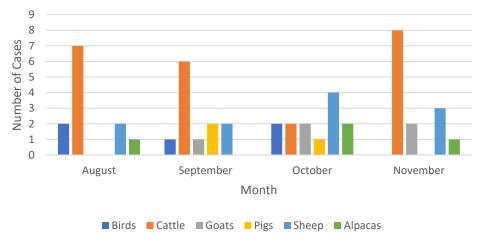


UoS VPC SURVEILLANCE QUARTERLY DIGEST

December 2022

Autumn overview

Autumn Surveillance cases at University of Surrey Veterinary Pathology Centre (UoS-VPC)



Meet the pathologists Josue Díaz Delgado

Hi! My name is Josué Díaz Delgado. I am a veterinary pathologist working as a teaching fellow (years 1, 2, 3 and 5) and as the director of the veterinary anatomic pathology residency (VAPR) program at the University of Surrey (UoS). Half-way through my degree in veterinary medicine (some time ago...), I decided I wanted to become a pathologist so... I conducted a masters on swine respiratory disease, a PhD and a postdoc on pathology of cetaceans, and a VAPR at Texas A&M



University. Shortly after, I worked for the Brazilian National Surveillance Program for Arboviruses. Before moving to UoS, I worked as a full-time diagnostic pathologist in Texas. My specialities/areas of interest are diagnostics and research on pathology of wildlife (with emphasis in marine organisms), zoo and exotic animals, as well as infectious diseases and their impact on public health.



Inside this issue:

- Case submissions summary, Autumn 2022
- Meet the pathologists
- Case Highlights
 - Sheep: Erysipelas
 - $_{\odot}~$ Cattle: Yew Toxicity
 - Cattle: Acorn toxicity

HOLIDAY SCHEDULE

All post mortem examination submissions will be received <u>up to 3 pm on Friday 23rd</u> <u>December 2022.</u>

The Centre will be closed from Monday 26th December thru Monday 2nd January 2023.

During this time, please refer cases to APHA Starcross Tel: 03000 600020

The VPC will reopen on Tuesday 3rd January at 9 a.m.

Best wishes to all in the year ahead!



Lab location:

Veterinary Pathology Centre School of Veterinary Medicine Francis Crick Road Guildford, GU2 7AQ tel: 01483 689 823 email: <u>vetpath@surrey.ac.uk</u>

LETTER FROM THE EDITOR

Greetings to the wider APHA community! We've had a busy fall here at the University of Surrey, Veterinary Pathology Centre and have an almost entirely new team of dedicated pathologists.

We are looking forward to the new year when we plan to add two additional new pathologists and a new resident to the growing team.

Our commitment to providing the highest quality diagnostic services to clients, as well as training services for final year veterinary students and postgraduate residents remains steadfast.

We strive to improve the services we provide to our referring practices, so should you have any comments or suggestions, please do not hesitate to contact us: email: <u>vetpath@surrey.ac.uk</u> tel: 01483 689 823



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Sai Fingerhood

Sai* Fingerhood (they/them) moved to this beautiful island nation and began work at the UoS-VPC in late August, 2022 (just in time for blackberry season!). Their educational background is quite varied, graduating from Princeton University with a degree in Anthropology and Dance in 2013 and proceeding to enter veterinary school at the University of California, Davis in 2015. During their final year of veterinary school they externed at multiple veterinary pathology diagnostic labs across the continental United States (WADDL, CSU VDL, the OSU CDL, JHU



MCP) and through that gained insight into the wealth of different ways labs can successfully run. After graduating from veterinary school (2019) they stayed in Davis for a residency in Anatomic Pathology, ending in July 2022. During their residency they worked closely with the UCD orphan kitten project and completed a research project characterizing *Clostridium piliforme* in orphaned kittens (in review). Their pathology-related areas of interest include diseases of production and intensively housed animals, as well as more broadly, the human-animal interface and the diseases that arise within this socio-ecological niche. They are interested in the multitude of ways humans can learn from one another and from the animals and environment around them; they are acutely aware of the important role educators have in helping to shape not only future veterinarians, but also community members.

*pronounced "say"

Cecilia Gola

Cecilia graduated from the University of Turin, Italy, with her degree in veterinary medicine in 2015. She began to develop an interest in pathology while undertaking her master's research component and has been highly committed in pursuing her training in this field ever since. After graduating, Cecilia worked in clinical practice and concurrently became a visiting graduate researcher at the University of Zurich and IZSTo. Her interest in diagnostic and research oncologic pathology progressed to the next level when she returned to the University



of Turin to pursue her PhD from 2018 to 2021, focusing on the molecular mechanisms of canine osteosarcoma. Contextually to her PhD, she decided to start her ACVP residency training in anatomic pathology along side her research activities and passed ACVP Phase I examination in April 2021. Cecilia joined the Veterinary Pathology Centre at the University of Surrey as a Resident in Anatomic Pathology in November 2021 and is aiming to sit ACVP Phase II examination in later this year in August.

Other members of the team:

<u>Kate English</u> is a clinical pathologist and our fearless section leader, working tirelessly behind the scenes. <u>Sarhad Alnajjar</u> is a lecturer and researcher, who you will hear more about come lambing season!





Fig. 1 Rumen filled with leaves and stems of *T. baccata*



Fig. 2 Heart with multifocal, acute presumptive myocardial necrosis and haemorrhage

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Yew toxicity in cattle

Here we report pathological data on four fatalities associated with common yew (*Taxus baccata*) toxicity in Highland cattle. Specifically, two outbreaks of sudden death involving a total of eight animals from Hampshire are documented.

A 3.5-year-old cow from an outdoor rearing farm from a group of 30 (total heads: 70) was submitted for autopsy to the UoS-VPC. Some were found dead in the morning while others were seen keeling over, gasping for breath and dying very quickly. Deaths were only recorded in that group. Of note, this home bred group had recently broken out and had nibbled a yew tree; some ivy growing nearby also. These animals were not vaccinated and not treated; no previous diagnoses were readily available. Anthrax test was negative.

The steers were three (3-year-old) animals, from a group of six, that had died over a 24h period. Two steers were found dead in the morning; one was still alive, convulsing, expelling ruminal content from the nose and mouth, and exhibited severe abdominal bloat. This steer was initially treated with closamectin and euthanised shortly after. These animals had been moved from grassland to a new site two days prior to death. No previous diagnoses were readily available. All three steer were submitted for autopsy at VPC-UoS. On post-mortem examination, in the cow, the main gross pathological findings were confined to the gastrointestinal and respiratory systems and included a distended rumen with abundant complete and masticated leaves and stems of *T. baccata* (Fig.1) and small quantities in the remaining forestomachs and proximal small intestine, as well as and ruminal acidosis (pH 5.0), and moderate, diffuse pulmonary congestion primarily of the cranioventral lobes. Similarly, in the steers, the main gross pathological findings were ruminal distension with mild to moderate amount of T. baccata and Hedera helix content (Fig.1 inset). The steers also exhibited multifocal, acute presumptive myocardial necrosis and haemorrhage (Fig.2).

Based on the epidemiological, clinical and gross pathological findings, these cases were consistent with common yew (*Taxus baccata*) poisoning. Yew poisoning has been recognised in livestock and humans for hundreds of years. In ruminants, microbial degradation of taxines in the rumen is considered important. Yew plants contain a heterogenous mixture of cardiotoxic alkaloids; the major constituents being taxine A and taxine B. Taxine B and its derivatives constitute the main and most toxic group. Taxines act as calcium and sodium channel antagonists within cardiac myocytes and may induce cardiac arrythmia, atrioventricular block and diastolic cardiac arrest.⁷ Death through acute heart failure normally occurs in less than 24 hours. However, in cattle a subacute to chronic intoxication lasting up to 18 days has been reported.⁴ In such instances, remaining animals in the herd who might have had access to *Taxus* spp. should be carefully monitored. Careful examination of common grazing should be performed, and appropriate fencing and avoidance of contaminated areas should be encouraged.

The authors acknowledge Dr. Nicola Parry and Dr. Marvin Firth for their contributions to these cases.



Avian influenza (AI)

Currently all of Great Britain is in an avian influenza prevention zone. This means birdkeepers must follow strict biosecurity rules to prevent the spread of AI.



APHA Interactive Avian Influenza Disease Map



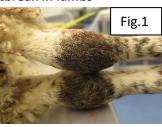
Follow this QR code for updates and guidance on AI in Great Britain.



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Erysipelothrix rhusiopathiae polyarthritis outbreak in lambs

In late September, 2022, UoS-VPC received report of an outbreak of swollen joints in post-weaned, 4-month-old sheep. Out of a group of 40 sheep, within a herd of 900 total animals, 30 of the animals were affected. These animals had been treated intermittently with Penicillin-streptomycin.



Three of the affected group were submitted for post-mortem examination. The tarsal and carpal joints were variably mildly to moderately swollen in all of these animals (Fig.1). One of the sheep demonstrated a severe fibrinosuppurative polyarthritis and tenosynovitis with extensive eburnation, while the other two sheep demonstrated mild, chronic, erosive polyarthritis (Fig.2). Cytology of the joint fluid confirmed a suppurative and histiocytic arthritis. A swab from the tarsal joint of the most severely affected animal grew a few small colonies of *Erysipelothrix rhusiopathiae*.



Histopathology confirmed a severe neutrophilic and lymphohistiocytic arthritis and tenosynovitis with extensive cartilage loss and underlying osteomyelitis.

E. rhusiopathiae is a ubiquitous, Grampositive, short rod-shaped bacterium that is widespread in distribution and hardy in the environment. It is a known, common cause of polyarthritis in sheep (typically 2- to 6-month old lambs), though the most common reservoir for the bacteria is swine, with 30% to 50% of healthy pigs harbouring the bacteria in lymphoid tissue.² Routes of infection include

cutaneous wounds (docking, shearing, injections), oral ingestion, and umbilical infections. In this outbreak no cutaneous lesions were present, but there was enteritis and colitis associated with intestinal parasitism; speculatively we wonder if this could have predisposed these animals to dissemination of the bacteria through ingestion.

In a recent review of arthritis in sheep covering the VIDA database from 2002-2020, *E. rhusiopathiae* was considered the aetiology in 11.8% of the cases (n=1,085).⁶ As in this case, the majority of sheep in the review were post-weaned, whereas the majority of neonatal and pre-weaned ovine polyarthritis cases were attributed to *Streptococcus dysgalactiae*. Despite the reported antimicrobial susceptibility profile including susceptibility to penicillin and erythromycin in this case, it is of note that this herd did not respond to the Pen-strep antibiotic administration. Recommendations for prevention of bacterial polyarthritis in lambs include maintaining proper hygiene during injections as well as within lambing pens, dipping facilities, and handling pens, monitoring trace element and vitamin levels to ensure optimal immune function, dipping navels in strong iodine solution at birth and four hours later, ensuring adequate colostrum intake, and monitoring tick burdens.⁶



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Contributors

Josue Díaz Delgado • Yew toxicity in cattle Sai Fingerhood

- Editor
- Erysipelothrix in lambs
- Acorn toxicity in cattle



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Acorn toxicity in cattle

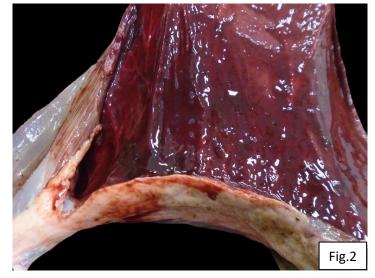
It is a mast-year for many of the seed and nut producing trees in the UK including oak trees (*Quercus robur* and *Q. petraea*), which means that large bumper crops of acorns have become available for consumption by grazing animals.^{5,8} We have seen evidence of this at the UoS-VPC, with both



surveillance and private cases of acorn toxicity submitted during the last three months.

One of these cases consisted of an outbreak of 5 deaths in a herd of 35 finisher Dexter cows coming from a farm in the Surrey-Hampshire area. Deceased animals were reported to have blood seeping from the oral cavity and rectum at the time of death; anthrax testing was pursued and was negative. One of the cows was submitted for post-mortem examination.

Pertinent gross post-mortem findings included moderate numbers of acorns within the rumen (Fig.1), marked haemorrhagic typhlocolitis with abundant intraluminal clotted blood



(Fig.2), a congested bloody spleen, and renal mineralization and tubular necrosis. Despite considerable autolysis, histopathology confirmed the presence of acute and chronic renal tubulointerstitial disease, characterized by acute tubular necrosis with intratubular haemorrhage, cellular casts, lymphoplasmacytic interstitial nephritis and interstitial fibrosis. The chronic renal changes are suggestive of chronic and acute toxicity, as has been described in previous reports of acorn intoxication.³

The mechanism of oak toxicity is complex, with the toxic principle being gallotannins. These tannins are hydrolyzed into metabolites that include tannic acid, pyrogallol, and gallic acid.¹ The lesions are thought to occur due to these active metabolites binding to endothelial cells, resulting in endothelial damage, leading to nephrotoxicity, and the oedema typical of this disease. The pathogenesis of alimentary lesions is putatively due to precipitation of proteins from the binding of gallotannins to peptide bonds, in addition to being a sequelae of disseminated intravascular coagulation.¹