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REVIEW ARTICLE

Sudden death syndrome in domestic ruminants: a review

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Abstract

This review provides a comprehensive exploration of factors contributing to sudden death (SD) in ruminants, focusing on cattle, sheep, and goats. The definition of unexpected death varies based on the farming system. In intensive systems, constant surveillance enables rapid detection of diseases, while in extensive systems, irregular checks may result in finding animals dead after a prolonged illness. SD syndrome definitions vary; some involve clear clinical signs, while others consider acute death in apparently healthy animals. Various infectious causes of SD are discussed, including enterotoxemia, hemorrhagic bowel syndrome, caudal vena cava thrombosis, and respiratory diseases. The review also covers nutritional disorders like acidosis, hypomagnesemia, and vitamin deficiencies. Poisoning, both from inappropriate drug use and toxic plants, is examined, as well as accidents and trauma, parasitism, stress, and miscellaneous causes such as aortic aneurysm rupture, congenital defects, and snakebites. Differential diagnosis is emphasized, and the importance of post-mortem examinations in understanding the cause of unexpected deaths is highlighted. The paper offers a detailed overview of the complex factors contributing to SD in ruminants, emphasizing the need for thorough investigations and necropsy examinations to identify the specific cause in each case.

Keywords Sudden death · Ruminants · Infectious factors · Nutritional disorders · Trauma · Poisoning · Parasitism

Introduction

The definition of unexpected death in ruminants varies depending on the individual approach and farming system type. In intensive industrial systems (such as dairy herds and feedlots), animals are under constant surveillance, which enables rapid detection of even peracute diseases. However, in extensive sedentary and transhumance systems, routine herd controls are irregular and infrequent, resulting in animals being found dead after a long period of illness, which may involve sub-acute or even chronic diseases that culminate in the perception of SD. The definition of SD syndrome remains ambiguous among authors, certain involve cases of animals that showed more or less obvious clinical signs before death (Lincoln 2012; Plumlee 2003) while others only consider an acute death of an animal apparently in good health (Anscombe 2016; Kelly 2003).

Studies of ruminant pathology suggest that the most common causes of SD are acute infectious diseases, digestive disorders, and nutritional deficiencies, but a number of cases of SD have been attributed to fatal poisonings, accidents, and certain parasitic agents.

This review aims to explore various factors that may contribute to SD in cattle, sheep, and goats. The paper highlights diseases that can manifest with clinical symptoms and their associated signs and etiopathogenesis. Additionally, particular attention is paid to the role of necropsy diagnosis in identifying the cause of SD in ruminants.

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Causes of sudden death in ruminants

Infectious etiology

Enterotoxemia

The term "enterotoxemia" was initially used to describe diseases caused by certain strains of *Clostridium perfringens* (Simpson et al. 2018). Enterotoxemia can be acute or peracute, and is a well-recognized cause of SD in what was otherwise considered to be very healthy animals (Lebrun et al. 2010). Recent investigations have reported that *C. perfringens* toxinotypes (A, B, C, D, and E) produce lethal toxins that can cause SD in ruminants (Fahimeh et al. 2018; Redondo et al. 2013; Simpson et al. 2018). Clostridia pathogen strains induce syndromes that can be classified as enteric, neurotoxic, and histotoxic (Santos et al. 2019). However, animals can die from enterotoxemia due to a single disorder or a combination of several syndromes (Simpson et al. 2018; Uzal et al. 2014). Toxemia is induced by the systemic spread of toxins in blood and to tissues, leading to intravascular hemolysis and capillary damage, as well as hepatic necrosis and cardiac effects (Lebrun et al. 2010; Uzal and Songer 2008). Furthermore, several other strains of the histotoxic *Clostridium* genus are involved in SD syndrome in cattle and sheep (Abbott 2018). Infections with histotoxic clostridia occur when wounds are contaminated with spores or vegetative forms; however, toxemia occurs when the toxins enter the bloodstream, resulting in shock and death (Junior et al. 2020). SD with severe hemorrhagic jejunitis and hemorrhagic intestinal contents observed at postmortem inspection is suggestive of clostridial enterotoxemia. Additionally, rapid putrefaction of the abdominal viscera with gas and putrid odor is characteristic of clostridial enterotoxemia (Lebrun et al. 2010). However, clinical, epidemiological, necropsy, and histological findings are essential for the accurate diagnosis of clostridial SD (Santos et al. 2019).

Hemorrhagic bowel syndrome (HBS), also known as jejunal hematoma syndrome, primarily occurs in adult cattle, with most clinical cases reported in dairy cows (Abutarbush and Radostits 2005; Kalender et al. 2007). HBS is an acute enteric disease that often occurs within the first 3 to 4 months of the lactation period (Underwood et al. 2015). The underlying causes are not well-known, but the most commonly discussed potential cause is infection with *C. perfringens* type A (Adaska et al. 2014; Simpson et al. 2018). Thus, it has been proposed that linked alpha and beta2 toxins may have a role in the occurrence of the disease; further, the implication of certain mycotoxins in the appearance in HBS has been reported (Mamak and Börkü 2019). Healthy cows affected by HBS can be found dead without prodromal signs. During necropsies, notable lesions include intestinal hemorrhage and ulceration (Kalender et al. 2007).

Caudal vena cava thrombosis (CVCT)

The most frequent cause of vena cava thrombosis in cattle is liver abscess rupture (Schild et al. 2017). Abscesses form in the liver due to the dissemination of bacteria from the rumen secondary to inflammation in the ruminal wall (Motta et al. 2016). Bacteria from the abscess can travel to the vena cava, causing septic thrombosis, and from there, septic emboli dislodge and are carried to and lodge in the pulmonary capillaries (Miller and Gal 2017). As a result, acute death occurs after a septic shock with evident hemoptysis after episodes of acute pulmonary hemorrhage (Motta et al. 2016). As well as a large septic thrombus may cause enough direct pulmonary infarction or a septic myocarditis leading to heart failure (Peek and Divers 2018). The most frequent pathogens isolated from liver abscesses are *Fusobacterium necrophorum* and *Trueperella* (*Arcanobacterium*) *Pyogenes pyogenes* (Motta et al. 2016; Nagaraja and Lechtenberg 2007).

Though, emboli may come from inflammatory processes such as deep digital sepsis, mastitis, or metritis and result in CVCT (Braun 2008; Schild et al. 2017); also, this process has been reported in acute rupture of reticuloperitonitis abscess (Gerspach et al. 2011). The diagnosis of CVCT requires careful necropsy when SD results and, in general, affected animals have appeared completely healthy before death (Peek and Divers 2018).

Infectious respiratory diseases

Glock and Degroot (1998) estimated that respiratory disease accounted for 47% of SDs in ruminants, highlighting its importance. Shipping fever complex, a type of acute pneumonia, is the most prevalent cause of SD (Glock and DeGroot 1998; Peek and Divers 2018). *Pasteurella multocida* and *Mannheimia haemolytica* are two common pathogens associated with pneumonia and septicemia in all ruminants, causing pasteurellosis; a bacterial disease characterized by bronchopneumonia, septicemia, and SD (Underwood et al. 2015). *Mannheimia haemolytica*, formerly known as *Pasteurella haemolytica* A1, is particularly associated with SD in sheep (Gilmour 1980) and can also cause septicemia in young lambs and kids (Brogden et al. 1998). Moreover, high mortality rates have been reported in cattle with acute pneumonia caused by *Bibersteinia trehalosi* (Cortese et al. 2012).

Histophilus spp. has also been associated with respiratory disease in sheep and goats, with death being typically the first sign of *Histophilus somni* pneumonia. Death can result

from septicemia and thromboembolic meningoencephalitis (Underwood et al. 2015). Additionally, the acute form of bovine respiratory syncytial virus (BRSV) infection has been reported as responsible for SD in severe cases (Scott et al. 2011). Bovine herpesvirus 5 (BoHV-5) is frequently associated with neurological disease and SD in young calves within 18 h of infection course (Lunardi et al. 2009). In goat herds, *Mycoplasma mycoides* subspecies *capri* or *Mycoplasma mycoides* subspecies *mycoides* has been found to cause high mortality rates (Debien et al. 2013).

Other infectious etiologies

Bacillus anthracis is the causative agent of anthrax or splenic fever. Sudden unexplained death is most commonly seen in grazing herbivore in contaminated vegetation or soil by *Bacillus anthracis* spores (Rao et al. 2019). Affected animals have nasal, oral, and anal or vulva hemorrhage (dark blood) and a few cases are ataxic in terminal septicemia (Abbott 2018; Blowey and Weaver 2011; Fasanella et al. 2010). Heartwater (cowdriosis) is one the most important tick-borne diseases of domestic ruminants in southern Africa, and it is caused by *Cowdria ruminantium* (Van de Pypekamp and Prozesky 1987). Fulminating fever in an apparently healthy animal is the main symptom, followed by paroxysmal convulsions, respiratory difficulties, and rapid collapse; however, it is often fatal and both goat and sheep may die suddenly (Van de Pypekamp and Prozesky 1987; Yunker 1996). Coliforms seem to be responsible for anaphylactic shock and SD (Andrews et al. 2008; Owens et al. 1998). Coliform septicemia is a disease that affects cattle, goats, and lambs and can cause SD in lambs under the age of 10 days (Pohl et al. 1993). Peracute toxic coliform mastitis, which occurs during the peripartum period or early lactation, can occasionally cause cow death, and many deaths related to endotoxic mastitis are likely due to disseminated intravascular coagulation (DIC) (Stalberger and Kersting 1988). In newborns, toxemia associated with particular pathogens, especially *E. coli*, are important causes of SD (Radostits et al. 2006). *Listeria monocytogenes* is the most common cause of bacterial infection affecting the central nervous system. Goats appear to be more sensitive to this infection than cattle or sheep, and neonatal listeriosis can lead to SD in 2- to 4-day-old calves (Blowey and Weaver 2011; Debien et al. 2013).

Foot-and-mouth disease (FMD), caused by an RNA *Picornavirus* of the genus *Aphthovirus*, is a highly contagious disease that is difficult to diagnose in sheep due to its subacute form. In very young animals, the virus can infect the myocardium, leading to SD (Ryan et al. 2008).

Malignant catarrhal fever is a severe disease caused by bovine herpesvirus 6 (BoHV-6) that mostly affects cattle and can lead to SD (Stanitznig and Wittek 2018; Underwood et al. 2015).

Nutritionals disorders

Acute and peracute acidosis

Acute and peracute acidosis have been associated with high-concentrate feeding regimes and production diseases such as SD in feedlot steers (Andersen 2003). SD usually occurs near the end of finishing without any signs of disease (Wilson et al. 1975). According to a study by Malafaia et al. (2016), a high level of lactate (9.13 mmol/L blood) in fattening cattle is considered dangerous as it can cause systemic metabolic acidosis (Malafaia et al. 2016). Endotoxemia is also considered an important factor in follow-on systemic metabolic acidosis leading to SD (Andersen 2003).

Red gut

Red gut is a fatal intestinal accident that can rapidly lead to death in weaned lambs and adult sheep, this condition may mostly occur in lush pastures (Abbott 2018); usually, lucerne (Barrell et al. 1989); white clover and ryegrass (Gumbrell 1997).

High intake of digestible forage leads to a reduction in rumenoreticulum and an increase in intestinal mass, which becomes mobile. Accidental twisting of the intestines occurs as a result of this mobility, leading to obstruction of the mesenteric blood vessels, causing death from shock (Barrell et al. 1989; Gumbrell 1997). Autopsy reveals an intense reddened or dark red intestinal mass, often displaced in the anterior abdominal cavity (Gumbrell 1997).

Hypomagnesemia

Hypomagnesemia is one of the differential diagnoses that should be considered in SD in cattle and sheep (Doncel et al. 2021). It is a biochemical disorder characterized by low serum magnesium (< 0.7 mmol/L) (Chevalier et al. 2012). The risk of hypomagnesemia increases when the diet is low in Mg and high in K, primarily in fast-growing lush grasses (Doncel et al. 2019). Clinical signs may be absent, and abrupt high mortality rates on pasture may be observed (Doncel et al. 2019). Like many metabolic disorders, hypomagnesemia can cause seizures responsible for SD in calves (Chevalier et al. 2012) and a certain diagnosis is only possible after biochemical investigation (Doncel et al. 2021).

Vitamin E and selenium deficits

Selenium deficiency, also known as white muscle disease, is often associated with a lack of vitamin E and can cause

paresis and SD, particularly in neonatal ruminants (Underwood et al. 2015). Calves are often born dead or die shortly after birth due to a congenital deficiency form (Davis and Myburgh 2016). Abbott (2018) stated that selenium deficiency can cause cardiac myopathy syndrome, which is liable to SD (Abbott 2018).

Vitamin B1 deficiency

Vitamin B1 deficiency can cause polioencephalomalacia (PEM) in ruminants; the disease can be peracute and lead to death (Jean-Blain and de Oliveira 1994). Animals are at risk when their diet contains thiaminases or when their feed or water is high in sulfates (Underwood et al. 2015). Major clinical signs of the disease in young dairy calves include incoordination, convulsions, and head retraction (Karapinar et al. 2010).

Poisoning

Toxic plants

While there are only a few poisonous plants that cause SD without predictive signs (King 1983), several plants can produce poisoning severe enough to cause death within 12 h (Burrows and Tyrl 1989). Certain cardiotoxic poisonous plants have been associated with the consumption of many species of the genera *Palicourea, Arrabidaea* and *Amorimia, Pseudocalymma*, *Elegans, Nerium Oleander* (Lima et al. 2019; Soares et al. 2011), and Gossypol (Plumlee 2003). Nitrate/nitrite poisoning, followed by gossypol, oleander, and pyrrolizidine alkaloids, accounted for most plant-associated poisonings (Varga and Puschner 2012). Poisonous plant consumption is one of the most common causes of SD in African livestock, according to Anaeto et al. (2009), with cyanogenic plants and yew being the most rapidly lethal (Anaeto et al. 2009; Burrows and Tyrl 1989). Traditional medicine can also lead to inappropriate dosing of herbal medicines, such as *C. occidentalis* (Nalule et al. 2011). Sorghums are among the most dangerous crops, as cyanide can be released during chewing and in the rumen or abomasum, leading to rapid intoxication (Burrows and Tyrl 1989). The entire oleander plant, whether fresh or dried, is poisonous, and just five to ten leaves can be fatal to adult cattle (Ceci et al. 2020) within 2–8 h (Varga and Puschner 2012). Gossypol intoxication causes myocardial necrosis in young ruminants. The cardiotoxic effects are gradual destruction of the heart muscle and interference with the conduction system (Plumlee 2003). However, it should be noted that poisoning by plants rich in cyanogenic glycosides can cause disorders similar to certain fatal nervous diseases (KAzI TAnI 2014), which can make diagnosis problematic. Ingestion of large amounts of oxalis causes severe hypocalcemia, central nervous system depression, and death from cardiovascular collapse (Mohammedi et al. 2014).

Monofluoroacetate (MFA) is a deadly toxic component of some plants and causes SD in livestock; of these, *Amorimia pubiflora*, which contains high concentration of AMF and cause fatal poisoning at all vegetative stages (Lima et al. 2019).

Nitrate poisoning is a serious problem only in ruminants. Plants that accumulate nitrates, such as sorghum, can cause SD (Burrows and Tyrl 1989). Similarly, cabbage plants and fertilizers are potential sources of nitrates (Scott et al. 2011). White sweet clover and yellow sweet clover produce the hemorrhagic syndrome (Burrows and Tyrl 1989). Nitrite is responsible for tissue anoxia by converting hemoglobin to methemoglobin (Scott et al. 2011; Varga and Puschner 2012). Further, non-protein nitrogen (NPN) (e.g., urea) induces ammonia toxicosis, with a rapid onset of clinical signs and SD (Peek and Divers 2018). The peracute death of lambs from ammonia poisoning may erroneously suggest clostridial enterotoxemia (Plumlee 2003).

Taxus baccata (European yew) causes SD from acute cardiac arrest and diagnosis is based on the detection of alkaloids in the liver, urine, or blood (Cortinovis and Caloni 2015).

Conium maculatum (hemlock) is one of the most toxic members of the plant kingdom and Hemlock alkaloids cause central nervous system depression in livestock (Vetter 2004).

Agricultural chemicals

Chemicals such as insecticides, herbicides, and rodenticides can also be toxic to animals (Delano et al. 2002). According to Caloni et al. (2018), pesticides were the leading cause of poisoning in Europe from 2010 to 2016 in most species (Caloni et al. 2018). Among ruminants, cattle are the species most frequently exposed to poisoning due to their undemanding feeding behavior (Cowan and Blakley 2016).

Metalloids

Sulfur poisoning in ruminants can cause polioencephalomalacia (PEM), which is characterized by brain damage (De Sant'Ana and Barros 2010). PEM is primarily caused by a thiamine deficiency, as mentioned earlier. Acute arsenic toxicosis occurs when livestock have access to old and waste materials containing arsenic, often in combination with chromium or lead and the primary clinical outcome is SD (Bertin et al. 2013).

Cattle are more susceptible to lead poisoning than other ruminant species (Caloni et al. 2018). Neurological dysfunction and respiratory failure can lead to death within 24 h (Cowan and Blakley 2016). When the clinical signs

are overlooked lead poisoning can result in SD and cattle may be found down or dead-on pasture (Bates and Payne 2017). Excessive exposure to copper can lead to high mortality, with death occurring within 12 to 72 h. Acute clinical signs of copper overdose include diarrhea and central nervous system disorders, shortness of breath, and depression (Varga and Puschner 2012). In rare cases, water intoxication has been reported in sheep, leading to acute deaths resulting from the consumption of large quantities of water after several days of deprivation (Abd-Elrahman et al. 2020).

Accident and trauma

Mortality due to trauma and accidents can be high in calves and young stock in beef cattle (Mõtus et al. 2017). Generally, sheep handling is safe; however in certain practices, such as shearing, accidental wounding resulting in lethal outcomes has been reported (Irandoust et al. 2013). A rupture of the small intestine can lead to rapid deterioration and often manifests itself in SD, similar situation is observed in newborn calves trampled by adult animals. Alike, a rectal laceration can result in SD from an abrupt rectal touch; a tear in the thickness of the rectal mucosa can result in fatal septic peritonitis within 24 h (Peek and Divers 2018). During investigations, veterinarians should not exclude the possibility of criminal intent, especially following suspicious death cases (Archibald and Smith 1965).

Bloat

Bloat is a common cause of SD in cattle due to physical obstruction by a foreign object in the alimentary body or esophagus. Gas is trapped in the rumen, causing acute free-gas bloat. Bloat occurs as well in pastured beef cattle and feedlot cattle, due to the production of a large amount of stable foam (Galyean and Rivera 2003; Radostits et al. 2006). When the bloat becomes severe enough, death is likely caused by suffocation when the distended rumen pushes against the diaphragm and stops breathing (Majak et al. 2003).

Traumatic reticuloperitonitis

Hardware disease is primarily a disease of cattle and is rarely seen in smaller ruminants (Underwood et al. 2015). SD can occur due to heart failure (Buczinski et al. 2010) or by fatal hemorrhage when metal wire migrates and punctures the heart coronary vessels or other large vessels like the reticular vein (Radostits et al. 2006).

Perforated abomasal ulcer

SD may occur in dairy cattle with perforated abomasal ulcer and death usually occurs in 48–96 h from shock and dehydration (Andrews et al. 2008; Radostits et al. 2006).

Electrical injuries

The accidental contact with electrical currents can cause instantaneous death in grazing animals. This is due to the disruption of neural regulatory impulses or heart failure (Schulze et al. 2016). Dead animals may be found with fresh food in their mouth and scorch marks of burned hair on their coat, especially the legs (Blowey and Weaver 2011).

Inhalation pneumonia

The major cause of pulmonary aspiration in cattle is the inhalation of foreign material into the larynx and then the lower airway. The aspiration of a significant amount of liquid or solid material can lead to instant death due to mechanical asphyxiation (Shakespeare 2012). Inhalation pneumonia may also result following hypocalcaemia, especially if the cow becomes cast, during general anesthesia, and after the faulty administration of drenches (Scott et al. 2011).

Parasitism

Haemonchus contortus is sometimes associated with SD in sheep, particularly in cases of hyperacute haemonchosis, which is due to a heavy *Haemonchus* species infection (20,000–30,000 worms) (Besier et al. 2016; Gebresilassie and Afera Tadele 2015). Clinical signs of acute disease are anemia, dark-colored feces, edema "bottle jaw," and weakness (Besier et al. 2016; Roeber et al. 2013). Acute visceral cysticercosis mainly affects lambs and goat kids and can cause SD without premonitory signs. It has been very sporadically cited in calves (Koutsoumpas et al. 2013). *Echinococcus* spp. is the causal agent of hydatid cysts, the rupturing of the cysts can result in pulmonary artery embolism and SD due to massive giant pulmonary artery embolism (Bayaroğullari et al. 2013). In young calves during the first month of their lives, a hyper infestation with *Strongyloides papillosus* worms can be fatal (Kváč and Vítovec 2007). *Dictyocaulus viviparus* is a lungworm that can be responsible for SD in severely affected growing cattle under severe challenge. In feedlot steers, death can occur suddenly in animals without exhibiting signs of clinical illness (Scott et al. 2011). Cysts containing *Fascioloides magna* found in the

liver and lung can liberate flukes that involve fatal pulmonary hemorrhage (Wobeser and Schumann 2014).

SDs caused by trypanosomiasis have been reported in small ruminants. An acute hemorrhagic syndrome has also been seen sporadically in cattle infected with some isolates of *T. vivax*, mostly in East Africa, the syndrome can be rapidly fatal (Spickler 2018). SD may also occur with *Babesia bovis* and *Babesia bigeminia* infection in cattle (Underwood et al. 2015).

Stress

In newborn calves, the threat of mortality increases following difficult parturition and adverse climatic conditions. In cold environments, hypothermia often occurs and may cause the death of weaker calves (Roland et al. 2016; Vermorel et al. 1983). According to Cox et al. (2016), heat stress is not only specific to young animals; significant cold- and heat-related increases in dairy cattle mortality have been observed. Farm animals, such as cattle, are known to suffer from temperature extremes (Cox et al. 2016). For some authors, high on-farm mortality is seen as a potential indicator of poor cow welfare (Sarjokari et al. 2018). Moreover, Vitali et al. (2015) established a link between cow age, temperature, and death (Vitali et al. 2015). Older dairy cows (5 to 8 years) are at high risk of death following exposure to long heat waves (> 11 days), likely with a high humidity index (Vitali et al. 2015).

SD is likely caused by severe stress during the transport of animals over long distances, in poor conditions or high stocking densities in pens, and a diet that is high in grain and low in fiber (Malafaia et al. 2016). Severe stress conditions can cause SD in lambs, even without diarrhea (King 1983).

Other causes of SD

The occurrence of aortic aneurysm rupture in goats, sheep, and cattle is sporadic and must be considered a significant cause of SD (Souto et al. 2017). Most animals are reported as being found dead, but those seen alive often show a sudden reduction in milk yield, with colic and recumbency preceding a rapid death (Crawshaw et al. 2011). Hereditary defects in fibrillin metabolism, known as Bovine Marfan syndrome, have been identified as potential causes of aneurysm formation in cattle (Potter and Besser 1994), especially in mature female of Holsteins breed (Lamm et al. 2007). Obstruction by hematoma of the aortic lumen or an aortic branch may result in acute myocardial infarction and SD (Amalinei and Căruntu 2013).

In newborns, congenital defects that are incompatible with life, prematurity, septicemia due to poor immune status, and hypothyroidism are important causes of SD (Radostits et al. 2006). Similarly, SD has been associated with frequent congenital heart disease in calves (Buczinski et al. 2010). In dairy calves, acute heart failure related to the anticipation of milk feeding has been reported and death may occur during feeding or immediately after, but the exact pathophysiology remains unknown (Jones 2014). In African livestock, snakebites are among the most commonly cited causes of SD. In species with only neurotoxic venom, the signs may be limited to puncture wounds and neurological deficits (Anaeto et al. 2009). Peracute death following snakebite is due to cardiovascular troubles leading to cardiac arrest (Plumlee 2003).

Differential diagnosis

SD in ruminants is a complex syndrome that can result from multiple factors interacting simultaneously, making diagnosis challenging. When causal factors are related to each other, it can pose a problem for both clinical and necropsy diagnosis, and the pathogenesis cannot be clearly defined (e.g., acidosis, acute pneumonia, and enterotoxemia). The diagnosis of SD in ruminants must involve two steps: first, investigation of the herd, including careful observation of the remaining animals for signs of acute clinical disease, which may provide key diagnostic information; second, individual post-mortem examination and autopsy of the deceased animal. The primary reasons for conducting post-mortem examinations on domestic animals are typically to determine the cause of unexplained individual animal deaths or sudden increases in herd mortality rates (Wäsle et al. 2017). Stockholders often expect a dramatic post-mortem result and clear instructions on how to prevent further losses after a tragic event such as SD (Kelly 2003). For veterinary investigators, a careful history and a prompt post-mortem examination can provide valuable diagnostic information in most cases of unexpected ruminant death (Pierson et al. 1976). If the animals really have died quickly, with no signs being observed, some assumptions about the necropsy findings may be made (Table 1). Most importantly, many of the pathological findings are likely to be unspectacular and nonspecific (Kelly 2003). In the case of infectious or toxic causes, the main objective of the necropsy examination is to collect and process samples for further examination (Wäsle et al. 2017).

Table 1 Postmortem findings and confirmation tests of principal causes of sudden death in ruminants

Cause	Species	Postmortem findings	Confirmation test
Infectious etiology			
Clostridium perfringens type C.	Cattle, sheep, goats	• Acute necrohemorrhagic enteritis particularly of the jejunum and ileum • The peritoneal cavity contains a small quantity of serous blood-stained fluid, and • Excess pericardial fluid and pulmonary interstitial edema • Petechial hemorrhages on serous membranes	ELISA (identification of beta toxin in intestinal contents) Gram stains, IHC Fecal cytology to determine spore counts and detect bacilli Polymerase chain reaction (PCR) Anaerobic bacterial culture
Clostridium perfringens type D. (pulpy kidney)	Sheep and goats	• Pulmonary congestion and edema • Serofibrinous pericardial effusion • Congested, soft, pulpy kidneys • Focal and bilaterally symmetric encephalomalacia most often affecting the basal ganglia, internal capsule, substantia nigra, and dorsolateral thalamus; less often within the white matter of frontal gyri	Identification of epsilon toxin in the blood or ileal contents
Bovine anthrax	Cattle	• Splenomegaly (very large soft spleen with "blackberry jam" consistency on the cut section that exudes thick black-red blood that brightens on exposure to air • Ulcerative hemorrhagic enteritis in the small intestine • Most severe lesions are over lymphoid tissue • Mesentery is infiltrated with gelatinous fluid from lymphangitis	Blood smear (precede autopsy) (tip of the tail, coronet) Old methylene blue (MacFadean's) stains capsule pink, bacteriologic culture from putrified exudates
Caudal vena cava thrombosis Perivascular hepatic abscesses	Cattle	• Thrombus fully occluding the lumen of the cranial vena cava • A sequel to rumenitis and hepatic abscessation • Thrombophlebitis • Valvular endocarditis is an occasional sequel • The abscess may erode the wall of the vena cava wall and rupture into the vena cava, causing massive septic embolization and sudden death	Post-mortem examination
Histophilus somni	Cattle, sheep, and goats	• Myocardial necrosis or infarction, myocardial abscesses may be present in some cases	CSF analysis: Increased protein neutrophils, Bacterial culture

Table 1 (continued)

Cause	Species	Postmortem findings	Confirmation test
BVD type II	Cattle in all age groups	• *Acute BVD*: In the mild form, mild erosions or shallow ulcerations of the muzzle and oral cavity; Peyer's patches outlined by coagulated blood and overlain by fibrin; fibrinohemorrhagic typhlocolitis • *Mucosal disease*: o Linear esophageal ulcerations o Erosions and ulcerations of mouth, tongue, oral and ruminal papillae, reticulum, omasum, abomasum, cecum/colon "Tiger stripe" colonic lesions o Peyer's patches swollen, necrohemorrhagic, ± diphtheritic membrane o Erosive-ulcerative interdigital dermatitis and coronitis	Virus isolation; immunofluorescent antibody assays; viral RNA detection using PCR; antigen detection using immunohistochemistry; serum virus neutralization; antigen capture ELISA Skin biopsies are often used for IHC antigen detection
Nutritionals disorders			
Acute and peracute acidosis	Cattle	• The presence of grains in the rumen and reticulum • Rumen filled with milky fluid with a sour odor and the mucosa of rumen and reticulum	Post-mortem examination, frothy content in the rumen
Hypomagnesemia	Cattle and sheep	• Multiple petechiae and ecchymoses are observed in the spleen capsule, epicardium, endocardium, subcutaneous tissue, and lung pleura	Mg concentrations < 1 mg/dL (0.4 mmol/L) from the CSF within 12 h of death or from the vitreous humor of the eye within 24–48 h after death are indicative of hypomagnesemic tetany
Vitamin E and selenium deficits	Cattle, sheep, and goats	• The cardiac form is often acutely fatal • Steatitis: o Yellow to yellow–brown subcutaneous and cavitary fat o Firm, lumpy, gritty fat o Subcutaneous edema o Fishy odor	Post-mortem examination
Vitamin B1 deficiency	Cattle, Sheep and goats	• Swollen cerebrum; flattened gyri; narrow sulci; prominent cerebral cortical necrosis with unaffected cerebellar cortex; cerebral cortical atrophy; rare tentorial herniation and coning of the cerebellum in severe cases • Yellow/brown discoloration of cerebrocortical gray matter; affected areas autofluoresce under ultraviolet light; possible due to ceroid-lipofuscin or mitochondrial ATP synthase • Hydrocephalus ex vacuo occurs in long-term cases	Post-mortem examination, elevated blood pyruvate; decreased erythrocytic transketolase activity

Table 1 (continued)

Cause	Species	Postmortem findings	Confirmation test
Accident and trauma			
Bloat	Cattle	• Congestion and hemorrhage of the lymph nodes of the head and neck, epicardium, and upper respiratory tract are marked • The lungs are compressed, and intrabronchial hemorrhage may be present • *Bloat line* demarcation; the cervical esophagus is congested and hemorrhagic, but the thoracic portion of the esophagus is pale and blanched • The rumen is distended, but the contents usually are much less frothy than before death • The liver is pale because of expulsion of blood from the organ	Post-mortem examination
Traumatic reticuloperitonitis	Cattle	• Focal suppurative or granulomatous inflammation in the wall of the reticulum, with or without minor overlying peritonitis • Perforation of one of the larger regional arteries • Penetration of the myocardium or rupture of a coronary artery. (sudden death)	Long, thin, and sharp foreign body, usually a wire or nail, penetrating the reticular wall
Perforated abomasal ulcer	Cattle	• Ulceration is most common in the fundic region in adult cattle and the pyloric antrum in milk-fed calves • The affected artery is usually visible after ingesta and necrotic tissue are removed from a bleeding ulcerated area • Most cases of perforation are walled off by the omentum, which forms a cavity 12–15 cm in diameter that contains degenerated blood and necrotic debris • Material from this cavity may infiltrate widely through the omental fat • Adhesions may form between the ulcer and surrounding organs or the abdominal wall	Post-mortem examination, transabdominal ultrasonography Laboratory testing

Table 1 (continued)

Cause	Species	Postmortem findings	Confirmation test
Electrical injuries	Cattle, sheep, and goat	• Rigor mortis develops and passes quickly • Postmortem distention of the rumen occurs rapidly and must be differentiated from antemortem ruminal tympany; in both conditions, • The blood tends to clot slowly or not at all • The mucosae of the upper respiratory tract, including the turbinates and sinuses, are congested and hemorrhagic; • Linear tracheal hemorrhages are common, and large blood clots are occasionally found in the trachea, but the lungs are not compressed as in bloat • All other viscera are congested, and petechiae and ecchymoses may be found in many organs • Close examination of the feet may provide physical evidence of pathology	Post-mortem examination, examination for burn injuries
Inhalation pneumonia	Cattle, sheep, and goat	• Pneumonia is usually in the cranioventral lobes of the lung • May be unilateral or bilateral and centered on airways • Aspiration in a standing patient often involves the right cranial and right middle lung lobes • In the early stages, the lungs are markedly congested with areas of interlobular edema • Bronchi are hyperemic and full of froth • The pneumonic areas tend to be cone-shaped, with the base toward the pleura • Suppuration and necrosis follow • Foci become consolidated and soft or liquefied, reddish brown, and foul smelling • Acute fibrinous pleuritis with pleural exudate	Pulmonary tissues do not deflate and do not float in formalin
Parasitism			
Haemonchus contortus	Cattle, sheep, and goat	• Dark red-brown contents with multifocal mucosal hemorrhages in the abomasum have • Presence of characteristic nematodes in the abomasum • Widespread subcutaneous edema; mostly in submandibular soft tissues "bottle-jaw" • Hydrothorax, hydropericardium, and ascites • Lymph nodes draining the abomasum may double in weight	Presence of characteristic nematodes in the abomasum, post-mortem examination
Strongyloides. papillosus	Suckling ruminants	• Catarrhal enteritis with petechiae, ecchymoses, and villus atrophy • Parasitic adults may be visible grossly	Identification of eggs in feces collected from the rectum, mucosal scrapings at necropsy,

Table 1 (continued)

Cause	Species	Postmortem findings	Confirmation test
Ophthalmomyiasis "uitpeuloog" or "gedoelstial myiasis"	Cattle, sheep, and goat	• Vary from transient mild conjunctivitis to destructive ophthalmitis with orbital or periorbital edema or abscessation affecting one or both eyes • Thrombosis may be very extensive, and may involve the jugular vessels and endocardium, and when coronary vessels are affected (sudden death)	Post-mortem examination, identification of parasite
Eosinophilic myositis	Cattle and sheep	• Well-demarcated, green, focal stripes or patches that fade to off-white when exposed to air • Lesions may be widespread through all muscles, including the heart • Individual lesions may be 2–3 mm to 5–6 cm in diameter in both heart and skeletal muscle • Green color to the lesion (abundance of eosinophils) • Eosinophilic myositis in sheep tends to occur in young animals	A heat-stable, eosinophil-chemotactic substance has been isolated from affected bovine muscle in which eosinophilic myositis lesions were present
Dreschlera spp. Acute bovine liver disease	Cattle	• Serosal petechiae and/or gastrointestinal hemorrhage, and hepatomegaly with a pronounced • Hepatic reticular pattern • The characteristic histologic lesion involves a periportal hepatocellular necrosis with bile duct proliferation	The presence of mycotoxin(s) produced by the fungus *Dreschlera biseptata* growing in some plants has been suggested to be the principal toxicant or cofactor in the syndrome
Other causes of SD			
Neuronal inclusion-body diseases	Japanese brown cattle (females)	• There are no gross lesions of significance • Single or sometimes multiple, eosinophilic cytoplasmic inclusion bodies in large neurons of the midbrain, pons, and medulla • The inclusions are mostly in the axon hillock region, are oval, and about 18 μm in greatest diameter	
Jejunal hematoma (hemorrhagic bowel syndrome, intestinal hematoma)	Cattle	At postmortem examination, there are one or more short jejunal loops with intramural hemorrhage. The latter usually distends the intestinal mucosa to the point of complete or partial obstruction of the intestinal lumen (Fig. 1-55B). Intraluminal hemorrhage is also present in some cases	

Table 1 (continued)

Cause	Species	Postmortem findings	Confirmation test
Pulmonary thromboembolism and liver abscesses	Cattle	• Animals are found dead with blood flowing from the nares, and expectorated blood is often detected in the stomach • Liver abscesses in cattle may extend into hepatic veins, spreading to the lungs via the caudal vena cava to cause pulmonary thromboembolism, acute interstitial lung disease, or chronic lung abscesses • Abscesses occasionally erode pulmonary vessels, causing massive hemorrhage, epistaxis, and exsanguination (sudden death)	Because the clinical features may be vague, the branches of the pulmonary arteries must be opened at autopsy in all animals with clinical evidence of respiratory disease
Dilated cardiomyopathy	Cattle (Japanese black calves younger than 30 days)	• Marked cardiac enlargement with left ventricular dilation, hydropericardium, hydrothorax, ascites, pulmonary edema, and hepatic and splenic congestion • Extensive myocardial degeneration and necrosis with fibrosis are present in the left ventricle, particularly affecting the papillary muscles • The right ventricle is similar but less often involved	Post-mortem examination, test for an autosomal recessive

Conclusion

The diagnosis of SD in ruminants is challenging. Multiple factors are involved and several causes may be present simultaneously. In addition, diagnosis can be further complicated by variability in husbandry systems and intervals between herd visits. The definitions provided by Kelly (2003) and Anscombe (2016), "an acute death of an animal or the discovery of dead, well-fed, non-perinatal animals within two days of observation, apparently in good health" and "the sudden and unexpected death of an apparently healthy animal," respectively, appear to be the most appropriate for SD syndrome in ruminants (Anscombe 2016; Kelly 2003). Similarly, in human medicine, there is no universally accepted definition of SD, but it is generally used to refer to deaths not preceded by significant symptoms (Sessa et al. 2021). The relevant definition of SD syndrome would be the sudden and unexpected death of an apparently healthy animal, excluding cases of loss due to diseases with obvious clinical signs. This means that livestock farmers must adopt modern means of control to better monitor herds, such as video surveillance and the widespread use of electronic chips to quickly identify sick animals and know the conditions in which sudden death occurred.

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