Emorragia e scolo nasale: un challenge diagnostico. Casi clinici significativi

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Giornate Boehringher Ingelheim 2018

Epistassi e scolo nasale sono un sintomo clinico, non una patologia a se stante

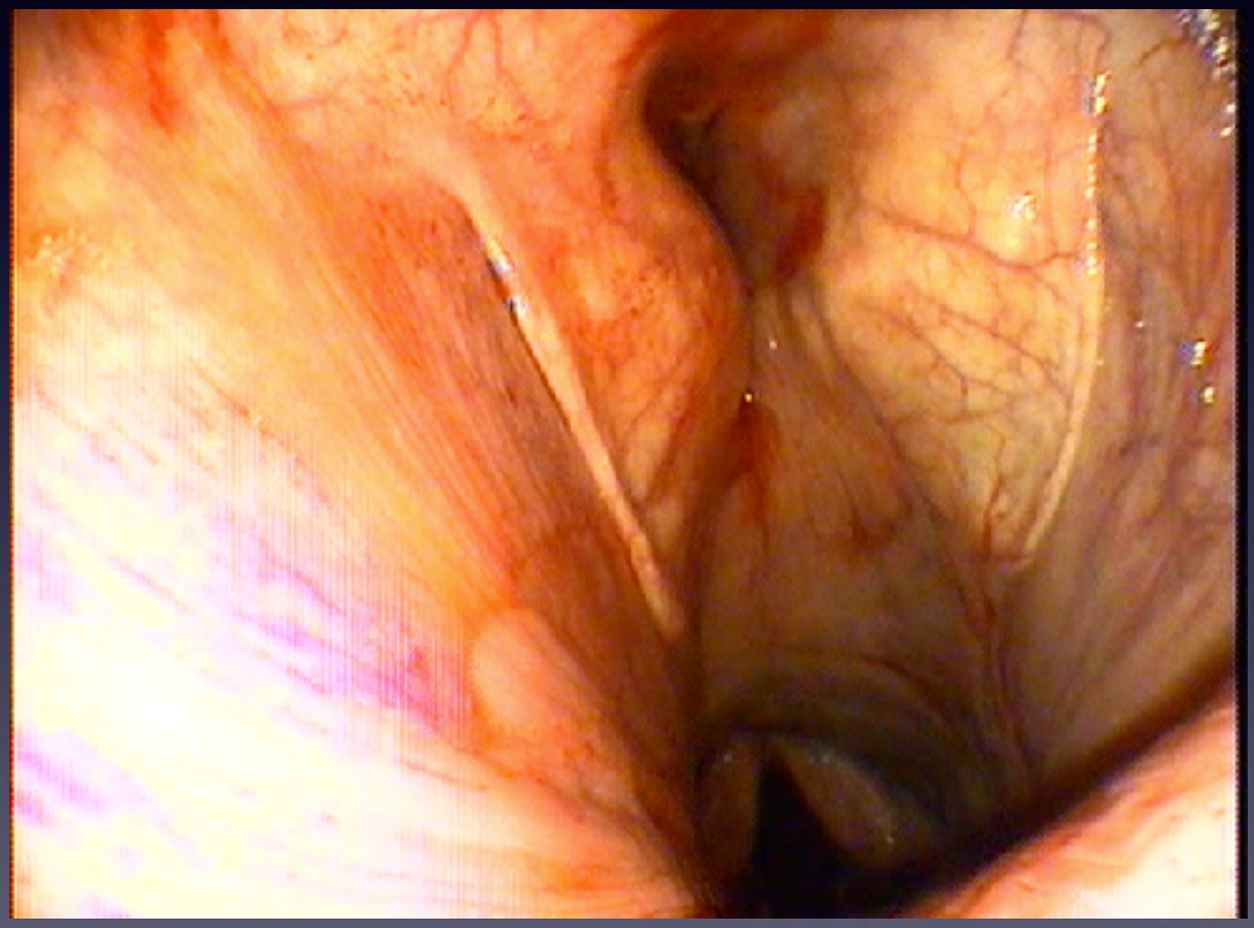
- Anamnesi
  - Durata, volume, uni- o bi-laterale, colore
- Visita clinica completa (situazione cardio-vascolare!)
- Endoscopia
- Ecografia
- Radiografia
- Tac/ RM
- Biopsia ed esame istologico

	ORIGINE					
SEGNI CLINICI	Cavità nasale e seni	Naso faringe	Tasche gutturali	Polmoni	Cavità pleurica	**TECNICHE D'ESAME**
EPISTASSI (monolaterale)	+++	++	+++	+	-	Esame clinico, endoscopia, rx., esami sangue
EPISTASSI (bilaterale)	+	++	+++	+++	+	Esame clinico, endoscopia, rx, ecografia, esami sangue
SCOLO (monolaterale)	+++	++	+++	+	-	Esame clinico, endoscopia, rx. Tampone, ago aspirato, biopsia
SCOLO (bilaterale)	++	++	+	++	+	Esame clinico, endoscopia, rx, ecografia
DISFAGIA	-	+++	+++	-	-	Esame clinico, endoscopia, rx, Tac, ecografia

Caso clinico n° 1

- Castrone 12 anni, da passeggiata
- Epistassi mono laterale di lieve entità da vari giorni
- FC:44, FR:14, Mucose: Rosa, Sensorio: Vigile, Temp: 37,6°,
- HT:34, PT:7,0, Lattato: 1,5, WBC:8,1 Neutrofili 67%, Fibrinogeno: 307
- Ascoltazione normale

Caso clinico n° 1



DX

SN

EMATOMA PROGRESSIVO DELL'ETMOIDE

- È caratterizzata da una massa incapsulata che origina normalmente dalla submucosa dell'etmoide. Tende a crescere progressivamente, è la più comune massa della testa del cavallo. Più frequentemente riscontrata in cavalli PSA e PSI di mezza età (8-12 anni), è caratterizzata da scolo nasale serosanguigno monolaterale. La diagnosi richiede esame endoscopico del naso faringe.

Caso clinico n° 1

ULTERIORE APPROFONDIMENTO DIAGNOSTICO

- RX
- TAC

Caso clinico n° 1

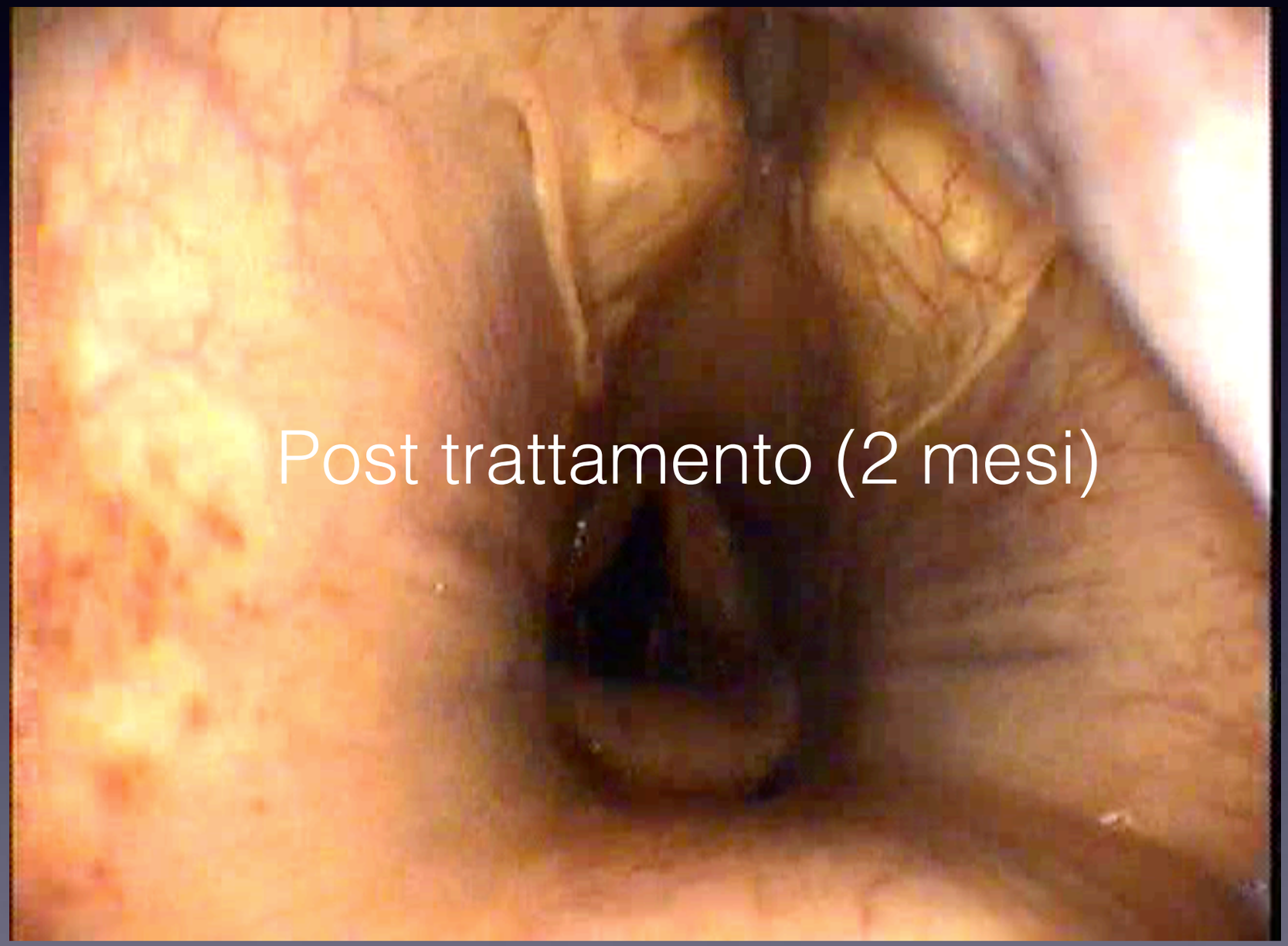
TERAPIE

- Ablazione chimica (formalina 4%) per via endoscopica. Controllo endoscopico dopo 3/4 settimane
- Nd:YAG laser per via endoscopica, lesioni di massimo 5 cm
- Ablazione chirurgica

Caso clinico n° 1

Post trattamento (1 mese)

Caso clinico n° 1



Post trattamento (2 mesi)

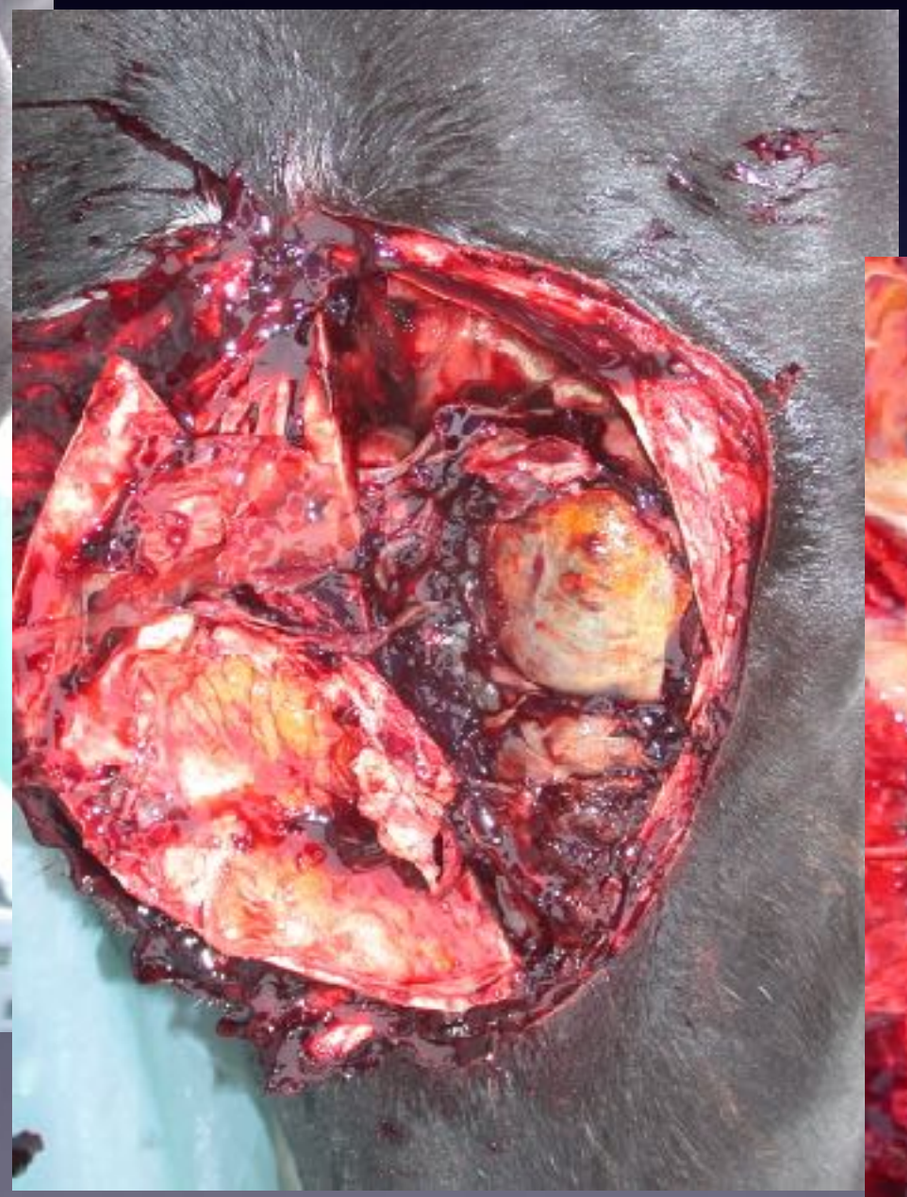
Caso clinico n° 1

PROGNOSI

- Indipendentemente dalla terapia scelta, la prognosi a lungo termine è riservata, per il rischio di recidiva, stimato, in varie pubblicazioni, attorno al 45%.

- È raccomandato un controllo endoscopico ogni 3/6 mesi, per almeno 5 anni

EMATOMA ETMOIDE

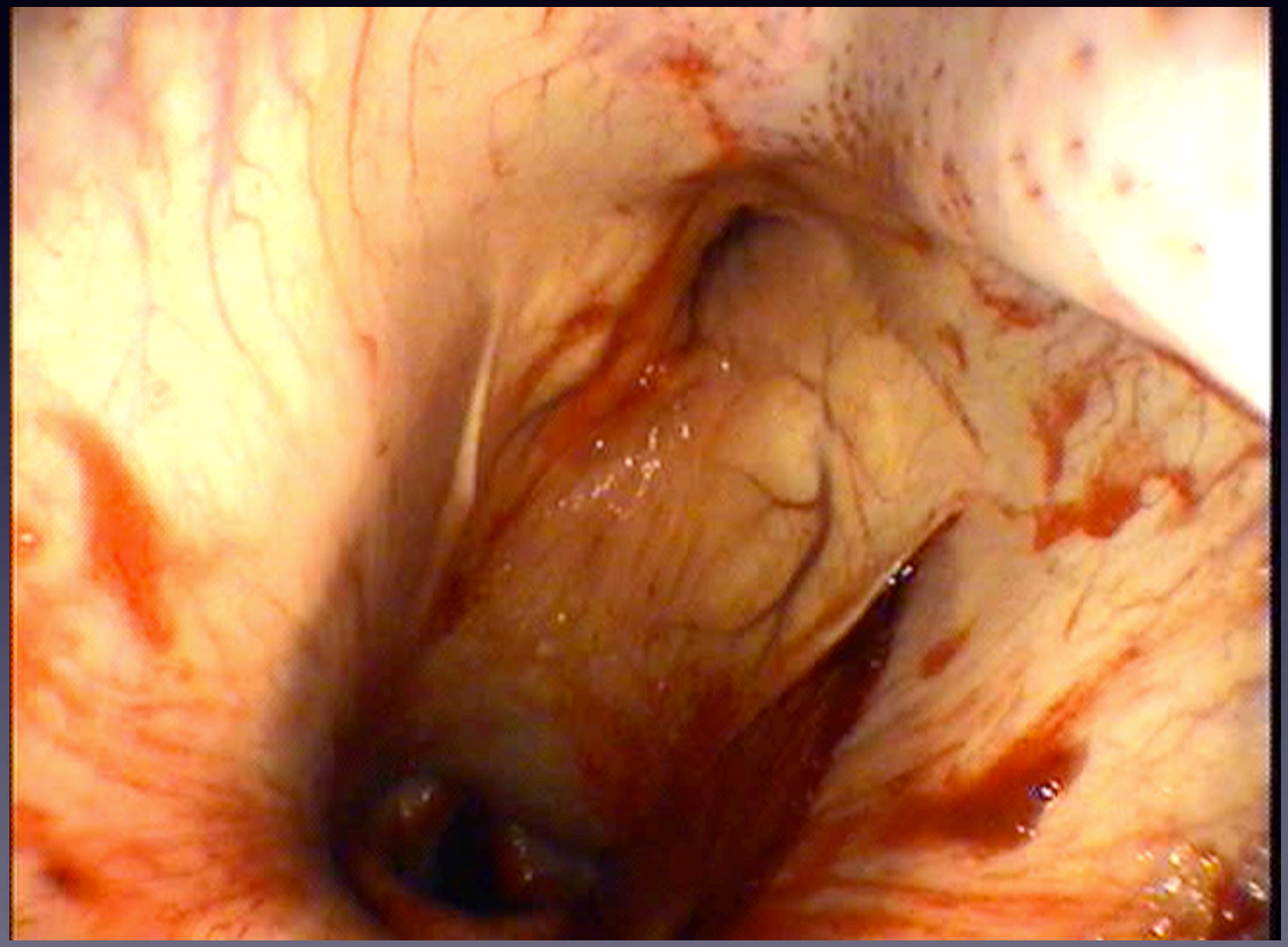


Post mortem

Caso clinico n° 2

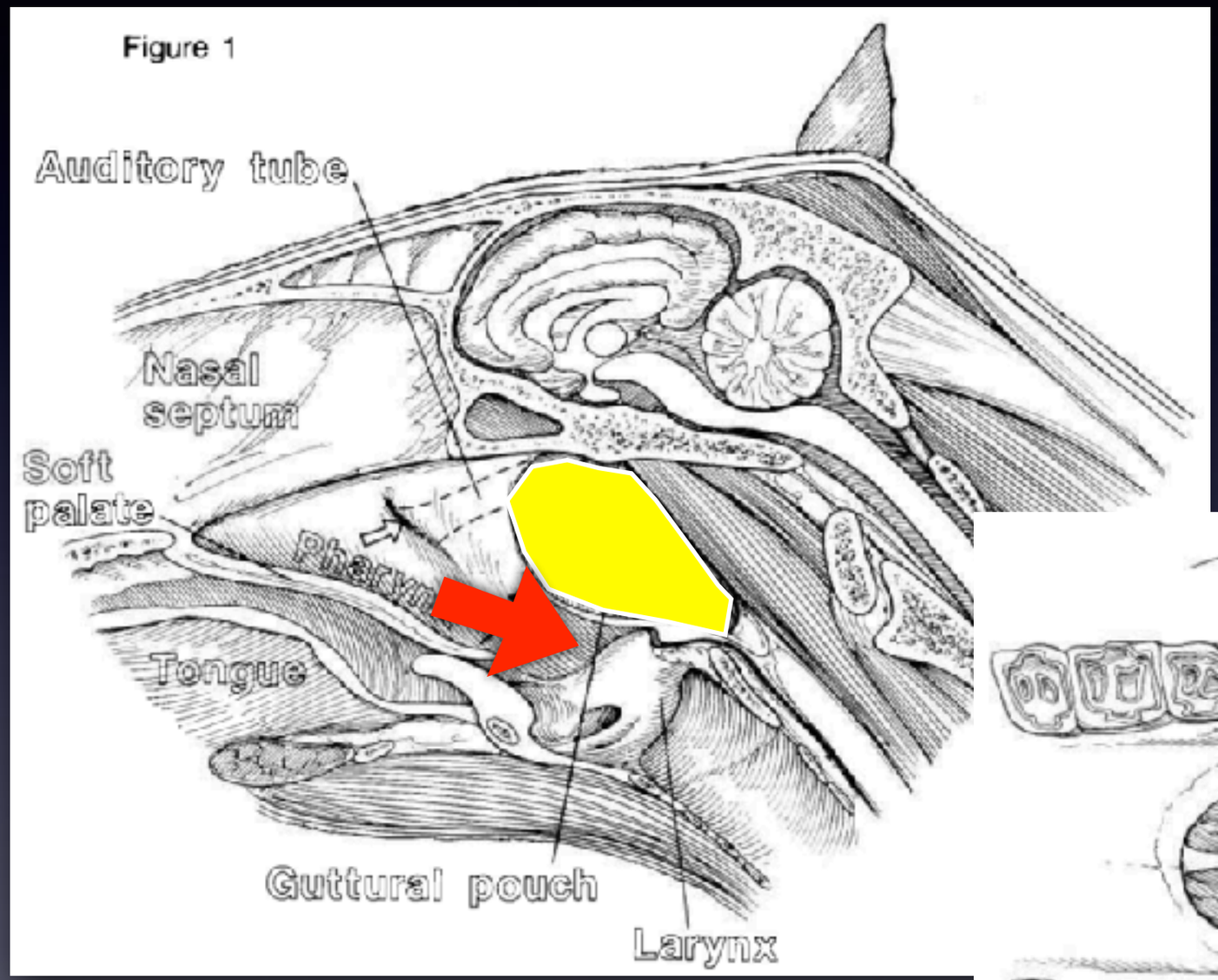
- Femmina, 13 anni, PSA
- Epistassi di media entità, bilaterale da un giorno
- FC:48, FR:14, HT:34, PT:7.0
- Ascoltazione normale

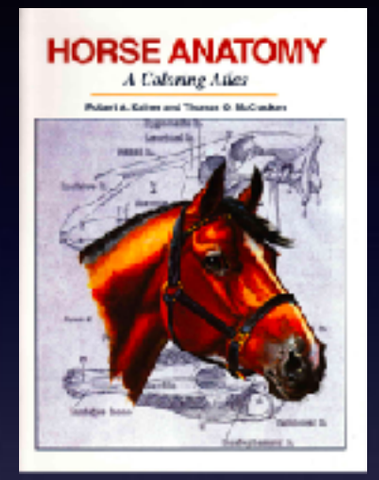
Caso clinico n° 2



Caso clinico n° 2

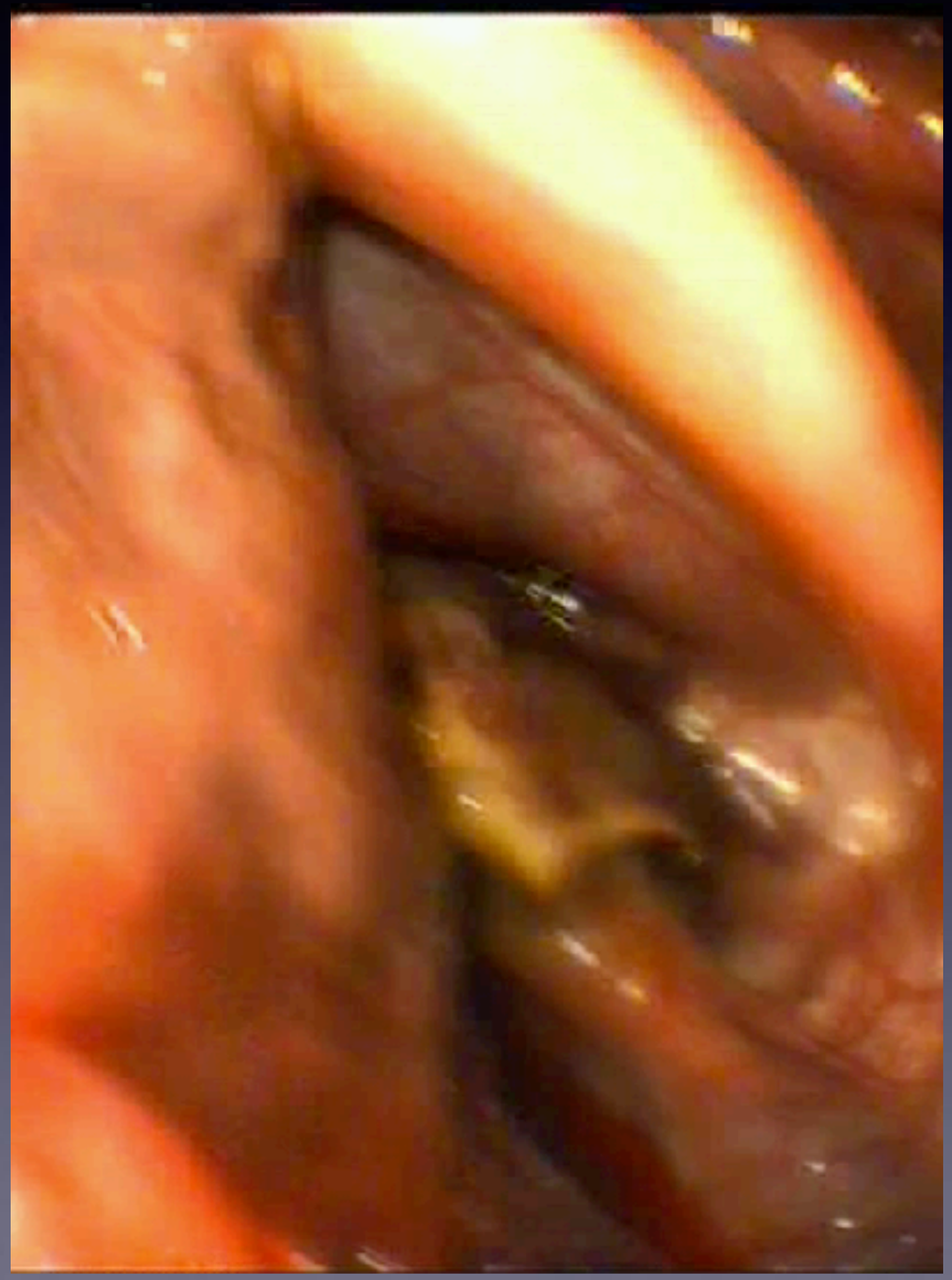
Caso clinico n° 2





Caso clinico n° 2

Caso clinico n° 2



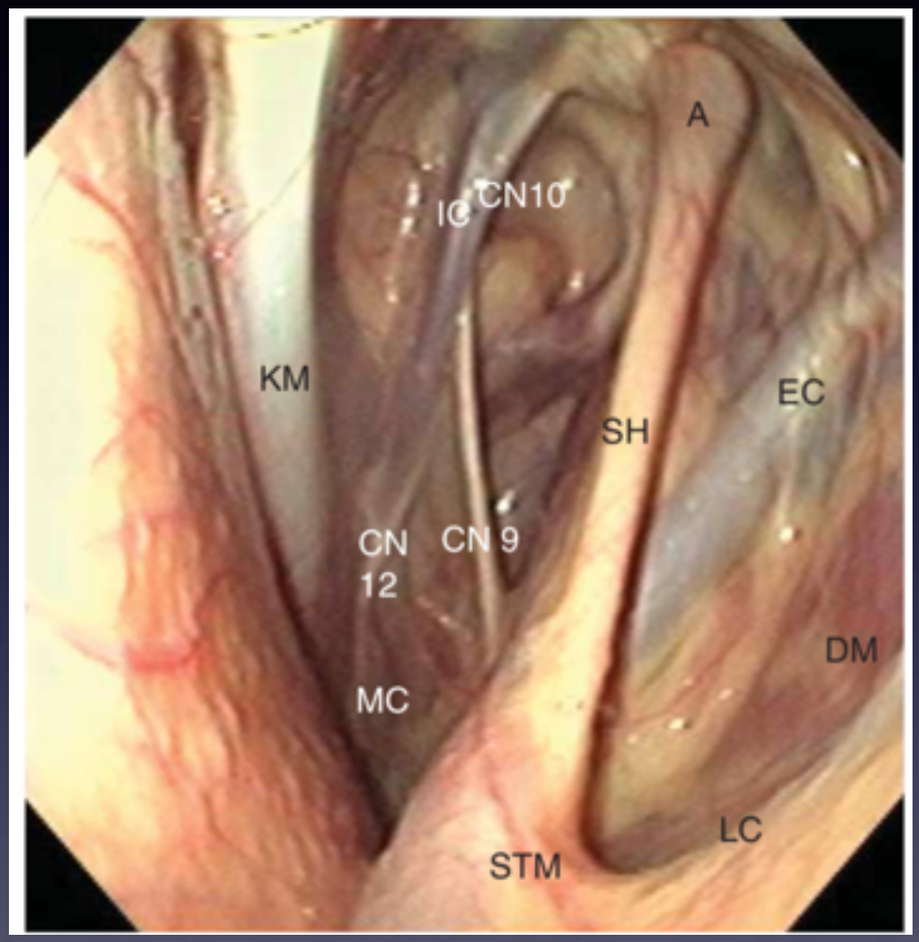
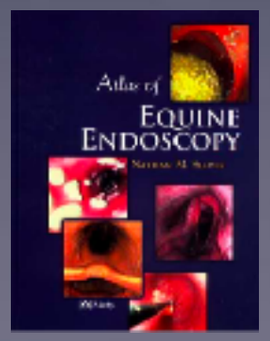


FIGURE 5-5. Normal left guttural pouch. *A,* Articulation of stylohyoid and petrous part of temporal bone; *CN9,* cranial nerve 9; *CN10,* cranial nerve 10; *CN12,* cranial nerve 12; *DM,* digastricus muscle; *EC,* external carotid artery; *IC,* internal carotid artery; *LC,* lateral compartment; *LCM,* long capitis muscle; *MC,* medial compartment; *SH,* stylohyoid bone; *STM,* stylopharyngeus muscle.



Caso clinico n° 2

TERAPIE

- Intervento chirurgico di occlusione del vaso interessato (varie tecniche)
- Trattamento topico con fungicidi
- Chiusura carotide comune

Caso clinico n° 2

PROGNOSI

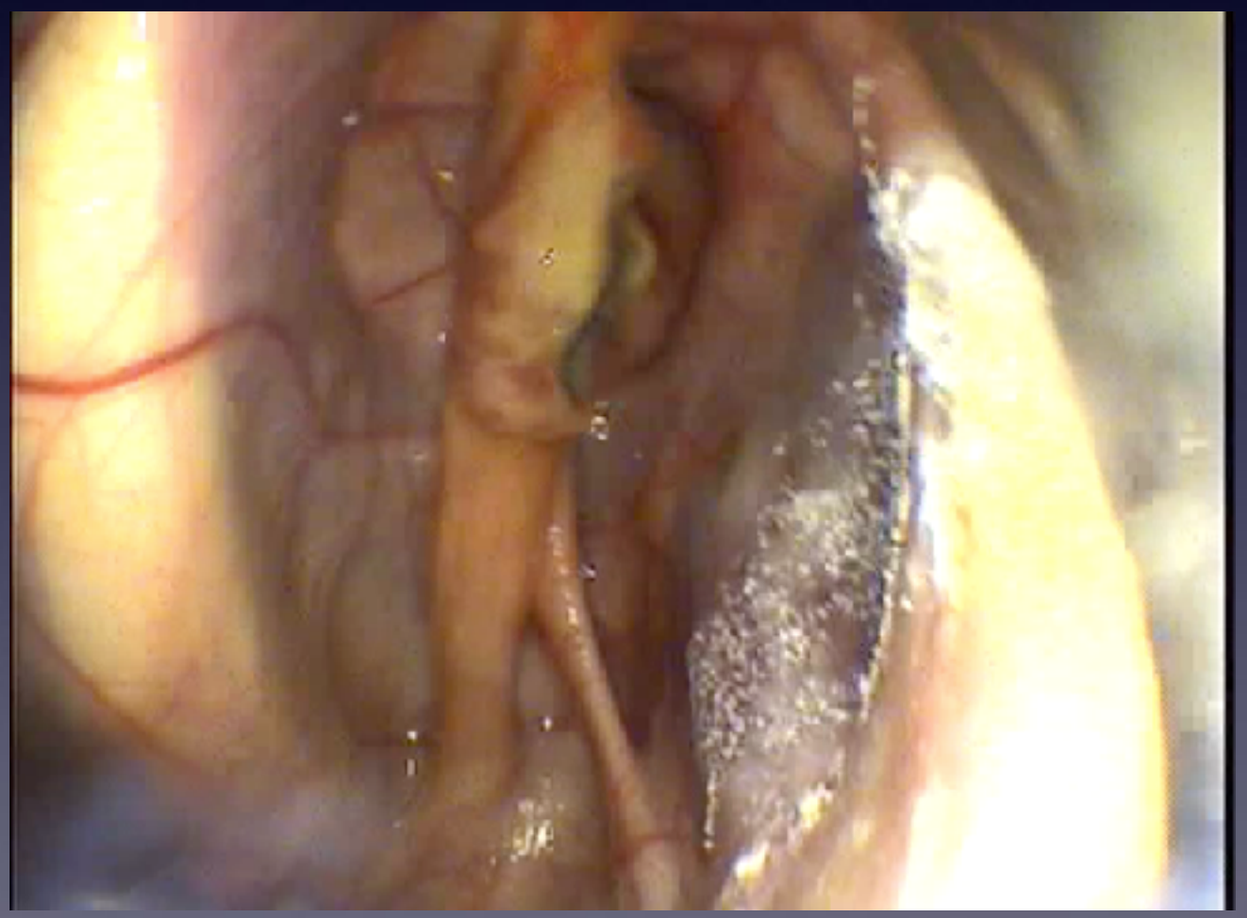
- 47,8% mortalità senza intervento chirurgico

  Cook, W. R., et al. (1968) The pathology and aeti- ology of guttural pouch mycosis in the horse. Vet Rec 83, 422–428, Freeman, D. & Hardy, J. (2012) Guttural pouch. In Equine Surgery. 4th edn. Eds J. A. Auer, J. A. Stick. St Louis: Saunders. pp 623–642

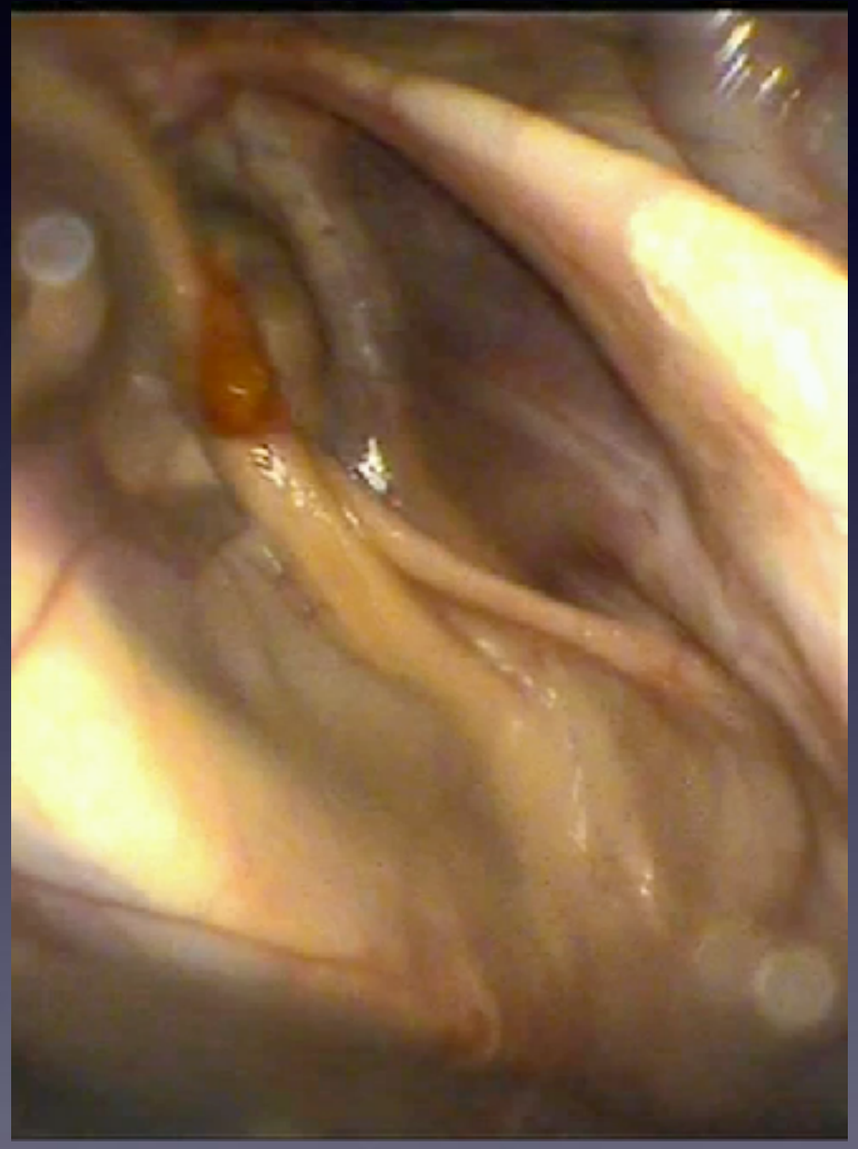
- 16,6% mortalità con legatura carotide comune e trattamento topico
  M. Cousty, et al. (2016) Ligation of the ipsilateral common carotid artery and topical treatment for the prevention of epistaxis from guttural pouch mycosis in horses. Vet Rec. 2016 Jan 9;178(2):44

- 0%-11% mortalità con varie tecniche chirurgiche

Caso clinico n° 2



13 giorni post it



24 giorni post it

Caso clinico n° 2

THE VETERINARY CLINICS Equine Practice

Vet Clin Equine 19 (2003) 123–158

Diseases of the guttural pouches

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Anatomy

Guttural pouches are large diverticula of the eustachian tubes that connect the pharynx to the middle ear. They are present in perissodactyls, such as equids, tapirs, some species of rhinoceros (except for the white rhinoceros), some bats, a South American forest mouse, and hyraxes [1–3]. Each pouch has a capacity of approximately 300 to 500 mL and is lined with a pseudostratified ciliated epithelium containing goblet cells [4]. Several mucous glands as well as lymphoid nodules are present in the young horse. Each guttural pouch is separated into a medial and lateral compartment by the ipsilateral stylohyoid bone, over which each pouch is draped. The epithelium overlies several vascular, neural, and lymphoid structures, and knowledge of these structures is important in understand-

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22nd SIVE INTERNATIONAL CONGRESS

GLI EFFETTI E L'UTILIZZO DELL'OSSIGENO IPERBARICO NELL'ASPERGILLOSI DELLE TASCHE GUTTURALI DEL CAVALLO: STUDIO PRELIMINARE

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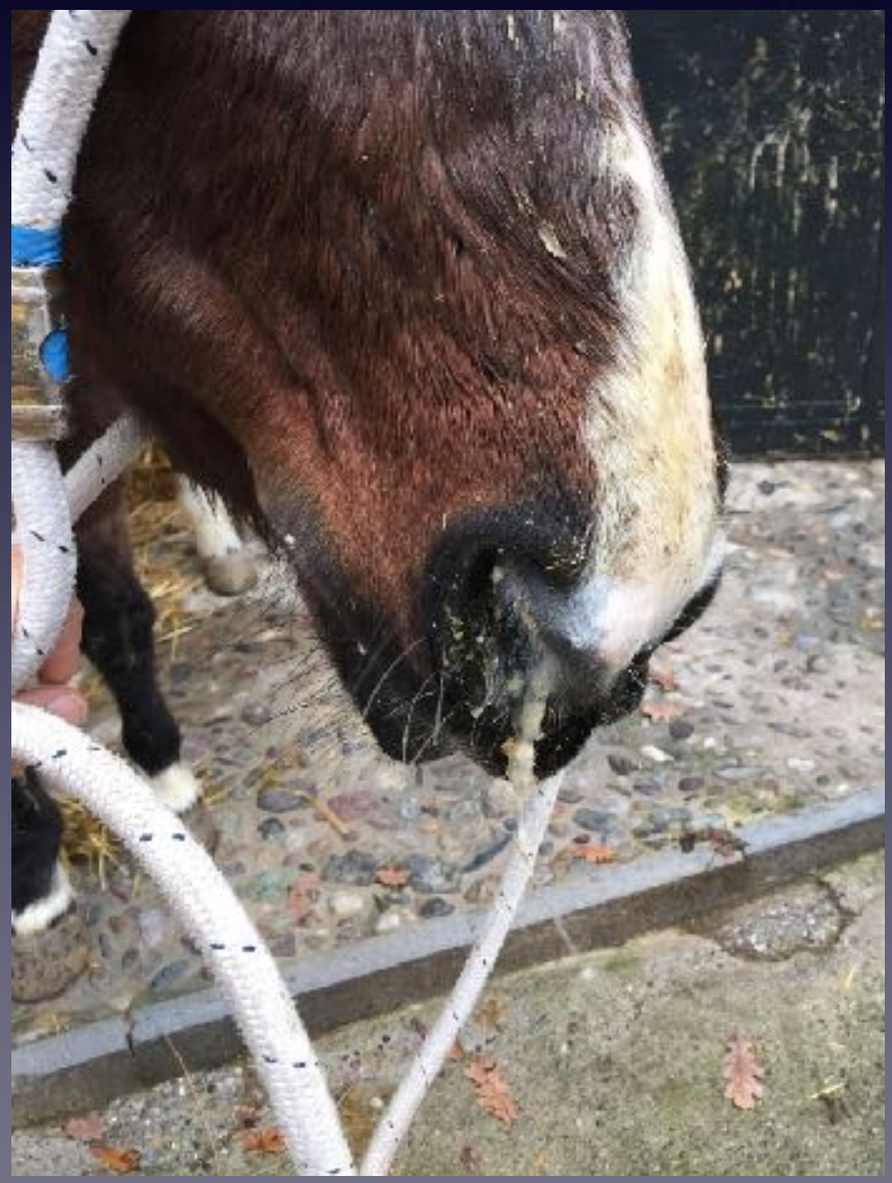
Tipologia: **Ricerca Originale**
Area di interesse: **Chirurgia**

Aspetto pratico

Caso clinico n° 3

- Femmina, 15 anni, al paddock
- Scolo nasale bilaterale e disfagia
- FC:50, FR:24, Mucose: Rosa, Sensorio: Vigile, Temp: 37,8°,
- HT:36, PT:7,2, Lattato: 1,3, WBC: 7,6 Neutrofili 68%, Fibrinogeno: 235
- Qualche rantolo

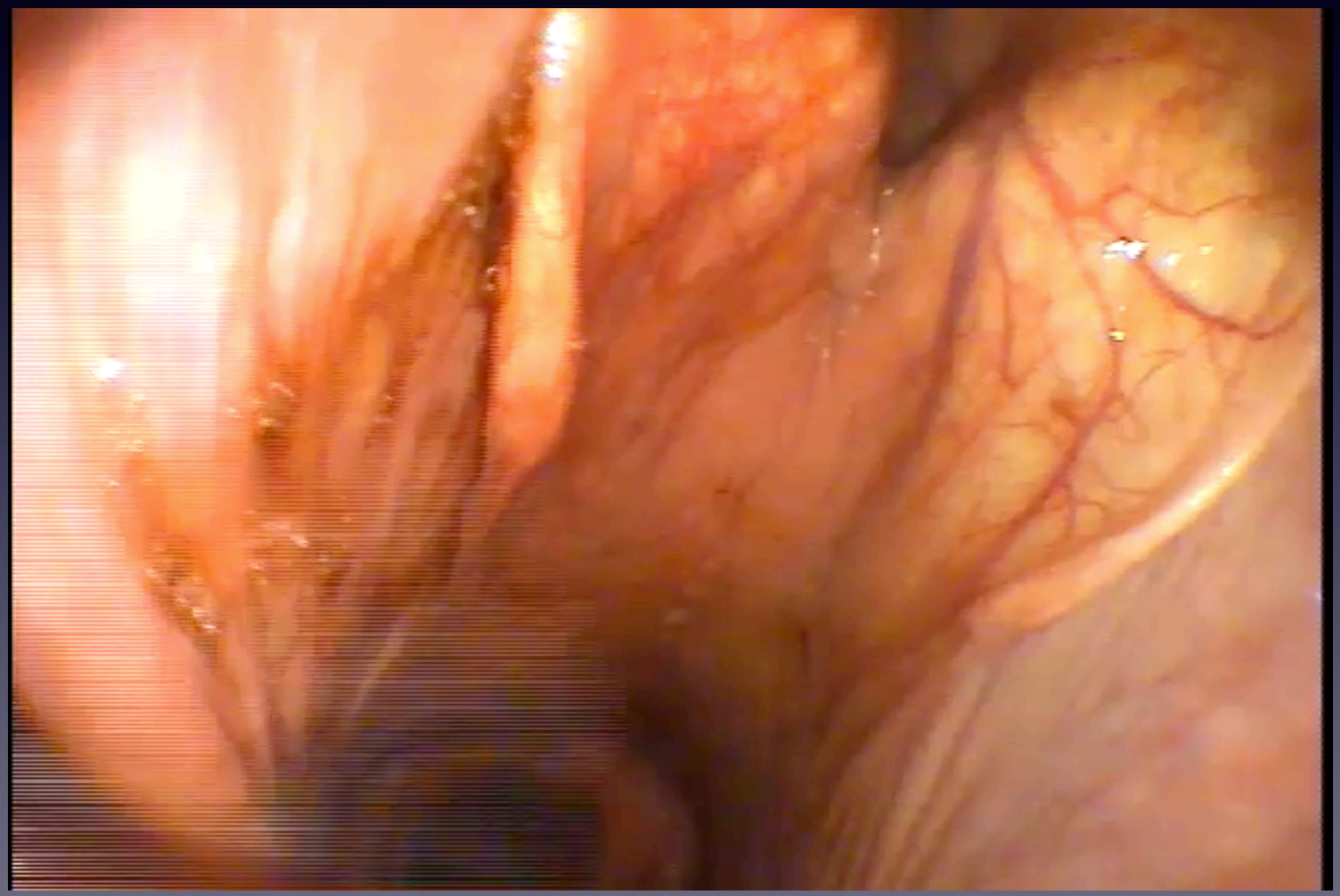


Caso clinico n° 3

Caso clinico n° 4

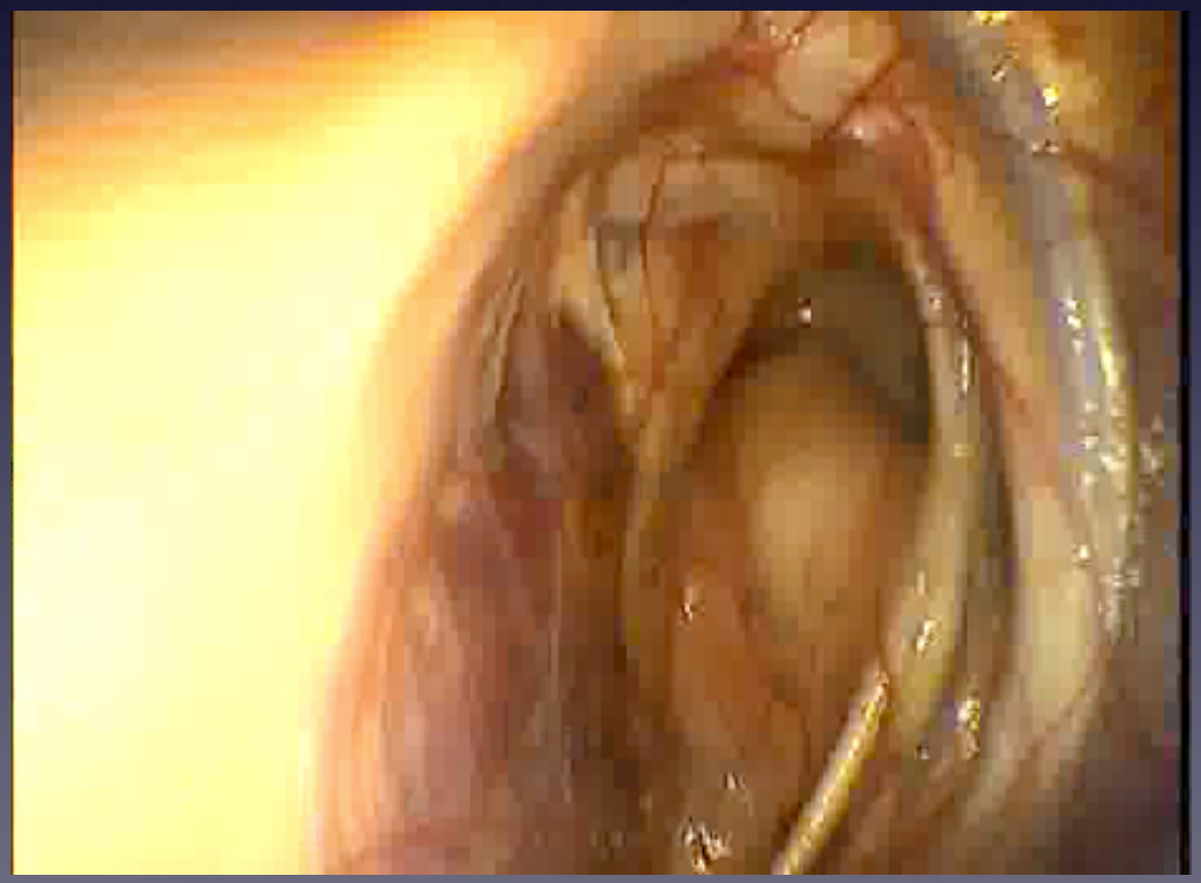
- Castrone, 7 anni, trotter, in lavoro
- Epistassi lieve, monolaterale, da 1 giorno
- FC:44, FR:12, Mucose: Rosa, Sensorio: Vigile, Temp: 37,8°,
- HT:36, PT:7,4, Lattato: 1,2 WBC:7,7 Neutrofili 71%, Fibrinogeno: 345
- Ascoltazione polmonare normale

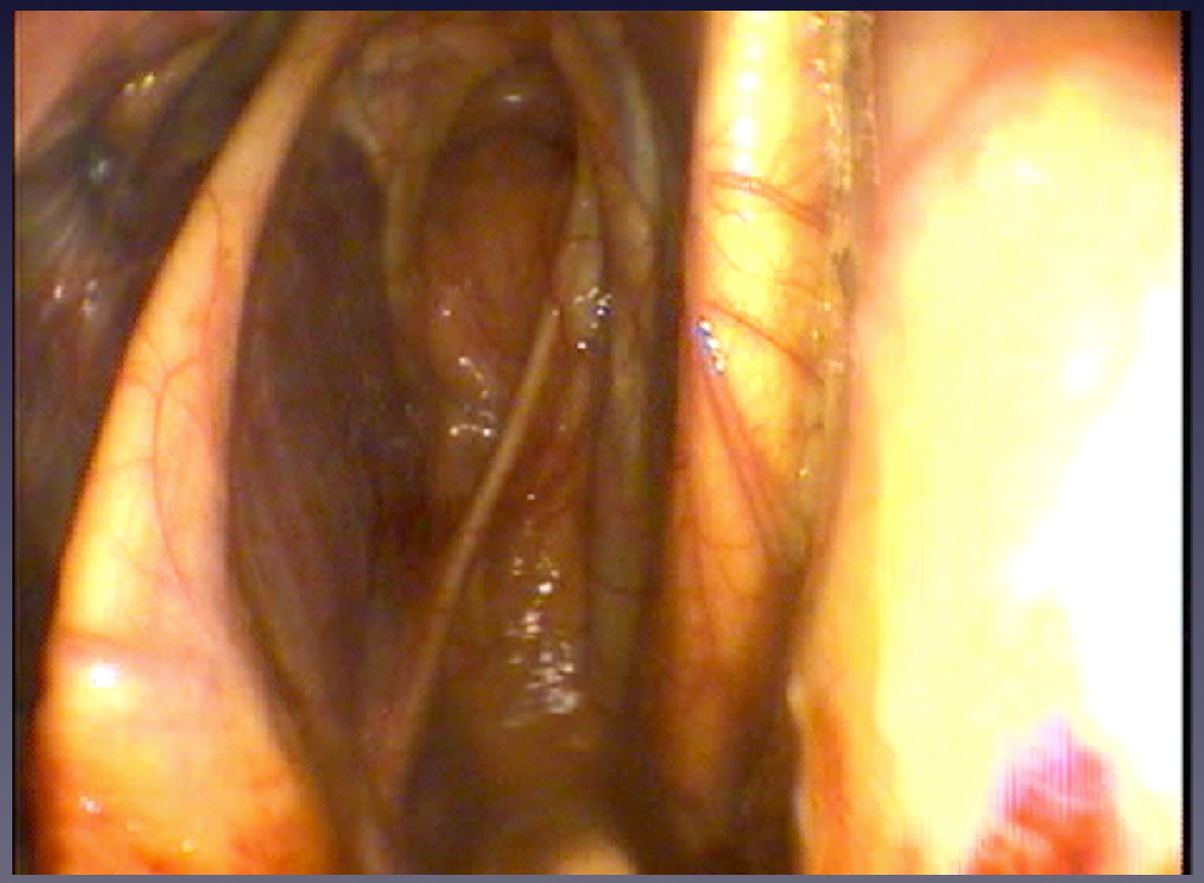
Caso clinico n° 4



Caso clinico n° 4

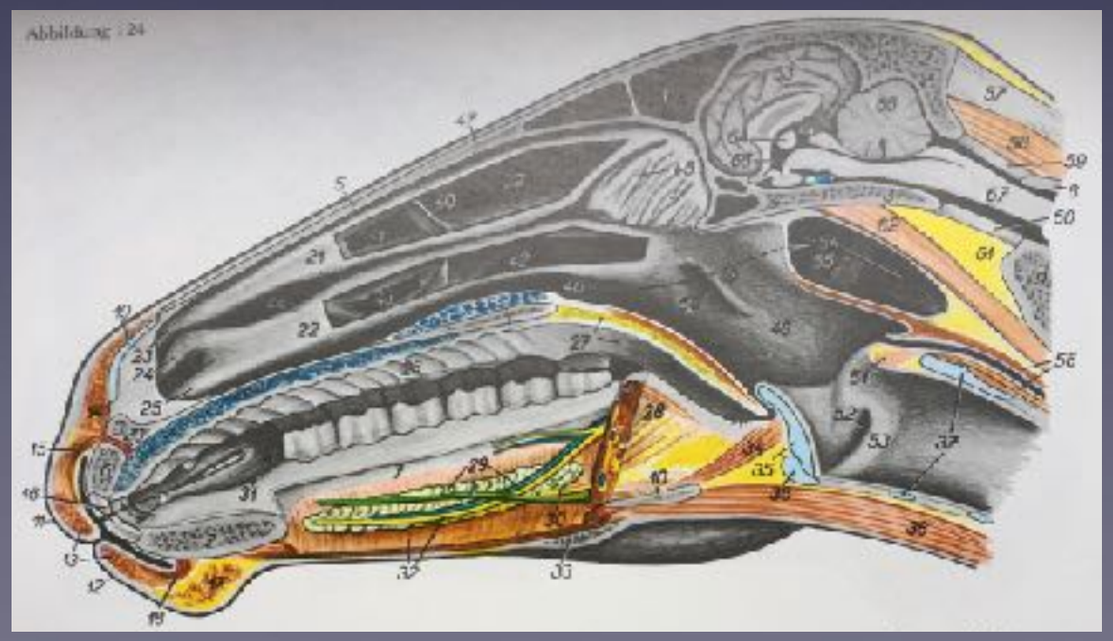
Emorragia a carico dell'inserzione dei muscoli longus captis e rectus capitis



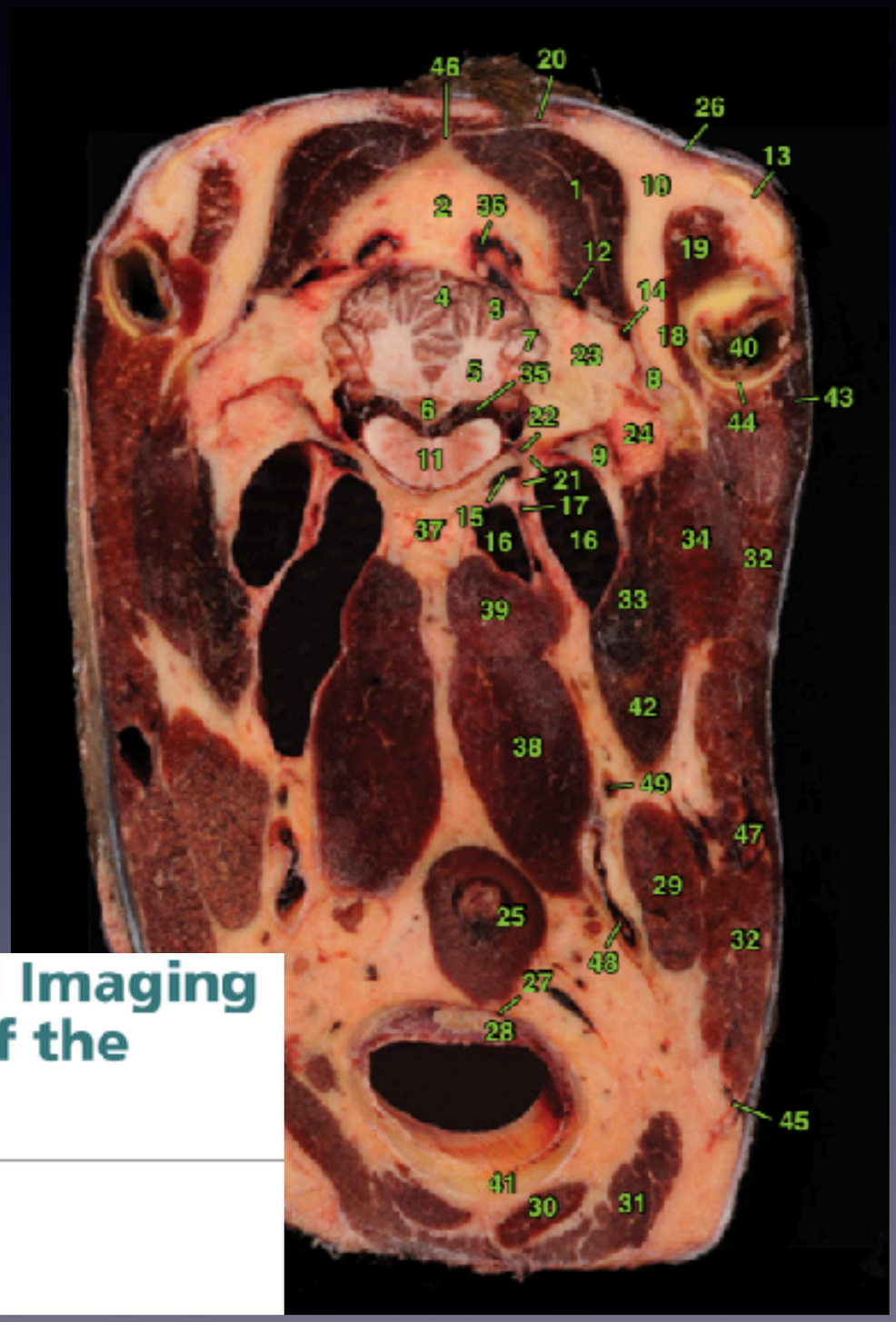


Caso clinico n° 4

- I muscoli longus captis e rectus capitis sono muscoli flessori della testa che originano dai processi trasversi della 3^, 4^ e 5^ vertebra cervicale e s'inseriscono sulla porzione basilare dell'osso occipitale e sul corpo del basisfenoide.



Caso clinico n° 4



Atlas of Clinical Imaging and Anatomy of the Equine Head

Larry Kimberlin, DVM, FAVD, CVPP

Alex zur Linden, DVM, DACVR

Lynn Ruoff, DVM

Caso clinico n°5

- Mc 24 anni, Sella, a riposo
- Epistassi bilaterale, circa 10 gg
- Eog nella norma, non ha mai avuto alterazione della °t

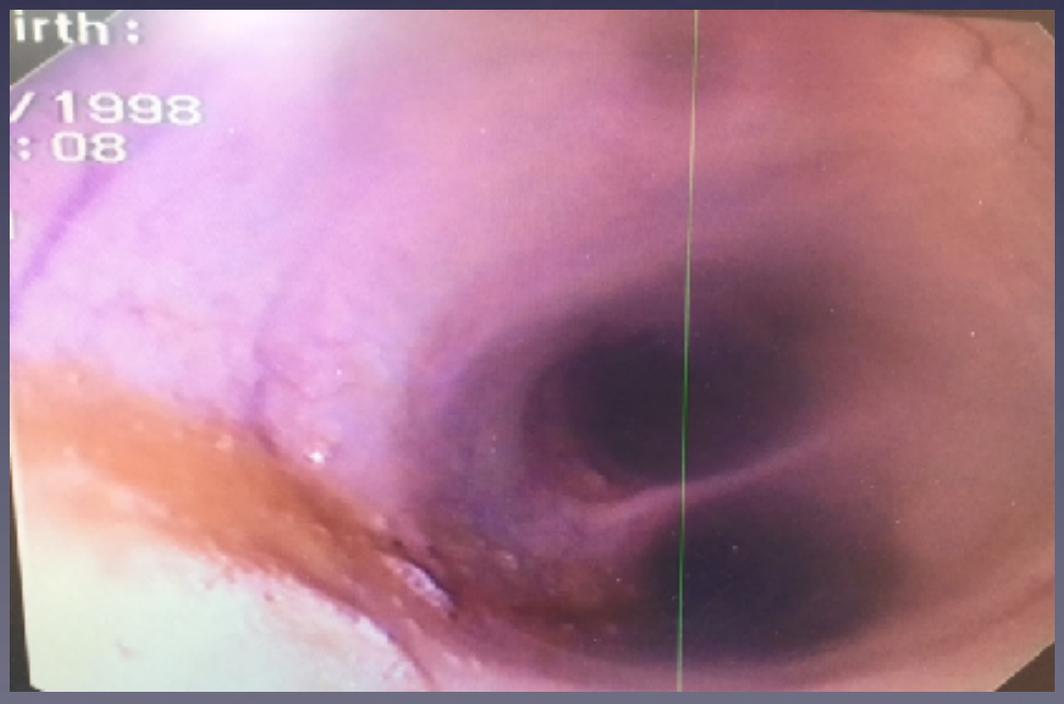
Caso clinico n°5

- Emocromo: eosinofilia relativa ed assoluta al di sopra dei limiti di normalità (Wbc 5200, eos 12,1%)
- Ematochimica nei limiti di normalità
- Feci coprologico: sterile

Caso clinico n°5

Eop respiratorio:

- ampiezza inspiratoria ridotta, murmure fortemente rinforzato, quadranti cranioventrali
- Esame endoscopico: muco biancastro e denso misto a sangue proveniente dai bronchi lobari di sx



Caso clinico n°5

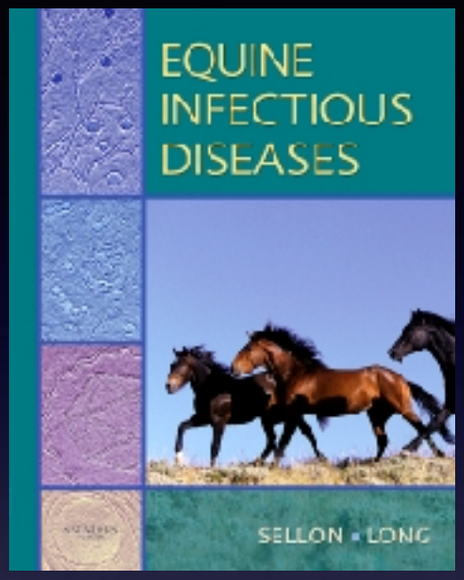
Eop respiratorio:

- Esame ultrasonografico torace: area cranio ventrale di sx, irregolarità della superficie pleurica, aree ipoecogene con eco di rinforzo, artefatti a coda di cometa

Caso clinico n°5

BAL

- ✓ Eosinofili 26%
- ✓ Emosiderociti 8%
- ✓ Cellule giganti (+++)



- ✓ Discreta quantità di materiale vegetale di natura fungina (spore cilindriche) libero e fagocitato all'interno dei macrofagi

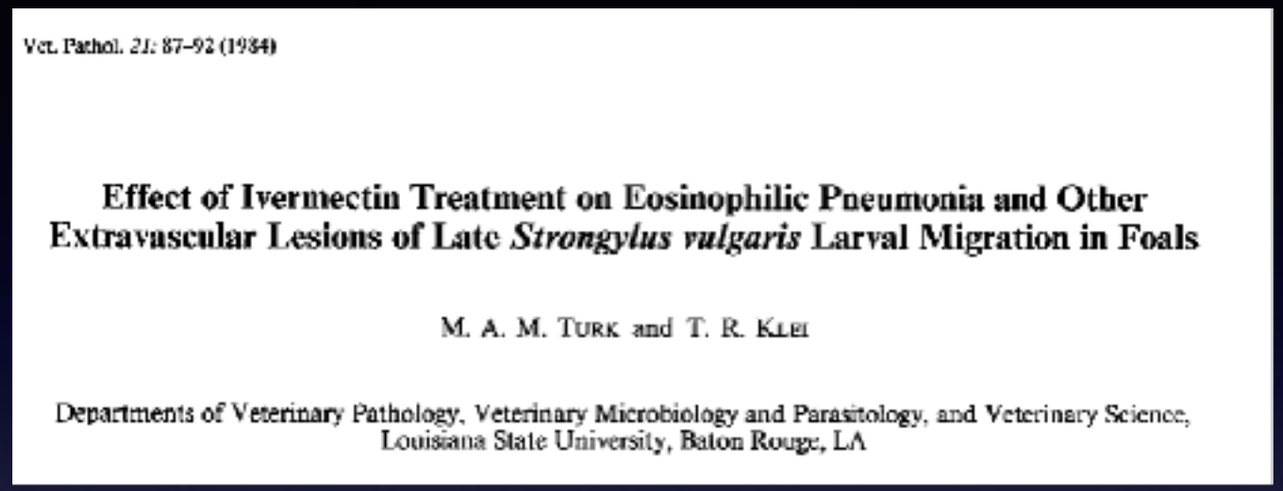
Esame microbiologico at: sterile

Esame micologico: sterile (attenzione alla conservazione 4 °C)

Caso clinico n°5

Diagnosi differenziale:

- Polmonite eosinofilica
- Meed
- Asma equina
- Polmonite parassitaria



Vet. Pathol. 21: 87–92 (1984)

Effect of Ivermectin Treatment on Eosinophilic Pneumonia and Other Extravascular Lesions of Late *Strongylus vulgaris* Larval Migration in Foals

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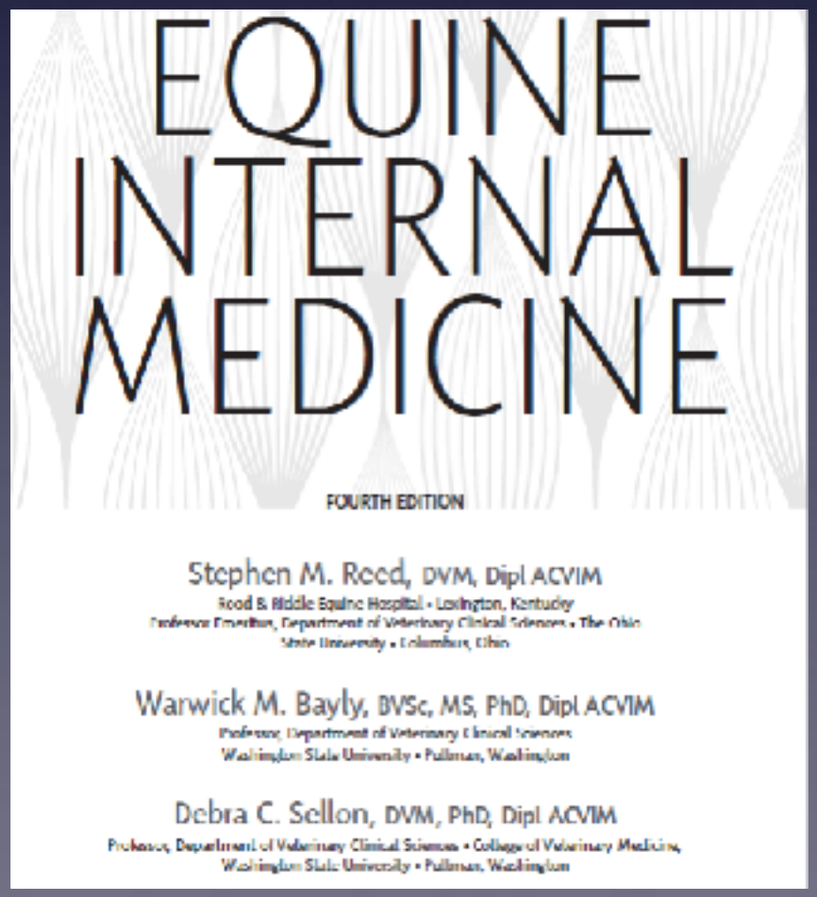

EQUINE INTERNAL MEDICINE

FOURTH EDITION

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Caso clinico n°5

Polmonite micotica

- Immunodepressione (aspergillus, candida, Pneumocystis Jirovecii)
- 86% complicazione patologia gastrointestinale
- Possibili patogeni primari: Blastomyces dermatitidis, coccidiodes immitis, Histoplasma capsulatum, Cryptococcus neoformans
- Febbre, Tachipnea, Rinforzo del murmure
- Epistassi
- Spore non significative nella citologia
- Conservazione campione per esame micologico ???

Caso clinico n°5

Polmonite parassitaria

- Parascaris equorum (larve) e Dictyocaulus arnfieldi (adulti)
- Eosinofili nel Bal e nel circolo ematico
- Scarsa condizione fisica
- Effusioni pleuriche, granulomi o ascessi
- Rinforzo del murmure

Caso clinico n°5

Problema: ridotta disponibilità economica

Terapia

- Prednisolone 2 mg/kg per 5 volte q 48h seguito da 1 mg/kg q 48h per 5 volte ed infine 0,5 mg/kg q 48h per 5 volte
- Ivermectina due somministrazioni a distanza di 30 gg
- al termine della terapia e per i sei mesi successivi non ha più avuto episodi di epistassi
- Controllo endoscopico negativo a 30 gg ed a 60 gg
- Eco torace: persistono piccole code di cometa cranioventrali

Caso clinico n° 6

- F 16 anni, Sella, scuola
- Epistassi monolaterale, scolo purulento e maleodorante
- Eog nella norma, non ha mai avuto alterazione della °t

Caso clinico n° 6

esame endoscopico

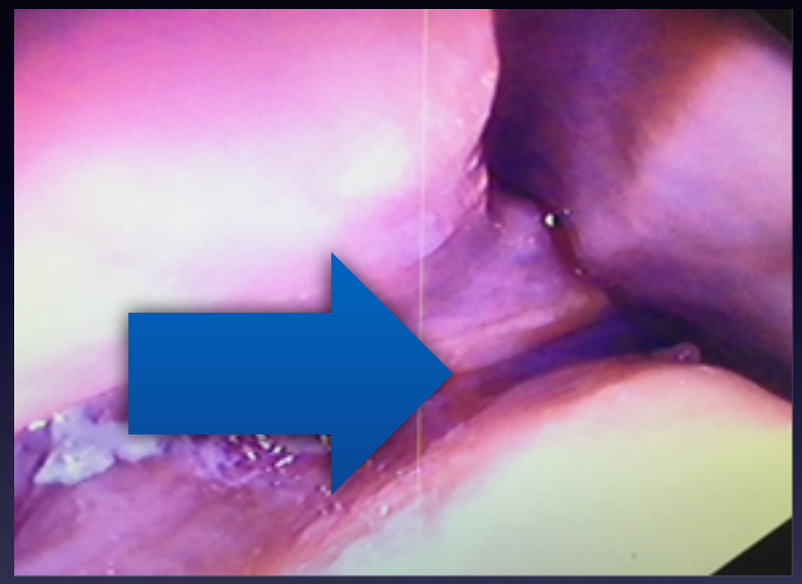
- Gemizio sanguinolento dalla conca

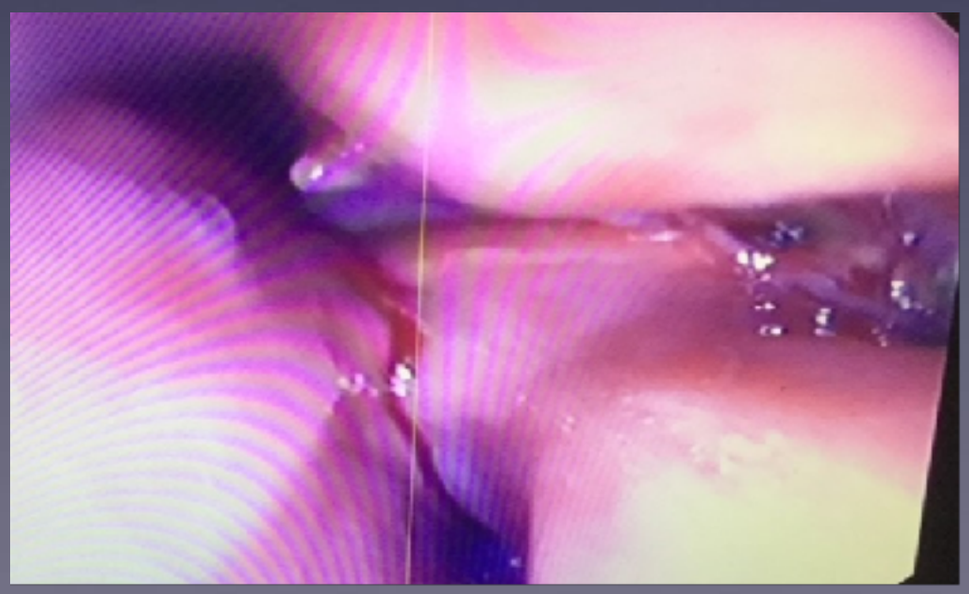
dorsale

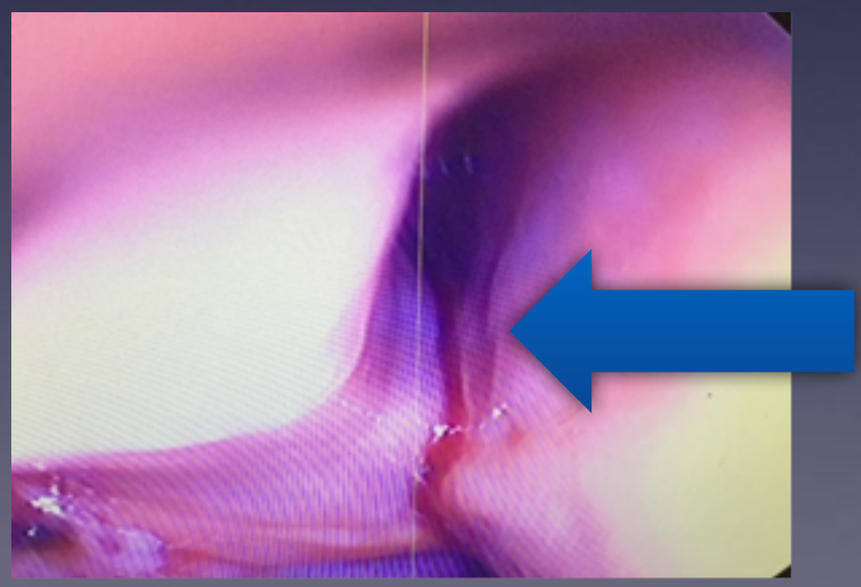
- Non è possibile osservare

l'etmoide per la deformazione della conca

dorsale







Caso clinico n° 6

rx

Area radiopaca seno

frontale di dx

Caso clinico n° 6

TAC

- neoformazione a carico della cavità nasale/seni paranasali di destra, compatibile in prima ipotesi con una cisti dei seni nasali/ paranasali.

- sinusite secondaria a carico del seno mascellare rostrale e caudale, del seno sfenopalatino, del seno frontale e della conca ventrale ipsilaterali ed osteolisi verosimilmente secondaria all'azione compressiva/infiammatoria esercitata dalla neoformazione sulle strutture ossee adiacenti.

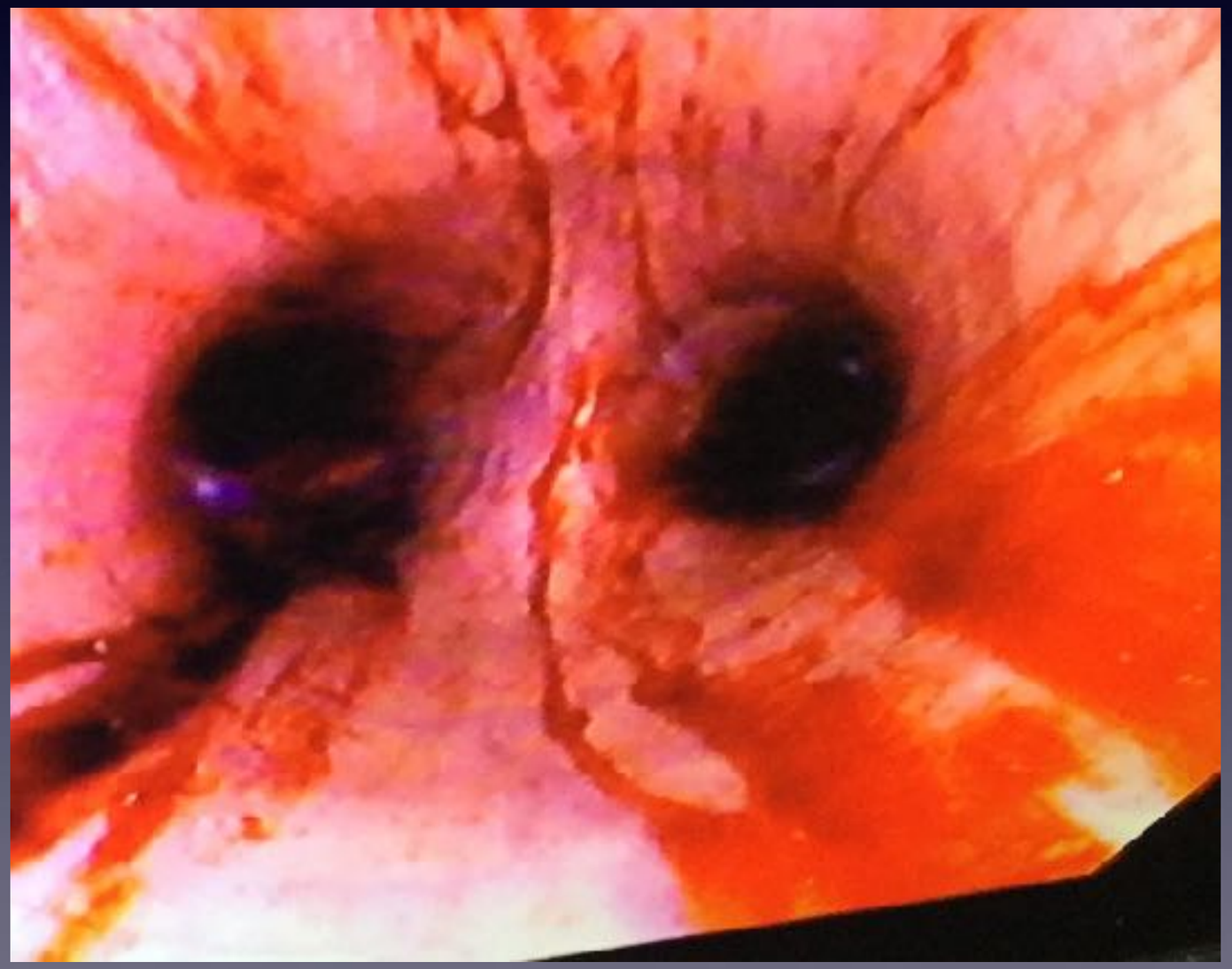
- Possibile eziologia neoplastica

- Chirurgia e biopsia

Casi clinici EIPH

Premesse

- Letteratura internazionale
- Consensus statement
- Opinioni personali



Casi clinici EIPH

Premesse

Letteratura internazionale e consensus statement

- Ricerche sul web:

Parola chiave: bleeders in horse 45.000 risposte

Parola chiave: eiph, 113.000 risposte

Parola chiave: lasix and horse 890.000 risposte

Parola chiave: furosemide and horse 905.000 risposte

Casi clinici EIPH

Premesse

Letteratura internazionale e consensus statement

- Ricerche sul web scientifico

Parola chiave: “exercise-induced pulmonary hemorrhage” or “exercise-induced pulmonary hemorrhage” AND “horse” 368 risultati

Parola chiave: “EIPH” AND “horse” 224 risultati

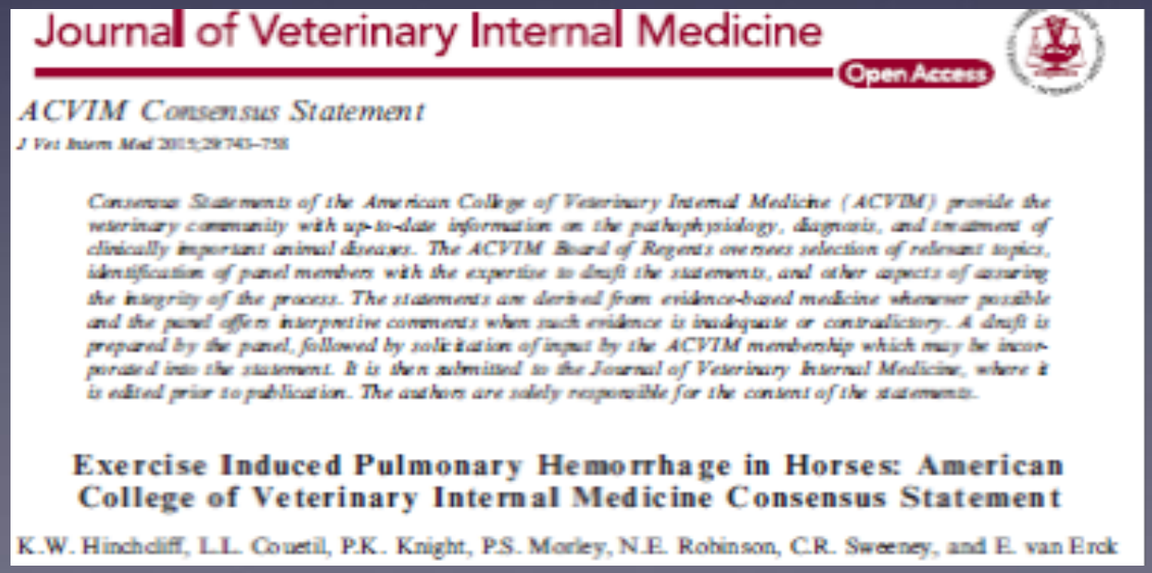
Parola chiave: furosemide and horse 367 risultati

Casi clinici EIPH

Premesse

Consensus statement

- Presenza di sangue nelle basse vie aeree (trachea e bronchi)
- Presenza di globuli rossi nel lavaggio broncoalveolare
- Entrambe le condizioni



Journal of Veterinary Internal Medicine

Open Access

ACVIM Consensus Statement

J Vet Intern Med 2015;29:743–758

Consensus Statements of the American College of Veterinary Internal Medicine (ACVIM) provide the veterinary community with up-to-date information on the pathophysiology, diagnosis, and treatment of clinically important animal diseases. The ACVIM Board of Regents oversees selection of relevant topics, identification of panel members with the expertise to draft the statements, and other aspects of assuring the integrity of the process. The statements are derived from evidence-based medicine whenever possible and the panel offers interpretive comments when such evidence is inadequate or contradictory. A draft is prepared by the panel, followed by solicitation of input by the ACVIM membership which may be incorporated into the statement. It is then submitted to the Journal of Veterinary Internal Medicine, where it is edited prior to publication. The authors are solely responsible for the content of the statements.

Exercise Induced Pulmonary Hemorrhage in Horses: American College of Veterinary Internal Medicine Consensus Statement

K.W. Hinchcliff, L.L. Couetil, P.K. Knight, P.S. Morley, N.E. Robinson, C.R. Sweeney, and E. van Erck

Casi clinici EIPH

Premesse

Consensus statement

Gli autori si pongono 4 domande

- Eiph altera lo stato di salute e benessere del cavallo?
- Eiph riduce le performance?
- Esistono trattamenti profilattici efficaci per l'eiph?
- La somministrazione preventiva di Furosemide migliora le performance?

Casi clinici EIPH

Consensus statement: risposte

1. Non altera lo stato di salute e benessere (segni clinici)
2. Non altera gli scambi dei gas respiratori (test su treadmill)
3. Non altera la concentrazione di lattato dopo esercizio
4. Non è causa di morte dopo esercizio (casuale)

Casi clinici EIPH

Consensus statement: risposte

5. Eiph grado II/III non riduce la durata della carriera mentre grado IV moderatamente

6. Ridotta possibilità che l'infiammazione delle basse vie aeree causi Eiph e ridotta possibilità che viceversa, l'eiph determini infiammazione delle basse vie aeree

7. Determina caratteristiche lesioni polmonari (rimodellamento vascolare)

Casi clinici EIPH

Consensus statement: risposte

8. Non è trasmissibile geneticamente

9. Valutazioni varie su quanto determini un alterazione della performance

10. Bassa relazione tra score eiph ed alterazione della performance

11. Elevata relazione tra somministrazione di furosemide e riduzione della gravità ed incidenza eiph

Casi clinici EIPH

Consensus statement: risposte

12. Acido aminocaproico, nasal strep, pentossifillina, broncodliatatori, corticostroidi: ridotta influenza sulla gravita e prevenzione eiph

13. Moderata relazione tra somministrazione di furosemide in corsa e risultato

14. Viste le lesioni polmonari è da considerare una malattia

15. Poche evidenze sulla progressività

Casi clinici EIPH

Osservazioni personali su:

- Circa 800 cavalli ‘’significativi’’, ripetutamente valutati durante la loro carriera agonistica
- Circa 1000 esami endoscopici delle vie aeree post esercizio massimale o competizione
- Circa 600 Lavaggi broncoalveolari ‘’significativi’’

Casi clinici EIPH

Osservazioni personali

1. Caratteristiche fisiologiche del cavallo atleta *(VO2 max, accoppiamento andatura/respirazione ecc)*

2. Intensità e tipo di esercizio che hanno determinato l'EIPH

- *Cavalli da corsa: in gara o in allenamento (intensità dell'allenamento)?*

- *Cavalli da salto ostacoli: in gara o in allenamento?*

3. Malattia professionale nei cavalli da corsa:

nel 100% dei lavaggi broncoalveolari sono presenti emosiderociti (anche nei cavalli con ottima performance), anche nei puledri dopo i primi allenamenti alla velocità di soglia anerobica

Casi clinici EIPH

4. Spesso causa polifattoriale:

- Infiammazione/infezione respiratorie
- Zoppie o dolore ortopedico
- alterazioni biomeccaniche
- Miopatie
- Aritmie cardiache
- Condizioni della pista/campo di gara
- Temperamento
- Svolgimento della competizione (parziali)

Casi clinici EIPH

5. Profilassi: Furosemide, acido etacrinico e adeguamento della metodologia di allenamento (carichi di lavoro)

6. Senza un adeguata profilassi drastica riduzione della durata della carriera agonistica

7. Scarsa relazione tra infezioni virali ed eiph

8. Elevata relazione tra condizioni della pista ed eiph

9. Elevata relazione tra alterazioni ortopediche, metodologie di allenamento ad alta intensità, temperamento ed eiph

Casi clinici EIPH

10. Dati consensus statement relativi alle ricerche sul web: non è possibile trovare una soluzione univoca (protocollo), probabilmente non esiste una soluzione perché spesso non si trova la causa

Casi clinici EIPH

Cavallo da concorso:

1° episodio marzo 2017

Eop cardiocircolatorio: nella norma

Eop respiratorio

- Caratteri del respiro nella norma
- Non sono rilevabili rumori respiratori patologici
- Numerosi artefatti a coda di cometa
- Iad neutrofilica mastocitaria

Casi clinici EIPH

Cavallo da concorso:

Terapia

- Corticosteroidi aerosol (fluticasone 4mg/kg q 12 h) per 15 gg
- Broncodilatatore aerosol (ipratroprio bromuro 3 mcg/kg ogni 12 h) per 15 gg
- Ceftiofur 1 g ogni 12 h per 10 gg
- 6 mesi prima di rientrare in gara
- 1° concorso: eiph

Casi clinici EIPH

Cavallo da concorso:

Cosa non ha funzionato?

1. La causa dell'eiph non era e non è l'infiammazione delle basse vie aeree
2. Probabilmente un fattore predisponente
3. Cavallo molto emotivo e con evidenti problemi biomeccanici
4. Test a casa previa somministrazione di acepromazina (prequillan 0,3 ml ev 30' prima). 20 salti submassimali

Casi clinici EIPH

Cavallo da concorso:

Esame endoscopico: negativo

Soluzione

- La soluzione del problema è di carattere tecnico e non veterinario

- Noi possiamo intervenire per il contenimento dell’infiammazione delle basse vie aeree

Casi clinici EIPH

Cavalli da corsa galoppo e trotto

- Nel 100% degli esami citologici dei Bal dei cavalli da corsa sono presenti emosiderociti e la concentrazione non è sempre correlabile alla performance

- Spesso nei casi con riduzione della performance, condizione irrisolvibile con progressiva riduzione delle prestazioni

- Il nostro scopo è principalmente quello di allungare la carriera del cavallo

Casi clinici EIPH

Cavalli da corsa galoppo e trotto

- Nel 100% degli esami citologici dei Bal di cavalli da corsa sono presenti emosiderociti e la concentrazione non è sempre correlabile alla performance

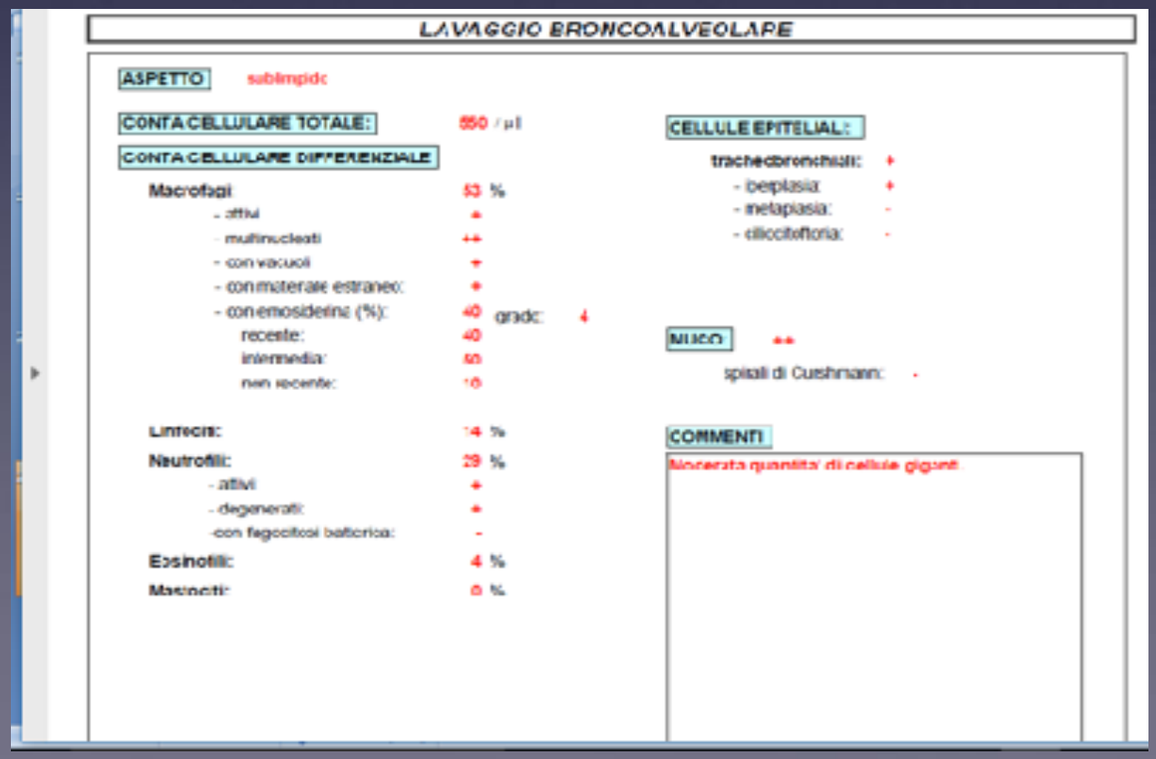
- Spesso nei casi con riduzione della performance, condizione irrisolvibile con progressiva riduzione delle prestazioni

- Il nostro scopo è principalmente quello di allungare la carriera del cavallo

Casi clinici EIPH

Cavalli sottoposti ad esercizi di alta intensità

- Fondamentale la sinergia veterinario/allenatore
- Attenzione ai numeri
- Clinica e diverse osservazioni



LAVAGGIO BRONCOALVEOLARE

ASPETTO sublimpido

CONTA CELLULARE TOTALE: 650 / µl

CONTA CELLULARE DIFFERENZIALE

Macrofagi 53 %
- attivi +
- multinucleati ++
- con vacuoli +
- con materiale estraneo: +
- con emosiderina (%): 40 grado: 4
recenti: 40
intermedia: 60
non recente: 0

Linfociti: 14 %
Neutrofili: 29 %
- attivi +
- degenerati: +
-con fagocitosi batterica: -
Eosinofili: 4 %
Mastociti: 0 %

CELLULE EPITELIALI:
tracheobronchiali: +
- iperplasia: +
- metaplasia: -
- discheratosi: -

MUCO ++
spirali di Curschmann: -

COMMENTI
Moderata quantità di cellule giganti

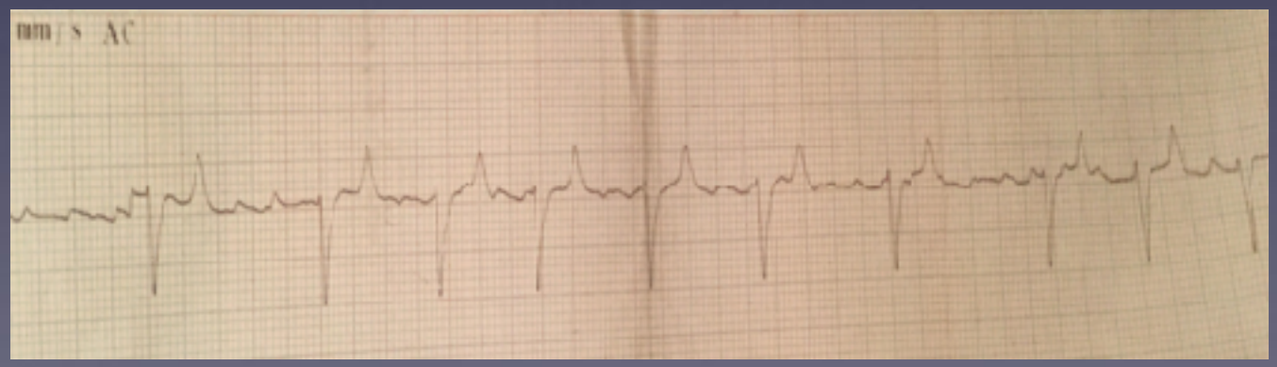
Casi clinici EIPH

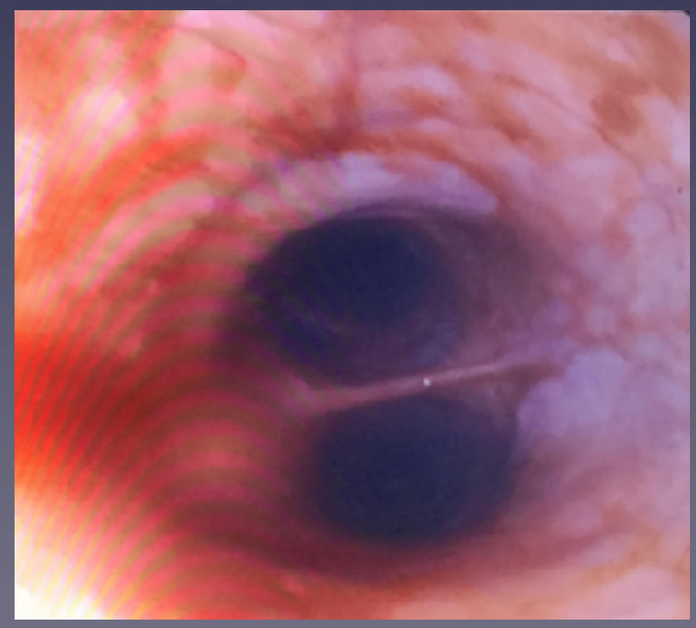
Cavalli sottoposti ad esercizi di alta intensità

Femmina 5 aa, trottatrice

Ottime performance in carriera

Eiph in allenamento di intensità moderata

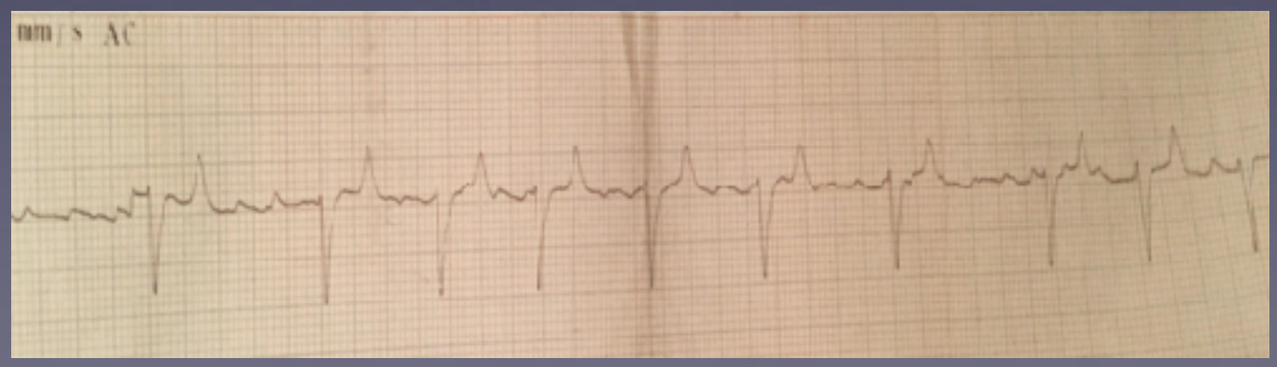




Casi clinici EIPH

Cavalli sottoposti ad esercizi di alta intensità

- Ecocardio
- Cardioversione con Chinidina solfato
- Recupero completo delle performance precedenti



Casi clinici EIPH

Cavalli sottoposti ad esercizi di alta intensità

J Appl Physiol (1985). 2013 Apr;114(7):869-78. doi: 10.1152/japplphysiol.01170.2012. Epub 2013 Jan 31.

Distribution of venous remodeling in exercise-induced pulmonary hemorrhage of horses follows reported blood flow distribution in the equine lung.

Williams KJ[1], Robinson NE, Defeijter-Rupp H, Millerick-May M, Stack A, Hauptman J, Derksen FJ.
Author information

Equine Veterinary Journal

Equine Veterinary Journal ISSN 0425-1644
DOI: 10.1111/evj.12458

Analytical Clinical Studies

Bar shoes and ambient temperature are risk factors for exercise-induced pulmonary haemorrhage in Thoroughbred racehorses

E. J. CRISPE, G. D. LESTER*, I. D. ROBERTSON and C. J. SECOMBE

College of Veterinary Medicine, School of Veterinary and Life Sciences, Murdoch University, Murdoch, Western Australia, Australia

*Correspondence email: g.lester@murdoch.edu.au; Received: 31.07.14; Accepted: 04.05.15

Equine Veterinary Journal

Equine Veterinary Journal ISSN 0425-1644
DOI: 10.1111/evj.12760

Effectiveness of furosemide in attenuating exercise-induced pulmonary haemorrhage in horses when administered at 4- and 24-h prior to high-speed training

H. K. KNYCH*†, W. D. WILSON‡, A. VALE¶, P. H. KASS#, R. M. ARTHUR†† and J. H. JONES‡‡

Equine Veterinary Journal

Equine Veterinary Journal ISSN 0425-1644
DOI: 10.1111/evj.12671

The association between exercise-induced pulmonary haemorrhage and race-day performance in Thoroughbred racehorses

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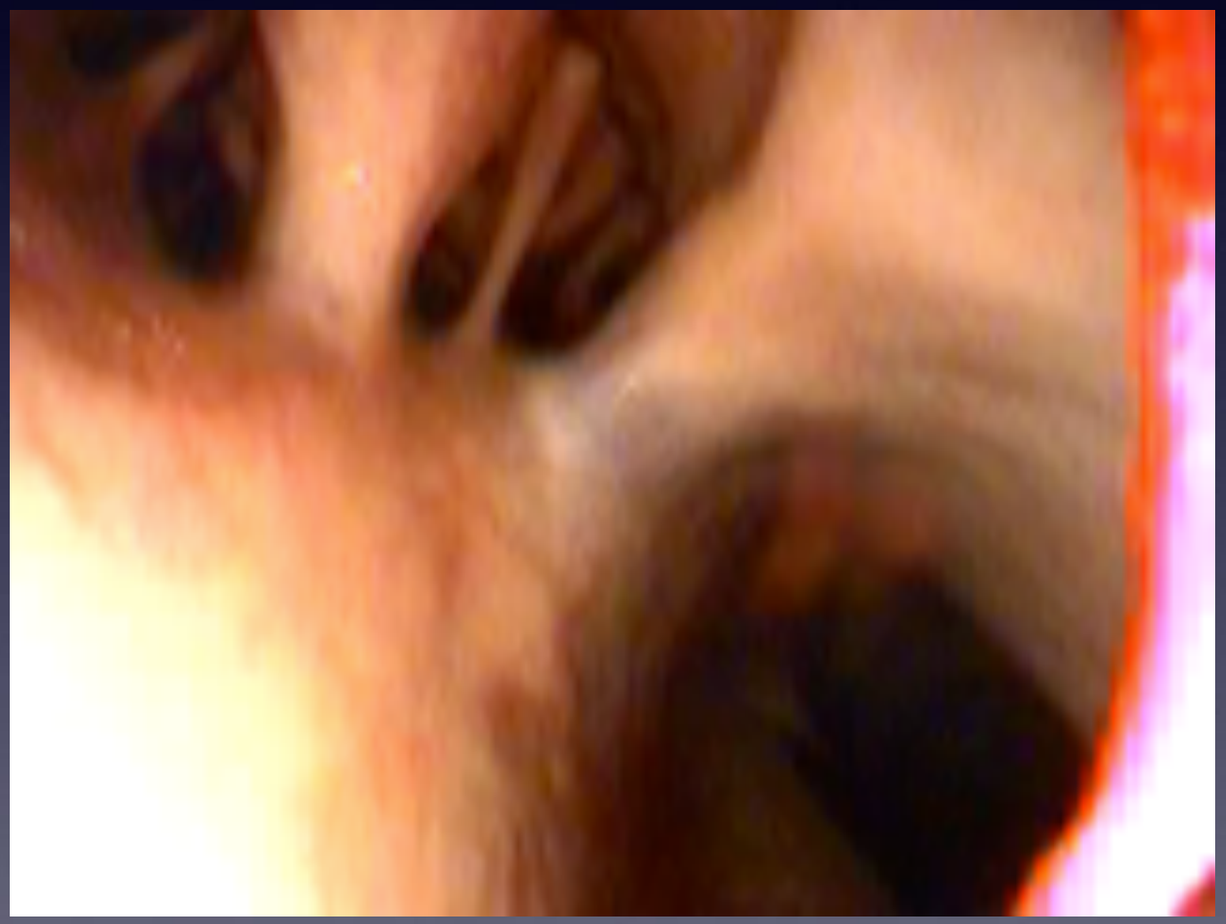
*Correspondence email: g.lester@murdoch.edu.au; Received: 26.08.16; Accepted: 04.01.17

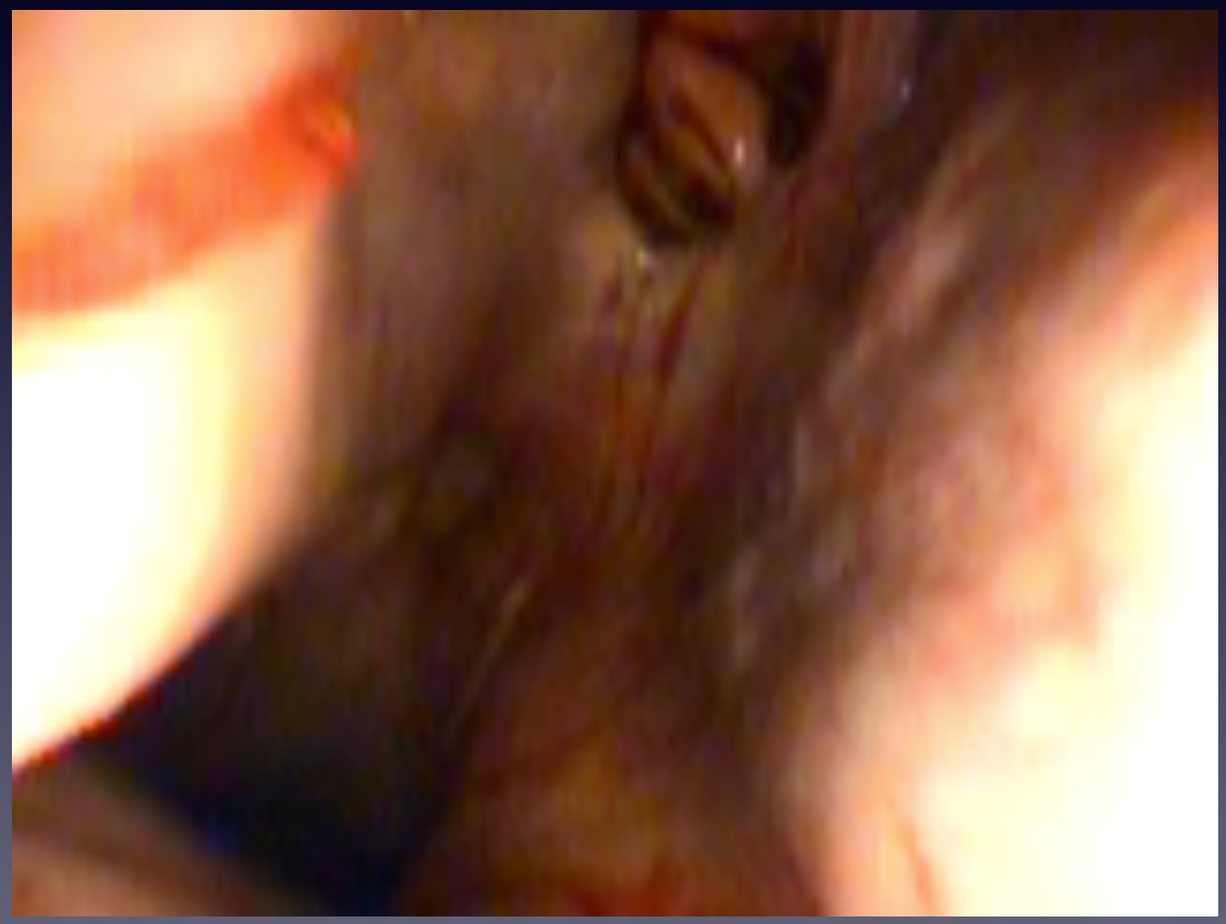
Caso clinico n° 7

- Castrone 22 anni, sella, al paddock
- Epistassi monolaterale di moderata entità da questa mattina
- FC:50, FR:16, Mucose: rosa, RT: 2”, Sensorio: vigile, Appetito:+
- HT:40, PT:7.0, Lattato: 3,0
- Ascoltazione polmonare normale

Caso clinico n° 7

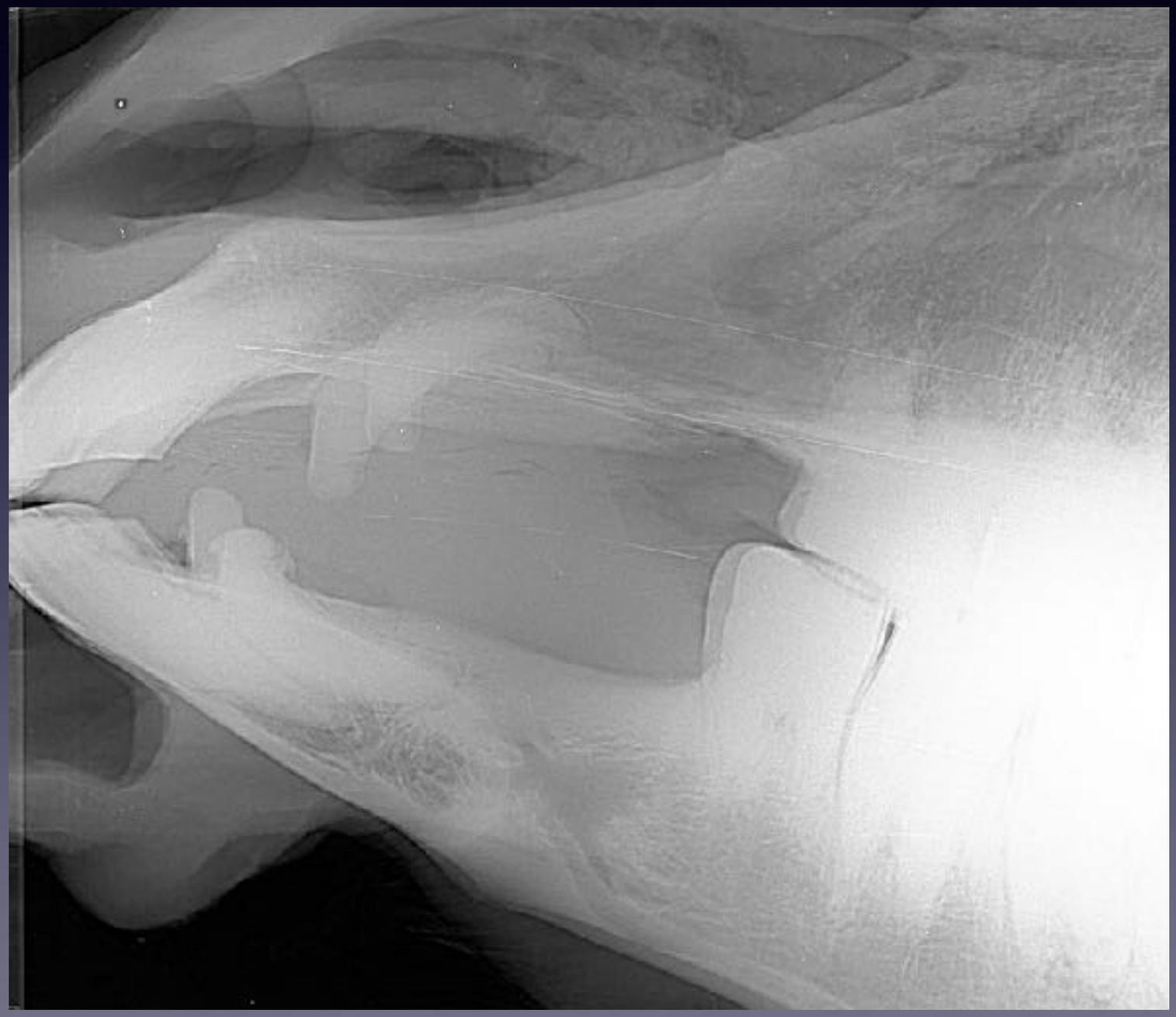


Dx



Sn

Caso clinico n° 7



Caso clinico n° 7

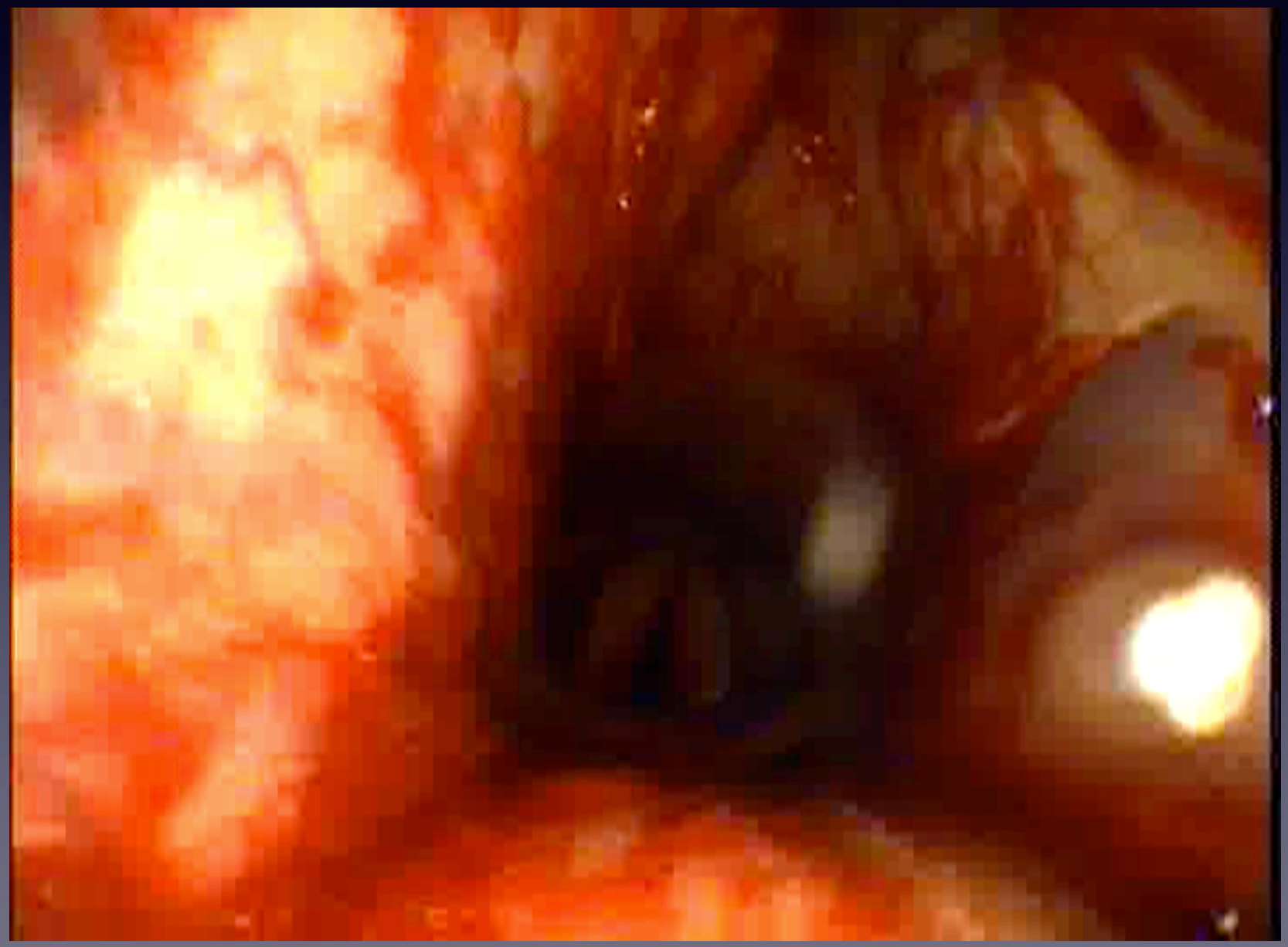
TERAPIA

- Flunixin meglumine: 1,1 mg/kg ev sid, per 3 gg.
- Acido Tranexanico: 5 mg/kg im, ev, dose singola
- Desametazone 0,08 mg/kg ev, per 2 gg.

Caso clinico n° 8

- Castrone 14 anni, sella, in lavoro regolare da maneggio
- Epistassi bilaterale abbondante, improvvisa, durante un lavoro leggero, fermatasi spontaneamente.
- FC:60 (44), FR:24, RT:2”, Mucose: Rosa, Sensorio: Vigile, Temp: 37,6°,
- HT:29,7, HGB:10,9, PT:4,8, Lattato: 5,5, WBC:9,9 Neutrofili. 85%, Fibrinogeno: 189, Piastrine: 189, SAA: 0,42
- Ascoltazione polmonare: rinforzo del murmure vescicolare localizzato in alcune aree polmonari
- Ascoltazione addominale nella norma

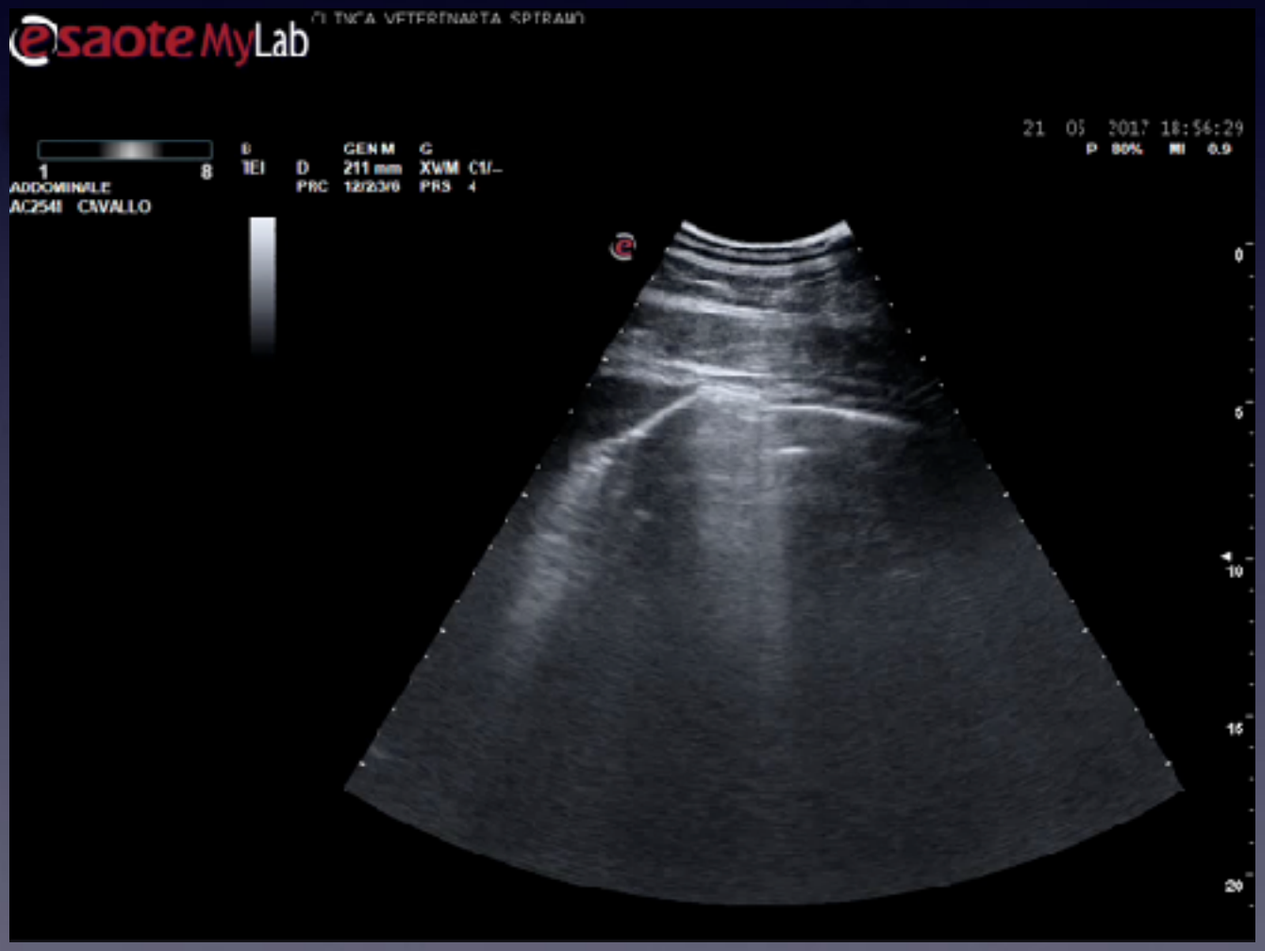
Caso clinico n° 8



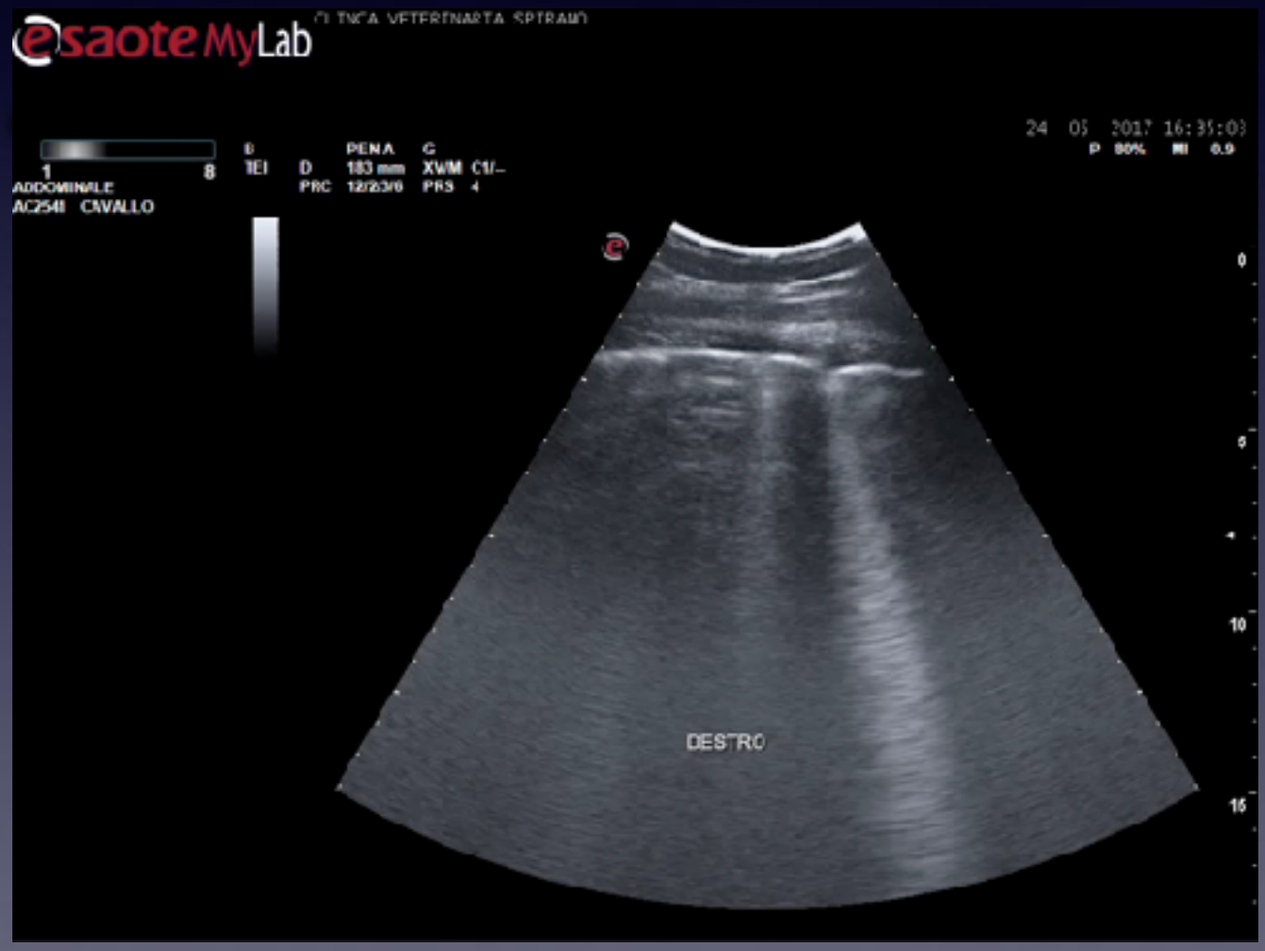
Caso clinico n° 8 bis

- Castrone 17 anni, sella, in lavoro leggero
- Epistassi bilaterale di media entità 24 ore prima, leggeri sintomi colici
- FC:78, FR:40, RT:2”, Mucose: Rosa, Sensorio: Vigile, Temp: 38,6°,
- HT:23, PT:5,8, Lattato: 5,5, WBC:18,0 Neutrofili. 93%, Fibrinogeno: 307
- Ascoltazione polmonare: assenza di rumori respiratori nei lobi anteriori, ventralmente, aumento dell’area di ascoltazione cardiaca
- Ascoltazione addominale nella norma

Caso clinico n° 8 bis



Polmone sn



Polmone dx

Caso clinico n° 8 bis

LIQUIDO PLEURICO

- HT: 26
- PROT. TOT.: 5,2

SANGUE

- HT: 23
- PROT. TOT.: 5,

Caso clinico n° 8 bis

TERAPIA

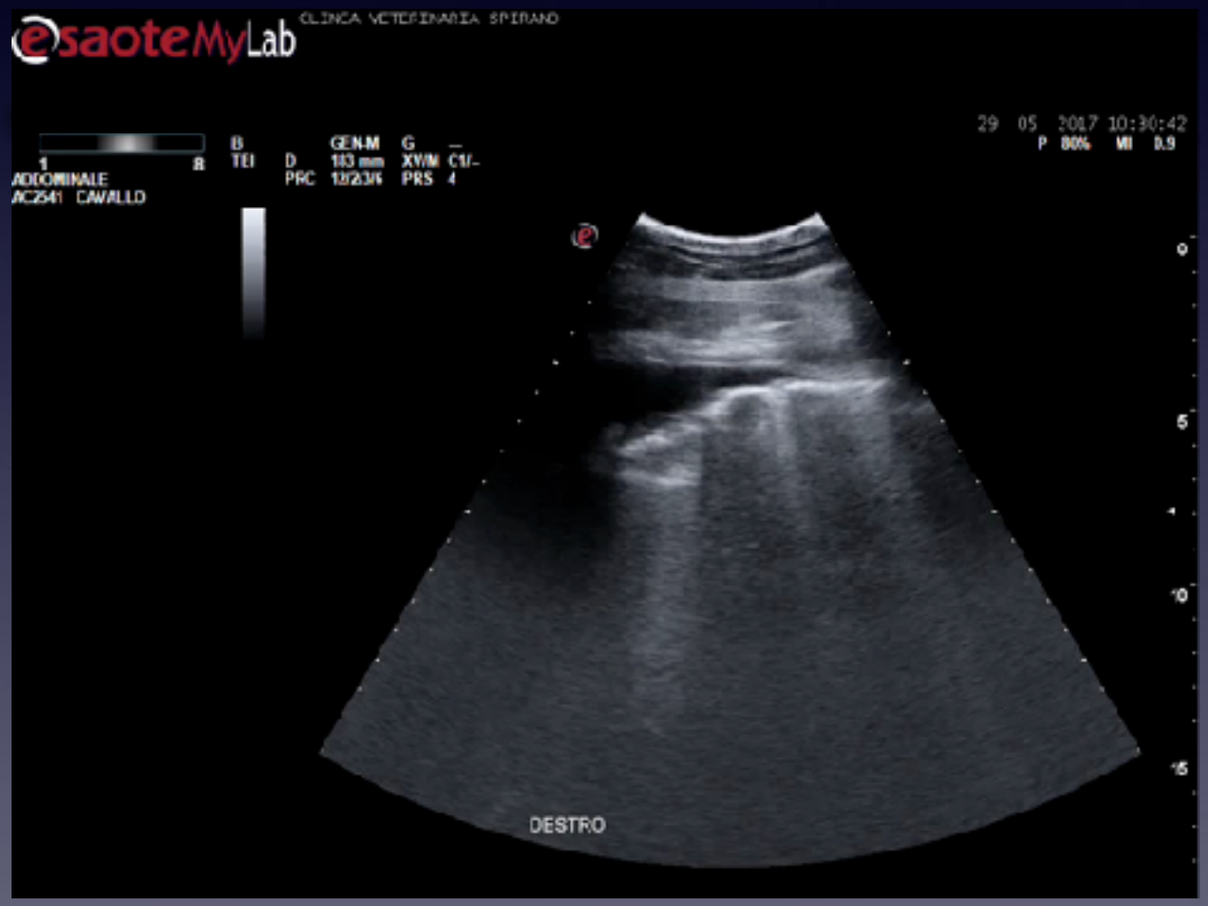
- ACIDO TRANEXANICO: 5-25 mg/kg im, ev, sq, bid
- ENROFLOXACIN: 5,5 mg/kg ev sid
- ACIDO ASCORBICO: 30 mg/kg ev bid

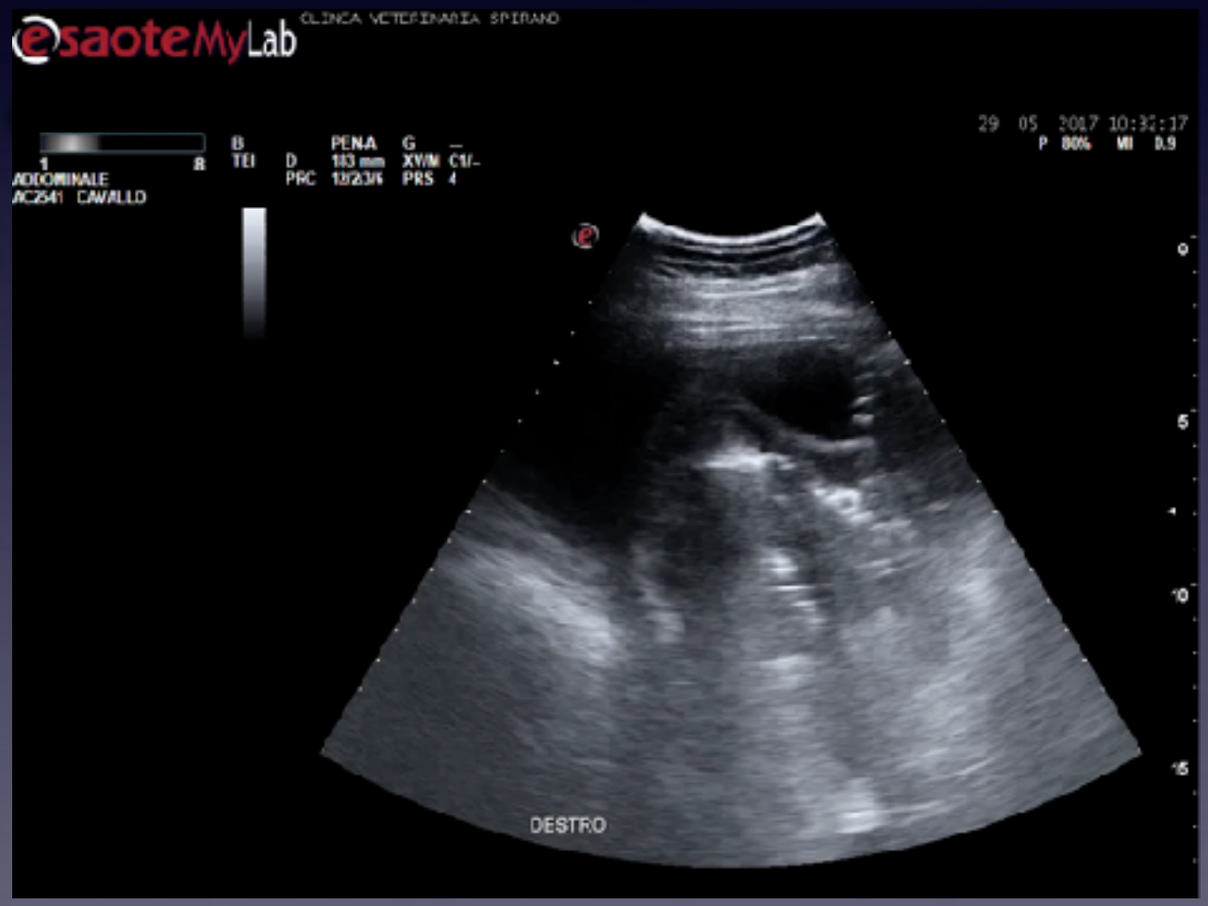
Caso clinico n° 8 bis

IL GIORNO SUCCESSIVO

- FC: 46
- FR: 18
- MUCOSE: Rosa
- RT: 2”
- SENSORIO: Vigile
- APPETITO: +
- FECI: +
- URINE: +
- TEMP. RETT.: 37,1
- HT.: 20
- PROT. TOT: 5,2

Caso clinico n° 8 bis





Dopo 8 gg.

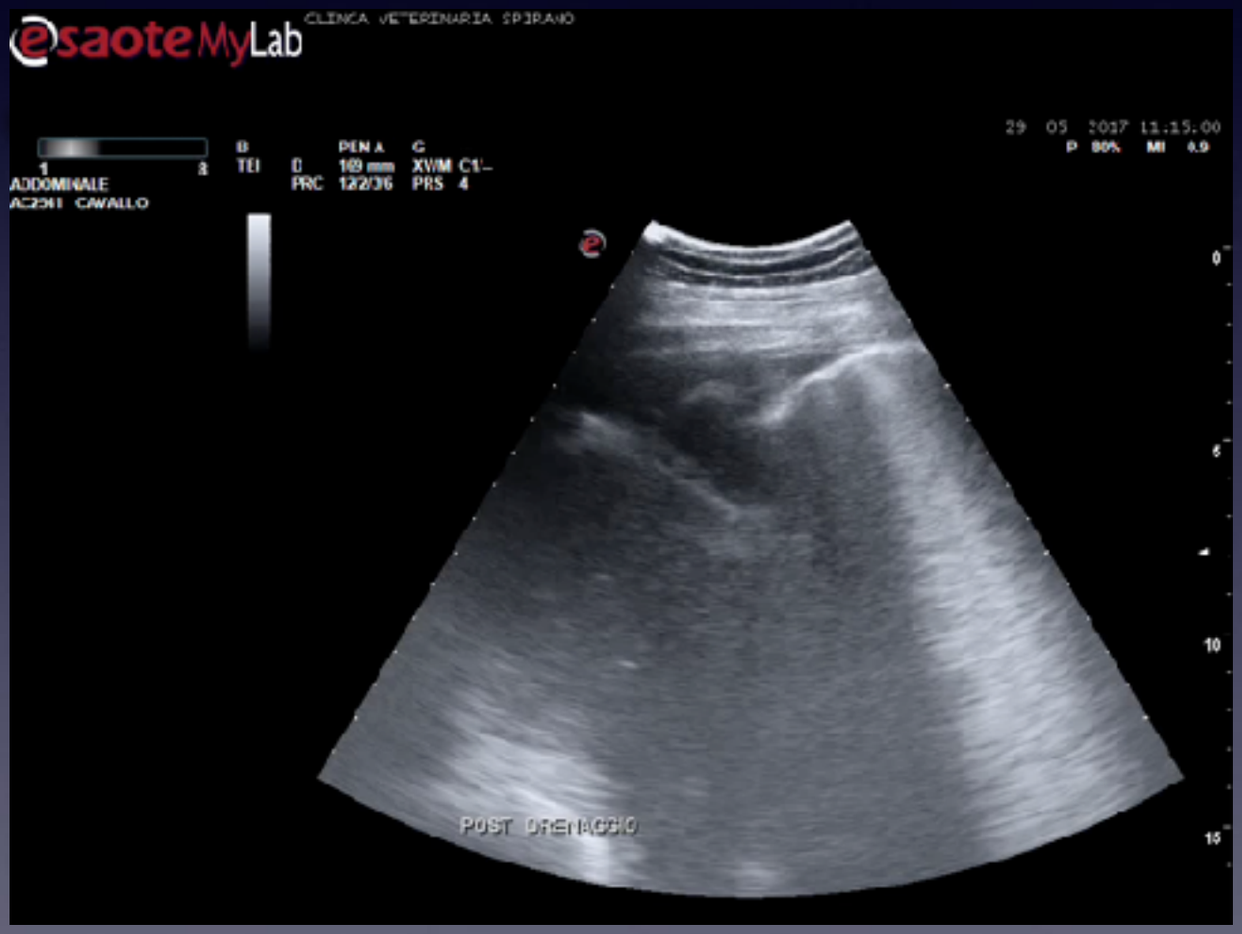
Caso clinico n° 8 bis

DOPO 8 GG.

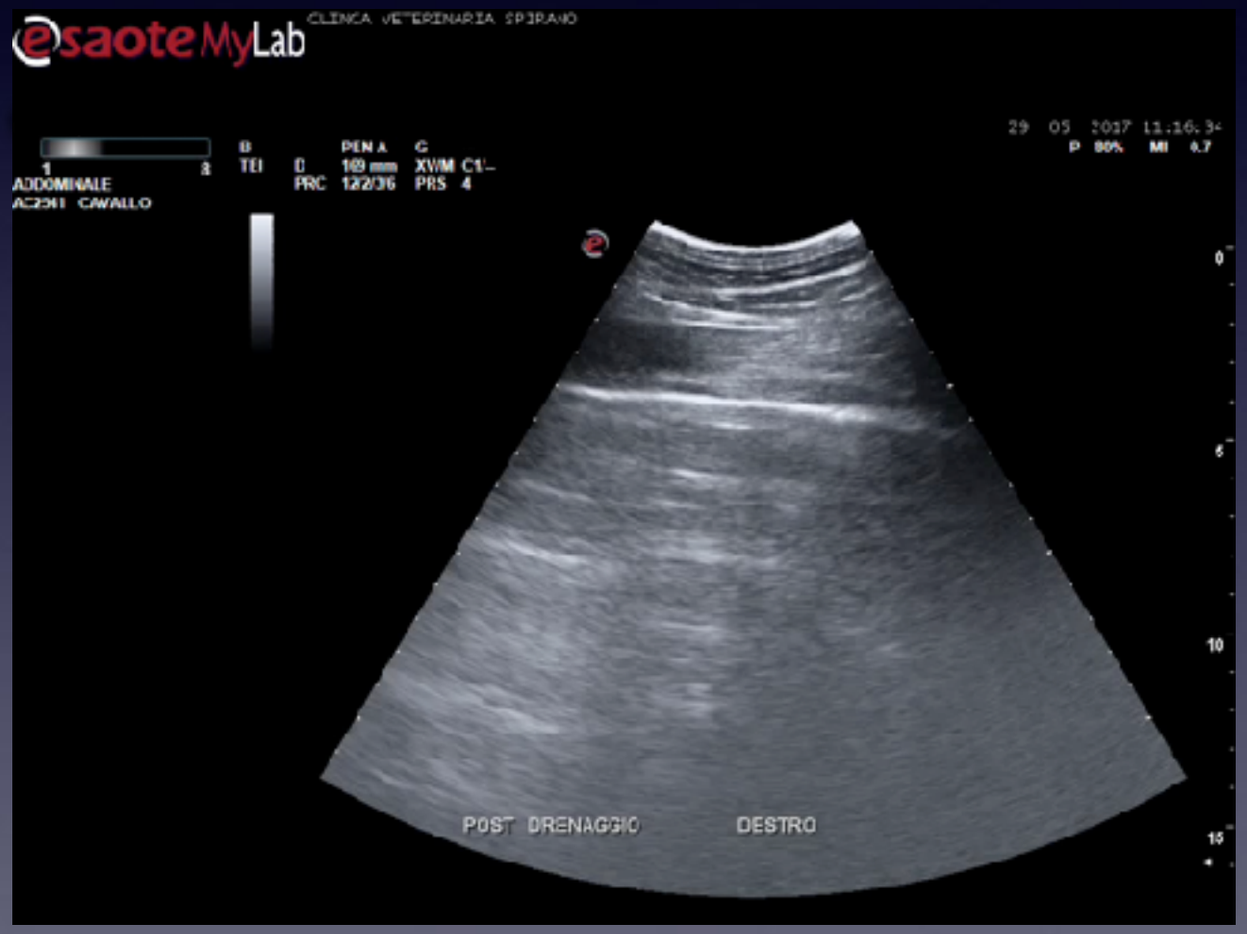
- FC: 48
- FR: 22
- MUCOSE: Rosa
- RT: 2”
- SENSORIO: Vigile
- APPETITO: +
- FECI: +
- URINE: +
- TEMP. RETT.: 37,5
- HT.: 21
- PROT. TOT: 7,0

Caso clinico n° 8 bis

Post drenaggio



Polmone sn.



Polmone dx.

Caso clinico n° 8 bis

Hemothorax in 2 Horses

Gillian Perkins, Dorothy M. Ainsworth, and Amy Yeager

J Vet Intern Med 1999;13:375–378

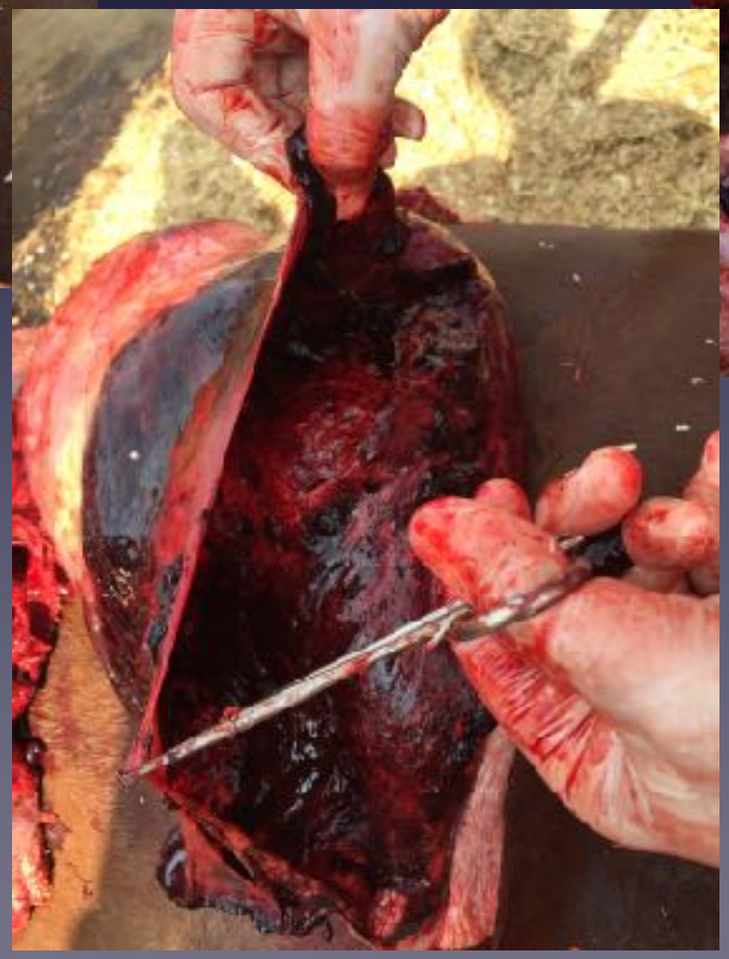
Drenato 6 lt di sangue dall'emitorace sn.

Caso clinico n° 8 bis

1 giorno dopo drenaggio

- FC: 40
- FR: 22
- MUCOSE: Rosa
- RT: 2”
- SENSORIO: Vigile
- APPETITO: +
- FECI: +
- URINE: +
- TEMP. RETT.: 37,0
- HT.: 24
- PROT. TOT: 7,4

Caso clinico n° 8



Caso clinico n° 8

Recombinant Tissue Plasminogen Activator (rTPA)?
2-10 mg in 2 lt NaCl

Australian VETERINARY JOURNAL
THE JOURNAL OF THE AUSTRALIAN VETERINARY ASSOCIATION LTD
EQUINE

CASE REPORT AND CLINICAL REVIEW

Combination fibrinolytic therapy in the treatment of chronic septic pleuropneumonia in a Thoroughbred gelding

DI Rendle,* SK Armstrong and KJ Hughes

This report documents the treatment of a case of chronic pleuropneumonia in a 3-year-old Thoroughbred gelding. A recombinant tissue plasminogen activator (tenecteplase) and a recombinant deoxyribonucleic acidase (alphadornase) were infused into the pleural cavity as adjunctive therapy in the early stages of treatment. Instillation of fibrinolytic drugs was associated with a subjective reduction in the amount of fibrin deposition and decreased fluid accumulation within the pleural cavities. Fibrinolytic therapy may be a useful adjunctive therapy in selected cases of intrapleural disease in horses.

Keywords deoxyribonuclease; fibrinolysis; parapneumonic effusion; pleuropneumonia; *Salmonella*; tissue plasminogen activator

Abbreviations APTT, activated partial thromboplastin time; PA, plasminogen activator; PT, prothrombin time; rhDNAase, recombinant deoxyribose nucleic acidase; TNK, tenecteplase; TNCC, total nucleated cell count; TPA, tissue plasminogen activator

manufactured mutants of the natural forms that have longer half-lives, greater fibrin specificity and greater resistance to natural inhibitors, such as plasminogen activator inhibitor-1.[7] Recombinant TPAs are also less immunogenic and are associated with a lower rate of treatment complications than streptokinase or urokinase.[8] The most widely used recombinant PAs are alteplase and tenecteplase (TNK). Alteplase (also known as rTPA) has been superseded in the treatment of thrombosis by TNK, which has a longer half-life and greater fibrin specificity.[7] Recent clinical reports in people and experimental animal models indicate that concurrent use of recombinant PAs and recombinant drugs that lyse deoxyribose nucleic acid (known as rhDNAases) may be more effective than using either class of drug alone.[8,9,13] The most widely used rhDNase is alphadornase, which is less immunogenic than the original wild-type streptococcal preparation, streptodornase.[8]

The use of alteplase, streptokinase and streptodornase in the treatment of equine pleuropneumonia has been reported previously[11–13]

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J Vet Intern Med 2015;29:1403–1409

The Use of Recombinant Tissue Plasminogen Activator (rTPA) in The Treatment of Fibrinous Pleuropneumonia in Horses: 25 Cases (2007–2012)

J.E. Tomlinson, E. Byrne, N. Pusterla, K. Gary Magdesian, H.G. Hilton, B. McGorum, E. Davis, A. Schoster, L. Arroyo, B. Dunkel, H. Carslake, R.C. Boston, and A.L. Johnson

Background: Information about treatment protocols, adverse effects and outcomes with intrapleural recombinant tissue plasminogen activator (rTPA) use in horses with fibrinous pleuropneumonia is limited.

Hypothesis/Objectives: Describe factors that contribute to clinical response and survival of horses treated with rTPA intrapleurally.

Animals: Horses with bacterial pneumonia and fibrinous pleural effusion diagnosed by ultrasonography, that were treated with rTPA intrapleurally.

Methods: Retrospective multicenter case series from 2007–2012. Signalment, history, clinical and laboratory evaluation, treatment, and outcome obtained from medical records. Regression analysis used to identify associations between treatments and outcomes.

Results: Thirty three hemithoraces were treated in 25 horses, with 55 separate treatments. Recombinant tissue plasminogen activator (375–20,000 μg/hemithorax) was administered 1–4 times. Sonographically visible reduction in fibrin mat thickness, loculations, fluid depth, or some combination of these was seen in 32/40 (65%) treatments. Response to at least 1 treatment was seen in 17/20 (85%) horses with sonographic follow-up evaluation after every treatment. Earlier onset of rTPA treatment associated with increased survival odds. No association was found between cumulative rTPA dose or number of rTPA doses and survival, development of complications, duration of hospitalization or total charges. Clinical evidence of hypocoagulability or bleeding was not observed. Eighteen horses (72%) survived to discharge.

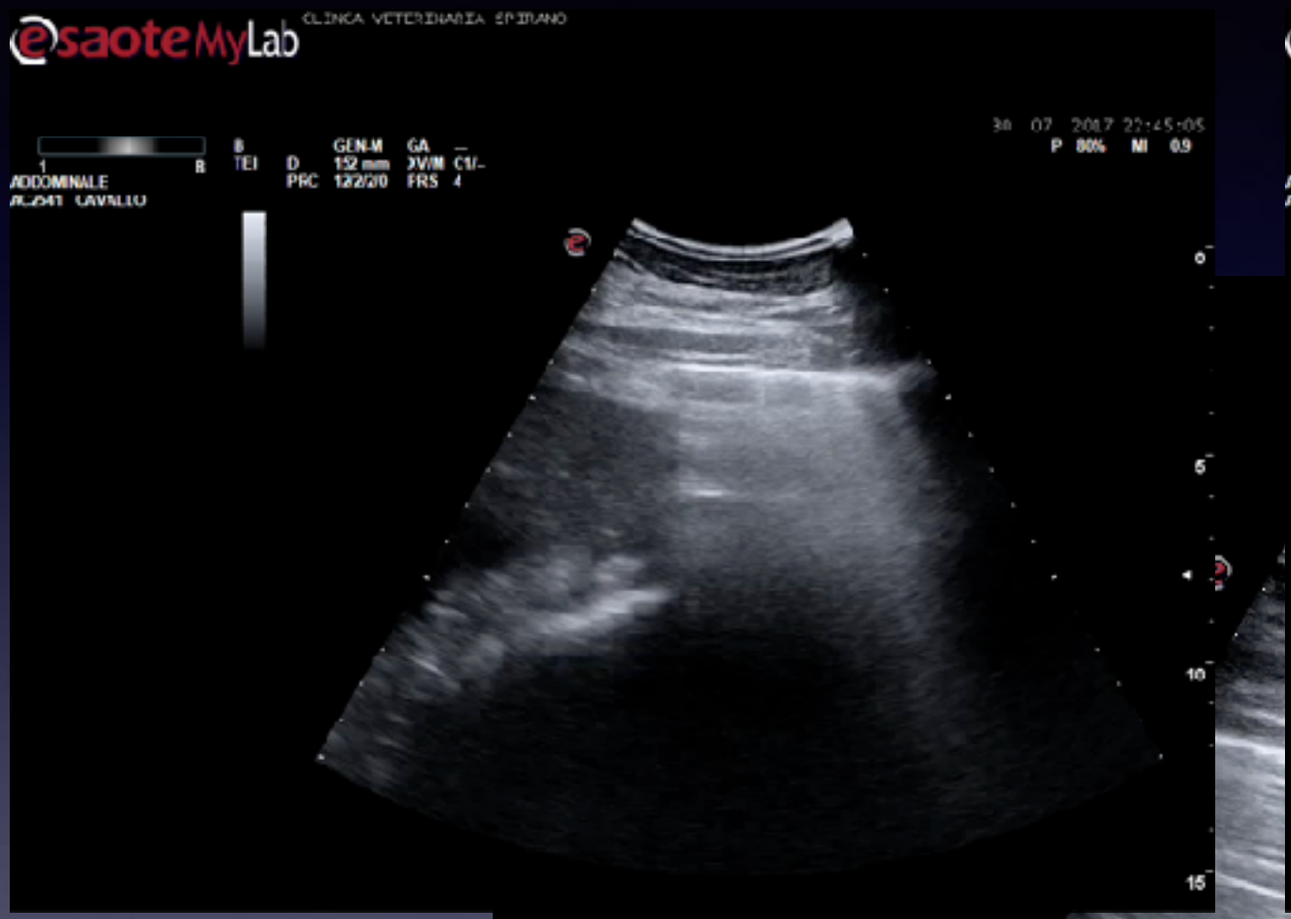
Conclusions and clinical importance: Treatment with rTPA appeared safe and resulted in variable changes in fibrin quantity and organization within the pleural space. Recombinant tissue plasminogen activator could be a useful adjunct to standard treatment of fibrinous pleuropneumonia, but optimal case selection and dosing regimen remain to be elucidated.

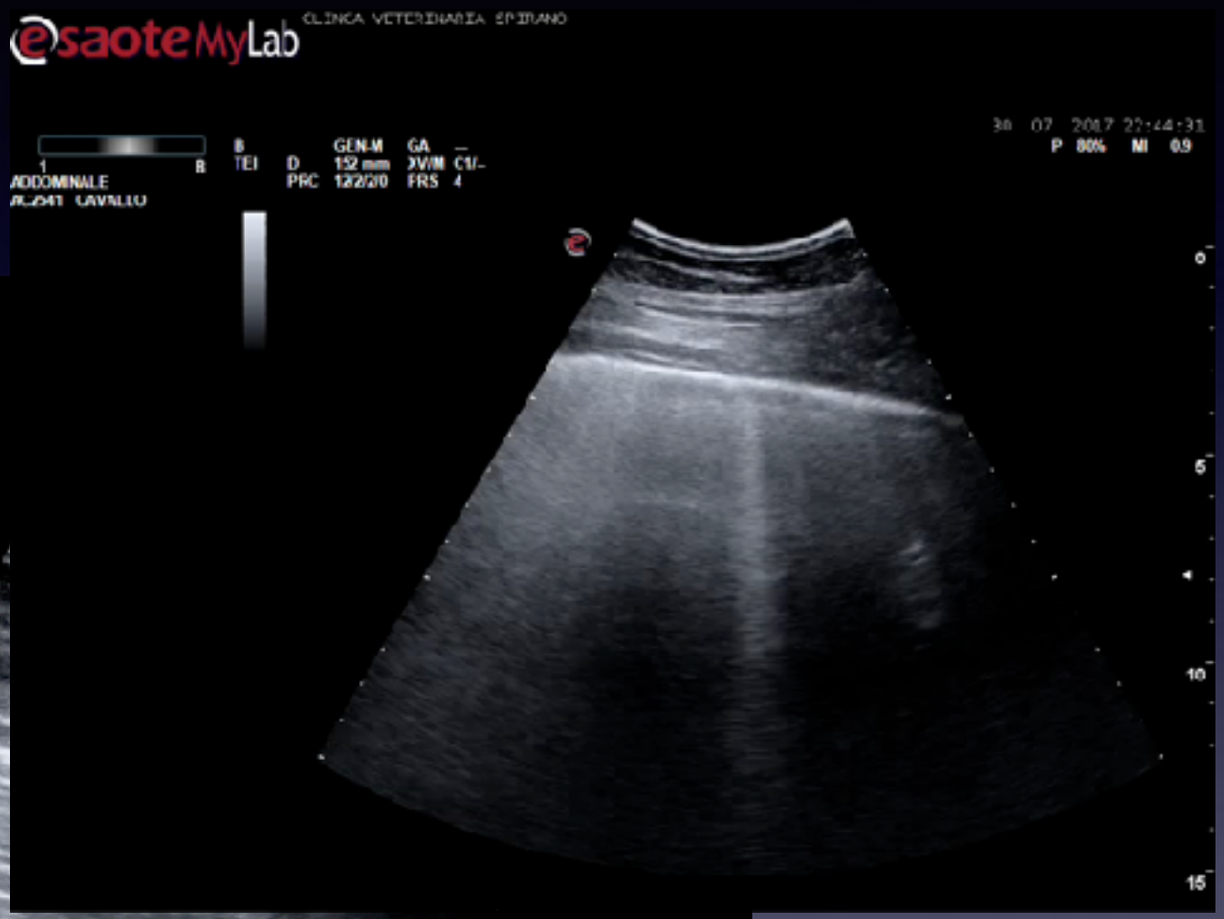
Key words: intrapleural; fibrinolysis; alteplase; pleuritis.

Caso clinico n° 9

- Castrone 16 anni, da concorso ippico, lavoro da maneggio
- Epistassi leggera, bilaterale, da qualche ora
- FC:88, FR:24, Mucose: rosa, RT: 2”, Sensorio: vigile, Appetito:+
- HT:38, PT:5,8, Lattato: 4,4
- Ascoltazione polmonare nella norma

Caso clinico n° 9



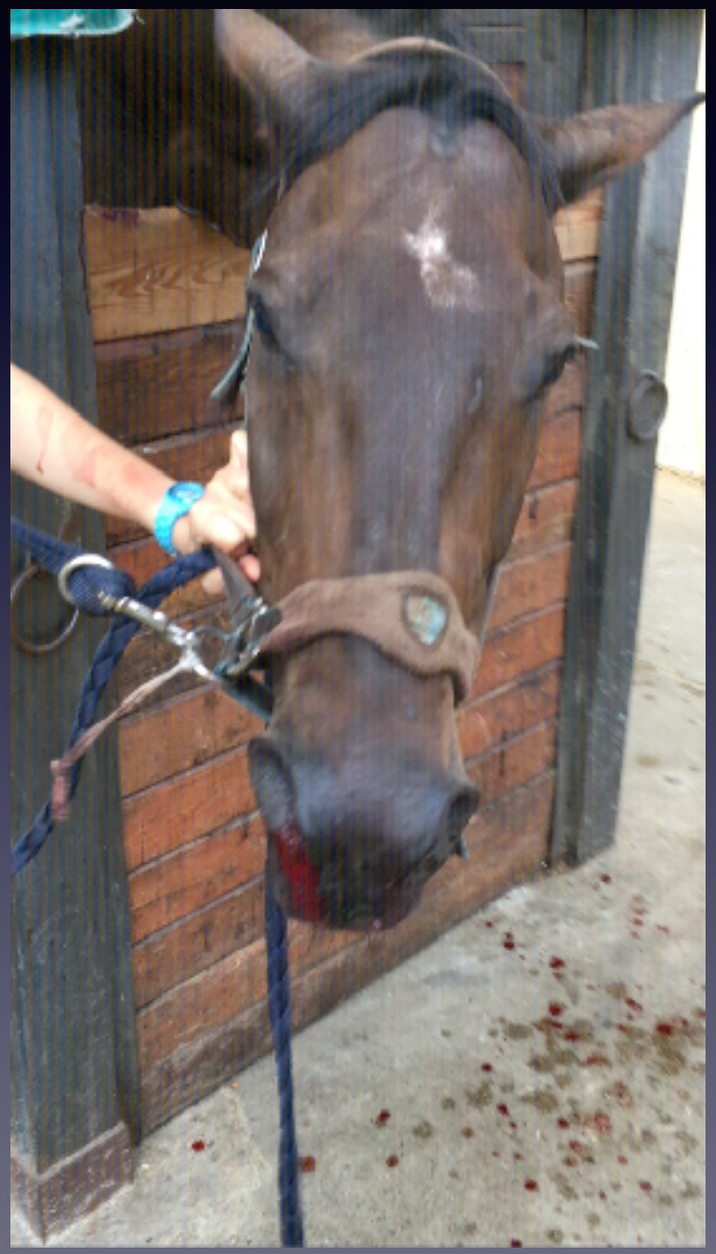


Caso clinico n° 9

TERAPIA

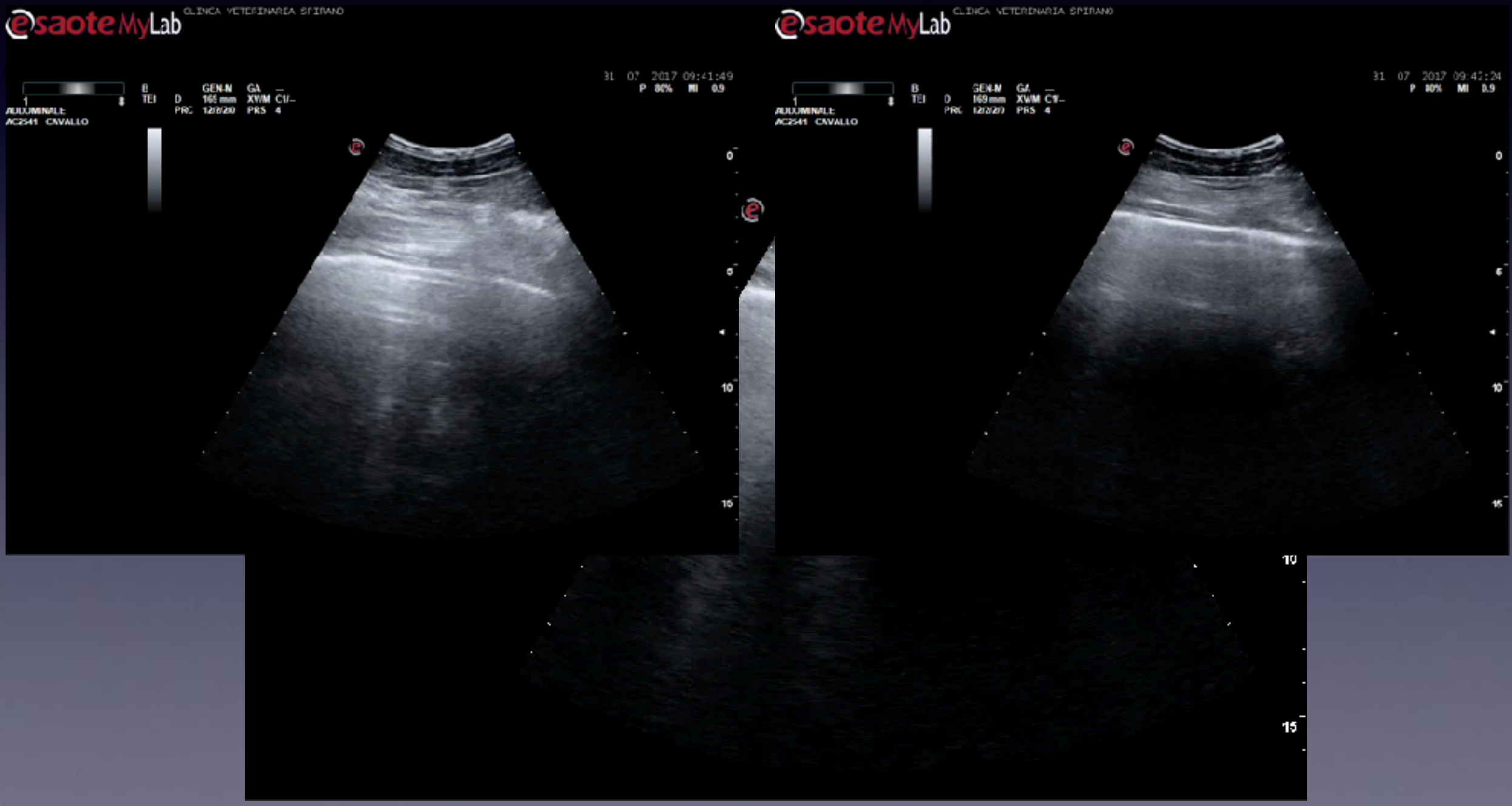
- Antibiotico terapia: Penicillina + streptomicina

Caso clinico n° 9



- FC: 88
- FR: 60
- HT: 33
- HGB: 11,1
- PLT: 32
- WBC: 16,4
- % GRANS: 84%
- % LYMPHO/MONO : 16%
- FIBRINOGENO: 44
- LATTATO: 6,7
- SAA: 432,56
- GLU: 224
- BUN: 26
- CREA: 2,2
- AST: 161
- ALP: 147
- GGT: 15
- TBIL: 1,08
- CK: 195
- PT: 4,7
- ALB: 1,4
- GLOB: 3,3

Caso clinico n° 9



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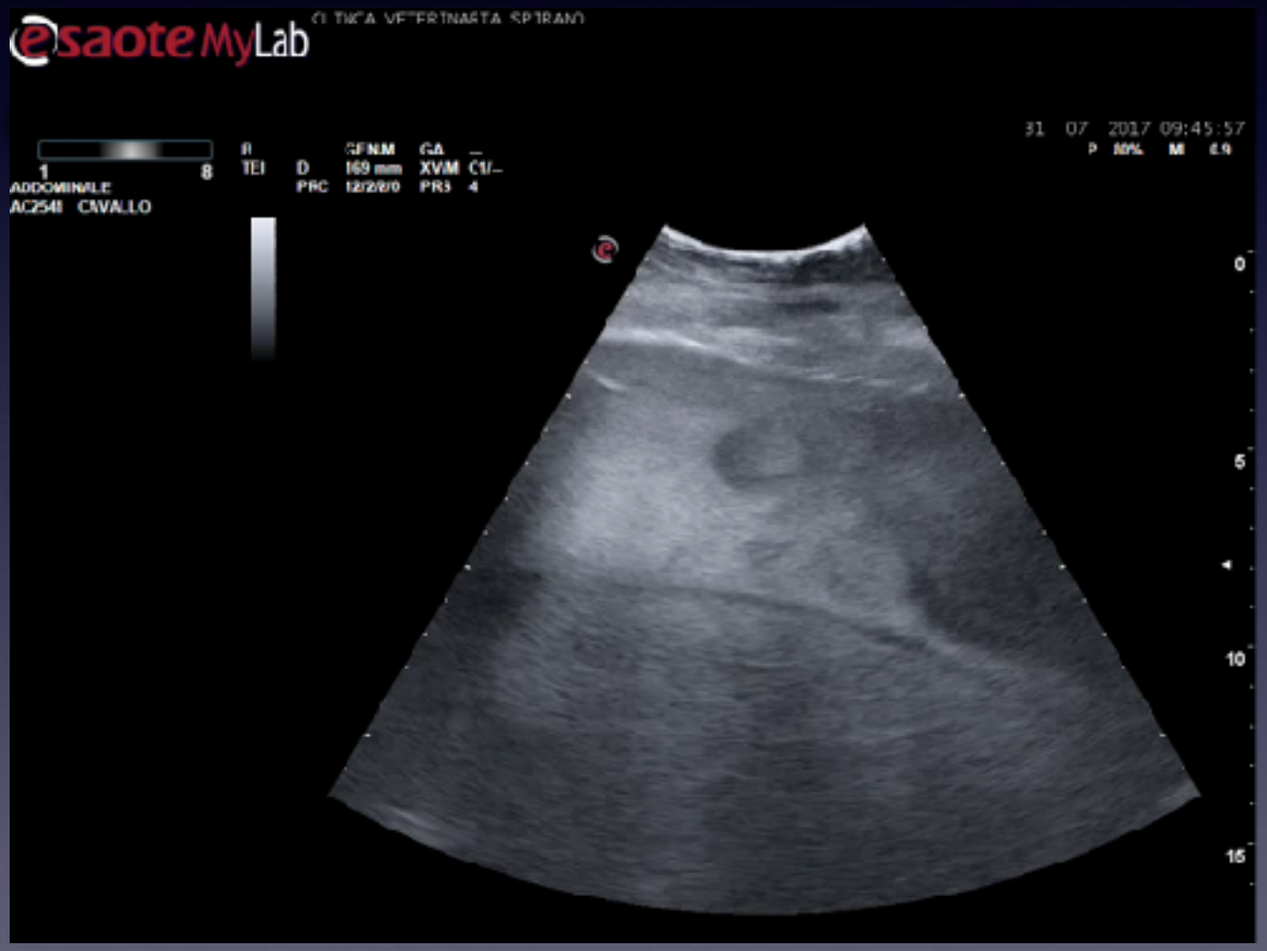
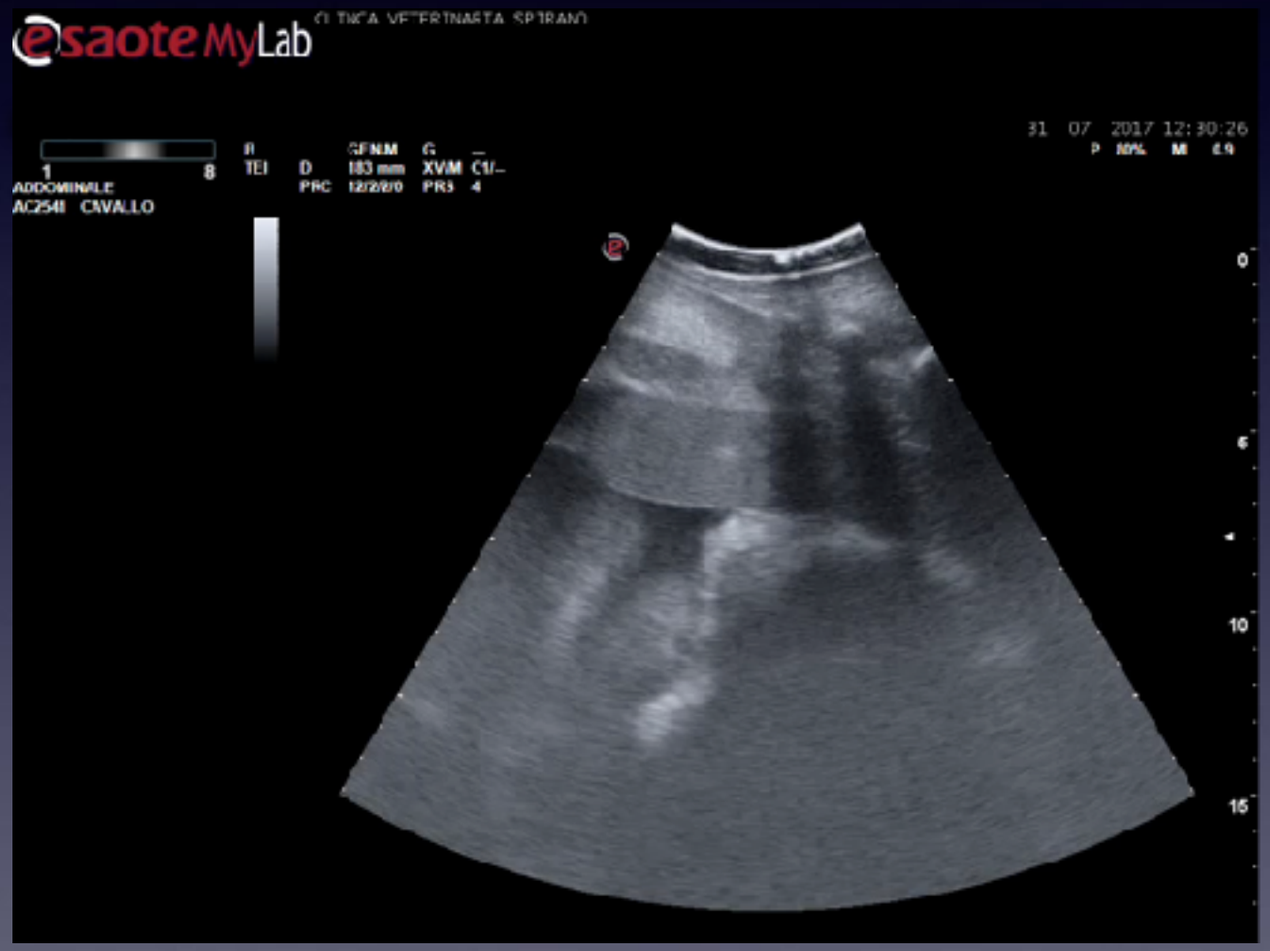




Caso clinico n° 9

- APTT: non determinabile (secondi)
- PT: non determinabile (secondi)
- FIBRINOGENO: > 30 mg/dl

Table 14-4 Reference Ranges for Equine Hemostatic Values*

Parameter	Units	Value	Reference
Platelet count	per µl	75,000-300,000	NCSU†
Fibrinogen	g/dl	<400	NCSU
PT	seconds	8.5-9.9	NCSU
APTT	seconds	30-44	NCSU
FDPs	mg/dl	<20	NCSU
AT III	% PNEP	63-131	Johnstone et al.‡
AT III	% PNHP	218 ± 18	Bernard et al.§
Plasminogen	% PNEP	64.6-155.9	Welles et al.‖
Protein C	% PNEP	104.5 ±13.8	Welles et al.¶

PT, Prothrombin time; *APTT*, activated partial thromboplastin time; FDPs, fibrin degradation products; *AT III*, antithrombin III; *PNEP*, pooled normal equine plasma; *PNHP*, pooled normal human plasma.
*These numbers are for reference only. Each laboratory should establish its own normal equine values.
†Data from *NCSU*, North Carolina State University College of Veterinary Medicine Clinical Pathology Laboratory normal equine reference values;
‡Johnstone IB, Physick-Sheard P, Crane S: Breed, age, and gender differences in plasma antithrombin-III activity in clinically normal young horses, *Am J Vet Res* 50:1751, 1989;
§Bernard W, Morris DD, Divers TJ, et al: Plasma antithrombin-III values in healthy horses: effect of sex and/or breed, *Am J Vet Res* 48:866, 1987;
‖Welles EG, Prasse KW, Duncan A: Chromogenic assay for equine plasminogen, *Am J Vet Res* 51:1080, 1990;
¶Welles EG, Prasse KW, Duncan A, et al: Antigenic assay for protein C determination in horses, *Am J Vet Res* 51:1075, 1990.

Caso clinico n° 9

TERAPIA

- Antibiotico terapia: Penicillina + streptomicina
- Flunixin meglumine: 1,1 mg/kg ev
- Acido Tranexanico: 5-25 mg/kg im, ev, sq, bid
- Desametazone 0,08 mg/kg ev, sid
- Vitamina K1 (Fitomenadione): 0,6 mg/kg im

Caso clinico n° 9

Caso clinico n° 9

- Ricerca anticoagulanti (Brodifacoum, Bromadiolone, Chlorophacinone, Coumachlor, Coumafuryl, Coumatetralyl, Difenacoum, Difethialone, Diphacinone, Flocoumafen, Warfarin)

NON RILEVATI

Caso clinico n° 9

- FC: 88
- FR: 60
- HT: 33
- HGB: 11,1
- **PLT: 32** **100-250 x 10^9 /lt**
  Emorragie spontanee se < 20- 50 x 10^9 /L
- **WBC: 16,4** **5.400-11.500 /uL**
- **% GRANS: 84%** **51-65 %**
- **% LYMPHO/MONO : 16%** **29-41 %**
- **FIBRINOGENO: 44** **100-400 mg /dL** **CID?**
- **LATTATO: 6,7**
- **SAA: 432,56** **0-20 mg /L**

DISCUSSIONE

- **GLU: 224**
- BUN: 26
- CREA: 2,2
- AST: 161
- ALP: 147
- GGT: 15
- TBIL: 1,08
- CK: 195
- **PT: 4,7** **5,8-7,7 g /dL** **CID?**
- **ALB: 1,4**
- GLOB: 3,3

Caso clinico n° 9

DISCUSSIONE

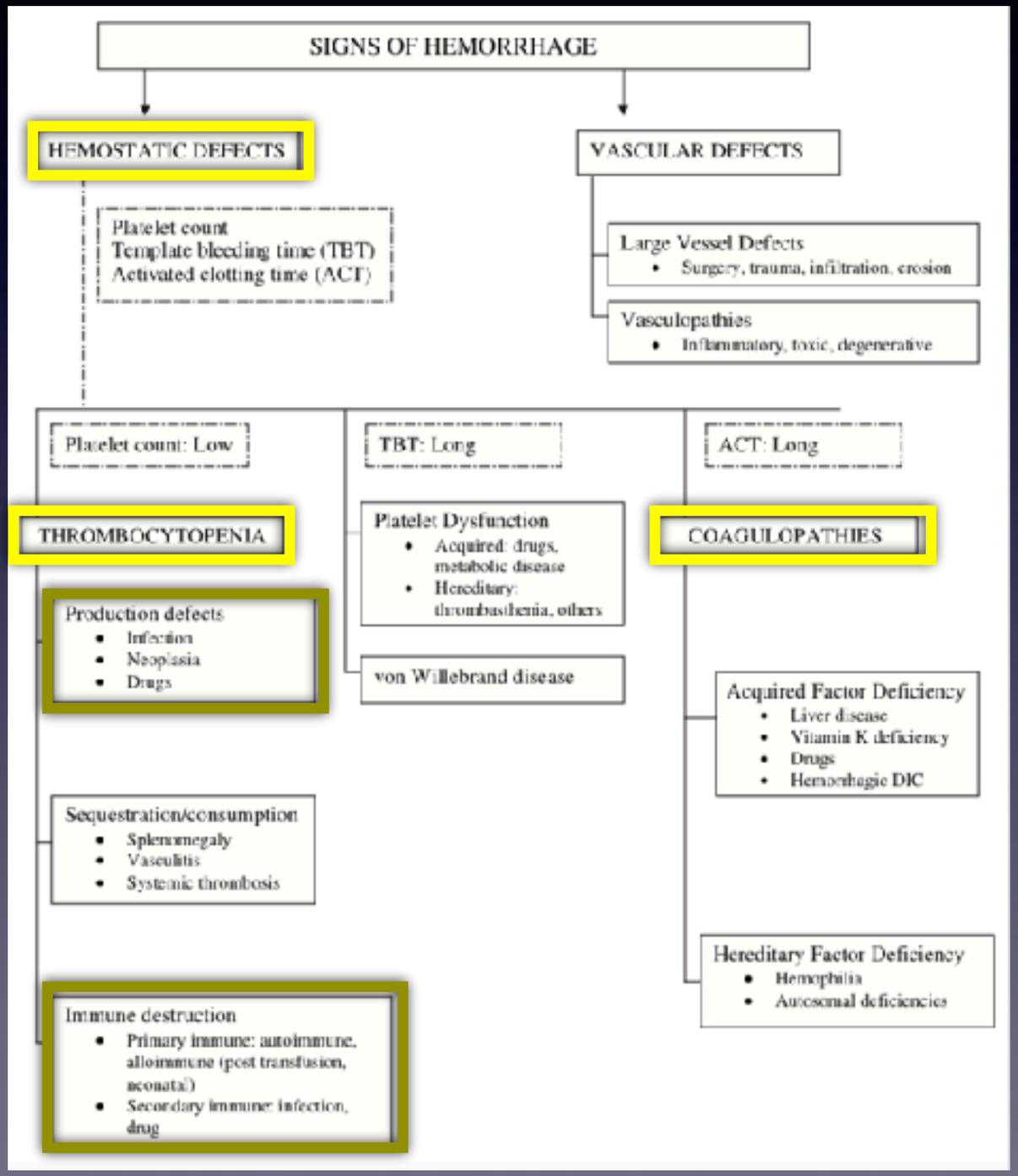
Table 3
Acquired hemostatic defects

Classification	Pathogenesis
Thrombocytopenia	Decreased production (bone marrow disease infectious, neoplastic, and aplastic) Increased use or sequestration (splenomegaly, vasculopathies, and systemic thrombosis) Increased destruction (primary or secondary immune-mediated and alloimmune)
Platelet dysfunction	Metabolic disease (uremia, hepatic failure, hyperproteinemia, and severe anemia) Drugs (nonsteroidal anti-inflammatory agents, plasma expanders, and sulfa drugs) Hepatic synthetic failure (acute hepatitis, necrosis, and chronic cirrhosis) Vitamin K deficiency (cholestatic disease and vitamin K antagonist toxicities) Disseminated intravascular coagulation: factor activation/secondary depletion (systemic inflammatory syndromes, neoplasia, and severe tissue injury)

Equine Coagulopathies

Marjory B. Brooks, DVM

Caso clinico n° 9



Equine Coagulopathies

Marjory B. Brooks, DVM

Vet Clin Equine 24 (2008) 335–355

Caso clinico n° 9

10.20-10.40

Clotting disorders

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The more we learn about the mechanisms involved in clotting, the more complicated it becomes. Classically, factors involved in clotting include platelets, the intrinsic and extrinsic clotting cascade, which are now known to interact significantly and the antifibrinolytic system.

PLATELET FACTORS

Associated with increased buccal mucosal bleeding time and petechial and ecchymotic haemorrhages, epistaxis, increased bleeding after venepuncture and rarely melaena and hyphaema (blood in the anterior chamber). Clinical signs not seen until count <30 x 10^9/l.

Reduced platelet production

Potential causes

Bone marrow (BM) aplasia, neoplastic infiltration and severe inflammation. In addition, myelosuppressive drugs including phenylbutazone (chloramphenicol) and oestrogens can do this. Rare causes include a familial neutropenic, thrombocytopenic syndrome reported in Standardbreds causing BM hypoplasia (Kohn *et al.* 1995).

Abnormal platelet distribution (sequestration)

e.g. splenomegaly

Increased platelet consumption or destruction

ELSEVIER
SAUNDERS

VETERINARY
CLINICS
Equine Practice

Vet Clin Equine 24 (2008) 335–355

Equine Coagulopathies

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Normal hemostasis: cell-based model

The original "cascade" model of coagulation involves two distinct series of activation reactions (the intrinsic and extrinsic pathways) that converge to produce thrombin, which, in turn, transforms soluble plasma fibrinogen into an insoluble fibrin clot. Although this model is still useful for understanding in vitro coagulation, a more complex "cell-based" model has been developed to reflect better the in vivo formation of a hemostatic plug [1,2]. In this model, the availability and activation status of a variety of cell types regulate the localized formation of a platelet and fibrin plug. The cell-based model consists of three overlapping phases: initiation, amplification, and propagation (Fig. 1). The initiation phase is localized to cells expressing tissue factor (TF), a membrane antigen generally restricted to

Caso clinico n° 10

- Femmina 6 anni, Q.H.
- Febbre di origine sconosciuta nelle settimane precedenti, riacutizzatasi da qualche giorno.
- FC:44, FR:20, Mucose: Rosa, Sensorio: Vigile, Temp: 37,5°
- Ascoltazione polmonare normale
- Ecografia toracica e polmonare normale

Caso clinico n° 10

Al ricovero

Caso clinico n° 10

- HT: 15,4
- HGB: 5,7
- PLT: 17
- WBC: 2,5
- % GRANS: 60%
- % LYMPHO/MONO : 40%
- FIBRINOGENO: 581
- PROTEINE TOT: 8,5

DOPO 4 GG

- HT: 16
- HGB: 5,9
- PLT: 17
- WBC: 2,4
- % GRANS: 62%
- % LYMPHO/MONO : 38%
- FIBRINOGENO: 642
- PROTEINE TOT: 8,9

DOPO 10 GG

- HT: 12,7
- HGB: 4,7
- PLT: 16
- WBC: 2,2
- % GRANS: 68%
- % LYMPHO/MONO : 32%
- FIBRINOGENO: 555
- PROTEINE TOT: 8,9

Caso clinico n° 10

TERAPIA

- Antibiotico terapia (Ceftiofur 2,2 mg/kg ev, im, bid poi Ossitetraciclina 10 mg/kg ev, sid)
- Desametazone 0,08 mg/kg ev, sid
- Azatioprina 2 mg/kg per os, sid
- Terapia di sostegno (complesso vitaminico)

Caso clinico n° 10

TEST DI COOMBS DIRETTO (Anemia emolitica autoimmune):

- NEGATIVO

TEMPO DI PROTROMBINA

- 12,1 sec. (10/15 sec)

aPTT

- 35,9 sec. (39-63 sec)

TEMPO DI TROMBINA

- 21,5 sec. (21/29 sec)

FIBRINOGENO

- 351 (132/310 mg/dl)

DOPO 11 GG

- HT: 17
- HGB: 5,7
- PLT: 7
- RBC: 3,1
- WBC: 1,3
- % GRANS: 13%
- % LYMPHO/MONO : 87% (presenza di linoociti reattivi, in parte ingrossati)

PCR BABESIA spp, ANAPLASMA PHAGOCYTOPHILUM

- NEGATIVO

Caso clinico n° 10

Table 3
Acquired hemostatic defects

Classification	Pathogenesis
Thrombocytopenia	Decreased production (bone marrow disease: infectious, neoplastic, and aplastic)
	Increased use or sequestration (splenomegaly, vasculopathies, and systemic thrombosis)
	Increased destruction (primary or secondary immune-mediated and alloimmune)
Platelet dysfunction	Metabolic disease (uremia, hepatic failure, hyperproteinemia, and severe anemia)
	Drugs (nonsteroidal anti-inflammatory agents, plasma expanders, and sulfa drugs)
	Hepatic synthetic failure (acute hepatitis, necrosis, and chronic cirrhosis)
	Vitamin K deficiency (cholestatic disease and vitamin K antagonist toxicities)
	Disseminated intravascular coagulation: factor activation/secondary depletion (systemic inflammatory syndromes, neoplasia, and severe tissue injury)

Caso clinico n° 10

ESAME CITOLOGICO DEL MIDOLLO OSSEO

- DESCRIZIONE: il prelievo appare povero di cellule. Su un fondo emorragico si osservano essenzialmente rari leucociti di derivazione ematica probabile e squame di cheratina da contaminazione. Assenza di grani midollari e di megacariociti. L'esame citologico dello striscio conferma la pancitopenia segnalata dall'emogramma e mette in evidenza la presenza di una discreta poichilocitosi.

- INTERPRETAZIONE: a causa della paucicellularità del prelievo, la citologia non è in grado di emettere una diagnosi.

- COMMENTO: si consiglia la ripetizione dell'esame e l'invio di altri 2-3 vetrini. Si consiglia anche di effettuare una biopsia per controllo istologico, in quanto è forte il sospetto di anemia aplastica e di escludere in priorità una infezione da Leishmania ed Ehrlichia.

Caso clinico n° 10

“Bone Marrow Suppression

Anemia caused by selective bone marrow suppression of erythropoiesis is unusual in the horse. Selective erythroid hypoplasia has been reported as a sequela to administration of recombinant human erythropoietin to horses. Anemia is thought to result from production of antibodies that cross-react with erythropoietin, inhibiting erythropoiesis. Treatment is with blood transfusions and corticosteroids. Prognosis for recovery is fair to guarded.

Pancytopenia (decreased circulating erythrocytes, leukocytes, and platelets) has been described in horses with **aplastic bone marrow disorders** and with myelophthisic diseases. Myelophthisis is a reduction in the cellular elements of the bone marrow, frequently a result of metastatic neoplasia or myelofibrosis, a proliferation of fibrous tissue in the marrow. Myelodysplasia implies accumulation of abnormal cells in the bone marrow. These cells are not usually dysplastic in the truest sense of the word, but rather they result from myeloproliferative neoplasia. Hematopoietic neoplasia is rare in the horse and is discussed in more detail later in this chapter. These conditions usually result in concurrent anemia, leukopenia, and thrombocytopenia. Diagnosis is by identification of abnormal cells in bone marrow aspirates.

“Bone marrow from horses with **aplastic anemia** is generally devoid of hematopoietic precursor cells because of a congenital or acquired failure of stem cell function. **Acquired aplasia may be associated with bacterial and viral infections**, chronic renal or hepatic disease, neoplasia, irradiation, or **drug therapy. In most cases a predisposing factor is not identified**. Because of the shorter life spans of the cells, leukopenia and thrombocytopenia usually precede anemia. Diagnosis depends on bone marrow biopsy. If one cannot obtain adequate marrow by serial aspirates, then one must obtain a core biopsy sample. Hypoplastic marrow is yellow because of fatty infiltration with a mixture of cell types, but the number of erythroid, myeloid, and megakaryocytoid cells is decreased. Aplastic anemia is thought to be an autoimmune disorder in some cases, and some horses have apparently benefited from therapy with corticosteroids and anabolic steroids. These drugs stimulate erythropoietin production and increase sensitivity of the stem cell receptors to erythropoietin.

Temporary or permanent bone marrow aplasia has been associated with the administration of a variety of drugs in other species.

“ Transient hypoplastic anemia after phenylbutazone administration has been reported in one horse. Aplastic anemia has been induced experimentally by feeding of trichloroethylene-extracted soybean oil meal to horses or by irradiation. Chloramphenicol produces transient and irreversible forms of bone marrow dysfunction in human beings, and similar reactions are possible in other species. One always should handle chloramphenicol with caution because human toxic reactions appear to be idiosyncratic and may be “associated with absorption of low amounts of the drug. Any ongoing drug therapy should be discontinued in horses with aplastic anemia, and supportive therapy should be administered. Some cases of drug-induced bone marrow failure are temporary, and hematopoietic function returns to normal with time if secondary complications are not severe. One may administer compatible whole blood transfusions to horses with severe anemia or thrombocytopenia, and systemic antibiotics and strict isolation from other animals may be considered for horses with severe leukopenia.

Bone marrow erythropoiesis frequently is suppressed in horses with chronic renal disease. Suppression generally is attributed to a decrease in erythropoietin production by the kidneys. Recombinant erythropoietin has been used successfully in cats and human beings with severe anemia associated with renal disease.”

Stephen M. Reed, DVM, Dipl ACVIM , Warwick M. Bayly, BVSc, MS, PhD, Dipl ACVIM , Debra C. Sellon, DVM, PhD, Dipl ACVIM. “Equine Internal Medicine”.

Caso clinico n° 10

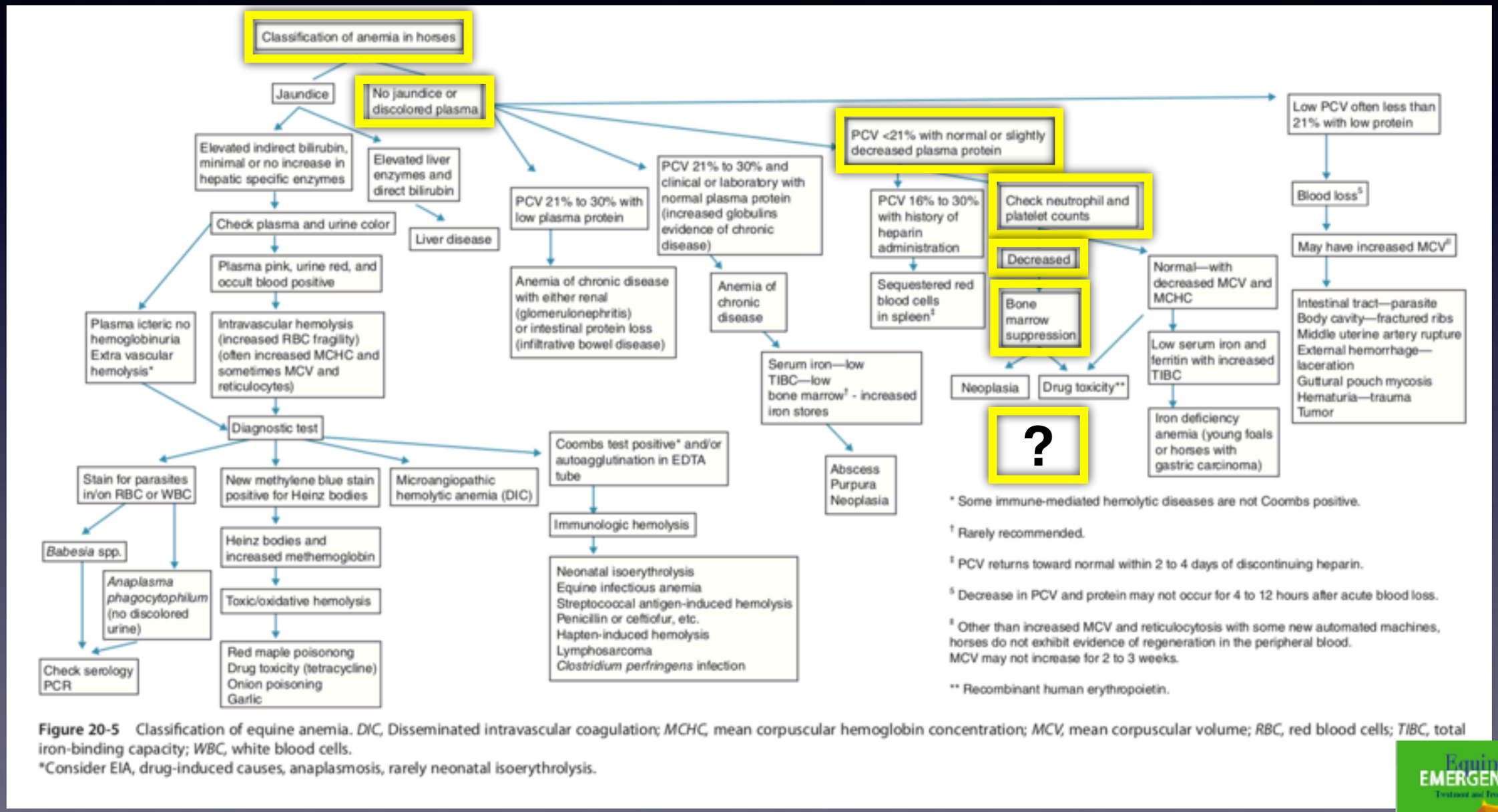
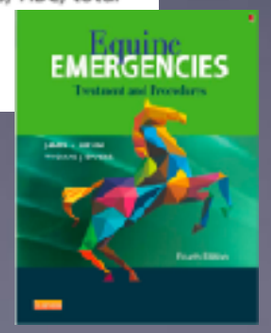


Figure 20-5 Classification of equine anemia. *DIC,* Disseminated intravascular coagulation; *MCHC,* mean corpuscular hemoglobin concentration; *MCV,* mean corpuscular volume; *RBC,* red blood cells; *TIBC,* total iron-binding capacity; *WBC,* white blood cells.
*Consider EIA, drug-induced causes, anaplasmosis, rarely neonatal isoerythrolysis.



Caso clinico n° 10

Equine Veterinary Education

EQUINE VETERINARY EDUCATION
Equine vet. Educ. (2012) ** (**) **–**
doi: 10.1111/eve.12000

Case Report

Pancytopenia in two horses following administration of potentiated sulfonamide antimicrobials

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Keywords: horse; potentiated sulfonamide; adverse drug reactions; pancytopenia; anaemia; thrombocytopenia

How to Obtain a Diagnostic Bone Marrow Sample From the Sternum of an Adult Horse

Debra C. Sellon, DVM, PhD, Diplomate ACVIM

AAEP PROCEEDINGS Vol. 52 2006 621

Corticosteroids and Immune Suppressive Therapies in Horses

Mathilde Leclere, DVM, PhD

Vet Clin Equine 33 (2017) 17–27

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Journal Web site
How to Submit

Can J Vet Res. 2012 Jan; 76(1): 52–56. PMCID: PMC3244288

Language: English | Frenc

Comparison of bone marrow aspiration at the sternum and the tuber coxae in middle-aged horses

Uta Delling, Katrin Lindner, Iris Ribitsch, Henriette Jülke, and Walter Brehm

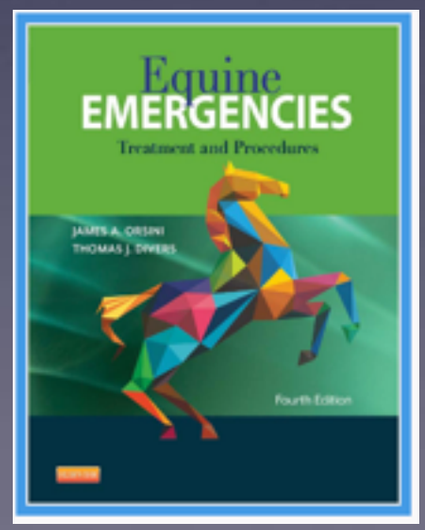
Equine immunologic disorders

Thomas J. Divers

DVM, DACVIM, DACVECC
College of Veterinary Medicine, Cornell University, Ithaca, NY

Trasfusione di sangue

- *Practice Tip: Perform blood transfusion in the following clinical situations:*
  - PCV decreases to <20% in the first 12 hours, and bleeding or hemolysis is ongoing.
  - PCV decreases to <12% over 1 to 2 days; hemoglobin of <5 g/dL generally has a detrimental effect on tissue oxygenation.
  - Elevated lactate and low PvO_2 and/or SvO_2 exists in addition to the above.
  - In peracute cases, death from hemorrhage can occur without a decrease in PCV.
    In these cases, the need for transfusion is based on:
    - The presence of severe tachycardia
    - White to gray mucous membranes
    - Signs of hypotension—-weak pulses, “cold sweat,” general weakness, and evidence of severe bleeding



Trasfusione di sangue

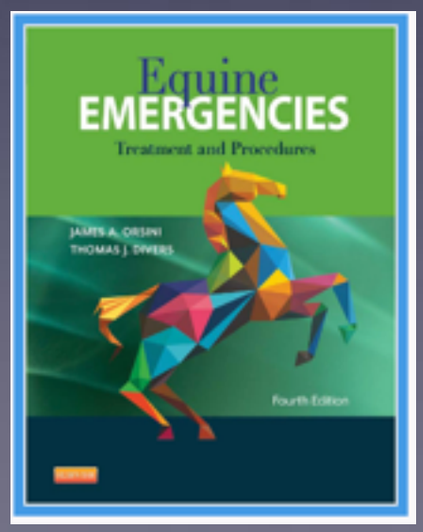
. **How Much Blood to Administer**

Practice Tip: For hemorrhage in an adult horse it is usually necessary to administer a minimum of 6 to 8 L in an adult or at least 30% to 40% of the estimated blood loss.

• *Note:* Decreases in CVP (jugular II) and increases in blood lactate occur with 15% to 20% of blood loss, but heart rate increases and blood pressure changes may not occur until there is at least 20% of blood loss. A 500-kg horse has approximately 45 L of blood.

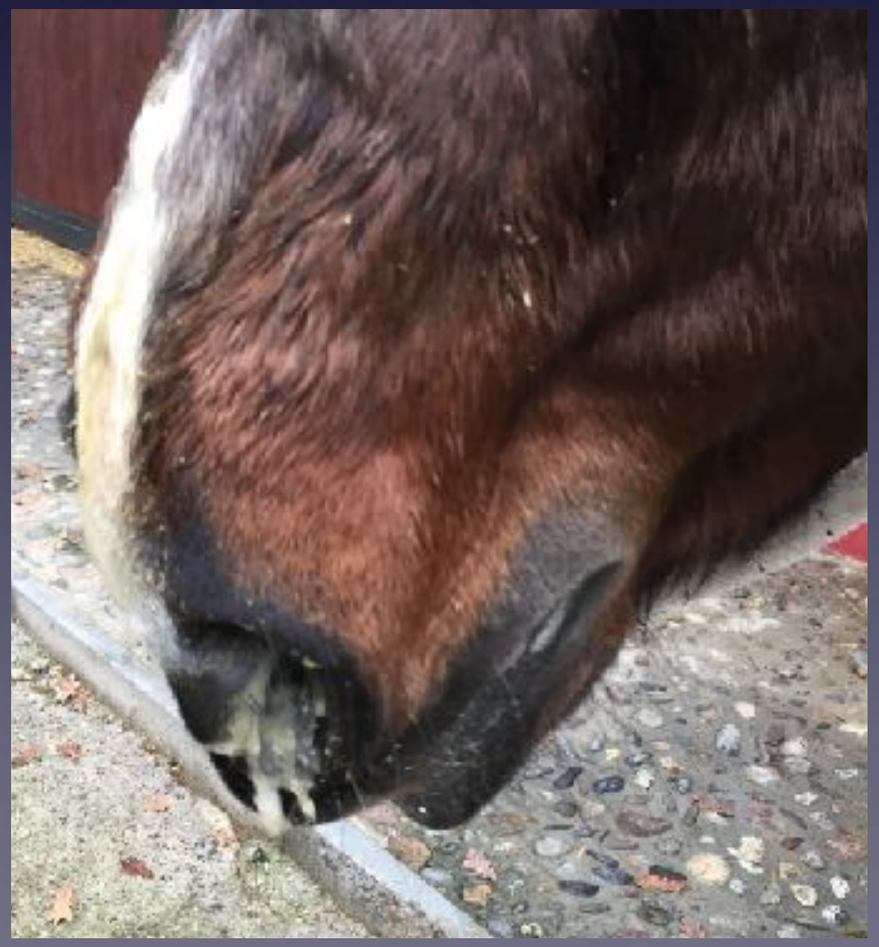
Administer concurrent polyionic uids and plasma.
For hemolysis use the following equation to calculate blood volume needed:

Desired PCV − PCV recipient × (0.08 × Body weight in kilograms of recipient)
PCV of donor = Liters of blood needed

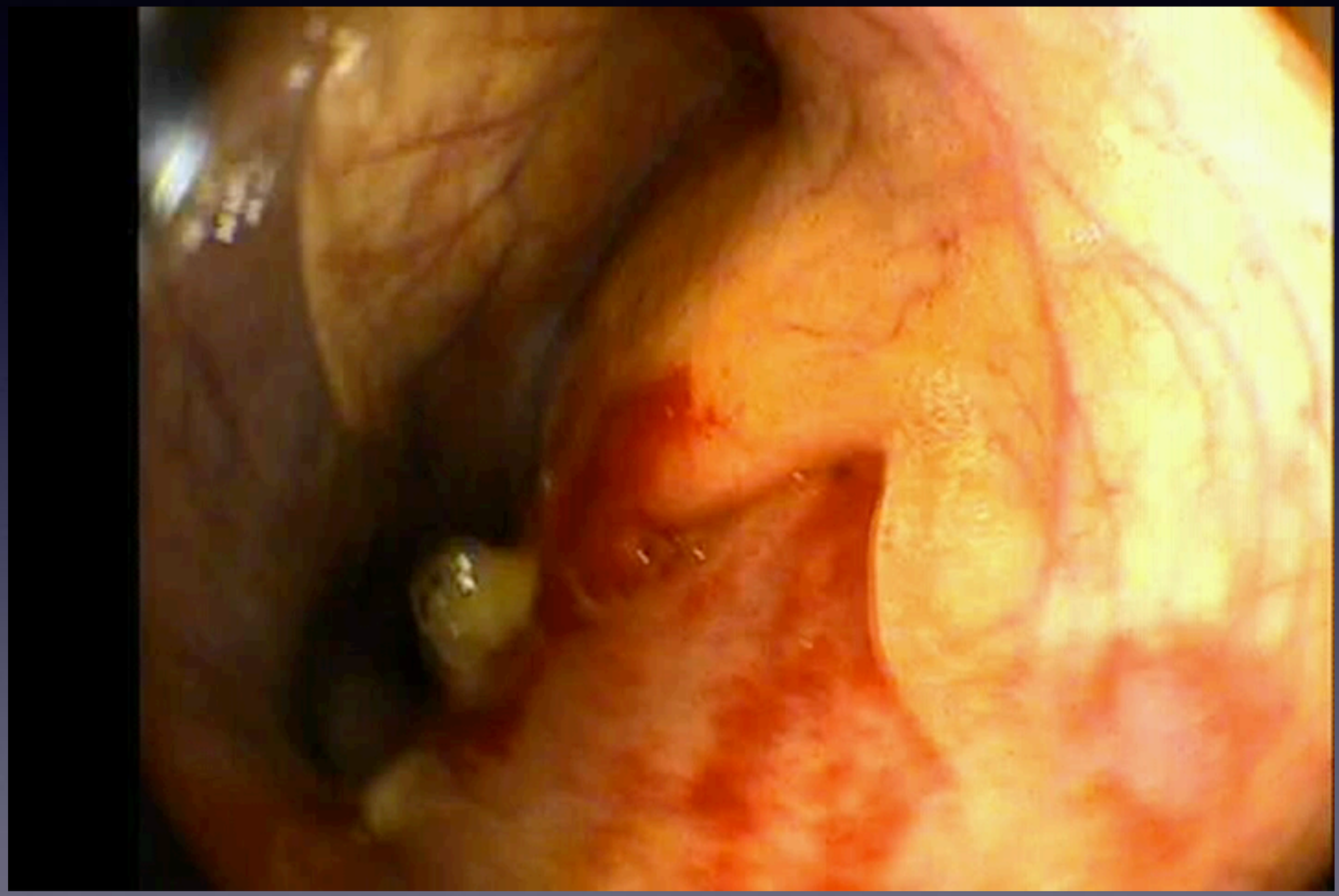


Caso clinico n° 11

- Femmina 18 anni, Sella, fattrice
- Disfagia da un paio di giorni, peggiorata nelle ultime 24h
- FC:48, FR:35, Mucose: Rosa, Sensorio: Vigile, Temp: 38,7°,
- Aumento del murmure vescicolare



Caso clinico n° 11



Caso clinico n° 11

Caso clinico n° 11

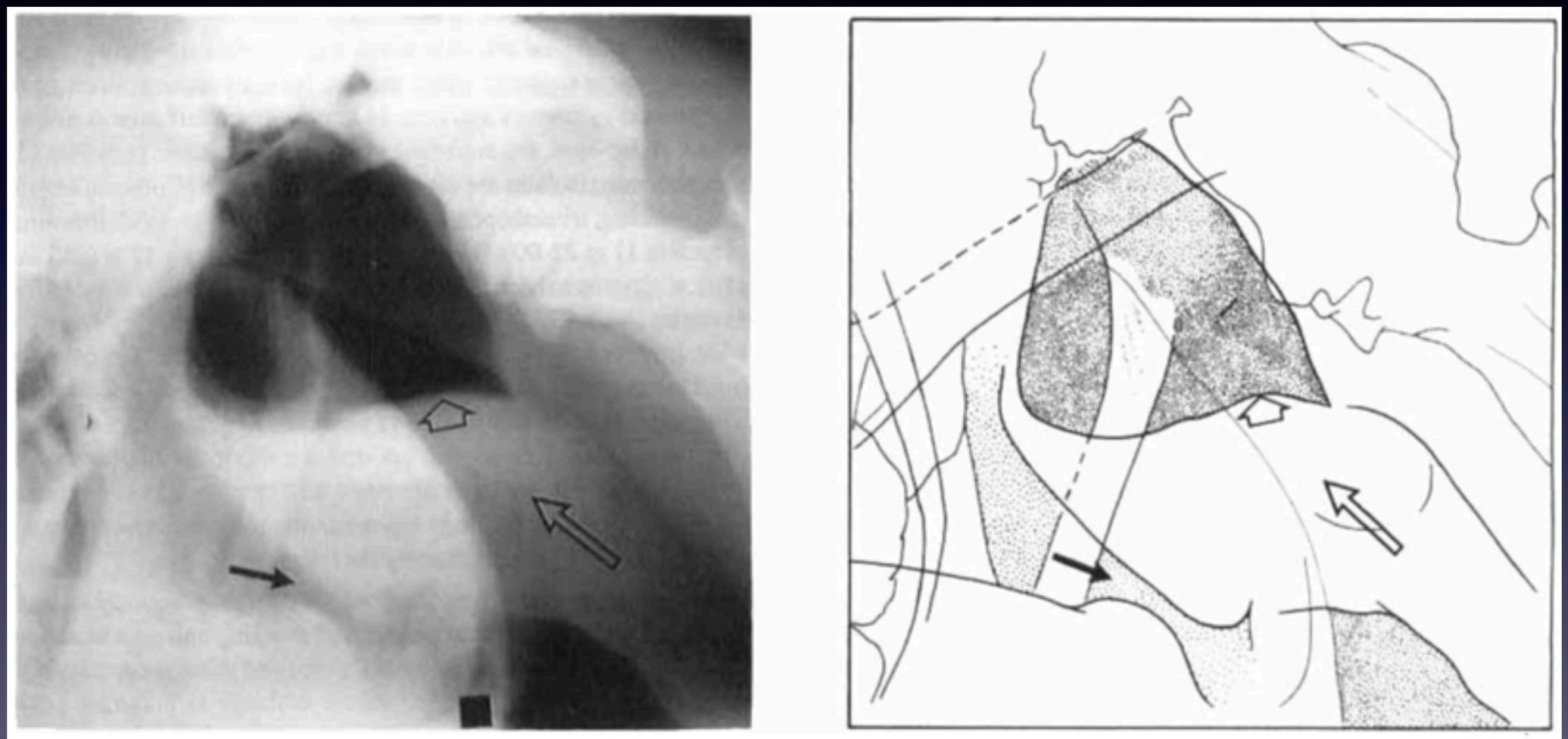


Figure 1. Lateral radiograph (left) and schematic drawing (right) of a horse with a large retropharyngeal mass (long open arrow). Note the reduced pharyngeal airway (closed arrow) and dorsal displacement of the ventral border of the guttural pouch (short open arrow).

Retropharyngeal lymph node infection in horses: 46 cases (1977-1992)

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Rural Veterinary Centre, Department of Animal Health, The University of Sydney, Werombi Road, Camden, New South Wales 2570

Caso clinico n° 11

TERAPIA

- Antibiotico terapia: Penicillina + streptomicina
- Flunixin meglumine: 1,1 mg/kg ev

Caso clinico n° 11

23° CONGRESSO INTERNAZIONALE SIVE

EQUINE DYSPHAGIA: A REVIEW OF 17 CASES

B. Conturba, DVM[2], E. Salsecci, DVM[3], S. Ceriotti, DVM[1,2], L. Stucchi, DVM[1,2], G. Stancari, DVM, PhD[1,2], E. Zucca, DVM, PhD, Researcher[1,2], F. Ferrucci, DVM, PhD, Associate Professor[1,2]

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Work type: **Clinical Case**

Topic: **Internal Medicine**

Caso clinico n° 11

EQUINE VETERINARY EDUCATION
Equine vet. Educ. (2006) **18** (4) 199-204

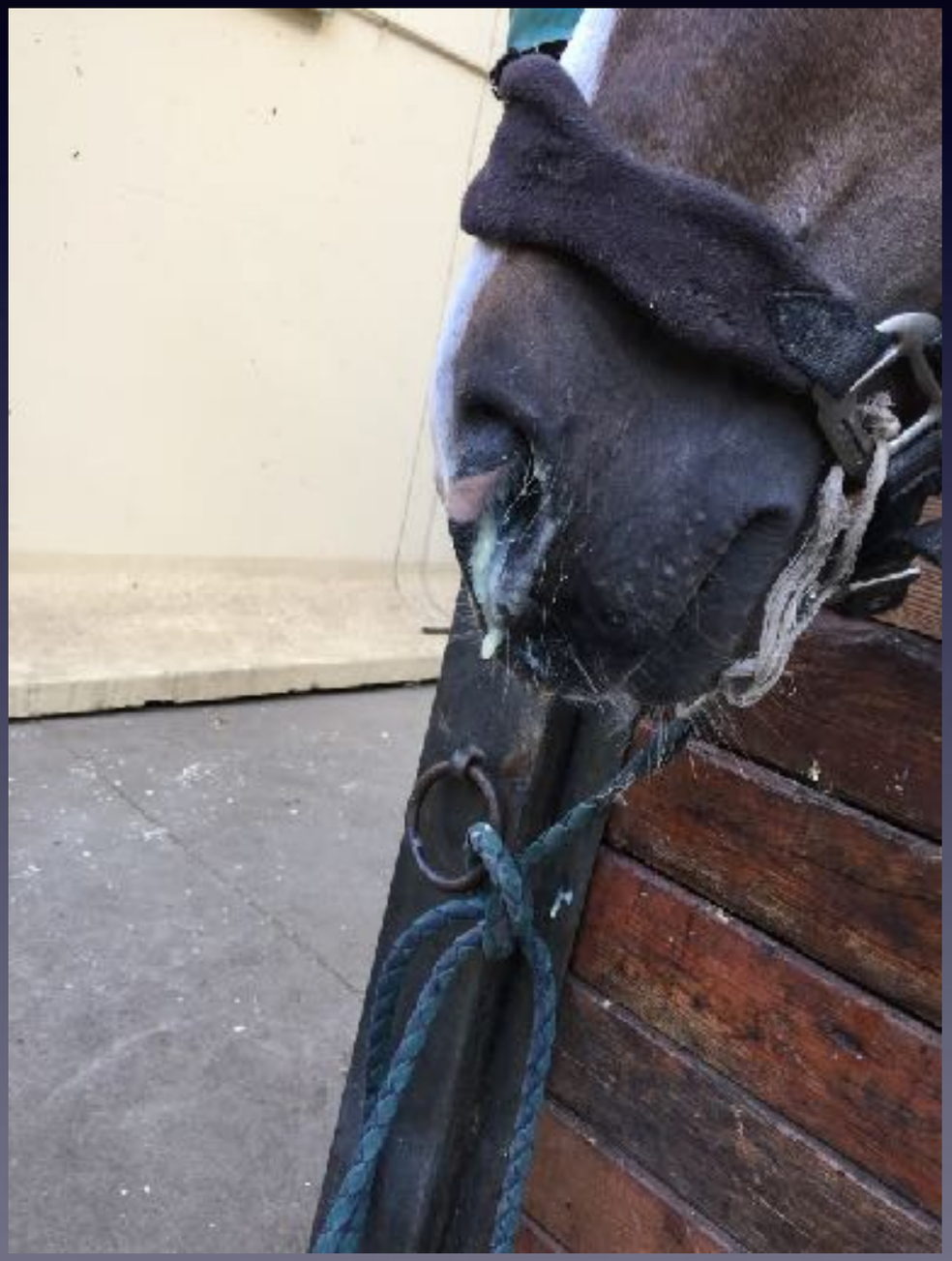
Case Report

A review of 4 cases of dysphagia in the horse: buccal abscess, lingual abscess, retropharyngeal foreign body and oesophageal obstruction

N. P. H. HUDSON*, B. C. MCGORUM AND P. M. DIXON

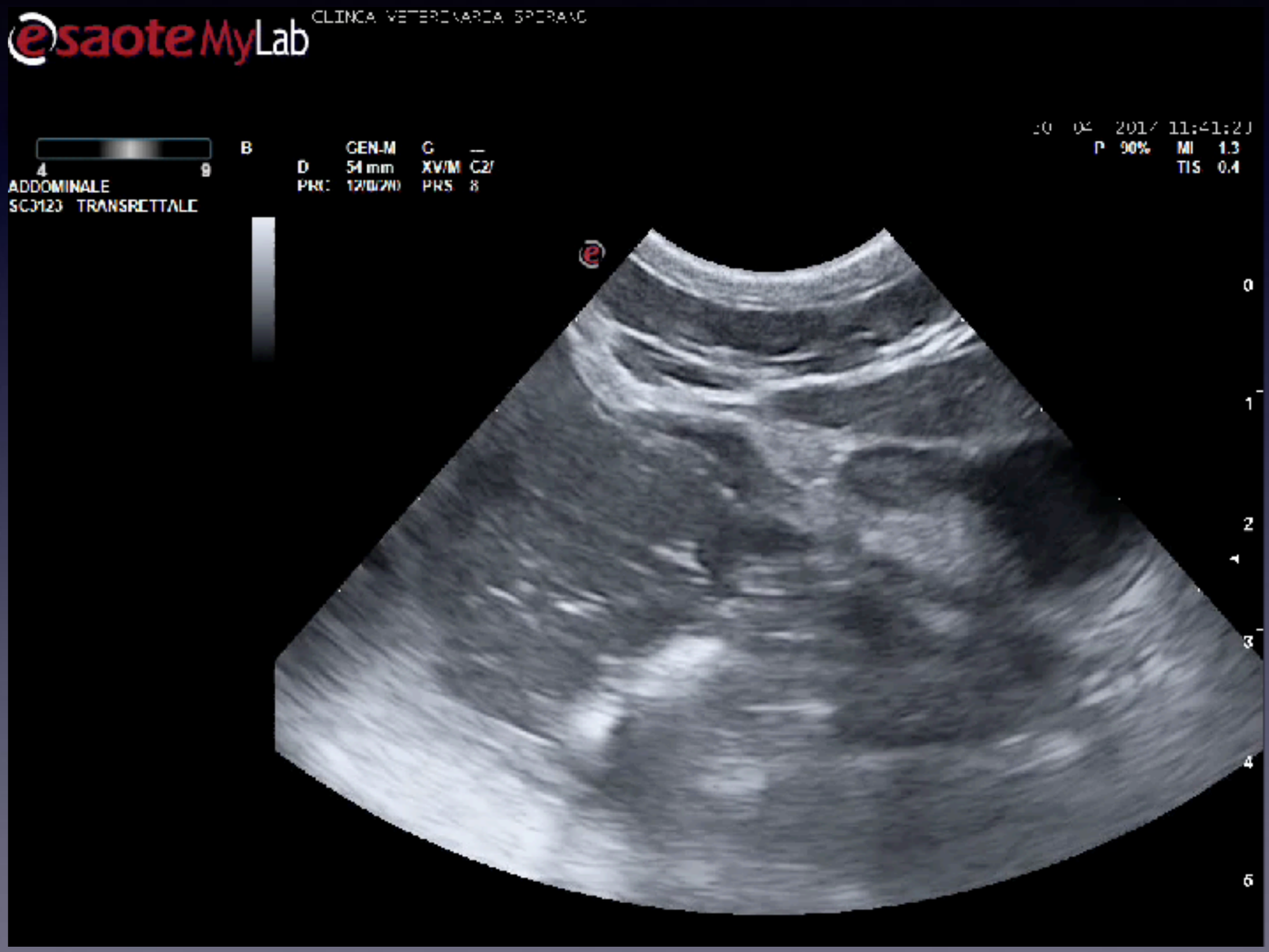
Caso clinico n° 12

- Femmina 11 anni, Sella, in lavoro (concorso ippico)
- Febbre da 2 settimane, nessuna risposta agli antibiotici
- Scolo nasale bilaterale da qualche giorno
- FC:38, FR:12, Mucose: Rosa, Sensorio: Vigile, Temp: 40,5°,
- HT:30, PT:7,9, Lattato: 1,5, WBC:14,5 Neutrofili 87%, Fibrinogeno: 605
- Siero Amiloide A: 148,90
- Ascoltazione polmonare normale



Caso clinico n° 12

Caso clinico n° 12



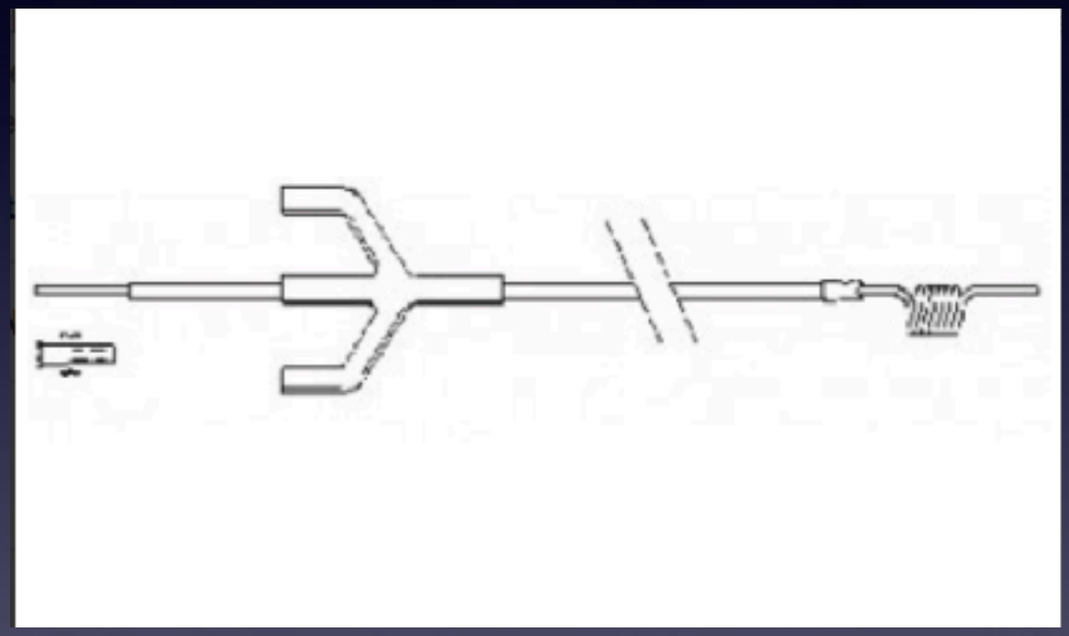
Caso clinico n° 12

Caso clinico n° 12

TERAPIA

- Antibiotico terapia (Ceftiofur 2,2 mg/kg ev, im, bid) per 7 gg
- Flunixin meglumine 1,1 mg/kg ev, sid, per 3 gg
- Lavaggio locale (2 lt. Sol. Fisiologica + 30 ml. Acetilcisteina) per 5 gg

Aspetto pratico



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Medical Instrumentation for Animals

Aspetto pratico

Aspetto pratico

Caso clinico n° 12

Caso clinico n° 13

- Castrone 22 anni, Sella,
- Disfagia da vari giorni
- FC:20, FR:20, Mucose: Rosa, Sensorio: Vigile, Temp: 37,9°,
- HT: 26, PT:7,3, Lattato: 1,4 WBC:6,5 Neutrofili 82%, Piastrine: 204 Fibrinogeno: 329
- Siero Amiloide A: 3,26
- Ascoltazione polmonare nella norma

Caso clinico n° 13

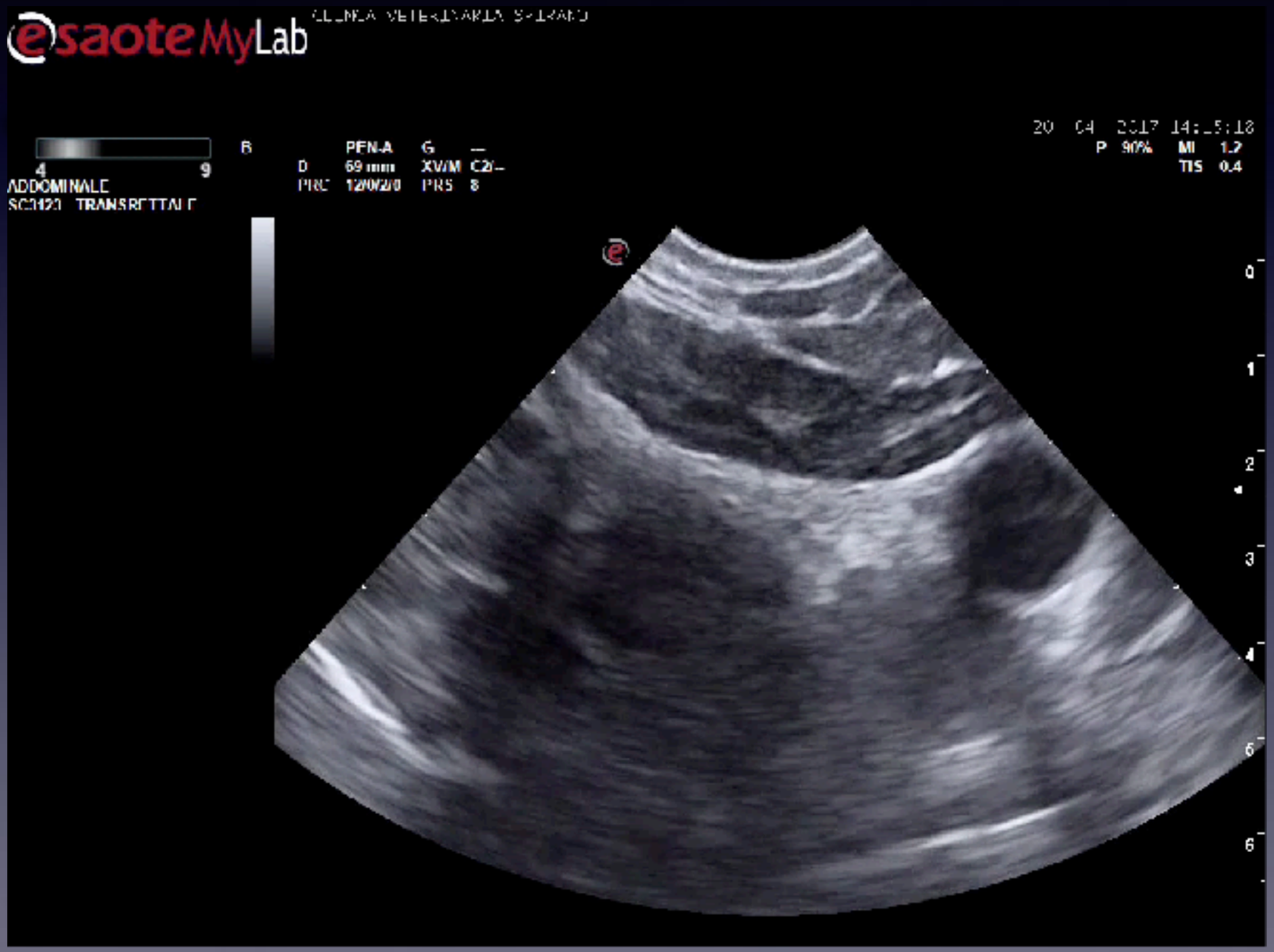
Caso clinico n° 13

Tasca gutturale dx.

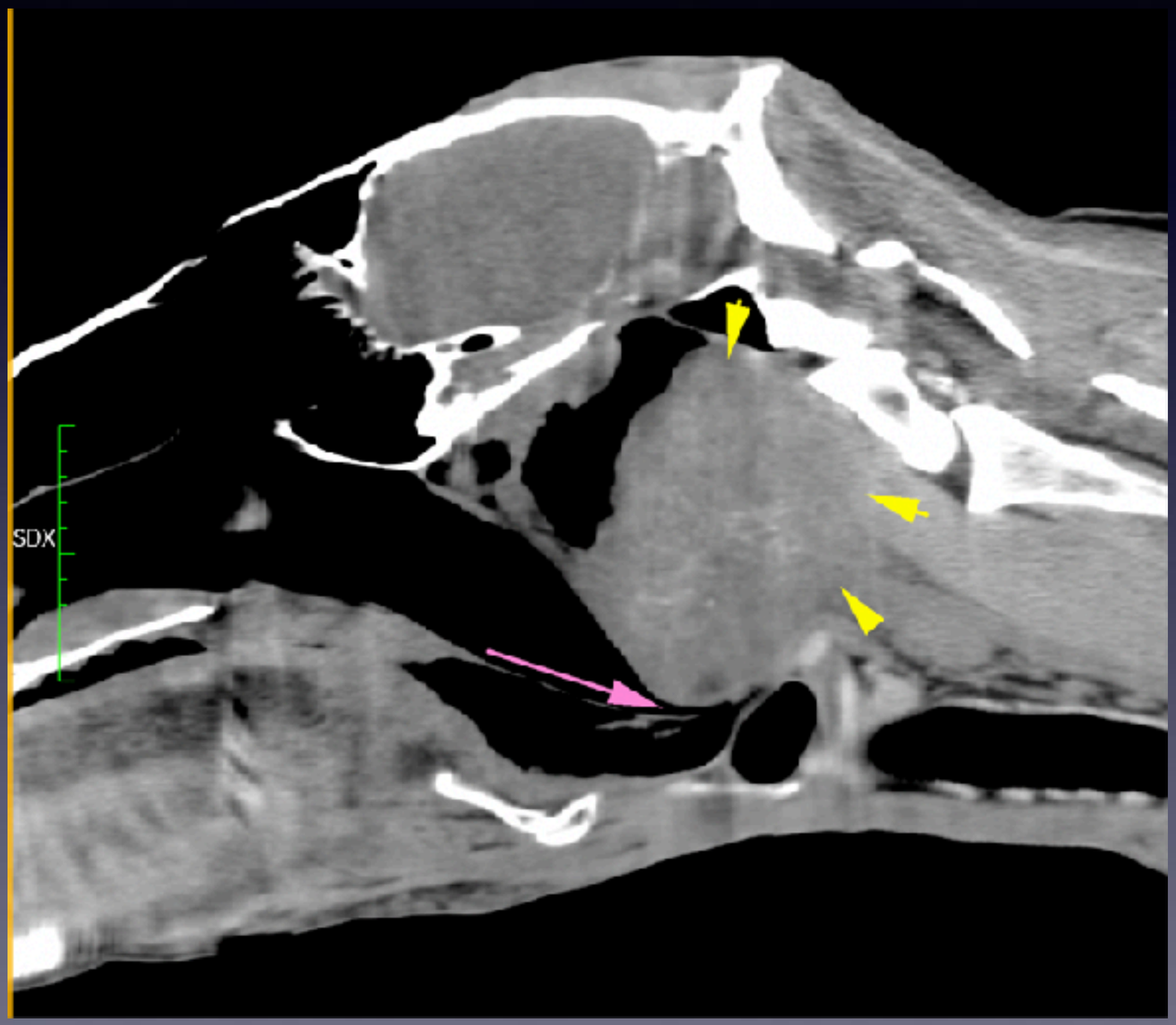
Tasca gutturale sn.

Caso clinico n° 13

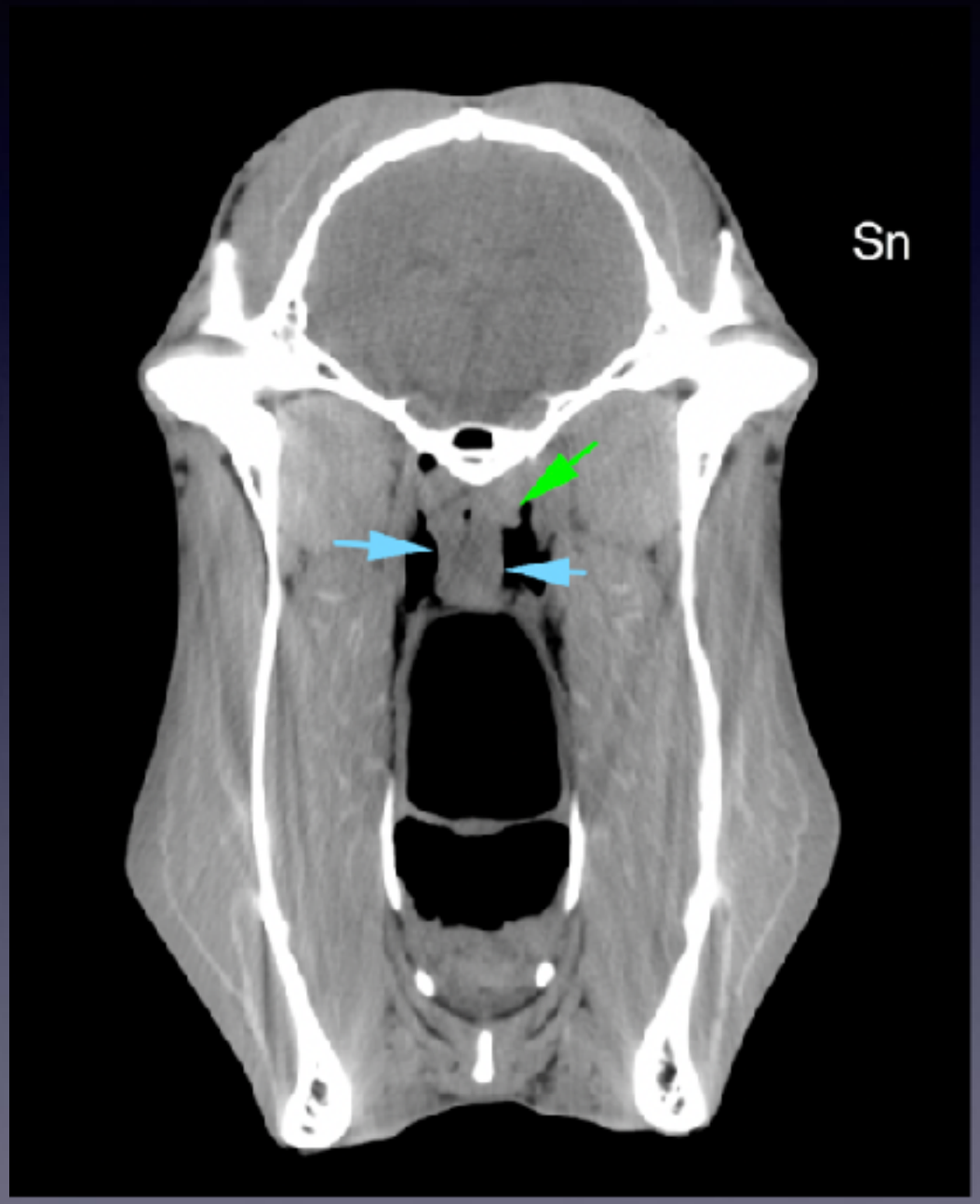
Caso clinico n° 13



Caso clinico n° 13



Caso clinico n° 13



Caso clinico n° 13

REPERTI:

- **presenza di un espanso (frecce gialle) a densità eterogenea, caratterizzato da lieve ipodensità e concomitante presenza di aree iperdense, e da forma globosa con profilo bozzellato (diametri max 14 x 14 x 8 cm). L'espanso presenta margini definiti e si estende tra parete laterale sinistra del rinofaringe e stiloideo di sinistra, portandosi caudalmente sino a livello di dente dell'epistrofeo, prevalentemente nel contesto della tasca gutturale di sinistra. La neoformazione determina significativa compressione della volta faringea in corrispondenza dello sfintere esofageo superiore (che non appare riconoscibile) a cui si associa dislocazione destrorsa del tratto esofageo prossimale, compressione dorso-laterale sinistra del nasofaringe, della laringe (frecce rosa), dislocazione destrorsa del setto delle tasche gutturali e del muscolo retto del capo di sinistra;**
- **si osserva un marcato ispessimento a carico dell'aspetto dorso-mediale della parete della tuba uditiva di sinistra, verosimilmente in corrispondenza della tonsilla rinofaringea sinistra (freccia verde) e delle pareti del recesso faringeo (frecce azzurre), che appare notevolmente ridotto di volume;**
- **dislocazione del palato molle sull'epiglottide;**
- **aumento di volume in assenza di evidenti alterazioni della densità a carico dei linfonodi retrofaringei mediali e laterali di sinistra.**

. **COMMENTO**

L'esame tomografico ha evidenziato la presenza di una neoformazione di verosimile natura neoplastica che occupa buona parte della tasca gutturale di sinistra esercitando significativo "effetto massa" sulle strutture laringee e faringee. Si associa marcata tumefazione della tonsilla rinofaringea sinistra e delle pareti del recesso faringeo, riconducibili in prima ipotesi a fenomeni reattivo-infiammatori.

Caso clinico n° 13

Diagnosi citologica:

Il quadro citologico è compatibile con una neoplasia maligna non differenziata. La presenza concomitante di gruppi cellulari di probabile origine epiteliale suggerisce una medesima origine delle cellule più marcatamente atipiche (carcinoma). Considerato il basso grado di differenzianzione, si suggeriscono ulteriori indagini istopatologiche per meglio caratterizzare il processo neoplastico.

Caso clinico n° 13

TERAPIA

- Flunixin meglumine: 1,1 mg/kg ev sid
- Prednisolone: 1 mg/kg per os sid

PROGNOSI

- Infausta, il cavallo è stato soppresso dopo un paio di mesi circa

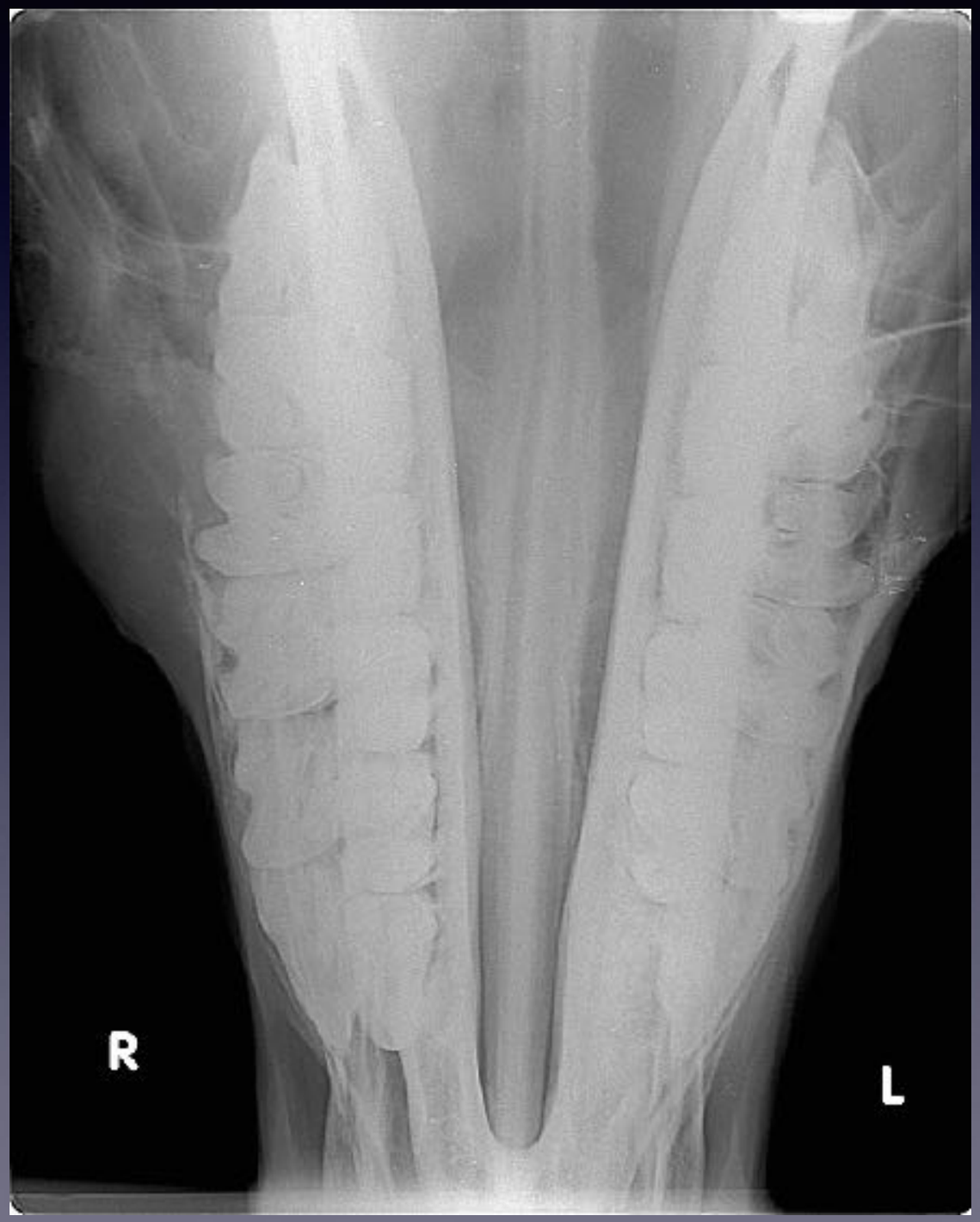
Caso clinico n° 14

- Castrone 20 anni, Sella,
- Tumefazione reg. zigomatica dx. da qualche settimana, con leggera epistassi ipsilaterale
- FC:44, FR:14, Mucose: Rosa, Sensorio: Vigile, Temp: 37,7°,

Caso clinico n° 14

Caso clinico n° 14

Caso clinico n° 14



Caso clinico n° 14

ESAME ISTOLOGICO

Tessuto osseo e tessuto fibroso con una proliferazione di cellule neoplastiche epiteliali, che si estende ai margini delle sezioni esaminate, organizzate in isole e cordoni. Le cellule neoplastiche sono poligonali con una quantità da scarsa ad abbondante di citoplasma eosinofilo e nuclei ovali con 0-1 nucleoli evidenti. Le cellule presentano differenziazione squamosa. Anisocitosi e anisocariosi sono da lievi a moderate. Le mitosi
sono meno di 1 per campo a forte ingrandimento.

DIAGNOSI *Carcinoma squamoso*

COMMENTO
Si tratta di una neoplasia epiteliale maligna.

Caso clinico n° 14

TERAPIA

- Flunixin meglumine: 1,1 mg/kg ev sid
- Prednisolone: 1 mg/kg per os sid

PROGNOSI

- Infausta, il cavallo è stato soppresso dopo un mese circa

Bibliografia

Epistaxis in the horse

S. Z. Barakzai* and P. M. Dixon

EQUINE VETERINARY EDUCATION 207 *Equine vet. Educ.* (2004) **16** (4) 207-217

Spontaneous bilateral epistaxis associated with the administration of phenylbutazone in a horse

A. MUÑOZ[1,2]*, P. TRIGO[2], C. RIBER[1,2], F. CASTEJÓN[2]

Revue Méd. Vét., 2011, **162**, 8-9, 421-424

GRAZIE A TUTTI PER L'ATTENZIONE!

dott. Marco Salvadori
Lp Milano

dott. Massimo Magri
Clinica Veterinaria Spirano

Giornate Boehringher Ingelheim 2018