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Swine Disease

Abstracts

Moderator:

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Saskatoon

Case #1 **Enrique Aburto, WCVM**

Congenital hyperostosis

Clinical history: One-day-old piglet born with a swollen leg that is hard (almost like a bony callus); farrowing manager had noticed around 15 piglets with similar presentation in the last few months.

Gross examination: A fresh leg and fixed tissues were submitted for histopathology. The skin was previously removed from the limb and the muscles showed several cuts and missing fragments of muscle. The muscles of the limb were very firm with extensive areas of edema and partially replaced by white tissue (fibrosis).

Histopathology: Cross sections of bone from the leg reveal a thick, circumferential layer (up to 5 mm) of radiating woven bone trabeculae extending out from the cortex and elevating the overlying periosteum. The trabeculae are separated by loose myxomatous connective tissue. The periosteum is thickened by dense fibrous tissue, and the inner periosteal margin is lined by multiple layers of osteoblasts separated by variable amounts of osteoid. The reactive periosteum merges and fuses with adjacent areas of edematous or myxomatous connective tissue which contains numerous loosely and haphazardly arranged spindle to stellate cells, separated by abundant pale blue-grey, mucinous matrix. This tissue infiltrates the adjacent skeletal muscle bundles. The myofibers within the muscle bundles are markedly shrunken (atrophy) and often replaced by fibrous tissue. Poorly defined lobules of cartilage and bone trabeculae are occasionally seen within the myxomatous connective tissue.

Morphologic diagnosis: Leg (bones and skeletal muscle): Periosteal woven bone formation (hyperostosis) with edema, fibrosis, and muscle atrophy.

Comments: The gross and histologic findings in the submitted leg are characteristic of congenital hyperostosis (aka congenital porcine cortical hyperostosis, diaphyseal dysplasia, congenital thick foreleg, and “thick legs”). The pathogenesis of this condition is unknown. The possibility of an autosomal recessive trait has been proposed but not confirmed. It has also been suggested that local circulatory disturbances of the limbs due to abnormal positioning of the fetus in the uterus leads to chronic edema and extensive periosteal bone proliferation. Arteriosclerotic (hypertensive) vascular lesions have been described in the radioulnar region of affected piglets. Some aspects of congenital hyperostosis of pigs resemble those of Caffey’s disease (Infantile cortical hyperostosis) in children.

References:

Linden E. Craig, Keren E. Dittmer, and Keith G. Thompson: Bones and Joints. In Jubb, Kennedy and Palmer’s Pathology of Domestic Animals, Vol. 1, 6th ed. Ed M. Grant Maxie, Elsevier, St. Louis, Mo, 2016, p. 53.

Shanika Kurukulasuriya and Edward Clark, WCVM

Case # 2

Malignant melanoma, with metastasis to regional lymph nodes

Gross findings:

Inspection of the carcass of a female Duroc pig of market age (approximately 25 weeks) revealed enlarged (2-3 times their normal size), black colure multiple lymph nodes. A large piece of skin, sub-cutaneous lymph nodes and associated fat and muscles were submitted. The skin was dark grey to black with a nodular, 12 X15 cm diameter, plaque like mass. The lymph nodes were various in size and diffusely grey to black.

Histopathology:

Skin: The deep dermis and sub-cutaneous are occupied by variably sized, un-encapsulated, densely cellular, infiltrative, heaps or lobules separated by thick fibrous stroma. The lesion comprises sheets of highly pigmented neoplastic melanocytes which are round to polygonal shape and contain distinct cell margins, moderate amount of cytoplasm with numerous brown, coarse, variable sized round cytoplasmic granules (melanosomes). Nucleus is round with finely reticular chromatin, and round 1-2, purple nucleoli. Anisocytosis and anisokaryosis are moderate (~2-3 folds). No mitotic figures are observed across 10 high power fields (2.37 mm^2).

Lymph node: Lymph node is effaced by (~ 95%) a neoplastic melanocytes population with similar cytological criteria mentioned above.

Diagnosis

Skin: Malignant melanoma (superficial spreading variant)

Lymph node: Metastasis of a malignant melanoma

Ancillary test: IHC staining for CD18 to identify presence of melanophages to demonstrate the potential signs of regression.

Comment

In slaughter pigs, melanocytic tumors most frequently found in Duroc and Duroc crossbreeds. The majority of melanocytic tumors in these breeds are benign, and as in the miniature breeds, they often undergo spontaneous regression which is characterized by massive infiltration of pigmented macrophages and lymphocytes. Additionally, it's common for regional lymph nodes to show black pigmentation which can be associated with the metastatic spread of pigment-producing malignant melanomas or to the presence of pigment-laden macrophages from melanomas that are regressing.

References

Hordlnsky MK, Ruth G, King R. Inheritance of melanocytic tumors in Duroc swine. Journal of Heredity. 1985 Sep 1;76(5):385-6.

Vincent-Naulleau S, Le Chalony C, Leplat JJ, Bouet S, Bailly C, Spatz A, Vielh P, Avril MF, Tricaud Y, Gruand J, Horak V. Clinical and histopathological characterization of cutaneous melanomas in the melanoblastoma-bearing Libechov minipig model. Pigment cell research. 2004 Feb;17(1):24-35.

Case #3 **Kylie Pon, WCVM**

Mycoplasma synovitis in a pig

Signalment: An 18-20 week old, 56 kg, intact, female, porcine.

Herd history: Rear leg lameness in 18-20 week old pigs that is refractory to treatment.

Gross findings: Bilaterally, stifles contained abundant red-orange, opaque, slightly viscous synovial fluid with clumps of friable, red material. The synovium was diffusely red, thick, with fibrillar luminal projections. Around the left stifle, surrounding connective tissues and skeletal muscle had patchy hemorrhage.

Histopathology:
Stifle joints – There is diffuse, moderate to severe, synovial hyperplasia and formation of prominent villus-like projections of synovium into the lumen. The synovial intima is thickened by increased layers of synoviocytes, and within the lumen there are occasional accumulations of fibrin admixed with neutrophils and cellular debris. The synovial subintima is markedly expanded by infiltration of moderate numbers of plasma cells, lymphocytes, fewer macrophages and neutrophils, plump fibroblasts, congested new blood vessels lined by plump endothelial cells, edema, and patchy areas of hemorrhage. Inflammatory cells infiltrate into the synovial intima as well as accumulate in low numbers around blood vessels in underlying adipose and muscular tissues. No bacterial organisms are observed.

Morphologic diagnosis:
Stifle joints: synovitis, fibrinohemorrhagic, proliferative, bilateral, severe, chronic.

Ancillary testing:
1. Synovial fluid bacterial culture: negative
2. *Mycoplasma hyosynoviae* PCR (synovial fluid): **Positive; Ct value = 27.21**

Etiology: *Mycoplasma hyosynoviae*

Comments:
Gross lesions, histopathology, and ancillary tests are consistent with severe chronic synovitis associated with *Mycoplasma hyosynoviae*. *M. hyosynoviae* is one of three pathogenic *Mycoplasma* spp. reported in swine and is associated with polyarthritis in pigs over 10 weeks of age. Other pathogenic Mycoplasma spp. are *M. hyopneumoniae* associated with enzootic pneumonia and *M. hyorhinis* which causes polyserositis and polyarthritis in younger pigs (3-10 weeks of age). Chronic arthritis in pigs is often multifactorial, with management, stress, genetics, and other infectious agents (*Erysipelothrix rhusiopathiae*, previous *Streptococcus* spp. infection, etc.) potentially contributing to clinical disease.

Reference:
Maxie, M. Grant, and Jubb, K. V. F. *Pathology of Domestic Animals*. Vol 1, Chapter 2: Bones and Joints. 6th Ed. Elsevier Saunders, 2015. Pg 148-153.

Case # 4 **Brenda Bryan, VDS, Manitoba**

Piglet Extraintestinal Colisepticemia Bacteremia

Clinical history: Received 2 piglets, 3 days of age from the same litter, with average body weight of 1.68kg. History describes 4 dead piglets overnight.

Gross findings: On necropsy, the skin of all extremities had well delimited, circumferential to lateral areas of purple to dark pink skin extending from the hock or elbow to the foot. Discoloration was also apparent on the ear, multifocally on the face, eyelids and body ventrum. Cut section of the affected skin revealed marked clear to yellow edema and diffuse congestion. There was similar edema in the subcutis and interstitium around the larynx. Claw tips back legs were purple to black.

Histopathology: Skin: superficial, deep dermis and deep and skeletal muscle interstitium diffusely expanded by edema, fibrin, congested dilated vessels, moderate numbers of interstitial, intravascular and perivascular small bacilli 2um long, neutrophils and macrophages, many with intracytoplasmic small bacilli. Few multifocal vessels had partially occlusive luminal fibrin thrombi.
Multiorgan: intravascular small bacilli approximately 2um long in multifocal vessels; lymph nodes, brain, skin, skeletal muscle, peritracheal tissues and may other tissues.
Heart: rare vessels held intravascular pink round hyalinised globules of fibrin (shock bodies).
Liver: Interstitium surrounding gall bladder wall expanded by ectatic lymphatic and multifocal myriad small bacilli. Rare tiny vessels had fibrinoid necrosis.
Lymphoid tissues: tonsil, Peyers' patches, lymph nodes (various) had mild to moderate diffuse lymphoid depletion.
Kidney: in the renal pelvis, myriad small bacilli and mononuclear cells infiltrated the interstitium. Bacilli were around and within vessels, and within the cytoplasm of many mononuclear cells. Multifocal vessels of the renal pelvis had small bacilli, leukocytes with intracytoplasmic bacilli and intravascular fibrin thrombi with partial luminal occlusion. Intravascular bacilli were apparent in the tiny vessels of the cortical interstitium.

Morphological diagnosis: Extra-intestinal Colisepticemia, Bacteremia, Disseminated Intravascular Coagulation (DIC)

1. Cellulitis, multifocal, focally extensive, distal extremities, head, ears, body ventrum, acute, severe with intralesional bacilli, intravascular fibrin thrombi (DIC)
2. Bacteremia, bacilli, multiorgan, intravascular, intracytoplasmic, peracute, cute, severe

Ancillary test:
Bacterial culture: Lung, heart, spleen, skin subcutis: E coli 3+
Brain: E coli 2+

Etiology: *Escherichia coli*

Comments**:** *E. coli* isolated from all culture organs, and microscopic findings of fibrinous polyserositis, bacilli in cytoplasm of leukocytes in many tissues, in vessels, and tissue interstitium was compatible with extra intestinal colisepticemia and bacteremia followed by DIC, and the cause of death of the piglets. *E coli* may enter orally through the intestinal tract, by inhalation or through the umbilicus. Colisepticemia may be associated with lack of colostrum ingestion or colostrum ingested lacks antibodies. Virulent *E coli* strains can develop rapid septicemia and death from endotoxic shock.

References:

1. Zachary JF. Pathologic Basis of Veterinary Diseases, 7th Ed. p104-157, p.171-292.
2. Maxie G. Jubb and Kennedy's Pathology of Domestic Animals. Vol 2 p 166-7.

Case # 5 **Brenda Bryan, VDS, Manitoba**

Cerebrospinal Meningeal (Atlanto-occipital) Iron Injection

Clinical history: Received 2 neonatal piglets with average weight of 776g. History described loss of 40 piglets immediately post injection. First piglet had sudden death post injection of Excede (ceftiofur sterile suspension), and the second piglet had sudden death post iron injection.

Gross findings: Gross exam was initially unrewarding until yellow brown staining was noted of the tissues around the larynx, extending to the chest. Further dissection revealed yellow brown staining in the subcutis behind the right ear, in the interstitium around the atlanto occipital joint. Additionally, there was yellow brown material and staining in the space between the dura and the brain, and in the meninges of the brain and spinal canal.

Histopathology: In the cerebrum, deep in rare sulci, and in rare cerebellar sulci there was brown to tan liquid pigment in the space between the pia mater and the leptomeninges and within vessels. Also observed at the ventral thalamus in leptomeningeal space. The lung had multifocal to coalescing mild alveolar high protein edema punctuated by open bronchioles and empty alveolar sacs. There was scant brown droplet material apparent in multifocal alveoli and airways. Mild high protein interstitial edema was apparent around the larynx.

Morphological diagnosis: Cerebrospinal Meningeal and Pulmonary Intralesional Foreign Material (iron supplement injection) and Suspected Anaphylactoid Reaction

Ancillary test: Perl's histochemical stain for ferrous iron revealed positive staining in vessels where there were blue stained globules in the lumen and lining the vascular intima in the brain, lungs, liver, and kidney. Globules of stained iron were also noted in lung alveoli.

Etiology: Foreign material leptomeningeal and intravenous injection (iron dextran)

Comments: Iron dextran injectable material (ferrous iron) was found in the musculature of the right neck, the intervertebral space at the base of the skull. The brown to yellow pigment covered the surface meninges of the spinal cord, medulla, cerebellum, ventral brain tissues and sulci of the cerebrum. Perl's histochemical stain revealed ferrous iron globules in the leptomeninges and blood vessels of the brain and brain stem, as well as in vessels of the kidney, lung, and liver. Death was suspected to be due to sudden increase in intracranial pressure and anaphylactoid like reaction incited by the complement cascade rather than conventional hypersensitivity type reactions. Prompt review of injection technique/procedure in neonatal swine was recommended.

References:

1. Rampton D. et al. Hypersensitivity reactions to intravenous iron: guidance for risk minimization and management. Guideline Article, Ferrata Storti Foundation. Haematologica 2014; 99 (11).

2. Tizard, IR. Veterinary Immunology An Introduction 8th Ed., 2009, p57-69.

3. Szebeni J. Hemocompatibility testing for nanomedicines and biologicals: predictive assays for complement mediated infusion reactions. Review. European Journal Nanomedicine. 2012; 4(1):33-53.

4. Zachary JF. Pathologic Basis of Veterinary Diseases, 7th Ed. 2022. P295-338.

Case # 6 **Brenda Bryan, VDS, Manitoba**

***Streptococcus equi* ssp *zooepidemicus* septicemia**

Clinical history: Received 3 dead sows average body weight 182kg. The history describes septicemia and sudden deaths. Submitted were 2 fresh sudden deaths and 1 euthanized sow. Death loss has been climbing since the weekend. Currently over 200 gilts/sows have died. Sows were on Pot Pen, followed by Amoxicillin, followed by tetracycline. So far antibiotics do not seem to be helping much. Sows that develop symptoms are unresponsive to injection with Ceftiofur or Ampicillin. This problem developed when gilts were introduced and *Streptococcus equi zooepidemicus* was cultured. It was assumed that this bacterium was the cause but may be something else.

Gross findings: On necropsy, the sows had dark pink ventrum and rump and ears were purple. The submandibular and cervical neck lymph nodes were enlarged approximately 2x, tan and dark red. In the chest, there was moderate amount of clear straw colored fluid with scant fibrin strands and the lungs were heavy pink to dark pink with prominent interlobular pulmonary edema. One pig (A) had a small fibrinous necrotic plaque on the papillary muscle of the right ventricle. Two of the pigs had large chicken fat clots in the right ventricles. All pigs had noticeable gall bladder wall edema.

Histopathology:
Heart: chorda tendonae endocarditic plaque measured approximately 10mm diameter x 2-3mm thick was expanded by edema, fibrin, and miliary cocci bacteria and myriad viable and degenerate neutrophils, covered by a thick layer of fibrin admixed with clusters of cocci bacteria and the surface lined by hemorrhage and fibrillar fibrin. Right and left ventricle had multifocal intravascular round hyalinised pink globules (shock bodies).
Lung: Alveolar septa were distended by miliary vascular fibrin thrombi, moderate numbers of neutrophils and septal macrophages. Large to capillary sized vessels had vascular fibrin thrombi and rarely, loose clusters of cocci bacteria. Alveoli multifocally had scant to small volumes of high protein fluid that occasionally curved along the alveolar septa, small clusters of loose fibrillar or globules of fibrin, few macrophages and rare multinucleated giant cells. Airway lumina contained sloughed autolyzed epithelium, rarely few neutrophils and globules of high protein. Interlobular septa and subpleura were slightly thickened by ectatic lymphatics, few random neutrophils.

Morphological diagnosis**:** Streptococcal Bacteremia/Septicemia, Disseminated Intravascular Coagulation (DIC)

1. Endocarditis, chordae tendonae right ventricle, plaque, fibrinosuppurative, acute, marked with miliary intralesional cocci bacteria
2. Pulmonary disseminated intravascular coagulation, marked, multifocal with intralesional cocci bacteria, pulmonary lobular septal edema, with marked, acute, interlobular pulmonary edema
3. Bacteremia, cocci, multiorgan, lung, kidney, joint synovium, brain, heart, thyroid, turbinates, liver, adrenal
4. Adrenal hemorrhage, multifocal to coalescing, with multifocal intravascular cocci bacterial and fibrin vascular thrombi

5. Lymphoid depletion, germinal centers, multifocal spleen, tonsils, lymph nodes, ileum
6. Lymphadenitis, mesenteric, bronchial, submandibular, lymph node, suppurative, moderate with moderate edema, multifocal vascular fibrin thrombi, germinal center lymphoid depletion

Ancillary test:
Aerobic Culture: Lung, heart, grain, spleen, liver, kidney, submandibular lymph node: *Streptococcus equi ssp zooepidemicus* 2 + to 4+
PCR , PCV2 lung: 2 of 3 pigs negative, 1 of 3 weak positive Ct 36.
PCR lung for PRRS, SIVA, and *Mycoplasma hyopneumoniae* was negative.

Etiology: *Streptococcus equi* ssp *zooepidemicus*

Comments: *Streptococcus equi ssp zooepidemicus* was isolated from numerous tissues supporting the diagnosis of Streptococcal bacteremia and septicemia, peracute to acute. In this situation, *S. equi ssp zooepidemicus* was extremely virulent, where affected pigs experienced a very rapid clinical course leading to organ failure and death within 24 to 48 hours. The main features in all pigs was massive DIC characterized by vascular fibrin thrombi in every organ/tissue examined along with intravascular tiny cocci bacteria in few chains, small loose clusters or singles and very few leukocytes despite the presence of bacteria.

References:

1. De Oliveira Costa M, Lage B. Streptococcus equi subsp zooepidemicus. Emerging Infectious Diseasess.www.cdc.gov/eid. vol 26, No. 10, October 2020, Research letters.
2. DeLay J, et al. Streptococcus equi subsp zooepidemicus septicemia: First confirmed case in Ontario swine, AHL Newsletter 2021:25(1):11.
3. Streptococcus equi subsp zooepidemicus, Swine Health Information Center, Feb 2021
4. De Oliveira Costa M, et al. Streptococcus equi subsp zooepidemicus infection of pigs leads to shedding in faeces and carrier state. Transboundary and Emerging Diseases.2022; 69:e1503-e1509.
5. Zachary JF. Pathologic Basis of Veterinary Diseases, 7th Ed. 2022 p.171-292, p 104-157, p839.
6. Jara LM, et al. Outbreak of Pathogenic Streptococcus equi subsp zooepidemicus in Guinea Pigs Farms of The Andean Region, Case Report, Pathogens, 2023,12,445.

Case #7 **Brenda Bryan, VDS, Manitoba**

Cutaneous Erysipelas

Clinical history: 15% of this batch of pigs have diamond skin lesions, mostly on the non-RWA (Raised Without Antibiotics) pigs. Suspecting Erysipelas infection. Submitted 2 pigs 1 RWA and 1 non-RWA/tagged pig. Received 2 pigs average weight of 36kg.

Gross findings: Both pigs had similar skin lesions characterized by multifocal rhomboidal to round, pink to red, slightly raised, plaque wheals occasionally with a central brown depression (targetoid). Fewer of the cutaneous lesions were slightly raised white plaques surrounded by red raised collars. Wheals were primarily on the hind ½ of the pigs, with few found on the front ½ of the body.

Histopathology**:** Skin: epidermis was hyperplastic (pseudocarcinomatous) with multifocal large intercorneal abscesses/pustules up to 0.2mm diameter and rare multifocal ulceration, with surface viable and degenerate neutrophils, and hemorrhage. The stratum spinosum was thickened by keratinocytes with pale expanded cytoplasm and multifocally many nuclei peripheralized by intracellular clear vacuoles (edema), rare reticulation, and transmigration of leukocytes, primarily neutrophils. Multifocally, the epidermal basilar region was effaced by neutrophils and edema. The superficial dermis was expanded by multifocal to coalescing neutrophils primarily with lesser numbers of lymphocytes, both interstitial and perivascular, edema, and rarely hemorrhage and by congested dilated vessels. In the deep dermis, there were perivascular neutrophils, edema and lesser numbers of lymphocytes. Apocrine glands were dilated and periadnexal interstitium expanded by mild edema and neutrophils.

Morphological diagnosis: Neutrophilic dermatitis, perivascular, marked, multifocal and focally extensive, subacute with suppurative adenitis

Ancillary test**:**
PCR on skin was positive for *Erysipelothrix rhusopathiae* Ct = 39.1 (Iowa State Veterinary Diagnostic Lab Erysipelas PCR *Erysipelothrix rhusopathiae, Erysipelothrix tonsillarum* and *Erysipelothrix sp2*)
PCR on lung was negative for PCV2 virus.

Etiology: *Erysipelothrix rhusopathiae*

Comments: PCR for *Erysipelothrix rhusopathiae* was positive in skin A at Ct 39.1 compatible with low molecular presence of the microbe and suspected cause of the “diamond skin lesions”. Both pigs had neutrophilic perivasculitis of the small vessels of the superficial dermis to the panniculus that led to the red to pink rhomboidal to round skin lesions. Differential etiology most common in pigs is the microbe *Erysipelothrix rhusopathiae* and not cultured in this case. However, other microbes may cause similar diamond to round shaped lesions on the skin such as *Actinobacillus suis*, and PCV2 infection (PCR negative). Lack of *E. rhusopathiae* isolation in tissues does not rule out the infection.

References:

1. Zachary JF. Pathologic Basis of Veterinary Diseases, 7th Ed. P.171-292, p104-157, p1086.
2. Maxie G. Jubb and Kennedy's Pathology of Domestic Animals. Vol 1 p151, vol 2 p20, 433, vol 3 p30.
3. Tizard, RI. Veterinary Immunology An Introduction, 8th Ed. 2009.
4. Gross TL, et al. Skin Diseases of the dog and cat. 2nd Ed. 2005.

Case # 8 **Marek Tomczyk, VDS, Manitoba**

Thrombocytopenic purpura

Clinical history
Second parity sow and a litter of weak and hemorrhagic piglets. 15 born alive, and 7 died and 8 were euthanized. Two dead piglets and two sedated but alive were received for evaluation and VDS in Winnipeg.
Gross findings: . Multiple petechial to small coalescing hemorrhages and blotches randomly distributed were seen on the head, ventral side of the abdomen and lateral side of the thorax and pressure points of the limbs. Pigs were pale.

Histopathology: Four pigs had very similar lesions, hence, be described as one. Skin: Severe and multifocal hemorrhages in deep dermis and muscularis were observed. Cerebral cortex: Multifocally sub meningeal hemorrhages elevated pia matter and extended into the superficial neuropil of the gray matter. Foci of hemorrhages were seen within sub meninges and within granular layer of the cerebellar folia. Tongue: Focal areas of ulceration and marked accumulation of neutrophils and macrophages, degenerated neutrophils and microcolonies were seen. Multifocal to coalescing interstitial hemorrhages were prominent. Heart: Multifocally foci of hemorrhages expanded myocardiocytes. There were multiple sub epicardial and endocardial foci of hemorrhages. Bronchial lymph node: Occasionally moderate accumulation of neutrophils and foci of hemorrhages were seen in the sub capsular and paratrabecular sinuses. Lung: Multiple foci of lobular pulmonary atelectasis were seen. Occasionally focally extensive foci of alveolar hemorrhages were observed. There were multifocal sub pleural hemorrhages which occasionally extended into interlobular septa.

Morphological diagnoses:
Thrombocytopenic purpura resulting in multiple tissues hemorrhages, acute, multifocal, with oedema and fibrin

Ancillary tests: Bacteriology, Hematology (CBC, chem profile), Virology (PCR for PRRS, SIV, PPV, CanSpot ASF)

Etiology: Neonatal alloimmune thrombocytopenia

Comments: The Iso-immunisation of pregnancy arises when incompatible fetal blood antigens cross the placental barrier. The dam becomes immunised against the incompatible cells and the iso-antibodies reach future piglets via colostrum. Hemolytic disease occurs if the iso-antibodies are directed against red blood cells or while directed against platelets – thrombocytopenic purpura will ensue. In this case moderate to severe anemia was regenerative, with erythrocyte morphology changes (possibly due to fetal hemoglobin or iron status) and lysis (ghosts) and severe thrombocytopenia. Also severe neutropenia with left shift and toxic changes indicating infectious or inflammatory disease or sepsis. Lymphocytes were reactive. Bacteriology revealed low numbers (few to 1+) of *Streptococcus suis, Streptococcus dysgalactiae* ssp. equisimilis, *Staphylococcus hyicus* and *Mannhemia haemolytica* from the lung and heart.

References
1. Joller, S., Häfliger, I.M., Drögemüller, C. et al. Thrombocytopenic purpura on an organic farm with pen mating: a case report on the re-emergence of an old disease. Porc Health Manag 6, 18 (2020). https://doi.org/10.1186/s40813-020-00157-z

Case # 9 **Marek Tomczyk, VDS, Manitoba**

Left liver lobe 360 degrees rotation at the hilus with liver capsule tear, hemorrhage, congestion and severe fibrinous peritonitis and peritoneal hemorrhage

Clinical history: In 2021 five farms indicated increased incidence of sudden death and abortions. Dead sows were submitted to VDS in Winnipeg for evaluation.

Gross findings: Animals had left liver lobe rotation. The affected lobe was dark red, markedly congested and covered with fibrin. The capsule was teared and covered with blood clots and fibrin adhered to the injured site. Approximately ten liters of dark red bloody fluid admixed with fibrin was in peritoneal cavity.

Histopathology: There was prominent accumulations of fibrin strands admixed with large numbers of neutrophils, macrophages and intralesional bacterial micro-colonies adjacent to the serosal side of the spleen, liver, serosa of the stomach, small and large intestines. Liver: In the affected liver lobe the congestion was severe; in the lobe which was not rotated there was marked oedema and vascular congestion; bile ducts pooled bile.

Morphological diagnosis:

1. Left liver lobe 360 degrees rotation at the hilus with liver capsule tear, haemorrhage, congestion and severe fibrinous peritonitis, peritoneal haemorrhage
2. Autolysis, marked

Ancillary tests: Bacteriology, Virology: PRRS, SIV, Mycoplasma hyopneumoniae - negative

Etiology: (Possible)

- Pigs are only being fed once a day, large volumes of wet feed and gas formation result in repeated stomach dilatation and in consequence changes of the liver space occupying volumes allowing for torsion
- Genetics
- Developmental liver ligament abnormalities
- Possible trauma, slippage, abrupt animal rotation

Comments: In recent years, VDS pathologists have encountered acute cases of liver lobe torsion upon necropsy. This diagnosis was made 10 times in 2021, and once in 2024. Partial or complete liver lobe torsion were reported in pigs, dogs, cats and horses. Torsed liver lobes undergo various degrees of ischemia, infarction, thrombosis. Affected animals usually die due to shock, haemorhage and septic peritonitis. Ischemia may favor *Clostridium* spp. overgrowth with development of necrosis and emphysema.

References:

1. Liver and biliary system, by J. M. Cullen and M. J. Stalker in Jubb, Kenedy, and Palmer's Pathology of Domestic Animals vol.2, edited by M. Grant Maxie, sixth edition, 2016; 268
2. Liver lobe torsion in dogs: 13 cases (1995-2004) by S.G. Hinkle Schwartz, S.L. Mitchell, J.H. Keating, D.L Chan in AVMA Volume 228: Issue 2, 2006; https://doi.org/10.2460/javma.228.2.242

Case # 10 **Marek Tomczyk, VDS, Manitoba**

Exudative pustular dermatitis with suppurative folliculitis & perifolliculitis

Clinical history: Piglets were becoming greasy 2-3 weeks after entry into nursery. The lesions were on the head, ventral neck, ventral abdomen, front and hind legs, inguinal area and dorsum. Three animals were submitted to VDS in Winnipeg for evaluation.

Gross findings: Skin lesions on the head and ventral neck, and lateral side of the chest. The ears are entirely covered with crusts and marked sub cutaneous oedema is felled on palpation. The lesions on the head, neck and thorax were round to coalescing ranging from 2 - 5 cm in diameter. There were thick crusts and reddening at the periphery of the lesion.

Histopathology: Multifocally variably acanthotic and oedematous epidermis was eroded, ulcerated or hyperplastic showing orthokerathotic hyperkeratosis with thick crusts of necrotic debris which covered deeper layers of sub epidermis and dermis, focally infiltrated by large numbers of neutrophils and macrophages mixed with islands of microcolonies of gram-positive rods and cocci. Follicular and perifollicular accumulations of neutrophils and macrophages mixed with bacteria, necrotic debris and keratin were observed.

Morphological diagnosis:
Exudative dermatitis, ulcerative with oedema, pustules formation, folliculitis, perifolliculitis and intralesional bacteria, severe, multifocal to coalescent

Ancillary tests
Bacteriology: Skin *Staphylococcus hyicus* & *Streptococcus equisimilis*
Virology: PRRS - negative, PVCV-2 - negative
Etiology
Staphylococcus hyicus & *Streptococcus equisimilis*

Comments: The lesions were consistent with exudative dermatitis. PCRs were negative for PRRS and PCV-2 In this case lesions were caused by *Staphylococcus hyicus* and *Streptococcus equisimilis*, although other staphylococci (S. scurii, S. chromogenes, S. aureus and MRSA) have also been sporadically implicated in outbreaks. Both virulent and avirulent strains of S. hyicus exist but appear unable to penetrate intact skin. Virulence is linked to production of exfoliative toxins (six of which have been identified) that reduce cell-cell adhesion of keratinocytes in the superficial epidermis. Abrasions on the face, feet, and legs or lacerations on the body precede infection. Contributing factors: abrasive surfaces, compromised immunity, poor hygiene, inadequate nutrition, presence of ectoparasites, stocking density and early weaning. The incidence is higher in gilt litters and newly established SPF herds Inapparent carriers like older pigs (sows, boars) may serve as a source of contamination for naive herds.

References:

1. 'Staphylococcosis' by T.S Frana & S.J. Hau in 'Diseases of Swine' edited by J.J. Zimmerman at al., VIII edition, 2019, pp 926-933
2. 'Integumentary System' by E.A. Mauldin & J. Peters-Kennedy in Jubb, Kenedy, and Palmer's Pathology of Domestic Animals vol.2, edited by M. Grant Maxie, sixth edition, 2016; pp 630-632
3. 'Staphylococcal skin disease in livestock' by A.P. Foster in Vet Dermatol 2012; 23: 342–351 DOI: 10.1111/j.1365-3164.2012.01093.x

Case # 11 **Marek Tomczyk, VDS, Manitoba**

Mulberry Heart Disease (Hemorrhagic cardiomyopathy)

Clinical history:

Swollen eye lids noticed yesterday; today two were in lateral recumbency and other progressing; three nursery pigs with swollen eye lids and nervous signs and one pig found dead.

Gross findings: Extensive epicardial haemorrhages; Hydrothorax and hydropericardium Pericardial oedema; background pathology observed included bronchopneumonia and eyelid oedema and conjunctivitis

Histopathology

Heart: Multiple to diffuse marked areas of interstitial haemorrhage, focal myocardiocytes early degeneration, and fragmentation involving full thickness of the left and right ventricle and the septum were seen. Multifocally vessels pool blood and occasionally fibrin and oedema fluid admixed with macrophages, lymphocytes, plasma cells were seen in the interstitium.

Morphological diagnosis

1. Mulberry Heart Disease (Haemorrhagic cardiomyopathy) resulting in epicardial & transmural haemorrhages, hydrothorax, hydropericardium & pericardial oedema
2. Bronchopneumonia with oedema and bronchiolitis obliterans
3. Conjunctival haemorrhage - C & Purulent conjunctivitis with intralesional bacteria - D

Ancillary tests

Bacteriology: a few *Streptococus suis* and *Trueperella pyrogens* from the heart C, no pathogens from the heart A/ B/ D; no pathogens isolated from the eyelids,

Virology: PCR Mycoplasma hyorhinis – A/ C weak positive ; B/ D positive

Etiology: Possible vitamin E and selenium deficiency

Comments: Mulberry heart disease (MHD) is a diagnosis of exclusion. It is considered that vitamin E deficiency plays a central role in the development of the mulberry heart disease. Some studies indicated lower levels of vitamin E in affected pigs, but others showed no difference. It is a possibility that MHD is precipitated following lack of vit E bioavailability in tissues, or possibly greater requirements for vit E under feeding diets high in unsaturated fat.

References:

1. 'Mulberry heart disease' in Cardiovascular system by W.F. Robinson & N.A Robinson in Jubb, Kenedy, and Palmer's Pathology of Domestic Animals vol.3, edited by M. Grant Maxie, sixth edition, 2016; pp 39-41
2. 'Vitamin E and selenium levels are within normal range in pigs diagnosed with mulberry heart disease and evidence for viral involvement in the syndrome is lacking' by Shen H, Thomas PR, Ensley SM, Kim WI, Loynachan AT, Halbur PG, Opriessnig T. in Transbound Emerg Dis. 2011 Dec;58(6):483-91. doi: 10.1111/j.1865-1682.2011.01224.x. Epub 2011 Apr 25. PMID: 21518323; PMCID: PMC7169668.

Case # 12 **Vasyl Shpyrka VDS, Winnipeg**

Swinepox virus

Clinical history or clinical history and gross findings Piglets were born this morning. 3 piglets in this litter had red dots over entire body. The remaining piglets had some dots, but not as bad. 2nd parity sow. 7 were born alive, 2 still born and 3 mummies. Fungal work up if possible? Please run other tests as necessary.
Carcass of one male new-born piglet without placenta was received and examined on October 17, 2023. The animal weighing 806 grams was in moderate body condition. Multifocal round to coalescing severe, erythematous umbilicated lesions with a wide ulcerated center and raised borders were covering the whole skin surfaces as well as the tongue. There were no other gross findings in the rest of the carcass.

Histopathology Skin. There is multifocal ulcerative loss of epidermis. Adjacent epidermis is detached from the dermis surrounded by necrotic cellular debris and inflammatory exudate. The inflammation in several location percolates into deeper dermis and affects several adnexal units. Multiple areas of non-ulcerated epidermis show a moderate increase in cellular thickness (hyperplasia), keratinocytes in hyperplastic areas have swollen cytoplasm (hydropic degeneration), with nuclei have marinated chromatin and central large clear vacuole. Affected keratinocytes frequently contain one or several pale eosinophilic intracytoplasmic inclusion bodies.
Tongue. Have similar lesions.
Morphological diagnosis
1.Skin. Dermatitis and folliculitis, multifocal, severe, subacute, proliferative and ulcerative with cytoplasmic inclusion bodies and pustules.
2. Tongue. Glossitis, multifocal, proliferative, with cytoplasmic inclusion bodies.

Ancillary test. Skin culture Escherichia coli 2 +; No fungi isolated; PCV2, PRRS, PPV PCR negative.

Etiology Swinepox virus

Comments. Swinepox occurs sporadically in pigs worldwide is endemic in regions with intensive swine production and is generally associated with poor sanitation. Pox viruses have host specificity, SwPV is not zoonotic and there is no evidence that SwPV is a threat in public health. Virus transition occurs either by direct contact or by mechanical vectors, such as the blood sucking swine louse (*Haematopinus suis*), mosquitoes or biting flies assisting infection by causing skin trauma. Vertical (transplacental) SwPV transmission is possible, as evidenced by sporadic cases of congenital infections. The presented case of a one-day-old piglet supports an intrauterine/congenital infection because the histopathology revealed reactive inflammatory changes indicative for a lesion duration of more than one day. In congenital infections, the affected piglets may be stillborn or die within a few days after birth, although the sows may appear clinically normal. Recovered on a skin culture *E.coli* is most likely contamination in this case.
References Diseases of Swine. Zimmerman J, et. al. 11th ed; .709-713., 2019

Case # 13 **Hélène Philibert, WCVM**

PCV2 related disease (postweaning multisystemic wasting syndrome)

Clinical history

A 14-week-old male pig was submitted alive to the diagnostic laboratory. Poor doing piglet. Infection with Porcine Circovirus suspected.

Histopathology

Tonsil, ileum, lung and kidney: There is marked lymphoid depletion in the tonsil and Peyer's patches with marked infiltration by macrophages and multinucleated cells. Rare macrophages contain large, basophilic blue intracytoplasmic inclusion bodies. The submucosa, and mucosa of the ileum are infiltrated by large numbers of macrophages and fewer multinucleated cells with loss of crypts and marked villous atrophy. Connective tissue of bronchi and bronchioles is infiltrated by small numbers of macrophages, fewer lymphocytes and interalveolar septae are mildly widened by similar cells. There is an increased numbers of macrophages in alveoli. Interstitium of the renal cortex contain few small aggregates of lymphocytes and macrophages.

Morphological diagnosis

Marked depletion of lymphoid tissue and granulomatous tonsillitis, ileitis, bronchointerstitial pneumonia.

Ancillary test

Abundant PCV2 antigen detected by immunohistochemistry within mononuclear cells.

Etiology

PCV2

Comments

It has been more than 30 years since the first outbreak of post weaning multisystemic wasting syndrome (PWMS) occurred in a small farm in Saskatchewan. The agent was not known at that time and it took a multidisciplinary approach of pathologists, virologists, immunologists to discover the relationship between a novel Porcine circovirus and this new syndrome. The histologic changes were striking - granulomatous inflammation with intracytoplasmic botryoid inclusion bodies in multiple organs and lymphoid depletion. PCV2 now occurs worldwide with different clinical manifestations, called PCV-2 related diseases (e.g PMWS, dermatitis and nephropathy syndrome, respiratory disease complex, reproductive failure). PCV2 is ubiquitous in herds and PCV2 related disease diagnosis requires three elements: presence of typical clinical signs, typical microscopic lesions and PCV2 antigen within lesions. Currently, there are two four species of PCV: PCV type 1, PCV type 2, PCV type 3 and PCV type 4 and are all pathogenic except PCV type 1.

References

1. J. Ellis. Porcine Circovirus: A historical perspective. Veterinary Pathology 2014. Vol 51 (2) 315-327.
2. H.K. Maity et al. Revisiting Porcine Circovirus Infection: Recent Insights and its significance in the piggery sector. Vaccines 2023, 11, 1308.

I would like to acknowledge Dr. Chris Wojnarowicz who did the diagnostic work-up on this case. He is a retired PDS pathologist.

Case # 14 Hemlata Gautam and Ivanna Kozii (WCVM and PDS)

Atrophic enteritis with intralesional coccidia

Clinical history or clinical history and gross findings:
Fixed tissue submitted from piglets of unknown age. Piglets showed signs of scouring and pneumonia

Histopathology
Small intestine: Circumferentially, there is a severe, diffuse, blunting and fusion of the mucosal villi (villous atrophy). Within the cytoplasm of mucosal epithelium, several coccidial organisms are present in various life stages including schizonts, macrogametes and microgametes. There is moderate increase in the mitotic figures within the crypt. Infrequently, in the crypt lumen and between the blunted villi there is eosinophilic to basophilic karyorrhectic nuclear debris. Frequently, few eosinophils are present in the lamina propria.

Morphological diagnosis
Atrophic enteritis, severe, diffuse with intralesional coccidia

Ancillary test: Real Time PCR positive for Rotavirus A (CT value=30.31)

Etiology: Rotavirus infection and coccidiosis

Comments:
Rotaviruses are responsible for post weaning white scours in 2-8 weeks old piglets. Predisposing factors include early weaning stress, reduced lactogenic immunity, and ubiquitous virus abundance. Rotaviruses primarily affect the small intestinal enterocytes by three mechanisms. First, virus multiplication induces cytolysis that results in sloughing and blunting of the enterocytes, hence, severe villous atrophy. Second, virus infected enterocytes release a vasoactive agent that causes villous ischemia and activation of the enteric nervous system. The third mechanism involves production of a secretory enterotoxin, non-structural protein (NSP4). Frequently, rotavirus infections are associated with other causes of diarrhea such as coccidiosis, adenovirus, and strongyloidiasis. Among several *Eimeria* species, *Cystoisospora suis* is the most common. It replicates in the distal third of the intestinal villi and completes their life cycle. Replication in the enterocytes further contributes to blunting and atrophy of enteric villi. This case is a classic example of porcine neonatal diarrhea caused by coinfection of commonly encountered porcine rotavirus A infection and coccidiosis.

References
Vlasova, A. N., Deol, P., Sircar, S., Ghosh, S., Jakab, S., Bányai, K., ... & Malik, Y. S. (2020). Animal rotaviruses. *Animal-Origin Viral Zoonoses*, 163-202.
Jubb, K. V. F., Kennedy, P. C., & Palmer, N. (2012). *Pathology of domestic animals*. Academic press, 234.

Case # 15 **Nicole Rose, WCVM**

Porcine cytomegalovirus rhinitis

Clinical history

A herd of 2500 piglets aged 23-27 days old. There has been an increase in the incidence of poor doing/fallback pigs. The piglets are unthrifty and approximately 2% are being held back in each batch. The herd is vaccinated for porcine circovirus 3 (PCV-3).

Histopathology

Nasal turbinates: Diffusely, the submucosa is moderately to markedly expanded and infiltrated by numerous lymphocytes, plasma cells, and fewer macrophages. Cells of the mucous glands and the respiratory epithelium are multifocally enlarged (cytomegaly) with abundant, foamy, eosinophilic cytoplasm and a single, basophilic, irregular, smudgy and granular, intranuclear inclusion body that fills and distends the enlarged nucleus. Multifocally the mucous glands are ectatic, lined by attenuated epithelium, and contain variable amounts of eosinophilic cellular and karyorrhectic debris (necrosis) with rare neutrophils. The respiratory epithelium is multifocally lost and necrotic. The concha contains evidence of bony remodeling and woven bone proliferation.

Morphological diagnosis

Nasal turbinates: Rhinitis, lymphoplasmacytic and necrotizing, subacute, diffuse, marked, with glandular epithelial karyomegaly due to intranuclear inclusion bodies

Etiology

Porcine cytomegalovirus.

Comments

Porcine cytomegalovirus is caused by Suid betaherpesvirus 2 and is found in herds worldwide. Infection is common but often inapparent in herds where there is widespread immunity. Piglets in naïve herds that acquire the infection congenitally or in the neonatal period can die from systemic infection. In contrast, clinical signs of inclusion body rhinitis tend to be seen in about 4-week-old suckling piglets. In this age group the disease is usually self-limiting and includes mild sneezing, catarrhal nasal exudate and coughing with dyspnea. The classic histologic lesions in this age group are 8-12 um basophilic intranuclear inclusion bodies, cytomegaly and karyomegaly in the nasal mucous glands.

This herd had a very low pathogenic strain of Porcine Reproductive and Respiratory Syndrome (PRRS) that the veterinarian had been trying to eliminate. During this time the herd had issues with septicemia caused by a toxigenic *Escherichia coli* and piglets showing clinical signs of inclusion body rhinitis possibly secondary to immunosuppression. The piglet in this case also had lesions of septicemia and cultured positive for extraintestinal pathogenic *E. coli* (positive for Aerobactin virulence factor).

References

Zimmerman, J. J. (2019). *Diseases of swine* (Eleventh edition.). Wiley Blackwell. https://doi.org/10.1002/9781119350927

Case # 16 Tat Chuan Cham, Enrique Aburto, WCVM

Vegetative endocarditis with septic thromboembolism

Clinical history: A female finishing pig was found dead, and its ears and nose were purple. There were other pigs in the same farm showing strep like symptoms and lameness.

Gross findings: The skin around the ears, ventral, cervical, axillary, and inguinal regions were purple red. Within the heart, the leaflets of pulmonary, aortic, and left atrioventricular valves were partially replaced by variably sized white to yellow, friable, cauliflower-like, friable masses covered by thin blood clots (vegetative endocarditis). Bilaterally, the tarsal joints were congested and markedly edematous. The lungs were mottled red and diffusely wet, heavy, and non-collapsed.

Histopathology: Affecting multiple organs (*i.e.*, lungs, heart, stomach, tarsal joint, skin), there were fibrin thrombi and bacterial emboli within the blood vessels, often associated with coagulative necrosis of the surrounding tissues.

Morphological diagnosis: Vegetative endocarditis with septic thromboembolism in multiple organs (lungs, heart, stomach, tarsal joint, skin)

Comments: This pig had vegetative endocarditis with septic thromboembolism affecting multiple organs. These lesions resulted from a generalized bacterial infection. *Streptococcus dysgalactiae* was isolated from the kidneys, spleen, and pulmonary valve. This bacterial organism has been previously associated with arthritis, endocarditis, and meningitis in pigs.[1,2]

References:

1. Karstrup C, Jensen H, Aalbæk B, Leifsson P, Boye M, Agerholm J. Endocarditis-associated brain lesions in slaughter pigs. *Journal of comparative pathology*. 2011;144**:** 289-295.

2. Kasuya K, Yoshida E, Harada R, et al. Systemic Streptococcus dysgalactiae subspecies equisimilis infection in a Yorkshire pig with severe disseminated suppurative meningoencephalomyelitis. *Journal of Veterinary Medical Science*. 2014;76**:** 715-718.

Case # 17 **Angie Magana, WCVM**

***Brachyspira hampsonii* mucopurulent and necrotizing colitis.**

Clinical history: A 10-week-old, castrated male, Yorkshire pig was euthanized after several days' history of mucohemorrhagic diarrhea.
A complete necropsy was performed with special attention to the stomach, duodenum, jejunum, ileum, mesenteric lymph nodes, spiral colon (proximal, apex and distal), cecum and rectum. The most severe gross lesions were in the mucosa of the cecum and proximal spiral colon and consisted of moderate to severe diffuse congestion and edema with large flecks of dark red material and mucous covering the mucosal surface. Lesions lessened in severity in the apex and distal spiral colon with only mild congestion and edema in the descending colon and rectum with sticky mucohemorrhagic contents.

Histopathology:

Proximal spiral colon: In a few areas, the lamina propria of the mucosa is minimally infiltrated by neutrophils, plasma cells, and lymphocytes. There is a focal area of epithelial necrosis surrounded by a mixed inflammatory infiltrate, composed primarily of degenerate neutrophils, and covered by a thick layer of mucus containing degenerate neutrophils, necrotic epithelial cells, and Warthin-Faulker positive bacterial colonies.

Morphological diagnosis: Proximal spiral colon: Mucopurulent and necrotizing colitis, severe, multifocal with spirochetes within the crypts and intralesional spirochetes.

Ancillary test: Warthin-Faulker stain, Brachyspira isolation on BJ agar from feces and colonic mucosa, qPCR.

Etiology: *Brachyspira hampsonii.* clade I (Canadian strain 30599).

Comments: *Brachyspira hampsonii* Clade I is a pathogenic spirochete responsible for mucohemorrhagic diarrhea and colitis in pigs, clinically indistinguishable from swine dysentery (*Brachyspira hyodysenteriae*) and *Brachyspira hampsonii* Clade II. Clade I strain 30599 was isolated in Western Canada in November 2011 from grow-finish pigs with mucous and bloody diarrhea. Histological lesions can be detected in the proximal and apical regions of the colon, as well as in the distal colon, and the spirochetes can be evident using silver stains such as Warthin-Faulker. The isolation of *B. hampsonii* can be performed from feces and colonic mucosa on BJ agar plates, where zones of β-hemolysis observed through dark-field microscopy confirm the growth of *Brachyspira*. Isolation followed by sequencing of the *nox* gene was set as the diagnostic gold standard for detection of *B. hampsonii* clade I strain 30599. The presence of this disease is associated with a decrease in feed efficiency, resulting in severe economic losses in the swine industry.

References

1. Costa, M. O., Hill, J. E., Fernando, C., Lemieux, H. D., Detmer, S. E., Rubin, J. E., & Harding, J. C. (2014). Confirmation that "Brachyspira hampsonii" clade I (Canadian strain 30599) causes mucohemorrhagic diarrhea and colitis in experimentally infected pigs. *BMC veterinary research*, *10*, 129. https://doi-org.cyber.usask.ca/10.1186/1746-6148-10-129

Case 18 **Marina Carla Bezerra da Silva, WCVM**

Porcine Sapovirus genogroup III

Clinical history or clinical history and gross findings: Ongoing scours in 4–7-day-old piglets. Received 4 live piglets. All piglets had slightly soiled perineum with white to yellow, creamy feces. Similarly, moderate amounts of yellow and watery feces were noted in the small and large intestine. There was approximately 70 mL of white curded milk in the stomachs. No other significant gross findings were present.

Histopathology: In the small intestine the villus-to-crypt ratio was decreased to 2:1 (normal is 7:1). There is marked crypt hyperplasia with increased mitotic figures with prominent nucleoli (regeneration). The lamina propria has mild numbers of lymphocytes and neutrophils. The enterocytes are mildly attenuated with moderate villous fusion and atrophy and mild vacuolation of the tip enterocytes. There is occasional crypt displacement to the submucosa (herniation). There is mild congestion of the submucosa and lamina propria and Peyer's patch hyperplasia.

Morphological diagnosis: Diffuse moderate villous atrophy with villous fusion and crypt regeneration.

Ancillary test: PCR for Porcine Sapovirus genogroup III was positive with Ct values ranging from 12.1 to 16.7

Etiology: Porcine Sapovirus genogroup III

Comments: Porcine Sapovirus (PoSaV), belonging to the *Caliciviridae* family, primarily affects neonatal and young pigs, with suckling and post-weaning pigs being most susceptible. Genogroup III is the most detected; however, eight genogroups of PoSaV are currently known. Histopathological findings in PoSaV infection are similar to other viral infections that cause enteritis in pigs, such as rotavirus and coronavirus, and include villus atrophy, epithelial vacuolation at villus tips, and crypt regeneration. In this case, culture, PCR, and histopathology were negative for other potential pathogens including rotavirus, PEDV, PDCoV, TGEV, bacteria, and *Cystoisospora suis*.

References

1. Nagai M et al. Porcine sapoviruses: Pathogenesis, epidemiology, genetic diversity, and diagnosis. Virus Res. 2020; 286: 198025.

2. DeLay J & Ojkic D. Porcine sapovirus: An emerging pathogen contributing to swine diarrhea. AHL Newsletter 2023; 27(1):15.

Case #19 Asha Perera, John Harding, Volker Gerdts, Susan Detmer, WCVM

Porcine epidemic diarrhea

Signalment: 6-days old, cross-bred, female, piglet.

History: These piglets were part of a vaccine study on porcine epidemic diarrhea. During this study, commercial crossbred pregnant sows received the PED vaccine or saline mixed with adjuvant intramuscularly. This piglet (VM54) was born to a control sow. All piglets born to these sows suckled from their dams. At 4 days of age, all piglets were orally inoculated with live porcine epidemic diarrhea virus (PEDV). This piglet was clinically sick with diarrhea but survived until euthanasia at 6 days of age. Tissues were immediately processed after euthanasia.

Histopathology:
Small intestine: Severe blunting and fusion of villi, multifocal epithelial necrosis, attenuation, cytoplasmic vacuolation in enterocytes, mild lymphoplasmacytic inflammation in the lamina propria.

Morphological diagnosis: small intestine: Enteritis, necrotizing, with villous blunting and fusion, and crypt hyperplasia

Ancillary tests: immunohistochemistry for PEDV was positive.

Etiology: Porcine epidemic diarrhea virus

Comments:

PEDV, an enveloped, positive-sense, single-stranded RNA virus belongs to family Coronaviridae, genus Alphacoronavirus. PED has been present in Europe and Asia from 1970s. PED is an emerging disease in North America and detected first in the United States in 2013. It was first found in Canada January 2014 on a farm in Ontario. It has since been detected in Manitoba, Quebec, Prince Edward Island and, most recently, Alberta in 2019. It is a provincially notifiable disease in Saskatchewan. There have been continuous efforts to eradicate PED in Canada with each new reintroduction.

In susceptible herds, PEDV infection causes watery diarrhea, vomiting, dehydration and high mortality (up to 100 %) in piglets of < 2 weeks of age. PEDV infection of older pigs results in considerably lower morbidity and mortality. It is clinically and pathologically indistinguishable from Transmissible Gastroenteritis virus (TGEV) infection. However, PEDV is antigenically distinct from TGEV and can be distinguished with diagnostic testing (i.e. RT-PCR, IHC). Thinning of small intestinal walls where contents are visible externally is common as gross lesions together with dehydration and a distended intestine filled with yellow fluid. Microscopically, villi blunting and fusion with multifocal epithelial necrosis and attenuation is present in the small intestine of affected piglets. Commercial vaccines are available for PEDV.

References:

1. Jung, K., Saif, L. J., & Wang, Q. (2020). Porcine epidemic diarrhea virus (PEDV): An update on etiology, transmission, pathogenesis, and prevention and control. Virus research, 286, 198045.
2. Makadiya, N., Brownlie, R., van den Hurk, J., Berube, N., Allan, B., Gerdts, V., & Zakhartchouk, A. (2016). S1 domain of the porcine epidemic diarrhea virus spike protein as a vaccine antigen. Virology journal, 13, 1-10.

Case #20 **Maria Spinato, AHL, University of Guelph**

Mycotic tonsillitis and lymphadenitis due to *Geotrichum* sp.

Clinical history: A herd of 115 grower/finisher pigs developed a problem affecting approximately 10% of animals in the finishing barn. Pigs presented with variably-sized serous-filled cutaneous swellings that were restricted to the cervical region, measuring up to 15 cm in diameter. No bedding was used in the finishing barn, and the feed was mixed on site. One of the affected pigs was euthanatized and samples were collected from internal viscera (no gross abnormalities noted), as well as the cervical cutaneous nodules. These were submitted to the Animal Health Laboratory for histopathologic examination and bacterial culture.

Histopathology: Tonsillar architecture is partially effaced and parenchyma expanded by focally extensive accumulations of infiltrating leukocytes that included eosinophils, lymphocytes, neutrophils and numerous multinucleated giant cells that often contained cross-sections of bulbous fungal hyphae. Sections of the cutaneous nodules were indeterminate regarding site; however, the presence of a distinct capsular boundary is most consistent with lymph node origin. These sections contained extensive foci of necrosis and numerous clusters of karyorrhectic leukocytes. A few scattered multinucleated giant cells containing fungal hyphae are also present. Fungal hyphae are accentuated in PAS-stained sections. A Ziehl Neelsen-stained section was negative for the presence of acid fast bacteria.

Morphological diagnosis: Granulomatous tonsillitis and cervical lymphadenitis with intralesional fungi

Ancillary test: Bacterial culture isolated mixed and variable growths of *Staphylococcus aureus*, *E. coli*, *Streptococcus suis* and *S. dysgalactiae* subsp. *equisimilis*. Since numerous fungal hyphae were seen on routine wet mount of the affected tissues, mycotic cultures were also set up. *Geotrichum* sp. was isolated from 3 out of 3 samples; *Mucor* sp. was also isolated from 1 of the 3 tissues.

Etiology: *Geotrichum* sp.

Comments: A single case report of mycotic tonsillitis in a free-ranging weaner pig from Australia due to *Geotrichum candidum* has been reported. *Geotrichum* sp. is a saprophytic fungus found on fruits, vegetables, and dairy products. The authors of this report speculated that the feeding of macadamia nut trash may have inoculated the tonsils of this pig with this uncommon organism. Sporadic cases of geotrichosis in other species, including humans, are often linked to immunosuppression. Addition history was obtained from the herd veterinarian in this case. The presumed source of infection was poor quality, moldy ensiled high moisture corn. Tonsillar inoculation following oral exposure with subsequent dissemination to cervical lymph nodes was the probable mechanism for development of the cervical cutaneous nodules.

Reference:

1. Lee EJ et al. Tonsillitis in a weaner pig associated with *Geotrichum candidum*. J Vet Diagn Invest 2011;23:175-177.

Case #21 **Rebecca Egan, AHL, University of Guelph**

Polioencephalomyelitis due to porcine sapelovirus

Clinical history: Nursery pigs (4-6 wks of age) have neurological abnormalities including ataxia, averaging 5 out of every 1000 pigs (per day). Not responsive to antibiotic therapy. Death occurring within 24 hours of first signs.

Histopathology: Situated predominantly within the grey matter of midbrain, brainstem and spinal cord (mostly concentrated in the ventral horns of cervical and thoracic segments), there are multiple glial nodules with rare neuronophagia present. Neutrophils are scattered among the glial cells in a few sites. Lymphocytic cuffs surround many blood vessels in adjacent neuroparenchyma. Low numbers of lymphocytes and plasma cells are scattered within the leptomeninges, and form narrow cuffs around a few meningeal blood vessels.

Morphological diagnosis: Non-suppurative polioencephalomyelitis

Ancillary tests: Porcine hemagglutinating encephalomyelitis virus (PHEV), rabies virus, and bacteria were not detected in brain by PCR (viruses) or culture (bacteria). Representative brain samples from 2 pigs were forwarded to the Iowa State University Veterinary Diagnostic Laboratory for porcine sapelovirus, porcine teschovirus, and porcine astrovirus 3 testing. Porcine sapelovirus DNA was detected by PCR in both brain samples, and porcine teschovirus nucleic acid was detected concurrently in the brain of 1 pig.

Etiology: Porcine sapelovirus

Comments: Historically, porcine enteroviruses (serotypes 1 to 11) have been members of the genus enterovirus in the *Picornaviridae* family. Recent reclassification has resulted in the generation of two new genuses: the teschovirus genus (serotypes 1-7 and 11-13, now porcine teschovirus), and the sapelovirus genus (serotype 8, now porcine sapelovirus A). Porcine enterovirus serotypes 9 and 10 (PEV-9 and PEV-10) remain in the enterovirus genus. Enteric infection with these viruses is often acquired post-weaning, coinciding with waning maternal antibodies and mixing of animals; however, most infections remain asymptomatic. Infection with virulent strains may produce clinical neurologic disease in 2 typical forms: a severe and highly fatal form (Teschen disease), and a milder form (Talfan disease). In recent years, similar cases of neuroinvasive sapelovirus infection have been reported in pigs in the United States and Europe. While entero-like viruses, including sapeloviruses, are commonly carried in the intestinal tract of healthy animals, factors precipitating spread to the central nervous system have yet to be elucidated. To date, sapelovirus-associated neurologic disease has not been reported in other species.

References:

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2. Arruda PHE et al. Detection of a novel sapelovirus in central nervous tissue of pigs with polioencephalomyelitis in the USA. Transboundary and Emerging Diseases 2017;64:311-315. https://onlinelibrary.wiley.com/doi/epdf/10.1111/tbed.12621
3. Arruda PHE et al. Identification of a divergent strain of sapelovirus associated with a severe polioencephalomyelitis outbreak in the US. Swine Health Information Center. https://www.swinehealth.org/sapelovirus/
4. Cantile C, Youssef, S. Enterovirus/teschovirus polioencephalomyelitis of pigs. In: Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, 6th ed. Maxie MG, ed. Elsevier, 2016; vol 2:372-373.

Case #22 **Rebecca Egan, AHL, University of Guelph**

Bronchointerstitial pneumonia due to porcine astrovirus 4

Clinical history: Several nursery pigs lost condition, and exhibited increased respiratory effort and coughing. Sick pigs were treated with antibiotics, and the mortality rate peaked at approximately 7%, with most of the losses occurring in the second and third weeks post-placement. On-farm postmortems were performed on nursery pigs from each barn, revealing lungs that failed to collapse.

Histopathology: There are multiple atelectatic lobules with bronchioles that are lined by variably attenuated to hyperplastic epithelium with intraepithelial neutrophils and luminal accumulation of dense aggregates of neutrophils admixed with occasional macrophages. Adjacent alveoli harbour many macrophages admixed with variable numbers of neutrophils. In large airways, respiratory epithelium often exhibits loss of cilia and multiple discrete foci of epithelial jumbling, and there are frequent intraepithelial neutrophils accompanied by occasional aggregates along the epithelial surface. In these lobules, there are also mild to moderate peribronchiolar and perivascular to interstitial lymphohistiocytic infiltrates. In the most prominently inflamed areas, edema with neutrophilic and macrophagic inflammation mildly expands interlobular septa as well.

Morphological diagnosis: Necrotizing bronchointerstitial pneumonia

Ancillary tests: PCR tests were negative for PRRSV, PCV-1,2,3, influenza A, *Mycoplasma hyopneumoniae*, ASFV and CSFV. IHC was also negative for PRRSV, PCV-2 and Influenza A. Lung samples were forwarded to Iowa State diagnostic lab for porcine astrovirus 4 PCR (positive) and porcine parainfluenza virus (negative). PoASt-4 RNAscope testing revealed the presence of scant to moderate staining within small and large airway epithelium in samples from 2 pigs. Bacterial culture was not performed.

Etiology: Porcine astrovirus 4

Comments: Not long after this diagnostic investigation, the cough in the sow barn resolved and nursery performance improved. It was hypothesized that introduction of large numbers of gilts following PRRSV eradication earlier that year may have led to reduced immunity in the gilt litters, which combined with viral respiratory infection in suckling pigs, may have predisposed animals to various infections in the nursery.
At present, there are five known lineages of porcine astrovirus, causing neurologic disease, enteric disease, or asymptomatic infection. More recently, detection and characterization of a novel genotype of PoAstV4 from nasal swabs obtained from suckling pigs with acute clinical respiratory disease has been reported, and while a causal link between PoAstV4 and respiratory disease in pigs has not been fully established, there is research to suggest that infection may contribute to the development of clinical disease.

References:

1. Rahe MC, et al. Porcine Astrovirus-4 detection in lesions of epitheliotropic viral infection in the porcine respiratory tract. Transbound Emerg Dis 2023, Article ID 9113355.
2. Padmanabhan A, Hause BM. Detection and characterization of a novel genotype of porcine astrovirus-4 from nasal swabs from pigs with acute respiratory disease. Arch Virol 2016;161(9):2575–2579.

Case #23 **Savannah Goldstein, WCVM**

Atypical Porcine Pestivirus in Pig Experimentally Infected with Swine Influenza A

Clinical history: 9-week-old male neutered pig (*Sus scrofa*) experimentally infected with swine influenza A virus (IAVS) presented with sudden onset, episodic bouts of full body fasciculations for few weeks' duration. Necropsy revealed well demarcated, patchy, dark red discoloration and atalectasis in cranioventral lung, with no other gross lesions.

Histopathology: Multifocally and superficially, throughout the cerebral cortex, mild infiltrates of lymphocytes, plasma cells with rare neutrophils and extravasated red blood cells are present within the Virchow-Robin spaces of vessels, occasionally admixed with scant pale, eosinophilic, amorphous material. Leukocytes rarely extend to surrounding neuropil. There is also mild, rare, multifocally and randomly distributed foci of gliosis. Luxol fast blue highlights normal myelination.

Morphological diagnosis:

1. **Brain:** Lymphohistiocytic perivasculitis, multifocal, subacute, mild with rare leukoencephalitis and gliosis
2. **Lung:** Necrotizing bronchiolitis, locally extensive, subacute, moderate with interstitial pneumonia, atalectasis, and secondary acute, cranioventral, suppurative bronchopneumonia **(slide not provided)**

Ancillary test: Polymerase chain reaction (lung, brain): IAV-s and atypical porcine pestivirus (APPV), respectively. Bacterial culture (lung): *Streptococcus suis,* few.

Etiology: Atypical porcine pestivirus (APPV) and swine influenza A (IAVS) with few *S. suis*

Comments: This case demonstrates clinical, gross, and microscopic pathologies associated with two viral conditions of varying commonality in domestic swine: IAV-S and APPV. Focus placed is placed on APPV, which is considerably less common. This small, enveloped, RNA virus (family *Flaviviridae*) is known to cause "congenital tremors" in piglets globally, with important economic and welfare implications. In its typical case presentation, neonatal pigs are infected vertically during early gestation, demonstrate severe full body tremors and inability to suckle colostrum (failure to thrive), as well as histological evidence of myelin destruction in the CNS. In this case, APPV was diagnosed using PCR, despite an older age at presentation and no evidence of myelin destruction. Changes associated with IAV-S, an enveloped RNA virus (family *Orthomyxoviridae*) with zoonotic potential, are discussed briefly in this case, and demonstrate classical gross and histological changes associated with this infection.

References:

1. I. R. Honorato Gatto, K. Sonálio, L. G. de Oliveira, Atypical porcine pestivirus (APPV) as a new species of pestivirus in pig production. Front. Vet. Sci. 6, 1–8 (2019).

2. L. Schwarz, et al., Congenital infection with atypical porcine pestivirus (APPV) is associated with disease and viral persistence. Vet. Res. 48, 1 (2017).

Case #24 **Josepha DeLay, Animal Health Laboratory, University of Guelph**

***Brachyspira murdochii*-associated colitis in growing pigs**

Clinical history: Chronic intermittent diarrhea in 1-2 month old pigs in a 1200 head commercial herd, fall-backs, with evidence of colitis on field necropsy. Previous diagnosis of ileitis.

Histopathology: Colonic superficial mucosal epithelium is multifocally attenuated or cuboidal. Clusters of spiral bacilli are present at the mucosal surface in few sites. Subjacent lamina propria is infiltrated by moderate numbers of densely packed macrophages, lymphocytes, and plasma cells, occasionally mixed with neutrophils. Mucosal crypts are intact and subjectively elongated, with increased numbers of mitotic figures, and crypt epithelium is well differentiated and intermingled with many goblet cells. Colonic lesions are similar in other pigs from the group and also include focal ulceration and attenuation of crypt epithelium.

Morphological diagnosis: Erosive colitis

Ancillary tests (similar results for all 3 pigs in group)**:**

Bacterial culture (colon): *E.coli* 2+, no *Salmonella* spp isolated by enrichment, no *C.perfringens* isolated.

Brachyspira spp PCR (colon): *Brachyspira* spp pos (Ct 26.38), *B.murdochii* pos (Ct 21.70), *B.innocens* pos (Ct 25.47) – *B.hyodysenteriae, B.pilosicoli, B.hampsonii* not detected. *Brachyspira* spp IHC: small to large numbers of positive-staining spirochetes on colonic surface and in crypts.

Lawsonia intracellularis PCR (feces): pos (Ct 37.29); *Lawsonia intracellularis* IHC: *L. intracellularis* antigen not detected in colon.

Fecal flotation: no parasites identified.

Etiology: *Brachyspira murdochii*

Comments: Colitis in this group is attributed to *Brachyspira* spp, specifically *B.murdochii*. This organism is considered mildly pathogenic. IHC for *Brachyspira* spp identified variable numbers of spirochetes in affected areas of colon, supporting but not confirming contribution of the organism to colonic lesions. Some non-pathogenic *Brachyspira* spp may be commensal organisms of swine colon, complicating interpretation of IHC findings here. The pathogenicity of less pathogenic spirochetes is at least in part dependent on a combination of host, microbial, and dietary factors. Contribution of *Salmonella* spp and other common enteric pathogens to colonic lesions was excluded on the basis of negative bacterial culture and other ancillary test results. Although Lawsonia intracellularis was detected in feces by PCR, histologic lesions compatible with *L.intracellularis* infection were not identified in ileum or colon, and *L.intracellularis* antigen was not detected in enterocytes by IHC.

References:

1.Costa MO *et al*: Subclinical colitis associated with moderately hemolytic *Brachyspira* strains. J Swine Health Prod 27(4): 196-209, 2019.

2.Jensen TK, AS Christensen, M Boye. *Brachyspira murdochii* colitis in pigs. Vet Pathol 47(2): 334-338, 2010.

Case # 25 **Roa'a Basyouni**

Porcine reproductive and respiratory syndrome (PPRS)

Clinical history :

Formalin fixed samples from a 1-week-old, weaned piglet. Clinical signs described: not eating well (mainly water) with watery scours. Treated with 2kg Chlor 100, 2kg Tilmicosin and Respotil in water. Gross findings described by the submitting veterinarian: terrible lungs and enlarged lymph node.

Histopathology

Lung: 80% of alveoli within the section are collapsed or filled with macrophages, fewer lymphocytes and plasma cells, eosinophilic cellular and basophilic karyorrhectic debris (necrosis) and clumps of basophilic cell-free chromatin. Similar infiltrate and necrotic material are also present within bronchioles. Less affected areas display thickening of interalveolar septa by mononuclear cells.

Morphological diagnosis

Lung: interstitial pneumonia histiocytic, with intra-alveolar and bronchiolar macrophage necrosis and free chromatin.

Ancillary test

Histopathology, Immunohistochemistry, PCR

Etiology

Lung: PPRS positive by PCR and IHC

Comments

Histologic lesions were consistent with severe interstitial pneumonia with extensive necrosis of macrophages admixed with clumps of free chromatin. These lesions are very suggestive for an infection with porcine reproductive and respiratory syndrome (PRRS). PRRS is caused by an *arterivirus* which is transmitted via multiple routs including inhalation and ingestion. The virus infects macrophages causing their apoptosis and necrosis and is spread via viremia to lymph nodes causing lymphadenopathy . The virus causes a decrease in the phagocytic and functional capacity of macrophages which often results in secondary infections. Reproductive abnormalities can also be seen in affected herds.

References

(Cao, Sugimura et al. 2022)
Pathologic Basis of Veterinary Disease, 7th Edition (VetBooks.ir).

Case # 26 **Kinda Ghubari, WCVM**

Enterococcus durans

Clinical history or clinical history and gross findings: Portions from a piglet of unspecified age received. Scours in parity 1 and 2 litters mainly starting 2-3 days of age reported by the submitting veterinarian. Clinical signs of yellow watery diarrhea, dehydration, and vomiting, followed by death described by the vet. Dilated fluid filled small intestine found on necropsy by the vet.

Histopathology: Jejunum, mild villous atrophy with multifocal bacterial attachment to the apical surfaces of the intestinal villi and crypts.

Morphological diagnosis: Jejunum: Villous atrophy with apical surface bacterial attachment

Ancillary test: *Clostridium perfringens (1+)* and *Escherichia coli* (2+) *were isolated using bacterial culture.* PCR testing was negative for E.coli toxins (AIDA-*I*, *Eae*, *F18*, *F4*, *LT*, *STa*, *STb*, and *Stx2e)*. PCR for *Clostridium perfringens* toxin typing confirmed the presence of alpha and beta2 toxins. PCR testing for Porcine Deltacoronavirus, Porcine Epidemic Diarrhea Virus, and Transmissible Gastroenteritis Virus was negative.

Gram stain on small intestinal sections: Bacteria suggestive of *Enterococcus durans* was seen.

Etiology: Most consistent with *Enterococcus* seen adhered to apical surface of enterocytes on histology and with grams stain

Comments: Gram staining on intestinal tissue sections revealed the presence of high numbers of gram-positive cocci attached to the apical surface of the jejunal enterocytes, which is highly indicative of *Enterococcus durans. Enterococcus durans* can be part of the normal intestinal microbiota in pigs. While generally not pathogenic, it has been linked to cases of diarrhea in neonatal piglets.[i] The underlying mechanism of diarrhea associated with adherent *Enterococcus durans* in piglets remains poorly understood. **It** does not induce diarrhea through enterotoxin production or significant intestinal damage. Instead, it likely disrupts nutrient absorption and digestion by reducing the activity of brush border enzymes, such as lactase and alkaline phosphatase.[ii]

No association was found between the positive *Clostridium perfringens* results and the observed histological lesions in this case.

References:

[1] UZAL, F. A.; PLATTNER, B. L.; HOSTETTER, J. M. Chapter 1 - Alimentary System. In: MAXIE, M. G. (Ed.). Jubb, Kennedy & Palmer's Pathology of Domestic Animals: Volume 2 (Sixth Edition): W.B. Saunders, 2016. p.1-257.e2. ISBN 978-0-7020-5318-4.

[1] Cheon, D., & Chae, C. (1996). Outbreak of diarrhea associated withEnterococcus duransin piglets. *Journal of Veterinary Diagnostic Investigation*, *8*(1), 123–124. https://doi.org/10.1177/104063879600800123

Case #27

Belarmino E. Lopes-Neto,
Postdoctoral Fellow, WCVM

Glasser's disease in a piglet

Clinical history: A three-month-old, male, "Piau" breed piglet presented with slight incoordination, mouth breathing, apathy and anorexia. The owner had recently acquired 14 piglets, but only one piglet was clinically affected. The pig died a few hours later. Postmortem examination revealed fibrinous polyserositis, including severe fibrinonecrotic epicarditis and pericarditis, abdominal visceral peritonitis, fibrinonecrotic and suppurative pleuritis and pleuropneumonia.

Histopathology:

Lung: Multifocally, moderate, infiltrating the alveolar septa are neutrophils, macrophages, lymphocytes, and fibrin. There are polynucleate inflammatory exudate in the lumen of the bronchus. The lymphoid tissue adjacent to bronchi and bronchioles is moderately hyperplastic. The pleura is multifocally expanded by fibrin, hemorrhage, and necrosis (not shown on slide).

Morphological diagnosis:
1. Severe interstitial pneumonia with fibrinonecrotic and suppurative pleuritis.
2. Bronchopneumonia

Ancillary test: Bacterial culture of lung t yielded *Glaesserella parasusis*

Etiology: *Glasserella parasuis*.

Comments: *Glasserella parasuis*, formerly *Haemophilus parasuis*, is a small, pleomorphic Gram-negative bacterium responsible for Glasser's disease in piglets, and pneumonia in adult pigs. The infectious disease occurs sporadically among swine herds and might be related subsequently to stressful events such as weaning, changes in environment, commingling, or as coinfection with other disease agents, especially Porcine Circovirus-2 (PCV-2) and Porcine Reproductive and Respiratory Syndrome (PRRS).

In this case, we were able to observe the main manifestation of the disease in piglets, mostly the polyserositis. The affected piglet likely had an underlying immunodeficiency, as it was the only animal of the herd that became ill and with acute and severe symptoms, which could be a result of an immunosuppressive viruses.

I would like to thank Dr. Thalita Oliveira for kindly share this case to present.

References:
1. Dickerman A., Bandara A.B., Inzana T.J. Phylogenomic analysis of Haemophilus parasuis and proposed reclassification to Glaesserella parasuis, gen. nov., comb. nov. Int J Syst Evol Microbiol 70:180–186, 2020

Sumit.J., Rojina N., Astha T., Subash

Case # 28 **Glenna McGregor, BC Ministry of Agriculture and Food**

Sarcoptic mange and glomerulonephritis and vasculitis in a pet miniature pig

Clinical history or clinical history and gross findings:

Nine-month old miniature pig presented to submitting vet for skin issues and ill-thrift and died during sedation. On gross post-mortem examination she was in thin body condition and the skin was diffusely thickened with an abundant brown adherent crust with multiple small irregular frond-like structures hyperkeratosis).

Histopathology

Kidney: Glomeruli are expanded by large amounts of eosinophilic material (membranous glomerulonephritis) and there is frequent cellular loss and glomerular senechiae. The tubules are often ectatic and lined by attenuated and sometimes degenerate epithelium. The tubular lumen multifocally contains abundant viable and degenerate neutrophils mixed with necrotic cellular and nuclear debris. Markedly expanding the interstitium and replacing, surrounding and separating glomeruli and tubules is a large amount of fibrous tissue infiltrated by moderate numbers of lymphocytes, plasma cells and fewer macrophages and neutrophils. Vessels are very frequently surrounded by moderate numbers of lymphocytes, plasma cells, macrophages and fewer neutrophils and reactive fibroblasts. The vessel wall is disrupted by a large amount of deeply eosinophilic material (fibrin) and moderate amounts of necrotic debris and degenerate neutrophils, lymphocytes, plasma cells and macrophages.
Congo Red Special Stain: There is no apparent significant amyloid within the kidney.

Skin: Diffusely, the epidermis is markedly hyperplastic with acanthosis, prominent rete ridge formation, spongiosis, and multifocal orthokeratotic and parakeratotic hyperkeratosis that contains embedded adult arthropods and eggs. Occupying the stratum corneum, occasionally within tunnels, adult arthropods are ovoid, 200-300 x 100-150 um and possess a spiny chitinous exoskeleton, jointed appendages, striated muscle, a body cavity (hemocoel), and intestinal and reproductive structures. The eggs are oval, thin-shelled, and 70 x 40 um. Multifocally there are intracorneal pustules composed of degenerate neutrophils, necrotic cellular debris, and proteinaceous fluid. Diffusely the superficial dermis is expanded by clear space(edema) and few lymphocytes, plasma cells, neutrophils, and eosinophils. There are few ectatic hair follicles.

Morphological diagnosis

Suppurative dermatitis with severe epidermal hyperplasia and hyperkeratosis with embedded *Sarcoptes scabei* adults and eggs (Sarcoptic mange)

Severe membranous glomerulonephritis and chronic interstitial nephritis with acute suppurative tubulonephritis

Severe fibrinonecrotizing vasculitis, most severe in the kidney

Ancillary test

African Swine Fever PCR – negative
Porcine Circovirus 2 PCR – negative in-house and at Prairie Diagnostic Services (PDS)
Porcine Circovirus 3 PCR – negative at PDS
Mineral panel – unremarkable
Aerobic culture – mixed bacteria in multiple organs
Fecal Flotation – 1+ coccidia, 1+ strongylidae

Etiology

Skin lesions: Sarcoptes scabei

Renal lesions: unknown, possible hypersensitivity reaction to *Sarcoptes scabei*

Comments

The skin lesions were consistent with Sarcoptic mange due to infection with *Sarcoptes scabei* var. *suis*.

In addition to the sarcopic mange there was a severe membranous glomerulonephritis, a dramatic fibrinonecrotizing vasculitis, that was most severe in the kidney, but also in other organs as well, and a severe chronic fibrosing myocarditis. PCV-2 infection was considered the most likely differential for this constellation of lesions; however, PCR on pooled spleen and lymph node was negative for PCV-2 in-house, and negative for both PCV-2 and PCV-3 at Prairie Diagnostic Services (PDS) in Saskatoon. An alternative explanation for these lesions was that it was secondary to the sarcoptic mange. Scabies in humans has been associated with glomerulonephritis and myocardial fibrosis and inflammation (called Rheumatic heart disease) likely due to immune dysregulation, hypersensitivity reactions and immune complex deposition, potentially related to *Streptococcus* spp. infection of the skin secondary to the Scabies; however, I could find no case reports of this in swine. Other hypersensitivity reactions due to other antigens including autoantigens also cannot be ruled out.

References

Wang D, Li L, Wei L, Liu Y, Sun S. Acute postinfectious glomerulonephritis associated with scabies in the elderly: A case report. Parasitol Int. 2017 Dec;66(6):802-805.

Case # 29 **Glenna McGregor, BC Ministry of Agriculture and Food**

Mycobacterial abortion

Clinical history or clinical history and gross findings:
Three piglets from a litter where all were stillborn. A previous litter also had all 18 piglets stillborn. Sporadic vaccine use on farm. Sows are housed indoors for farrowing but otherwise kept outdoors with access to sheds. Piglets weighed 0.6, 1.1, and 1.5 kg and were variably autolyzed.

Histopathology:

There is severe generalized autolysis and freeze-thaw artifact.

Liver: there are multifocal randomly-distributed foci of inflammation and necrosis characterized by hepatocyte loss and aggregates of necrotic cellular and nuclear debris, large numbers of viable and degenerate neutrophils and smaller numbers of macrophages including some Langhans-type and foreign-body type multinucleated giant cells and moderate numbers of lymphocytes and plasma cells. Portal triads are multifocally infiltrated by moderate numbers of lymphocytes and plasma cells and smaller numbers of neutrophils and macrophages. There are small numbers of acid-fast bacilli in macrophages and multinucleated giant cells.

Spleen: there are multifocal aggregates of viable and degenerate neutrophils mixed with necrotic cellular and nuclear debris. There are often frequent aggregates of macrophages and foreign-body and Langhans-type multinucleated giant cells. There are small numbers of acid-fast bacilli in macrophages and multinucleated giant cells.

Morphological diagnosis

Pyogranulomatous hepatitis and splenitis with intrahistiocytic acid-fast bacterial rods (Mycobacteriosis)

Ancillary test
Nocardia consensus PCR negative
Mycobacterium consensus PCR Positive. DNA sequencing results indicate best match to *Mycobacterium avius* subsp. hominissuis.
PCR negative for PRRS, PCV-2, African Swine Fever, Porcine Parvovirus

Etiology
Mycobacterium avius subsp. Hominissuis

Comments
Mycobacterium avium subsp. Hominissuis is a member of the M. avium complex and is regarded as an opportunistic pathogen for pigs and humans. It is typically derived from an environmental source. Infection in pigs is typically characterized by granulomatous lesions in lymph nodes associated with the digestive system, but granulomatous lesions in internal organs like the liver, lungs, and kidneys may also occur. There are reports of association with abortion, but no description of the associated fetal lesions to my knowledge. In Norway it is associated with the use of peat for bedding. It was also found in a single sawdust sample. There was a previous *Mycobacterium* spp. abortion diagnosed at the Animal Health Centre prior to 2013, associated with a pig herd using moss for bedding.

References
Hulinova Stromerova N, Faldyna M. Mycobacterium avium complex infection in pigs: A review. Comp Immunol Microbiol Infect Dis. 2018 Apr;57:62-68.

Case # 30 **Dominique Fournier, MAPAQ**

Multifocal ulcerative and necrotizing dermatitis with leukocytoclastic vasculitis and thrombosis of undetermined cause

Clinical history and gross findings:

Two weeks after arriving at a grower-finisher unit, ten pigs were found dead presenting reddish skin lesions on the ventral abdomen and buttocks. These pigs had received a water treatment (tylvalosin) for three days for *Mycoplasma hyosynoviae*-related lameness. Other clinical signs were also observed: constant tail movements, subcutaneous edema between the hindlegs, and tremors (shivering). Only a few groups were affected in one section of the farm.

Two 12-week-old pigs were submitted dead for necropsy. Both pigs showed similar cutaneous lesions consisting of irregular wine-red macules and plaques. These lesions were more extensive on the hind limbs as well as inguinal and ventral areas. Lymph nodes were markedly hypertrophied.

Histopathology:

The epidermis shows extensive areas of erosion and ulceration covered by thick serocellular crust composed of degenerate leucocytes, erythrocytes and keratin as well as small clusters of cocci bacteria. There is necrosis of the underlying dermis with loss of cellular details (coagulated appearance) with thrombosis of small capillaries as well as infiltration of a small number of granulocytes (potentially neutrophils), predominantly surrounding blood vessels. The wall of a small number of these vessels is also infiltrated by a few to a small number of granulocytes (vasculitis) with pyknotic nuclear debris/dust.

Morphological diagnosis:

Multifocal ulcerative and necrotizing dermatitis with leukocytoclastic vasculitis and thrombosis

Ancillary tests:

Special stain: gram stain confirms the presence of gram-positive cocci in the crust.

Bacteriology: no significant findings

PRRSv PCR: negative
Porcine circovirus type 2 PCR: positive (CT: 24.01)
Porcine circovirus type 3 PCR: negative
Porcine circovirus immunohistochemistry (IHC): negative

Etiology:

Undetermined

Comments:

A tylvalosin formula was used on this farm just before the occurrence of the clinical signs. A thorough investigation was then conducted by the pharmaceutical company marketing this product. According to their report, the severe cutaneous reaction seen in some of these grower pigs was not associated with the usage of this compound. The cause for these severe lesions remains uncertain. A few cases of leukocytoclastic vasculitis associated with the usage of macrolide antibiotics have been reported in human medicine literature. We cannot exclude the possibility that the lesions were associated with the presence of porcine circovirus type 2; however, immunohistochemistry for this agent was negative in the skin lesions. Nonetheless, this virus can be a cause of vasculitis. Consequently, it remains a differential even though there was no evidence of renal lesions in this pig as seen with porcine dermatitis and nephropathy syndrome. Furthermore, vascular lesions can be reproduced experimentally with porcine circovirus type 2 virus infection but are not necessarily seen with the presence of renal lesions. An immune-mediated vasculitis from another or unknown cause was also considered, but no potential cause could be identified. Almost one year after this event, there are still no other pigs on this farm that have subsequently presented similar clinical signs.

References:

1) Langohr I. M. et al: Vascular Lesions in Pigs Experimentally Infected With Porcine Circovirus Type 2 Serogroup B. Vet Pathol; 47:140-147, 2010.
2) Odemis E. et al: Azithromycin-Induced Leukocytoclastic Vasculitis. J of Rheumatol; 30:10, 2003.

Case # 31 Josephine Lee, Helene Philibert, Yanyun Huang, WCVM/PDS

Systemic Porcine Cytomegalovirus

Clinical history and gross findings: Tissues from a 5 month old pig from a research study on myocardial infarction. It was treated with immunosuppressive drugs. This animal developed lethargy, thrombocytopenia, severe leukopenia, tachypnea, poor peripheral perfusion and acute respiratory distress 2 weeks after surgery on the heart. On post-mortem exam, there was severe pulmonary edema, and 120 ml of clear fluid in abdomen. Abdominal lymph nodes were enlarged and reddened and kidneys were pale with petechial hemorrhage. Suspected acute heart failure.

Histopathology:
Kidney**:** There are multifocal, acute interstitial hemorrhage, more pronounced in the medulla, associated with rare necrosis of endothelial and tubular epithelial cells. Occasional endothelial and epithelial cells are markedly enlarged and contain large, deep purple, intranuclear inclusion body. There is focal necrosis of glomerular tufts with protein rich fluid in Bowman's space and tubules and rare intranuclear inclusion body in endothelial cells of the tufts. Proximal straight tubules are lined by attenuated epithelium.

Liver: There are multifocal, well delimited areas of congestion and atrophy/loss of hepatic cords and rare necrosis, hemorrhage in the centrolobular region. Scattered individual endothelial and hepatocytes throughout the parenchyma contain large intranuclear inclusion body. Lymphatic vessels in capsule and portal tracts are distended.

Morphological diagnoses:
1. Renal hemorrhage, acute, extensive and intranuclear inclusion bodies in endothelial and tubular epithelial cell with acute necrosis
2. Acute centrolobular congestion, hemorrhage, necrosis and atrophy of hepatic cords

Etiology: Suid herpesvirus 2 (Porcine Cytomegalovirus)

Comments: Porcine Cytomeglovirus (PCMV) is a high morbidity, low mortality disease primarily affecting piglets 3 – 5 weeks of age. Piglets developing systemic disease are mostly infected under 3 weeks of age, and disease in older animals tend to present as a mild rhinitis. Like other *herpesviruses*, reactivation of latent infections can occur during times of stress. This animal was either latent or newly exposed to PCMV, and most likely developed a systemic infection due to immunosuppression. Other sites of infection in this piglet were the lymph nodes, nasal turbinates and the lungs where it caused an acute interstitial pneumonia. The zonal pattern in the liver would be most suggestive of acute passive congestion rather than hemorrhage solely due to cytomegalovirus infection.

References

1. Maxie, M. G. (2016). *Jubb, Kennedy, and Palmer's Pathology of Domestic animals*. Saunders Limited.
2. Edington, N., Plowright, W., & Watt, R. (1976). Generalized porcine cytomegalic inclusion disease: Distribution of cytomegalic cells and virus. Journal of Comparative Pathology, 86(2), 191–202. https://doi.org/10.1016/0021-9975(76)90043-8

Case # 32 **Magaly B-Pepin, MAPAQ**

Marked subcutaneous edema associated with PRRS infection in newborn piglets.

Clinical history:

A commercial farrow-to-finish pig farm began to observe several piglets born with very marked subcutaneous edema. Several litters were affected but not all piglets from the same litter were affected. Two, one-day-old piglets were euthanized and submitted for necropsy. The most striking gross lesion was marked subcutaneous edema, more prominent on the eyelids and ears, with swollen and enlarged lymph nodes.

Histopathology:

Histological lesions included rare foci of necrosis in the brain, moderate dilation of lymphatic vessels in multiple organs as well as edema of subcutaneous tissue and lymph nodes. Marked extramedullary hematopoiesis was also noticed in multiples organs but was considered an incidental finding.

Ancillary test:

Routine bacterial culture (viscera) : Negative
PCRs :
PCV2, PCV3, Senecavirus, Herpesvirus : Negative
PRRSv: Positive (CT:26.81)
IHC (PRRSv): Mild positivity in lymph nodes

Etiology :

- Porcine respiratory and reproductive syndrome (PRRS) ; probably

Comments:

Porcine reproductive and respiratory syndrome virus (PRRSv) is commonly diagnosed in our laboratories. This arterivirus is associated with reproductive impairment and most of the time, in our necropsy cases, with respiratory disease in young and growing pigs. PRRS virus infection generally results in mild to severe lesions in the lungs with interstitial pneumonia often combined with areas of proliferative and necrotizing pneumonia. Many other lesions, such as periocular and subcutaneous edema has been reported with PRRSv but are less common. In this particular case, two one-day-old, euthanized piglets were submitted for necropsy with marked subcutaneous edema that was more prominent on the eyelids and ears with swollen and

enlarged lymph nodes. Histological lesions included foci of necrosis in the brain, moderate lymphatic vessels dilation in multiples organs and edema in subcutaneous tissue and lymph nodes. These lesions are presumed to be associated with PRRS virus infection as this virus was detected by PCR on a pool of tissues from these piglets. No other significant pathogen was detected. Congenital heredity lymphedema, edema disease, Cytomegalovirus infection were also considered in the differential but were ruled out with ancillary test and histological findings. Subcutaneous edema is a possible manifestation of PRRSv infection in neonatal piglets.

References

Van der Putte, S., (1978) "CONGENITAL HEREDITY LYMPHEDEMA IN THE PIG", *Lymphology* 11(1), 1-9.

Lunney JK, Benfield DA, Rowland RR. Porcine reproductive and respiratory syndrome virus: an update on an emerging and re-emerging viral disease of swine. Virus Res. 2010 Dec;154(1-2):1-6.

Raymond P, Bellehumeur C, Nagarajan M, Longtin D, Ferland A, Müller P, Bissonnette R, Simard C. Porcine reproductive and respiratory syndrome virus (PRRSV) in pig meat. Can J Vet Res. 2017 Jul;81(3):162-170.

Rossow KD, Collins JE, Goyal SM, Nelson EA, Christopher-Hennings J, Benfield DA. Pathogenesis of Porcine Reproductive and Respiratory Syndrome Virus Infection in Gnotobiotic Pigs. *Veterinary Pathology*. 1995;32(4):361-373.

Rossow KD. Porcine Reproductive and Respiratory Syndrome. *Veterinary Pathology*. 1998;35(1):1-20.

Case #33 **Hannah Walker, Oregon State University**

The complexity of swine enteric disease - Proliferative ileitis, colitis, and enteric villous atrophy

Clinical history and gross findings: Samples from 6, 10-week-old, York x Landrace piglets. All piglets were submitted live for necropsy and presented with dehydration, watery diarrhea, and runted growth. The 1200 head feeder operation experienced repeated episodes of scouring and weight loss in younger pigs. Farm had changed production to "natural" 12 months prior. Grossly, caudal ileal mucosa was diffusely thickened to 2mm and slightly granular, intestinal contents watery. Colonic and cecal contents had thick aggregates of yellow, rubbery materials.

Histopathology: Diffusely, small intestinal villi are moderately to severely blunted and many are fused. Ileal segments have a thickened lamina epithelialis mucosae with long, branching crypts. The lamina propria mucosae has multifocal, mild, neutrophilic infiltrates. Peyer's patches are moderately depleted. The colon has multifocal crypt necrosis and dilation and segmental ulceration with massive bacterial colonization. Comma-shaped bacteria colonize the apical aspect of affected ileal enterocytes.

Morphological diagnosis: Severe, diffuse, proliferative ileitis (with intraepithelial, comma-shaped bacteria as per special stain). Moderate to severe, diffuse villous atrophy: SI. Moderate, multifocal, acute, fibrino-necrotizing colitis with intralesional colonies of mixed bacteria.

Ancillary test: Coronavirus detected via electron microscopy (ileal mucosa). *Lawsonia intracellularis* detected via immunohistochemistry (ileum). PCR positive for PCV 2.

Etiology: Coronavirus, *L. intracellularis,* secondary bacterial colonization and septicemia

Comments: Coronaviral enteritis infections in pigs can be attributed to transmissible gastroenteritis virus, porcine epidemic diarrhea virus, and porcine deltacoronavirus[1]. Coronaviruses are known to target and destroy villus enterocytes, resulting in the villous atrophy seen in this case. *L. intracellularis,* a known obligate intracellular bacterium that replicates in the cytoplasm of immature enterocytes is the cause of proliferative enteropathy, more commonly referred to as ileitis[2]. Characterized by the proliferation of epithelial cells and thickening of intestinal mucosa in the ileum[2]. While more commonly seen in finisher pigs, this bacterium is suspected of using mechanisms of other infections to aid in its transmission between enterocytes[3]. For this case, *L. intracellularis* PCR was not used. This case is from 2005 and diagnostic work-up for porcine enteric disease has since been approached differently due to availabilities of tests. Further diagnostics and quantification of the suspected etiologies was not pursued, and a direct coronaviral cause was not determined. Lack of culture growth suggests a chronic disease process. Special thanks to Dr. Christiane Loehr for her expertise on this case.

References

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3. Vannucci FA, Gebhart CJ. Recent Advances in Understanding the Pathogenesis of Lawsonia intracellularis Infections. *Veterinary Pathology*. 2014;51(2):465-477.

Isabelle St-Pierre, MAPAQ (no case number and no slide)

Investigation of rhinitis in weaned/post-weaned piglets

Clinical history

In Quebec Animal Health Provincial Laboratory, we regularly diagnose rhinitis in weaned and post-weaned piglets. In some cases, histologic lesions of rhinitis are present without a specific viral or bacterial cause. Swine practitioners from the Porcine Reseau team asked us to investigate the presence of *Mycoplasma hyorhinis* in rhinitis cases because this agent is prevalent in their herds, and they think it might contribute to the development of rhinitis.

Rhinitis in weaned and post-weaned piglets can cause upper respiratory signs, sneezing, decreased food consumption and wasting. *Mycoplasma hyorhinis* is part of the upper respiratory tract commensal flora of pigs and is associated with polyserositis and polyarthritis in post-weaned pigs. It has also been associated with conjunctivitis, otitis media, pneumonia and more recently with meningitis. We designed an investigation protocol to evaluate the presence and distribution of *M. hyorhinis* in the nasal conchae of weaned and post-weaned piglets and its potential role as a contributing pathogen in rhinitis. A standard necropsy work-up was performed in 3- to 7-week-old piglets received for necropsy. *Mycoplasma hyorhinis* PCR on nasal swabs, histologic examination of the nasal conchae, bacterial culture on nasal swabs and In Situ Hybridization using RNAscope assay on a few positive *M. hyorhinis* PCR cases, were included in our protocol.

Results

Mycoplasma hyorhinis PCR on nasal swabs and histologic examination of the nasal conchae, were performed on 56 piglets.

	Histo rhinitis+	Histo rhinitis -
PCR *M. hyorhinis*+	34	4
PCR *M. hyorhinis*-	7	11

Odds ratio: 13,4 (confidence interval: 3,3 to 54,4)

Bacterial culture on nasal swabs was performed on 33 piglets with evidence of rhinitis on histolologic examination.

Most prevalent combinations of agents:

PCR *M. hyorhinis*+ cytomegalovirus (Inclusion Body (IB) rhinitis) + *Past. multocida* +: 7/33 (21%)

PCR *M. hyorhinis*+ *Past. multocida* +: 6/33 (18%)

PCR *M. hyorhinis*+ *Bordetella bronchiseptica* +: 4/33 (12%)

PCR *M. hyorhinis*+ *Bord. bronchiseptica*+ *Past. multocida* +: 4/33 (12%)

PCR *M. hyorhinis*+ *Bord. bronchiseptica*+ cytomegalovirus (IB rhinitis) +: 4/33 (12%)

M.hyorhinis RNAscope on 7 cases (6 with PCR + and 1 PCR – control):

Positive staining of variable severity was observed on the surface epithelium and in the lumen of PCR+ cases.

Comments:

The odds ratio (OR) is a measure of how strongly an event is associated with exposure to a particular variable, so in this study, the likelihood of having histologic lesions of rhinitis in a pig with positive *Mycoplasma hyorhinis* PCR. The odds ratio is >1 meaning that a positive *M. hyorhinis* PCR and the presence of rhinitis on histology, are correlated. The presence of rhinitis on histology increases the probability of a positive *M. hyorhinis* PCR and, symmetrically, the presence of a positive *M. hyorhinis* PCR increases the probability of rhinitis on histology. Odds ratio >1 doesn't involve causality meaning that it doesn't confirm that *M. hyorhinis* is a cause of rhinitis. In conclusion, there is a positive correlation between a positive *M. hyorhinis* PCR result and the presence of rhinitis on histology. Further research is needed to determine the importance of *M. hyorhinis* in the development of rhinitis lesions and define its role.

References

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