Swine Diagnostic Pathology

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A working relationship between the laboratory and a practitioner is very important for diagnosis. The practitioners should be aware of what tests are available, what can be expected and how to interpret the results. Timeliness of the results reporting should be considered. Proper sample collection with sufficient history, packaging, and shipping help assure the accuracy of the laboratory results. When faced with a disease outbreak, occasionally, a strong presumptive disease diagnosis can be made based on clinical signs and epidemiological data before doing the necropsy.

However, necropsy is not the only tool to determine a definitive diagnosis. Development of antemortem methods such as agent isolation, serology tests and nucleic acid-based diagnostic methods becomes available for epidemiological investigation and rapid diagnosis. In addition, there are many limitations of postmortem diagnostic procedures. Firstly, necropsy is costly and not applicable to large scale screening. Lesions may be resolved by the time at necropsy or the changes may be subtle and not grossly visible. In most cases, a list of differential diagnoses should be documented after postmortem examination since many swine diseases often appear clinically similar. Since intensive swine production systems often commingling pigs from different sources that have different health and immune status, genetic background and many endogenous respiratory and gut floras, swine diseases are commonly attributable to more than one pathogen. In this manuscript, respiratory and enteric diseases are discussed in term of diagnostic pathology.

Upon necropsy, a pathologist should examine any discharge from the nostrils to facilitate diagnosis. Epistaxis is a clinical term used to denote presence of blood flow from the nose. Two methods are used to examine the nasal structures by making a midsagittal cut through the head or by making several transverse sections of the nose. In case of atrophic rhinitis, severity can be scored by several methods depending on the purpose of the study. Before the thorax has been opened, the diaphragm is punctured through the abdominal cavity to check whether negative pressure is present. Lack of the negative pressure may be an indication of advanced pneumothorax, pyothorax, pleural effusion, or presence of non-collapsible lung due to pulmonary edema or pneumonia. After removing the rib cage, pleural adhesion or abnormal thoracic contents must be observed or quantified. The tongue, pharynx, larynx, trachea, lung, and heart should be removed as a whole. The airway must be examined by opening along the dorsal midline from larynx to trachea and then extending the incision into the large bronchi of the caudal lobes. Tracheal swab can be done at this point in order to see the presence of the respiratory bacterial pathogens. Presence of foamy fluid indicates pulmonary edema. Foreign materials such as food contents may suggest broncho-aspiration. Blood aspiration is commonly seen at the slaughter-houses.

The lungs should be inspected for changes in color, texture, and distribution of lesions. Postmortem reports must also contain an estimate of the extent of the pulmonary lesions, preferably expressed as a percentage of the lungs affected.

- Lungs from exsanguinated and anemic animals are paler than normal due to the reduced blood volume in pulmonary tissue. Color changes can be various shades of red indicating congestion, hemorrhage, pneumonia, or atelectasis. Fibrotic lung is white pale and contracted.
- Palpation is required to differentiate the feeling of normal lungs from the pneumonic lungs. Texture is determined by gently palpating the surface and parenchyma of the lungs. Pneumonic lungs will have firm, hard, rubbery, or crepitant texture depending on exudates. Pulmonary abscesses can also be located by palpation.
- Distribution of lesions is generally described as focal, multifocal, locally extensive, or diffuse. Average lung lesion scoring can be done at this point in order to evaluate the severity of the lesion. Several bacterial organisms cause pneumonia and a few have characteristic lesions. Cranioventral pneumonia seen in Mycoplasma pneumonia is classified by its topography. When complicated with Pasteurella multocida (PM), exacerbation of the lesion called enzootic pneumonia is expected. Fibrinous pleuritis seen in those affected pigs can be caused by PM, Streptococcus suis or Haemophilus parasuis (Glasser’s disease). E. coli septicemia may produce fibrinous polyserositis coupled with fibrinous polyarthritis similar to Glasser’s disease and Streptococciosis. Bacterial culture and identification should be considered for the definitive diagnosis as well as sensitivity test. Fibrinous pericarditis is relatively common and accompanies those diseases mentioned before. Mycoplasma hyorhinis previously considered non-pathogenic respiratory agent may produce similar polyserositis and polyarthritis as well. In suckling pigs, scattered lobular red hepatization of lung strongly suggests Bordetella bronchiseptica (BB) infection. If survive, the affected lung heal with the fibrous tissue. In

S79
older pigs, BB is commonly associated with porcine respiratory disease complex (PRDC) mostly involving with respiratory viral pathogens such as porcine reproductive and respiratory syndrome virus (PRRSV), porcine circovirus type 2 (PCV2) or swine influenza virus (SIV) etc. underneath. Diffuse pneumonia caused by virus pneumonia or occasionally induced by septicemic salmonellosis should be determined by other sophisticated tests such as immunohistochemistry (IHC), in situ hybridization or polymerase chain reaction (PCR). Acute fibrinonecrotizing pleuropneumonia is commonly produced by *Actinobacillus pleuropneumoniae* or more frequently found in *Actinobacillus suis*-infected pigs lately.

Viral enteritis in suckling piglets can be differentiate from the bacterial enteritis by the absence of lacteal due to villous atrophy caused by virus pathogens. However, it should be done promptly with the presence of milk curd in the gut in order to avoid postmortem autolysis. Sections from different areas of the affected gut can be examined under the microscope. Those enteric diseases in grow-finish pigs include epizootic TGE, proliferative enteritis, enteric salmonellosis, whipworms, swine dysentery, and intestinal spirochetosis as well as classical swine fever or PCV2 infection. Since many enteric diseases mentioned above do not present initially with high mortality, a diagnosis by evaluation of clinical symptoms and collection/testing of fecal samples is very helpful. However, none of the fecal-based tests are as sensitive as the same tests when run on intestinal or colonic mucosa selected from the lesions especially in case of *Lawsonia intracellularis* PCR. Generally, lesions of the small intestine result in a normal frequency of high volume of stools that are watery, where as, lesions of large intestine result in a high frequency of a low volume of stool containing mucus. If affected both small and large intestine, a mixture of those clinical signs may be expected. When blood originates from the stomach or the small intestine, the feces appear brown due to being digested (dark in color) and mixed with normal green of feces. If originates from the large intestine, it remains undigested (red) in feces.

Besides necropsy, the most widely used techniques for enteric diseases are: direct smear of intestinal mucosa, microbiology (plus antibiotic sensitivity tests), detection of antibodies, histopathology, IHC, immunofluorescence (frozen tissue), and PCR tests on intestinal mucosa or feces. When conducting post-mortem examination, only acutely affected and typical untreated animals should be done. Careful postmortem examination especially of the jejunum, the entire ileum, the cecum and multiple segments of the spiral colon followed by testing for the specific pathogen is of useful for differential diagnosis. Enzootic TGE may cause the intestine dilated with watery contents (maldigestion). Diagnosis is accomplished by demonstrating villous atrophy in the small intestine or the presence of TGE antigen by IHC. Distribution and type of lesions may allow a tentative diagnosis. Lesions of severe ileitis caused by *Lawsonia intracellularis* are most consistent in the ileum followed by the proximal spiral colon, cecum and distal jejunum. Gross lesions of ileitis include thickening of the mucosa or having large amounts of undigested blood and sometimes having fibrinonecrotic debris in chronic cases. However, mild lesions are more difficult to diagnose. Microscopic lesions with silver stains like the Warthin-Starry stain are characteristic and diagnostic. PCR using ileal mucosa and immunohistochemistry on ileum sections are the two most sensitive and specific methods for this disease. Prevalence of ileitis can be conducted using in house IFA or a commercially available ELISA. Rare case of coccidiosis caused by *Eimeria spinosa* was reported in the boar stud with the clinical signs of mild, sporadic, transient diarrhea. Fibrinonecrotic ileitis was also found in this case. When antibiotic therapy does not appear to affect the clinical course of the disease, coccidiosis should be considered.

Fibrinonecrotic enterocolitis can be caused by many pathogens including *Salmonella* spp., *Brachyspira hyodysenteriae* or *Trichurus suis*. Other specific tests should be done in order to narrow down the cause of the diseases. Demonstration of *Salmonella* spp. either by culture or PCR with typical microscopic lesions in the intestine and liver may suggest Salmonellosis. In case of acute *B. hyodysenteriae* infection, gross and microscopic lesions are fairly unique and are often nearly diagnostic. Warthin-Starry stain demonstrating typical large snake-like spirochetal bacteria in the mucosa accompanied with culture, IHC or PCR are required for the diagnosis. No specific serologic assay is commercially available for the diagnosis of pigs with swine dysentery. Colonic spirochetosis is caused by attachment of *B. pilosicoli* to epithelial cells and persistence of *B. pilosicoli* in the cecal and colonic crypt lumina. Chronic inflammation is caused by spirochetal invasion into the subepithelial lamina propria and translocation to extraintestinal sites. Diagnosis for *B. pilosicoli* also requires demonstration of typical microscopic lesions and *B. pilosicoli* in the mucosa and by culture or PCR. Isolation of both *B. hyodysenteriae* and *B. pilosicoli* is also required for a definitive diagnosis. Direct smear on the lesion of the intestinal mucosa may help determine type of organisms especially *Balantidium coli*. When found in the trophozoit form, *B. coli* can be an opportunistic organism aggravating the lesions especially in the button ulcers (chronic salmonellosis). In addition, PCV-2 is able to induce clinical diarrhea in growing pigs and PCR or antigen-captured ELISA can be used to detect the presence of PCV-2 in the fecal sample.
If laboratory results do not explain the observed clinical situation or lesions, the possibilities below should be considered:

- detected microorganism is not the primary cause of the problem
- detected microorganism results from contamination
- detected microorganism is the true cause of the problem, however, on-farm diagnosis should be reconsidered.

In conclusion, the goals of diagnostic testing generally include reliably identifying the presence of a disease or disease agent in a pig farm. The ideal diagnostic test should be accurate, reliable, sensitive, specific, and consistent and be cost effective. The practitioners should be aware of the diagnostic process of any available laboratory in their areas in order to yield and make the most use of the laboratory and results.