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Diagnosis of Canine Viral Infections

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Canine viral infections may be tentatively diagnosed on the basis of clinical signs, hematologic findings, and/or gross pathology; however, definitive diagnosis generally requires laboratory assistance. Laboratory diagnosis of these infections relies on one or more of the following procedures: histopathology, virus isolation, serology, and the detection of virus or viral antigens, using electron microscopy (EM), fluorescent antibody techniques (FAT), immunoperoxidase techniques (IPX), and enzyme immunosorbent assays (EIA). The approach to diagnosis frequently is dictated by the presenting clinical signs and the virus suspected.

Diagnostic methods such as EM, FAT, IPX, and EIA have begun to supersede the use of other diagnostic methods, particularly virus isolation and serology, because these methods are rapid and relatively inexpensive. However, FAT, IPX, and EIA require specific, specially prepared reagents; therefore, these techniques are utilized primarily for the diagnosis of commonly encountered virus infections such as canine distemper. Virus isolation and serology often tend to be relegated to those infections for which specific reagents are not available.

Electron microscopy is a particularly useful method for diagnosing cases of virus-induced enteritis. In general, virus isolation is not a logical approach to diagnosis, because the viruses that cause these infections are difficult to propagate in laboratory host systems.

Serologic diagnosis requires the measurement of the patient's antibody response to a specific virus. Traditionally, this has been accomplished using serum collected near the onset of clinical signs (acute sample) and a second sample collected 10 to 14 days later (convalescent sample). Serologic diagnosis using this approach requires the demonstration of a rising antibody titer in these paired serum samples. This approach to diagnosis has two distinct problems: the frequent absence of paired serum samples and the long delay in diagnosis when paired samples are available. An alternate serologic approach, which circumvents these two problems, involves the detection of antiviral IgM antibody in the acute serum sample. Antiviral

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IgM antibody is present only for a short period of time after infection (less than 30 days); therefore, its detection indicates recent or current infection.⁵ Use of this serologic approach requires knowledge of the patient's vaccination history; recent vaccination also results in a virus-specific IgM antibody response.

Specimens for virologic analysis (Table 1) are selected based on the patient's presenting clinical signs and the virus suspected. Tissue for histopathologic examination should be placed in 10 per cent buffered neutral formalin (10 volumes to 1 volume of tissue); these may be transported to the laboratory without refrigeration. Specimens for virus isolation, FAT, and EM should be chilled as soon as they are collected and transported to the laboratory with minimal delay. These may be shipped on ice or frozen with dry ice. If dry ice is used, specimens for virus isolation must be placed in sealed containers because carbon dioxide released from the dry ice may inactivate virus. Blood smears and slides prepared from tissue scrapings should be air dried and may be shipped without refrigeration.

Antemortem specimens for virus isolation may be submitted on cotton swabs. These should be immersed in 2 to 3 ml of virus-transport medium immediately after collection and refrigerated as described previously. Several transport media for viruses are commercially available.*

Specimens for viral diagnostic analysis should be collected early in the disease process. Virus and viral antigens rapidly disappear from tissues as the immune response is generated; therefore, the probability of making a diagnosis, based on virus isolation and antigen detection, decreases as the disease progresses. Likewise, the timing of serum sample collection is important; a rising antibody titer may not be detected if the acute serum sample is collected late in the acute phase of infection. The acute serum sample must be collected early, before a significant antibody response is generated.

CANINE DISEASES WITH SYSTEMIC MANIFESTATIONS

Canine viruses, which affect multiple organ systems and, therefore, induce a variety of clinical signs, include canine distemper virus (CDV) and canine adenovirus type 1 (CAV-1), the cause of infectious canine hepatitis. Systemic viral infections affecting neonates will be considered separately.

Canine Distemper

Canine distemper virus (CDV) is a highly contagious disease of dogs caused by a paramyxovirus. This virus is essentially pantropic, replicating in epithelial, lymphoid, and nervous tissue. The presenting clinical signs of CDV infection are dependent upon the degree of virus-induced damage in these various tissues. Clinical signs may consist of coryza, conjunctivitis, bronchitis, catarrhal pneumonia, gastroenteritis, and neurologic disturbances. Lymphopenia commonly is present early in the course of the infection.¹⁸

Infection with CDV is initiated by droplet and aerosol exposure. The

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Table 1. *Diagnosis of Canine Viral Infection: Selection of Specimens*

	HISTOPATHOLOGY	VIRUS ISOLATION	ELECTRON MICROSCOPY	SEROLOGY	FAT	MISCELLANEOUS
Systemic Infection						
Canine distemper virus	Brain, lung, kidney, stomach, bladder	IA*	IA	IgM, paired serum samples, CSF	Buffy coat and CSF cells, conjunctival scrapings, brain, lung, kidney, stomach, bladder	—
Infectious canine hepatitis	Liver, spleen, lymph nodes	Liver, kidney, urine	IA	IgM, paired serum samples	Kidney	—
Respiratory Infection						
	Trachea, lung	Nasal swabs, trachea, lung	IA	Paired serum samples	IA	—
Enteritis						
Canine parvovirus	Intestines, thymus, spleen	Fecal swabs, feces, intestines	Feces	IgM, paired serum samples	Intestines	Feces (EIA, HA)
Canine coronavirus	Intestines	IA	Feces	IA	IA	—
CNS Infection						
Rabies	Brain	Brain	IA	IA	Brain	—
Canine distemper virus	Brain	IA	IA	CSF	Brain	—
Canine paramyxovirus		CSF, brain				
Pseudorabies virus	Brain, spinal cord	Brain, spinal cord	IA	IA	Brain	—
Neonatal Infection						
Canine parvovirus	Heart, lung, liver, spleen, kidney, brain, intestines	Heart, lung, spleen, kidney, brain, intestine	IA	IA	Heart, lung, liver, spleen, kidney, brain, intestines	—
Canine herpesvirus	Heart, lung, liver, spleen, kidney, brain	Heart, lung, liver, spleen, kidney, brain	IA	IA	Heart, lung, liver, spleen, kidney, brain	—

*IA = infrequently applied to diagnosis.

virus replicates initially in macrophages and lymphocytes of the respiratory tract and local lymph nodes, then spreads within a few days to other lymphatic organs. At this point, the outcome of the infection is dependent upon the host-immune response to the virus. Restricted antibody formation, as a result of virus-induced immune suppression, results in continued viral replication and spread.¹⁸ Unrestricted spread of the virus results in infection of the respiratory, urogenital, and alimentary tracts and ultimately the brain. Death frequently occurs in these dogs 3 to 4 weeks after infection owing to a demyelinating encephalitis.

In those dogs that immunologically respond with the progressive production of antibody, CDV infection is mild to inapparent, and these dogs invariably recover.¹ However, CDV may remain in a latent state within the brains of these animals and manifest itself years later as a demyelinating encephalitis, frequently referred to as "old dog encephalitis."¹¹

Diagnosis. Canine distemper often is presumptively diagnosed on the basis of presenting clinical signs and history—an unvaccinated puppy, for example. Additional evidence in support of such a diagnosis may be obtained from hematologic findings. Hematologic findings consistent with CDV infection include lymphopenia and the presence of inclusion bodies in blood and bone marrow cells.^{13,18,24} Inclusion bodies may be detected in leukocytes, erythrocytes, and their precursors in blood and bone marrow smears stained with Wright's stain. In lymphocytes and neutrophils, these appear as large, gray, oval structures, singular in lymphocytes, and often multiple in neutrophils. In erythrocytes and reticulocytes, inclusions are light blue and slightly larger than Howell-Jolly bodies.^{13,24}

Laboratory approaches to the diagnosis of CDV infection include pathology, the detection of CDV-specified antigens in tissues, and serology. Virus isolation is infrequently utilized owing to the availability of faster and more reliable diagnostic approaches.

Antemortem diagnosis of CDV infection frequently is based on the detection of CDV antigens in cells and tissue scrapings, principally using FAT. The FAT may be applied to buffy coat cells, cerebral spinal fluid cells, and scrapings of the conjunctiva, prepuce, and vagina.³ False-negative results may occur if this approach to diagnosis is applied late during the course of the infection.

Serologic diagnosis may be accomplished using traditional methods—that is, paired serum samples or via the detection of anti-CDV IgM antibody. When IgM antibody detection is used, attention must be given to the vaccination history of the patient. Animals inoculated with CDV vaccine within the preceding 3 weeks may have a measurable IgM antibody titer to CDV.⁵

The serologic detection of CDV-specific antibody in cerebrospinal fluid often is useful in detecting CDV infection in animals presenting with neurologic disturbances.¹⁵ Antibodies are present in cerebrospinal fluid as a result of local production but also may be present owing to nonspecific damage to the blood-brain barrier. Local production of CDV-specific antibody, and its appearance in cerebrospinal fluid, occurs subsequent to central nervous system infection. Studies by Appel indicated that CDV-specific antibody could be detected in the cerebrospinal fluid of dogs with subacute CDV encephalitis but not in dogs that recovered early or died acutely from

infection. In addition, cerebrospinal fluid antibody to CDV was not detected in vaccinated dogs.^{2,3} Nonspecific damage to the blood-brain barrier, resulting in leakage of serum antibodies into the cerebrospinal fluid, may be determined by comparing serum and cerebrospinal fluid antibody titers to other canine viruses, such as CAV-1 and canine parvovirus.⁴

Postmortem diagnosis of CDV infection may be established using pathologic findings or by detection of CDV-specific antigens in tissues. Gross pathology is generally unremarkable; thymus atrophy and interstitial pneumonia may be observed. Histopathologic examination is much more informative and may reveal the presence of eosinophilic inclusion bodies; these are intracytoplasmic and intranuclear in the brain and in epithelial cells of the lung, stomach, bladder, and kidney. Inclusion bodies also may be detected by FAT; this technique is considered to be more sensitive than histopathologic detection.²¹

Infectious Canine Hepatitis

Infectious canine hepatitis (ICH) is a disease of dogs caused by canine adenovirus type 1 (CAV-1). Antigenically, this virus is closely related to canine adenovirus type 2 (CAV-2), a cause of respiratory disease in dogs, and the virus from which a vaccine for ICH has been derived.

The character of CAV-1 infection may range from a mild, inapparent infection to one that is severe and rapidly fatal. The severe form of the disease primarily occurs in young dogs near the time of weaning. Two forms of CAV-1 infection are recognized: a localized respiratory infection and a generalized infection manifesting primarily as hepatitis.^{23,26} Generalized infection (ICH) follows oral exposure to the virus, after which the virus becomes widely disseminated and replicates in endothelial and hepatic cells primarily.²³ Virus-induced damage to these cells is manifested clinically as edema, hemorrhage, and hepatitis. In addition, CAV-1-infected dogs frequently present with vomiting, diarrhea, anorexia, conjunctivitis, and leukopenia.²³

Diagnosis. Diagnosis of generalized CAV-1 infection may be based on pathologic changes, virus isolation, serology, and the detection of CAV-1-specific antigens in tissue. Pathology findings in these infections reflect endothelial and hepatic cell damage; these include hemorrhage, edema, and an enlarged, mottled liver. Microscopically, eosinophilic, intranuclear inclusion bodies and degeneration are seen in endothelial cells, Kupffer's cells, and hepatocytes.

Virus isolation and paired serum samples serology may be utilized in diagnosis; however, these methods are relatively slow. Isolation of the virus is readily accomplished; urine, kidney, and portions of the liver from affected dogs should be submitted when one wishes to attempt virus isolation. Virus isolation should not rely on liver tissue alone because the presence of hepatic arnase in this tissue may interfere with recovery of the virus.⁷

Rapid diagnosis of CAV-1 infection may be obtained by the detection of a CAV-1 IgM antibody titer or by the detection of CAV-1 antigen in the liver, primarily using FAT.

RESPIRATORY VIRAL INFECTIONS

Several viruses have been associated as causes of respiratory infection in the dog; these include CDV, CAV-1, CAV-2, canine paramyxovirus, canine herpesvirus, and reovirus types 1 and 2. These viruses, with the exception of CDV, cause localized infections of the respiratory tract that are similar in clinical presentation and pathology.

Respiratory infection by these viruses is generally mild in character and often inapparent; however, severe disease, including pneumonia, may occur when viral infection is complicated by other respiratory pathogens, particularly *Bordetella bronchiseptica*. Clinically, these infections are characterized by fever, nasal discharge, and a mild to severe cough.

Infectious tracheobronchitis, commonly referred to as "kennel cough," is usually a mild, upper respiratory infection characterized by the presence of a harsh, dry, paroxysmal cough. Occasionally, these infections progress to bronchopneumonia.

Diagnosis of Respiratory Infection

A tentative determination of an infectious etiology in cases of canine respiratory disease may be based on clinical signs and epidemiologic information. The presence of fever and the contagious nature of these infections often are useful in distinguishing infectious causes of respiratory disease from other causes such as airway obstruction, congestive heart failure, allergies, and parasitisms.

Pathologic changes present in cases of virus-induced respiratory disease frequently are similar; therefore, other laboratory procedures are required to differentiate these infections. Gross lesions are confined to the respiratory tract and consist of hemorrhage, congestion and consolidation. Microscopically, bronchitis, bronchiolitis, and interstitial pneumonia may be present.

Definitive diagnosis is usually determined by virus isolation and serology; antigen detection techniques are applied to these cases infrequently owing to the requirement for virus-specific reagents. The viruses associated as causes of canine respiratory infection, with the exception of CDV, are readily isolated. Suitable specimens for virus isolation are nasal swabs and respiratory tissues.

Serologic diagnosis of these infections primarily is accomplished using paired serum samples. The detection of IgM antibody as an approach to diagnosis has been limited to CDV and CAV-1 infections; however, this approach also should be applicable to the diagnosis of other canine respiratory infections.

ENTERITIS

Canine parvovirus (CPV) and canine coronavirus (CCV) are causes of enteric infections in the dog. Rotaviruses and astroviruses have been detected in the feces of dogs with diarrhea; however, the importance of these viruses as causes of canine enteritis has not been established.

Canine Parvovirus

Canine parvovirus (CPV) causes two distinct disease syndromes in the dog, namely enteritis and neonatal infection.²⁰ The enteric form of infection primarily occurs in dogs 8 weeks or older; severe enteric infection occurs in young puppies 8 to 16 weeks of age. Infection initially begins in the pharynx, after which a viremia develops that disseminates the virus to other tissues. Typical of parvoviruses, this agent replicates and induces damage in cells that are mitotically active. Consequently, clinical signs and pathologic changes reflect virus-induced damage to intestinal crypt epithelial cells, bone marrow stem cells, and lymphoid cells.²⁰ Clinical signs associated with CPV enteritis include fever, depression, vomiting, and a diarrhea that is frequently hemorrhagic.

Canine Coronavirus

Canine coronavirus (CCV) causes a localized infection of the intestinal tract similar to other enteric coronaviruses, such as bovine coronavirus and transmissible gastroenteritis virus of pigs.¹⁶ Replication of the virus and virus-induced damage are restricted to mature villous epithelial cells. Clinically, CCV infection may mimic CPV-induced enteritis.

Diagnosis of Viral Enteritis

A tentative diagnosis of CPV-induced enteritis frequently is based on clinical signs, principally a severe enteritis in conjunction with fever. However, other agents that cause similar clinical signs, such as CCV, *Salmonella* spp., and *Leptospira* spp., must be considered. Clinical pathology may provide corroborative evidence; CPV infection is frequently accompanied by the presence of neutropenia, lymphopenia, or pancytopenia.

Several methods are available for confirming a diagnosis of CPV enteritis. These include pathology, serology, the detection of CPV in feces by virus isolation, electron microscopy (EM), or enzyme immunosorbent assays (EIA), and the detection of CPV antigens in intestinal tissue using fluorescent antibody techniques (FAT).

Pathologic changes due to CPV infection are characteristic and consist of crypt epithelial necrosis in the small intestines and dilated lymphatics. In advanced cases, extensive loss of epithelial cells, crypt dilatation, and an inflammatory cell infiltrate of the lamina propria may be present. Intracellular inclusion bodies infrequently are detected. Lymphoid depletion often is present in the thymus, Peyer's patches, lymph nodes, and spleen.

The virus may be detected in feces by various means, including virus isolation, HA, EM, and EIA.^{8,9,22} In addition, CPV antigens may be detected in tissues by FAT. Diagnosis of CPV infection using these methods is successful only when feces and tissue samples are collected early in the course of the infection.²⁷ An immune response to CPV is generated rapidly, beginning 4 to 5 days after infection.²⁷ As a consequence, the virus can be detected in feces and tissues of infected dogs for only a short period of time after the onset of clinical signs.

Hemagglutination is widely utilized to detect the virus in feces owing to the speed, simplicity, and sensitivity of this method. Nonspecific HA

reactions occur; however, specificity of the reaction is determined by hemagglutination inhibition using CPV-specific antiserum. Electron microscopy is relatively insensitive; in addition, it cannot distinguish between CPV and a nonpathogenic parvovirus, minute virus of dogs, which may also be present in feces. Recently, a commercially available EIA has been developed, that is rapid, simple to perform, and almost as sensitive as HA.²² This assay is similar to the commercially available EIA for detecting feline leukemia virus infection and conceivably will be used by veterinary practitioners for the same purpose.

Serologic methods infrequently are applied to the diagnosis of CPV enteritis. However, diagnosis based on IgM antibody detection has been successfully applied to these infections and has particular value in those cases in which diagnosis is sought relatively late during the course of the infection, when virus cannot be detected by other methods.^{5,27}

Methods currently available for the diagnosis of CCV enteritis are limited primarily to pathology and EM; CCV is difficult to isolate and virus-specific reagents frequently are not available. Microscopic pathologic changes are characteristic and include villous atrophy, villous fusion, flattening of epithelial cells, and deepening of intestinal crypts. Electron microscopy is a valuable approach to the diagnosis of CCV infection, but caution must be exercised in the interpretation of EM findings, because artifacts resembling coronaviruses are commonly present in feces.

CENTRAL NERVOUS SYSTEM INFECTION

Viruses causing central nervous system infection in the dog include CDV, rabies virus, pseudorabies virus (PrV), and canine paramyxovirus. The diagnosis of CDV-induced neurologic disease is discussed in a previous section of this article.

Rabies Virus

Canine rabies virus infection occurs after intramuscular or subcutaneous inoculation of the virus, generally the result of being bitten by an infected animal. The virus may penetrate nerve endings immediately; however, local replication in myocytes generally occurs, after which the virus penetrates nerve endings and travels centripetally to the central nervous system within the axoplasm of nerves. Viral replication within the central nervous system is followed by centrifugal spread of the virus to non-neural, peripheral tissues; these tissues include the salivary glands and highly innervated tissues, such as the cornea and tactile hair follicles.

Clinical signs of infection generally develop as a progression of stages; a prodromal stage is followed by either the furious form or the dumb form of rabies. The prodromal stage of infection, characterized by changes in temperament, frequently is not recognized except in closely observed pets. Viciousness, seizures, and altered phonation, due to partial paralysis of the vocal cords, occur during the furious form of the infection. The dumb form is characterized by a progressive paralysis that begins in the head and neck and results in facial paralysis, drooling, and an inability to chew or swallow.

Diagnosis. Approaches to the diagnosis of rabies virus infection include histopathology, FA, and virus isolation using mice. Histopathologic diagnosis is accomplished by the detection of eosinophilic, intracytoplasmic inclusion bodies called Negri bodies. These inclusions may be diffusely scattered throughout the brain but are more commonly found in the pyramidal cells of the hippocampus. The presence of Negri bodies establishes a definitive diagnosis of rabies virus infection; however, in approximately 15 to 20 per cent of infected animals, these inclusions may not be detected.²⁵ Negri bodies are formed relatively late during the course of the infection and, therefore, may not be detected in those animals that die or are euthanatized prior to their formation.¹⁷

The FAT is a sensitive and more accurate method of diagnosis than Negri body detection; this technique will detect infection in up to 98 per cent of affected animals.¹⁷ Antemortem diagnosis of rabies virus infection also may be achieved by applying this technique to corneal scrapings and tactile hair biopsies.⁶ However, false-negative results may be obtained if antemortem diagnosis is attempted early during the course of the infection, prior to centrifugal spread of the virus to peripheral tissues.

Isolation of the virus by intracerebral inoculation of mice is almost as sensitive as FAT; however, diagnosis using this procedure requires several days. This approach to rabies virus diagnosis is used infrequently, principally to confirm histopathologic and FAT results.

Pseudorabies Virus

Pseudorabies virus (PrV) infection is a relatively uncommon cause of canine neurologic disease. Infection is acquired by direct contact with infected swine or by the ingestion of raw, contaminated pork. Clinically, PrV infection may resemble rabies; infected dogs exhibit pharyngeal paralysis, profuse salivation and, occasionally, convulsions. However, it differs from rabies in that PrV-infected animals do not become vicious and they frequently are afflicted by an intense pruritus. The pruritus is manifested by self-mutilation, which is frequently so severe that animals exhibit maniacal behavior. The course of the disease is quite short, death generally ensuing within 48 hours after the onset of clinical signs.

Diagnosis. The presenting clinical signs, in conjunction with a history of recent exposure to swine, may be used for determining a tentative diagnosis of canine PrV infection. Definitive diagnosis is obtained by histopathology, virus isolation, or by the detection of PrV antigens in tissue; serology is of little value owing to the rapidly fatal progression of this infection.

Gross pathology is of little value; pulmonary congestion, pulmonary edema, and skin lesions due to pruritus may be observed. Central nervous system lesions are confined to the brain stem primarily. Microscopically, these include perivascular cuffing, gliosis, and the presence of eosinophilic, intranuclear inclusion bodies within astrocytes and neurons.

Pseudorabies virus antigens may be detected in the tissues of infected animals by FAT. This technique is used frequently owing to the availability of specific reagents otherwise used for the diagnosis of this disease in swine. Similarly, virus isolation frequently is applied to the diagnosis of canine PrV

infection. Brain and tonsils are the preferred specimens for these tests; these should be submitted whenever canine PrV infection is suspected.

Canine Paramyxovirus

Recently, neurologic infection due to canine paramyxovirus has been described. However, the importance of this virus as a cause of neurologic disease in the dog has not been established.¹² Clinical signs attributed to central nervous system infection by this virus consisted of incoordination and posterior paresis. Diagnosis was based on the isolation of the virus from cerebral spinal fluid.¹²

NEONATAL INFECTION

Canine herpesvirus (CHV) and canine parvovirus (CPV) have been identified as causes of canine neonatal infections. Diagnosis of these infections is achieved by similar approaches.

Canine Herpesvirus

Canine herpesvirus (CHV) induces a severe, frequently fatal, infection of young puppies; in older dogs, infection is mild and frequently inapparent.¹⁴ Severe infections occur only in puppies less than 2 weeks of age.¹⁰ The disease in young puppies is characterized by anorexia, dyspnea, and pain manifested by crying. Clinical signs of infection generally begin 5 to 14 days after birth. The course of the disease is quite short; affected puppies usually die 24 to 48 hours after the onset of clinical signs.

Canine Parvovirus

Canine parvovirus (CPV) may cause an acute, generalized infection in puppies less than 2 weeks old. This form of CPV infection can occur as a consequence of intrauterine infection or as a result of exposure to the virus soon after birth. A high level of mitotic activity is present in virtually all canine tissue types during the first 2 weeks of life; therefore, CPV replication and damage are not restricted to the bone marrow, intestinal tract, and lymphoid tissues as is the case in older dogs.¹⁹

Young dogs, 3 to 8 weeks of age, may develop myocarditis as a result of CPV infection. This is believed to be a late sequela of neonatal CPV infection. The dogs that survive neonatal infection subsequently may develop heart failure due to an immunopathologic response to CPV-infected myocytes.¹⁹ These animals often die suddenly without manifesting clinical signs; however, dyspnea, crying, and electrocardiographic abnormalities may be detected.

Diagnosis of Neonatal Infection

Diagnosis of canine neonatal infections is based principally on pathology, virus isolation, and the detection of virus-specific antigens in tissue. Pathologic changes in puppies with CHV infection, or generalized CPV infection, consist of disseminated focal necrosis in most organs. Characteristic of CHV infection is the presence of hemorrhage in these organs; subcapsular hem-

orrhages of the kidneys are especially prominent and appear as bright red spots. Microscopically, intranuclear inclusion bodies may be detected in either of these infections.

Pathologic changes in cases of CPV myocarditis are localized primarily to the heart and lungs. Characteristic of these infections is the presence of focal areas of fibrosis within the myocardium, which appear as pale streaks. Myocardial lesions are characterized microscopically by edema and a mononuclear infiltrate primarily composed of lymphocytes and plasma cells. Lung lesions consist of congestion, hemorrhage, edema, and mild interstitial pneumonia.

Canine herpesvirus and CPV may be isolated from various tissues including heart, lung, liver, spleen, kidney, and brain. In addition, CPV may be isolated from intestinal tissues. These tissues should be submitted when infection due to these viruses is suspected. Although CHV is readily isolated, CPV is relatively difficult to isolate and may require several cell culture passages.

Antigen detection techniques, principally FAT, may be applied to the diagnosis of these infections whenever virus-specific reagents are available.

SUMMARY

The diagnosis of canine viral infection frequently requires the correlation of clinical, hematologic, pathologic, and laboratory findings. When laboratory diagnostics are utilized, appropriate specimens must be collected. In addition, correct timing of specimen collection and the proper handling and transport of these specimens are essential.

Whenever possible, the practitioner should not rely on any one laboratory procedure for making a diagnosis; rather, a combination of techniques should be used. Submission of tissue for histopathology, virus isolation, and antigen detection techniques (FAT), serum for serologic analysis, and feces whenever enteric infections are investigated will maximize the chances of making a successful diagnosis.

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