Poult Enteritis and Mortality Syndrome (PEMS)

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Experimental poult day 3 post exposure (PE). Note fecal staining of feathers and watery brown droppings leaking out the vent. Mortality on day 3 is uncommon; peak mortality occurs on days 5-7 PE.
Dehydration and marked decrease in body weight rapidly follow onset of diarrhea. Affected bird (left) compared with Control (right). Note smaller size, dark shanks, and diarrhea.
Severe stunting is seen in PEMS survivors. Both poults are hatchmates, 21 days of age. Small poult is the only survivor out of a group of 14 (93% mortality) poults exposed by contact. Large poult is an unexposed control with body weight closest to the average for the group. Small bird weighed 28% of the average control weight.
Typical droppings (from the dead poult in the previous image). Note the florescent nature of the droppings indicative of a high protein content.
Occasional survivors have abnormally brittle feathers, which gives the "helicopter" appearance seen here and also described in runting/stunting.
Typical mortality curve for a severely affected flock. This was the "index" case. Mortality at 6 weeks was 43%. Normally a flock this badly affected would have been destroyed when mortality reached approximately 50%, but this flock was held longer to obtain samples and for study. Mortality during a 7 hour period on day 19 was 5%. A flock with total mortality due to PEMS of 96% has occurred.
Appearance of dead bird early in the course of disease. The abdomen is distended because of swollen, fluid-filled intestines. Dehydration and stunting are not apparent yet.
Note loss of muscle mass compared to normal Control (left). Not only is the Affected turkey (right) failing to grow and develop, it apparently is consuming its own tissues to survive.
Abdomen opened to show the pale, thin-walled, fluid-filled intestines typically seen in poulets with PEMS. These changes are not specific but can also be seen in many forms of enteritis in young turkeys.
Acute enteritis, day 4 PE. Saccular, fluid-filled, pale, thin intestines are typically seen in enteritis affecting poults including PEMS. A soft yellow-brown cast is present in one section.
Experimental PEMS, day 7 PE. Ceca markedly distended with watery, pale brown fluid.
Thymus of normal Control.
Thymus of a bird with PEMS. Note the marked atrophy. Although bursal atrophy and, to a lesser degree, splenic atrophy occur in poult's with PEMS, the thymus is generally the most severely affected of the lymphoid organs.
PEMS affected birds that are examined later in the course of the disease occasionally have bursal cores composed of inflammatory exudate, necrotic debris, and bacterial colonies. Although not entirely diagnostic, PEMS should be considered likely when this lesion is found. Current evidence indicates bursal cores form because of epithelial changes induced by turkey coronavirus.
Crop mycosis is often seen in PEMS-affected birds. This disease is believed to indicate immunologic dysfunction. PEMS is one cause of immune dysfunction in young turkeys.
Experimental PEMS, day 4 post-exposure (PE). Villi are contracting giving them a pleated appearance. Excess protein is present in the lumen. Note pale, swollen degenerating enterocytes (black arrow), and infiltration of inflammatory cells in the lamina propria (white arrows) at higher magnification.
Experimental PEMS; day 8 PE. Villi are no longer evident and there is crypt hyperplasia. Note abundant luminal contents, which contains very high numbers of bacteria.
Experimental PEMS, day 4 PE (Giemsa Stain). Often colonies of adherent bacteria can be identified associated with microerosions and inflammation expanding the villus giving it a "club" shape. In our studies they have not been observed prior to day 4. Shortly afterwards a marked heterophilia occurs and there are small foci of necrosis with intralesional bacteria in the spleen. Current research indicates the bacteria are enteropathogenic *Escherichia coli*, which can interact with viruses to cause PEMS.
Control, 21 days of age. Normal thymus.
Experimental PEMS, day 14 PE. Thymus is shrunken and has little, if any, recognizable cortical tissue. This change results from a combination of both atrophy and hypoplasia.
Control, 14 days of age. Normal bursa. Compare with bursa from a 15 day old poult exposed to PEMS 8 days previously. Magnification of both slides is the same.
Experimental PEMS; day 8 PE. Bursal follicles are markedly depleted of lymphocytes. As with the thymus, this results from the combination of loss of existing tissue (atrophy) and failure of the bursa to develop normally (hypoplasia).
Experimental PEMS, day 4 PE. Degeneration, necrosis, and inflammation of the epithelium are seen in turkeys with PEMS that are also infected with turkey coronavirus. Note heterophils.
Control, 21 days of age. Normal spleen. Note density of lymphocytes in periarteriolar lymphocytic sheaths (PALS).
Experimental PEMS, day 14 PE. Lymphocytic depletion; germinal centers are less affected than PALS lymphocytes.
Control, 21 days of age. Normal pancreas; note amount of zymogen in acinar cells and the uniform distribution of these cells.
Pancreas, experimental PEMS, day 3 PE. Acinar cells are markedly distended with zymogen. Note islet.
Pancreas, experimental PEMS, day 6 PE. Acinar cells surrounding islets remain engorged while those that are more peripheral are depleted and undergoing vacuolar degeneration with zymogen.
Thyroid, control, 21 days of age. Normal.
Experimental PEMS, day 14 PE 21 days of age. Thyroid follicles are distended with colloid and have flattened epithelium. Low levels of both T3 and T4 have been demonstrated in birds with these changes indicating hypothyroidism.
Cryptosporidia are commonly associated with severe outbreaks of PEMS. Experimental studies have not demonstrated a causal role for cryptosporidia in the disease. They may be another indicator of the immune dysfunction that occurs in PEMS.
Intestinal flagellates, especially *Cochlosoma* and *Spironucleus* (*Hexamita*) are also frequently associated with severe outbreaks of PEMS. Numerous *Cochlosoma* are apparent in the intestinal lumen.
Abnormal leukocytes in peripheral blood of a PEMS-affected poult. Identity and significance of these calls has not been determined.
Turkey coronavirus (TCV) visualized by direct electron microscopy. TCV occurs in some but not all cases of PEMS. The disease has been reproduced experimentally in birds exposed to TCV and enteropathogenic *Escherichia coli*.
Fluorescent antibody test for turkey coronavirus in bursal epithelium stains heavily for viral antigens. Note cytoplasmic fluorescence and absence of significant staining in underlying lymphoid follicles.
Indirect immunoperoxidase test for coronavirus in bursal epithelium. Viral antigen is not present in lymphocytes of the bursal follicles.
Proposed pathogenesis of PEMS. Interaction of at least two agents is required. One or more viruses initiate the process and cause stunting and immune dysfunction, which increases susceptibility to bacterial infections. Bacteria overwhelm the host and cause death. Damage to intestinal epithelium from other viruses (e.g. coronavirus), intestinal protozoa (e.g. cryptosporidia or flagellates), or possibly bacteria seems to also be necessary.
Potential points of intervention to reduce impact of PEMS on a flock. Biosecurity and good management practices have the greatest impact on disease control. Antibiotics can reduce impact on disease control. Antibiotics can reduce mortality but have little or no affect on stunting.
(Note: Older flocks and environment are not currently believed to be the primary means for introduction of causative agent. A reservoir with a vector is most likely.)

Reservoir - vector hypothesis. How many outbreaks result from introduction of causative agent via a vector from the reservoir and how many from subsequent secondary spread via movement of people among flocks is unknown.