OPHIDIAN PARAMYXOVIRUS (OPMV)

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■ INTRODUCTION

Historically, gram-negative microorganisms including *Pseudomonas* spp., *Providencia* spp., *Proteus* spp., *Salmonella* spp., *Aeromonas hydrophila* and *Escherichia coli* have been the most commonly isolated bacteria from the respiratory tract of reptiles with clinical signs of pneumonia. Prior to 1976, relatively few viral infections of reptiles had been reported, and virtually nothing was known about viral infections of the respiratory tract of reptiles.

In 1972, a respiratory epizootic spread through a collection of lance-headed vipers (*Bothrops moojenii*) at a serpentarium in Switzerland. Initially *Pseudomonas* and *Aeromonas* were isolated from the respiratory tract of dead snakes and the disease was originally thought to be bacterial in origin. Eventually a virus with morphological and biochemical properties of certain myxoviruses was isolated and was tentatively placed in the paramyxovirus subgroup 2.

Since its first description, ophidian paramyxovirus (OPMV) has surfaced as an extremely important pathogen of viperid snakes. In 1979 the first outbreak was reported in a private collection in the United States, and since that time numerous outbreaks have been seen in collections of viperid snakes in the United States and Mexico. A similar virus has been isolated from a black mamba (*Dendroaspis polylepis polylepis*) and multiple species of rat snakes including corn snakes (*Elaphe guttata*), beauty snakes (*Elaphe taeniurus*) and Moelendorff's rat snakes (*Elaphe moellendorffi*). In the Federal Republic of Germany, myxovirus-like agents were recently recovered from a red-tailed rat snake (*Gonyosoma oxycephala*), diamond python (*Morelia spilotes*) and rhinoceros viper (*Bitis nasicornis*).

In general all viperid species should be considered susceptible to infection and there also have been reports of infection in colubrid, boid, and elapid snakes. Both juveniles and adults are affected. There are no reports of infection in neonates.

The virus appears to be distributed worldwide in zoological and private collections including The United States, Mexico, Argentina, and Germany.

■ CAUSATIVE AGENT

The infectious agent was identified as a virus and called ophidian paramyxovirus (OPMV). Based on several characteristics it appeared to belong to the virus family *Paramyxoviridae* to which also the measles and mumps viruses belong.

Paramyxoviruses are very small particles, 100 to 200 nanometre (nm) in diameter (one nm is one millionth of a millimetre). They consist of genetic material (RNA) which is surrounded by a protein layer and a so-called envelope. The shape of the virus particles is pleomorphic, ranging from spheroidal to filamentous. Viruses depend on other living cells for their multiplication. They use the machinery of living cells to replicate and they spread by the process of budding in which many newly formed virus particles are excreted through the outer layer of the cell, the cell membrane.

Newly formed viruses will infect healthy cells and these cells will in turn produce a large number of progeny virus particles.

■ CLINICAL SIGNS OF THE DISEASE

In the original epizootic involving lance-headed vipers, clinical signs lasted 5 to 12 days and progressed through 4 stages. During stage 1 there was a loss of muscle tone, with affected snakes exhibiting a "stretched out" linear posture with the head slightly elevated. During stage 2, which lasted 1 to 2 days, snakes showed abnormal activity. Affected snakes crawled about restlessly and kept their mouths partially opened. Their tongues were incompletely withdrawn into the sheathes and their pupils were extremely dilated. Stage 3 was seen from several hours to one day preceding death. The mouth was kept completely open and the snakes expelled a purulent material from the glottis. Stage 4 was seen from several minutes to one hour preceding death. The mouth

was kept fully opened, the pupils were dilated, and animals were excessively active.

In an epizootic involving rock rattlesnakes (*Crotalus lepidus*), a new breeder male was introduced into an established collection without being quarantined. Ultimately this snake was in contact with 8 other rock rattlesnakes, 7 of which died. On day 3 following introduction the new snake developed head tremors and loss of equilibrium; it died on day 14. Over the next 2 1/2 months, 4 females and 3 males died after manifesting clinical signs. Only one rattlesnake remained healthy and survived.

In those snakes seen in the terminal stages of the disease, immediately preceding death, these animals generally manifest a convulsive behaviour. This should not be confused with primary central nervous system disease described in rock rattlesnakes. These are agonal signs and are rather non-specific. Snakes may twist around, become flaccid and quiet for a period of time, and initiate these death-throws all over again.

In many of the outbreaks on OPMV infection, minimal or no clinical signs are noted by the keeper/owner. Often snakes will be found dead in their cage early in the morning, having died the night before. Many appear to be in good health with good weight and normal behaviour prior to death. Clinical signs can be subtle or non-specific such as off feed for one to two weeks. Although clinical signs in the earlier stages of the disease are often subtle, abnormal respiratory sounds are audible when ill snakes are manually restrained. If the oral cavity is examined, exudate may be seen within the glottal opening. Some snakes die with blood expelled from the glottis and filling the oral cavity. In a group of Siamese cobras (*Naja naja kaouthia*), the major consistent clinical signs was polyuria (increased urination). These snakes became ill during a die-off of rattlesnakes in the same room; paramyxovirus was isolated from dead rattlesnakes.

■ PATHOLOGY

It is the respiratory system that appears to be targeted by OPMV infections. Gross changes ranged from diffuse haemorrhage of the lung and air sac system to diffuse to focal accumulations of caseous necrotic cellular debris. Other organs which may be involved on a gross level are the pancreas and liver. Pancreatic hyperplasia is not uncommonly encountered in infected crotalid snakes. The authors have seen this quite commonly in timber rattlesnakes, *Crotalus horridus*. In the liver, areas of necrosis and formation of multifocal firm nodules (granulomas) may be seen. In the liver, lesions range from areas of caseation necrosis to granuloma formation. By special staining, gram-negative microorganisms are often demonstrated in these lesions. A variety of bacterial organisms have been isolated from these lesions with *Pseudomonas* spp. being the most common isolates. Bacterial organisms can invade the liver either from showering of bacteria from the gastrointestinal tract or from secondary bacterial invaders in the respiratory tract. Paramyxoviruses in mammals are known to have immunosuppressive effects and most likely results in a compromised immune system in snakes. Thus it is not surprising that these snakes often succumb to secondary pathogens.

Occasionally, OPMV infection may be manifested as an encephalitis. In a rock rattlesnake, demyelination and some degeneration of axon fibres with moderate ballooning of axon sheaths were seen in the brainstem and upper spinal cord. However, signs of central nervous system disease are not typically seen. When seen in a boa or python, consider inclusion body disease in the differential.

■ TRANSMISSION AND EPIDEMIOLOGY

According to reports in the literature and our recent experience with epizootics in private collections, once snakes start dying of OPMV infection, the mortality within a collection generally progresses fairly rapidly and peaks at about one month following initial deaths. It then declines through 2-3 months. In each epizootic we have investigated, although several species may comprise the collection, the virus seems to target a particular species. In some die-offs, the disease may result in the death of a large number of snakes over a more prolonged period time.

Although OPMV infections have occurred throughout the year, in many cases, epizootics have been experienced from January to May. Replication of the virus *in vitro* has been demonstrated to be temperature-dependent with an optimum temperature for growth at 30°C and a range of temperature for growth of 23°C to 32°C. Thus, possibly a latent infection may become activated if snakes are kept at suboptimal environmental temperatures. This may account for many of the epizootics occurring during

cooler times of the year or following hibernation.

Transmission most likely occurs by virus being expelled into the air as droplets from the respiratory system. Virus gaining access to water bowls and pools of water may persist for considerable periods of time. Transmission of virus via the digestive tract through faeces is also a possibility. Although transovarian or transuterine transmission has not been firmly established, this may also be involved in the spread of the virus.

The natural host for OPMV is unknown. Since rat snakes have been found to harbour a similar agent, possibly a non-viperid snake could be the source of infection. Although we have isolated this virus from recently imported snakes, there have been none isolated from snakes in the field. Snakes are an extremely mobile group of animals in the pet and zoo trade and this probably has accounted for this virus being introduced into many species of native snakes.

■ DIAGNOSIS

Diagnosis is generally difficult. Snake keepers will have to depend on close observations of their animals for the typical OPMV symptoms described above. Presumptive diagnosis of OPMV infection can be made upon finding characteristic light microscopic changes in lung tissue. Since lesions in the lungs can be segmented, sections of cranial, mid, and caudal lung should be examined. This can only be done by an expert. Specific diagnosis will depend upon isolation of the virus and multiplication in cell cultures. By electron microscopy, virus can be seen in infected cells. Virus replication in these cell cultures (and presumably in infected snakes) is temperature-dependent with the highest amount of virus achieved at incubation temperatures of approximately 23°C to 32°C. The upper temperature limit for virus replication was in all cases less than 37°C.

A test has been developed to determine the presence of specific antibodies to OPMV in blood plasma/sera of exposed snakes. Snakes, like all mammals, try to fight infections through their immune system. Upon an infection special blood cells will produce specific molecules, called antibodies, that recognize and help to destroy the bacteria or viruses that cause the infection. The presence of antibodies against OPMV in the blood, called a positive titre, is simply indicative of exposure to OPMV. Based upon a single sample, it would be impossible to make a statement about presence of virus and shedding status. If two samples are obtained from the same animal at a 2-4 week interval, and an increase in the amount of specific antibodies can be demonstrated, this would be supportive evidence for recent OPMV infection.

■ TREATMENT

There is no specific treatment for snakes showing clinical signs of OPMV infection. Since most affected snakes die with severe gram-negative respiratory tract infections, treating ill snakes with appropriate antibiotics is indicated. The aminoglycosides, gentamin and amikacin, in combination with a cephalosporin such as ceftazadime, are the drugs of choice.

Cages of ill snakes should be cleaned and completely disinfected with a solution of 0.15% sodium hypochlorite. Chlorox is 5.25% sodium hypochlorite; a 1:33 dilution can be used. Cages should remain empty for at least two weeks before introducing new animals. Additionally, as a rule, new snakes should not be introduced into a colony of snakes in which there is active OPMV infection. Minimally, two months should lapse following the last death from OPMV before introducing new animals. Needless to say, ill snakes should be removed from the collection and placed in a quarantine room.

Currently, there is no vaccine available for protecting snakes against OPMV.

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