APICOMPLEXA 3

Apicomplexa of Blood Pathology

Babesia, Cytauxzoon

***Babesia vogeli* (aka *Babesia canis,* or *B. canis vogeli*)**
Large Piroplasm of Canids

A. Morphology
- Trophozoites in RBC: pair of large tear-shaped organisms (piroplasms)

B. Indirect Life Cycle (vertebrate host and Ixodid tick host)
1. Canid Intermediate Host
   a. Transmission -- Sporozoites injected by tick host
   b. Invasion -- Sporozoites invade erythrocytes (RBC)
   c. Asexual reproduction
      i. Merogony (schizogony) [multi-nuclear division followed by cytoplasmic division]
      ii. Merozoites burst out of the RBC and infect other RBC's, then goes through merogony again.
      iii. The mass destruction of RBCs during multiple asexual cycles is cause of disease.
   d. Sexual reproduction
      i. Some merozoites exit the RBCs, infect other RBCs, and go through gametogony (production of gametes). Macrogametes (egg) and Microgametes (sperm)
      ii. The gametes within the RBCs are then ingested by a tick host with its blood meal.
2. Tick Definitive Host and also the Vector
   a. *Rhipicephalus sanguineus* (Brown Dog Tick, aka Kennel Tick)
      i. Can reproduce and live indoors
      ii. 3-host tick => hard to eradicate
   b. Transmission -- Gametes are ingested by tick host
   c. Fertilization – Microgametes fertilize Macrogametes in the tick gut
   d. The resulting zygote goes through Sporogony
   e. Sporozoites invade various tick organs and cells
      i. Salivary glands => transmission to the next dog
      ii. Ovaries => Transovarian & Transstadial transmission
         1. Transovarian = parasite from mother tick to offspring (trans-generational)
         2. Transstadial = tick retains parasite infection throughout its life stages - from egg to larvae to nymph to adult tick
   f. When the tick feeds it transfers sporozoites to the dog host.
3. Can be transmitted via blood transfusions

C. Pathogenesis
1. Pathology results from the mass destruction of the RBCs, and subsequent host immune response.

D. Clinical Disease in Canids
1. Sub-clinical to clinical disease
2. Clinical disease usually acute
   a. Fever, thrombocytopenia (more common), anemia, depression, anorexia, lethargy, splenomegaly
   b. Puppies more likely to show clinical disease, present with anemia
   c. Infected RBCs are sticky and stick together forming micro-emboli, thus potential ischemia in CNS or Kidneys.

E. Diagnosis
    1. Blood smear demonstrating intra-erythrocytic organisms
        a. Use capillary blood -- 1st drop from skin puncture
    2. PCR molecular assay most senstivie and can Id species (species id important for appropriate treatment -tx different for large vs. small *Babesia* spp.)
    3. Serology using IFA; 5% of dogs in NC are sero-positive
F. Treatment
    1. Imidocarb dipropionate injections (large *Babesia* sp.)
    2. Don't immunosuppress while treating because the drugs are static and need immune system to help clear pathogen.
    3. Supportive fluid therapy
G. Control
    1. Tick control in US
        a. Spot-ons, collars, systemics, etc.
        b. Bio-surveillance and control for ticks in kennels.
    2. Surveillance of Transfusion Blood
    3. Vaccines in Europe
H. Epidemiology
    1. In general, disease widely distributed
    2. But high prevalence in Florida Grey hounds
        a. Ecology -- ?? Dog racing kennels infested with the kennel tick ??
    3. Reinfection can occur

==========

Babesia gibsoni
Small Piroplasm of Canids

A. Morphology
- Trophozoites in RBC: single, small round to oval organisms (piroplasms)

B. Natural Indirect Life Cycle (vertebrate host and Ixodid tick host)
    1. Definitive Host – Canids
    2. Intermediate Host (Vector) - *Rhipicephalus sanguineus? Haemaphysalis longicornis?*
        a. *Rs* = Brown Dog Tick, aka Kennel Tick;
        b. *Rs* can reproduce and live indoors
        c. 3-host tick => hard to eradicate
        d. IH = *Haemaphysalis longicornis* (Asian Longhorned tick) -not native to US (relatively new in US since 2010, ~11 states including NC); not clear if *H. longicornis* transmits *B. gibsoni* in the US.
        e. 3-host tick => hard to eradicate
        f. Females can reproduce without mating
    3. See *Babesia vogeli* notes above for detailed Life Cycle
    4. Can be transmitted via blood transfusions

C. Behavioral "Direct" Life Cycle
    1. Definitive Host – Canids
    2. "Reservoir" Host --- Infected Canids
    3. Behavioral "Direct"
        a. Direct transfer of organism via wounds received during dog fights
        b. High prevalence in American Pit Bull Terriers

D. Pathogenesis
    1. Pathology results from the mass destruction of the RBCs, and subsequent host immune response.

D. Clinical Disease in Canids
    1. Sub-clinical to fatal
    2. Clinical disease usually acute
        a. Fever, thrombocytopenia (low platelets), hemolytic anemia, hyperglobulinemia, splenomegaly, lymphadenomegaly, depression, anorexia, lethargy, vomiting
    3. History of a past dog fight is suspect
E. Diagnosis
    1. Blood smear demonstrating intra-erythrocytic organisms
    2. PCR molecular assay most sensitive and can Id species
F. Treatment
    1. Atovaquone and Azithromycin combination, oral treatment
    2. Don't immunosuppress while treating because the drugs are static and need immune system to help clear pathogen.
    3. Supportive fluid therapy
    4. Effect a recovery and decrease parasitemia; but may not totally cure; may remain carriers.
G. Control
    1. Tick control in US
        a. Spot-ons, collars, systemics, etc.
        b. Bio-surveillance and control for ticks in kennels.
    2. Canine behavioral "modifications"
    3. Surveillance of Transfusion Blood

==========

Cytauxzoon felis
Piroplasm of Felids

A. Morphology -- Trophozoites in RBC: single, small round to oval organisms (piroplasms)
B. Indirect Life Cycle (vertebrae host and Ixodid tick host)
    1. Definitive Host – Felids
    2. Intermediate Host (Vector) – *Amblyomma americanum* /*Dermacentor variabilis*
        a. Lone Star Tick / American Dog Tick
        b. Outdoor ticks
        c. 3-host ticks => hard to eradicate
    3. See *Babesia vogeli* notes above for detailed Life Cycle
C. Pathogenesis
    1. Early disease: Minimal pathology - destruction of RBCs, and some host immune response.
    2. Quickly progresses to death due to giant macrophages packed with schizonts occlude small blood vessels causing multi-organ failure and death.
D. Clinical Disease in Felids
    1. Often an acute rapidly-developing fatal disease
    2. Clinical disease
        a. Acute Fever, Pancytopenia [anemia (low RBCs), leukopenia (low WBCs), thrombocytopenia (low platelets)], hyperbilirubinemia, icterus, depression, anorexia, dehydration, death
        b. Death occurs within a few days
E. Diagnosis
    1. Clinical Signs + Acute febrile disease
    2. Blood smear may demonstrate intra-erythrocytic organisms (piroplasms)
    3. Late stage blood films may demonstrate giant macrophages packed with schizonts (most often seen toward the feathered edge)
F. Treatment
    1. Atovaquone + Azithromycin (preferred) OR Imidocarb
    2. Supportive fluid therapy
    3. May or may not effect a recovery (~60% survive with A+A tx)
G. Control
    1. Tick control in US
        a. Spot-ons, collars, systemics, etc.
H. Epidemiology
    1. In general, sporadic, in South Central and Southeastern US
    2. Sylvatic host – Bob Cats (*Lynx rufus*)
    3. Large Cats – Florida Panthers and a captive Tiger - fatal cases

==========