
 - Habronema
 - One site at a time – variable intensity
 - Insect bite hypersensitivity
 - Atopy
 - MsEED
 - Insect worry / stings
 - Parasites (Oxyuris equi)



Courtesy M Sloet



34

Stop the pruritus.... Cure the problem....

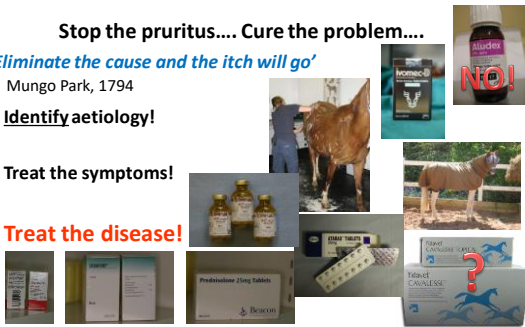
'Eliminate the cause and the itch will go'

Mungo Park, 1794

• **Identify aetiology!**

• **Treat the symptoms!**

• **Treat the disease!**



35



36

Some comments on *Oxyuris equi*

- Increasing problem
 - Not “detected” by worm egg counts
 - Stable acquired infestation (HYGIENE)
 - Strategic worming takes no account of it
 - Avermectins have poor efficacy
 - High dose pyrantel / fenbendazole / albendazole
 - Piperazine
 - Levamisole
 - “Resistance”?
- Bum wipes?
- Rectal wormers?



37



38

BUT.....

- Perineal pruritus?
 - Lice
 - Mites
 - Food hypersensitivity
 - Proctitis
 - Intermammary irritation



39

Summary

- Pruritus is an important clinical sign
- Most are parasitic
 - Differentiate worry from pruritus
- Neurologic causes can be extremely serious
- Fungal infections often mildly pruritic
- Non-infectious causes are important
 - Hypersensitivity (Sweet Itch)
 - Atopy
 - Paraneoplastic pruritus
 - Hepatocutaneous Syndrome



**BE CAREFUL
BE METICULOUS
CONSIDER ALL POSSIBILITIES**

40



41

What is a sarcoid?

DEREK C KNOTTENBELT

EQUINE MEDICAL SOLUTIONS

www.equinesarcoid.co.uk

App: Equine Medical Solutions

EQUINE MEDICAL SOLUTIONS

1

What is a Sarcoid?

- Common [skin] tumour of equidae
 - "VIRAL induced" fibroblast tumour
 - IT'S CANCER!
- Worldwide incidence
 - 2-10% of horses
- Clinical spectrum
 - Different phenotypes
- Pathological spectrum
 - Wide range of terminology!
 - Adds to problems

EQUINE MEDICAL SOLUTIONS

2

Why is the sarcoid important?

- Genuine neoplasm
 - Not .."JUST a virus disease"
 - IT'S CANCER!
- Welfare issue
- Affects usefulness of the horse
- Affects the commercial value
- Treatment costs are always significant
- PROGNOSIS IS GUARDED - ALWAYS

EQUINE MEDICAL SOLUTIONS

3

What "causes" the disease?

- Overwhelming evidence for a BPV1 / 3 relationship
- No consistent / convincing vegetative virus has been found yet
- BPV causes "pseudo-sarcoid"
 - Rapid proliferative change BUT rapid "self cure" and seroconversion
- Affected horses do not spontaneously seroconvert to BPV
- Vaccination against BPV does not prevent sarcoid
- Genetic susceptibility genes may be important
 - Familial / breed susceptibility varies

EQUINE MEDICAL SOLUTIONS

4

The BPV_{1/4} conundrum

EQUINE MEDICAL SOLUTIONS

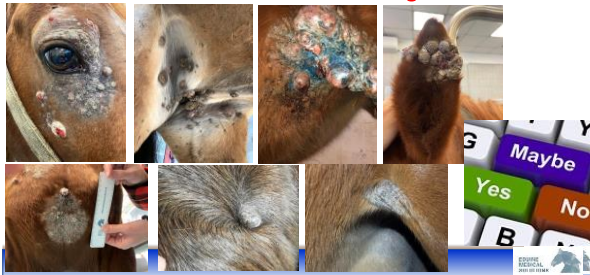
5

Is this the same as the bovine viral papilloma? Is this a 'papilloma' at all?

EQUINE MEDICAL SOLUTIONS

6

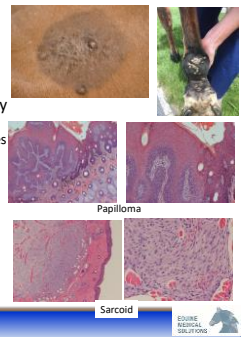
Can we justify a diagnosis of viral disease here?
Can we attribute this to a virus 'blowing on the wind'?



7

Interesting sarcoid facts

- Changes in keratinocyte behaviour are probably secondary
 - Sarcoid is a tumour of fibroblasts NOT keratinocytes
 - Mediator driven epidermal hyperplasia / atrophy
- **Circular appearance almost always at first!**
 - Diffusion from central active area
 - Not always the most obvious proliferative site!
 - Gene mediator expression is a likely factor
 - Not a patent virus response
- PV's → no primary effect on fibroblasts



10

Does the disease progress or is each type spontaneous?

- Progression is the norm!
 - Natural progression over days / weeks / months / decades
 - SEVERITY V EXTENT V SPEED
- SLOW → FAST → variable
- TRAUMA REF → invariable
- Interf →



13

Progression?



14

Progression ...

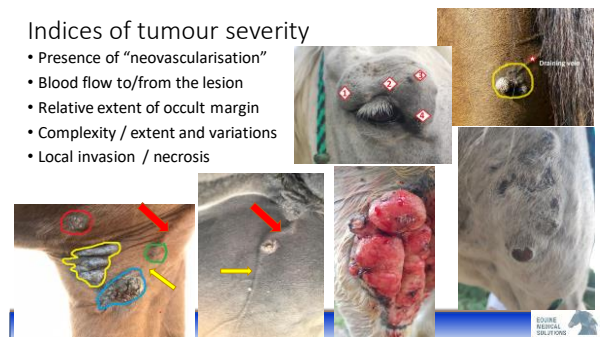
- It is almost invariable!
- NOT inevitable – can remain static for many months / years
- Unpredictable triggers and variable rates of progression
- Ill-advised interference makes it **faster / worse** and **less treatable!**



15

Indices of tumour severity

- Presence of "neovascularisation"
- Blood flow to/from the lesion
- Relative extent of occult margin
- Complexity / extent and variations
- Local invasion / necrosis



16

If the disease is so important what are the principles of diagnosis?

- CLINICAL FEATURES ARE PARAMOUNT
- MULTIPLE SARCOIDS MAKE IT EASIER
 - **BEWARE OF CO-MORBIDITY**
- Pathologic diagnosis
 - Definitive histology?
 - Cutaneous involvement?
 - Immunohistochemistry
- Surface swabs – BPV PCR
 - Probably not safe - ? helpful
 - **Also +ve on a wide variety of inflammatory /neoplastic lesions**



17

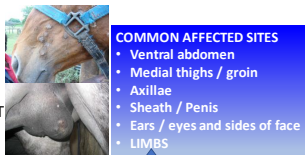
'I have been told it's unsafe to biopsy'



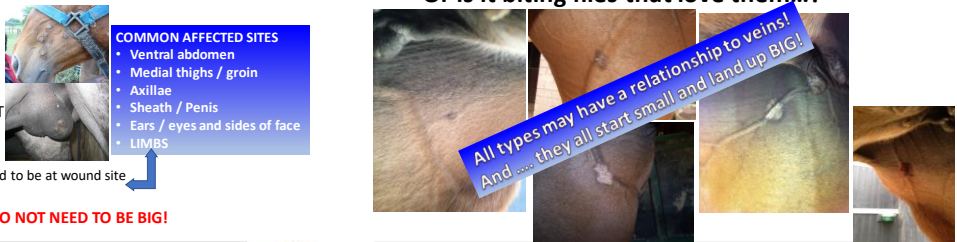
18

Location – are there truly 'preferred' sites?

- THIN SKINNED REGIONS
- GLABROUS/ THINLY HAired SKIN
- SWEAT
- FLY ATTACK WITHOUT RESENTMENT
 - SEASONALITY OF ONSET?
- WOUND SITES
 - Sarcoids on distal limb can be assumed to be at wound site
- BITING FLY SITES



REMEMBER WOUNDS DO NOT NEED TO BE BIG!



25

26

BUT ... this has to be differentiated from NEOVASCULARISATION – implication?

1. Incidental relationship to vessels?
2. Obstructed vessels?
3. Enlarged draining vessels ?
 - Aggressive growth rate of tumour but no new vessels
4. Neovascularisation?
 - Aggressive growth rate and
 - NEW VESSELS / ABNORMAL VESSELS



27

Anatomic location is a critical factor...

- **Anatomic considerations**
 - What is involved?
 - How does this impact on therapeutic options
- **Pathologic behaviour**
 - Subtle differences in same regions
 - BIG differences in different regions
- Therapeutic options
- Logistics
- etc....



28

Transmissibility

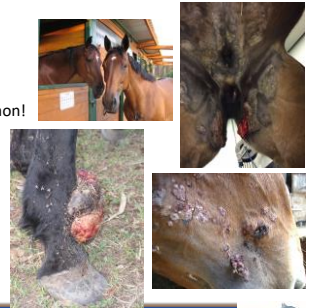
- Flies?
 - Virus
 - Cell transmission
 - Virus infection
 - Mechanisms for *de novo* sarcoids
 - Immunologic aspects
 - MHC status
- F-F-F-S**
- Beware of fly feeding frenzy sites



29

TRANSMISSION

- Horse to horse transmission IS common!
 - Factors that allow this to occur?
 - MHC related self-protection
 - MHC receptor blocking by BPV
- No need for patent virus infection?
- VECTOR DRIVEN
 - SUMMER / FLY SEASON ONLY
- Is there another explanation?



30

Are flies involved in transmission?

- BITING FLIES?
 - [Hogezlager M et al., The possible role of Stomoxys calcitrans in equine sarcoid transmission. Vet J. 2019 Jun;231\(6\):12. doi: 10.1016/j.tvjl.2017.11.009. Epub 2017 Nov 22](#)
 - ... obligate blood feeder
 - ... but biting flies do not feed on sarcoid... e
 - ... how did they become PCR positive?
- SURFACE FEEDING FLIES?
 - Musca spp., Haematobia spp
 - ALWAYS prefer sarcoid!
 - Prefer wound sites too!
- BOTH TOGETHER?



31



32



33

How can we explain this.... ?

- Does it fit a virus infection 'blowing around in the wind'?
- The first lesion identified 12 months previously was the red-outlined area
- 12 months later in spring the new lesions became progressively more obvious and by late summer the lesion is as shown here!



35

Differential Diagnosis



- Each type has its own set of differentials to consider
 - NOT always easy to tell the difference but its **IMPORTANT** to know what it is
- **CLINICAL ASSESSMENT IS ESSENTIAL**



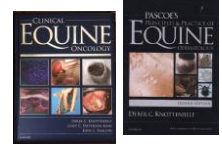
Very common cause of litigation!

36

Classification matters



- Common description between professionals
 - We understand each other and can communicate properly
- We can compare results and treatments
- We can be more scientific in publications
 - We can get better evidence based information
- We can recognise changes for better / worse



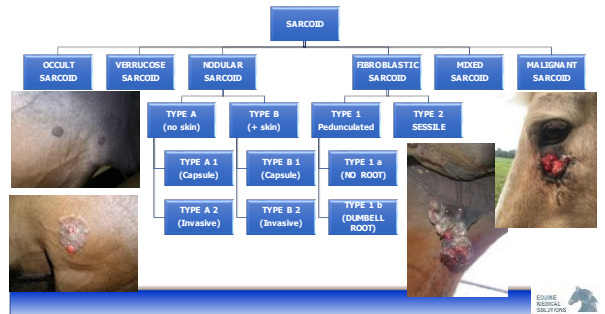
Knottenbelt DC (2005) A Clinical Classification of the Equine Sarcoid. *Clinical Techniques in Equine Practice* 4: 278-295

37

Treatment choices vary
.... so classification matters



38



39

Problems ?



- Continuum rather than defined category boundaries
 - Biological systems seldom have well defined categoric boundaries!
- Most if not all sarcoids are 'mixed' to some extent
- **PREDOMINATE TYPE IS USED**
- 'Mixed Sarcoid' only used if no dominant type is present
- Variations in every type and subtype
- **UNDERSTANDING IS IMPORTANT!**

40

Occult sarcoid

- 'Earliest' / mildest form
 - Alopecia (CIRCULAR?)
 - Epidermal hyperplasia
 - Hyperkeratinisation → scaling / flaking
- Easily overlooked
- Easily mistaken for other conditions
 1. Rubs
 2. Alopecia areata
 3. Dermatophytosis
 4. Pemphigus foliaceus ... etc



41



★ Pre-purchase litigation cases

42

Why the occult zones?

- Is it.....
 1. Part of the tumor itself?
 2. The true extent of the lesion
 3. Incidental skin changes
 4. PARACRINE EFFECTS relating to BPV genomic expression



43

Occult sarcoid doesn't matter TRUE OR FALSE?

- It is **IMPORTANT** and must not be disregarded
- Its an early warning of doom!



EQUINE MEDICAL SOLUTIONS

44

The Verrucose Sarcoid

- 'Wart'-like
- Ill-defined margins
- Variable extent
- Alopecia
- Epidermal hyperplasia
- Heavy flaking / scaling
- Differentials include
 - Pemphigus / Papilloma / Epidermal naevus / Ringworm etc



EQUINE MEDICAL SOLUTIONS

45

The diagnostic halo!



46

It's not a wart!



EQUINE MEDICAL SOLUTIONS

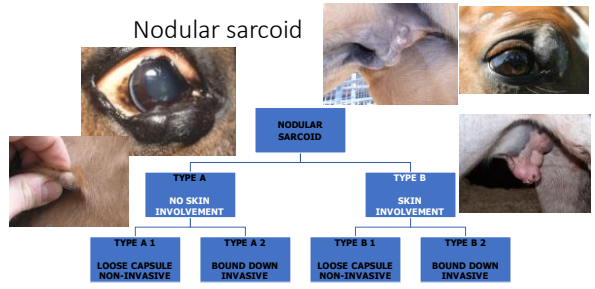
47

They can get very extensive....
Therapeutic implications?



48

Nodular sarcoid



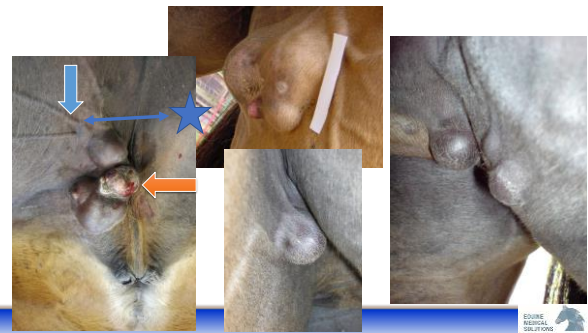
49

Key facts about nodular sarcoids....

- IDENTIFY TYPE!
 - MAKES A SIGNIFICANT DIFFERENCE TO TREATMENT / PROGNOSIS
- CAN BE SOLITARY or MULTIPLE
 - Variations on same horse
 - Nesting nodules
- ULCERATION IS COMMON
 - SKIN ATROPHY (TYPE A)
 - SKIN DESTRUCTION (TYPE B)
- TYPES A2 / B2 can have extensive root development



50



51

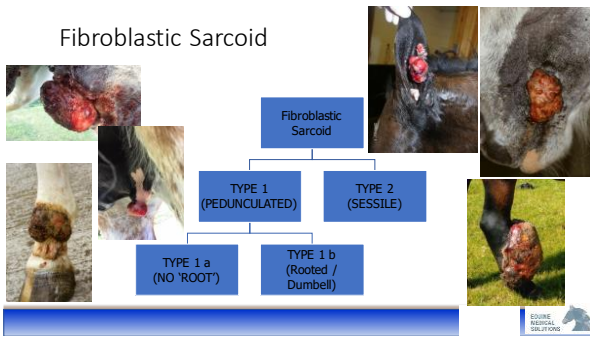


52



53

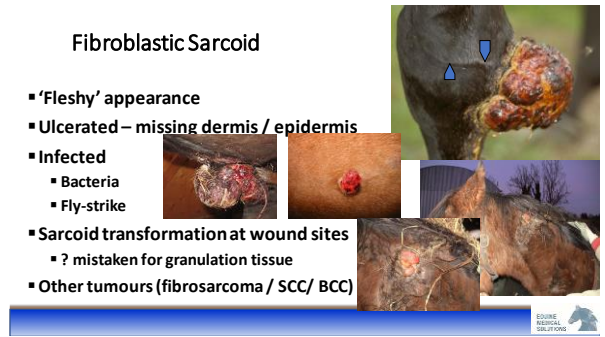
Fibroblastic Sarcoid



54

Fibroblastic Sarcoid

- 'Fleshy' appearance
- Ulcerated – missing dermis / epidermis
- Infected
 - Bacteria
 - Fly-strike
- Sarcoid transformation at wound sites
 - ? mistaken for granulation tissue
- Other tumours (fibrosarcoma / SCC/ BCC)



55



56



57



58

Mixed sarcoid

- Most sarcoids are mixed
- Definition only used when there no predominate type
- ? Reflect varying stages of development



59



60

The Malignant Sarcoid

- Clinically recognisable
- Locally invasive
 - Face
 - Groin
 - Elbow
- Histology
 - Lymphatic invasion
 - Local invasion
- NO METASTASIS!



61



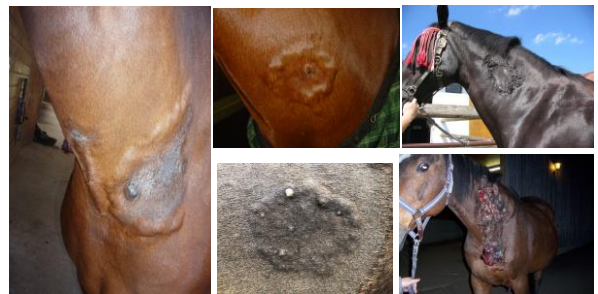
62



63



64



Malignant expanding ring nodular sarcoid

65

Sarcoid Transformation at Wound Sites

- Dangerous state
- Incipient wound healing failure
- Insidious expansion
- Rapid expansion

Is it granulation tissue?...
or is it sarcoid...?
or is it both?



66



67



68

What is it?



69

Prognosis

- **RULE 1:**
The more they have the more they get
- **Rule 2:**
The fewer they have the fewer they get
- **Rule 3:**
Multiply over summer & grow over winter
- **Rule 4:**
A single sarcoid implies (genetic) susceptibility

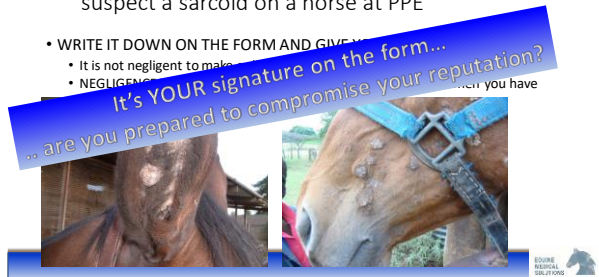
RULES!

1. You **SHALL!**
2. You **WILL!**
3. You **MUST!**



What should I do if I detect a sarcoid or if I suspect a sarcoid on a horse at PPE

- WRITE IT DOWN ON THE FORM AND GIVE IT TO THE VET
- It is not negligent to make a diagnosis
- **NEGLIGENCE** is what you do when you have a suspicion and do nothing



70

71

Key points for purchasers...

- The horse should not necessarily be “excluded” from purchase so long as the owner understands the implications
- 1. SARCOID IS A FORM OF SKIN CANCER BUT IT DOES NOT SPREAD TO OTHER ORGANS
- 2. A horse with even one sarcoid is *ipso facto* liable to the disease
 - This [genetic] liability remains with the horse for life!
- 3. Sarcoids are inclined to progress if neglected or treated inappropriately
- 4. The more they have the more they get – the fewer they have the fewer they get!
- 5. They multiply over the summer and “grow” over the winter
- 6. They can remain static for years –
 - THE ONLY PREDICTABLE THING ABOUT THE SARCOID IS THAT IT IS UNPREDICTABLE!
- 7. TREATMENT COSTS ARE SIGNIFICANT
- 8. SARCOID IS TRANSMISSIBLE TO OTHER HORSES TO SOME EXTENT

72 

SARCOID CAN RESULT IN LITIGATION

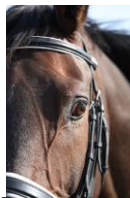
- Pre purchase examination
- Animal Welfare Act legislation
 - UK
 - Elsewhere
- Cruelty by neglect or inappropriate treatments
- DANGER TO OTHER HORSES – CIVIL PROCEEDINGS?



73 

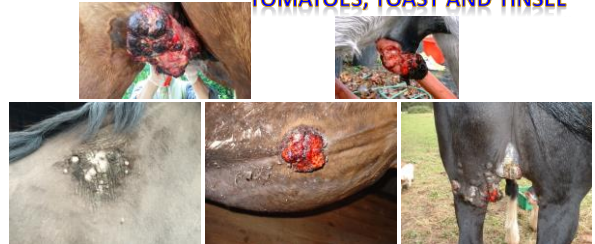
SUMMARY


- DO NOT IGNORE THEM!
- DO NOT INTERFERE UNLESS YOU HAVE A PLAN!
- APPLY THE BEST TREATMENT IMMEDIATELY – DON'T FIDDLE WITH THEM!
- UNDERSTAND THE DISEASE & KEEP UP TO DATE



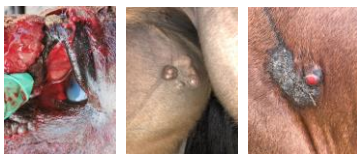
74 

“TURMERIC, THUJA, TOOTH PASTE, TOMATOES, TOAST AND TINSEL”



75 

So your diagnosis is right...



The problem is what to do THEN!



"Hello, doc. This is the 'hypochondriac'. Guess where I'm calling from?"

76 

Therapeutic problems

- Low success rates overall
- Lack of knowledge
 - Lack of “understanding”
 - Failure to recognise as “cancer”
- Lack of availability of treatments
 - Different countries have different problems
 - Logistical
 - Regulatory

Can we realistically give a 100% prognosis

Wisdom is the power to see the truth without attempting to fit it into any mold.

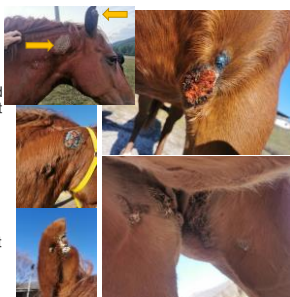


77 

Owner idiocy!

"Mare has had sarcoids for 5 years now, owner treated it herself without vet. She tried turmeric ointment, vitamins, leeches, the last was zinc ointment (august), which helped when I compare photos before and after treatment, but **owner wants to treat it with medication now and not with alternative medicine anymore**

Lesions are very extensive so I would like to ask you for your best advice. Lesions are on the right ear, right side of the neck, in the left axillary region, in the flanks/medial thighs"



78

Typical frustrated vet!



• "There are two fibroblastic chin lesions of 2x3 cm diameter, two flat lesions on her nose 5 cm in diameter (appear not to be changing), and a small 0.5 cm nodule above the right eye, which is 0.3 cm in diameter and possibly fibroblastic. *These have failed to respond to numerous cisplatin oil and bead treatments. The horse has no inflammatory response to treatment. Imiquimod has also been used with minimal reaction. With continued treatment failure the lesions are getting larger.*"



79

A lesson in oncology....

"Owner noticed a little lump (1-2 cm diameter) a couple of weeks ago; not been bothering Jasmin. Over last few days grew drastically and developed an ulcerated surface. Will be extremely difficult to tie anything around sarcoid. Also, Jasmin is difficult to inject to sedate."



Early intervention is always best... shame owners and vets don't appreciate that!

80

Frustration leads to desperation....

"About 8 years ago, ***** had a fist-sized sarcoid on his chest. The owner said it was triggered by a tick bite
We treated with xxTerra, but we could not get it in [country] any more

Then it was tied, sprayed with tarantula and iced up 3 times. However, the wound never really healed and it has remained a wound for the last 2 years. For the last two years, I smeared CompX (Yellow Bloodroot Ointment) without success
The sarcoid continued to spread in the area and also developed down the right front leg.

I found on the Internet a supplement with herbs, which I fed him and lubricated aloe vera gel, whereupon the growth came to a halt but unfortunately did not improve. In addition, I fed red beets, dried rose hips, and cat claw powder and additionally adjusted the concentrate to cereal-free. Then I decided to try the Indian Black Ointment. Unfortunately, after about 1 year, it was not really successful

In the meantime, I also tried Thuja outwardly and internally, and also aloe vera gel and various immune products. Now I'm smearing a blood ointment like xxTerra for a year now. It has become smaller in size, but unfortunately has not reduced

81

Treatments

- Surgery / Cryo / Laser / Ligation
- 'Immune' therapy
 - BCG Injections
 - Vaccines (autogenous / viral)
 - Cytokine / immunotherapy
- Chemotherapy / Electrochemotherapy
 - Cytotoxics (topical & injection)
 - Antimitotics (topical)
- Photodynamic therapy
- Radiation
- Homeopathy / Natural medicine / Phytotherapy



Why so many treatments?

82

Treatment selection criteria

1. SARCOID TYPE
 - a) LOGISTICS OF TREATMENT
2. SARCOID EXTENT
 - b) VALUE OF HORSE
3. SARCOID LOCATION
 - c) COST OF TREATMENT
4. SARCOID DURATION
 - d) DURATION OF TREATMENT
 - Time to return to work
5. PREVIOUS INTERFERENCE?
 - e) ANIMAL COMPLIANCE
6. PROGNOSIS
 - f) OWNER COMPLIANCE



83

Spontaneous resolution

- Rare occurrence
 - 0.1% in UK?
 - 10% in Sweden?
 - Up to 60% in Franche Montaigne breed... very
- Abrupt/total resolution V Gradual?
- SOLID IMMUNITY!
 - No further lesions
- Smaller long standing lesions
 - Single or few
 - Nodular → fibroblastic lesions
- Reason unknown: could this be useful!



85

Surgical options

- CONVENTIONAL SURGERY
 - High rate of recurrence
- LASER SURGERY
 - Less recurrence – slow healing
- HARMONIC / Plasma knife
- CRYOSURGERY
- SMART SURGERY
- LIGATION



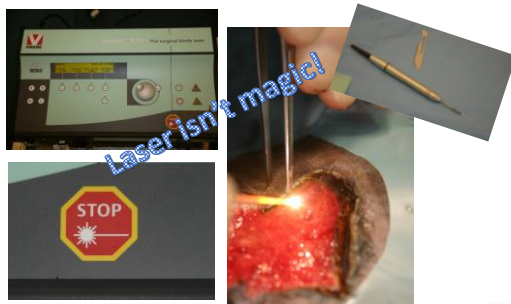
86



87



88



89

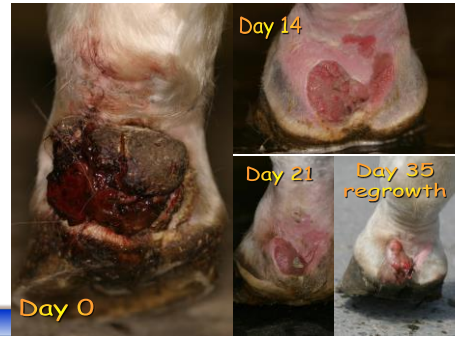


90



Type 1/a pedunculated fibroblastic sarcoid

91



92

What are the constraints on tumour surgery?

1. Defining the extent of the tumour
2. Anatomic constraints
3. Evidence based "margin of safety"
4. Bleeding
5. Cell seeding / contamination
6. Closure V safety / efficacy
 - Primary "centrifugal" contraction → widening of site
 - Fear of closure challenges
 - The "TWO TEAM" approach
 - CUTTERS V CLOSERS!

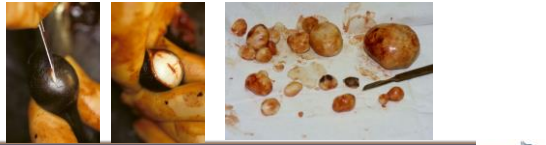


But remember...
You are on the same side!

93

Surgical "shell out"

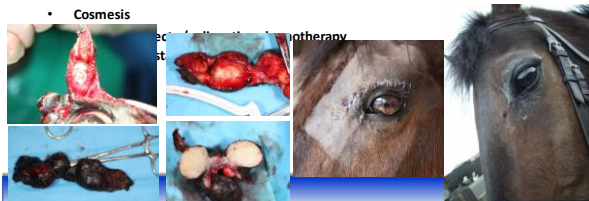
- Applicable to Type A1 nodules only!
- Dangerous as sole method for others
 - Ligation preferred for Type B/1 nodules



94

SHARP Surgery (conventional)

- Careful thought / preparation
- Anatomic considerations
- Cosmesis



95

The ultimate surgical option?



96

Surgical debulking – does it help or hinder?



97

RISK OF SEEDING / RESIDUAL TUMOUR

- **MARGIN DEFINITION**
 - Fear of closure challenge
- **SEEDING RISKS**
 - Instruments
 - Swabs
 - Gloves
 - Suture needles



98

Post Surgical Recurrence

- [Very] high rates for SARCOIDS! Jackson, 1936!
- Reasons for recurrence
 - **INADEQUATE MARGIN** → residual tumour
 - **SEEDING OF TUMOUR CELLS**
- **ALWAYS CONFIRM MARGIN SAFETY!**
 - **Do not economise!**



99

ROOT RECURRENCE V SEEDING

- Different implications!
- **ROOT RECURRENCE**
 - MORE PROBLEMATIC BUT UNSAFE MARGINS PRE-EMPTIVELY IDENTIFIABLE by histology
 - Starts deep and expands to surface
 - Widespread expansion deep below wound first → greater treatment challenges
 - Wound dehiscence - healing inhibition (contraction/ epithelialisation)
- **SEEDING**
 - Starts at surface of surgical site and expands INWARDS
 - LESS root expansion / MORE LOCALISED
 - Often develops within healing wounds
 - Can occur late if vector involved → scar-based recurrences



100

NOTE **Is sarcoid still present?**

- BE SUSPICIOUS IF.....
- 1. Your surgical wound breaks down for no obvious reason
- 2. HEALING IS SLOWER THAN YOU WOULD EXPECT
- 3. THERE IS INHIBITION OF EPITHELIALISATION
- 4. THERE IS LITTLE OR NO CONTRACTION OF THE SITE
- Inhibition within a treated / surgical site can be focal or general in either the margin or the wound bed OR BOTH



101

What happens if you fail?

1. RECURRENCE
2. EXACERBATION
3. REDUCED PROGNOSIS
4. BEWARE OF WOUND BREAKDOWN



102

Improving the outcome in TUMOR SURGERY

• “SMART” SURGERY

- Minimal contamination surgical tumour reduction
- Avoids seeding of tumour cells into the site
- Improves surgical prognosis by 5- 10% overall
- NOT 100% success rate!!
- SELECT YOUR CASES CAREFUL
- Define the likely margin!



103

SMART SURGERY PROVIDES A SOLUTION

1. FASTER SURGICAL PROCEDURE
2. LIMITED (ZERO) BLEEDING
3. NO SEEDING
4. NO PRIMARY CONTRACTION



The wound is closed before you make your incision!

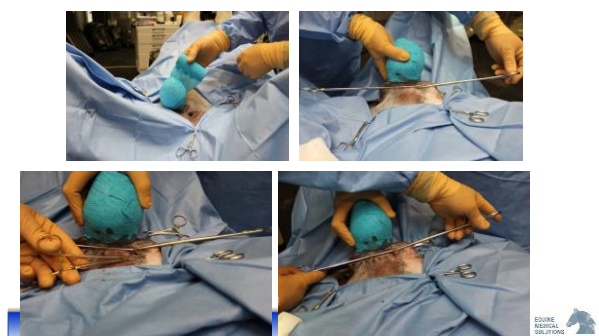
BUT – you must still have margin?



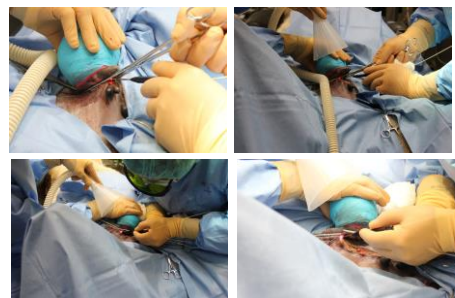
104



105



106



107



108



109

TYPE B NODULE

SMART (sharp) SURGERY

Staples NOT sutures

Healing / fly control

6 Pathology report

COMMENT
The histopathology demonstrate a spindle cell discrete mass which appears fully excised. This is compatible with a sarcoma, as per your suspicion. Sarcomas are the most common skin neoplasms of the horse. They are thought to be associated with bovine papilloma virus infection following injury or insect bites. The lesion can occur anywhere on the body but is most commonly found on the head, neck, legs and ventrum. Sarcomas do not metastasize, but recurrence post-excision is common, although some are reported to regress spontaneously. Since excision appears complete in this case, the prognosis is probably good.

110

It is useful but does not compensate for bad surgery!

THE WORLD'S MOST DANGEROUS BORDER

- **Sarcoid- high risk of seeding**
 - Carcinoma less / nil risk
 - Haemangiosarcoma some risk
- **Margin definition remains a problem**
 - **MARK THE MARGIN IF NECESSARY**
 - **Histology is STILL essential**
 - Laser / harmonic scalpel helps?

Martens et al., Polymerase chain reaction analysis of the surgical margins of equine sarcoids for bovine papilloma virus DNA. Vet. Surg. 2001; 30(5):460-467

111

Cryosurgery

- Time consuming
- Experience helps!
- Cryo-antigen effects?
- 'Crude-pour' techniques?
- **COMBINATIONS BETTER!**
 - Chemotherapy
 - Surgery

112

Cryosurgery combined with 5-Fluorouracil

113

Chemotherapy Options

- **TOPICAL CHEMOTHERAPY**
 - CYTOTOXICS / CYTOSTATICS / ANTIMITOTICS
 - ESCHAROTICS (metal chlorides)
 - HERBAL REMEDIES
 - PHYTOTHERAPY
- **INTRALESIONAL**
 - Antimitotics
- **SYSTEMIC**

TOPICAL	INTRALESIONAL
5 FU	SFU
BLEOMYCIN	MMC
AW5	CARBOPLATIN
IMQUIMOD	
BLOOD ROOT	

115

Topical Cytotoxics

- Few available
- 1. Formalin (Lotagen)
- 2. Arsenic Paste
- 3. 5% 5-Fluorouracil
- 4. AWS
- 5. Xterra/ Bloodroot



Advantages	Disadvantages
<ul style="list-style-type: none"> - Convenient - owner applied? - Cheap / Easy - ? Minimal dangers 	<ul style="list-style-type: none"> - Repeat applications - Penetration - Collateral damage - Exacerbation



116

Topical 5-FU Therapy

Pre treatment

Post treatment

Eufudix[®] cream
Fluorouracil 5%
20 g

117

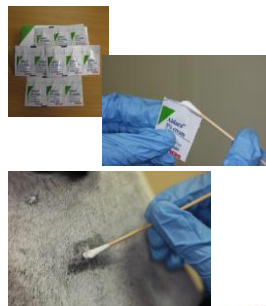
AWS

119

120

Imiquimod (Aldara[®])

- Antiviral / Anti-tumour effects
- Single reported study
- Prolonged applications
 - q48h x 10 / q 72h x 20
- Effect at 12 – 14 months
- 80% improvement in 60%
- 56% resolution



121

122



124



125



126

Aciclovir

- Specific herpes virus medication
- Bystander effect on suicide gene
- Irrational?
 - Extremely selective
 - Low [cytotoxicity](#)
 - Poor oral [bioavailability](#) (15–30%)
 - Poor penetration ability in tumors
- Applied q 4h at least to have any significant effect



128

Bleomycin

- Anti-tumor antibiotic
- Single/ double strand DNA breaks → inhibited DNA / RNA synthesis
- Normal cells have reparative mechanisms but possibly not tumour cells
- Bleomycin hydrolase in skin / other organs
 - → Lack of efficacy unless high dose / IV
 - Safer to use / handle?
 - Overcome by using topical ultra-deformable nano-liposome (patent)
- PAIN FREE / INFLAMMATION FREE / EASY TO APPLY!
 - Improved prognosis after surgery
 - Pre-medication with 5% 5FU ointment → excellent long term outcome
- Still testing – so far >600 cases with good efficacy / adjunctive for surgery but still some challenges



Kottenbelt et al., A pilot study on the use of ultra-deformable liposomes containing bleomycin in the treatment of equine sarcoid. Equine Veterinary Education 2018 21 June 2018 <https://doi.org/10.1111/eve.12950>

129



130

Intralesional Antimitotics

- Cisplatin / Carboplatin
- 5-FU
- Mitomycin C
- I-L essential!
- Restrict blood supply
 - Washout < 2 minutes



Advantages

- Convenient
- Low numbers
- Difficult sites

Disadvantages

- Stable S-R emulsions
- DANGEROUS
- CARCINOGENIC
- Repeated injections
- GA ?

131

Slow release local chemotherapy

- Stable emulsions using medical grade oils / stabilisers
1. Cisplatin
 2. Carboplatin
 3. 5 Fluorouracil
 4. Mitomycin C

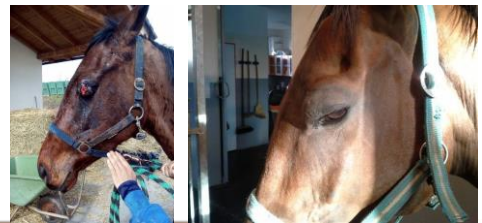


132

Spinal needles work best!

134

Carboplatin s-r x 5 at 2 week intervals



135

Slow release carboplatin emulsion

Is it getting WORSE?

136

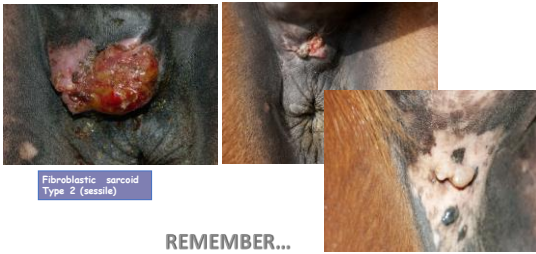
Cisplatin beads

- Available USA
 - Matrix III™, Royer, USA
- Implanted into tumour bulk or post surgical sites
- Relatively safe but still require care
- Limited personal experience
- Improved prognosis described



Hewes CA, Sullins KE Use of cisplatin-containing biodegradable beads for treatment of cutaneous neoplasia in equidae: 59 cases (2000-2004). J Am Vet Med Assoc. 2006 229(10):1617-1622.

137



Fibroblastic sarcoid Type 2 (cellule)

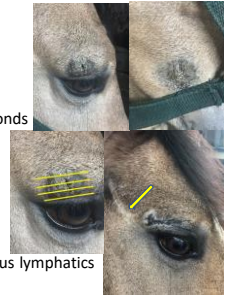
REMEMBER...

Anticancer drugs can have a pro cancer side effect!

138

Mitomycin C

- Aqueous MMC is washed out within 30 – 60 seconds
 - VERY high doses required
 - Repeat injections at short intervals
 - One paper presented at ECEIM (McKane S, 2016)
- MMC POLYMER
 - Thermolabile (multiple methods of administration)
 - Preliminary results encouraging
- SECONDARY CHANGES in pigment along cutaneous lymphatics



139

Electrochemotherapy

- Electrically induced increase in cell uptake of chemo agents
 - Electroporation → increased drug concentration
- Reported cisplatin only
- Repeated GA required – up to 6 times
- Good results reported
- Limited personal experience of it
- Does it do any more than a slow release form of the drugs?

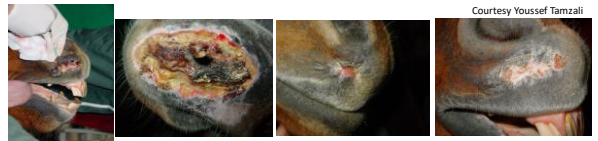


Mazères S et al., Non invasive contact electrodes for in vivo localized cutaneous electropulsation and associated drug and nucleic acid delivery. J Control Release. 2009 Mar 4;134(2):125-31. doi: 10.1016/j.jconrel.2008.11.003. Epub 2008 Nov 19.

140

Electrochemotherapy

- Non responsive to repeated surgical / cryosurgical treatment
- 6 procedures



Courtesy Youssef Tamzali

Tozon N et al, Electrochemotherapy as a single or adjuvant treatment to surgery of cutaneous sarcoid tumours in horses: a 31-case retrospective study. Vet Rec 2016 Dec 17;179(24):627. doi:10.1136/vr103867. Epub 2016 Oct 7

141

IMMUNOTHERAPY

- Vaccines (BPV / mRNA vaccines)
 - BCG
 - AUTOGENOUS VACCINES
 - ESPY AUTOGENOUS TISSUE IMPLANTAT
- PHYTOTHERAPY (Iscador P)
- HEMOTHERAPY
- MEDIATOR THERAPY

Grassarth-Maticek R et al., Use of Iscador, an extract of European mistletoe (Viscum album), in cancer treatment: prospective nonrandomized and randomized matched-pair studies nested within a cohort study. Altern Ther Health Med. 2001 May-Jun;7



142

BCG Immunological Therapy

- Repeated injections
 - Live bacillus best
 - PPD (Imunicidin) – volume related dose
- Risk of anaphylaxis?
- Method critical to success
 - Different proteins have different requirements
- Best results around eye
 - Nodules / fibroblastic
- **NOT DISTAL LIMB [F-B] LESIONS**

It still works!



143

BCG Immuno-therapy

- Nodules / fibroblastic lesions around the eye
- Periorbital better than face > body
 - **CONTRAINDICATED ON LIMBS**
- Technique critical (Knottenbelt, 2009)
- INTRALESIONAL INJECTION ESSENTIAL
 - Not always easy for solid tumours
- RISK OF ANAPHYLAXIS ! – take precautions!
- Expanding week protocol is immunologically better
- Some local side effects possible



144



145

ESPY AUTO TUMOR IMPLANT

- Autologous grafting after liquid nitrogen freezing of tumor tissue blocks
- Reports of success but also failures (of course, as for all other treatments)
- Mechanism is uncertain and rationality is not easy to understand!
 - Tumour derived antigens?
 - Remote effects reported
- High efficacy reported but also failures
- Different outcomes from different users
- 3 partial 'successes' out of 16 cases



Espy BMK How to treat Equine sarcoids by autologous implants. Autologous grafting treatment for equine sarcoid. Proceedings of the American Association of Equine Practitioners AAEP 2008, San Antonio. 68-73

146

ESPY TECHNIQUE



147

Phytotherapy

- Iscador P
 - Repeated injection locally
 - Long course / painful injections
- Blood root
 - Commercially prepared with 15-30% zinc chloric
 - Topical applications
 - Owner applied



Christos Giannidis et al. Treatment of clinically diagnosed equine sarcoid with a mistletoe extract (*Viscum album austriacus*). [J Vet Intern Med.](#) 2010; Nov-Dec;24(6):1483-9.

Wilford S et al., Owners' perception of the efficacy of Newmarket bloodroot ointment in treating equine sarcoids. [Can Vet J.](#) 2014 Jul;55(7):683-6.

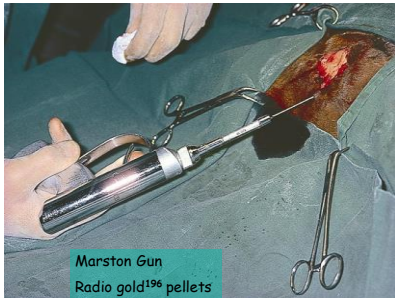
148

Radiation

- Beta / Gamma radiation
- Plesiotherapy
- Interstitial brachytherapy
- Teletherapy

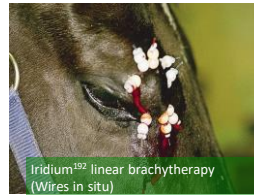


149



150

Low dose Radiation (LDR) Iridium 192



151

Low dose Radiation (LDR) – iridium 192



152



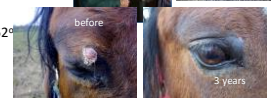
153



154

What other treatment options are there?

- Photodynamic therapy
 - Photoactive agents (Hypericin / ALA)
- Hyperthermia
 - Repeated elevation of temp to 60° - 62°
- Actual cautery
 - "Burn them out" with firing irons etc
- Electrochemotherapy
- HIFU



Courtesy Jeremy Kemp Symonds



155



156



157

Homeopathic / Natural Remedies

First learn to spell!!!

- TURMERIC
- TOOTHPASTE
- TOMATO / Toast
- Tea Tree Oil
- Thuja
- 'Camrosa'
- Cautery (acid/alkali/copper sulphate Silver nitrate)

158

Immune "stimulants"

- 'Sarc-ex' / Echinacea etc.!
- Irrational choice
 - Would you ask for this therapy for yourself?
- If it was as easy as this we need nothing else!

159

Therapeutic optionschoose carefully

- Combination therapy is a really good option
 - Variations to therapy are common even when the lesions look the same and are situated in broadly the same anatomic region
- Limitations to choices are inevitable
 - Facilities / regulatory
 - Cost
 - Logistics

160

Implications of failure?

- Exacerbation
 - Transition to fibroblastic / malignant
- Regrowth at site
 - Recurrence / new lesions
- Reduced prognosis for next treatment
 - 40% for each failure
 - Biopsy counts as failure?

First treatment attempt MUST be the best available option
Economy seldom pays!

161

It's not the successes that matter..
...its the failures!

If you always
do what you
always did, you
will always get
what you
always got.
- Albert Einstein

"If your treatment works – use it!

But IF IT DOESN'T, then CHANGE IT QUICKLY!



EQUINE
MEDICAL
SOLUTIONS

162



EQUINE
MEDICAL
SOLUTIONS

163

Afternoon Session



EQUI FOCUS POINT BELGIUM

Dr. Annick Gryspeerdt



1

1

Organisation

- Sponsors



- Daily operation:



Annick.gryspeerdt@gmail.com

www.efpb.be

0485 34 28 72

2

Activities & news



✓ Routine diagnostics:

- Advice concerning tests and sampling
- Stimulation and sponsoring of diagnostical tests (abortion and respiratory diseases)
- Advice in case of outbreaks of diseases

✓ Stimulation of research of several infectious diseases:

- Rotavirus project (2012)
- Respiratory project (2014 and 2016)
- Abortion project (2015 and 2016)
- Guidance in case of outbreak (Rhino, 2015 and 2019)
- Serological screenings Lyme and Lawsonia (2016)
- Free diagnostics for equine abortion and respiratory disease (2017)
- Partially sponsored diagnostics for equine abortion and respiratory disease (2018 till June 2023)

✓ 2024:

- Depending on budget: sponsoring of several diagnostical tests in different cooperating laboratories
- Guidance in case of outbreaks

3

3

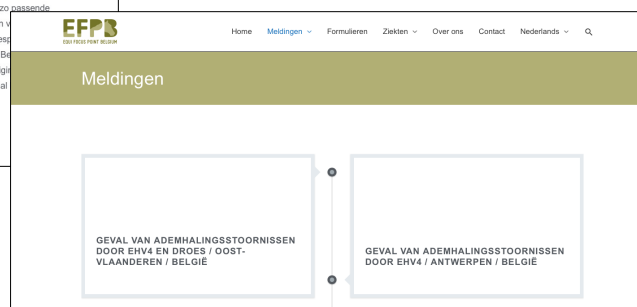
Website



www.efpb.be

- Alerts
- Forms
- Newsletters
- Diseases
- Inscriptions

30 632 unique visitors in 2023



4

Inscriptions

Schrijf u hier in om onze nieuwsbrief te ontvangen

Naam*

E-mail*

Archief

Interessante links

<http://veterinaryrecord.bmj.com/>
www.respe.net
 Latest Defra/AHT/BEVA Equine Disease Surveillance Report

Kies uw taal

Nederlands
 Français

5

Activities & news



- 892 inscriptions: 559 Flemish vets, 215 Walloon vets, 118 other
- individual and summarizing alerts:

Pathogen	Respiratory disorders	Abortion	Nervous system disorders	
EHV1 (18)	3	10	3	2
EHV4 (10)	6	4		
EHV2 (5)	5			
EHV5 (3)	3			
Influenza (7)	7			
Strep equi (4)	4			
Strep zooep (1)		1		
TOTAL	28	15	3	2

- News letters

6

Alerts 2023

15th of April 2023: unusual large outbreak of EHV1 (East Flandres)

Combination of symptoms in 1 premise:

Abortion	>15	death
Neonatal foal death	8	death
Neurological disease	4 infection in first days	survival
	13 quadriplegia	death
	3 neurological symptoms	survival

> 36 dead horses

7

Cause of big outbreak ...?

- Gathering of risk groups:
 - young horses → Low immunity
 - pregnant mares → High level of viral excretion
 - pregnant mares → Sensible for abortion
- Large amount of horses, no separation of risks groups
- No vaccination

→ Ideal circumstances for rapid viral spread

Low population immunity + high viral spread
= high risk factor for neurological disease

8



Pastern Dermatitis What's in a name?

EQUINE MEDICAL SOLUTIONS



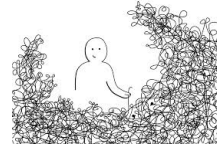
Email: office@equinesarcoid.co.uk
 Website: www.equinesarcoid.co.uk
 Facebook: <https://www.facebook.com/equinemedicalsolutions/>
 App: Equine Medical Solutions (on-line or off-line use)

Derek C Knottenbelt

1

Pastern Dermatitis Syndrome

- ▶ Complex group of disorders
- ▶ Commonly over-simplified
 - ▶ poorly understood
 - ▶ poorly investigated
 - ▶ poorly managed (owner / vet)



2

On the balance of probability what can we say about these?

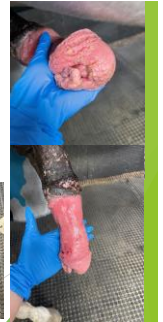


PCLCCV SLE-like Syndrome Staph dermatitis Dermatophilosis

3

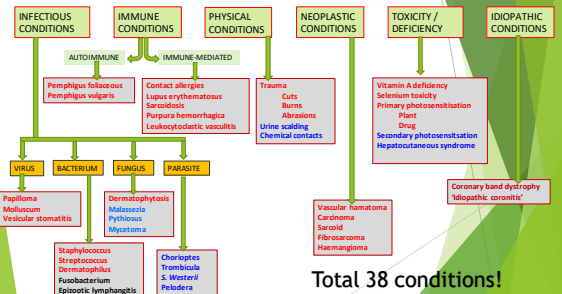
So what about this "Mud Fever?"

- ▶ 6-month history of persistent dermatitis on cannon and medial distal tibia - distribution?
- ▶ Little response long-term but repeated heavy washing helps! - implication?
- ▶ History and clinical examination! - necessary?
- ▶ Sheath discharge noted - significant?



4

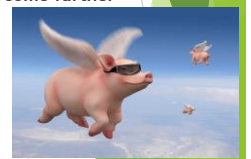
PASTERN DERMATITIS AND CORONITIS



5

How likely is it that ...

- ▶ There will be a single effective treatment / management?
- ▶ Every condition will respond to a single / every drug / wash?
- ▶ A diagnosis can be made in every case?
- ▶ A diagnosis will not require at least some further investigation?



6

DON'T FORGET TO REMEMBER

All cases with inflammatory dermatitis restricted to the white areas **MUST** be tested for hepatic function

- ▶ even if lesions are restricted to limbs
- ▶ even if everything else is apparently normal
- ▶ even if there are other causes of actinic dermatitis

7

Always consider all possibilities!

Logic, logic and logic

... no matter how rare...



8

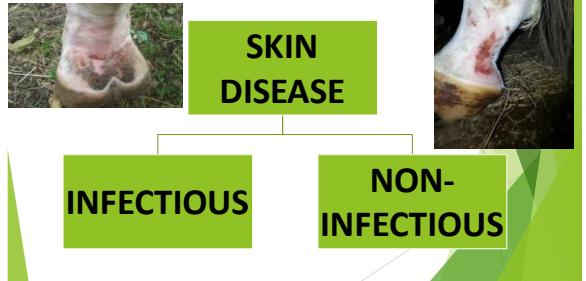
The 3 requirements of clinical investigation

1. Logic, logic and logic
2. Thoroughness, thoroughness and thoroughness
3. Knowledge, knowledge and knowledge



9

Diagnostic Scheme



10

- INFECTIOUS
 - VIRUS
 - BACTERIA
 - FUNGUS
 - PROTOZOA
 - PARASITES
 - ECTOPARASITES
 - MITES
 - LICE
 - OTHERS
 - ENDOPARASITES



□ NON-INFECTIOUS

- Genetic
- Immunological
- Nutritional
- Allergic
- Neurological
- Developmental
- Traumatic
- Hormonal (ENDOCRINE)
- Neoplastic
- Iatrogenic / Idiopathic
- Chemical / Toxic
- Cardiovascular

11

Viral causes of pastern dermatitis

- ▶ Papilloma
 - ▶ EcPV1
 - ▶ Young horses
 - ▶ Spontaneous resolution
 - ▶ Concurrent papillomata elsewhere
 - ▶ 'Vaccination' effective
 - ▶ EcPV2
 - ▶ Non/weakly immunogenic
 - ▶ Can be very serious
 - ▶ Vaccination non-effective
- ▶ Molluscum contagiosum
- ▶ ? Sarcoid..... not really a viral disease!

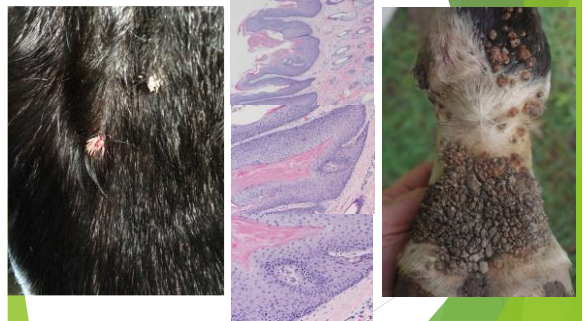


12

Viral papilloma of pastern region



13



14

Management

- ▶ HIGHLY problematical
 - ▶ No detectable antibody responses
- ▶ Some DO resolve spontaneously eventually
- ▶ Antiviral creams
 - ▶ IMIQUIMOD.... Not acyclovir!
 - ▶ Podophyllin and other wart creams
- ▶ SURGERY / CRYO / LASER ABLATION



15

Bacterial Pastern Folliculitis

- ▶ Follicular inflammation + debris
 - ▶ *Staph. aureus* / *hyicus* / *intermedius*
- ▶ Degeneration of hair follicles
 - ▶ infection of surrounding dermis (farunculosis)
- ▶ Coalescence of expanding pastern / coronet pyoderma
- ▶ Possibly fungal complications....



IT'S REALLY PAINFUL!

16

Bacterial Pastern Folliculitis

Clinical Signs

- ▶ COMMON
- ▶ Usually limited to heel bulbs & palmar / plantar pastern
- ▶ One or more [white] limbs
- ▶ Extensive ulceration / exudation
- ▶ Extensive matting of hair
- ▶ Oedema of limb **with pain**
- ▶ Lymphangitis / cellulitis
- ▶ ? Lameness but few systemic effects

OUCH!

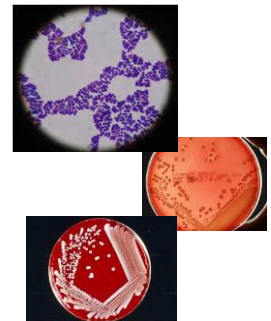


17

Bacterial Pastern Folliculitis

Diagnosis

1. Typical appearance
2. Pain / character of lesions
3. Smears
 - ▶ possible pure Staphylococcal infection
 - ▶ mixed organisms common if long standing
 - ▶ BPVI
4. Culture / biopsy (beware!)
 - ▶ Collection technique is vital!



18

Bacterial Pastern Folliculitis

▶ **IMMEDIATE Treatment**

- ▶ **Sedation / analgesics / ?GA if > 1 leg**
- ▶ **CLOSE CLIP WHOLE AFFECTED AREA**
- ▶ **WARM / HOT skin** [antiseptic] washes (repeated)
 - ▶ chlorhexidine / povidone
 - ▶ hygiene essential to prevent skin wetting / maceration
- ▶ ? antibiotics (TMS/Penicillin)



19

Bacterial Pastern Folliculitis



▶ **LONG TERM Treatment**

- ▶ Prolonged antibiotics
 - ▶ Potassium iodide orally (NON-PREGNANT!)
- ▶ Local clipping / antiseptic washes
 - ▶ INFREQUENT / **ONLY IF NEEDED!**
- ▶ Silver dressings?
- ▶ Avoid creams & lotions



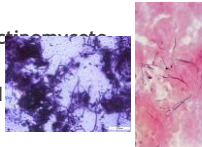
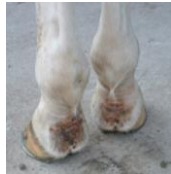
WARNINGS !

1. **DO NOT PROMISE QUICK RESPONSES**
2. **CAN BE PAINFUL or even VERY painful!**

20

Dermatophilosis

- ▶ Important / common disease
- ▶ Moist conditions (but not always ?)
 - ▶ Hygiene / management related aetiology
 - ▶ Affects other species as well - cattle...
- ▶ **D congolensis:**
 - ▶ Gram +ve, filamentous, branching actinomyces
 - ▶ coccoid / motile zoospores
- ▶ Humoral & cellular responses required
 - ▶ Immune compromised horses ?



21

Dermatophilosis (Mud Fever)

- **Clinical signs**
 - Lesions distributed over 'wetable' areas
 - Heels / palmar pastern ("greasy heel")
 - Matted coat with exudate
 - Can be legs only / single limb?
 - Worse on white haired legs ?
 - Pain / lethargy / mild worry (pruritus)
 - **Silvery skin + purulent material**
 - **Paint brush matting of hair coat**
 - Not pastern or cannon forms



22



23

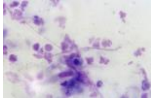


24

Dermatophilosis

▶ Diagnosis

- ▶ Clinical appearance pathognomonic?
 - ▶..... OR IS IT?
- ▶ Smear stained with Methylene Blue (+ others)
- ▶ Culture - difficult (consult with lab)
 - ▶ Sampling technique
 - ▶ Microaerophilic
- ▶ Treatment results
 - ▶ if it doesn't get better think again



25

Dermatophilosis

▶ Treatment

- ▶ CLIPPING ESSENTIAL
- ▶ Gentle warm / hot antiseptic wash
- ▶ SILVER DRESSINGS / Flamazine
- ▶ KEEP DRY thereafter!
- ▶ Skin emollients
 - ▶ White lotion / zinc and castor oil ointment etc.
- ▶ Topical corticosteroids not advised
- ▶ Systemic antibiotics ? (penicillin / TMS)



26

Dermatophilosis

▶ Control

- ▶ Recognise cases early
 - ▶ Beware secondary infections
- ▶ Isolate cases
- ▶ Use dedicated equipment / tack
 - ▶ Fumigate / wash tack etc.
- ▶ Avoid over grooming of diseased horses
- ▶ Sunlight is good but rain is bad!
 - ▶ SOME CASES EXACERBATED BY SUNLIGHT



Roberts DS. 1963 Chemotactic behaviour of the infective zoospores of *Dermatophilus dermatonomus* Australian Journal of Agricultural Research 14(3) 400 - 411

27

Fungal Pastern Dermatitis



28

Dermatophytosis

- ▶ Trichophyton / Microsporum spp.
- ▶ Common skin infection
- ▶ Rare on pastern / cannon
- ▶ Usually concurrent problem at other sites
 - ▶ NOT ALWAYS
- ▶ Sampling
- ▶ Treatment



29

Malassezia folliculitis

.. Or is this an opportunistic infection?



30

Management

▶ **Topical**

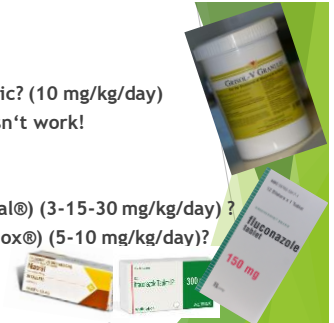
1. Imaverol® (Enilconazol) 0.2% 2xweekly
2. Mycophyt ® (Natamycin) 2x per week
3. Malaseb Shampoo (Chlorhexidine 2%, Miconazol 2%) 2x per week



31

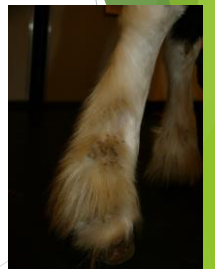
Systemic Medication

- ▶ Griseofulvin fungistatic? (10 mg/kg/day)
 - ▶ Not available / doesn't work!
- ▶ Azoles - fungicidal
 1. Ketoconazol (Nizoral®) (3-15-30 mg/kg/day) ?
 2. Itraconazol (Sporanox®) (5-10 mg/kg/day) ?
 3. Fluconazol ???



32

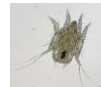
Parasitic Pastern Dermatitis



33

Chorioptes equi

- ▶ Common
- ▶ Feathered horses more often
 - ▶ NOT EXCLUSIVELY
- ▶ Severe infestations develop
 - ▶ Over winter / Stabled horses
- ▶ Carrier status
 - ▶ Transmission by bedding / brushes / contact
- ▶ Pruritus not proportional to infestation severity



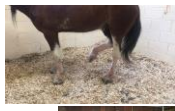
34

Chorioptic Mange

▶ **Clinical Signs**

▶ **PRURITUS!**

- ▶ Irritation / stamping / bizarre behavior
- ▶ Worse warm weather / warm stables
- ▶ Self inflicted trauma
- ▶ Secondary infections
 - ▶ Dermatophilus / Staphylococci / Streptococci
- ▶ Lichenification & patchy alopecia
 - ▶ Broken hairs
- ▶ Scaling / flaking / exudation / crusting



35

36



37

Chorioptes equi

- ▶ Management
 - ▶ Identify affected horses in winter
 - ▶ Low infestations may → severe pruritus
 - ▶ High infestations may → slight pruritus
 - ▶ Identify carriers in autumn
 - ▶ Treat all affected / in contact horses - repeatedly
 - ▶ Fipronil (*Frontline*)
 - ▶ Doramectin (intramuscular)
 - ▶ Pyrethroids

NOT AMITRAZ



39

Harvest/Forage/Poultry Mites

- ▶ Scale feeders
 - ▶ Forage mites
- ▶ Suckers (blood / plasma)
 - ▶ Trombicula spp.
 - ▶ Dermanyssus gallinae
- ▶ Contact with source
 - ▶ e.g. poultry
- ▶ Seasonal / geographical
- ▶ Legs / head (severe) pruritus



40



41



42

Harvest/Forage/Poultry Mites

- ▶ Management
 - ▶ Avoidance of contact / sources
 - ▶ Eat the chickens!!
 - ▶ Change the hay / field!!
 - ▶ Tend to be seasonal/geographical
 - ▶ Washes
 - ▶ Fipronil (*Frontline*)
 - ▶ Pyrethroids

NOT AMITRAZ



43

Parasitic Dermatitis (larval nematode)

Strongyloides westerii

- ▶ Poor hygiene (wet yards / mud)
- ▶ Foals < 4 months
- ▶ Poor worming history
 - ▶ NOT ALWAYS
- ▶ Usually several involved
- ▶ Accumulates to end of season

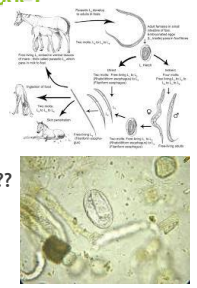


44

Parasitic Dermatitis (larval nematode)

▶ **Diagnosis**

- ▶ Washings from limbs
 - ▶ parasite easily seen in numbers
 - ▶ dissecting microscope
 - ▶ black tile method
- ▶ Clinical appearance pathognomonic ??



45

Non-Infectious Pastern Dermatitis

- ▶ **IMMUNOLOGIC**
 - ▶ Localised
 - ▶ Generalised
- ▶ Neurologic
- ▶ **TRAUMATIC DERMATITIS** (injuries / burns)
- ▶ **NEOPLASTIC DISEASE**
- ▶ **IATROGENIC IDIOCY**
- ▶ **CARDIOVASCULAR COMPROMISE**
- ▶ **CHEMICAL / TOXIC**



46

Pemphigus Foliaceus

- ▶ Autoimmune disorder
 - ▶ Type II hypersensitivity (cytotoxic response)
 - ▶ Vesiculo-bullous disease
 - ▶ Junctional auto-antibody response
 - ▶ dermo-epidermal junction
 - ▶ Appaloosa predisposed
 - ▶ NOT exclusive!

47



Crusting Scaling
Pemphigus foliaceus

48



49

Coronitis / Immune-mediated coronary band [junctional] disorder

Clinical Signs

- ▶ Extensive or localised inflammatory changes
- ▶ Coronary band only ?
- ▶ Recurrent / Persistent
 - ▶ Periods of remission ?
- ▶ Related to vitamin A ?
- ▶ Pemphigus group ?



50



51



52

Diagnosis

- ▶ BIOPSY
 - ▶ Punch biopsy from CB is safe!
- ▶ Eliminate pemphigus...
 - ▶ Not easy since pathology is NOT / [never] clear
- ▶ Consider Coronary band Dystrophy
 - ▶ Dystrophy
 - ▶ "any of various bodily disorders, characterized by wasting of tissues"
- ▶ **What is this condition?**



53

Management

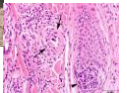
- ▶ Few possibilities for treatment
- ▶ Management usually the target of therapy
- ▶ CORTICOSTEROIDS → some responses - temporary
- ▶ IMMUNOSUPPRESSIVES → better control
 - ▶ Methotrexate / azathioprine / pentoxifylline
- ▶ Local exfoliants / keratolytics
- ▶ Cell Stimulants / blisters / oils / creams
- ▶ Dietary supplements (oils / biotin)



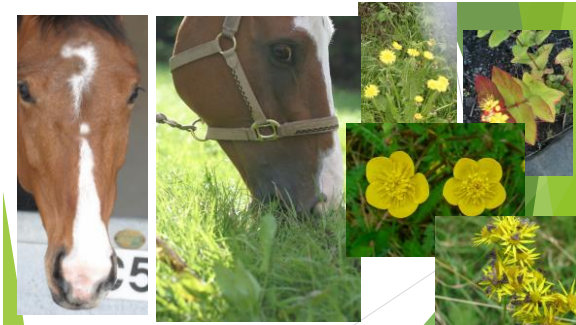
54

Pastern & Cannon Leukocytoclastic Vasculitis

- ▶ sporadic disease / ?UV related
- ▶ more common than realised ?
- ▶ mature horses
 - ▶ non-pigmented extremities
 - ▶ outer aspects of non-pigmented hind limbs!
 - ▶ Other areas also!
 - ▶ summer months
- ▶ early lesions: IgG+C3 deposition in vessels



55



56



57

**Pastern & Cannon
Leukocytoclastic Vasculitis**

► Clinical Signs

- Acute onset erythema / oozing / crusting
 - Severity often only revealed by clipping
 - Chronic case / lesions
- Clearly demarcated
 - Lateral aspect of white (hind!) pastern / cannon
 - Lat hind > lat front > medial hind > medial front
 - Pigmented limbs seldom if ever affected
- pain rather than pruritus
 - scabs / crusts difficult to remove



58

59



60



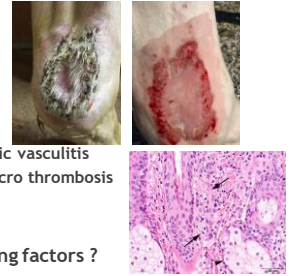
61



62

Pastern & Cannon Leukocytoclastic Vasculitis

- ▶ **Diagnosis**
 - ▶ History important
 - ▶ first site / nature
 - ▶ CLIP → pathognomonic?
 - ▶ BIOPSY
 - ▶ non specific leukocytoclastic vasculitis
 - ▶ vessel wall necrosis / micro thrombosis
 - ▶ CHECK LIVER FUNCTION
 - ▶ usually normal
 - ▶ No primary photosensitising factors ?



63

Pastern & Cannon Leukocytoclastic Vasculitis

- ▶ **Treatment**
 - ▶ Clip carefully / **protect from sunlight**
 - ▶ Bandage / stable - sunblock NOT enough
 - ▶ Check for photo-active plants etc. REMOVE!
 - ▶ *Hypericum perforatum* (St John's Wort)
 - ▶ Remove all drugs - drugs don't help!
 - ▶ High dose prednisolone (2 - 3 mg / kg q 12h)
 - ▶ Taper over weeks: METDD ⇒ MEADD
 - ▶ Parenteral antibiotics (?)
 - ▶ Some recur (several times!) but most are single episode

64



65



No chemicals / no drugs

66

Systemic Lupus Erythematosus-Like Syndrome

- ▶ No true lupus cells found in horses
 - ▶ LE cell (Lupus Erythematosus cell) = neutrophil or macrophage that has phagocytized (engulfed) denatured nuclear material of another cell
- ▶ Some have anti-nuclear antibody
- ▶ Multiple organ involvement typical
- ▶ Multifactorial aetiology?
 - ▶ Genetic / immunological
 - ▶ Systemic disease (lymphosarcoma etc.)
 - ▶ Trigger factors (temperature / pregnancy / work)
 - ▶ Iatrogenic (DRUG RELATED)
 - ▶ ? VIRUS (not in horses?)

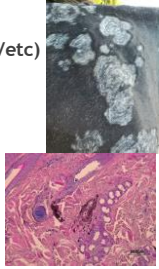


67

Systemic Lupus Erythematosus -Like Syndrome

▶ Clinical Signs

- ▶ Multiple organ involvement (eyes/joints/etc)
- ▶ Petechiation / pyrexia / wt loss etc.
- ▶ Uveitis
- ▶ Pastern changes:
 - ▶ Erythema / scaling / alopecia
 - ▶ Lichenified / thickened skin
 - ▶ Non-pigmented skin most affected ?
 - ▶ Photosensitised skin tendency



68

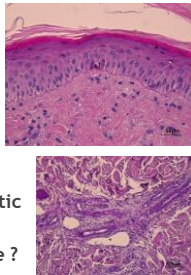


69

Systemic Lupus Erythematosus -Like Syndrome

▶ Diagnosis

- ▶ Very difficult
- ▶ Clin. Path non-specific
 - ▶ ↑ Fibrinogen / γ glob
- ▶ Multi-organ / system involvement
 - ▶ Did you do a full clinical ?
- ▶ Biopsy
 - ▶ immunofluorescence characteristic
- ▶ ANA antibody
 - ▶ Coomb's test anti equine positive ?



70

Systemic Lupus Erythematosus-Like Syndrome

▶ Treatment

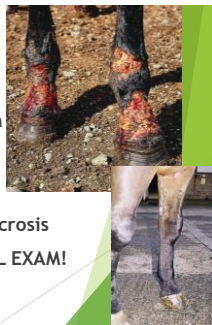
- ▶ remove any possible underlying cause
 - ▶ NB REMOVE ALL DRUGS!
- ▶ antibiotics then corticosteroids
 - ▶ long courses but complications of drug eruptions

71

Purpura haemorrhagica

▶ Clinical signs

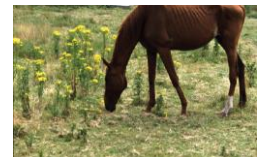
- ▶ Urticarial wheals and limb oedema
- ▶ Cerebral and pulmonary oedema
- ▶ Petechial haemorrhages
- ▶ Extensive distal limb erosions / necrosis
- ▶ CHECK HISTORY AND DO A CLINICAL EXAM!



72

Photosensitisation (Actinic Dermatitis)

- ▶ Ultraviolet radiation
- ▶ Non pigmented skin
 - 1) Sunburn (expected outcome)
 - 2) Photosensitisation (unexpected outcome)
 - a) primary photodynamic agent in skin
 - b) secondary hepatic failure



73

Photosensitisation (Actinic Dermatitis)

► Clinical Signs

- Severe cutaneous destruction restricted to white areas ONLY - well demarcated
 - skin necrosis & sloughing
 - exudation and pain
 - coronary band often worst affected area
 - generalised lesions (hepatic involvement?)
 - limb lesions alone (P&CLCCV ?)
 - face only (sunburn?)

74



75

Photosensitisation (Actinic Dermatitis)

► Diagnosis

- ALL horses showing dermatitis restricted to white areas MUST be tested for liver function
- investigate for possible photodynamic agent ingestion
- Hepato-cutaneous Syndrome



76

Photosensitisation (Actinic Dermatitis)

► Treatment

- treat primary cause
 - pastern lesions are often not the major concern
- protect from sunlight (ESSENTIAL)

77

Contact Dermatitis

- Irritant contacts
- Iatrogenic
 - Mercury / ether blister
 - Cantharides Blisters
- Persistent wetting
- Urine scalding
 - Bladder paralysis
- Serum exudation
- Bandage Dermatitis / necrosis



78

**Verrucose / Progressive Pastern Dermatitis
Chronic progressive lymphedema**

- Historically devastating
- Heavy horses
- Feathered legs
- Chronic possibly hereditary / genetic disorder
- Can be prevented
- Common cause of litigation against owners



79



80



81



82



83



84



Management

- ▶ VERY CHALLENGING but can be rewarding
- ▶ REMOVE ALL HAIR and keep it away!
- ▶ HOT WASH and then keep dry
- ▶ Exercise
- ▶ Mite control
- ▶ HYGIENE

A RIVER CUTS THROUGH ROCK, NOT BECAUSE OF ITS POWER, BUT BECAUSE OF ITS PERSISTENCE.



85

Neoplastic conditions → pastern disease

▶ WOUND RELATED USUALLY!

▶ Sarcoid

▶ Carcinoma

▶ Fibrosarcoma

▶ Hemangiosarcoma



86

When we hear the sound of hooves coming towards us

We should think HORSES

.....
not ZEBRAS

Unless of course you are in parts of Africa where there are NO horses!!



87

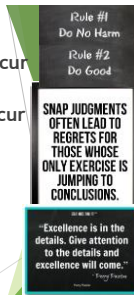
Notes:

▶ Common things are common because they occur more often

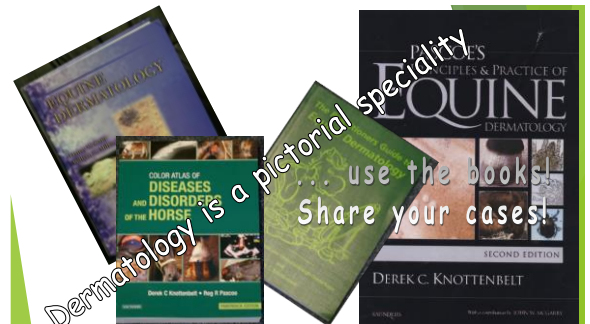
▶ Rare disorders are only rare because they occur rarely - they are NOT impossible

▶ Always:

1. Take your time
2. Be logical
3. Consider every possibility however remote
4. Remember to consider systemic disease
5. Focus treatment on disease not presenting signs



88



89



1



DEPARTMENT OF LARGE ANIMAL SURGERY, ANAESTHESIA AND ORTHOPAEDICS

PRACTICAL GUIDELINES TO EXAMINE THE EQUINE EYE

Eline Vercruyse



2

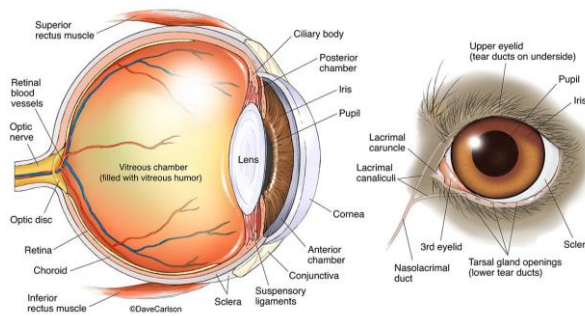
“MORE IS MISSED BY NOT LOOKING THAN NOT KNOWING”

THOMAS McCRAE (1870-1935)

3

GENERAL RULES

- Be systematic and take your time!
- Knowledge of the anatomy (eye + adnexa)



DOG EYE ANATOMY

Illustration of canine eye anatomy. Image © copyright by Dave Carlson.

4



Examination conditions



Routine equipment and supplies



Advanced examination tools



General order of steps



5

5

EXAMINATION CONDITIONS

- Room with closed doors
 - Restricts disturbance
 - Permits darkness
- Stal, barn aisle or corner of a shed
 - NOT in a pasture
- Person holding the horse/head
 - Stocks
 - Head support



6

ROUTINE EQUIPMENT AND SUPPLIES



7

ROUTINE EQUIPMENT AND SUPPLIES

– Sedation

Molecule	Dosage	Effect
Xylazine	0.5 – 1 mg/kg IV	Moderate sedation
Detomidine	0.02 – 0.05 mg/kg IV	Profound sedation
Butorphanol	0.01 – 0.02 mg/kg IV	Sedative and opioid



8

ROUTINE EQUIPMENT AND SUPPLIES

- Local anesthetics
 - Lidocaine or mepivacaine
 - 0.5% proparacaine or 1% tetracaine
- Mydriaticum - 1% tropicamide



9

9

ROUTINE EQUIPMENT AND SUPPLIES

- Fluorescein colouring (strips or minims)
- Sterile eyewash
- Graefe fixation forceps



10

10

ROUTINE EQUIPMENT AND SUPPLIES

- Sampling items
 - Sterile culture swab
 - N°15 scalpel blades/Kimura spatula/cytobrush
 - Microscope blades



ROUTINE EQUIPMENT AND SUPPLIES

- Halogen Finnoff transilluminator
- Small focal pen light
- Direct ophthalmoscope
- Monocular slit lamp
- Cell phone/camera



ADVANCED EXAMINATION TOOLS

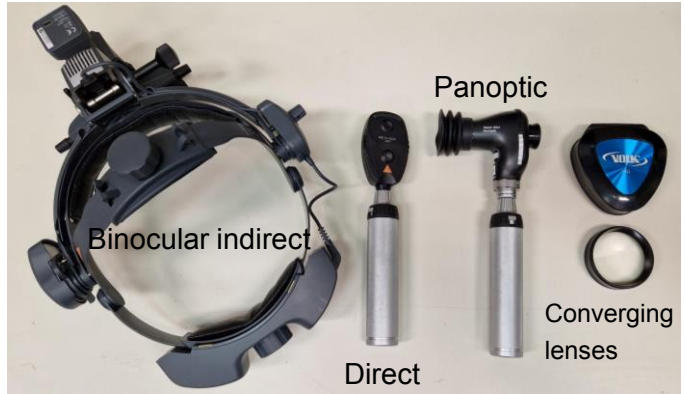
Tonometer



Slit-lamp



Ophthalmoscopes



ADVANCED EXAMINATION TOOLS

- Tonometer
- Handheld slit-lamp biomicroscope
- Binocular indirect ophthalmoscope
- Handheld converging lenses (15-20, 30D or 2.2 pan-retinal)
- Panoptic ophthalmoscope
- High frequency ultrasound equipment
- Electroretinogram
- Infrared camera

GENERAL ORDER OF STEPS

1. Obtain medical/ocular history
 - Motive of the owner
 - Signalement
 - Intended use
 - Environment and management
 - Duration and severity presenting signs
 - Other (systemic) disorders
2. Examine horse in environment
 - Lead or loose in a stall
 - Performing



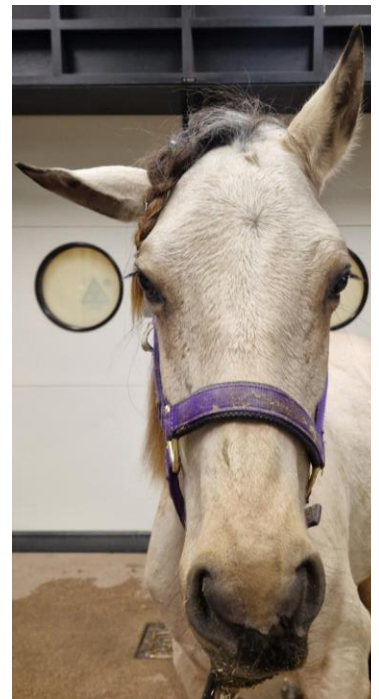
15

15

GENERAL ORDER OF STEPS

BEFORE SEDATION:

3. Evaluate symmetry from the front
 - Globe – orbit – pupils – eyelash direction – ear and lip position
4. Neuro ophthalmic exam
 - Menace response
 - Palpebral reflex
 - Dazzle reflex
 - Pupillary light reflexes



16



17

17

GENERAL ORDER OF STEPS

5. Schirmer tear test (STT) and/or tonometrie (IOP)
 - If indicated



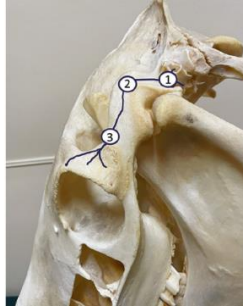
18

18

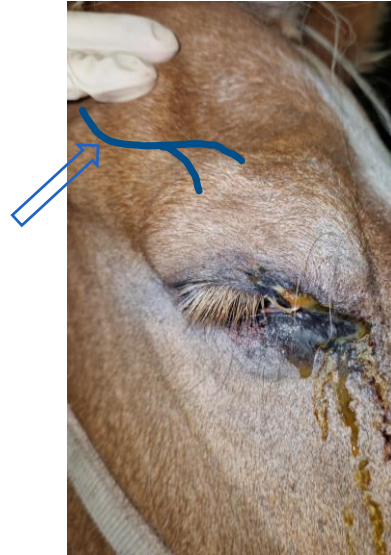
GENERAL ORDER OF STEPS

6. SEDATION

- ± palpebral nerve block
- ± topical anaesthetic



Equine Ophthalmology, 4th Edition. Brian C. Gilger (Editor).
ISBN: 978-1-119-78225-4 July 2022 Wiley-Blackwell, page 12.



GENERAL ORDER OF STEPS

7. Gross examination of the anterior segment via direct transillumination:

- Eyelids – conjunctiva - cornea – anterior chamber – iris



GENERAL ORDER OF STEPS

8. Detailed examination of the anterior segment via biomicroscopy, transillumination and retroillumination:



GENERAL ORDER OF STEPS

9. Induce mydriasis

- For purchase – 0.5% tropicamide
- Miosis in case of uveitis – 1% atropine



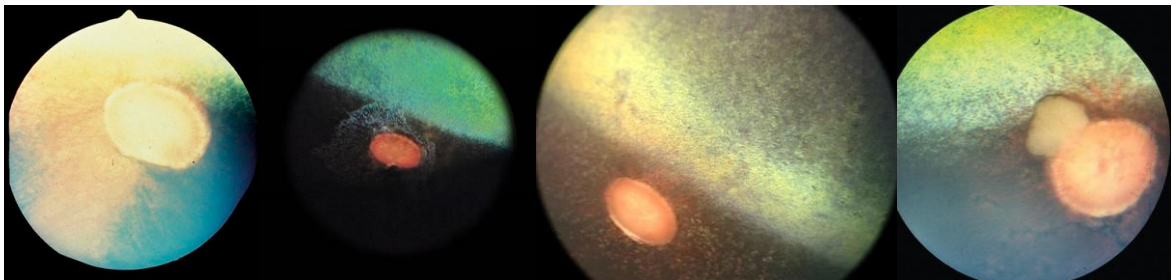
GENERAL ORDER OF STEPS

10. Detailed examination of the lens and vitreus via transillumination, retroillumination and biomicroscopy:



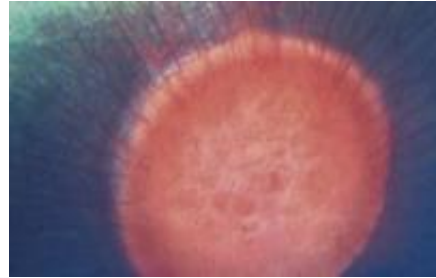
GENERAL ORDER OF STEPS

11. Perform direct and indirect ophthalmoscopy
 – Identify and examine optic nerve - retinal vasculature - nontapetal fundus - tapetal fundus



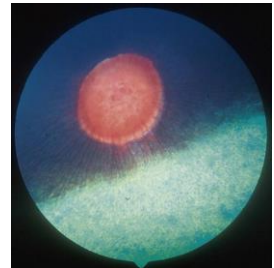
GENERAL ORDER OF STEPS

11. Perform direct and indirect ophthalmoscopy
 – Image upright and magnified several times



GENERAL ORDER OF STEPS

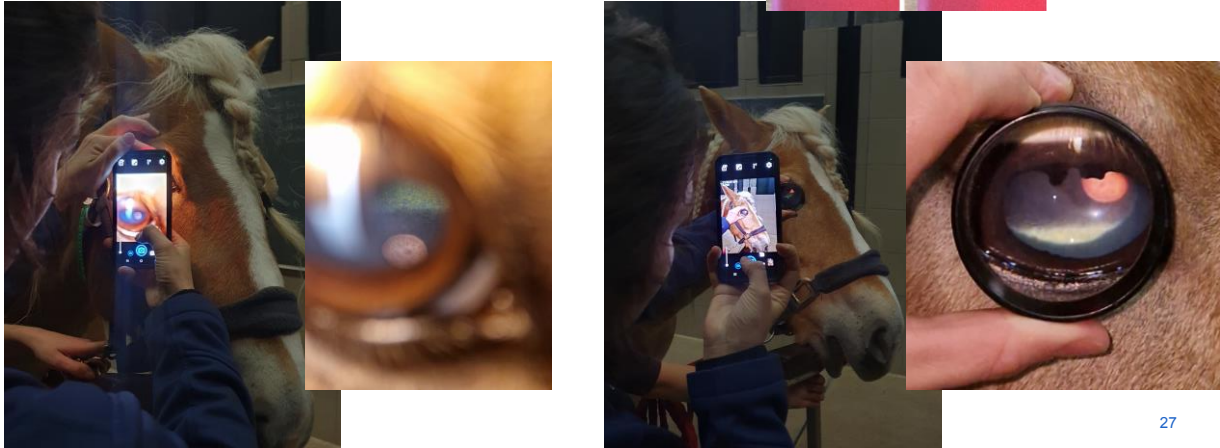
11. Perform direct and indirect ophthalmoscopy
 – Monocular or binocular
 – Image upside down and expanded view



GENERAL ORDER OF STEPS

11. Perform direct and indirect ophthalmoscopy

- Smartphone – “open camera”



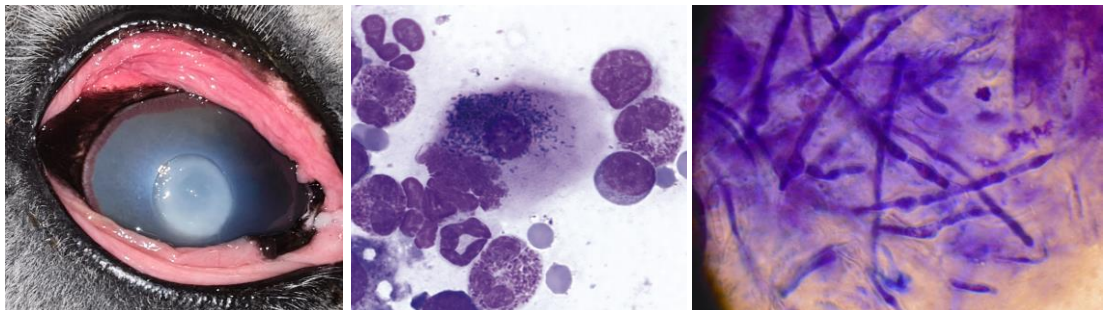
27

27

GENERAL ORDER OF STEPS

12. Sampling if indicated

- Culture
- Cytology



28

28

GENERAL ORDER OF STEPS

13. Topical fluorescein
14. Irrigate nasolacrimal duct(s) if indicated
15. Investigation of the third eyelid



29

29



Eline Vercruysse

DVM, Dipl ECVO, Phd student

DEPARTMENT OF LARGE ANIMAL SURGERY,
ANAESTHESIA AND ORTHOPAEDICS

E Eline.vercruysse@ugent.be

T +32 9 2647618

Universiteit Gent

@ugent

@ugent

Ghent University

www.ugent.be



30

What does it mean when gamma-GT is too high?

Emmanuelle van Erck-Westergren DVM, PhD, ECEIM, ECVSMR
Equine Sports Medicine Practice
83 avenue Beau Sejour, 1410 Waterloo, Belgium
evanerck@esmp.be

Blood sample analysis are routinely performed in equine sports medicine as part of the clinical follow-up of equine athletes. Changes in liver enzymes are not uncommonly identified in horses in training and the challenge for the practitioner is triple: understand the underlying cause for the elevation, define their clinical relevance and evaluate compatibility with performance. Regular blood sampling in the fit individual horse during a competitive season can help establish baseline values and identify more subtle abnormal deviations that would prompt further investigation. Because severe liver diseases usually develop on the long term and are debilitating, they are rarely diagnosed in active athletes. However due to the large functional reserve of the equine liver, active horses can sustain significant hepatic damage and still fail to display obvious clinical sign for a significant period. A comprehensive diagnostic approach, including a detailed medical history and physical examination is important in all cases and can lead to other laboratory investigations and ultrasound imaging.

Primary hepatic injury can be caused by various factors, including infectious diseases or exposure to toxic substances (medications, plants, or mycotoxins). Elevation in liver enzymes can also be linked to secondary, non-hepatic conditions such as musculoskeletal injuries, or metabolic or gastrointestinal disorders. Intense exercise, particularly endurance competitions or prolonged strenuous training, can also lead to temporary increases in liver enzyme levels.

GGT syndrome

« GGT syndrome » is a particular condition described in racehorses where serum GGT activity is elevated (>50UI/L) without a significant increase in other liver or muscle enzymes. The relationship of GGT syndrome with poor performance remains unclear and elevated GGT values have been reported in racehorses with decreased performances but also in presumably healthy performance horses. Altered demeanor and stamina, reduced time to fatigue and weight loss have also been reported [1], conditions typically associated to overreaching syndrome in humans.

In several recent studies, a series of analyses were conducted in fit Thoroughbred racehorses, including liver enzymes, vitamin E and selenium levels, viral load, and metabolomics and horses elevated GGT were compared to those with normal values [2,3]. Results suggested that high GGT could be related to excessive oxidative stress. Intense cumulative workload as well as increased racing frequency result in a marked increase in reactive oxygen species production. Oxidative stress occurs when the resulting oxidation is not fully compensated by endogenous or dietary antioxidants. As a result, GGT levels often return to normal values once the horses are rested and had fully recovered. Supplementation with antioxidants could contribute in reducing GGT. A genetic component has also been investigated but remains to be further clarified [3].

Exposure to toxins and dietary issues

Hepatic function can be challenged in response to the consumption of an imbalanced diet, particularly when there is a lack of knowledge in basic nutrition of performance horses and inadequate supplements are used cumulatively. However, the most common cause of perturbation in liver enzymes in adult sport horses probably originates from the consumption of forages contaminated with mycotoxin-producing microscopic molds. Durham has shown that over 80% of forages used in the UK contained mycotoxins, with a series of 10 that could be related to high GGT values and evidence of associated hepatic disease. Various fungi from the *Aspergillus*, *Penicillium*, *Alternaria* and *Fusarium*

species can produce harmful mycotoxins (aflatoxin, zearalenone, ochratoxins, trichothecenes, and fumonisins) [4]. As all the horses from the same stable are exposed to the same forage, blood tests should be conducted on several other individuals to confirm this hypothesis. The presence of fungi and mycotoxins can also be assessed by specific laboratory tests on the hay, straw but also on the cereals stored in silos. The presence of fungi can cause asthma and horses should be carefully assessed for concomitant respiratory disorders as these are a primary cause of poor performance.

Another source of intoxication can come from toxic plants (e.g., mugwort, clover, sunflowers...), ingested when horses have access to outdoor grazing. A thorough environmental and nutritional investigation is important to eliminate the potential sources of hepatotoxins.

Finally, the use of drugs that are metabolized in the liver, such as corticosteroids, benzimidazoles or antibiotics such as rifampin can be hepatotoxic and cause liver disease in susceptible horses.


Infections and infestations

Other possible causes hepatopathy outbreaks affecting several horses in the same barn include infectious pathogens. Elevated hepatobiliary enzymes are detected in hepatotropic virus carriers even in the absence of clinical disease. Yet a study failed to show an association between high GGT and SDH activities and viruses. According to context, testing for equine herpesvirus hepatitis, equine hepacivirus, and equine parvovirus is possible [5].

Inflammatory diseases of the digestive tract can lead to cholangiohepatitis. In this case, several liver enzymes will be elevated, and the horse can display clinical signs of digestive disease. Parasitic hepatitis (strongyles, ascarids) is common despite the use of anthelmintics, as resistant species are emerging. A review of the horse's worming program is usually insufficient proof of the absence of parasites and coprology should be performed regularly on samples harvested over 3 days. If negative, blood or saliva testing for parasites such as cyathostomes, tapeworms etc... can be considered.

In conclusion, several factors can cause temporary increase in liver enzymes in equine athletes; most are related to non-hepatic factors such as intense and cumulative exercise or environmental exposure to toxins. A comprehensive approach to this issue is necessary to avoid damage to the liver and digestive tract and limit impact on performance. Because therapeutic options are limited, training, dietary and environmental management are key in resolving the issue.

- [1] McGowan CM, Whitworth DJ. Overtraining syndrome in horses. *Comp Exerc Physiol* 2008;5:57–65. doi:10.1017/S1478061508979202.
- [2] Mann S, Ramsay JD, Wakshlag JJ, Stokol T, Reed S, Divers TJ. Investigating the pathogenesis of high-serum gamma-glutamyl transferase activity in Thoroughbred racehorses: A series of case-control studies. *Equine Vet J* 2022;54:39–51. doi:10.1111/evj.13435.
- [3] Peng S, Magdesian KG, Dowd J, Blea J, Carpenter R, Ho W, et al. Investigation of high gamma-glutamyltransferase syndrome in California Thoroughbred racehorses. *J Vet Intern Med* 2022;36:2203–12. doi:10.1111/jvim.16582.
- [4] Durham AE. Association between forage mycotoxins and liver disease in horses. *J Vet Intern Med* 2022;36:1502–7. doi:10.1111/jvim.16486.
- [5] Lyons S, Kapoor A, Schneider BS, Wolfe ND, Culshaw G, Corcoran B, et al. Viraemic frequencies and seroprevalence of non-primate hepacivirus and equine pegiviruses in horses and other mammalian species. *J Gen Virol* 2014;95:1701–11. doi:10.1099/vir.0.065094-0.

BEPS 
BELGIAN EQUINE PRACTITIONERS SOCIETY

**PSSM in horses:
What is it?**

Irene Tosi, MSc, PhD,
ECVSMR initial applicant
Center of sports medicine,
Faculty of Veterinary
Medicine, University of
Liège

1

PSSM stands for...

Polysaccharide Storage Myopathy
... also called...

Equine Polysaccharide Myopathy (EPSM)
Monday morning disease
Tying-up
Azoturia...

2

Myopathy? Genetic testing?

PSSM 1? Rhabdomyolysis? Hair?

P2? P3? P4? Blood?

MF? PSSM2?

3

Which kind of disease is PSSM?

- **MUSCLE** pathology (myopathy)
 - Occurs during **EXERCISE** (exertional)
 - **STORAGE** disorder
 - **GLYCOGEN** (polysaccharides)
- Cause of exertional ... Rhabdomyolysis => increased CK
Myopathy=> normal CK

4

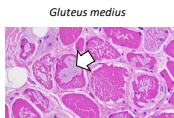
Polysaccharide Storage Myopathy: history

- Originally reported in QH and Belgian Drafts (90's)
- Now > 30 breeds described (Europe, UK, NA)
- Originally diagnosed by muscle biopsy

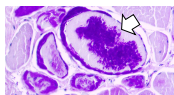


5

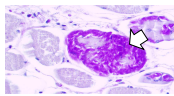
Hematoxylin and eosin



Periodic-acid-Schiff (PAS)



PAS + amylase digestion



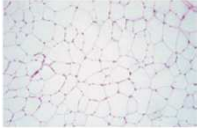
Gluteus medius

Polysaccharide inclusions in the muscle cell

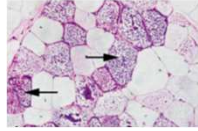
Not broken down in the normal manner (amylase)

6

Polysaccharide Storage Myopathy



Polysaccharides digested by amylase (amylase-sensitive):
type 1 PSSM



Polysaccharides not digested by amylase (amylase-resistant):
type 2 PSSM

7

This is an old classification!!!!

Things changed in 2008....

8

2008: PSSM has a genetic basis!

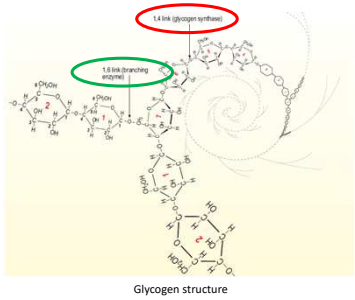
> *Genomics*. 2008 May;91(5):458-66. doi: 10.1016/j.jygeno.2008.01.011. Epub 2008 Mar 20.

Glycogen synthase (*GYS1*) mutation causes a novel skeletal muscle glycogenesis

Molly E McCue ¹, Stephanie J Valberg, Michael B Miller, Claire Wade, Salvatore DiMauro, Hasan O Akman, James R Mickelson

9

- GSY1 enzyme : α -1,4-bonds
- GBE: of α -1,6-bonds
- PSSM: gain-of-function mutation GYS1
- GYS1: permanently « switched-on »
- GBE normal activity



Glycogen structure

10


Can I test PSSM1?

YES!

11

PSSM1 or PSSM2?

- Test **positive**: PSSM1 independently on histopathology
- Test **negative**: PSSM2 with PSSM features on histopathology



12

PSSM 1	VS	PSSM2
<ul style="list-style-type: none"> • Known etiology • GYS1 mutation • Diagnosis: testing VS histopathology • Clinical signs <ul style="list-style-type: none"> • Acute ER • Chronic: lack of energy under the saddle, reluctance to move forward, stop to urinate • In SJ chronic back pain, failure to round over fences, pain upon palpation • Elevated CK/AST, even at rest • QH and European Draught Horses (+ others) 		<ul style="list-style-type: none"> • Unknown etiology • No (recognized) genetic mutation • Diagnosis: histopathology vs testing • Clinical signs <ul style="list-style-type: none"> • In QH and light breeds: chronic ER, abnormal CK/AST • WB: abnormal gait, sore muscles, undiagnosed HL lameness, stiffness, (ER less frequent), normal CK/AST • Arabians and WB (>80% and 100% of PSSM cases)

13

Can I test PSSM2?

NO!

14

Home Company Technology Blog Buy Contact

Learning Center > Tests > Polysacch

EquiSeq Has Developed Tests for the Predisposition to PSSM2/MFM

The tests will give a definite genotype.

The P2 (MYOT) test will give a genotype of n/n, n/P2, or P2/P2. Most n/P2 horses will develop late-onset PSSM2, some n/P2 horses will not develop PSSM2 even through the second decade of life. P2/P2 horses will develop early-onset PSSM2.

The P3 (FLNC) test will give a genotype of n/n, n/P3, or P3/P3. Horses that are n/P3 will develop late-onset MFM, a subtype of PSSM2. P3/P3 horses will develop early-onset PSSM2/MFM.

The P4 (MYO2) test will give a genotype of n/n, n/P4, or P4/P4. Horses that are n/P4 will develop late-onset MFM, a subtype of PSSM2. P4/P4 horses will develop early-onset PSSM2/MFM.

The P5 (PHRYOX1) test will give a genotype of n/n, n/P5, or P5/P5. Horses that are n/P5 will develop late-onset MFM, a subtype of PSSM2. P5/P5 horses will develop early-onset PSSM2/MFM.

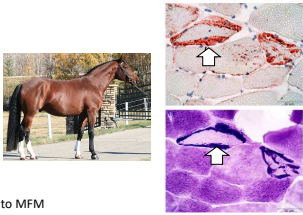
15

What is MFM?



16

Myofibrillar Myopathy

- Described in 2017 (Valberg)
- Histopathologically similar to MFM in humans
- Clinical signs:
 - Exercise intolerance
 - Stiffness, reluctance to move
 - Poorly localized lameness
 - CK/AST OK
- Diagnosis: histopathology
 - Myofibrillar degeneration
 - Desmin accumulation, PAS-positive
 - Z-disc and myofibrillar degeneration
- Potentially inheritable
- PSSM2 may be an early indicator of/predispose to MFM



17

 <p>MFM exists in humans</p> <p>MFM is due to mutations in genes encoding for structural proteins in the muscle cell</p> <p>PSSM2 could be an early stage of MFM</p> <p>Commercial testing are available in humans, can be applied in equids</p> <p>Horses can be tested for variant alleles in genes encoding for structural proteins : « P variants »</p> <p>Horses with P2 - P3a - P3b - P4 - P8 variants will develop early onset PSSM2-MFM</p>	 <p>It is a relatively rare disorder</p> <p>In 50% of MFM cases in humans the molecular basis remains unknown</p> <p>Not firmly established</p> <p>Genetic testing is regulated in humans, not in vet species</p> <p>P variants were present in equids before breed formation</p> <p>No association between any of the test variants with the presence of PSSM2/MFM. Sensitivity of genetic testing <33% (Valberg et al., 2021)</p>
---	--

18

The main problem is...

In human medicine several analyses (association, segregation analyses, modelling, functional studies) are necessary to determine that genetic variants identified by sequencing are actually **causative** of disease.

Unlike in human medicine, there is **no regulation** of veterinary genetic testing.

Source: Valberg et al., 2021

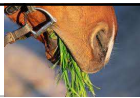
19

Take home message?

- PSSM1: you can test on EDTA blood or hair (with follicles)
- PSSM2: there's no **validated** genetic testing
- PSSM1 has a known etiology, not MFM nor PSSM2
- Commercial tests proposed for PSSM2 and MFM are **NOT** associated with histopathology
- PSSM2 remains a histopathological entity, probably the endpoint of different diseases (inherited or not...)
- PSSM2 and MFM may be pathologically linked

20

Treatments?



PSSM1


- Regular aerobic exercise (daily basis)
- Hay <12% NSC, NO concentrates or low NSC
- Limited access to grass
- Fat supplementation but prevent weight gain
- AA supplementation if symmetric topline atrophy

PSSM2

- Respond well to low NSC diet (QH)
- Less evidence of LS-HF diets effective for PSSM2
- Regular exercise is important but no clinical trial
- AA supplementation if topline symmetric atrophy


21

Prognosis



- At least 70% of horses with PSSM1 and PSSM2 well managed improve, many return to acceptable levels of performance
- 80% PSSM2 improve with advices for PSSM1 but 53% still not advancing as expected (reluctant to forward/collect) (Williams et al., 2018)
- Most PSSM1 and PSSM2 horses can be successful pleasure horses but they may not perform correctly in high level dressage or barrel racing

22



Comparative Neuromuscular Diseases Laboratory - Diagnostic Services

At RVC we have a dedicated laboratory providing a comprehensive range of diagnostic services for the evaluation of equine neuromuscular disease.

Validating a genetic test

When a test is used, it is essential to select a suitable panel for publication. The primary reason for this is to ensure that the test is accurate and reliable. This is done by the development of a test panel that is representative of the population of horses that are likely to be affected by the disease. This is done by identifying horses that are affected by the disease and then testing them for the presence of the mutation. The results of these tests are then compared to the results of the test panel to ensure that the test is accurate and reliable.

PSSM Type 1 (PSSM1)

PSSM1 is a genetic muscle disease that causes horses to have episodes of stiffness, muscle tremors and to lose weight. It is caused by a mutation in the *PP1R3B* gene. The mutation is inherited in an autosomal recessive manner. The test panel for PSSM1 consists of 10 horses that are affected by the disease and 10 horses that are not affected by the disease. The results of these tests are then compared to the results of the test panel to ensure that the test is accurate and reliable.

PSSM Type 2 (PSSM2)


PSSM2 is a genetic muscle disease that causes horses to have episodes of stiffness, muscle tremors and to lose weight. It is caused by a mutation in the *PP1R3B* gene. The mutation is inherited in an autosomal recessive manner. The test panel for PSSM2 consists of 10 horses that are affected by the disease and 10 horses that are not affected by the disease. The results of these tests are then compared to the results of the test panel to ensure that the test is accurate and reliable.

[RVC Comparative Neuromuscular Diseases Laboratory - Diagnostic Services](#)

23

Thank you for your attention


24



Update on West-Nile virus

Dr Carla Cesarini

PhD, Dipl. ACVIM-LA, Dipl. ECEIM
EBVS® Veterinary Specialist in Equine Internal Medicine



1



WEST NILE VIRUS An Update

- Brief review of the disease
- Epidemiology:
 - Current situation in Europe and Belgium
- Implications for the equine practitioner
 - How to recognize the disease?
 - How to confirm the diagnosis?
 - How to treat a horse with WNV?
 - Prognosis and prevention
- Take home messages



Carbon Black and White Line Drawing of a Doctor Horse
©2005 by Don Lehmman

2



WEST NILE VIRUS For further details



**JOURNAL OF
VETERINARY INTERNAL MEDICINE**
Open Access

CONSENSUS STATEMENT |  Open Access | 

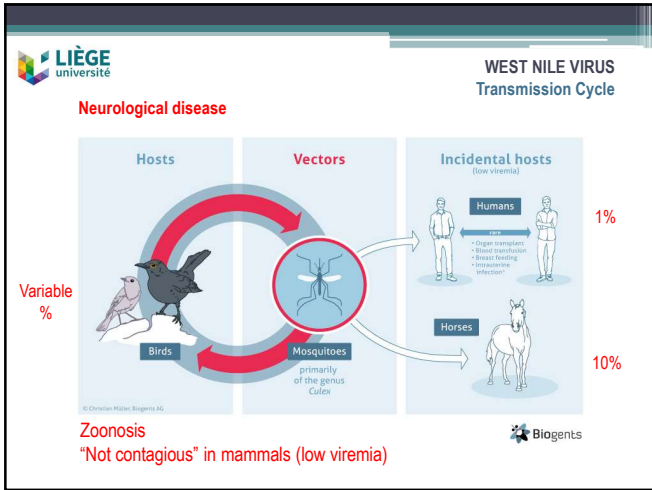
**European College of Equine Internal Medicine consensus
statement on equine flaviviridae infections in Europe**

Jessika M. V. Cavalleri, Orsolya Korbacska-Kutasi, Agnès Leblond, Romain Paillot, Nicola Pusterla,
Eike Steinmann, Joy Tomlinson

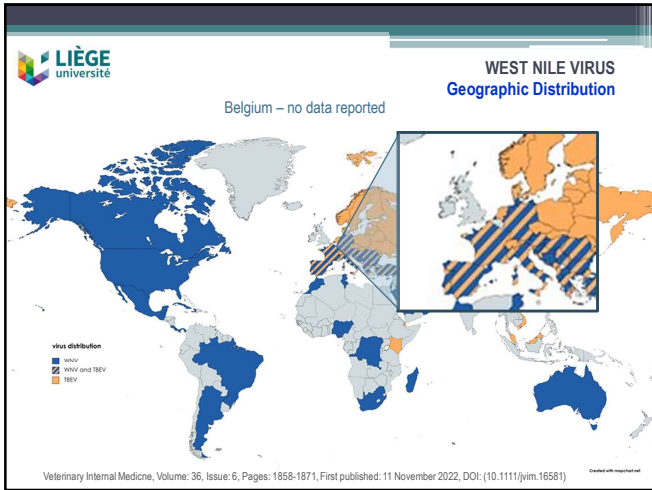
First published: 11 November 2022 | <https://doi.org/10.1111/jvim.16581> | Citations: 2

Consensus Statement
Open Access
2022

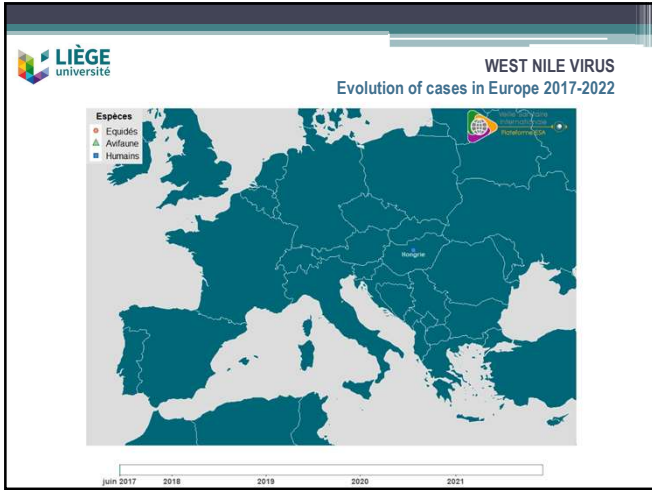
3



4



5



6

LIÈGE université

WEST NILE VIRUS
How to recognize the disease?

Courtesy of Dr Eduard José Cunilleras, Universitat Autònoma de Barcelona

10

LIÈGE université

WEST NILE VIRUS
How to confirm the diagnosis?

ANTEMORTEM

- Serum : anti-WNV IgM MAC-ELISA
IgG (x4, cross-reactions)
- Cerebro Spinal Fluid : anti-WNV IgM MAC-ELISA

PCR? *Less useful*
If done, EARLY after clinical signs

Viral isolation?

Fig. 2 Viremia and antibody kinetics in West Nile virus infection

Zeller HG et al. Eur J Clin Microbiol Infect Dis. 2004.

11

LIÈGE université

WEST NILE VIRUS
How to confirm the diagnosis?

ANTEMORTEM

Clinical signs	Risk status	Biochemistry, CBC, CSF	Serology
Low grade fever Ataxia Muscle fasciculations Weakness	Endemic region Mosquito season Inadequate vaccine	Rule out organ dysfunction, e.g. liver Rule out other neurologic causes	WNV IgM-capture ELISA WNV IgG

Flowchart for Serology:

- IgG+ IgM-**: Historical vaccination, Historical infection, Cross-reaction with other flavivirus. *→ Virus neutralization test to confirm flavivirus. Or retest in 1 week if strongly suspect WNV. Or test CSF.*
- IgG+ IgM+**: Recent vaccination, Recent infection.
- IgG- IgM+**: Recent vaccination, Recent infection.
- IgG- IgM-**: WNV negative.

12

LIÈGE université

WEST NILE VIRUS
How to confirm the diagnosis?

POSTMORTEM

WNV RNA detected by RT-PCR in:
fresh or formalin-fixed neural tissue

thalamus, hypothalamus, pons/medulla and spinal cord

13

LIÈGE université

WEST NILE VIRUS
Nationally Reportable Disease

AFSCA
Agence fédérale pour la sécurité de la chaîne alimentaire

FAVV
Federaal Agentschap voor de veiligheid van de voedselketen

EFPPB
EQUI FOCUS POINT BELGIUM

DECLARE

14

LIÈGE université

WEST NILE VIRUS
How to treat a horse with WNV?


- Most horses can fully recover with at-home care
- 30% of horses may experience a relapse of signs after initial improvement
- In severe cases, horses require hospitalization
- Supportive treatment:
 - Fluid therapy
 - Nonsteroidal anti-inflammatory agents (flunixin-meglumine: 1.1 mg / kg, q12h IV)
 - Short-acting glucocorticoids (dexamethasone sodium: 0.05-0.1 mg/kg q24h IV)
 - Other as needed:
 - Slings to reduce bed sores
 - Head and leg protection
 - Assisted feeding

15

LIÈGE université

WEST NILE VIRUS
Prognosis and Prevention

- **Prognosis**
 - survival rate is high (55%-70%) compared with other infectious encephalitis
 - 10-40% of horses will have residual neurologic deficits
- **Prevention**
 - Mosquito Control
 - Vaccines : 2 available in Belgium
 - ✓ Safe and protective against severe illness
 - ✓ Protective antibody levels 6 to 12 months
 - Need to be administered before exposure
 - ✗ Some horses respond poorly, WNNND can occur in vaccinated animals
 - ✗ Interferes with diagnosis: IgM antibodies up to 52 days after vaccination



16


LIÈGE université

WEST NILE VIRUS
Take Home Messages

- Equine cases in Belgium are not reported *but could emerge with time.*
- One horse on ten infected by WNV will develop neurological disease
...but neurological sequels and dead are possible.
- Horses affected by WNV are not contagious to other horses or people
...but EHV1 neurological form are, so isolate until confirmation.
- Declare the case: *horses serve as epidemiological sentinels.*
- Specific antibodies (IgM) are more useful than PCR for antemortem diagnosis...*but vaccination may interfere with IgM titers during 2 mo.*
- Vaccine is safe and effective. Interest of use may vary depending on exposure risk (travelling horses?).

17

LIÈGE université



Creator: Anderson, Nick. 1995. Date: 2001-08-30. Finding number: CGA.A.C.PE.140

18
