CONTROL ID: 2019481

TITLE: Variant Strain of *Porcine Epidemic Diarrhea Virus* Caused Mild Histological Lesions in the Small Intestines of Piglets

ABSTRACT BODY:
Narrative: In January 2014, we detected a variant strain (OH851) of PEDV in samples from a swine farm in Ohio. Genome sequence comparison showed that there was a high nucleotide similarity in either the complete genome (99%) or the full-length spike (S) gene (97%) between variant PEDV and currently circulating PEDV strains from US, whereas a low nucleotide identify (<= 89%) was observed in the first 1,170 nt of the S1 region between them. Importantly, the S1 domain of the OH851 strain is closely related (99% identical) to another PEDV strain (CH/HBQX/10) reported in China, indicating at least two genotypes of PEDV circulate in the US. Histological changes of piglets infected with the variant strain of PEDV were observed in the small intestines (jejunum, ileum), including mild segmental to multifocal villous atrophy, villous fusion, and superficial enterocyte attenuation. Mild lymphoid depletion in 1/5 pigs was observed in colonic lymph nodes. These microscopic changes were much milder than anticipated for PEDV associated infection. No evidence of infection of virulent PEDV, rotaviruses, and TGE virus. No colibacillosis, clostridial enteritis, coccidiosis or cryptosporidiosis was observed microscopically in any of the 44 intestinal sections examined. Further research is needed to monitor the evolution of the variant PEDV as well as virulent PEDV in US swine populations.

CURRENT CATEGORY/DISCIPLINE: Pathology | Virology

AUTHORS/INSTITUTIONS: Y. Zhang, L. Wang, J. Hayes, B. Byrum, Ohio Department of Agriculture, Animal Disease and Diagnostic Laboratory (ADDL), Reynoldsburg, Ohio, UNITED STATES;

AWARDS:

Trainee Letter:

CONTROL ID: 2018991

TITLE: Histological Lesions in Piglets Associated With a *Swine Deltacoronavirus*

ABSTRACT BODY:
Narrative: *Porcine deltacoronavirus* is a member in the genus Deltacoronavirus of the family *Coronaviridae*. The virus was first recognized in pigs in Hong Kong in 2012. Between the end of January and the beginning of February 2014, samples from piglets and sows were received from outbreaks of diarrheal disease resembling porcine epidemic diarrhea (PED) or transmissible gastroenteritis (TGE) from several pig farms in Ohio. Mortality ranged from 30 – 50% in piglets. All samples were negative for PED and TGE viruses. Subsequently, we detected *deltacoronavirus* from all samples. Phylogenetic study indicated that the newly detected virus was closely related to the *porcine deltacoronavirus* reported in Hong Kong. Further study showed this virus was detected in samples from nine out of ten states, demonstrating wide distribution of this virus in the US. Sequence analysis of all isolates from the nine states suggested that a single genotype is circulating in the US. Histopathologic alterations, such as attenuation and cytoplasmic vacuolation of superficial enterocytes, villus atrophy and villus fusion in small intestinal sections were similar to, but less severe, than those observed in piglets affected with PED virus infection.

CURRENT CATEGORY/DISCIPLINE: Pathology

AUTHORS/INSTITUTIONS: Y. Zhang, L. Wang, J. Hayes, C. Sarver, B. Byrum, Ohio Department of Agriculture, Animal Disease Diagnostic Laboratory, Reynoldsburg, Ohio, UNITED STATES;

AWARDS:

Trainee Letter:

CONTROL ID: 2022839

TITLE: Treatment-Associated Pathologic Findings in Wisconsin Cattle

ABSTRACT BODY:

Narrative: Most pathologies of cattle are due to infectious disease. One our most common pathologic submission in Wisconsin (>90% of cases) is the 7-14-day-old dairy calf with hypotonic dehydration, metabolic acidosis and electrolyte imbalance secondary to diarrhea caused by *Cryptosporidium parvum, rotavirus* and/or *coronavirus*. Intestinal lesions may be subtle due to rapid autolysis, though villous atrophy or mild inflammation (tips of villi) may be found.

In addition, lymphoid depletion (stress, acidosis, utilization), abomasitis (dehydration, improper lubrication of mucosa), and mild, culture-negative cholangitis (possible ascending infection) are often seen. Most calves do not have failure of passive transfer (FPT). Treatment-associated lesions include ruminitis, which in some cases is associated with tubing with milk, aspiration pneumonia due to improper tubing, nephrosis (dehydration; gentamicin toxicity), renal papillary necrosis (rare, non-steroidal anti-inflammatory drugs, NSAIDs), antibiotic-induced yeast or clostridial overgrowth, and iron, selenium or sodium toxicity. Recently, a notable trend identified older calves with shipping fever bronchopneumonia being treated with dexamethasone, a long-acting corticosteroid. Most calves with respiratory disease are 6 weeks of age or older—perhaps early detection is difficult because sick calves continue to drink milk. Thus, some calves thought to be in the acute stage clinically, actually have advanced disease. Furthermore, treatment with dexamethasone may alter pulmonary defense mechanisms, decreasing lung clearance and eventually, worsening their already advanced state of disease. Adult cattle are submitted less often to our laboratory. Undesirable outcomes have included a few vaccine reactions (anaphylaxis due to bacterins; abortion due to modified-live IBR vaccines), enteritis due to *C. difficile* following multiple antibiotic courses, and post-surgical peritonitis.

CURRENT CATEGORY/DISCIPLINE: Pathology

AUTHORS/INSTITUTIONS: M. Behr, Wisconsin Veterinary Diagnostic Laboratory, University of Wisconsin-Madison, Madison, Wisconsin, UNITED STATES;

AWARDS:

Trainee Letter:

CONTROL ID: 2016785

TITLE: Descriptive Epidemiology, Gross and Histologic Lesions of Toe-Tip Necrosis in Western Canadian Feedlot Cattle

ABSTRACT BODY:

Narrative: Toe-tip necrosis (TTN) is a disease of feedlot cattle involving any combination of inflammation and necrosis of the corium; inflammation, necrosis and lysis of P3; or both. TTN typically affects hind feet and more commonly lateral claws. Toe-tip necrosis syndrome (TTNS) refers to sequela associated with TTN that includes lameness and unthriftiness due to arthritis, tenosynovitis, cellulitis and embolic pneumonia. Therefore, TTNS is of concern to feedlot operators and veterinarians for financial and humane reasons.

Studies reported herein include a computerized health record search; analysis of health records of animals dead or euthanized because of TTNS; and the post-mortem examination of hind feet of feedlot cattle. A search of health records for 501,438 feedlot cattle, under the care of a beef industry consultancy, compiled over the 6 year period 2007 to 2012, inclusive, found that about 2/10,000 animals died or were euthanized due to TTNS, but that incidence varied between 1 and 24/10,000 at different times and feedlots. These figures are conservative as

only animals that died or were euthanized because of TTNS were included. Some animals with TTNS likely survived without being diagnosed or survived following treatment. Retrospective analysis of 702, veterinarian diagnosed, cases of TTNS found 55% were yearlings and 45% were calves <1 year; and 71% were steers and 29% were heifers. Of those animals with a post-mortem diagnosis of TTNS, the mean and median interval between arrival and death was 42.7 and 27.0 days, respectively. Deaths due to TTNS occurred in all months, but were most common between September and November.
The post-mortem study was initiated in the fall of 2012. Three veterinary feedlot practices collected 1 or both hind feet from cattle dead or euthanized because of TTNS. The same practices also submitted hind feet from other cattle in the same feedlots, but that had died or been euthanized because of other disease, e.g., bronchopneumonia and myocarditis. In total, 1 or more feet were submitted from 79 animals believed to be affected with TTNS and 67 animals that died or were euthanized with other diseases and believed to be free of TTN. During the 'blind' examination of 39 feet, the presence of apical white line separation (AWLS) correctly predicted the presence of TTN in 100% of claws; and the absence of AWLS correctly predicted the absence of TTN in 97% of claws. A gross exam of other claws with TTN also found AWLS; that lesions progressed proximally from the tip; and no displacement of the P3 relative to the hoof wall. The histologic exam of 6 affected claws revealed no evidence of vasculitis, which might be consistent with BVD virus infection; no evidence of laminitis, which might be consistent with a metabolic disturbance related to feeding a high grain ration and ruminal acidosis; and no evidence of embolic disease. Studies to identify risk factors for the development of AWLS of the hind feet of feedlot cattle are ongoing.

CURRENT CATEGORY/DISCIPLINE: Pathology

AUTHORS/INSTITUTIONS: L.A. Gyan, A.L. Allen, Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskachewan, Saskatoon, Saskatchewan, CANADA;
C.D. Paetsch, M.D. Jelinski, Department of Large Animal Clinical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, CANADA;

AWARDS:

Trainee Letter:

CONTROL ID: 1996802

TITLE: Oak Poisoning in Yearling Cattle

ABSTRACT BODY:

Narrative: A herd of approximately 200 yearling cattle were driven from high mountain pasture in the early autumn. The animals were allowed to overnight in an area containing a mixture of Juniper trees, Sagebrush and oak brush. A small pond was also present to provide a water source for the night. The following morning, six yearling heifers were found dead along with numerous sick animals, many of which had moderate to severe dyspnea, blood in the feces and straining. The animals were then moved further down the mountain onto winter pasture. Several more animals died during the next few days, then the deaths resided.
The significant renal lesion was characterized by renal tubular necrosis of the convoluted tubules with the formation of granular and hyaline casts. In some tubules, necrosis of the epithelium was severe, leaving only a few tubular cells and bare basement membranes. In less severely affected tubules, fewer numbers of tubular cells were necrotic or missing. Intratubular hemorrhage was also observed along with necrotic epithelial cells within some of the tubules. Some tubular cells were less severely affected and contained only degenerative changes with no necrosis. The completeness of necrosis in groups of tubules with intratubular hemorrhage distinguishes the nephrosis of acute oak poisoning from that of most other causes. The clinical history of exposure to oak (*Quercus* sp.) and the presence of acute renal nephrosis

is diagnostic for oak poisoning. Oak poisoning in ruminants, and occasionally horses can be caused by the ingestion of oak leaves, stems and acorns. The toxic substances are gallotannins which are hydrolyzed to tannic acid, gallic acid and pyrogallol which appeared to be the active toxic metabolites. In Western Colorado cattle graze areas abundant with oak brush each year without incident. However, if the feed is limited and they graze heavily on the oak leaves or acorns oak toxicity can occur. Interestingly, the black bear (*Ursus americana*) graze heavily on oak brush in the late fall consuming large numbers of acorn with little or no effect

References:
1. Maxie MG, Newman SJ: 2007, UrinarySystem. In: Jubb, Kennedy and Palmer's Pathology of Domestic Animals, ed. Maxie MG. pp. 473. Saunders Elsevier, Philadelphia, PA.

CURRENT CATEGORY/DISCIPLINE: Pathology

AUTHORS/INSTITUTIONS: D. Kitchen, Department of Pathology, Colorado State University, Grand Junction, Colorado, UNITED STATES;

AWARDS:

Trainee Letter:

CONTROL ID: 2015935

TITLE: Pathology of Select Organs after Zilpaterol Supplementation and Variation in Select Hematologic and Anatomic Parameters

ABSTRACT BODY:

Narrative: Supplementation of zilpaterol hydrochloride (ZH; Zilmax®) to cattle has been unscientifically implicated as having a negative impact on cattle well-being. Currently, there is no scientific evidence to support or refute these claims. This study was designed to determine the effects of supplementation on hematologic profiles, select organ weights, organ histopathology and cardiac anatomic features at slaughter. Heifers (n=20; 556±7 kg BW) were separated into two groups: Control (CON): no ZH, or 2) Zilpaterol treated (ZIL): supplemented with ZH at 7.56 g/ton (DM basis). The trial was 25 d (-2 to 22d) in duration, with 3 blood-collection periods [-2 to 3d (ZH supplementation started on d 0); 12 to 15d, and 20 to 22d (withdrawal period)]. For each collection period, blood was collected via indwelling jugular catheters (inserted on d -3, 11, and 19) into EDTA tubes and submitted for a complete blood count. At slaughter animal, carcass, liver, kidney, lung, adrenal and heart weights were recorded. Heart necropsy included right ventricular weight, combined left ventricle and septal weights. The right AV, pulmonic, left AV, and aortic circumferences were recorded. Thickness of right and left ventricular free walls and interventricular septum were measured. Cardiac weight ratios and valve diameter ratios were compared. Organ to body carcass weight ratios were determined. No differences were found between groups in any of the standard leukogram parameters excepting hemoglobin ($p=0.01$) which was slightly higher in the ZIL group but within normal reference range and was this difference was present prior to treatment and remained constant. Liver ($p=0.02$) and kidney ($p=0.02$) weights as percent of carcass weight were reduced in treat cattle. Raw liver weight ($p=0.08$) and kidney ($p=0.11$) weights tended to be less and carcasses tended to be heavier in the ZH group. The data demonstrates a metabolic effect evidenced by reduction in hepatic and renal mass as a percentage of carcass weight. Other organ (heart, lung, adrenals) to carcass weight ratios remained similar suggesting this was not a pure carcass weight impact. No treatment associated histopathologic lesions were seen in the organs sampled. No pathologic effects of treatment were found. As cattle may be presented for necropsy late in the feeding period it is important to know the effects if any that beta agonist might have. The absence of any cardiac anatomic differences suggests that beta agonist is not likely a direct contributor to increased reports of cardiac failure in feedlot cattle.

CURRENT CATEGORY/DISCIPLINE: Pathology | Toxicology

AUTHORS/INSTITUTIONS: D. Steffen, School of Veterinary Medicine and Biomedical Science, University of Nebraska, Lincoln, Nebraska, UNITED STATES;
T. Schmitd, S. Jones, J. Buntyn, Department of Animal Science, University of Nebraska, Lincoln, Nebraska, UNITED STATES;
J. Carroll, Livestock Issues Research Unit (LIRU), USDA-ARS, Lubbock, Texas, UNITED STATES;

AWARDS:

Trainee Letter:

CONTROL ID: 2022927

TITLE: Sodium Toxicity in Neonatal Dairy Calves

ABSTRACT BODY:

Narrative: Ten dairy herds from 5 states submitted tissues from calves <14 days of age to the lab for work-up; all calves were fed milk replacer (MR); some calves had a history of abnormal gait, stiffness, muscle twitching, or seizures, but most had no neurologic signs other than lethargy and depression; morbidity and mortality rates ranged from 25% to 100%; most calves had diarrhea, but many were negative for our most common agents of diarrhea (*Cryptosporidium*, *rotavirus* and *coronavirus*). No consistent gross or histologic lesions were found; brains were almost always non-lesional; acute neuronal necrosis ("dead reds") was found rarely; no eosinophilic meningoencephalitis was seen; a few calves had pneumonia. When available, serum sodium level was found to be >155 mmol/L: calves are at risk of neurologic disease when serum sodium values exceed 160 mmol/L. Brain sodium levels were 1800 ppm or higher (wet matter basis); normal brain sodium value is <1400 ppm, and levels >1800 ppm are confirmatory of sodium toxicosis. In 6/10 herds, milk replacer or electrolytes were the source of excessive dietary sodium, often because softened water was used with a high sodium concentration (>500 ppm); mixing errors were also found. Water that has passed through a softener can have very high levels of sodium, particularly when the water is very hard; it should not be used to mix up commercial MR or used as a source of potable water unless it has been tested and verified to have low levels of sodium <100 ppm. In 4/10 herds, heat stress and feeding MR were thought to be the cause of sodium toxicosis. Since neonatal calves don't drink much water during the first week of life, mild dehydration and feeding MR may be sufficient to cause sodium toxicity in some herds. Submission of fresh brain to the laboratory is essential; serum, plasma, ocular fluid and cerebrospinal fluid (CSF) are also appropriate samples. MR can be submitted to the lab for percent total solids, Na concentration, and osmolality. Sodium toxicosis should be considered if a calf dies within 4-24 hours of onset of signs, with no evidence of neonatal calf diarrhea or other diseases (abomasal tympany or ulcer, omphalophlebitis, pneumonia, sepsis, meningoencephalitis). The likelihood of sodium toxicity increases if the calf has neurologic signs. Histologic cerebrocortical lesions of salt poisoning of neonatal calves may differ from those seen in pigs.

CURRENT CATEGORY/DISCIPLINE: Toxicology | Pathology

AUTHORS/INSTITUTIONS: D.C. Sockett, M. Behr, Wisconsin Veterinary Diagnostic Laboratory, University of Wisconsin-Madison, Madison, Wisconsin, UNITED STATES;

AWARDS:

Trainee Letter:

CONTROL ID: 2013882

TITLE: Hepatogenous Chronic Copper Toxicity in a Charolais Heifer

ABSTRACT BODY:

Narrative: A six-month old Charolais heifer presented for a two day history of lethargy and inappetence. The calf had been orphaned and was currently unweaned, received 6 L of commercial milk replacer twice daily and was kept in a 1-acre paddock of unimproved "weedy" pasture. On presentation, the calf exhibited severe icterus and was estimated to be 10% dehydrated with hard, pelleted feces. Blood collected for an assessment of the packed cell volume (PCV; 64%) and total solids concentration (6.0 mg/dL) was dark brown in color. Nitrate toxicity is the most common cause of methemoglobinemia in cattle but does not commonly cause icterus. Furthermore, clinical signs of nitrate toxicity were not evident. The owner was unaware of exposure to other exogenous oxidants that may cause methemoglobinemia. A complete blood count and serum biochemical profile revealed a mature neutrophilia and lymphocytosis and elevated concentrations of GGT, CK, AST, and bilirubin consistent with a hepatopathy. The heifer developed hemoglobinuria the following day; potential causes include leptospirosis, copper toxicity, water intoxication, alloimmune or autoimmune hemolytic anemia (IMHA), bacillary hemoglobinuria, postparturient hemoglobinuria, eperythrozoonosis, babesiosis and ingestion of toxic plants. Antibiotic therapy was initiated due to the possibility of bacterial infection; abdominal ultrasound was unremarkable and a liver biopsy was delayed due to the ongoing hemolysis. The calf was subsequently found to be seronegative for leptospirosis and anaplasmosis and the calf was treated with dexamethasone due to the possibility of IMHA. However, hemolysis continued over the next 72 hours and the PCV fell to 12%; a blood transfusion was performed using 3.5L of whole blood. The following day the patient appeared to stabilize and a liver biopsy was performed. Histopathologic analysis of the biopsy sample revealed extensive bridging fibrosis with lymphoplasmacytic pericholangitis suggestive of toxic hepatopathy. A plant-based etiology for the observed changes was suggested by pigmentary change from chlorophyll pigments. The copper concentration was found to be significantly elevated (210 ppm). Analyzed copper levels in the milk replacer were not elevated and local soils are not deficient in molybdenum or sulfate, which moderate copper levels by binding copper molecules. Hepatogenous chronic copper toxicity results after ingestion of hepatotoxic plants (e.g., *Senecio*, *Heliotropium* spp.) leads to liver damage and an increase in hepatocyte affinity for copper. This diagnosis was made by the presence of *Senecio* spp. in the pasture, elevated liver copper, histopathologic changes indicative of a plant based toxic hepatopathy, and absence of other copper sources. This case demonstrates the disease can be a diagnostic challenge and should be included on the differential list for patients exhibiting methemoglobinemia and signs of liver disease.

CURRENT CATEGORY/DISCIPLINE: Toxicology

AUTHORS/INSTITUTIONS: B. Newcomer, T. Passler, K. Joiner, Department of Pathobiology, College of Veterinary Medicine, Auburn University, Alabama, UNITED STATES;
B. Newcomer, D. Wolfe, M. Chamorro, T. Passler, Department of Clinical Sciences, College of Veterinary Medicine, Auburn University, Alabama, UNITED STATES;

AWARDS:

Trainee Letter: