Pathology of Swine

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Respiratory System
Diagnosis of Respiratory Diseases in Swine

• Complete Necropsy on ACUTE pigs
• Submit both fixed and refrigerated samples:
  – Lung, trachea, nasal turbinates
  – Brain, spleen, tonsil, liver, kidney, lymph nodes,
  – Serum
• Serology: coincident seroconversion
Gross Examination of the Lung Distribution

- Diffuse $\Rightarrow$ vascular or viral
- Dispersed multifocal $\Rightarrow$ vascular and/or embolic
- Lobular $\Rightarrow$ airway, segmental
- Cranioventral $\Rightarrow$ dependent
- Hilar $\Rightarrow$ App
Gross Examination of the Lung

**Color**

- Pink $\Rightarrow$ blood + air = normal
- Purple $\Rightarrow$ blood - air = atelectatic
- Red $\Rightarrow$ blood (congestion or hemorrhage)
- Red spots or blotches $\Rightarrow$ hemorrhages
- White $\Rightarrow$ WBC (Pus), Fibrin, Albumin
Gross Examination of the Lung

Consistency

- Soft, resilient $\rightarrow$ normal
- Fluctuant $\rightarrow$ edematous
- Firm, resilient $\rightarrow$ fibrinous
- Firm, unyielding $\rightarrow$ cellular infiltrates
- Hard, unyielding $\rightarrow$ scarring
- Crepitant $\rightarrow$ emphysema
Gross Examination of the Lung
Cross-Section

- Dry, friable $\Rightarrow$ necrosis
- White interlobular septae $\Rightarrow$ fibrin
- White grape-like clusters $\Rightarrow$ pus in alveoli
- Red blotches (interlobular septae) $\Rightarrow$ hemorrhage
- Glistening stringy clear or white material in airways $\Rightarrow$ mucous +/- WBC
Gross Examination of the Lung
Integration

- Lesions in lungs → differentials
- Lesions in other organs → differentials
- One disease? Systemic disease?
- Multiple diseases?
- Adequate samples?
normal
Progressive atrophic rhinitis

- Toxigenic *P. multocida* (usually type D) +/- co-infection with *B. bronchiseptica*
- *P. multocida* produces dermonecrototoxin > demise of osteoblasts > enhanced osteoclast activity > turbinate atrophy > distortion of nasal septum > possibly shortening and twisting of upper jaw
- Clinical signs: Sneezing (1-8 weeks of age) +/- epistaxis, blockage of lachrymal ducts with tear staining, mucopurulent nasal discharge, shortening of upper jaw and corrugation of the skin of snout
- Pathology: Rhinitis, turbinate atrophy and nasal distortion
- Diagnosis: Clinical signs confirmed by culture of nasal swab for toxigenic *P. multocida*
Atrophic rhinitis
Atrophic rhinitis
Atrophic rhinitis
Inclusion body rhinitis

- **Etiology**: Porcine cytomegalovirus
- **Clinical signs**: Sneezing in pigs <3 weeks of age, nasal discharge and epiphora. If chronic, may lead to otitis media
- **Pathology**: Rhinitis and conjunctivitis
- **Diagnosis**: Turbinates plugged with mucus/debris, basophilic intranuclear inclusion bodies in nasal mucosa
- If herd is naïve, (e.g. a new herd) any age can be affected and 25% mortality in affected litters may be seen
MAYBE NEXT TIME YOU'LL TRY A LITTLE SUNSCREEN...
Interstitial Pneumonia in Swine

• Viral
  – Swine influenza
  – Pseudorabies
  – PRRSV
  – PRCV
  – Porcine Circovirus

• Septicemia
  – S. cholerasuis
  – H. parasuis
  – S. suis
  – Other

• Allergic
  – Ascarid larval migration
Salmonella choleraesuis
Hemophilus parasuis
Influenza A

- Enveloped, negative sense RNA viruses that encode 10 major viral proteins on 8 independent segments of RNA
- Two major structural proteins (determine A, B, C):
  - Nucleoprotein
  - M1 matrix protein
- Envelope glucoproteins:
  - Hemagglutinin (H) – 15 subtypes
  - Neuraminidase (N) – 9 subtypes
- Historically only limited number of subtypes in mammals
- Subclinical infection of waterfowl with all subtypes
Swine Influenza

- Epizootic and endemic form
- Replicates in:
  - Epithelium of small airways within 2 h
  - Epithelium of nasal cavity, trachea, alveoli by 24 h > alveolar macrophages
- Small bronchi blocked by neutrophil-rich exudate
- Alveolar necrosis/bronchial epithelial hyperplasia causes clinical signs
- Type A
  - H1N1 later H3N2
  - also H1N2, H4N6
SIV Induced Disease

• Rapid course
  – incubation: 12-24 hours, almost 100% of animals
  – shed in nasal secretions for 4-5 days
  – recovery, if uncomplicated, in 7 days

• Symptoms
  – coughing
  – apathetic, prostrate, erythema of skin, anorexia
  – dyspnea, sneezing, pyrexia, “drop-off”
  – reddened eyes and conjuntival discharge
  – abortions
SIV Lesions

• **Gross**
  – “checkerboard” cranioventral pattern
  – diffuse interstitial pattern
  – firm (proliferative) cranioventral pattern

• **Microscopic**
  – necrotizing bronchiolitis
  – proliferation of type II pneumocytes
  – peribronchial and perivascular cuffing
Diagnosis of SIV

- Typical lesions
- Demonstration of SIV or antigens
  - FA/IFA lung
  - VI lung, nasal swabs
  - IHC fixed lung
  - ELISA nasal swabs, bronchial swabs
- Serology: paired samples
  - HI
SIV
Lung
As the two friends wandered through the snow on their way home, Piglet grinned to himself, thinking how lucky he was to have a best friend like Pooh.

Pooh thought to himself: ‘If the pig sneezes, he’s f**ken dead.'
Swine Influenza in Humans

- Pigs are important role in inter-species transmission, because they have receptors to both avian and human influenza virus strains: “mixing vessel”
- Outbreaks and sporadic human infection with swine influenza have been occasionally reported
- Influenza outbreak caused by swine H1N1 virus in Fort Dix, New Jersey in 1974
- Outbreak in Wisconsin in 1988 resulted in multiple human to human infections
- People in contact with swine have higher antibody levels
- Swine influenza viruses have been isolated from turkeys, indicating transmission between pigs and avian species
- Pigs can be infected with the highly pathogenic avian influenza (HPAI) H5N1 virus
Porcine Respiratory Coronavirus

- Deletion mutant of TGE virus
- Replicates in epithelium of nasal cavity and airways
- Shed in nasal secretions for up to 10 days
- Virus is transient in lungs
Symptoms and Lesions of PRCV

• Symptoms
  – usually none **seroconversion to TGE**
  – mild dyspnea, pyrexia, coughing

• Lesions
  – slight increased firmness of apical, cardiac lobes
  – mild interstitial pneumonia with necrosis of epithelium of terminal airways
Diagnosis of PRCV

- Typical lesions
- Demonstration of PRCV or antigen
  - FA lung
  - VI nasal swabs, lung
  - Differentiate from TGE: cDNA probe
  - PCR followed by cDNA probe
- Serology
  - SN for TGE and on sera > or = 1:64
  - Competitive ELISA
Pseudorabies
SIV + PRRSV
Ascaris suum
Ascaris suum
Ascaris suum

Lung
A. suis
Aspergillus fumigatus
Aspergillus fumigatus

Lung
Aspergillus fumigatus

Lung
Aspergillus fumigatus in Lung
Mycobacterium avium
Mycobacterium avium
Bacterial Pneumonia in Swine

Infection versus Disease

- subclinical upper respiratory bacterial infection ≠ pneumonia
- risk factors for bacterial pneumonia
  - impaired lung defenses
  - level of challenge
  - virulence of bacterial organism
Bacterial Initiators of Porcine Respiratory Disease Complex

Nursery Grower

“17-18 Week Wall”
increased morbidity
increased mortality
Lung Defenses

Native
Mucociliary Apparatus
Alveolar macrophages
Inflammation

Acquired
IgA
CMI
IgG
Damage to Lung Defenses

Mucociliary Apparatus
- M. hyopneumoniae
- SIV
- PRV
- PRCV
- PCV?
- Chilling
- high levels of ammonia

Macrophages
- PRRSV, APP, PCV?
- Stress, endogenous steroids
PRDC is a “Complex”

- Primary pathogens (Initiators)
  - *M. hyopneumoniae*
  - *A. pleuropneumoniae*
  - SIV
  - PRRSV
  - PRV
  - PRCV
  - PCV

- Secondary pathogens (Followers)
  - *P. multocida*
  - *S. suis*
  - *H. parasuis*
  - *B. bronchiseptica*
  - *A. pyogenes*
  - **A. pleuropneumoniae**
Risk Factors for PRDC

Primary Pathogens

Exposure Dose

Immunity
Prevention and Control of PRDC

- focus on Initiators
- farm or system specific
  - identify initiators
  - evaluate shedding and immunity
  - evaluate management practices, exposure
  - develop plan
  - work plan
  - monitor and re-evaluate
Increased Bacterial Challenge

- High stocking density
- Frequent mixing of pigs
- Large variation in age in pens or rooms
- ☠ 200 pigs per air space
- Low ventilation rates
- Non-solid partitions between pens
- Breaches in biosecurity
Virulence of Organisms

- **Primary Inhaled**
  - M. hyopneumoniae
  - A. pleuropneumoniae
  - B. bronchiseptica

- **Primary Blood-borne**
  - S. choleraesuis
  - A. suis
  - A. pyogenes

- **Secondary Inhaled**
  - P. multocida
  - S. suis
  - H. parasuis
  - M. hyorhinis*
  - A. pyogenes*
Mycoplasma hyopneumoniae

- fastidious pleomorphic bacterium
- infects only pigs
- colonizes respiratory epithelium - cilia
  - catarrhal bronchopneumonia - resolves ☞75d
  - secondary purulent bacterial bronchopneumonia
    - “Enzootic pneumonia”
  - protracted disease
- marked reduction in rate-of-gain
Mycoplasma hyopneumoniae

Epidemiology

• Transmission is inefficient
  – Nose-to-nose
    • Clinical disease 😞 Subclinical disease
    • Older animals 😞 Young animals
  – Aerosol

• Disease is dose-dependent
  – Subclinical infection vs. clinical disease
  – Incubation period: 11 days to 6 weeks
**Mycoplasma hyopneumoniae**

**Diagnosis**

- **Clinical signs**
  - Slowly spreading nonproductive cough
  - Depression in growth rate
  - Dyspnea, anorexia, death
- **Lesions:** Catarrhal bronchopneumonia
- **Confirmation:**
  - Antigen: Tissue FA, IHC
  - Nucleic Acid: PCR
  - *Antibodies: CF, ELISA*
Mycoplasma hyopneumoniae
Mycoplasma hyopneumoniae
Mycoplasma hyopneumoniae

Mycoplasma hyopneumoniae
Mycoplasma hyopneumoniae

Lung
Secondary Inhaled Pathogens
Purulent Bronchopneumonia

- upper respiratory commensals
- follow Mycoplasma or viral infections
- “Enzootic Pneumonia” - may not be!
- most common:
  - P. multocida
  - S. suis
  - H. parasuis
- diagnosis: culture
Pasteurella multocida

- common in nearly all swine herds
- non-toxigenic and toxigenic strains
- most common bacterial isolate from pneumonic lungs in slaughter swine
- lung isolates
  - most are capsular type A
  - most are serotypes 3 or 5 (of 16 total)
  - toxin as a virulence factor??
  - some strains: pleuritis, abscessation
Enzootic pneumonia
Enzootic pneumonia
A. pyogenes
Bordetella bronchiseptica

- primary or secondary inhaled pathogen
- Pathogenesis: Colonisation and destruction of cilia in upper respiratory tract, may colonize lung causing broncho-pneumonia (cranial and middle lobes)
- primary pathogen: first few weeks of life
- lobular necrohemorrhagic bronchopneumonia
  - Coughing, sneezing +/- epistaxis and mucopurulent nasal discharge, mild (reversible) turbinate atrophy (regressive atrophic rhinitis), death
- chronic progressive bronchopneumonia
  - Coughing and poor growth
Bordetella bronchiseptica
Bordetella bronchiseptica
Bordetella bronchiseptica

Lung
Bordetella bronchiseptica

Lung
Actinobacillus pleuropneumoniae

- fastidious encapsulated coccobacillus found only in swine
- biovar 1: require NAD for growth
- biovar 2: NAD not required for growth
- 15 serotypes
  - predominant serotypes vary by region
  - serotypes and strains vary in virulence
Actinobacillus pleuropneumoniae

**Virulence Factors & Epidemiology**

- **Virulence factors**
  - Capsule
  - Endotoxin
  - Exotoxins: APX I, II, III
    - Hemolysin APX I, II
    - Cytolysin APX I, II, III
- **Epidemiology**
  - Carriers: tonsil, nasal cavity, lung
  - Carriers may be seronegative
### Actinobacillus pleuropneumoniae Diagnosis

- **Clinical signs:**
  - most common in 3-6 month-old pigs
  - peracute - chronic
  - pyrexia, dyspnea, hemoptosis, death, anorexia

- **Lesions:** Fibriononecrotic hemorrhagic pleuropneumonia

- **Confirmation:**
  - Culture: fresh lung from untreated animals
  - Antigen: Agglutination, Co-agglutination, ELISA
  - Antibodies: CF, HN, ELISA
Actinobacillus pleuropneumoniae
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Actinobacillus pleuropneumoniae
Actinobacillus pleuropneumoniae
OH, D-D-DEAR DEAR... I SEEM TO HAVE SPILLED YOUR D-DINNER ALL OVER M-ME...

WINNIE THE POOH, ABOUT TO INVENT THE FIRST HONEY-ROASTED HAM
S. suis
Fumonisn intoxication
Fumonisin intoxication
Septicemia
<table>
<thead>
<tr>
<th>Cause</th>
<th>Type of Pneumonia</th>
<th>Ages Commonly Affected</th>
<th>Is Coughing Typical?</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aujeszky’s virus</strong></td>
<td>Interstitial pneumonia</td>
<td>Any age</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Swine Influenza virus</td>
<td>Interstitial pneumonia</td>
<td>Any age</td>
<td>Yes (loud, harsh)</td>
</tr>
<tr>
<td>PRRS virus</td>
<td>Interstitial pneumonia</td>
<td>Any age</td>
<td>No</td>
</tr>
<tr>
<td>Ascarid larval migration</td>
<td>Interstitial pneumonia</td>
<td>After weaning</td>
<td>No</td>
</tr>
<tr>
<td><strong>S. choleraesuis septicemia</strong></td>
<td>Interstitial pneumonia</td>
<td>1 – 5 months</td>
<td>No</td>
</tr>
<tr>
<td><strong>Mycoplasma hyopneumoniae</strong></td>
<td>Bronchopneumonia</td>
<td>1 ½ – 6 months</td>
<td>Yes (dry, soft)</td>
</tr>
<tr>
<td><strong>Actinobacillus pleuropneumoniae</strong></td>
<td>Pleurropneumonia</td>
<td>Any age</td>
<td>No</td>
</tr>
<tr>
<td><strong>Bordetella bronchiseptica</strong></td>
<td>Bronchopneumonia</td>
<td>1-5 weeks</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Pasteurella multocida</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Streptococcus suis</strong></td>
<td>Secondary purulent bronchopneumonia</td>
<td>Any age</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Haemophilus parasuis</strong></td>
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<tr>
<td><strong>Actinomyces pyogenes</strong></td>
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</tr>
</tbody>
</table>
Metastrongylus elongatus
Metastrongylus elongatus
Metastrongylus elongatus

Lung
Diaphragmatic hernia
Carboxyhemoglobinemia
Cardiovascular System
Mulberry heart disease
Mulberry heart disease
Mulberry heart disease
Mulberry heart disease

Heart
Gossypol intoxication
Ascaris suum
Erysipelas
Actinobacillus suis
A. pyogenes
Arcanobacterium pyogenes
Hypervitaminosis D