Advances in Life Science and Technology
ISSN 2224-7181 (Paper) ISSN 2225-062X (Online)
Vol.65, 2018
www.iiste.org

Review on Dog Neonatal Mortality

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Abstract
The canine neonatal period refers to the first 2–3 weeks of life.After this time, the hepatic and renal drug elimination mechanisms can be expected to approach adult values. Or it is defined as the transitional phase from foetal to adult life. During this interval changes in structure and function occur as the animal moves from the physically, chemically and microbiologically protected environment of the uterus to face the adaptive requirements for survival in a complex environment. Neonatal mortality in dogs has caused a lot of loss dogs and has always attention is given on the survival of young dogs.Neonatal mortality occurs in dog is related to different factors, such as management conditions, malnutrition, congenital abnormalities, parasitism, and infectious diseases. In general the causes of the neonatal mortality in dog is classified as neonatal, maternal, environmental and management risk factors.
Keywords: Mortality, Dogs, Puppy

1. Introduction

Pup mortality, both during parturition and in the neonatal period, is asignificant clinical problem that is poorly documented in the veterinary literature. Neonatal mortality in the canine species (within the first3 weeks after birth) is highly prevalent ranging between17 and 26% (Tønnessen *et al*., 2012; Indrebø *et al*.,2007). The majority of pup losses are stillbirths and deaths within the first week of life, that is, perinatal mortality (Gill, 2001). Lawler; 1989) specified 65% of the losses occurred in the first week with about half of these being stillbirth.

In veterinary medicine, the neonatal mortality rate ranges from 17%-30% in dogs and is the highest during the first 7 days of life. Neonatal mortality is associated with several factors, including stillbirths, maternal neglect, and agalactia as well as congenital and acquired conditions (Inderbo A. Trargerd C.More L, 2007); however, many losses are a result of inadequate reproductive management and could be avoided.

Mortality in the neonatal period can be related to many factors including dystocia, maternal neglect or carelessness, lack of nutrition, congenital abnormalities, environmental conditions or infectious agents (Münnich, 2008; Indrebø *et al*., 2007). Others have reported that infectious diseases, mainly bacterial infections, are the second most important cause of neonatal mortality (Münnich, 2008).Bacterial infections are one of the major causes of neonatal deaths in many dog-breeding kennel (Greene, C. E., and T. F. Prescott, 1998). Factors that predispose a puppy to bacterial infections include endometritis in the bitch, a pro- longed delivery or dystocia, vaginal discharges, and environ- mental exposure. Such infections may lead to diarrhea, pneumonia, peritonitis, septic arthritis, septicemia, and other clinical conditions which are usually debilitating and are often fatal. Staphylococci and gram-negative bacteria, especially Staphylococcus aurous and Escherichia coli, are the most common bacteria associated with systemic infections in newborn puppies (Sager, M., Remmers, 1990). Additionally, it has been shown that the “Fading puppy syndrome” contributes to the Canine Herpesvirus-1 Infection in Neonatal Dogs neonatal losses (Gill 2001; Indrebø *et al.*,2007; Tønnesen *et al*., 2012).Canine herpesvirus type 1 most frequently isolated from neonates dying within the first week after birth (Münnich, 2008; Dahlbom *et al*., 2009).

Losses among puppies and kittens may occur in utero, during expulsion, immediately after birth, in the first weeks of life and after weaning. The rate of perinatal death (stillborn puppies and neonates) is highly variable but highest during parturition, immediately after birth and in the first days of life. All together, morbidity and mortality range from 5 to 35 %, depending on factors like quality of labor, occurrence of dystocia, time and kind of intervention during birth, in-breeding, genetic defects and malformations, maternal disturbances, vaccination status of the mother, low birth weight, environmental conditions (temperature), or infectious agents. (RDS / Asphyxia, Hypothermia, Hypoglycaemia, dehydration, gastrointestinal diseases, Neonatal Isoerythrolysis in kittens). Most non-infectious causes predispose the puppies and kittens for infections.

Based on the above background, this review paper is prepared with the objectives to:

- Summarize the principal causes of puppy death in dogs briefly and
- To provide owners and veterinarians with information and advices regarding management and care of neonatal dogs

2. Neonatal Mortality in Dog

2.1. Classifications of Neonatal Mortality

The Abnormal Pup: A pup was classified as abnormal if it was mummified, had died prior to birth, was

premature, small for date or had gross congenital defects. Foetal mummification was identified by the birth of dry foetal mass surrounded by shrivelled, dried foetal membranes. Pups were classified as having died prior to birth if they were fully developed but had evidence of autolysis indicating that they had died within days before the commencement of labour (Arthur, 1973).

Fetal Asphyxia: Asphyxia refers to a condition of hypoxemia, hypercarbia and acidosis (James and Adamsons, 1964). Tissue acidosis and dysfunction concurrently occur, and will, if not rapidly corrected, lead to irreversible organ damage and death (Jacobs and Phibbs, 1989). The direct cause of death from asphyxia is an insufficiency of oxygen supplied to the tissues by the blood (Potter & Craig, 1976).

The Live Normal Pup: The live normal pup was one considered both physically and clinically normal following birth and which subsequently died (Arthur, 1973).

2.2. Risk Factors of Neonatal Mortality in Dog

2.2.1. Fetal Risk Factors

Neonatal Immaturity: The newborn puppy is an immature animal, dependent on its dam for survival in the first three weeks and as a consequence the etiology of neonatal death is frequently complex and often undetermined (Chandler, 1990; Blunden, 1998).Newborn puppies are born with an immature immune system that needs to be built up over time, starting with their mother's milk. The immature status of the immune system makes them highly vulnerable to infections (Day, 2007) and environmental, nutritional, and metabolic factors. Young puppies have poorly developed immune systems. They acquire antibodies from the milk the mother dog produces directly after birth, called the colostrums. Puppies that fail to nurse adequately will not receive these antibodies and are much more likely to catch a serious infection.. Because of the immature status of the newborn puppy, a sick neonate may rapidly become hypothermic, hypoglycemic, dehydrated and hypoxic resulting in die – regardless of the initiating insult (Gunn Moore, 2006). The newborn puppy is particularly vulnerable because of four major factors. These are, their thermoregulatory mechanism is poorly developed, and there is a risk of dehydration, a risk ofhypoglycemia and immunological immaturity (Blunden, 1998).

Neonatal Thermoregulation: Newborn puppies have a poorly developed thermoregulation which makes them susceptible to hypothermia (Simpson et al., 2004).The newborn puppy is a true homeotherm that substitutes compensatory thermal conduction for its lack of compensatory thermogenesis. The shivering reflex and vasoconstriction mechanism are not operant in the newborn. Poor maternal instinct may result in neglect of puppies at the time of birth so that the initial hypothermia is not corrected. Weak or premature pups may be unable to establish the necessary physical contact with the dam to achieve normothermia.If the periods of contact are intermittent, moderate hypothermia may persist.Young animals do not yet have strong body temperature regulation, and their body temperature can fluctuate profoundly in response to changing environmental temperatures and humidity (Simpson et al., 2004).

Neonatal Glycogen Reserves: The puppies have relatively small reserves of glycogen in the liver. In failure to suck or in lack of nutrition, the puppies may rapidly develop hypoglycemia (Simpson et al., 2004). The newborn relies almost exclusively on hepatic glycogen for energy for the first 24 hours. Hepatic glycogen stores may be low at birth owing to intrauterine malnutrition associated with excessive multiple pregnancy or maternal malnutrition. Within 8-12 hours after birth most hepatic glycogen has undergone glycogenolysis and the newborn is forced to rely on nutritional intake to maintain euglycaemia. It is during this interval that the newborn isexquisitely susceptible to the development of hypoglycaemia (Center et al., 1990). Failure to suck results in rapid depletion of the liver reserve of glycogen and the development of hypo glycaemia by the second day.Neonates have minimal body fat reserves and limited metabolic capacity to generate glucose from precursors. Glycogen stores are depleted shortly after birth, making adequate nourishment from nursing vital. Even minimal fasting can result in hypoglycemia. Hypoglycemia can also result from endotoxemia, septicemia, portosystemic shunts and glycogen storage abnormalities (Simpson et al., 2004).

Neonatal Ontogeny: Within the first week of life newborn animals are more susceptible to infection than older animals because they lack a well-developed microflora and because they have a non-selective protein transport mechanism that facilitates the absorption of immunoglobulins. This protein transport mechanism may also transport pathogenic microorganisms across the mucosal barrier. Maternal immunity is essential in providing protection during this critical period (Greene, 1984a).

Neonatal Renal Function: Due to the immature function of the kidneys they are at increased risk of dehydration (Simpson et al., 2004). The kidney of the newborn contains an outer zone of non-differentiated tissue in the cortex which requires 2-3 weeks to undergo nephrogenesis and become functional (Mosier; 1978). Functional tests of normal puppies have established that the glomerular filtration rate at birth ranges from 21-50% that of the adult and the tubular secretion rate at eight weeks of age ranges from 12-15% that of the adult. This leads to a slow clearance of fluids, increased sodium loss and inability to conserve fluids. There is a positive correlation of glomerular filtration rate and mean arterial blood pressure (Robinson, 1983). Neonates have a greater extracellular water and total water compartment than adults (Jones, 1987). This difference is due mostly

to extracellular fluid. As 82% of bodyweight is water and kidney function is immature, the neonate is particularly susceptible to dehydration (Blunden, 1998). Water turnover rate is twice that of the adult. Neonatal puppy fluid maintenance requirements are approximately 132 - 220 mls/kg/day (Mosier; 1978).

2.2.2. Neonatal Diseases

Fading Puppy Syndrome: Fading puppy syndrome is a term used to describe the situation where puppies are apparently normal at birth but gradually "fade" and die within the first two weeks of life. A puppy that is apparently normal at birth but fails to survive beyond 2 weeks of age is often referred to as a fader. Sometimes puppies appear to be healthy at birth but die suddenly in the first few weeks of life. Veterinarians define this as fading puppy syndrome or sudden death in puppies. Breeders use the term "Fading Puppy Syndrome" for puppies that are apparently born healthy and then fails to thrive and suddenly die within the first weeks of life (Ranjan, 2010).

Causes of fading puppy syndrome**:** The etiology is diverse and includes a whole range of causes as, hypothermia, mismothering, inadequate nutrition and uptake of colostrum, trauma, congenital abnormalities, low birth weight, bacterial and viral infections (Indrebø *et al.*, 2007; Ranjan, 2010). CHV-1 appears to be one of the important viral agents in "Fading Puppy Syndrome" (Ranjan, 2010).

*Inadequate maternal care and Lack of milk production in the mother***:** Lack of mothering instinct coupled with poor hygiene can often result in neonatal septicemia (systemic infection) in a very short time. Although some maternal immunity is conferred to the puppy in utero or while it is developing in the mother's womb, the majority of this immunity is acquired via the colostrum or first milk. If the puppy does not drink an adequate amount of this first milk, it is more vulnerable to infection. Overweight or older dams are more likely to experience neonatal loss. Maternal neglect—maternal neglect can be exhibited by a dam's reluctance to lie with and warm the neonates, refusal to permit nursing, or lack of sufficient milk production. Large-breed or barrel-bodied dogs (Ranjan, 2010).

*Low weight at birth (runt of the litter***):** Birth weight is considered as an important survival determinant in most mammalian species (Gatel*et al*., 2011). Low birth weight is accompanied by immature development and adaptive postnatal failure. Low birth weight puppies are physiologically immature when compared to litter mates of average birth weights. They are also at greater risk from hypothermia and cannot compete well for milk against their larger litter mates.The birth weight was related to maternal size, weight and age as well as breeds and litter size with heavier puppies in small rather than larger litter from medium sized breeds (Gropetti *et al*., 2015). The birth weight of new born is also reported to be influenced by variety of factors such as genetics, environment, nutrition and fetal uterine position (Bautista *et al*; 2015). The definite role of genetics influencing the birth weight was reported in case of boxer puppies (Nielen *etal*., 2001).

Clinical sign: Failure to suckle, Crying and crawling from being uncomfortable, Diarrhea, Cyanosis or "Blue Baby", Occasional sloughing of tail and toes, Lie away from the group, Act restless, Vomiting, Salivation, Difficulty breathing

Diagnosis: The veterinarian will examine the puppy physically for signs of infection, birth defects or other problems. Blood, urine, and feces samples may also be taken. Often death occurs too suddenly for diagnosis or treatment. In this case, it's a good idea to take the puppy for posthumous examination anyway, so the cause can be determined since some conditions could pose a threat to other members of the litter. Also the veterinarian will likely need to examine the dam to check for signs of breast or uterine infection, as well as glandular problems that can cause low blood calcium during lactation and lead to poor milk production. Bloodwork and urine tests will likely be needed (Rota *et al*., 2007).

Treatment: Because the exact causes of fading puppy and kitten syndrome is seldom known, your veterinarian will initially focus on supportive care and diagnostics. Initial therapy will include providing supplemental warmth, nutrition and especially glucose, broad spectrum antibiotic, and fluids will all be needed to stop the fading until the cause is identified. Blue babies may get a blood transfusion and oxygen when needed and antibiotics will be started. It is important to ensure that the puppy receives adequate fluid and is kept warm. Puppies should not be allowed to become chilled and an environmental temperature of slightly above 103OF (39OC) should be maintained. If bacterial septicemia develops, antibiotics may benefit the puppy, but strict hygiene and good management procedures are also critical. It is also important that the mother is examined for teat (breast) discharge and possible mastitis (breast infection), metritis (uterine infection) or other illness.Treating "Fading Puppy Syndrome" with Plasma: One important use of blood plasma is to provide a source of globulins (plasma protein antibodies) to protect weak, fading or orphan newborns against the common infectious agents to which they are exposed. Plasma treatment [canine fresh-frozen plasma (FFP)] for orphaned puppies or for those receiving only minimal colostrum after birth should be given three times in the first 24 - 48 hrs. of life (1st at birth, 2nd in 12 hours and 3rd time in 12 hours) (Greene, 2012; Rota *et al*., 2007).

Treatment for healthy newborns may be repeated at 5 to 14 days of age and then again at 3 to 4 weeks of age. For sick newborns, more frequent transfusions of FFP may be necessary. These transfusions are usually given intraperitoneally (IP), but they can also be given orally (by mouth) in the first 24-36 hours of life [as FFP

is salty, it should be followed with a little drop of honey or syrup on the tongue. When puppies are two days of age or older, the route of administration must be IP (or IV or subcutaneously) and not oral as the antibodies in plasma will no longer be absorbed through the gastrointestinal tract. For kittens, the same protocol is followed using feline FFP. The recommended dose is 3-5 mL per pound of body weight 0.25 x Weight of Puppy in Ounces = The Amount of Plasma given in mL or cc. This is given to each puppy orally, IP or subcutaneously. Do not give more than 10 mL at one time (Dodds, WJ, 1993).

Neonatal Infections includes late gestational abortion and neonatal death can be associated with numerous viral, bacterial and parasitic infections. Infectious diseases accounts for only a relative small percentage of deaths (Greene, 2012; Rota et al., 2007) and the majority of these deaths occurred in the late neonatal period. This time frame for neonatal infections may be related to the fact that in the absence of the ingestion of colostrum the puppy is probably protected for at least one week by the in utero transfer of immunoglobulins. Possible routes of infection are oral, via the umbilicus, by the ingestion of vaginal discharge during the process of parturition or from the infected environment, by inhalation and across the placenta (Evans, 1978).

Bacterial Infection: Brucellosis, the bacterium Brucella cane's (small, aerobic Gram-negative coccobacilli,) stands out as one of the main bacterial causes of pregnancy loss and neonatal mortality in bitches . The main source of infection is through vaginal and seminal secretions from infected animals, although bacteria are shed in faeces, milk, saliva, and nasal and ocular secretions (Givens MD, Marely MSD, 2008). The B. canis may be present for a long time in dogs without exhibiting clinical signs. After the initial exposure, the bacteria reach the bloodstream in about three weeks. Subsequently, the pathogen can infect the genital tissues enabling a continuous release of the agent, which may be recurring for months or even years

Pathogenesis**:** After the initial exposure, the bacteria reach the bloodstream in about three weeks. Subsequently, the pathogen can infect the genital tissues enabling a continuous release of the agent, which may be recurring for months or even years. In turn, canine brucellosis can result in infertility, difficulties in pregnancy, early embryonic death, foetal resorption and late abortion.

Clinical signs**:** The clinical signs associated with Brucellosis in dogs are not pathognomonic and due the lack of the lipopolysaccharide antigen associated with endotoxemia in bitches, it is rarely systemic ill and fever (Graham EM, Taylor DJ, 2012). Clinical signs reflect the localization of the bacteria in extra reproductive tract sites such as the eye, intervertebral disc spaces, and reticuloendothelial system. Brucellosis causes spontaneous late abortion in a healthy bitch, most commonly occurs from days 30 to 57, accompanied by a vaginal discharge lasting up to 6 weeks. Earlier abortions can occur but may be incorrectly reported as conception failure since the bitch typically ingests aborted fetuses. Early embryonic death and foetal resorption can occur within 10 – 20 days post-mating. Many bitches that abort will subsequently have normal litters, although puppies born to infected bitches contain both live and dead pups, although most live pups die shortly thereafter. Aborted puppies usually appear partially autolysed, with lesions of generalized bacterial infection, including subcutaneous oedema and degenerative lesions in the liver, spleen, kidneys and intestines(Graham EM, Taylor DJ, 2012).

Parasites: Intestinal parasites because roundworms and hookworms are transmitted through the placenta, most pups are born with these parasites. Kittens and pups can acquire roundworms through the dam's milk. Hookworms are transmitted to kittens and puppies through the placenta and mammary glands. In addition, some protozoan parasites cause diarrhea in the young. While rarely fatal, they can contribute to illness and put a neonate at higher risk of additional infection (Zimmer JF, Pollok RVH, 1987).

External parasites like fleas and ticks can also weaken a puppy by taking vital blood and nutrients away from the developing puppy. Infested puppies often fail to gain weight and slowly "fade" and die (John S. Parker, 2007). Ectoparasites in young puppies and kittens are often a sign of poor management and hygienics. Coccidia and neospora are also known pathogens in dogs.

Protozoon diseases such as Toxoplasma infection in dogs at various gestational stages can cause mortality of the puppies from the 4th to 75th postnatal days. Stillbirth, abortion and foetal death has been observed, in the middle third and final pregnancy in dogs (Dubey JP, Lappin MR, 2015).

Viral Infection: Canine Herpesvirus (CHV): Exposure of an immunologically innocent bitch to canine herpesvirus during the last 3 weeks of gestation can result in late term abortion or neonatal death within the first 3 weeks of life. CHV-1 is an enveloped double stranded DNA virus belonging to the family Herpesviridae and the subfamily Alphaherpesviride, genus Varicellovirus (Dubovi & Maclachlan, 2010). The canine herpesvirus 1 (CHV-1) has a worldwide distribution and is associated with respiratory and reproductive diseases in dogs (Evermann JF *et al*., 2011). CHV-1 is phylogenetic similar to those of α- herpes viruses affecting other species but specific receptors on the cell surface causes the virus to have a restricted host range to domestic dogs or others of the Canidae family (Greene, 2012; Nakamichi *et al*., 2000).

The virus is inactivated at pH < 5 and >8 and by the exposure of most disinfectant, to lipid solvents and to heat above 40°C (Greene, 2012). Because the virus is sensitive and quickly destroyed when exposed to environmental factors, transmission occurs by direct contact with mucosal secretions.Like other herpesviruses, it becomes latent after a primary infection and is shed periodically, primarily in nasal or rarely in genital

secretions. Moreover, the disease manifests itself in seasonal way, accentuating in cold weather because the virus is unstable and sensitive to higher temperatures (Green, C.F, 2012; Buonavogila and Martella V, 2007). Among the infectious diseases of viral origin, the CHV-1 stands out as one of the main viral cause of abortion and neonatal mortality in dogs (Dahlbom, *et al.,* 2009). The infection caused by this virus during pregnancy can lead to abortion, stillbirth, embryonic resorption, premature birth and neonatal death (Ronsse V *et al.,* 2005).

The disease is usually asymptomatic in puppies exposed to CHV after 1-2 weeks of age. However, CHV infection is generally fatal in neonatal pups (1-4 weeks old) that lack maternal immunity. These pups may be infected during passage through their infected dam's birth canal or, more commonly, by contact with oronasal secretions of the dam or other dogs in the kennel or home. Infected littermates, or neighboring dogs that are shedding virus, also can be sources of infection. The incubation period is about 6 - 10 days, and duration of illness in newborn pups is 1-3 days(Creevy, Kate E, 2017).

Transmission: Vertical transmission occurs from mother to foetus through the placenta [Megid and Souza TD, 2016]. In some cases the infection can reach the uterus resulting in foetal death and still birth of the offspring (Carmicheal LE *et al.,* 1965). Newborn puppies may acquire the infection in utero or from passage through the birth canal but more commonly, the puppies are suspected to be infected from oronasal secretion from the dam or by infected littermates and surrounding dogs (Rootwelt *et al;* 2009; Greene, 2012) pointed out that puppies, older than two weeks, at the time of infection, are relatively unaffected by the virus and the infection is generally associated with localized infections in the upper airways. After infection, the virus enters the bloodstream and replicates in vascular endothelial cells lining small blood vessels (Poulet *et al.,* 2001).infected adult dogs often do not show apparent symptoms. In them, the infection is often subclinical. However, in newborns and puppies with 1–2 weeks of life may develop systemic disease that may result in a generalized necrotizing haemorrhagic disease (Megid and Souza TD, 2016; Carmicheal LE *et al*., 1965).

The incubation period varies from four to ten days and most of the affected puppies are less than three weeks old with signs of anorexia, dyspnea, and painupon abdominal palpation, incoordination and a typical soft, yellow-green feces. There may be serous or hemorrhagic nasal discharge. Petechia (small pinpoint hemorrhages) are common on the mucous membranes, and thrombocytopenia (low platelet count) may occur. Rectal temperatures are usually not elevated (Creevy kate E, 2016).The mortality of the litter can be high and may reach a mortality rate up to 100% [(Decaro *et al.,* 2008). Carmichael,1970)] summarized in an article that after oronasal inoculation of puppies, the primary site for virus replication was the nasal epithelium and the tonsils. After three to four days, the virus enters the bloodstream resulting in a leukocyteassociated viremia probably through the uptake of macrophages. The virus spreads through the blood and replicates in vascular endothelium lining small blood vessels, leading to necrotizing vasculitis with secondary diffuse hemorrhage in several organs including the kidneys, adrenal glands, liver, spleen and lungs (Poulet *et al.,* 2001).

Clinical signs: Clinical signs of canine herpesvirus are: Lethargy, Decreased suckling, Diarrhea, Nasal discharge, Conjunctivitis, Corneal edema, Red rash, rarely oral or genital vesicles, Soft, yellow-green feces, Notable absence of fever(Poulet *et al.,* 2001).

Diagnosis: Information obtained from the clinical history of the puppies compared with high mortality of the litter during the neonatal period, usually gives the veterinarian reason to suspect a CHV-1 infection. In order to determine CHV-1 infections, diagnosis must be based on more specific pathological and histopathological findings and detection of viral DNA in the organs of stillborn or dead neonatal puppies. Obtaining the history and medical records of these animals is also essential for a complete and accurate diagnosis (Lamm CG and Njaa BL, 2016).

Treatment: The treatment of this disease in puppies is difficult due to the rapid development of infection and mortality that occurs before the diagnosis is established (Green CE, 2015). Successfully use of acyclovir in therapy and management of neonatal puppies infected with CHV-1 is described in a case report from California (Davidson et al., 2003). However, signs of toxicity have been reported in dogs from accidental ingestion of acyclovir with doses of 40 mg/kg (Richardson; 2000). Intraperitoneal injection of 1 – 2 ml of immune sera obtained from seropositive dogs or an elevation of the puppies body temperature to reduce viral replication, seems to lower the mortality of those puppies where a generalized infection has not yet been manifested (Greene, 2012). In general, therapy for neonatal dogs with signs of generalized CHV-1 infection is limited and with poor prognosis.

Prevention: Vaccination and appropriate sanitary measures are essential to prevent viral spread among animals in kennels [Montero *et al*., 2016]. An inactivated vaccine is licensed in Europe (Eurican® Herpes 205, Merial, France) which contains a specific surface protein (gB – glycoprotein) from CHV-1 (EMEA, 2002). The vaccine is administrated subcutaneously to pregnant bitches in a two dose regimen, to ensure a satisfactory neutralizing antibody level at the time of whelping. First vaccination should be at estrus or seven to ten days after mating, and second vaccination one to two weeks before whelping (EMEA, 2002). Vaccination provides passive maternal immunity to the puppies, when absorbing IgG from the colostrum and milk within the first 12-36 hours of life (EMEA, 2002).

In order to protect neonatal dogs from infection a suggestion of a prophylactic regimen in larger kennels, could be to improve hygiene and isolation of the dam and puppies (to lower the risk of CHV-1 infections from surrounding dogs). Additionally, good environmental conditions to ensure an elevation of the body temperature may provide some protection to uninfected puppies as the virus replication is reduced at higher temperatures (Carmichael, 1970). Furthermore, colostrum intake within the first few hours of life is essential to the puppies in receiving maternal antibodies as protection against infections.

2.2.3. Maternal Risk Factors

Maternal Infections: Maternal infections are well known causes of perinatal morbidity and mortality.Maternal infections with Canine Parvovirus, Herpes Virus, Distemper, Brucella Canis and toxoplasmosis are all reported to cause foetal and neonatal mortality (Green CE, 2015;Bresciani KDS et al., 2003)

Maternal Behaviour: The maternal behaviour post whelping can significantly affect pup mortality.maternalfactors and mismothering, which included trauma, excessive licking, lactational failure and cannibalism, caused the death pups born and are the principal causes of pup mortality.The lack of effective maternal care allows environmental influences, particularly the environmental temperature, to affect the puppies (Crighton, 1968).

Dystocia: Dystocia is defined as difficult birth or theinability to expel the foetus or fetuses through thebirth canal without assistance (Jackson, P.G.G, 2004) and constitutes aserious reproductive problem in the bitch and also the associated hypoxia or anoxia are very significant causes of earlydeath in the dog. Although the incidence of dystocia in dog is low, it is life –threating to both the mother and foetuses when it occurs (Lawler, 1989).

3. Conclusions and Recommendations

The overall incidence of neonatal mortality defined as the death of puppies occurring from time of delivery to the time of weaning. In comparison to other species, prevalence of canine neonatal mortality is considerably higher and is related to many factors including prolonged labor, maternal neglect or carelessness, lack of milk, congenital abnormalities and acquired disorders of neonates. Optimal husbandry impacts neonatal survival favorably and involves managing labor and delivery to reduce stillbirths, controlling parasitism and reducing infectious diseases, preventing injury and environmental exposure, and optimizing nutrition of the dam and neonates. Proper genetic screening for selection of breeders minimizes inherited congenital defects. Prevention is the best way to manage early death in puppies. Regular vaccinations in the mother dog can help to reduce the puppies' likelihood of exposure to viruses. The dam should be monitored for bacterial infections or canine herpesvirus during pregnancy. Maintaining her health throughout the gestation and lactation period is extremely important for the puppies' survival. Practicing good hygiene when handling the puppies can also help to minimize the spread of infection. Some early deaths may be unavoidable, however, especially if the puppy has a problem at birth.

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