CANINE BABESIOSIS IN THE DEVELOPING WORLD

VECTOR BORNE DISEASE

MSD Animal Health

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Andrew Leisewitz graduated from the Veterinary Faculty of the University of Pretoria at Onderstepoort (and the only veterinary school in South Africa) in 1987. He gained an Honors degree soon after this and then completed a residency and Masters degree in small animal medicine in 1995. He went on to become a Diplomat of the European College of Internal Medicine in 2003. He started as a junior academic in the Veterinary Faculty in 1990 and his research focus has been canine babesiosis since then. He has published widely on this disease (over 50 publications) and as an active clinician has spent over 30 years managing this common cause of morbidity and mortality in the Veterinary Academic Hospital. He is currently a full professor of small animal medicine and runs an active research program in canine babesiosis.

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WHERE IS THE DISEASE MOST LIKELY TO BE FOUND?

Geography

Canine babesiosis in **sub-Saharan Africa** is responsible for significant morbidity and mortality.

Veterinary care in most of this region of the world is very limited and basic care such as vaccination, ecto- and endoparasite control and treatment for common infectious disease is out of reach for the average dog owner.

Surveys of blood parasites infecting domestic dogs in the developing world are comparatively few; therefore, the geographic occurrence of canid *Babesia* in these regions is less well defined than for the developed world.

Climate change (and with it altered vector biology and distribution) and pet owner mobility mean that there is an ever-increasing need for awareness of **infections circulating in domestic dogs** in these relatively neglected areas.

Sub-Saharan Africa

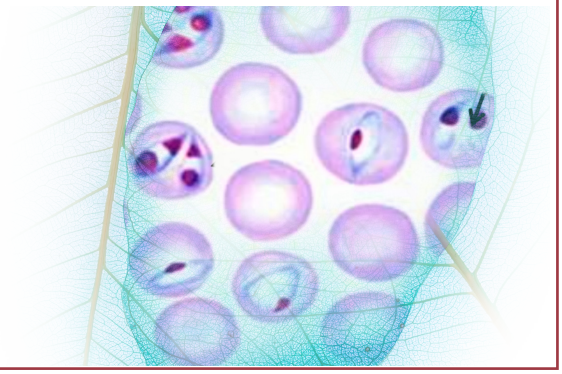
Although the traditional developed world view for why people own and keep dogs is not typical in this region of the world, most dogs in African settlements are owned and cared for by households and are kept for reasons that focus on security, shepherding, hunting as well as (although to a lesser degree) companionship.

Infections circulating in domestic dogs

The presence of *Babesia rossi* (the most pathogenic of the *Babesia* parasites infecting dogs) is unique to sub-Saharan Africa where it has a wide distribution and affects a massive dog population.

Babesia vogeli also has a wide distribution in this region but it is not as clearly a cause of disease, particularly in adult dogs.

There are no reports of *B. canis* or *B. gibsoni* being endemic to this part of the world.



WHERE IS THE DISEASE MOST LIKELY TO BE FOUND?

Local environment

Most reports of canine babesiosis emanate from **South Africa** where around 10% of dogs seeking veterinary care in the summer are diagnosed with this disease.

Because most dogs in Africa have no or minimal access to veterinary care, there is almost no data on the incidence of babesiosis on the continent.

> Thirty-one percent of dogs presenting to the Outpatients clinic for veterinary care at the Onderstepoort Veterinary Academic Hospital that were diagnosed with babesiosis were sick enough to warrant admission.

Therefore, the risk of this disease generally remains high because **most dogs live in underprivileged areas** and are not treated for ectoparasites.

The impact of climate change, environmental degradation, human migration, poverty and political instability on human vector borne disease (such as malaria) indicates there is every reason to believe that this dog disease has the potential for continuing spread.

Favorable climatic conditions

In South Africa most of the country is a summer rainfall area and *B. rossi* is more common in the summer months. In the small area of the country that has a winter rainfall, the disease peaks in winter.

Climatic change could be a factor leading to increased occurrence with longer periods of warm temperatures.

Evidence of disease spread

There is **no evidence** that *B. rossi* is spreading. The parasite is vector specific and the disease distribution mirrors the distribution of the single known tick vector.

> There is one report of an imported case in Texas, USA.

It is not known whether ticks non-endemic to sub-Saharan Africa that are competent vectors of other dog *Babesia* parasites could also transmit *B. rossi.*

HOW DOES A DOG **BECOME INFECTED?**

Vector

B. rossi is only transmitted by the ixodid tick *Haemaphysalis elliptica* which has a distribution restricted to the warm moist regions of sub-saharan Africa.

Proportion of vectors infected

Experimental work suggests that <20% of *H. elliptica* are infected. It has been shown that almost all dogs diagnosed with *B. rossi* carried *H. elliptica.*

Reservoir(s)

The hosts of adult *H. elliptica* are various carnivore species, among which are the domestic dog, domestic cat, lion *(Panthera leo)*, and leopard *(Panthera pardus).*

The hosts of the immature stages are diverse rodent species, and they may very occasionally be present on the same hosts as the adults.

The **Black-Backed Jackal** (*Canis mesomelas)* is the natural wildlife reservoir host, carrying the parasite but without clinical signs.

The **domestic dog** appears to be a much more recent spill-over host and hence the severe disease caused by infection.

Black-Backed Jackal and parasite

The jackal and parasite have apparently co-existed for millennia and as a result the host-parasite interaction no longer causes disease. The domestic dog appears to be a much more recent spill-over host and hence the severe disease caused by infection.

Domestic dogs

Dogs can become infected and develop a transient parasitemia which then self cures resulting in sero-conversion. Some dogs can be positive on blood smear but disease free. Such dogs may act as an infection reservoir. Very low infectious doses of parasite may be cleared by the host without developing disease.

Probability of transmission

A study that used *H. elliptica* ticks fed on *B. rossi* infected dogs to transmit the infection to naïve dogs found that 10 of 13 dogs developed clinical disease and a patent parasitemia.

Transmission was slow and took longer than 24 hours following tick attachment.

Transmission mechanics

The tick vector is by far the most common means of parasite transmission.

Only adult ticks (not immature stages) apparently transmit the infection although the parasite is transferred transovarially within the tick population.

Other routes of transmission

Transmission by blood transfusion is plausible, but there is no evidence that this has occurred. There are anecdotal reports of transmission *in utero* in dogs. There is no evidence of transmission by dog bite.

Transmission from an attached tick requires a longer period of attachment, greater than 24 hours and possibly greater than 48 hours.

WHAT BEHAVIORS
PUT A DOG AT RISK FOR THE DISEASE?

Activities

Behavior that increases tick exposure risks logically increases infection risks.

- Animals from environments where there is minimal veterinary care would also be at increased risk as ectoparasite control is poor in these areas.
- The increased adoption of isoxazoline compounds for ectoparasite control in urban areas has apparently reduced the disease burden within these communities.

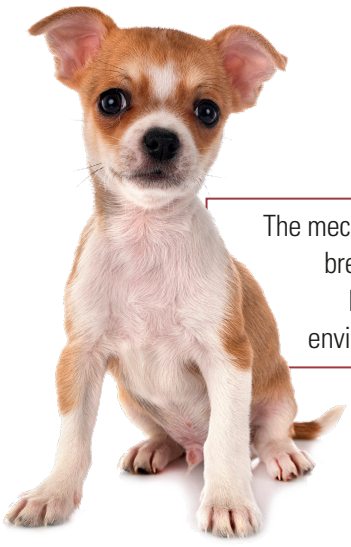
Time of day for increased exposure

The extended tick feeding time required for transmission means that there is no association with time of day and risk exposure.

Breed-related risks

- In one South African study, intact male, neutered male, and neutered female dogs were at increased risk for canine babesiosis compared to intact females.
- In another study the median age of 320 cases was 20 months with significantly more males presenting.
- In a study of 662 hospitalized dogs with babesiosis the traditional 'fighting breeds' (Staffordshire Terriers, Bull Terriers and Pitt Bull Terriers) were overrepresented in the group that developed complicated disease and died.

Several dog breeds, notably **toy breeds**, had a lower risk of babesiosis in a hospital population of dogs in South Africa.



The mechanism by which toy breeds are protected is likely due to reduced environmental exposure.

WHAT BEHAVIORS PUT A DOG AT RISK FOR THE DISEASE?

Diet

No effect of diet has been shown and diet is not believed to be a factor in the risk of disease.

Contact with other animals

A diverse group of rodents are hosts for the **immature stages** of the vector (*H. elliptica*). On occasions, immature stages may also infest the same hosts as the adults.

The **adult stage** infests various domestic and wild carnivores.

The tropical and subtropical nature of sub-Saharan Africa means that these hosts are abundant, and the parasite is easily maintained and widely spread and can be expected to be seen especially in domestic dogs in peri-urban and rural settings.

CAN A DOG BE INFECTED AND **NOT SHOW SIGNS?**

Risk to the population from sub-clinically diseased dogs

Sub-clinically infected dogs could be a source of ongoing infection for the tick vector.

Risk of subclinical disease and tests that reveal a sub clinically infected dog

A small number of apparently healthy dogs in a field study were blood smear and PCR positive for *B. rossi* without clinical disease. A larger proportion of dogs in this study were sero-positive for *B. rossi*. Seropositivity indicates evidence of previous exposure and not necessarily disease. *B. rossi* prevalence is unknown in seropositive dogs in areas where there is no veterinary care and no treatment for ill dogs. Occurrence of *B. vogeli* (non-pathogenic) in the same geographic region may confuse the situation because these parasites cannot be distinguished from one another on a blood smear. Molecular methods are required to distinguish these two parasites.

WHAT CLINICAL SIGNS DOES A SICK DOG SHOW AND WHY?

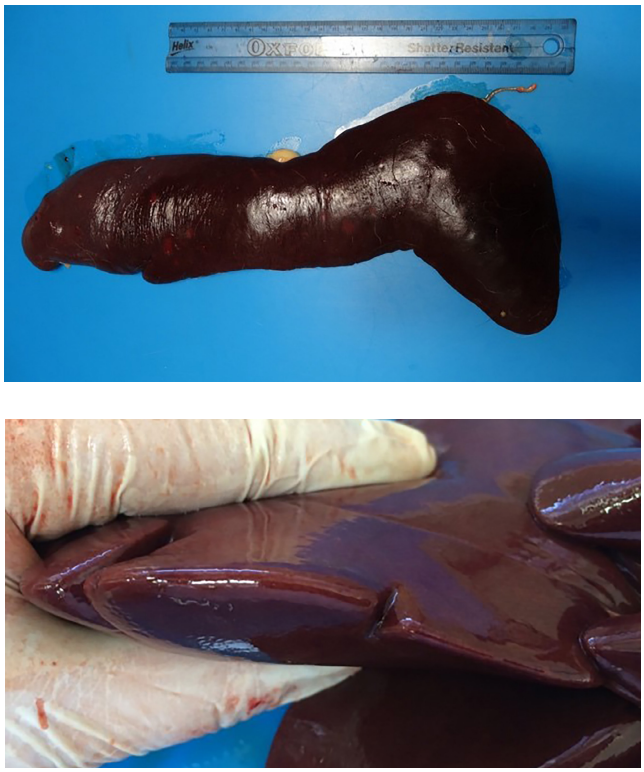
Early signs

- Loss of appetite with signs of nausea and depression is the **first sign** owners usually notice.

Most dogs will present for veterinary care after having become progressively more anorexic and depressed over 1-3 days.

At presentation most dogs are still ambulatory and walk into the hospital.

- Some owners describe dark or red urine (a result of bilirubinuria or hemoglobinuria).
- Hepatomegaly and splenomegaly are almost always detected on abdominal palpation.
- In rare cases the disease is hyperacute and dogs seizure (characteristic of cerebral disease or hypoglycemia) or are collapsed in shock (characteristic of the hemo-concentrating form of the disease).

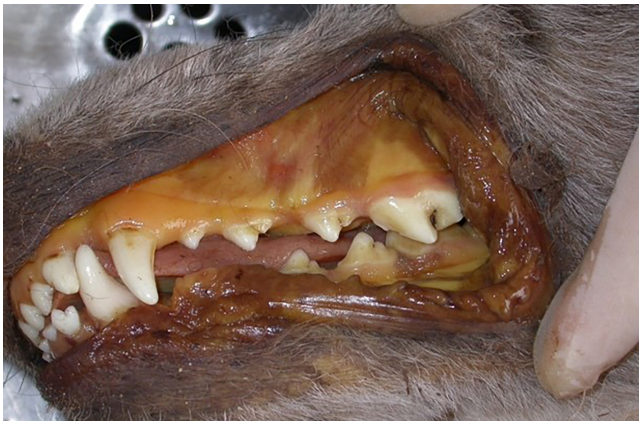


The very enlarged spleen and liver of a dog that succumbed to *Babesia rossi* infection. Splenomegaly is almost always easily detected on abdominal palpation.

Progression

- As disease progresses, dogs become weaker and their mucous membranes become more pale (consistent with worsening anemia).
- Icterus is a common finding in more established infections.

The rate of disease progression can vary widely from a rare per-acute form (a dog that was fine in the morning and found dead in the evening) to a much slower evolution over 10 to 14 days.



Icterus is common in more established disease and is largely prehepatic (as a result of hemolysis). Dramatic yellowing of the gums is easily seen in this image.

WHAT CLINICAL SIGNS DOES A SICK DOG SHOW AND WHY?

Prognostic factors

Compared to other babesial organisms that infect dogs, *B. rossi* is the most virulent.

Clinically the disease is classified as complicated or uncomplicated with the complicated forms having a significantly poorer prognosis.

Uncomplicated disease

Defined as a case well enough to be treated as an outpatient (not admitted) with a parasiticide and no other supportive treatment.

Complicated disease

Defined as evidence of single or multiple organ dysfunction or failure requiring hospital admission.

Collapse is a common finding in severe disease as a result of *Babesia rossi* infection and is associated with a poorer prognosis.

Mortality

Mortality is between 10 and 15% of hospital admitted cases. In a series of 320 naturally infected admitted cases, 22.5% of cases were collapsed (unable to stand unaided) at presentation and the odds ratio for death with collapse was 8.36. Almost all deaths occurred within 24 hours of hospitalization.

Factors that had a significant odds ratio for death

- Collapse at presentation
- A high band neutrophil count (consistent with the severity of the inflammatory nature of the disease)
- Hypoglycemia
- Hyperlactatemia
- Increased serum urea (which correlates poorly with creatinine and seems unrelated to renal function in many cases)
- Increased serum creatinine (which is always associated with an elevated urea)
- Hyperbilirubinemia
- Hypercortisolemia
- A low total thyroid hormone (TT4) concentration

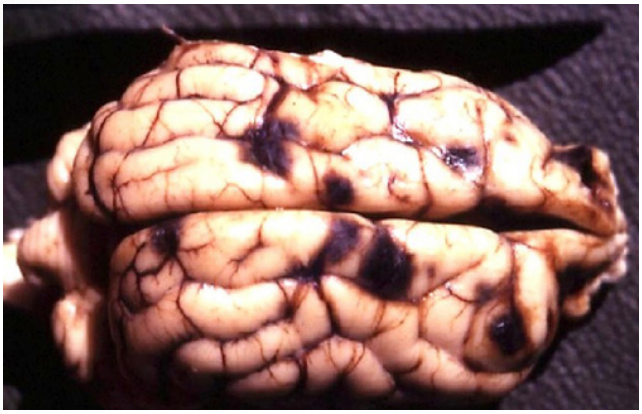
Receiver Operator Curves (ROC) highlight that TT4, total bilirubin, serum urea and cortisol at admission are most predictive of death.

Prognostic factors

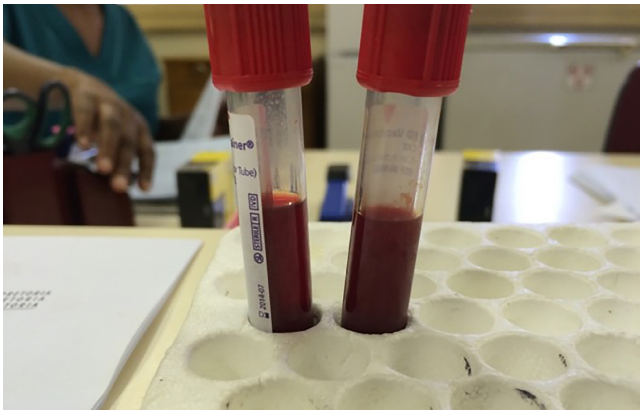
Complications that carry a very poor prognosis include **cerebral disease and hemoconcentration** (which occurs together with **severe hemolysis** and is usually associated with **acute respiratory distress syndrome** and/or **acute oliguric renal failure**).

Anemia is a very common presentation (>60% of 320 cases had a hematocrit below 0.25 L/L, 35% were below 15 L/L) but the odds ratio for death was not significant for anemia.

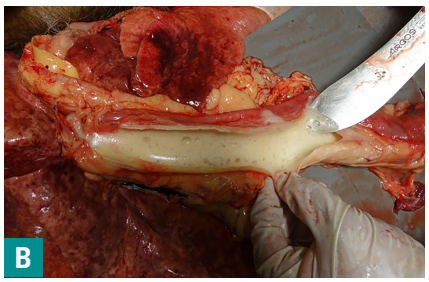
Severe anemia likely has a relatively minor effect on outcome because of the ease with which blood transfusion corrects this otherwise serious complication. Even severe anemia (hematocrit <10 L/L), if it is a sole complication, is not a poor prognostic indicator. The lowest hematocrit that the author has successfully treated was 0.04 L/L.

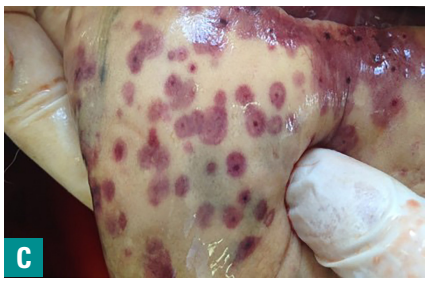


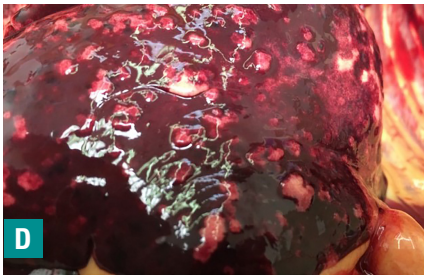
A *post mortem* image of the surface of the cerebral hemispheres of a dog that died per-acutely as a result of cerebral babesiosis. There are dark red areas of hemorrhagic encephalomalacia visible on the brain surface.



The severe hemolysis induced by *Babesia rossi* can result in profound hemoglobinemia, sometimes so severe that distinguishing the red cell – serum/plasma interface in centrifuged blood tubes becomes very difficult.

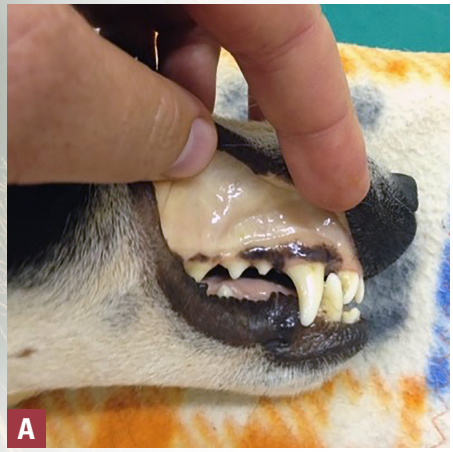


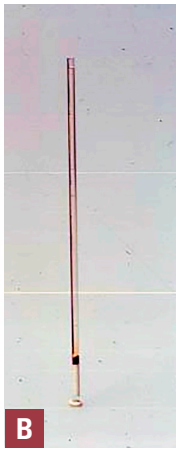




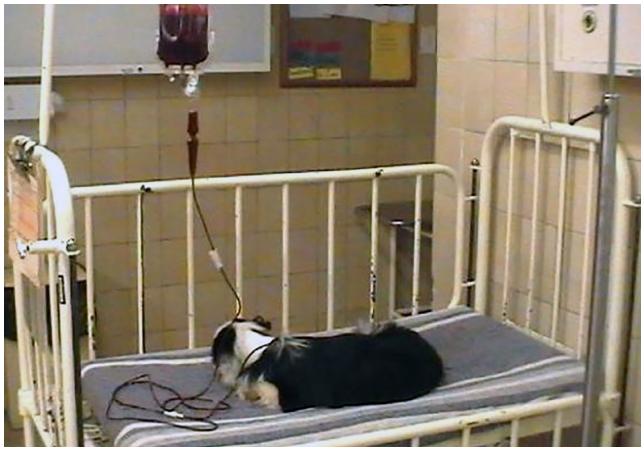
Agonal bloody foam expectoration is sometimes seen as a result of acute respiratory distress syndrome (A and B). The tissue changes associated with acute lung injury (C) and acute respiratory distress syndrome (D) are common in severe *Babesia rossi* infections.

CAN A DOG BE INFECTED AND **NOT SHOW SIGNS?**

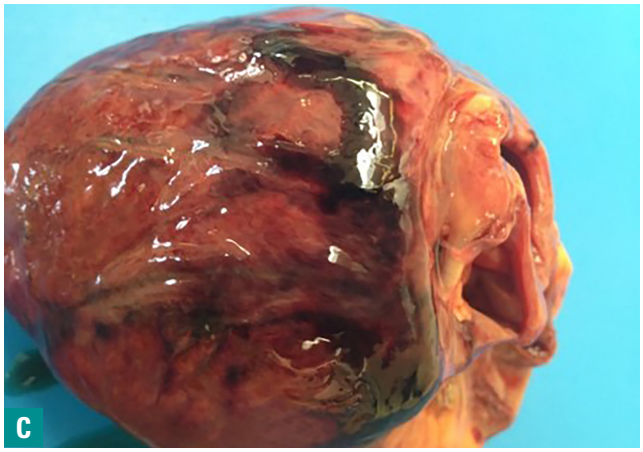


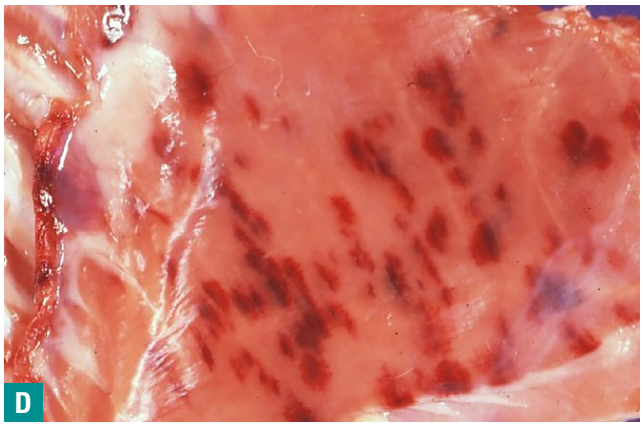


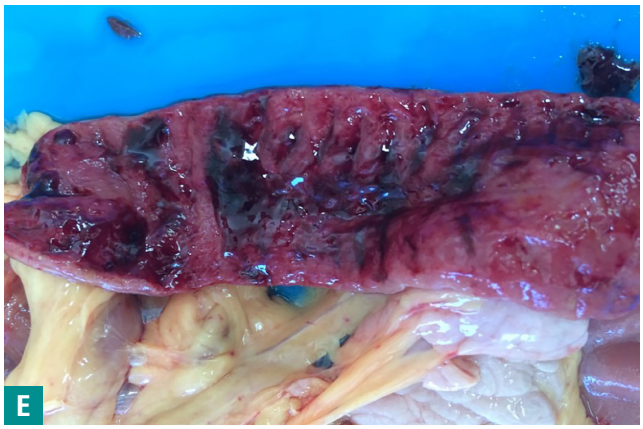
Anemia (causing the pale mucous membranes seen in this dog) is a very common clinical finding (A). Hematocrit is a simple and cheap means of determining the degree of anemia – which can at times be profound, as in this case (B).

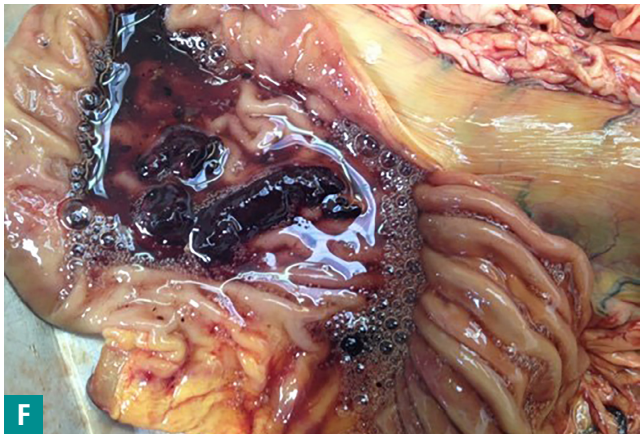


Blood transfusion (preferably packed cells) is a crucial and lifesaving intervention that has a significant effect on improving survival in *Babesia rossi* infected and anemic dogs (hematocrit <0.15L/L).









Macroscopic or clinical bleeding is a rare finding in the living dog, however, *Babesia rossi* causes obvious hemorrhage in a wide range of organs that can be seen on post mortem. (C) epicardial hemorrhage; (D) ecchymoses in the diaphragm; (E) hemorrhage into the small intestine; and (F) gastrorrhagia.

CAN A DOG BE INFECTED AND **NOT SHOW SIGNS?**

Prognostic factors

- **Thrombocytopenia** is present in close to 100% of cases but clinical hemorrhage is very rare although on post mortem examination nearly all cases have obvious bleeding in various organs.
- DIC is a described but rare complication with a poor prognosis.
- Severe mixed acid base abnormalities are usually seen in cases with a poor prognosis (concurrent metabolic acidosis and respiratory alkalosis). These changes are typically seen with **deep and labored breathing** (Kussmaul breathing).
- Warm in-saline-agglutination (ISA) is present in around 30% of cases but does not appear to impact on mortality.
- Prognosis has also been associated with the increased concentrations of various cytokines (TNF, MCP-1 and IL-6 in particular).

High parasitemia is associated with more severe disease.

Recovery indications

Uncomplicated cases (treated as outpatients) recover their habitus and appetite within 24 hours of treatment.

Complicated cases admitted for care typically spend 3 – 5 days in hospital with at least 2 of these in a high care or intensive care facility. Most deaths occur within 24 hours of hospitalization.

Recovery indications

Parasitemia

Parasitemia falls quickly following parasiticidal treatment and is usually cleared by 24 hours after treatment.

Pyrexia

Pyrexia resolves within the same time frame.

Anemia

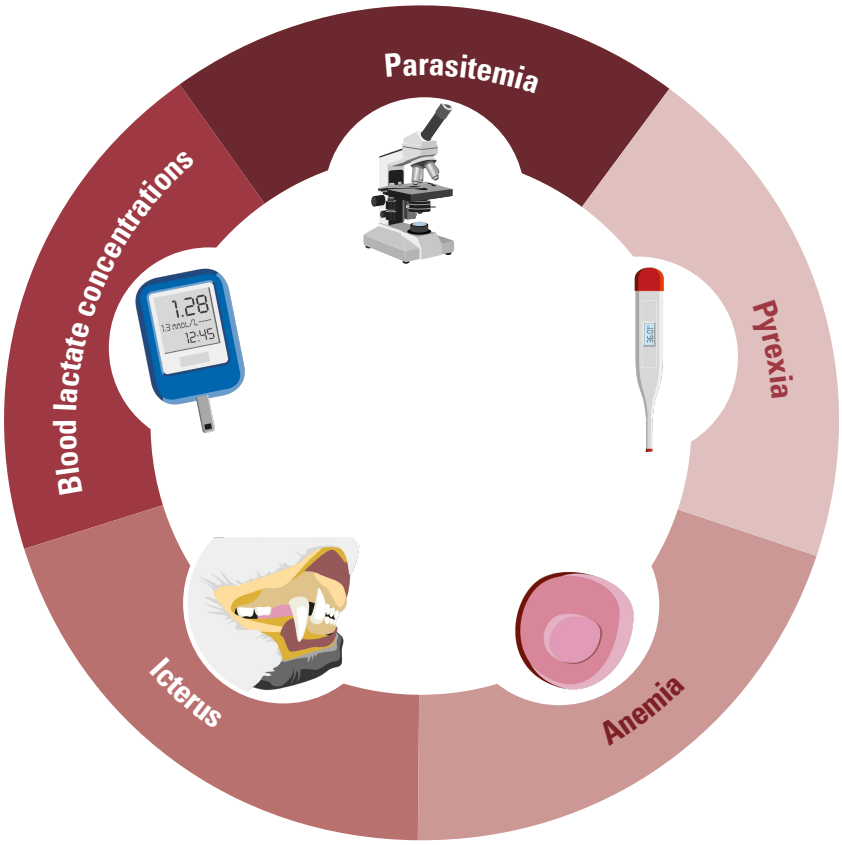
Dogs that are sufficiently anemic to require blood transfusion (hematocrits of <0.15 L/L) with no other complications may spend just 12–24 hours in an outpatient or day ward facility and are typically discharged within several hours of completing the transfusion.

Dogs that are warm in-saline agglutination (ISA) positive will remain so for days despite immunosuppressive treatment.

Despite this, once treatment halts the immune mediated hemolysis, the hematocrit will begin to rise and this is the important indicator of recovery, not the results of the ISA test.

Recovery is marked by a returning appetite and a spontaneously (i.e. not as the result of a blood transfusion) rising hematocrit.

Dogs that present with even profound anemia that is quickly corrected by transfusion will often recover sufficiently for discharge within hours.



Icterus

Dogs that become very icteric recover more slowly and require additional fluid, electrolyte and antiemetic supportive treatment.

Blood lactate concentrations

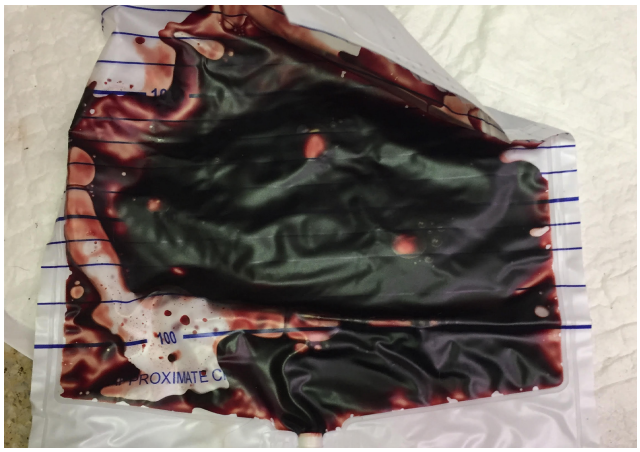
Blood lactate concentrations >5 mmol/L on admission, >2.5 mmol/L at 8, 16, and 24 hours after admission, that show an increase or <50% decrease by 8 and 16 hours after admission are associated with mortality. Concentrations persistently >4.5 mmol/L also indicated a very poor prognosis.

WHAT DIAGNOSTIC TESTS SHOULD BE RUN IN A DOG THAT IS SUSPECTED TO HAVE THE INFECTION/DISEASE?

Rapid, table-side

First, record a history and complete a **physical examination:**

- Rectal temperature (usually elevated),
- Femoral pulse (usually increased and described as 'bounding' if the dog is anemic) and respiratory rate
- Auscultate the chest for evidence of lung edema.
- Abdominal palpation. The abdomen may be tense on palpation due to splenomegaly (present in almost 100% of cases). The finding of a palpable urinary bladder is helpful as it reduces the concern of acute renal failure and means urine collection is possible.
- If urine can be expressed, a macroscopic appreciation of bilirubinuria or hemoglobinuria is possible.



Severe hemoglobinuria (seen in a urine collection bag) is not uncommon and occurs as the renal threshold for cell free hemoglobin in circulation is exceeded.

In hospital using microscope or similar equipment

It is routine to prepare a peripheral (capillary) **blood smear** from the ear margin in all dogs suspected of having babesiosis.

Packed cell volume, warm ISA test and blood glucose are routinely run as part of a minimum data base in all dogs that are blood smear positive.

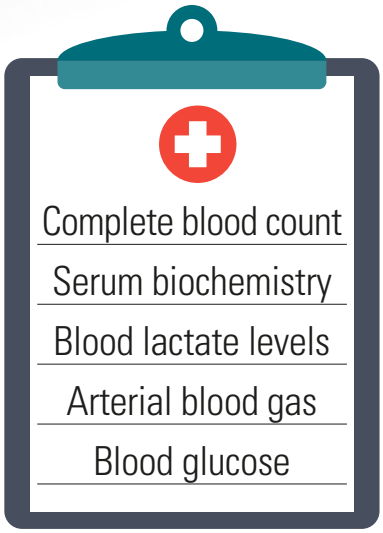
If urine is available (free-flow or cystocentesis) it is helpful to determine if there is hemoglobinuria present.

Babesia rossi parasites are easily detected in clinically ill dogs on thin capillary blood smears (400x, oil immersion, Diff Quick stain).

WHAT DIAGNOSTIC TESTS SHOULD BE RUN IN A DOG THAT IS SUSPECTED TO HAVE THE INFECTION/DISEASE?

Laboratory

Total serum proteins and albumin are also usually unremarkable (unless co-infection with *Ehrlichia canis* is suspected in which case the globulin concentrations are frequently raised). Thrombocytopenia and leukopenia are unhelpful as a means of diagnosing *Ehrlichia* coinfection while PCR remains the best means of ruling this organism in or out. Co-infection with *B. rossi* and *B. vogeli* occurs in <1% of cases and can only be determined by PCR but the co-infection is clinically insignificant as *B. vogeli* is not regarded as pathogenic. Serology is not used as a means of diagnosis for *B. rossi* infection.



All cases that require admission for care will require an extended data base to include a complete blood count (CBC) (usually including a reticulocyte count) and serum biochemistry (urea, creatinine, Na^+, K^+, Cl^-, total bilirubin and glucose).

Blood lactate levels are helpful in monitoring response to treatment.

Arterial blood gas analysis can be helpful to diagnose early acute respiratory distress syndrome.

Liver enzymes are not helpful in establishing a prognosis.

Test interpretation

Complete blood count

The degree of anemia is not a strong predictor of outcome but does **determine the need for a blood transfusion or not.**

Despite the anemia being obviously hemolytic, the reticulocyte count is typically consistent with an inappropriately regenerative response. Hemoconcentration (hematocrit >0.55 L/L), although rare, is a very poor prognostic indicator.

The degree of **left shift neutrophilia** correlates with outcome and indicates the very inflammatory nature of the disease.

Serum biochemistry

High serum urea is common, frequently associated with **normal creatinine*** (and hence unrelated to renal function) and is correlated with outcome.

Hypokalemia may develop in dogs that have prolonged recovery (especially if icteric) and should be corrected.

* Elevated creatinine is significantly less common than elevated urea and reflects renal function with elevations associated with poor outcome.

WHAT DIAGNOSTIC TESTS SHOULD BE RUN IN A DOG THAT IS SUSPECTED TO HAVE THE INFECTION/DISEASE?

Blood lactate levels

Blood lactate levels that remain **high** or fail to fall with treatment are associated with a **poor outcome**.

Blood glucose and bilirubin

Hyperbilirubinemia and hypoglycemia are correlated with a poorer outcome.

Arterial blood gas

Arterial blood gas evaluation provides information on **lung function** and assists in the early diagnosis of ARDS (typified by a ventilation/perfusion mismatch) although lung edema is usually acute and often only seen in cases that are agonal.

Acute *vs* convalescent

Dogs admitted for hospital care should have at least a daily hematocrit and serum electrolytes determined.

Convalescence is usually quick (within 24–48 hours) and there is generally little need for intensive monitoring in this period.

Death usually occurs within 24 hours of admission and in dogs that have indicators of a poor outcome in this period, even with intensive monitoring and treatment, there is usually little that can be done to alter this outcome.

Measures that have not been assessed but that may alter outcome and that should be investigated include:

- Early use of a mechanical ventilator in Acute Respiratory Distress Syndrome (ARDS)
- Use of dialysis in cases of acute oliguric renal failure.

Although almost all cases with cerebral disease die, survivors have been described but they were all left with devastating neurological deficits.

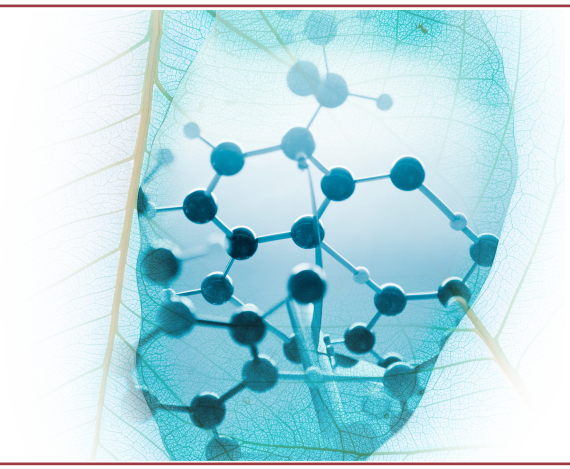
These interventions are expensive and dogs with this disease are frequently owned by people with limited financial means. Access to such advanced treatment modalities is also a limiting factor.

WHAT GENERAL TREATMENT STRATEGY IS RECOMMENDED FOR SICK DOGS?

Classes of drugs to use

Diminazene aceturate (an aromatic diamidine) and imidocarb (a urea derivative) are the **drugs** most commonly used to treat *B. rossi.*

Diminazene and imidocarb both have a narrow therapeutic index and their dose must be strictly calculated for dogs whose body weights are carefully established.



Drugs

Trypan blue was used widely until around the mid 1990's when it became apparent that there were no advantages to this use, and it is no longer available in South Africa.

Diminazene was discovered in 1944 and its use in dogs for *Babesia* first described in 1955.

Imidocarb was identified in 1969 and first described in the treatment of canine babesiosis in 1981.

Phenamidine (an amandine, like diminazene) is also used but less commonly than diminazene.

Mono or combination therapy

Some practitioners will follow a dose of diminazene up with a dose of imidocarb several days later and some will repeat the dose of imidocarb if they use imidocarb alone. There is no evidence that combining diminazene with imidocarb or repeating imidocarb is necessary.

The difficulty in diagnosing concurrent *E. canis* infection means that many practitioners will treat with daily doxycycline if this disease is suspected.

WHAT GENERAL TREATMENT STRATEGY IS RECOMMENDED FOR SICK DOGS?

Dogs that are In Saline Agglutination (ISA) positive should be immunosuppressed with a short acting **oral glucocorticoid.**

Prednisolone or prednisone are typically used. The starting dose is generally 1–2 mg/kg twice daily for the first 1–3 days and the dose is reduced rapidly after that. Treatment usually does not go beyond a week. The ISA positive reaction takes days to resolve (the agglutinating antibody takes several days to clear) but the hematocrit should rise spontaneously (and not because of a blood transfusion) in dogs that respond to treatment.

Dogs with complicated disease that require in-hospital treatment typically require a **blood transfusion** (sometimes several) to raise the hematocrit above 0.2 L/L.

Packed red cells are the blood product of choice (although whole blood is often used).

Additional treatments are typically aimed at supporting whichever organ or organs are dysfunctional or failing:

- Intravenous **fluid therapy** is almost always required following blood transfusion.
- Nasal oxygen may be helpful in cases that have low arterial oxygen tension.

IV fluids ensure adequate hydration and urine production which is important in cases clearing a significant intravascular cell free hemoglobin load. Electrolyte supplementation (especially potassium) is often required as these dogs have generally been anorexic for a while and will not start eating for at least a day or two. IV glucose supplementation (or bolus treatment in cases that are hypoglycemic) may be needed.

- A prokinetic drug is very often useful and maropitant or metoclopramide is frequently used.
- Although choleretics and various vitamin and mineral supplements are sometimes used, there is no evidence that these offer any benefit.
- Seizure management by means of general anesthesia will be necessary in dogs with cerebral babesiosis (non-neuroglycopenic central nervous system signs).
- Mechanical ventilation will be required in cases with ARDS.
- In dogs that have fragile lung function (usually determined by arterial blood gas),**conservative fluid treatment** is important.

Follow the 'run them dry' philosophy of managing Acute Respiratory Distress Syndrome.

- Dogs in acute renal failure may benefit from dialysis.
- Although severe tachyarrhythmias are frequently observed on ECG monitoring, treating these appears to make no difference to outcome.

WHAT GENERAL TREATMENT STRATEGY IS RECOMMENDED FOR SICK DOGS?

Monitoring for response to treatment

Uncomplicated cases

- Uncomplicated cases that receive a single diminazene or imidocarb injection seldom need further follow up monitoring or treatment.
- Dogs with a moderate anemia or ISA positive test will normally be followed up at least once **24 hours after initial diagnosis and treatment.**

At this point a brief history and clinical examination should be performed and the hematocrit determined. It is important that by this point the owners have noted an improvement in general habitus, an interest in food and that the hematocrit should not have dropped lower than what it was at diagnosis.

Complicated cases

- Dogs with complicated disease should have frequent (twice daily) hematocrit determination.
- Renal function should be followed biochemically. Urine production should be assessed closely by means of a closed urinary catheter and collection device.
- Serum electrolyte and glucose should be measured as needed to follow response to supplementation.

ARE OTHER PETS OR PEOPLE IN THE HOUSE AT RISK?

Advice to give owners of a sick dog

The disease is not a zoonosis and is not contagious. Tick exposure is the risk factor and effective ectoparasite control is crucial for disease control. This is the only means of prophylaxis. There is no point to treating other healthy dogs in the home for babesiosis.

Can cats get this infection/disease?

Cats are not at risk for *B. rossi* but can get a *Babesia* infection (seen quite commonly in a small geographic area in South Africa) caused by *Babesia felis*. This is a very different disease to the canine disease.

Close monitoring for signs of illness and early presentation for veterinary care is the best advice.

WHAT ARE SOME RECOMMENDATIONS AROUND PREVENTION STRATEGIES?

How to avoid the vector

Avoid tick exposure by staying away from wilderness or semi-wilderness areas. Fastidious compliance with ectoparasite control recommendations is crucial.

Is there a vaccine?

A vaccine available previously was withdrawn from the market because of the cost and need for frequent boosting.

Is routine testing recommended?

Routine testing for *B. rossi* is not recommended.

WHAT DOES THE FUTURE LOOK LIKE?

What are the changes being seen with the disease?

Because of the use of oral isoxazoline compounds the incidence of disease among dogs has dropped significantly among affluent populations. Disease incidence has not changed in rural settings and among people unable to afford tick prevention products.

How is the risk of disease changing?

Public awareness of canine babesiosis has risen in association with educational campaigns tied to ectoparasiticide protection recommendations, particularly among media-exposed people, and there is good awareness of the disease and the danger of ticks.

Dogs owned by economically disadvantaged people continue to be at risk.

There is a growing problem with tick resistance to dips used on cattle, and this cheaper means of ectoparasite control (sometimes applied in rural settings at primary veterinary care facilities) could become increasingly ineffective.

Has resistance to prevention or reduced treatment effect been seen?

Acaricide resistance is a global concern and Africa is no exception.

This has not been reported in *H. elliptica,* but it is possible and may be a problem in areas, particularly poorer communities, where dogs are frequently dipped in topical ectoparasiticides.

B. rossi has not shown resistance to either diminazene aceturate or imidocarb.

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