A clinician's guide to what kills adult sheep and goats, as diagnosed by necropsy

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Abstract

Techniques for efficient field necropsy of adult sheep and goat are described. Common conditions that kill adults, as well as older lambs and kids, are then presented, with emphasis on diagnosis by gross examination of the body. Additional laboratory tests that may be warranted and management considerations for the remainder of the herd are included to guide the practitioner in addressing the problems that have been identified. Conditions leading to the death of adult sheep include the following: anemia from haemonchosis, copper toxicosis, malnutrition, heavy strongyle burden, dental disease, paratuberculosis, liver flukes, caseous lymphadenitis, bacterial pneumonia, retroviral pneumonia, endocarditis, grain overload, enterotoxemia, intestinal accidents, toxic mastitis, dystocia with uterine rupture or retained fetuses, pregnancy toxemia, hypocalcemia, urolithiasis, polioencephalomalcia, listeriosis, and other neurologic diseases.

Résumé

Les techniques de champ efficace de l'autopsie de moutons et de chèvres adultes sont décrites. Conditions communes qui tuent les adultes, ainsi que les agneaux plus âgés et les enfants, sont ensuite présentés, en mettant l'accent sur le diagnostic par l'examen de l'organisme. D'autres tests de laboratoire qui peuvent être justifiées et considérations de gestion pour le reste du troupeau sont inclus à guider le praticien dans le traitement des problèmes qui ont été identifiés. Conditions conduisant à la mort de la brebis adulte sont les suivantes : anémie de haemonchosis, le cuivre de la toxicose, malnutrition, lourd fardeau strongyle, les maladies dentaires, paratuberculose, des douves du foie, lymphadénite caséeuse, pneumonie bactérienne, la pneumonie, l'antirétroviral, endocardite, surcharge grain entérotoxémie, accidents intestinaux, la mammite toxique, une dystocie avec rupture utérine ou conservé des fœtus, la grossesse la toxémie, hypocalcémie, urolitiasis, polioencephalomalcia, la listériose, et d'autres maladies neurologiques.

Introduction

Much useful information about the individual dead animal and health issues in the herd can be gleaned by performing a necropsy. However, autolysis occurs rapidly and makes interpretation of lesions difficult. The herd veterinarian should ideally examine the body within two (for gastrointestinal diseases) to four hours after death if timely submission to a diagnostic laboratory is not possible. This paper describes a protocol for performing a necropsy and typical lesions produced by common fatal conditions of small ruminants. Documentation with digital pictures of the necropsy findings will assist the veterinarian if consultation with a pathologist is required to make or confirm a diagnosis.

Necropsy Techniques

An excellent necropsy manual by King et al. describes a systematic but efficient technique for field necropsies.[12] Additionally, several journal articles[6,7] and many textbooks of small ruminant diseases[1,8,9,13,14,16] include information on performing a necropsy or list and illustrate numerous causes of sudden death. Videos of various necropsy techniques are also available on the internet. These include a sheep necropsy video targeting the feedlot lamb[10] and a complete cattle necropsy, also applicable for sheep and goats.[3] A protocol for collecting digital images of small ruminant necropsies is also available on line.[15] Images of suspected lesions can be shared with a veterinary pathologist or compared with an online databank of veterinary pathology lesions.[11]

Necropsies are best performed in a warm, well-lit area, on a surface that can be easily sterilized or a plastic sheet that can be disposed of afterwards. Disposable gloves are imperative.

Supplies such as sample containers, formalin, culture swabs, markers, and paper or a recorder for taking notes should be organized before the first body is opened. A digital camera to record lesions is desirable, especially if an assistant with clean hands is available to take the pictures. A sharp necropsy knife is ideal but a box cutter or even several size 22 scalpel blades can substitute for the knife, while a hatchet or tree branch pruner is used to cut ribs and a saw or hatchet can be used to open the skull.

Start by weighing the animal if possible and recording all ear tags and tattoos, sex, age, estimated weight, body condition score, and any grossly visible abnormalities as well as the history and clinical signs reported. Examine the conjunctiva for pallor or icterus and the eye for evidence of keratitis from exposure or infectious pinkeye. Examine the feet, palpate all joints and external lymph nodes, and examine the perineum for evidence of diarrhea, discharges, or prolapses. Check the incisor teeth to verify reported age.

The sheep or goat should be placed on its left side and the two right limbs should be reflected out of the way after cutting the skin and muscles over the axilla and inguinal area and the ligament to the head of the femur. The two openings in the skin are then connected and the remaining skin is peeled back off the side of the animal. The skin incision is also extended up the neck to the intermandibular space. Slice into both sides of the udder or scrotum and examine the contents. Next the abdominal cavity is entered through an incision just caudal to the costal arch. The abdominal wall is reflected and the diaphragm is severed along its attachment to the ribs. A branch pruner or (if the animal is immature) a necropsy knife is then used to cut each rib at the costochondral junction. The ribs can then be reflected dorsally by breaking them near the backbone or cutting them with the branch pruner. The thoracic and abdominal cavities are now exposed and individual organs can be examined. Open the pericardial sac and examine its contents before removing the pluck (tongue, larynx, trachea, esophagus, lungs and heart) from the body. The lungs are examined visually and by palpation. Open the esophagus and trachea longitudinally and follow larger airways into the parenchyma of the lung. The four chambers of the heart and the valves are examined, following the flow of blood.(King necropsy) Return to the abdomen, noting presence of fluid and omental fat. Both kidneys should be located and incised and the bladder and its contents examined. Locate and examine the uterus in females. The liver should also be incised, looking for abscesses and flukes. The gastrointestinal tract is opened last, with special attention being paid to rumen contents (feed material present, consistency, pH, presence of poisonous plants) and abomasal contents (*Haemonchus* worms, foreign bodies). Consider retaining rumen contents, a fecal sample for parasitology, liver and potentially kidney for trace mineral analysis, and samples from all lesions identified, for laboratory testing.

Several joints should be examined routinely during the necropsy. These include the right hip and shoulder and both stifle joints. Finally, the head can be disarticulated from the spine using a ventral approach to examine the atlanto-occipital joint and the skin is removed from the top of the skull in preparation for brain removal. Using a saw or hatchet and starting at the dorsolateral aspects of the foramen magnum, the skull cap is reflected to permit inspection of the brain. If the animal has horns, remove them flush with the skull before attempting to access the brain. Wear a face shield and double gloves in regions where rabies is endemic.

After the necropsy has been completed, the carcass should be disposed of safely and properly, according to state laws. Composting, deep burial and landfill disposal are typical choices to prevent contact with people, pets, other livestock, or scavengers. Equipment and surfaces should be sanitized and contaminated gloves disposed of properly, with due consideration for the risks of zoonotic diseases.

Haemonchosis

Blood loss to abomasal parasites should always be suspected in the animal with pale mucosa and muscles. In some cases thousands of 'barberpole' worms can be visualized in the abomasum to confirm this diagnosis or a quantitative fecal reveals thousands of strongyle eggs per gram. In other animals the worms are no longer present because of recent anthelmintic administration or because the animal was so anemic that the *Haemonchus* deserted the abomasum. The practitioner should check the FAMACHA score[2] of other animals on the farm, take additional fecal samples for quantitative analysis, and review previous treatments, nutrition and pasture management. A comprehensive parasite control program can then be developed, following the guidelines of the American Consortium for Small Ruminant Parasite Control.[2]

Copper Toxicosis

The body fat of sheep and goats should be white, including in the axilla where staining from gastrointestinal organs is not a concern. If icterus is identified, the most likely cause is copper toxicosis, although other liver diseases or even leptospirosis occasionally cause icterus. The liver may be discolored, the kidney may be a dark gray color, and any urine retained in the bladder may be coffee or port wine colored. Confirmation will require copper analysis of the liver, and of the kidney as well if liver copper concentrations are not elevated. Before leaving the farm obtain a thorough feeding history including tags from salts and concentrates fed and any possible access to poultry, swine, cattle, or horse feeds. Also determine if copper oxide wire particles have been administered for parasite control. Although sheep are more susceptible to copper toxicosis, goats can also be poisoned. The occasional animal dies of copper-induce liver failure without displaying icterus. Routine histology of animals with undiagnosed death and trace mineral testing, including copper, for flock monitoring purposes will aid in reaching a diagnosis.

The Emaciated Animal

Malnutrition in small ruminants can be the result of many diseases and management problems. Common deficiencies leading to a low body condition score include protein, energy, cobalt or copper. Abundant omental fat may remain in an emaciated animal, so always palpate the loin area before commencing the necropsy to determine the body condition score. With severe cachexia the fat in the marrow of the long bones and in the coronary groove is replaced by gelatinous tissue referred to as serous atrophy of fat.

A simple agroceriosis may occur with the feeding of low quality, late cut hay or inadequate quantities of feedstuffs.

Crowding at the feed bunk or commingling of animals of different sizes or horn status may cause starvation of some while others are in adequate body condition. This is especially true with goats, where the social order is strong.

Parasitism, especially with gastrointestinal strongyles, is a common cause of emaciation and hypoproteinemia, visible antemortem as edema below the chin, termed 'bottle jaw'. When *Haemonchus* is not the primary parasite involved, egg counts in the fecal sample harvested from the rectum or cecum may be modest. Poor quality feed and anthelmintic resistance will contribute to parasitism as a cause of death. In particular, adequate dietary selenium, vitamin E, and copper are needed for a proper immune response to parasites.

Dental disease will lead to emaciation of individual older animals, such as pets, but even young adults can suffer from periodontal disease and be unable to properly chew roughage. A 'gummer' who has lost incisors will have reduced ability to graze but can function well on harvested feeds. Often a palpable thickening of a mandible heralds a tooth root abscess. The cheeks should be slit at necropsy of any thin animal to permit a thorough examination of the molar teeth. Sharp points are normal, but missing or abscessed teeth will hinder mastication. When older animals are retained in a herd, owners should be instructed to evaluate body condition score frequently and provide pelleted roughages as needed.

Enzootic nasal tumor is a retroviral disease that can lead to emaciation as breathing becomes progressively more difficult for the sheep (or less commonly goat) with tumor occluding its nasal passages. There may be a history of stertor, nasal discharge, or bulging of an eye. Use the saw to open the nasal passages transversely if this condition is suspected.

Paratuberculosis or Johne's disease is common in sheep and goats over one year of age but less commonly diagnosed, as diarrhea occurs in only a small proportion of animals that die of the condition. Animals that die of paratuberculosis lack body fat and are usually moderately anemic. They frequently have an increased parasite load. Intermandibular edema and tricavitary effusion secondary to hypoproteinemia may be evident at necropsy. Sometimes dilated lymphatics are visible on the serosa of the ileum, and mesenteric lymph nodes may be mineralized. Testing will be required to confirm the diagnosis, with histology of the ileum, ileocecal junction, and adjacent mesenteric node ideal. An acid fast stained smear of the intestinal mucosa in one of these locations and a fecal culture or PCR for antigen are other options.

Liver flukes, especially *Fasciola hepatica* and *Fascioloides magna*, will lead to emaciation, anemia, and hypoproteinemia. Presence of the flukes and their black migratory tracts makes the initial diagnosis simple, but flukes may need to be submitted to a parasitologist to confirm the species identity, which is needed to determine drug dosages to use. This is because *Fascioloides* does not reach the bile duct (or establish patency), so higher doses of flukicides are needed. Wet areas of the pasture should be fenced off and an eight way clostridial vaccine administered to protect against sudden death from black disease, caused by the toxins of *Clostridium novyi*.

Abscesses from **caseous lymphadenitis** (*Corynebacterium pseudotuberculosis*) may be found in the lungs, liver or kidney and adjacent lymph nodes and in these locations they commonly cause emaciation. Involvement of just peripheral lymph nodes, by contrast, has little effect on the overall health of the infected sheep or goat. The pus in the abscess may be creamy or layered, and culture will be required to confirm the diagnosis so that a herd control program of culling and/or vaccination can be instituted. Differentials include *Trueperella pyogenes*, tuberculosis, meliodosis in other parts of the world, and necrotic neoplastic lesions. Lymphosarcoma is not rare in goats and can infiltrate lymph nodes or internal organs;[4] an impression smear of a lesion will allow differentiation from caseous lymphadenitis. Older animals that have reached 'tumor age' can have any imaginable neoplasm as a cause of emaciation.

Pneumonia

Bacterial pneumonia is a frequent cause of acute death or chronic debilitation in sheep and goats. *Mannheimia haemolytica* is more commonly involved than *Pasteurella multocida*. In some herds *Mycoplasma* species are an important contributing cause, but special culture techniques will be required to confirm their presence. These pneumonias are located in the cranioventral portions of the lung, especially on the right side where a separate bronchus serves the right apical lobe. The affected lung is firm on palpation and commonly has fibrin on the surface. Culture will confirm the organisms involved but as there are no appropriate vaccines against the strains affecting small ruminants, management will need to focus on improving nutrition and ventilation and, possibly, drenching techniques if the pneumonia began as an inhalation.

Retroviral pneumonias (ovine progressive pneumonia in sheep, caprine arthritis-encephalitis in goats) sometimes cause locally extensive areas of palpably firm pneumonia in animals over one year of age. These animals are usually thin and have a history of dyspnea and exercise intolerance. A bacterial pneumonia may also be present, as such an infection will upregulate/activate the virus. Confirmation will require histology. Positive serology is proof of a herd problem but does not prove that the virus was involved in the death of the animal.

Endocarditis may lead to embolic pneumonia or to congestion of the liver and fluid accumulation in the abdomen due to heart failure. Lesions will be evident on the heart valves. White muscle disease of the heart can also present as acute heart failure, often with well delineated white streaks in the myocardium of the pulmonary outflow tract or other parts of the heart or skeletal muscles.[12] The diet should be evaluated for selenium and vitamin E adequacy.

Gastrointestinal causes of acute death

Enterotoxemia due to the toxins of *Clostridium perfringens* type D can cause rapid death in inadequately vaccinated small ruminants allowed access to rapidly digested carbohydrates in the form of concentrates or lush pasture. A gross diagnosis is difficult to make, and even a laboratory diagnosis is usually only tentative, as the organism is a normal inhabitant of the intestinal tract and proliferates rapidly after death, producing toxin. Presence of fluid and a fibrin clot in the pericardial sac is strongly suggestive of enterotoxemia, in the absence of evidence of a bacterial pneumonia. The classic 'pulpy kidney' lesion of enterotoxemia is very subjective and varies with the postmortem interval. The practitioner can confidently advise a vaccination program (two doses of commercial vaccine 3 or 4 weeks apart, boosters prelambing, boosters at 6 month intervals if heavy feeding) in unprotected animals, even if enterotoxemia is not proven to be the cause of death of the animal necropsied. Just be aware that there is a 21 day meat withdrawal for the vaccine.

Animals that are properly vaccinated against enterotoxemia can still die of grain overload, or **lactic acidosis**. Sudden access to excessive quantities of grain (especially if finely ground, as in chicken or hog feed) will lead to the production of increased quantities of propionic and butyric acid in the rumen. High concentrate diets require less cud chewing, and less saliva reaches the rumen to buffer it. As the pH of the rumen contents drops, normal flora that digest roughages die off and are replaced by acid loving streptococci and lactobacilli and lactic acid is produced. The pH then drops further until it goes below 5.5 and the normal buffering system is overwhelmed. At necropsy these animals will have sunken eyes and a splashy rumen. Rumen contents often contain a lot of visible grain and are milky. In addition to checking pH, a gram stain of the rumen fluid can be done to demonstrate a preponderance of Gram positive cocci and bacilli. Control measures for the rest of the herd may include better locks on the grain storage area, smaller concentrate meals, less finely ground grain products, and providing high quality grass hay before concentrates are fed. Some farms believe that offering sodium bicarbonate free choice also helps to prevent acidosis.

Sheep and goats are occasionally afflicted by **intestinal accidents** such as intussusception, mesenteric torsion, or obstruction of the rumen, abomasum, or spiral colon with an ingested foreign body. These problems can be identified by opening the ruminoreticulum and abomasum and by carefully following the full length of the intestinal tract.

Acute liver fluke disease occurs in regions where *Fasciola hepatic* infests small ruminants. The grazing animal that has consumed large numbers of metacercaria may develop a rapidly fatal fibrinous peritonitis accompanied by liver necrosis and hemorrhage. Migrating flukes can also cause sudden death by creating anaerobic conditions in the liver that allow *Clostridium novyi* spores to germinate, leading to production of a fatal toxin in unvaccinated animals.

Poisonous plants occasionally lead to sudden death, and a careful history may indicate that the animal had access to yew (*Taxus*), members of the rhododendron family, or cyanide containing plants, amongst others. The rumen contents should be searched for plant fragments and the environment for toxic plants.

Mastitis

Toxic mastitis in small ruminant is most commonly caused by infection with *Staphylococcus aureus*, but *Mannheimia* from the pharynx of nursing lambs or kids or other organisms spread by the milking machine are also possible. The affected udder half will be swollen and firm at necropsy, often with gangrene of the skin of the udder and ventral abdomen or the presence of abscesses or discolored fluid in the udder parenchyma. A postmortem culture will confirm the causative bacterium, but the teat ends should be examined for evidence of trauma or infection with the contagious ecthyma (sore mouth) virus that might have predisposed to the entry of pathogens into the udder. The milking system and procedures should also be evaluated in dairy herds.

Uterine Causes of Acute Death

Dystocia can occur if a fetus is malpresented - breech, head back, transverse - or if multiple fetuses enter the birth canal at the same time. The head back position is a frequent cause of death either because of unskilled manipulation by an owner or because the dam can strain hard enough against the fetus to rupture its own uterus. Fetuses with arthrogryposis also lead to dystocia, and Cache Valley virus is one potential cause of these malformations. Fetal fluids need to be tested for antibodies to confirm the diagnosis. A retained fetus, for whatever reason, decomposes rapidly, killing the dam via toxemia.

Other less frequent causes of death related to parturition include the metabolic disease discussed below, uterine torsion, and rupture of the middle uterine artery.

Metabolic Diseases

Pregnancy toxemia should be suspected if a late pregnant or periparturient female dies with an almost empty rumen and a fatty liver. Generally two or more fetuses will have been present. Any urine present in the bladder may be tested for ketones, but it is more reliable to obtain ocular fluid (aqueous humor) to test for betahydroxybutyrate. The results are still valid 24 hours after death, and values greater than 2.5 mmol/L support a diagnosis of pregnancy toxemia.[5] Unless the individual animal had a specific problem that resulted in decreased feed consumption in late pregnancy, such as lameness or poor teeth, the diet of the remainder of the herd needs to be evaluated and corrected.

Hypocalcemia can rapidly kill females that are late pregnant or lactating heavily. There will probably be few or no gross lesions, but a suspicion based on the history or a lack of other diagnosis can be confirmed by testing the aqueous humor for calcium, with a value less than 1.0 mmol/L being diagnostic.[5]

Tetany **(hypomagnesemia)** is another metabolic disease that does not result in postmortem lesions but can be diagnosed with aqueous humor. In this instance, a magnesium concentration less than 0.33 mmol/L is diagnostic.[5] The history may include recent transport or access to lush pastures high in potassium.

Urinary Tract Diseases

Urolithiasis is a very, very common cause of death of intact and castrated male sheep and goats of all ages. Necropsy may reveal evidence of obstruction, including a distended, hemorrhagic bladder, hydronephrosis, urine in the abdomen from rupture of leakage through the distended bladder wall, or subcutaneous urine where the urethra has ruptured. The urethral process and the sigmoid flexure should be examined closely, as these are common sites for obstruction. Stones or precipitate collected at necropsy can be sent for free analysis at the University of Minnesota Urolith Center.[18] Gold colored round BB shaped stones do not need to be sent for analysis as they are invariably calcium carbonate. Dietary management for the remainder of males in the herd will include interventions such as increasing the consumption of water by adding salt to the hay or grain, avoiding the feeding of excessive minerals, and very commonly by not feeding grain to pet wethers or allowing them access to chicken feed.

Pyelonephritis, often the result of an ascending infection from the urachus or bladder, results in swollen kidneys with pus accumulated at the hilus. Toxins such as oxalates (from various poisonous plants or ethylene glycol) and copper can cause nephrosis in small ruminants. A whitish line of precipitate near the corticomedullary junction may be visible in the case of oxalates, and the kidneys are often dark when affected with hemoglobinuric nephrosis from copper toxicosis. Additionally, lambs that die of hyperthermia have been reported to have swollen, pale, moist kidneys in which severe tubular necrosis is visible histologically.[17]

Neurologic Diseases

The two most common neurologic causes of acute death in small ruminants are polioencephalomalacia (cerebrocortical necrosis) and listeriosis. Scrapie can also cause either neurologic signs or emaciation in sheep or goats. These conditions will be difficult or impossible to diagnose without laboratory support, but gross examination of the brain (with due care to avoid potential exposure to rabies) may reveal trauma or presence of a brain abscess.

A history of blindness or convulsions or of an indigestion accompanied by diarrhea is compatible with **polioencephalomalacia**. There may be coning of the cerebellum because intracranial pressure has forced the caudal part of the cerebellum against the foramen magnum. The cerebral cortex may appear yellowish and these areas often fluoresce under an ultraviolet light. The diet should be investigated to identify deficiencies of fiber or excesses of sulfur, possible predisposing causes of polioencephalomalacia.

Listeriosis is best diagnosed by histology and immunohistochemistry of the brain stem, but historical clues of facial nerve paralysis or circling or gross evidence of exposure keratitis would be suggestive. Sometimes the rumen contents are rather watery because the animal was inappetent in the days preceding death, but some sheep and goats die within 12 hours of first being noted ill. Exposure keratitis may suggest a facial nerve paralysis, a common sign of listeriosis.

Conclusions

A gross field necropsy will often lead to the proper diagnosis of cause of death in small ruminants. The practitioner who performs the necropsy on the farm is ideally placed to observe the remainder of the herd for signs of illness and the environment and diet for possible deficiencies. Additional animals can be tested if indicated. Then, with the aid of confirmatory tests from a diagnostic laboratory and consultation with pathologists, diagnosticians and standard textbooks, a plan can be formulated to control the problem in the remainder of the herd.

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