Sudden death in sheep

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doi:10.1136/inp.g5351

Several factors conspire to make the definition of 'sudden death' in sheep problematic. The most commonly accepted definition is death that occurs since the last inspection (ie, within 12 to 24 hours). However, it can be challenging for the shepherd to detect early clinical signs of disease in sheep due to extensive husbandry, limited supplementary feeding and sometimes, despite the legal requirements, infrequent inspection. In addition, group sizes tend to be large, making recognition of individual animals difficult. Consequently, veterinary investigation into any case where sheep have been found dead may not always reveal an acute cause. This article aims to consider such situations as well as those of truly 'sudden death'.

Investigation

Taking a thorough history is essential and allows a list of possible diagnoses to be drawn up before any animals are examined. Useful background information to gather is summarised in Box 1.

The ability to carry out a postmortem examination is a useful tool when investigating sudden deaths in sheep, with the size of carcases making it a fairly straightforward undertaking. Whether carried out on farm (Fig 1), at a fallen stock centre or at a veterinary laboratory, postmortem examination can provide an immediate diagnosis allowing steps to be taken to prevent further losses. Wherever possible, several carcases should be examined, as one may not be representative of the problem. Postmortem examinations should be carried out in a consistent fashion (see Table 1) to avoid missing anything significant. Samples should be collected in order to confirm a suspected diagnosis, and particularly if the cause of death remains unclear. Liver and lung are the most useful tissues to swab for bacteriology. Small sections of tissue can be collected for histopathology into leak proof containers of 10 per cent formol saline allowing a ratio of fixative:tissue of at least 10:1.

Sheep suffering from neurological conditions generally show clinical signs lasting for hours to days, but these may have been missed by the shepherd. Gross postmortem findings are generally absent, with diagnosis relying on histopathology.

Sudden deaths can be categorised by the age or type of sheep or the body system affected, but this article divides deaths into those caused by traumatic, infectious (bacterial, viral and parasitic), feed-related or toxic reasons.

Traumatic and accidental death

Sudden death due to injuries such as those inflicted by a predator or a road traffic accident may cause obvious external damage or rupture of internal organs and thus be simple to diagnose. In contrast, injury caused by fighting may cause a true sudden death with few obvious signs except perhaps haemorrhage and bruising in the cervical region.

The collection of a comprehensive history is very important: recent dipping could suggest an inhalation pneumonia, while a drenching injury may result in either a sudden or more prolonged death with distinctive damage in the pharyngeal area. Stress from recent handling may precipitate death from a metabolic disorder such as hypocalcaemia or from a pre-existing heart condition such as endocarditis (Fig 2).

The veterinarian is often asked to pronounce on suspected lightning strike due to its common inclusion in insurance

Box 1: General history relevant to cases of sudden death

- Age, breed, sex.
- Number of deaths, number in affected group(s).
- Time period over which deaths have occurred.
- Interval between last inspection and animal(s) being found dead.
- Expected lambing date.
- History of clinical signs in the dead sheep or others in the group.
- Body condition of the dead sheep compared to the rest of the group.
- Housed or at grass.
- Grazing/feeding history, including recent changes of field/diet.
- Recent weather events, eg, flooding, storms.
- Vaccination history including lambs and vaccination status of the dam.
- Treatment history, eg, anthelmintics, flukicides, boluses.
- Recent handling, eg, dipping, transport, weaning, mixing of groups.

Fig 1: Undertaking a postmortem examination on farm is usually an opportunity not to be missed

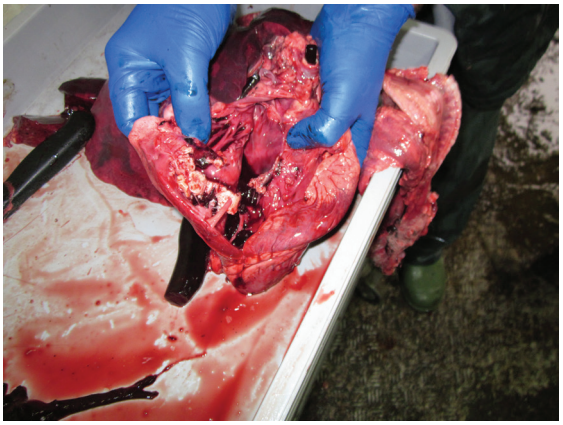


Fig 2: Pre-exisiting endocarditis may cause an apparently sudden death. There may be another obvious focus of bacterial infection; in this case, a chronically infected foot

policies. Circumstantial evidence, such as nearby trees, water or wire fencing, plus knowledge of a recent storm, is vital to diagnosis. Death is usually instantaneous with few distinguishing features postmortem. There may be singe marks on the skin, a rapid onset and disappearance of rigor mortis and rapid decomposition of the carcase with distension of the rumen. Haemorrhages and petechiation are usually notable on the epicardium and endocardium with congestion of coronary as well as peripheral blood vessels. Blood is usually normal in colour and poorly clotted. In housed animals, faulty electrical equipment may cause death by electrocution with similar postmortem signs.

Bacterial conditions

Sudden death may result from either an acute mastitis or metritis. Both conditions are obvious on gross postmortem examination.

Clostridial diseases

Despite the availability of effective vaccines, clostridial diseases kill large numbers of sheep every year, with pulpy kidney and lamb dysentery diagnosed most often. Affected animals are occasionally found alive and may show

Table 1: Outline of postmortem examination in sheep that have been found dead

Area of body	Assessment
External examination	Mucous membranes: check for anaemia/jaundice, haemorrhages, ulceration, vesicles Check for discharges from eyes, nose, vulva or evidence of faecal staining Check udder for mastitis Check for evidence of trauma, eg, dog bite, fracture Assess body condition Can collect aqueous/vitreous humour (see Edwards and others 2009)
Subcutaneous tissues	Check for subcutaneous fat, anaemia/jaundice, hydration (dry tacky if dehydrated) Check peripheral lymph nodes (size, haemorrhages, abscessation)
Circulatory system	Can collect blood for serology but not biochemistry Check for increased volume of pericardial fluid +/- fibrin clot, endocarditis, pericarditis Check myocardium for pale lesions of nutritional myopathy
Respiratory tract	Check larynx for obstruction, laryngeal chondritis Open up trachea and bronchi to check for froth, parasites, foreign body Check for pleural effusion, pleurisy Check lungs for consolidation, abscessation, neoplasia Check lymph nodes (size, abscessation, haemorrhage)
Digestive tract	Check pharynx for necrosis or signs of dosing gun injury Check oesophagus for ulceration or obstruction Check rumen fill and content for unusual leaves/material, excess grain (can check pH) Check abomasum for milk, abomasitis, ulceration Check intestines for torsion, neoplasia, intussusception, thickening, pigmentation Can collect abomasal and intestinal contents for worm counts or ileal contents for clostridial toxin testing Check for diarrhoea, can collect faeces for a worm egg count Check liver for fluke, black disease, colour change Can collect liver for copper/selenium/vitamin E/cobalt analysis
Urinary tract	Check for calculi in bladder or urethra, including vermiform appendage Check kidneys for nephrosis, hydronephrosis, colour change Assess degree of autolysis compared to the rest of the carcase Check colour of urine, eg, haemoglobinuria Can collect urine for analysis Can collect kidney for copper or lead analysis
Reproductive tract	Check for stage of pregnancy, evidence of impending abortion, metritis in females Check scrotum for evidence of hernia or inflammation
Musculoskeletal system	Check joints and examine joint fluid Check muscles for areas of discolouration
Central nervous system	Remove brain if no diagnosis has been reached or clostridial enterotoxaemia is suspected Check for cerebellar coning or fluorescence under UV light

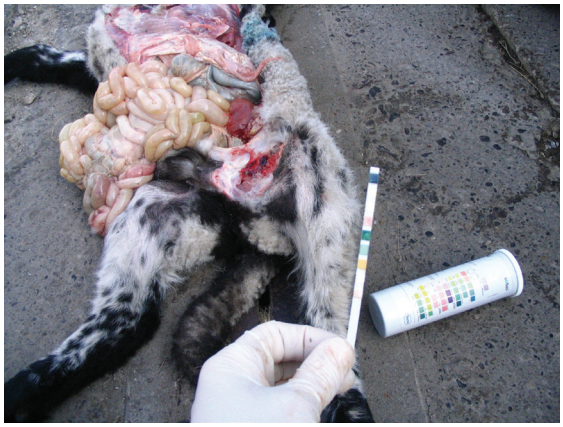


Fig 3: Dipsticks are useful to test for glucosuria in cases of pulpy kidney

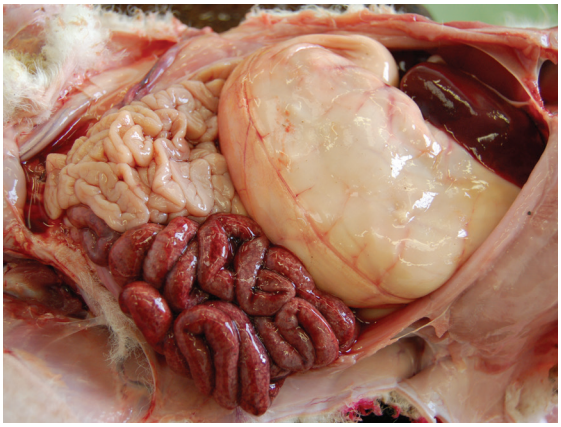


Fig 4: In this case of lamb dysentery, a section of jejunum is inflamed and the abomasum is full of milk

neurological signs such as opisthotonos due to the action of epsilon toxin, which damages vascular endothelium. General brain swelling leads to flattening of the gyri and cerebellar coning in some cases. Glucose may be detected in the urine (Fig 3) as a consequence of hyperglycaemia which is a response to toxaemia. Focal inflammation and necrosis of the small intestine is a typical finding in cases of lamb dysentery (Fig 4). Detection of clostridial toxins in small intestinal contents (E in cases of pulpy kidney, B and E in lamb dysentery) can support a diagnosis but can also be produced postmortem. In addition, toxins, particularly B toxin, are labile so false negative results can occur. General postmortem findings in cases of clostridial disease include effusions into body cavities, including the pericardium, which may contain a fibrin clot (Fig 5). Carcases autolyse rapidly, which can make the assessment of 'pulpy' kidney subjective. A summary of clostridial diseases is given in Table 2 and more detailed information can be found by referring to Lewis (1998).

Fig 5: Pericardial fluid accumulation with a fibrin clot in this case of pulpy kidney

Table 2: Clostridial diseases causing sudden death in sheep

Disease	Cause	Summary
Pulpy kidney	*Clostridium perfringens* type D	Fast-growing lambs after waning of maternal immunity Outbreaks can occur after a change to a better diet Sporadic cases in adult sheep particularly tups
Lamb dysentery	*Clostridium perfringens* type B	Lambs up to three weeks of age Unvaccinated dams High milk intakes can predispose Outbreaks can occur in unhygienic conditions Older lambs may show signs of abdominal pain and/or haemorrhagic diarrhoea before death
Black disease	*Clostridium novyi*	Migration of liver fluke predisposes to infection Pale area(s) of necrosis in liver surrounded by a darker ring
Abomasitis/ metritis	*Clostridium sordellii*	High milk intakes can predispose to infection in four- to 10-week-old lambs. Older lambs/adult sheep can be affected but show less severe changes postmortem Abomasal wall can be emphysematous and oedematous (Fig 6) Also reported to cause periparturient lamb death and fatal metritis sometimes following vaginal prolapse
Blackleg	*Clostridium chauvoei*	May follow an injury or assisted lambing (perineum swollen and oedematous). Affected areas of muscles appear dry and are dark in colour
Braxy	*Clostridium septicum*	Causes abomasitis possibly after ingestion of frosted forage
Struck	*Clostridium perfingens* type C	Uncommon clostridial enterotoxaemia causing a haemorrhagic enteritis mainly of adult sheep

Pasteurellosis

Pasteurellosis is a common reason for sudden death in sheep, caused by either *Mannheimia haemolytica* or *Bibersteinia trehalosi*, both of which are common commensals located in the pharynx and tonsils. There are many possible predisposing factors including recent handling, transport or mixing, changes in weather, improved nutrition, cobalt/selenium deficiency or underlying infections such as *Mycoplasma ovipneumoniae*. There is a clear age distribution, with *M haemolytica* septicaemia causing sudden death in lambs up to 12 weeks of age, often as colostral immunity wanes, but also causing fatal pneumonia in older sheep. Gross postmortem findings and even bacteriology may be equivocal, particularly in cases of peracute death. Following acute deaths, there may be signs of general septicaemia such as subcutaneous ecchymotic haemorrhages over the ribs and throat area, and lung oedema with froth in the airways. The lungs are purple-red, swollen and heavy (and will sink in water compared to a congested lung, which would float). Fibrinous pleuritis is seen in some cases and there may be lung consolidation, particularly cranioventrally. Systemic pasteurellosis due to *B trehalosi* typically affects weaned lambs in the autumn and early winter and the protection afforded by pasteurella vaccines is not absolute. *B trehalosi* can produce characteristic lesions including mucosal necrosis in the pharynx, oesophagus and stomach, and pin point foci of hepatic necrosis (Figs 7, 8).

Alarm bells should ring in flocks that regularly lose adult sheep to pasteurella pneumonia. The possibility of an underlying infection with either ovine pulmonary adenocarcinoma or maedi-visna should be investigated.

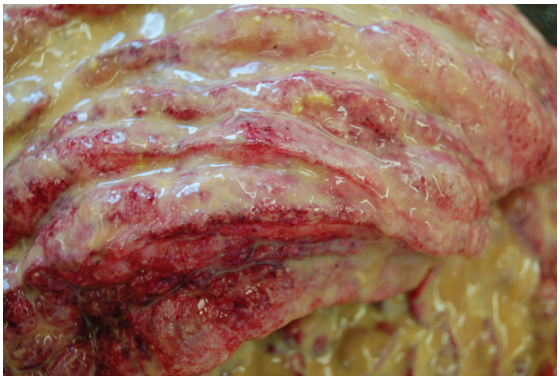


Fig 6: The mucosal surface of the incised abomasum is hyperaemic, oedematous and emphysematous in cases of *Clostridium sordellii* infection

Listeriosis

The clinical signs of listerial encephalitis are a consequence of microabscess formation around the pons and trigeminal nerve nucleus causing depression, ptyalism and facial paralysis, which is unilateral in most cases. Death usually occurs within a week of the onset of clinical signs. There may be a history of access to poor quality silage.

Anthrax

Anthrax due to *Bacillus anthracis* is an uncommon but notifiable disease in the UK; the last outbreak affecting sheep was confirmed in 1986, although there was a case affecting cattle in 2006 in Wales. If anthrax is suspected, the carcase should not be opened and should be reported to the AHVLA. Externally, the carcase may be distended and show rapid decomposition with dark blood oozing from the orifices. If a carcase is accidently opened, lesions associated with septicaemia are visible. These include a swollen friable spleen, lymph node enlargement, haemorrhages on serosal surfaces, oedematous red-tinged effusions and dark blood that fails to clot.

Viral conditions

Foot-and-mouth disease

The UK is currently foot-and-mouth disease (FMD)-free following the outbreaks in 2001 and 2007. FMD virus causes vesicular lesions and, in adult animals, mortality is generally low. Infection in young lambs, however, may lead to high mortality due to an acute myocarditis. Gross lesions may consist of grey or yellow streaks in the myocardium leading to the description of 'tiger heart' lesions. Suspicion of FMD must be reported without delay to the duty veterinary officer at the AHVLA.

Louping ill

The presence of ticks on a carcase may raise suspicions of louping ill, particularly if there is a history of ataxic sheep; this virus can cause coma and death within four days of a tick bite. The diffuse non-suppurative meningo-encephalomyelitis may be accompanied by secondary pneumonic lesions. Positive serology with a predominance of IgM antibodies confirms recent infection.

Parasitic conditions

Nematodirosis

Nematodirosis outbreaks occur when large numbers of *Nematodirus battus* eggs hatch simultaneously in fields grazed by six- to 12-week-old lambs. Synchronous hatching requires a period of chill followed by a warmer spell explaining why cases are traditionally seen in spring and early summer. However, outbreaks can occur later in summer and autumn so it should not be ruled out based on season alone. Severely affected lambs may be found dead, while others have evidence of scour. Clinical signs can develop in the prepatent period so a negative *N battus* egg count does not exclude the possibility of infection. At postmortem examination, tangled masses of *N battus* worms may be found in the distal small intestine.

Coccidiosis

Concurrent coccidiosis is not uncommon in the same age group and it is important to reach an accurate diagnosis to allow specific targeted treatment. The large number of non-pathogenic species means that faecal oocyst counts are of limited value unless speciation is carried out. Coccidiosis affects the ileum, caecum and colon (Fig 9) and may cause either a thickening or a thinning of the gut wall with gross changes that are often difficult to appreciate, although there may be pathognomonic raised white lesions in this area of the intestines.

Fasciolosis

The link between high summer rainfall and an increased risk of acute fasciolosis is well known. Numbers of encysted metacercariae generally peak in September or October and sheep grazing high risk pastures can ingest a fatal dose in less than a day. Animals are found dead three to six weeks later, with several thousand immature fluke in their livers. Migration through the parenchyma cause, widespread destruction and haemorrhage, with death ensuing before there is any loss of body condition. Carcases are anaemic and there may be free blood in the abdomen originating from hepatic haematomas (Fig 10). Cohorts may appear dull and lethargic with pale conjunctivae and signs of abdominal pain. Ingestion of smaller numbers of

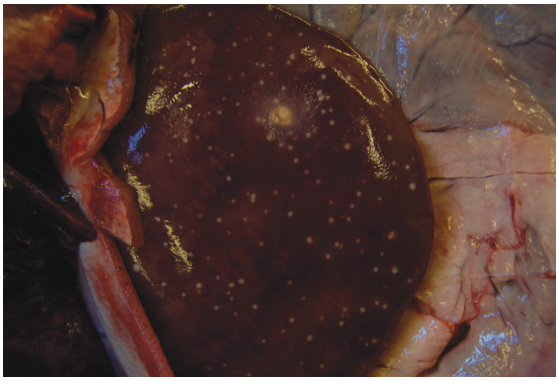


Fig 7: Focal hepatic lesions in a case of septicaemia caused by *Bibersteinia trehalosi*

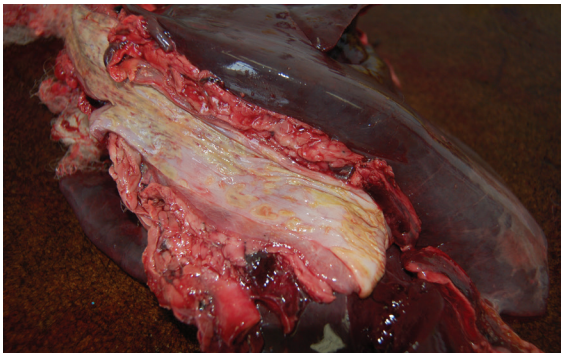
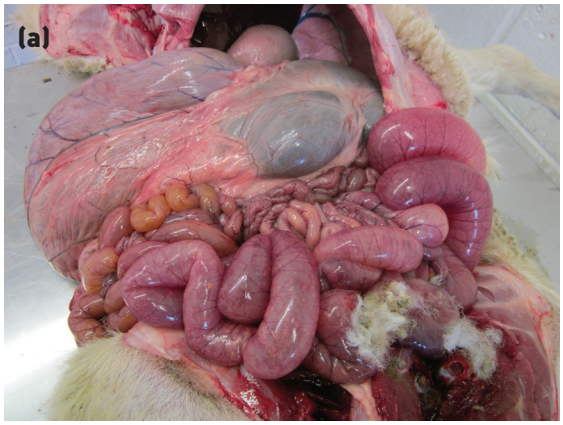


Fig 8: Oesophageal ulceration in a case of septicaemia caused by *Bibersteinia trehalosi*. The lungs are oedematous and congested



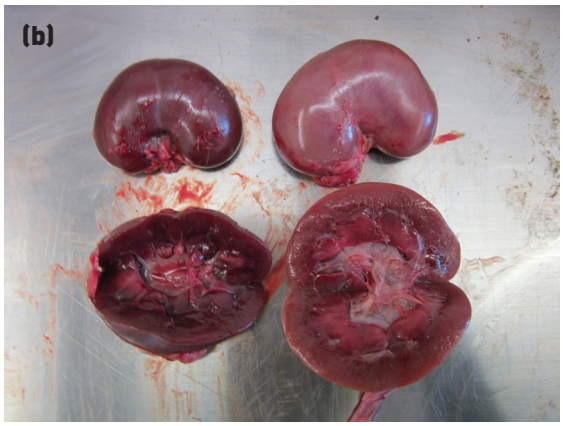


Fig 9: (a) Inflamed gas-filled small intestine of a lamb that had been treated for coccidiosis. (b) Nephrosis is often a complicating sequela notable by the enlarged pale kidney and indicates that this lamb was not a 'sudden' death

metacercariae over a longer period results in subacute fasciolosis, which can also present as sudden death (Figs 11, 12). The liver contains both immature and adult fluke and, in addition to anaemia, there can be significant weight loss and submandibular oedema as a consequence of hypoalbuminaemia.

Haemonchosis

Haemonchosis is a possible differential diagnosis in these cases due to the blood feeding activity of *Haemonchus contortus* worms. All ages of sheep can be affected and outbreaks of disease can follow a move to contaminated pasture. Female worms are highly fecund so very high worm egg counts (often more than 10,000 eggs per gram) are typical. The worms are easily visible within the abomasum of infected sheep and the intertwining of the uterus with the blood-filled intestine gives the classic 'barber's pole' appearance (Fig 13).

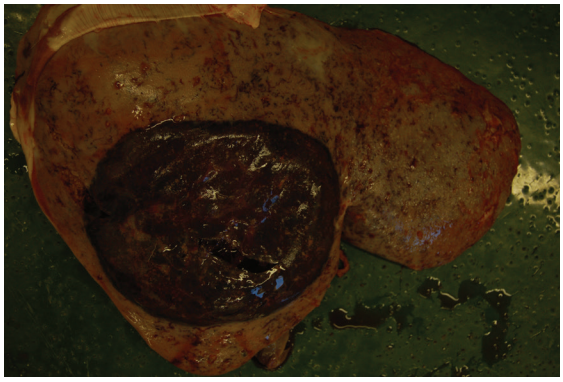


Fig 10: Immature liver fluke may cause considerable areas of haemorrhage, as well as widespread parenchymal damage

Feed-related deaths

Bloat

Antemortem bloat may occur quickly if a well-fleeced adult becomes cast on its back or following unstable ruminal fermentation. As ruminal microbes rapidly produce gases that distend the rumen postmortem, it is important to distinguish bloat as a cause of death. In this circumstance, the pressure of the distended rumen causes the liver and caudal oesophagus to look pale in comparison to the cervical oesophagus, which is generally congested.

Redgut

Redgut is a condition that may affect weaned lambs or adults, particularly when grazing legumes or lush pasture. The intestines become unstable due to rapid transit times, small rumen volume and large intestinal fermentation. Torsion occurs around the root of the mesentery (Fig 14) and death usually follows with few premonitory signs except perhaps acute abdominal pain, distension and toxic mucous membranes.

Acidosis

Acidosis or grain overload is a common cause of death in sheep often with a history of recent introduction or

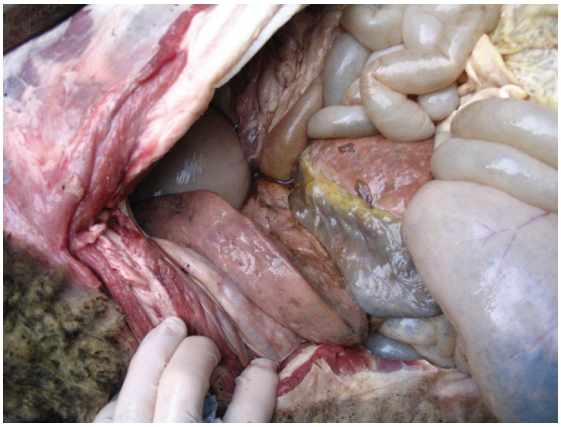


Fig 11: In this case of subacute fluke infection, adult fluke were immediately evident in the abdominal cavity, presumably following rupture of the gall bladder

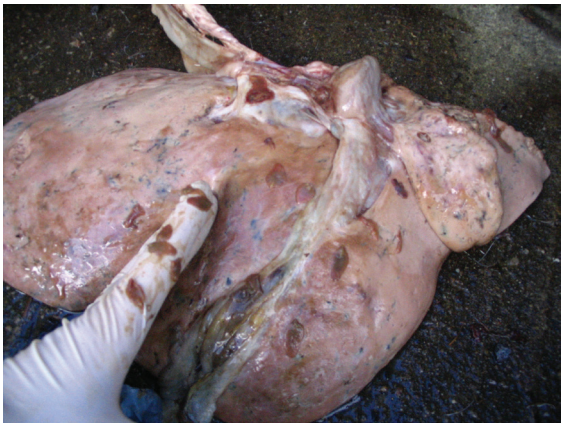
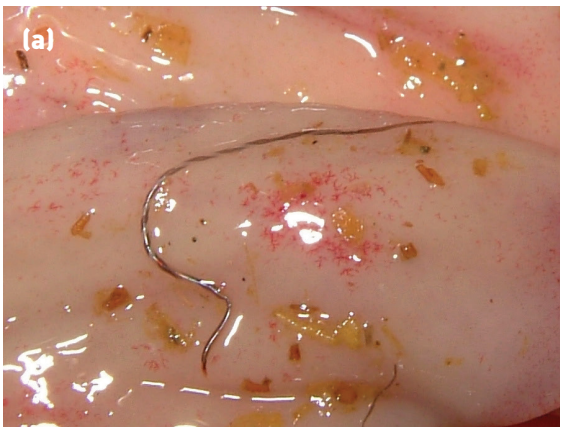


Fig 12: In subacute fluke infection, there may be numerous adult liver fluke in the bile ducts as well as evidence of considerable parenchymal damage



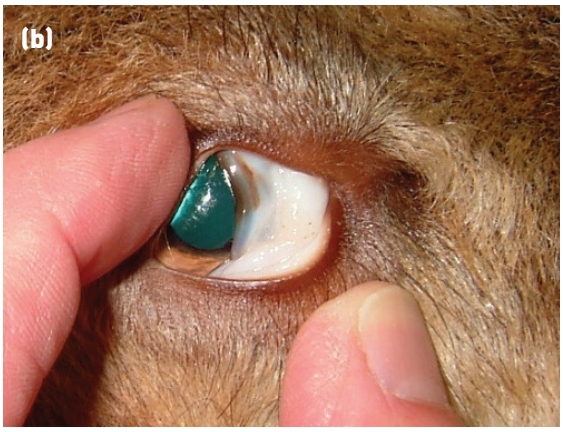


Fig 13: 'Barber's pole' *Haemonchus* worms cause pinprick haemorrhages on the (a) abomasal mucosa and (b) severe anaemia throughout the carcase, seen here in the eye

inadvertent access to grain or concentrate feed. Ingestion of large quantities of highly digestible feed leads to a rapid fall in rumen pH. Sheep may be found dead or appear listless, exhibit signs of abdominal pain and lie down frequently. Diarrhoea containing undigested grains may develop after 12 to 24 hours.

Postmortem examination reveals sour smelling rumen contents containing many undigested grains and watery small and large intestinal content. A rumen pH of 4.5 or less is supportive of a diagnosis, but note that rumen pH may rise after death.

White muscle disease

Diets deficient in selenium and/or vitamin E predispose to white muscle disease, which typically affects growing lambs. Sudden death may occur if cardiac (Fig 15) or respiratory muscle is affected. Stiffness and signs of muscle weakness may be observed if skeletal muscles are affected. Postmortem examination may show focal muscle pallor but gross changes can often be difficult to see. Rarely, a whole muscle body may be affected giving a 'fish flesh' appearance. Histopathology of affected muscle may confirm the diagnosis supported by estimation of liver selenium and vitamin E levels.

Hypomagnesaemia and hypocalcaemia

Hypomagnesaemia is uncommonly reported as a cause of sudden death in lactating ewes particularly on lush pasture. Sheep are typically found dead, but, if found alive, they may exhibit muscle tremors, recumbency, seizures and opisthotonos with death ensuing rapidly.

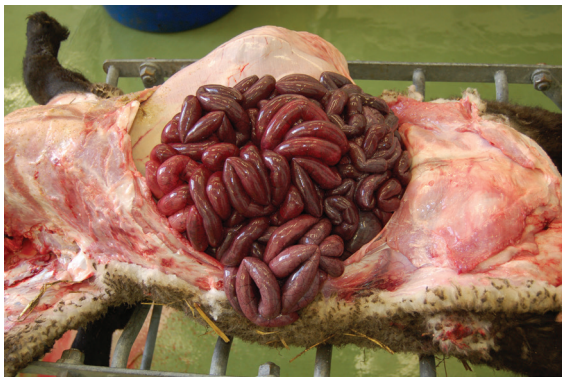


Fig 14: Torsion of the intestine in cases of redgut causes distinct hyperaemia, although both the duodenum and rectum are unaffected. Careful investigation allows the torsion to be traced to the root of the mesentery

Ewes with hypocalcaemia are usually recumbent for 24 to 48 hours before death, but it may be reported as sudden death. Cases are most common in late gestation, but may occasionally occur in early lactation. Disease may be precipitated by a change or reduction in feed intake, which may be associated with a stressful event such as movement, inclement weather or dog worrying.

Postmortem examinations are unlikely to reveal any significant gross findings and detection of low aqueous or vitreous humour magnesium or calcium concentrations may provide supportive evidence (Edwards and others 2009). Low serum calcium concentrations in typically affected live ewes is diagnostic.

Polioencephalomalacia

Cases of polioencephalomalacia (cerebrocortical necrosis) commonly occur about a fortnight after a diet change or anthelmintic drench, with clinical signs including blindness and opisthotonos (Fig 16). However, with these clinical signs appearing 24 to 36 hours after onset, it is rare that a case of polioencephalomalacia causes a truly 'sudden' death. Altered glucose metabolism damages the grey matter (polio) giving the cerebral hemispheres a dull yellow appearance. Affected areas may fluoresce under UV light.

Toxicities

Texel, Suffolk, Lleyn and North Ronaldsay sheep are particularly at risk of chronic copper toxicity, which may result in sudden death as stress precipitates the release of copper from over-burdened hepatic cells. The resulting intravascular lysis of red blood cells is evident in the live animal as jaundice, haemoglobinuria, shallow distressed

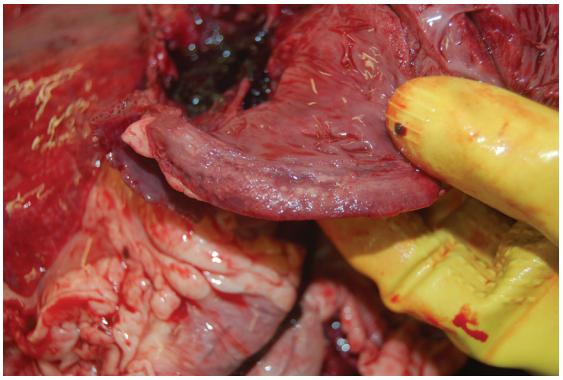


Fig 15: In this case of white muscle disease, the myocardium is notably pale

Fig 16: Opisthotonos may be the most obvious feature in cases of polioencephalomalacia although this does not cause a truly 'sudden' death

Fig 17: Red wine urine, black kidneys and a yellow/orange liver are the distinguishing features of copper poisoning

Fig 18: These leaves were removed from the rumen of a sheep that had access to both a wood and a garden. The single large leaf at the top is *Photinia* species which contains cyanogenic glycosides and was the cause of death in this case. The male fern leaf was thought to be irrelevant and the third leaf remained unidentified, despite taking it to the local garden centre

breathing and progressive nervous signs. Postmortem findings of a jaundiced carcase, yellow/orange liver and black kidneys are distinctive (Fig 17). The diagnosis can be confirmed by measuring liver and kidney copper content.

Acute copper toxicity may occur in any breed within a few days of an overdose as the high levels of circulatory copper overwhelm the liver's capacity to remove and store it. Live animals are pale and depressed with tachycardia, tachypnoea and green mucoid diarrhoea. Abomasitis is evident at postmortem examination as is petechiation, erosion and ulceration, with generalised and pulmonary oedema also seen.

Plant toxicities should always be considered if there is a history of sheep escaping, snow cover or garden trimmings being dumped in the field (Fig 18). Rhododendrons (and related azalea, *Pieris* and *Kalmia* species) contain toxic dipteroids that cause respiratory and central nervous depression, as well as hypotension. Clinical signs include vomiting, excessive frothy, green salivation, severe abdominal pain and nervous signs. Evidence of these signs, together with remnants of leaves in the rumen, is often sufficient for a postmortem diagnosis. Excessive salivation, vomiting, diarrhoea and nervous signs are also seen following alkaloid poisoning such as might be caused by the ingestion of hemlock.

Another well-known alkaloid-containing plant is yew (*Taccus baccata*). Taxine, found in yew, causes sudden death via a strong depressive effect on the heart and respiratory centre with leaves often still found in the buccal cavity at postmortem examination.

Acorns and oak leaves contain hydrolysable tannins, which initially cause constipation and then a black tarry diarrhoea or dysentery. Dark urine and oculonasal discharges can be seen and there may be oesophageal ulceration at postmortem examination. Uraemia as a consequence of kidney failure is a consistent finding.

Brassicas accumulate nitrates, particularly in the roots and stems during overcast conditions. Following conversion to nitrites in the rumen, absorption into the bloodstream leads to the formation of methaemoglobin, which is unable to transport oxygen, resulting in anoxia and dyspnoea. At postmortem examination, the blood and tissues appear brown, lungs are congested and there may be petechial haemorrhages on the heart and trachea. Brassicas also have the ability to cause haemolytic anaemia with acute cases resembling copper toxicity at postmortem examination.

A history of recent treatment or access to chemicals should always raise the possibility of iatrogenic fatalities. Nitroxynil toxicity can occur following an overdose, with respiratory distress, recumbency and death the most commonly reported signs. Inadvertent intravascular or intramuscular administration of tilmicosin may result in rapid death due to respiratory failure.

A description of all possible toxicities is beyond the scope of this article and other texts should be consulted for further information (for example, Angus 2007)

Conclusion

The causes of sudden death in sheep are many and varied. It is important to keep an open mind, but also to remember that common things are common. A good history will provide key pieces of information which, together with postmortem examination of fresh carcases, will enable a diagnosis to be reached in most cases.

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Further reading

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InPractice

Sudden death in sheep

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In Practice 2014 36: 409-417
doi: 10.1136/inp.g5351

Correction

Sudden death in sheep (*In Practice*, July/August 2014, vol 36, pp 409-417; doi: 10.1136/inp.g5351)

Figures 13a and 13b should have been accredited to Rudolf Reichel. The authors acknowledge his help for these. The error is regretted.

doi: 10.1136/inp.g5890