Canine Parvovirus Types

Canine Parvovirus-1 infection can occur via oronasal or transplacental transmission. After transmission the virus replicates within lymphatic tissues and intestinal epithelium. It is generally regarded as non-pathogenic as most infections are asymptomatic. Canine Parvovirus-1 can cause enteritis and diarrhea in neonatal puppies. It can also cross the placenta and cause early fetal death and abortion, or birth defects to fetal pups in dams infected within the first 30 days of gestation. Gastroenteritis has been seen in some adult dogs and there have been some reports of neurologic signs as well.

Canine Parvovirus-2 was the principal virus circulating in the dog population in the late 1970s. There has been controversy over whether Canine Parvovirus-2 had evolved as a direct mutant of Feline Panleukopenia Virus, or from a feline panleukopenia vaccine. This was finally settled when Dr. Truyen, et al published a paper in 1998 that proved Canine Parvovirus-2 was not a direct variant of Feline Panleukopenia Virus or the feline panleukopenia vaccine virus strain.

Initial cases of Canine Parvovirus-2 were characterized by a myocarditis in puppies. Others developed a mucoid diarrhea or hematochezia. Though morbidity was high the overall mortality rate was low for this virus, except in puppies less than 3-4 months old.

During the initial outbreak the Feline Panleukopenia vaccines were used in the emergency setting while homologous Canine Parvovirus vaccines were being developed. So, although Canine Parvovirus-2 appeared to be getting better controlled, some veterinarians began reporting outbreaks between the years 1981-1982. Puppies were noted to collapse in a shock-like state and die. Some never even developed enteric clinical signs whereas other puppies developed an acute, rapidly progressive illness characterized by a severe hemorrhagic diarrhea, dehydration, vomiting and shock. The severity of these clinical signs differed from the initial outbreak of Canine Parvovirus-2. Also of noted was that these puppies were poorly responsive to IV fluid therapy alone, which was a very reliable treatment in the initial outbreak. It was at this time that Canine Parvovirus-2a was emerging. Despite the fact that the clinical signs of disease for Canine Parvovirus-2a were more severe than the original Canine Parvovirus-2 initially the vaccines produced to protect against Canine Parvovirus-2 seemed to confer immunity against Canine Parvovirus-2a as well.

By the early 1980s the Canine Parvovirus-2b was noted. Canine Parvovirus-2a and 2b became the predominant types circulating in dog populations worldwide. The original Canine Parvovirus-2 disappeared around 1981. Within about 8 years after it emerged, Canine Parvovirus-2 had undergone evolutionary change, including an extended host range – cats! In 2000-2001, a third strain of the virus was isolated in Italy, Canine Parvovirus-2c. Canine Parvovirus-2c is distinguishable from Canine Parvovirus-2a and 2b by the substitution of Glutamic acid in lieu of Asparagine or Aspartic acid at residue 426 of the capsid protein VP2.

This area involves a major antigenic site and results in a change in antigenicity that has made it possible to differentiate between the three strains using monoclonal antibodies. This may also result in changes in vaccine efficacy and usefulness of some monoclonal antibody-type diagnostic assays. Despite anecdotal reports that Canine Parvovirus-2c is more pathogenic there is no evidence of different virulence between strains. Parvovirus does not affect all dogs equally. Different strains will result in varied effects based on the age of the animal, immunity status, breed, route of exposure and viral dose.