



Common Emergencies in Small Ruminants

Misty A. Edmondson, DVM, MS, DACT

Agenda



1. Obstructive Urolithiasis
2. Anemia due to Internal Parasitism
3. Pregnancy Toxemia
4. Dystocia



Obstructive Urolithiasis



Obstructive Urolithiasis: Clinical Presentation

- Often present as a “sick goat” – disease of males
- Restlessness, vocalizations, tail switching, colic
- Dribbling urine or anuria
- Urethral pulsations without urination (male)
- +/- urethral swelling at site of obstruction
- Crystals/blood on preputial hairs
- Preputial/rectal prolapse
- Signs of pain – stretching out bruxism, ↑ HR & RR; pain may subside if rupture



Figure 1: Dr. Dwight Wolfe, Auburn University

Obstructive Urolithiasis: Diagnosis



- **Most common & useful diagnostic → Ultrasound examination**

- Also examine kidneys for nephroliths, hydronephrosis, hydroureter

- **Clinical Pathology**

- ↑ creatinine, PCV
- Hyponatremia, hypochloremia, +/- hyperkalemia
- BUN normal until late (ruminants recycle urea)
- Possible evidence of inflammation

Obstructive Urolithiasis: Management/Treatment



- **TRUE EMERGENCY!**

- Avoid chronic distension of bladder, urethral rupture
- Feedlot animal → immediate harvest
- Pet/Breeding Animal → do NOT delay surgery
- Urethral rupture → prognosis for long-term survival declines
- Cystocentesis?

- Fluid support

- Administer during surgery; must have outlet if surgery delayed
- Isotonic sodium chloride
- Especially important if hyperkalemic

- **Non-Surgical Treatment**

- Urethral catheterization - be cautious, urethra friable
- Walpol's solution
 - pH 4.5
 - U/S-guided cystocentesis, 30-50 mL Walpol's lavage & leave
 - Urine flow in 24-36 hours, normal voiding 3-5 days

● **Surgical Treatment**

- Amputation of urethral process – high recurrence rate
- Salvage (Feedlot Animal) – temporary due to stricture formation (weeks to months)
 - Urethrostomy at site of obstruction
 - Penile amputation
- Breeding or Pet Animal
 - Tube cystotomy
 - Bladder marsupialization



Figure 2: Dr. Dwight Wolfe, Auburn University

Obstructive Urolithiasis: Prevention



- Females as pets
- Delay castration as long as possible (at least until puberty)
- Increase water intake
- Avoid excess protein (grain supplements, legume hay) in pets
- Ca:P ration of 1.5-2:1
- Urinary acidifiers - monitor pH



Anemia Due to Internal Parasitism

- *Haemonchus contortus* most significant as far as...

- Clinical disease
- Resistance to anthelmintics
- Greatest risk to kids & periparturient females

- Clinical signs:

- Unthrifty, rough hair coat, pale mucous membranes, submandibular edema,
- No diarrhea to pasty/watery feces
- Sudden death

Anemia & Internal Parasitism: Resistance



- Resistance to one drug in a class usually means resistance to all drugs in that class
- Resistance to multiple drug classes & all classes
- Most important factor affecting rate of development of resistance
 - Untreated portion of worms, REFUGIA, provides a pool of genes that are sensitive to dewormers
 - Dilutes out the frequency of resistant genes

- **Fecal Flotation**

- Qualitative (1+ to 4+)
- Okay but not information on resistance

- **Modified McMaster's Fecal Egg Count Reduction Test**

- Quantitative (EPG)
- 2 tests performed 10-14 days apart
- No resistance = 95-99% reduction

- **DrenchRite® Larval Development Assay**

1. What parasites are in the herd/flock?
2. Which drugs are effective on the farm?



Figure 3: <https://www.wormx.info/drenchrитеassay>

- DrenchRite® Larval Development Assay
- Combination Treatment
 - Strong evidence that using **combination treatment** is best method for using anthelmintics
 - 1. Additive effect with each drug used → efficacy of treatment increases with each additional drug given; and
 - 2. Higher efficacy means fewer resistant parasites that survive treatment → greater dilution of resistant parasites by susceptible portion of the population

Anemia & Internal Parasitism: Management/Treatment



Table 1: Impact of using dewormers in combination on the efficacy of treatments.
 The increases in efficacy are due to a simple additive effect as per the equation below:
 Where D1 = efficacy of dewormer 1, D2 = efficacy of dewormer 2, D3 = efficacy of dewormer 3, C2 = efficacy of D1+D2, and C3 = efficacy of D1+D2+D3
 $C2\% = D1\% + (100-D1\%)*D2\%$
 $C3\% = C2\% + (100-C2\%)*D3\%$

Drug 1 (%)	Drug 2 (%)	Drug 3 (%)	Combination (%)
80	80		96
80	80	80	99.2
90	90		99
90	90	90	99.9
60	95		98
60	60	95	99.2
99	99		99.99
60	60	60	93.6
50	50	50	87.5
40	40	40	78.4

Table 2: Impact of combinations on percent of resistant worms that survive.
 Table shows the % of worms killed by a single dewormer vs a combination treatment with two dewormers both with the same efficacy, ranging from 80% to 99%. The last column shows the magnitude of the difference between % of worms killed and % surviving when one or two dewormers in combination are used. Note that the higher the efficacy of the drugs, the smaller the difference in efficacy when used in combination, but the greater the difference in the % of resistant survivors.

Efficacy of Dewormer		Single Dewormer	2 Dewormers in Combination	Fold Difference
99	% Killed	99	99.99	1.01x
	% Surviving	1	0.01	100x
98	% Killed	98	99.96	1.02x
	% Surviving	2	0.04	50x
95	% Killed	95	99.75	1.05x
	% Surviving	5	0.25	20x
90	% Killed	90	99	1.1x
	% Surviving	10	1	10x
80	% Killed	80	96	1.2x
	% Surviving	20	4	5x

Figure 5: <https://www.wormx.info/combinations>; Ray Kaplan, Combination Dewormers: The Time is Now, January 2017.

Anemia & Internal Parasitism: Prevention



- Pasture Management
 - Fallow period (3-6 months)
 - Use **browse areas** as much as possible
 - Maintain forage height > 2 in
- FAMACHA[®]



Pregnancy Toxemia



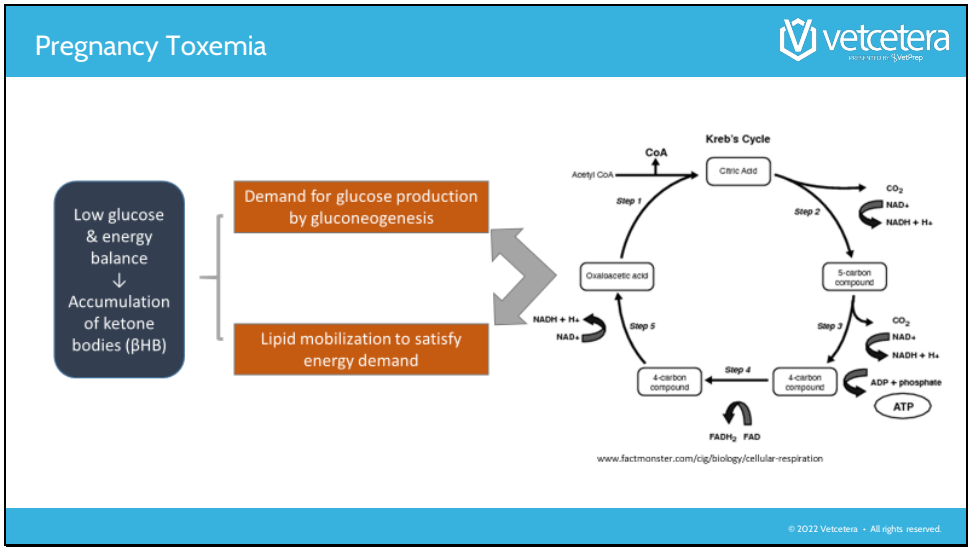
Pregnancy Toxemia




- Metabolic disease of pregnant ruminants
- Cause:
 - Abnormal metabolism of CHO and fats in late pregnancy (last 2-6 weeks)
- Predisposition:
 - Does carrying 2 or more fetuses
 - Lean ($BCS < 2$) or obese ($BCS \geq 4$)

Pathophysiology of disease

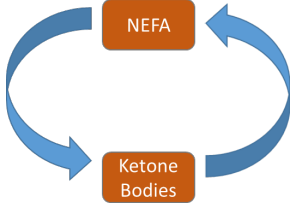
- Rapid fetal development in late gestation causes rapid mobilization of fat stores to assure adequate energy
- Liver also increases gluconeogenesis to facilitate glucose availability to the fetus(es).
- Negative energy balance → increased mobilization may overwhelm capacity of liver → hepatic lipidosis
- At same time, ketone bodies produced and accumulated → leads to excessive ketone bodies in blood which increases susceptibility to pregnancy toxemia



Pregnancy Toxemia 

Vicious Cycle

- Hypoglycemia
- Lipolysis
- Increased NEFAs
- Ketone formation
 - Ketones further suppress gluconeogenesis
 - Ketone bodies suppress appetite
- Resulting FFA mobilization suppresses liver function



© 2022 Vetcetera. All rights reserved.

Pregnancy Toxemia: Clinical Presentation



- **May be vague initially**
 - Weakness, lethargy
 - May separate from herd
 - Mild ataxia



Figure 6: Dr. Thomas Passler, Auburn University

Pregnancy Toxemia: Clinical Presentation

vetcetera
International Veterinary Peer-Review Journal


Veterinary World
Open access and peer reviewed journal

ISSN 0959-2688 2016 Aug; 9(8): 869-874.
Published online 2016 Aug 18. doi: 10.14202/vetworld.2016.869-874

Studies on clinical signs and biochemical alteration in pregnancy toxemic goats

Prasankumar R. Vasava¹, B. G. Jani¹, H. V. Goswami², S. D. Rathna³ and F. B. Tandel⁴


- Anorexia
- Depression/lethargy
- Neurologic signs
 - Opisthotonos
 - Dropped head
 - Periodic convulsion
 - Apparent blindness
- Bloat
- Bruxism
- Frothy Salivation
- Sweet, fruity breath




- Recumbency
- Coma

Figure 7: Vasava PR, Jani RG, Goswami HV, et al. Studies on clinical signs and biochemical alteration in pregnancy toxemic goats. Vet World, 2016 Aug; 9(8): 869-874.

© 2022 Vetcetera. All rights reserved.



Pregnancy Toxemia: Clinical Presentation

 **Veterinary World**
Open access and peer reviewed journal

JAN 2018 2018 Aug 9(8): 869-874
Published online 2016 Aug 18 doi: 10.14202/vetworld.2016.869-874
PMCID: PMC5021937

Studies on clinical signs and biochemical alteration in pregnancy toxemic goats

Prasenjit Kumar R. Vasava¹, R. G. Jani¹, H. V. Goswami², S. D. Rathna³ and F. B. Tandel¹

<u>Clinical Signs:</u>	<u>Pathophysiology of CS:</u>
Anorexia	→ Ketone bodies
Depression/lethargy	→ Low glucose
Neurologic signs	→ Cerebral hypoglycemia
Bloat	→ Hypocalcemia
Bruxism	→ Cerebral hypoglycemia
Frothy salivation	→ Frothy salivation
Sweet, fruity odor to breath	→ Ketone bodies
Recumbency/Coma	→ Low energy, hypocalcemia

Figure 8: Vasava PR, Jani RG, Goswami HV, et al. Studies on clinical signs and biochemical alteration in pregnancy toxemic goats. *Vet World*. 2016 Aug; 9(8): 869-874. © 2022 Vetcetera. All rights reserved.

Pregnancy Toxemia: Clinical Presentation



Figure 9: Dr. Thomas Passler, Auburn University

Pregnancy Toxemia: Diagnosis



- Dehydration
- Stress leukogram
- Metabolic acidosis
- Increased ketone bodies in urine
- Blood ketones



Figure 10: www.abbottdiabetescare.com

Pregnancy Toxemia: Diagnosis – Clinical Pathology



● Decreased

- Glucose
- 20-40 mg/dL
- Calcium

● Increased

- SGPT
- SGOT
- BUN
- Creatnine
- BHBA
- >3 mmol/L (x10.3 = 30.9 mg/dL)
- NEFA



ISSN 2020-2016 Aug; 9(8): 869-874
Published online 23 Aug 2020
DOI: 10.14202/vetworld.2016.869-874
PMCID: PMC6021637

Studies on clinical signs and biochemical alteration in pregnancy toxemic goats
Prasannkumar R. Visava,¹ R. G. Jani,¹ H. V. Goswami,² S. D. Rathva,³ and F. B. Tandel¹

Figure 11: Vasava PR, Jani RG, Goswami HV, et al. Studies on clinical signs and biochemical alteration in pregnancy toxemic goats. Vet World, 2016 Aug; 9(8): 869-874.

Pregnancy Toxemia: Management & Treatment



- Efficacy of treatment depends on...
 - Timely & correct diagnosis
 - Early treatment

- Fair prognosis early, poor once recumbent

- Treatment based on...

1. Eliminate negative energy source
 - Good quality hay & grain
 - Starch ferments to propionate in rumen
 - Propionate "Gluco-genic" VFA
 - Oral propylene glycol: 2-3 oz PO BID
 - 3-carbon energy source
 - Glucose precursor
 - Parasite issues, etc.?

- Treatment based on...

1. Eliminate negative energy source
 - Good quality hay & grain
 - Starch ferments to propionate in rumen
 - Propionate "Gluco-genic" VFA
 - Oral propylene glycol: 2-3 oz PO BID
 - 3-carbon energy source
 - Glucose precursor
 - Parasite issues, etc.?

Pregnancy Toxemia: Management & Treatment



● Treatment based on...

1. Eliminate negative energy source
 - Good quality hay & grain
 - Starch ferments to propionate in rumen
 - Propionate "Glucogenic" VFA
 - Oral propylene glycol: 2-3 oz PO BID
 - 3-carbon energy source
 - Glucose precursor
 - Parasite issues, etc.?
2. Removal of factors that affect energy requirements (3 Options)
 - 1) Prolonged medical support of dam
 - 2) Induction of parturition
 - 3) Immediate Cesarean section

Pregnancy Toxemia: Management & Treatment



Prolonged Medical Support

- Possible to save dam and fetuses
- Poor prognosis for both dam and kids
- Provide dextrose, fluids, calcium
 - IV dextrose infusions
 - 5-7 g q 3-4 hours, preferably CRI
 - 5% dextrose, 10-20 mEq/L of potassium, 25 mL of calcium borogluconate/L
 - Insulin +/-
 - Blocks hormone sensitive lipase
 - Dose to effect, monitor glucose
 - Approximately 4X/day or CRI

Pregnancy Toxemia: Management & Treatment



Induction of Parturition

- 10 mg of Lutalyse® in goats
- 15 mg dexamethasone in sheep
- Dam may not survive until parturition (36-48 hours)
- Fetuses may be premature

Pregnancy Toxemia: Management & Treatment



Immediate C-Section

- Best prognosis for dam
- Most common reported reason for C-section in small ruminants
- Recovery of dam often dramatic (within 24-48 hours)
 - Warn owners & be prepared to euthanize kids if premature

Pregnancy Toxemia: Prevention



- Increase nutritional plane in last 6 weeks of pregnancy
- Groups dams according to...
 - Number of fetuses (ultrasound)
 - Age
 - Body condition
- Prevent stress during pregnancy

Breeding Management

- High fecundity associated with increased incidence
- Breed at appropriate age – often delayed in show does
- Show does are over-conditioned
- Cull older does
 - Increased incidence with age/parity
 - Most first parity cases are show/pets → over-conditioned



Dystocia

Dystocia: Clinical Presentation



- **Dystocia**

- When does a normal delivery become a dystocia?
 - Doe in active labor \geq 30 minutes & no progress
 - Abrupt cessation of parturition
 - Delivery of placenta without kid/lamb
- Most common cause = ***fetal postural abnormalities***
- Other causes...
 - Incomplete cervical dilation
 - Simultaneous presentation of lambs or kids
 - Cervicovaginal prolapse
 - Uterine inertia
 - Fetal-maternal disproportion - singleton birth, over-weight ewe or doe

Dystocia: Diagnosis



- **Diagnosis**
 - Based on history & physical examination

Dystocia: Management/Treatment



- **Equipment**

- Soap & water
- Small hands
- Lubricant
- Head snare
- Sleeves/gloves
 - most causes of abortion are zoonotic!

Dystocia: Management/Treatment



- “Race to the Finish”

- More than one fetus attempting to exit the vagina at same time
- Figure out which legs belong to which fetus & untangle
- ID 1st kid, repel 2nd kid, deliver 1st kid

- Head Malposition

- **NEVER** deliver if the head is retained
 - may cause rupture of the uterus!
- Push fetus back to gain room to pull head around

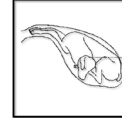
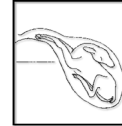


Figure 13: <https://goats.extension.org/goat-reproduction-parturition-kidding/>

● Front Leg Malposition

- One or both front limbs retained
- Some does may kid normally with one leg retained
- If both, only head may be protruding from vulva
 - Swollen tongue
- Elbow lock
 - Elbows are locked in flexion against pelvis
 - Repel fetus & digitally straighten one leg at a time

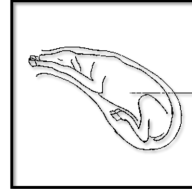
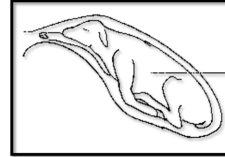


Figure 14: <https://goats.extension.org/goat-reproduction-parturition-kidding/>

Dystocia: Management/Treatment



● Breech Presentation

- Caudal longitudinal presentation, dorsosacral position, both rear limbs retained beneath fetus
- Requires straightening both rear limbs
- Push fetus cranially & to one side
- Requires pulling & rotating the hock while the foot is pulled ventrally & medially
- Careful with fetal hooves

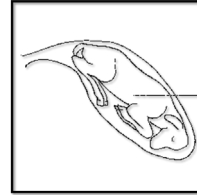


Figure 16: <https://goats.extension.org/goat-reproduction-parturition-kidding/>



Figure 15: http://www.ansci.wisc.edu/fjp1/ansci_repro/lab/lab13_09/calving/abnormal_positions.html

- Ringwomb

- Failure of cervix to dilate completely
- Relatively common (~23%)
- Active labor with no kids delivered
- Manual dilation unrewarding - may tear cervix

- Predisposing factors
 - Hypocalcemia, hormone imbalances, mineral imbalance, prolonged dystocia

- Treatment = Cesarean Section

- Fetal-Maternal Disproportion

- Fetus is too large or pelvis is too small of delivery through pelvis or vaginal canal

Doe:

- Over-conditioned doe/ewe
 - Too much fat in pelvis
- Small framed doe/ewe
 - Pelvis too small
- Injuries to pelvis

Kid:

- Singleton births - Large kid/lamb
- Deformed kid/lamb

- **Uterine Inertia**

- Inability of uterus to contract
 - May be cause or result of dystocia
- Primary uterine inertia
 - May be due to hypocalcemia, hormone imbalance
- Secondary uterine inertia
 - Due to fatigue
 - May be caused by ringwomb, fetal malposition

Dystocia: Management/Treatment



- **Cesarean Section**
 - May be required in prolonged labor
 - Cervix starts to close within 2-3 hours
 - Relatively simple surgical procedure
 - Often best option for both doe and kids

- **Fetotomy**
 - Dead fetus
 - Removing extremities to relieve dystocia
 - Head, forelimb, rearlimb

Dystocia: Management/Treatment



● Aftercare for Doe/Ewe

- Carefully check for additional fetuses
- +/- antibiotics
- +/- tetanus antitoxin
- Anti-inflammatories
- Good nutrition
- Check for sufficient colostrum/milk

● Aftercare for Kids/Lambs

- Remove fetal membranes
- Manual stimulation
- +/- oxygen
 - Meconium staining
- Keep warm
- Dip navel - 7% iodine
- COLOSTRUM!!!

Dystocia: Prevention



- Good breeding management
- Good nutrition - not too fat, not too thin
- Careful observation
- Client education - what's normal, when to call DVM



Thank you for choosing Vetcetera!

Misty A. Edmondson, DVM, MS, DACT
