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Canine parvovirus in Australia: A comparative study of reported rural and urban cases

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ABSTRACT

Canine parvovirus (CPV) is a highly contagious and often fatal disease reported worldwide. Outbreaks occur throughout Australia, and it has been suggested that disproportionally more CPV cases occur in rural locations. However, evidence to support this suggestion—and possible reasons for such a predisposition—has not existed until now. In this study a total of 4870 CPV cases reported from an Australian disease surveillance system between September 2009 and July 2014 were analysed. Australian postcodes were classified as rural or urban (based on human population density) and reported CPV cases were then categorised as rural or urban based on their reported home postcode. Parvovirus cases were predominately young (<12 months), entire, unvaccinated, mixed-breed dogs. More than twice as many of the reported cases were from a rural area (3321 cases) compared to an urban area (1549 cases). The overall case fatality rate was 47.2%; it was higher for those CPV cases reported from urban areas (50.6%) than rural areas (45.5%). A greater proportion of rural cases were younger, entire dogs compared to urban cases. The final multivariable model of CPV cases being reported from a rural area included age (<12 months) and vaccination status (never vaccinated) as significant predictors. Poor socioeconomic status might be a reason for the decision of rural owners not to vaccinate their dogs as readily as urban owners. The excess reporting of rural CPV cases compared to urban cases and the predictive risk factors identified in this study can be used by veterinarians to reduce the incidence of CPV by educating owners about the disease and promoting better vaccination programs in rural areas. This study also supports that the increased risk of CPV in rural areas may necessitate a need for increased vigilance around preventing CPV disease spread, additional care with puppies which are the most susceptible to this disease and tighter vaccination protocols, compared to urban areas.

1. Introduction

Since its emergence in the late 1970s, canine parvovirus (CPV) has remained a common and important cause of morbidity and mortality in young dogs (Goddard and Leisewitz, 2010). It is a highly contagious disease and can occur as outbreaks. Infection is acquired via the faecal-oral route and virus replication occurs only in rapidly dividing cells such as lymphoid tissues, intestinal crypt epithelial cells, precursor cells in the bone marrow and rarely myocardiocytes in puppies within the first two weeks of life (Goddard and Leisewitz, 2010; Prittie, 2004). Intestinal tract damage increases the risk of bacterial translocation and subsequent coliform septicaemia, which may lead to the development of a systemic inflammatory response that can progress to septic shock and ultimately death (Schoeman et al., 2013). The estimated case fatality rate reported for CPV in studies conducted within Europe, North America and Australia ranges from 24 to 43% (Glickman et al., 1985; Kalli et al., 2010; Ling et al., 2012).

The treatment for parvovirus disease in individual dogs is supportive and symptom-based (Lamm and Rezabek, 2008), and often involves use of anti-emetics to manage severe vomiting, fluid therapy to restore hydration and administration of antibiotics to prevent secondary bacterial infection. Survival rates are considered to be significantly higher with treatment (64%), with the reported survival rate as low as 9.1% in the absence of treatment (Goddard and Leisewitz, 2010). It is thought that dogs which recover from CPV infection will generally retain lifelong protective immunity against the infection strain (Lamm and Rezabek, 2008).

Initial clinical signs of canine parvovirus are non-specific, and include anorexia, depression, lethargy and fever (Goddard and Leisewitz, 2010). This often progresses to severe vomiting and small bowel diarrhoea. Since a clinical diagnosis is not definitive,

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several laboratory methods—including ELISA, immunochromatographic, hemagglutination, virus isolation or polymerase chain reaction-based methods—have been developed to detect CPV in the faeces of infected dogs (Desario et al., 2005).

Certain predisposing factors (breed, age, seasonality) have been reported for CPV. Although this disease can be seen in dogs of any age, puppies between 6 weeks and 6 months of age appear to be more susceptible (Goddard and Leisewitz, 2010). Certain breeds have been shown to be at greater risk of developing CPV disease—including the Doberman pinscher, Rottweiler and German shepherd dog (Castro et al., 2007)—however, the reasons for possible breed susceptibility are unknown. Furthermore, a distinct seasonality to CPV disease has been reported in Australia, with a higher incidence of disease occurring during the warmer months of the year. The most commonly reported risk factor for CPV disease is lack of protective immunity (Ling et al., 2012).

CPV is highly stable and ubiquitous in the environment, as it is extremely resistant to pH and temperature changes and to treatment with lipid solvents, trypsin and most disinfectants (Decaro and Buonavoglia, 2011). Transmission of the virus is usually through direct contact from dog-to-dog, or indirectly via exposure to fomites or environments contaminated with faecal matter.

Surveillance of infectious disease amongst the Australian canine population has been rare and consequently few studies on the epidemiology of such diseases have been published to date. In January 2010, Virbac Animal Health in Australia launched Disease WatchDog, a prospective national disease surveillance project, to capture data on communicable disease cases and outbreaks (Ward and Kelman, 2011). This provided the first dataset of information on the epidemiology of CPV outbreaks, and other infectious diseases, at a national scale in Australia.

Anecdotally, Australian veterinarians have reported disproportionally more cases of canine parvovirus from rural areas. A recent media release (AVA, 2014) reported a rise in the number of parvovirus cases across the entire country during the preceding months. Tamworth in rural New South Wales was reported to have the highest number of cases at the time (50 cases), with other rural areas—such as Chinchilla QLD, Orange NSW and Albury NSW—also reported as disease hot spots. The aim of the current study was to determine if more cases of canine parvovirus are reported from rural versus urban areas of Australia, and to identify potential risk factors for the differential reporting of CPV cases from rural versus urban areas.

2. Materials and methods

2.1. Data source

All case data for the study was acquired from Disease WatchDog, a national disease surveillance program officially launched in January 2010 to capture data on diseases in dogs (including parvovirus) and cats throughout Australia (Ward and Kelman, 2011). The online database relies on the participation of veterinary practices around Australia and access to submit data is restricted to registered veterinarians and veterinary nursing staff at such clinics. In return, the database provides veterinarians and veterinary nursing staff with the ability to view maps of disease cases and outbreaks in real-time (Ward and Kelman, 2011).

Records with a reported case diagnosis date between 22 September, 2009 and 27 July, 2014 were extracted from the Disease WatchDog database. All cases were screened for duplicate entries prior to data analysis. Any case in which the recorded postcode was invalid and not matched to an Australian postcode (GDA 1994 coordinate system, Lambert Conformal Conic projection; ArcGIS v10. ESRI, Redland CA) was removed prior to data analysis. Each record was counted as a single case report, even if it was a report of a litter.

Each case report was allocated a case identification number and contained the following data fields: clinic name, veterinarian name, case date, animal name, suburb, state, region, postcode, country, species, breed, age (years, months, weeks), sex (male, female or unknown), neuter status (entire or neutered), disease (canine parvovirus), co-infection diagnoses, co-infection disease, case outcome (animal died, euthanased, tested positive but not clinically affected, treatment ongoing, or recovered), case diagnosis (clinical presentation, ELISA snap test, PCR, immunofluorescence or other), vaccination status (vaccinated, unvaccinated or unknown), the vaccine given and vaccination date. There was also a field available to record if the case was a litter and number of animals in the litter affected, however the latter was not used in this study.

2.2. Data management

The variables extracted from the Disease WatchDog database and analysed were case outcome, age, sex, neuter status, breed and vaccination status. These epidemiological risk factors were categorised for statistical analysis. Case outcome was recorded as 'animal recovered', 'treatment ongoing', 'animal died', 'animal was euthanased' and 'animal tested positive but not clinically affected'. For data analysis, the categories 'animal died' and 'animal was euthanased' were combined to create a single outcome variable 'animal died'. The categories 'animal recovered', 'treatment ongoing' and 'animal tested positive but not clinically affected' were retained.

Age data extracted from Disease WatchDog was recorded in years, months and weeks. For data analysis, all ages were converted into weeks only, based on the assumption that 1 month consisted of 4 weeks and 1 year consisted of 52 weeks. Sex was recorded as male, female or unknown. Neuter status was recorded as entire or neutered.

Specific breed information was categorised for data analysis based on the Australian National Kennel Council breed standards. Breeds were allocated to one of seven categories and cases were classified by breed as 'toy', 'terrier', 'gundog', 'hound', 'working dog', 'utility' or 'non-sporting'. Any cases reported as crossbreds or mixed breed were categorised as 'mixed'. Some breeds reported in the Disease WatchDog are not recognised by the Australian National Kennel Council; these were subsequently classified as non-sporting (Pitbull, American Pitbull, Tibetan Spaniel), working (Bull Arab, Koolie, Australian Queensland Heeler, Blue Heeler, Red Heeler, Catahoula Leopard Dog, Cardigan Welsh Corgi, Smithfield), and hound (Dingo, Wolf). Cases reported as 'other canine' were categorised as 'unknown'.

Vaccination status was classified as vaccinated, unvaccinated, unknown or incomplete. Based on reported information regarding date of vaccination, dogs classified as vaccinated were further categorised as vaccination incomplete (i.e. last recorded vaccination before 16 weeks of age), vaccinated within the previous 12 months, non-recent vaccination (last recorded vaccination greater than 12 months—but less than 3 years—prior to infection), or vaccinated more than 3 years prior to the case report date. For analysis, vaccination was further categorized into never vaccinated versus vaccinated.

Any missing information for the above variables in the reported cases were categorised as 'unreported' for the purposes of data analysis.

Australian postal codes were classified as either rural or urban based on information from the 2006 Australian census, made available by the Australian Bureau of Statistics (ABS, 2014). For each postcode, the 'usual residential population' was divided by postcode land area to calculate postcode population density (residents per square km). Postcodes with a population density of