PCV2-associated foetopathy in pigs
APHA Disease Surveillance Report
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- Mucosal disease outbreak
- Cerebellar cortical degeneration in a Limousin cross heifer
- Rickets in lambs
- BVD in sheep

CATTLE

Alimentary disease

Mucosal disease: Starcross diagnosed an outbreak of mucosal disease in suckler calves which were housed with their dams. The fifth animal to die from a group of 50 Devon-cross calves, aged seven months, was received for postmortem examination. The clinical signs reported by the submitting veterinary surgeon included pyrexia, listlessness, a purulent nasal discharge, crusty eyes and bloody scour which eventually progressed to death in three to four days. Attempted treatment with various antibiotics and anti-inflammatory drugs had proved unsuccessful. Within the oral cavity there was severe superficial, coalescing, ulceration of the dental pad and palatine ridges. Some of the ulcers were overlaid with yellow necrotic material (fig 1). Necrotic, haemorrhagic ulcers were also present within the Peyer’s patches of the small intestine. Diagnosis of mucosal disease was confirmed by PCR testing of the spleen which identified BVDv-1 infection. History and other presenting signs ruled out foot and mouth disease, which should always be considered. APHA field veterinarians and Veterinary Investigation Officers are available for consultation for unusual, novel and unresolving cases.
Nervous disease

**Pseudomonas sp meningitis:** An outbreak of disease in unweaned dairy calves was investigated at Langford. It was reported that calves aged around nine to 14 days exhibited sudden onset collapse with at least eight animals on the 650 cow dairy farm similarly affected. The calves had shown no signs of illness prior to being found in lateral recumbency with opisthotonos. Calf management was reported to be good with 3.5 litres of colostrum administered by stomach tube at birth and the navels dipped. The calves were subsequently moved into groups of 20 and machine-fed powdered milk. Postmortem examination of three calves was undertaken. One had gross evidence of meningeal opacity with purulent deposits. *Pseudomonas* spp. were isolated in mixed culture from the brain and histological examination confirmed a severe, suppurative meningitis and encephalitis. *Pseudomonas* spp. are not typically associated with meningitis; other environmental bacteria, such as *E. coli* and streptococci, are more commonly associated with meningeal infections at this age. Enteritis was present in the two other calves which had liquid yellow intestinal contents; mesenteric lymphadenopathy and the digestive tracts were relatively empty suggesting inadequate feed intake. Cryptosporidia were identified in the faeces and ZST results indicated insufficient colostral administration. The range of findings indicated a need for improved management of the calves on this large dairy farm.

**Cerebellar cortical degeneration** or ‘abiotrophy’ was diagnosed in a three-quarter bred Limousin heifer. The animal had been born normally and grew well initially, but at around four months of age it developed ataxia and a hypermetric gait, which particularly affected the hind legs. There was no response to treatments and the animal spent increased time recumbent. It was euthanased and examined postmortem. Only non-specific gross pathology associated with its recumbency was evident but histopathological examination of the brain identified multifocal Purkinje neuronal loss with associated gliosis. There was also loss of the Purkinje dendritic trees resulting in narrowing of the molecular layer and prominence of the radial glial
processes. These changes were considered indicative of cerebellar cortical degeneration which is associated with the premature or accelerated degeneration of formed brain elements, presumed due to an intrinsic metabolic defect. It is believed to be inherited and has previously, rarely, been reported in Limousin cattle.

Respiratory disease

Mannhaemia haemolytica pleuropneumonia: Outbreaks of pleuropneumonia caused by Mannheimia haemolytica were reported by two VI Centres. At Carmarthen seven cows in a dairy herd died after exhibiting acute respiratory distress. This spring around 200 of the 300 cows had calved in a loose housed area. Following calving the cows were moved into cubicles where the seven animals developed acute respiratory distress and died despite attempted treatment with antibiotic. One was examined postmortem which confirmed extensive anteroventral lung consolidation with a fibrinous pleuritis and pronounced widening of interlobular septae. Pure cultures of M. haemolytica were isolated, with tests for viral agents including BVDv negative. At Penrith postmortem examination revealed similar pathology in a dairy cow, one of two animals which died of seven affected with severe respiratory signs. No underlying ‘trigger’ for either of the outbreaks was identified. Cases of M. haemolytica pleuropneumonia in adult dairy cows have previously been identified, usually occurring between November and March (Harwood and others 1995).

SMALL RUMINANTS

Musculoskeletal disease

About 30-40 from a group of 500 homebred 10-month-old lambs were described as “going slow”. Three were severely affected and recumbent but still bright, alert and eating. The group was on stubble turnips and had been most of the winter. Vitamin supplements were given three times at intervals of about 3 - 4 months. They had been wormed and fluked but not vaccinated. A lamb was submitted for post-mortem examination to Thirsk. The distal tibia and radius was ‘flared’ with an increased diameter at the site of the growth plate. The bones fractured easily at the site of the growth plate and evidence of necrosis was visible (fig 2). The joint fluid appeared normal and no lateral deviation of the long bones was detected. Histological examination of rib revealed moderate to severe chronic metaphyseal osteopathy. There was associated inter-trabecular patchy prominent fibroplasia and lack of haematopoiesis. The physis appeared widened and the hypertrophic zone in the growth plate cartilage was somewhat irregular and narrow. In view of the nature and location of the metaphyseal lesions, this was very likely due to the resolving phase of rickets.
Fig 2. Necrosis at the growth plate of a store lamb with rickets.

**Alimentary disease**

*Rotavirus* was diagnosed on ten occasions at Shrewsbury, often in lambs only a few days old. On one farm the lambs from yearlings and triplets were reported to be severely affected. The individual pens used after lambing had an earth floor so although they were cleaned out between sheep they did not dry out completely and *E. coli* was also identified which was resistant to many of the commonly used antibiotics. Over half the lambs were affected in a pedigree Texel flock.

**Systemic disease**

*Anaemia secondary to administration of cow colostrum* was thought to be the cause of death in mule cross lowland lambs. Seven had died from a group of 200. A lamb submitted to Shrewsbury was three weeks old and one of twins. The carcase was pale and slightly jaundiced, with moderate liver enlargement, marked mesenteric lymph node enlargement and enlarged, pale and firm kidneys. The pallor and jaundice seen are consistent with haemolytic anaemia due to an auto immune reaction following administration of cow colostrum.

*Selenium toxicity:* Ewe deaths due to selenium toxicity were diagnosed following the submission of a carcase and samples to Carmarthen. A soil fertiliser containing selenium had been used on the fields where the affected animals had grazed. The manufacturers indicated there was no need to remove sheep from the fertilised land and the weather was not significantly different to previous years when this substance was used without incident. Twelve deaths were reported at the time of submission. At postmortem examination of a ewe, a large amount of free sero-sanguinous fluid was present in the pleural and peritoneal cavities and a smaller amount in the pericardial sac. Lungs were dark red in colour. Liver selenium concentration was measured in the toxic range 612, 499, and 462 µmol/kg DM in three animals, with toxicity occurring >250 µmol/kg DM. The farmer agreed to animals from this group not being slaughtered for human consumption for one month to protect the human food chain (selenium does not accumulate within the animal). The pattern of reported deaths implicated a point source of exposure with no further deaths after 2 weeks of the fertiliser being spread. The remaining ewes
lambed without incident. It was advised to analyse the fertiliser to determine if increased selenium had been accidentally incorporated. It was also advised that animals should not be immediately exposed to fertilised fields in the future to minimise the likelihood of this occurring again.

**BVD in sheep:** A two-day-old lamb was submitted alive for postmortem examination. The animal was displaying tremor affecting the whole body. The brain was examined post euthanasia and it looked grossly normal. All other body systems were unremarkable. PCR for Bovine Viral Diarrhoea (BVD) virus on brain tissue was positive for BVD type I. A small percentage of suspected Border disease cases are recognized as being caused by BVD virus. This is an important consideration for cattle eradication programmes.

**Enterotoxaemia in goats:** Dairy goats had shown signs of scour and drop in milk production. The majority were recovering over a few days but samples were received from one of two that had died. It had become recumbent with a painful abdomen and was dehydrated before death. The caecal contents were noticeably watery and had mucus. Tests on small and large intestinal content were positive for the epsilon toxin of *Clostridium perfringens* consistent with enterotoxaemia due to *C. perfringens* type D.

**PIGS**

*Reproductive disease*

**Severe reproductive disease due to porcine circovirus 2:** A diagnosis of porcine circovirus 2-associated reproductive disease was made following submission of several litters of mummified and stillborn piglets delivered at term to Thirsk. This is only the second confirmed GB case of PCV2-associated fetopathy, but has been reported elsewhere in the field in Europe and North America and following experimental infection of pregnant sows. Disease manifested on two linked units as severe SMEDI, with mummified pigs and stillbirths (figure 3), not abortions and sows were well. PCV2 antigen was detected by immunohistochemistry associated with severe myocarditis lesions in foetal hearts. No other infectious agent was identified. Both units were recently established young sow herds. The sows had been vaccinated for PCV2 as weaners and, following the diagnosis, were revaccinated and PCV2 vaccination of replacement breeding gilts prior to service has been instituted. PCV2-associated reproductive disease is only likely to occur following primary infection of the sow at service or during pregnancy suggesting that there had been no PCV2 challenge of the sows prior to the units being set up until this disease event. This case confirms that PCV2-associated fetopathy can occasionally occur under certain management conditions and surveillance for this disease is undertaken routinely in undiagnosed fetopathy cases if suitable material is available.
Enteric Disease

**Gastric ulceration occurring during Glässer’s outbreak:** Fatal haemorrhage from ulceration of the *pars oesophagea* was found to be the cause of death of an eight-week-old pig from a batch of 850 recently treated for Glässer’s disease. Since then occasional deaths totalling 26 had occurred over a ten-day period of which the pig submitted to Bury St Edmunds was an example. The carcase was markedly anaemic and cranioventral pulmonary consolidation was present affecting about 15% of the lungs, no viral or bacterial pathogens were identified although the recent antimicrobial treatment may have affected culture results. One can speculate that earlier *Haemophilus parasuis* infection caused a period of inappetance and predisposed to the gastric ulceration in the pig and a submission with a similar history had been reported the previous month. Submission of further deaths would be worthwhile and these cases will be reviewed to see if there are any common features.

**Coccidiosis with *Salmonella* causing diarrhoea and death in replacement gilts**
A problem of diarrhoea and some deaths was reported in batches of nine-month-old replacement gilts after arrival on a commercial outdoor breeding unit. Six gilts had died in the last six batches delivered, deaths occurring around nine days after arrival. Two gilts were submitted to Starcross and both had a severe extensive necrotic enteritis (figure 4) particularly affecting the distal small intestine and proximal large intestine.
Coccidial counts of 6200 and 2300 oocysts/gram were detected in caecal contents and *Salmonella* Reading was isolated from the intestinal contents of one. Intestinal histopathology confirmed coccidia as the main cause of the necrotic enteritis and there was no evidence of involvement of *Brachyspira* species. Coccidiosis at this age is unusual in pigs and involves certain pathogenic *Eimeria* species. When it does occur, it is typically seen in a similar scenario to that present in this case; in replacement breeding pigs soon after moving onto commercial pig units. This is likely to be due to their exposure to a contaminated environment soon after arrival and has been described previously (Gaudie and others 2005). Outdoor training paddocks into which replacement breeding gilts are introduced on arrival can be the source of infection if used long-term.

**Systemic Disease**

**PCV2-associated myocarditis in preweaned pigs following disease in sows:** Swabs for culture and fixed hearts for histopathology were submitted from two pigs as part of an investigation into sudden deaths prior to weaning in three-week-old piglets. On-farm postmortem examinations revealed lesions suggestive of septicaemia and *Streptococcus suis* was cultured from one pig. In the second pig, histopathology revealed severe chronic active nonsuppurative myocarditis and significant labelling of PCV2 antigen by immunohistochemistry, consistent PCV2-associated myocarditis. The pig was the progeny of sows affected by PCV2-associated fetopathy described above and had been from a batch affected at birth (25% mummies and stillbirths), the chronic nature of the heart lesions suggested that the disease in this pig was residual from in-utero infection although it was not possible to determine the timing of the myocarditis lesions.

**Urinary Disease**

**Smallholder sow with pyelonephritis:** A sow was submitted from a smallholding with nine sows and one boar. She was weaned three weeks...
earlier and had died following three days lethargy and anorexia despite antibiotic and anti-inflammatory treatment. Severe chronic bilateral pyelonephritis was found from which *Escherichia coli* was isolated mixed with a *Streptococcus* species from some sites but in heavy pure growth from one – the *E. coli* was considered likely to be the significant clinical isolate and is one of the main pathogens involved in urinary tract infections in pigs. The chronic nature of the lesions indicated disease had been ongoing for several weeks and it is important that sows, especially farrowing/lactating sows are encouraged to get up to drink and urinate at regular intervals. Ensuring good hygiene and access to clean water at all times was also advised. Pyelonephritis is much less common now on commercial units than in the past.

**MISCELLANEOUS EXOTIC AND FARMED SPECIES**

**Bronchioloalveolar carcinoma in alpaca**: A 6 year old Huacaya alpaca was presented to the Royal Veterinary College with a 6 month history of increased respiratory rate which was non responsive to symptomatic treatment. Skin and serological tests for bovine TB were negative. Thoracic radiography showed a diffuse miliary interstitial pattern predominantly in the caudo-dorsal lung lobes. Clinical signs progressed to include inappetance and weight loss and the animal was euthanased. On postmortem examination, the lungs were pale, firm with a mottled white/pink surface and numerous white military foci throughout. The caudal lung lobes were most severely affected and were grey and glistening. Associated lymph nodes were generally unremarkable. Although no acid fast organisms were detected on examination of ZN stained smears of the lung the possibility of tuberculosis could not be fully ruled out and consequently the case was discussed with the local Animal Health Office. Histopathology identified a slow growing, multifocal bronchioloalveolar carcinoma and a diffuse, moderate, suppurative and histiocytic bronchopneumonia: the latter was likely to be secondary to the neoplasia. Immunohistochemistry was carried out at Moredun Research Institute and was negative for Jaagsiekte sheep retrovirus (JSRV), the causative agent of Jaagsiekte/Ovine Pulmonary Adenocarcinoma in sheep. A review of the Veterinary Investigation Diagnosis Analysis (VIDA) data over the last 10 years did not identify any similar cases. Although unusual, this diagnosis along with chronic infectious conditions such as tuberculosis should be included as a possible differential diagnosis in alpaca with chronic respiratory disease.

**BIRDS**

**Broilers**

**Spinal abscess**: Chronic spondylitis (“spinal abscess”) was seen in a submission of 33-day-old broilers with a history of lameness. Postmortem examination revealed large ventral swellings of the spine centred on the body of the free thoracic vertebra (“T4”), in all five birds examined. Bacterial cultures produced good growths of *Enterococcus*–like colonies with
biochemical features consistent with *Enterococcus cecorum* from the spinal lesions. *E. cecorum* is recognised as a cause of vertebral abscesses in broilers, and infection is likely to have arisen as a result of environmental contamination with this organism.

**Commercial Layers**

**Mortality in layers:** Grumbling mortality in a flock of 4,500 19-week-old point of lay pullets prompted the submission of five birds. Two to three birds had been found dead each day for a week, which then increased to seven. Affected birds were initially seen dull with ruffled feathers. They had moved onto the site three weeks previously. At postmortem examination all birds were found to have fibrinous peritonitis and enlarged spleens, and three birds had fibrinous pericarditis. The ovaries were active. The findings were suggestive of *E. coli* septicaemia, which was confirmed bacteriologically. It was thought the stress of coming into lay may have precipitated disease in the birds.

**Erysipelas:** Mortality was reported in a group of 3,500 free range 51-week-old layers in a mobile unit. Two other houses on the premises were unaffected. Over a four day period there had been mortality of 30, 17, 16 and 16 birds. The birds had been permanently housed for the last three weeks. Feather wear was marked and red mite (*Dermanyssus gallinae*) was known to be an issue. Egg production figures were not available and no clinical signs were described in the flock in general. The predominant findings in the dead birds were enlarged spleens, small amounts of pericardial fluid and discolouration or haemorrhage of some of the ovarian follicles. Bacterial culture produced heavy pure growths of *Erysipelothrix rhusiopathiae* from the liver and follicle samples confirming a diagnosis of erysipelas in the birds. Red mite is suspected to act as a reservoir for erysipelas and may have contributed to the transmission of the disease if the organism was present in the environment.

**Backyard**

**Marek’s disease and helminth infection:** Marek’s disease with concurrent *Davainea proglottina* and *Capillaria* sp. infection was diagnosed in a 26-week-old point of lay pullet from a group of 17 homebred birds, part of a backyard flock. Four of the group had died with a further two showing clinical signs including weight loss, going off legs, abnormal head and wing position and death within ten days. At postmortem examination intestinal smears detected very large numbers of the pathogenic poultry tapeworm *Davainea proglottina* in the duodenum and moderate numbers of *Capillaria* sp. nematode eggs. Marek’s disease was suspected from the clinical history and the diagnosis was confirmed by histopathological examination of brachial and sciatic nerves, which exhibited a typical mild focal lymphocytic neuritis. It is possible that the immunosuppressive effects of Marek’s disease may have contributed
indirectly to the heavy tapeworm and roundworm burden, but the reverse may also have occurred and a heavy helminth burden may have contributed to the development of clinical signs of Marek’s disease in birds infected with the virus. Under commercial conditions, vaccination would have been used to control Marek’s disease.

**Game birds**

*Respiratory disease*

**Respiratory disease in pheasants:** An investigation was undertaken into an ongoing problem of respiratory disease in adult pheasants early in the breeding season. Ten birds were examined, three of which showed swelling of the infra-orbital sinus adjacent to the eye, with turbid material evident within the sinuses found on postmortem examination. No evidence was found of *Mycoplasma gallisepticum* involvement by serology or DGGE testing; although *M. gallinaceum* was detected in pooled samples, the role of this organism as a pathogen in respiratory disease in game birds is uncertain. *Pasteurella*-like organisms were isolated on culture from the sinuses of two birds, and may have played an opportunistic role. However PCR testing was positive for avian metapneumovirus type B (aMPV; formerly turkey rhinotracheitis, TRT) in two of the birds suggesting the possible involvement of this agent. AMPV is recognized as a playing a primary role in some outbreaks of respiratory disease in pheasants, and is likely have been of significance in this case.

**References**


This summary is produced by the APHA and is drawn from reports provided at the time of reporting by the APHA laboratories at Bury St Edmunds, Carmarthen, Lasswade, Penrith, Shrewsbury, Starcross, Sutton Bonington, Thirsk and Weybridge and partner external postmortem providers to APHA (University of Bristol School of Veterinary Sciences, Royal Veterinary College, SAC Consulting Veterinary Services St Boswells). APHA monthly reports are available online at [https://www.gov.uk/government/publications/disease-surveillance-reports-2015](https://www.gov.uk/government/publications/disease-surveillance-reports-2015)