UoS VPC SURVEILLANCE QUARTERLY DIGEST

April 2023

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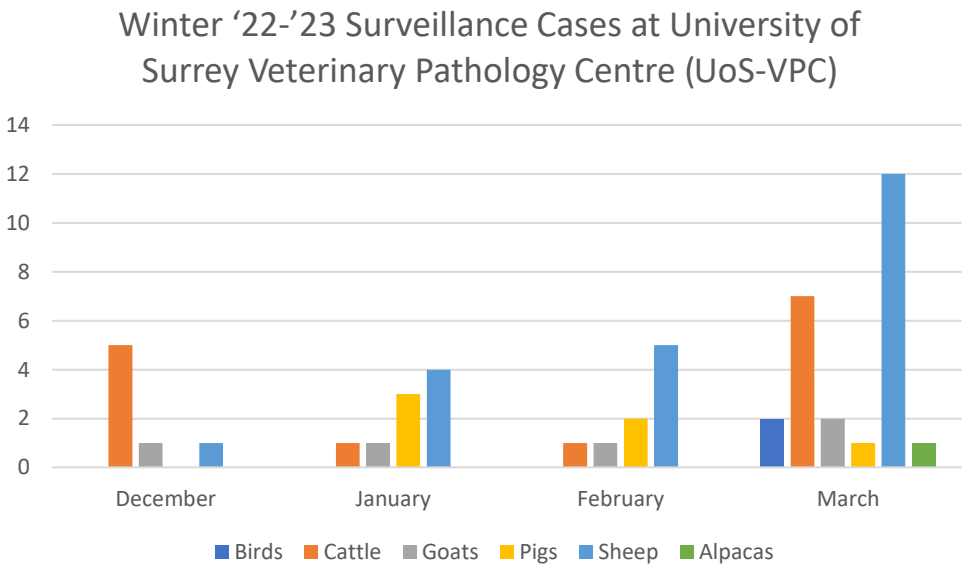
Winter overview



Meet the pathologists

Marta Hernandez Perez

Marta graduated from the University of Las Palmas de Gran Canaria (ULPGC), Spain, in 2015. While doing an externship in the pathology group studying causes of death in stranded marine mammals in the Canary Islands, she developed great interest in veterinary pathology, and gained a training scholarship to work at the Biopsy Service of the ULPGC for 2.5 years. She then moved to Scotland to complete her Residency in Anatomic Pathology in November 2018 at the University of Glasgow, where she stayed working as lecturer/pathology clinician for almost a year. Marta joined the Veterinary Pathology Centre at the University of Surrey in February 2023, and her specific areas of interest are infectious diseases in small animals and neoplasia.

Lab location:

Veterinary Pathology Centre
School of Veterinary Medicine
Francis Crick Road
Guildford, GU2 7AQ

LETTER FROM THE EDITOR

Greetings to the wider APHA community! We've had an exciting winter and have recently welcomed two amazing new pathologists and one wonderful new pathology resident to the team!

As you can see from the growing number of sheep submissions in our winter overview, it's now full on lambing season!

Hopefully you'll find the content of the newsletter interesting, educational, and relevant. If you want to get some hands on pathology experience yourselves, you can join us at our CPD event in May (see final page!).

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: vetpath@surrey.ac.uk
tel: 01483 689 823

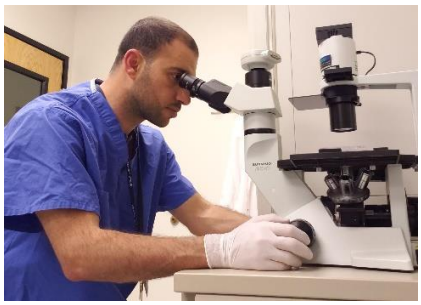
Javier Marrero

Hello! My name is Javier Deniz Marrero. Board eligible veterinary pathologist who has recently joined the University of Surrey Veterinary Pathology team. I started my veterinary studies in 2012 and halfway through, I realised that veterinary pathology was the area I gravitated to the most within the veterinary world. After finishing my Veterinary medicine degree in 2017 I decided to broaden my veterinary pathology knowledge by doing a "short" externship at the University of Utrecht in The Netherlands. This short stay ended up being a one year Veterinary Pathology internship and a 3 years veterinary pathology residency. After finishing my residency in 2021, I decided to take a year to enjoy my main hobby, rock climbing, as well as travel a bit through Europe by car and relax in my hometown in Gran Canaria. I will be sitting my ECVP exams next year. My interests within the veterinary pathology field are associated with the cardiovascular and respiratory system. Additionally, I would also like to broaden my expertise in fish pathology.

Pablo Diaz Santana

Hi everybody! My name is Pablo Diaz Santana, I am a graduated Veterinarian from Las Palmas of Gran Canaria, Spain. During the degree I took the chance of trying different areas of this profession, as well as studying in other Universities like The Autonomous University of Barcelona (UAB), finally focusing and developing a big interest in wildlife conservation, infectious diseases, pathology, and One Health approach. After finishing my degree in 2018, I started a PhD at the University Institute of Animal Health focusing in pathologies and causes of death in cetaceans stranded in the Canary Islands. This research, which is in its final stage, allowed me to expand my interest in the pathology field and perform various externships at the Pathobiology department of the University of Utrecht. Consequently, and once the path in my professional career was clarified, I have recently joined the pathology team (March 2023) at the UoS as a pathology resident aiming to sit the ACVP board exam. Pathology is a multidisciplinary tool which allows the unification of human and animal health and that represents a key point to prevent, describe, and solve future global problems due to climate instability and imbalanced anthropogenic activities. My passion concerning pathology coexist with the never-ending desire of meeting new cultures around the world and the eternal searching of new melodies and connections while playing guitar, which keep the balance in between all parts.

UoS Research Focus: Sarhad Alnajjar and Respiratory Syncytial Virus



Sarhad Alnajjar is a lecturer in veterinary pathology at UoS and is also part of Lambcure, LLC (a USA-based company), which facilitates research and development of preclinical safety and efficacy trials using a lamb model for respiratory syncytial virus (RSV) infections. Collaboration between researchers in different institutions worldwide is critical for research development and outcome. There are several models of RSV infection, but neonatal lambs were established years ago and appropriately mimic the RSV infection of human infants and provide a good model to evaluate the efficacy of anti-RSV therapeutics. In the end, our hope is to find a cure for RSV and have a world with happier healthier children.

Lungs of a lamb with diffuse interstitial pneumonia, associated with RSV infection. Picture Credit: S. Alnajjar

Human Respiratory Syncytial Virus (hRSV) is a common cause of respiratory infection with transient flu like symptoms in immunocompetent adults. However, in children under 5 years old and older adults >65 years old, RSV causes severe lower respiratory tract infections and hospitalization. Most children get infected with RSV by the age of 5 years. Research indicates that more than 57000 children are hospitalized annually due to RSV-associated illness, which is associated with many deaths, mostly in developing nations. Additionally, developing severe RSV infection has been linked to further respiratory conditions in adult life like asthma and COPD.

Until now, there was no vaccine for RSV, and very limited therapeutic choices available. Advances in vaccine development brought by Covid pandemic have facilitated the development of some vaccine strategies that are under testing. Treatment options are limited to one antibody which, in addition to being pricy, has limited therapeutic benefits and is given as prophylactic for highly susceptible patients such as infants born preterm or organ transplant patients.

Within veterinary species, bovine RSV (bRSV) is one of the primary agents of the bovine respiratory disease complex, along with other viruses such as PI-3, BoHV-1, BVDv and common bacterial agents (e.g. *Mannheimia haemolytica, Pasteurella multocida*, *Histophilus somni, Mycoplasma bovis*); calf infections have a significant welfare and economic impact within the global cattle industry. Vaccines that reduce bRSV clinical symptoms exist and are widely in use; research into vaccines that may be effective in eliminating viral circulation is ongoing.[1]

RSV is similar to other respiratory viruses and is transmitted by sneezing and coughing and direct contact. First, the virus gains access through the nose and replicates in the nasal sinuses and nasopharynx, mainly the upper respiratory tract causing bronchitis, sinusitis, nasal discharge, and congestion that persist for up to 14 days. In some individuals, the infection persists longer period and moves to the lung leading to severe bronchiolitis and pneumonia with devastating impacts on the patient, families, and health system. The huge impact of RSV across species has motivated academic institutions, pharma companies, and governments to continue research developing possible vaccines and treatments.

REFERENCE

1. Hägglund S, Näslund K, Svensson A, et al. Longitudinal study of the immune response and memory following natural bovine respiratory syncytial virus infections in cattle of different age. Abuelo A, ed. *PLOS ONE*. 2022;17(9):e0274332.

PRRSV-1 and *Streptococcus suis–Klebsiella pneumoniae pneumoniae* meningoencephalitis in piglets

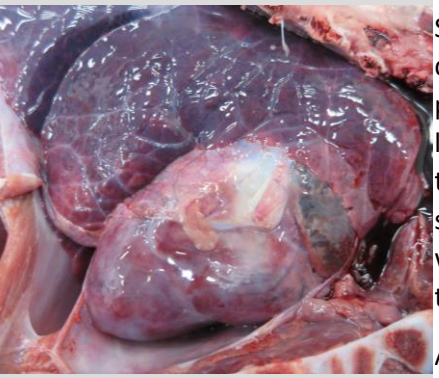


Fig. 1 Interstitial pneumonia and pulmonary oedema, pericarditis and epicarditis.

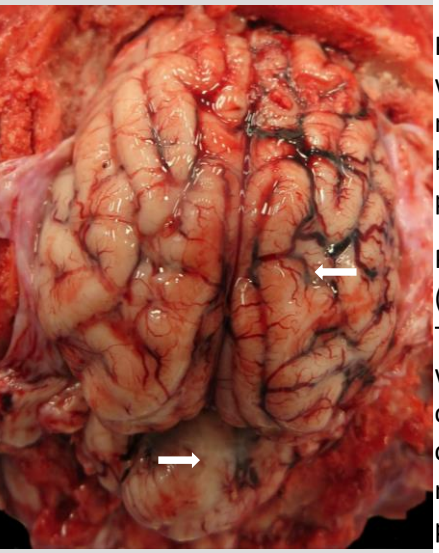


Fig. 2 Suppurative meningitis. Note the opaque meninges with variable amounts of exudate (arrows).

Sixteen piglets from a 1200 head group of pre-weaned piglets (10-12 weeks-old) died over the course of a couple weeks with no premonitory signs. Two of these piglets were submitted to the UoS-VPC for *post-mortem* examination. Major gross lesions included diffusely rubbery and dark red to purple lungs, pericardial thickening and opaque pericardial exudate, (Fig. 1), enlarged, firm, and dark red submandibular lymph nodes, and diffusely cloudy meninges (Fig. 2), the latter of which was consistent with suppurative meningitis (confirmed via touch imprints of the meninges).

Ancillary testing included lung bacterial culture (including *Mycoplasma* spp), Influenza A and porcine reproductive and respiratory syndrome virus (PRRSV) RT-PCR on lung, and bacterial culture of brain tissue. Within the respiratory tissue, PRRSV-1 was amplified. *Streptococcus suis* serotype 2 (SS2) and *Klebsiella pneumoniae pneumoniae* (Kpp) were cultured from the brain. The SS2 was resistant to tetracyclines and the Kpp was resistant to ampicillin and clindamycin.

Histologically, lymphohistiocytic interstitial pneumonia was confirmed, consistent with lesions expected in PRRSV infections. Additionally, a severe fibrinosuppurative meningoencephalitis was present in both pigs. Interestingly, only Gram-negative bacteria were associated with the meningitis, despite the culture of both Gram-positive (SS2) an Gram-negative *(*Kpp) bacteria.

PPRSV strains are divided into two distinct species based on genetic makeup (genotypes), PRRSV-1 (European strains) and PRRSV-2 (North American strains).[3] This virus can result in massive economic losses. Respiratory disease associated with the virus is one of the most common diagnoses in pigs submitted for diagnostic casework in the UK.[1] The common mode of transmission is direct nasal, oral, or coital contract. The economic impact of the virus is in part attributable to the reproductive losses associated with the virus; these occur due to endometritis, placentitis, and foetal infections of late-gestation sows which can result in weak neonates and abortions. Part of the devastating effects of the virus can be attributed to systemic immune suppression that accompanies infection, resulting in susceptibility to secondary infections, as may have been the case with these animals.

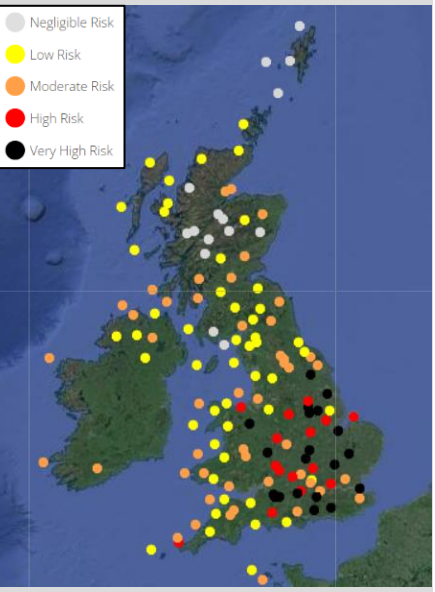
The viral damage to the lungs in addition to systemic immune suppression likely predisposed this animal to opportunistic infections resulting in the multi-bacterial meningoencephalitis. SS2 is a commensal inhabitant of the respiratory tract, and Kpp is a commensal of the intestinal tract; both bacteria can be present without associated disease, however virulent strains of both of these bacteria have been identified and are associated with increased pathogenicity. SS2 is a well known and prevalent cause of polyserositis and valvular endocarditis in pigs. Kpp infections can result in pneumonia, septicaemia, and occasionally meningitis in many species, including humans, where it is typically nosocomial. Kpp appears to be an emerging pathogen in pigs, strain ST25, in particular, has been identified in seasonal outbreaks of septicaemia in piglets in England.[2]

REFERENCES

1. APHA. GB Pig Disease Surveillance Dashboard. *Tableau Software*. 2023:
2. Bidewell C, Williamson S, Rogers J, et al. Emergence of Klebsiella pneumoniae subspecies pneumoniae as a cause of septicaemia in pigs in England. *PLOS ONE*. 2018;**13**:e0191958.
3. Wei C, Dai A, Fan J, et al. Efficacy of Type 2 PRRSV vaccine against challenge with the Chinese lineage 1 (NADC30-like) PRRSVs in pigs. *Sci Rep*. 2019;**9**(1):10781.

Nematodirus forecast

- Multiple areas in Southern England are currently at high risk of Nematodirus infections.
- Hatching of the larvae of this nematode is dependent on climatic conditions.
- The SCOPS forecasting map can help determine when a flock might be most at risk.



SCOPS Nematodirus Forecast Map

Follow this QR code to access the SCOPS Nematodirus Forecast

Mitral valve defect in a piglet mimicking polyserositis

A 3 week-old, male piglet from a litter of 10 died after exhibiting an increased respiratory rate, decreased appetite, and a normal body temperature. Two other piglets from the litter had died 1 week prior, with no conclusive cause of death found.

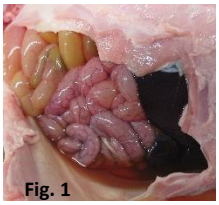


Fig. 1


Fig. 2

Gross *post-mortem* examination revealed moderate amounts of translucent, orange-tinged, watery fluid within the peritoneal cavity (Fig. 1), along with lightly adhered strings of fibrin (Fig. 2). Similar fluid and fibrin was present within the thoracic cavity and attached to the pleural surfaces.


Fig. 3

The heart had a globoid profile, with marked dilation of the left auricle and atrium (arrow in Fig. 3). The epicardium was diffusely, firmly adhered to the pericardium, and when the two layers were separated, the epicardial layer was roughened and irregular (Fig. 3); this was interpreted as fibrous adhesions and mesothelial hyperplasia that was confirmed histologically.

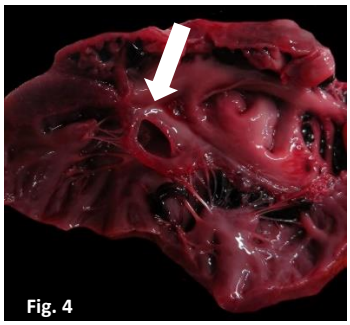


Fig. 4

The central portion of the aortic leaflet of the mitral valve contained a well demarcated, round (5 mm diameter) defect with mildly thickened edges (Fig. 4, arrow). Opposite the defect in the mitral valve, the left atrial endocardium was regionally roughened and thickened (Fig. 5, arrows), consistent with jet lesions from regurgitation of blood through the valve defect.

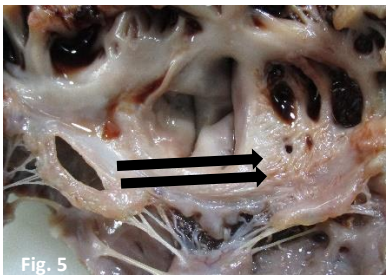


Fig. 5

Based on gross examination, an infectious cause for tricavitary fibrinous polyserositis was suspected, and culture of a swab of the thoracic pleura was pursued. However, histologically, an inflammatory process that could explain the tricavitary process was not present and all ancillary testing for infectious diseases were negative. Based on this, we concluded that the tricavitary fibrinous effusion with chronic fibrous pericarditis likely occurred due to chronic left sided heart failure which led to biventricular failure due to the congenital mitral valve defect. The severity of the fibrinous effusion (presumed transudate) and the chronic epicardial adhesions were impressive, and a reminder to the pathologists on the case that the presence of fibrin does not always equate to the presence of infectious agents.

The mitral valve defect described above is an unusual cardiac anomaly in any species, and has not been reported in pigs, to our knowledge. Embryologically, the atrioventricular valves develop from the endocardial cushions, and an abnormality in this process could have led to the defect; the chronicity of the changes are suggestive of a congenital process, as opposed to an acquired one, given the age of this animal, though lesions like this have been described in cases of resolved bacterial valvular endocarditis. More commonly reported congenital cardiac anomalies in pigs include tricuspid valve dysplasia, atrial septal defects, and subaortic stenosis.[1] As in this case, clinically significant cardiac anomalies are often identified in animals less than 2 months of age.

Thanks for reading!

Newsletter Contributors

Sai Fingerhood
- Editor
- Pig cases

Sarhad Alnajjar
- Research Focus: RSV

REFERENCE for *Mitral valve defect in a piglet*

1. Hsu FS, Du SJ. Congenital Heart Diseases in Swine. *Vet Pathol*. 1982;**19**(6):676–686.

Veterinary Pathology Farm CPD

Wednesday 3rd May 2023

08:45-16:30

The University of Surrey Veterinary Pathology Centre would like to invite you for a Farm Veterinary Pathology CPD Day.

❖Introduction and review on 'How to conduct a post-mortem'

❖Supervised post-mortem examination

❖Examination of respiratory and gastrointestinal organs

❖Discussion and overview of APHA Surveillance Programme – Fin Twomey: Head of Surveillance Intelligence Unit and Lead Scientist for Scanning Surveillance, AVTRW President.

Limited spaces available!

Register your interest at:
https://www.eventbrite.co.uk/e/veterinary-pathology-farm-cpd-tickets-553367215237

Contact j.kingswell@surrey.ac.uk for further information