

Adenovirus infection in bearded dragons

Fact sheet

Introductory statement

Adenoviral hepatitis is a common cause of neonatal and juvenile mortality in captive bearded dragons (*Pogona* spp.) in the USA. Although adenoviral infection has been reported in captive bearded dragons in Australia, the prevalence of disease is not known. Infection of wild reptiles with viruses having morphology consistent with adenovirus has not been confirmed in Australia (Ladds 2009). Ante mortem diagnosis is difficult and treatment usually unsuccessful. Free living reptiles and captive populations are at risk in Australia.

Aetiology

Adenoviruses are medium-sized (80–110 nm), non-enveloped viruses containing a double stranded DNA genome (Moormann et al. 2009). Adenoviral infections have been recorded from a large number of reptile species including snakes, dragons, skinks, geckos, chameleons, monitors, crocodiles and tortoises (Jacobson 2007). Adenoviruses are generally regarded as being species specific and the majority of infections in bearded dragons have been caused by Agamid adenovirus 1 (AgAdv-1), as confirmed by PCR (Wellehan et al. 2004; Kübber-Heiss et al. 2006; Wagner et al. 2007; Moormann et al. 2009; Doneley et al. 2014; Hyndman and Shilton 2016). However, there is one report of a helodermatid adenovirus infection in a western bearded dragon (*Pogona minor minor*), while AgAdv-1 has been found in a central netted dragon (*Ctenophorus nuchalis*), a species in the same subfamily as bearded dragons (Hyndman and Shilton 2011). Given the high prevalence of AgAdv-1 in bearded dragons overseas it seems likely that some, if not all, of the adenovirus infections in bearded dragons reported before the advent of PCR were due to AgAdv-1 virus (Julian and Durham 1982; Frye et al. 1994; Jacobson et al. 1996; Kim et al. 2002).

Natural hosts

Adenovirus infection has been reported predominantly in agamids of the *Pogona* genus. These include the eastern bearded dragon (*Pogona barbata*), central bearded dragon (*P. vitticeps*), Downs bearded dragon (*P. henrylawsoni*) and western bearded dragon. A case was also reported in a central netted dragon, which is in a different genus (Julian and Durham 1982; Frye et al. 1994; Jacobson et al. 1996; Hyndman and Shilton 2011;

Doneley et al. 2014). AgAdv-1 infection has been confirmed by PCR in the central bearded dragon and central netted dragon (Wellehan et al. 2004; Kübber-Heiss et al. 2006; Wagner et al. 2007; Moormann et al. 2009; Hyndman and Shilton 2011; Doneley et al. 2014). A similar virus was reported in a group of genetically related pygmy bearded dragons, *Pogona henrylawsoni*, in the USA (Frye et al 1994). Adenoviral infection has also been reported in a savannah monitor (*Varanus exanthematicus*) that died without showing premonitory signs. In the USA PCR studies also confirmed the presence of adenovirus in a blue-tongued skink (*Tiliqua scincoides scincoides*).

World distribution

Adenovirus first emerged as a cause of hepatitis in an illegally imported, confiscated eastern bearded dragon in New Zealand in the early 1980s (Julian and Durham 1982) and is now recognised as a common cause of sudden death in captive bearded dragons in the United States (Kim et al. 2002; Wellehan et al. 2004; Jacobson 2007; Wagner et al. 2007) and Europe (Kübber-Heiss et al. 2006; Moormann et al. 2009; Kubiak 2013). Cases of adenovirus infection in USA have been reported in central bearded dragons and Downs bearded dragons, while all the European cases occurred in central bearded dragons.

Occurrences in Australia

Adenoviral infection occurs in captive dragons in Australia. Published reports have identified adenovirus infections predominantly in central bearded dragons, but also in eastern bearded dragons, a western bearded dragon and a central netted dragon (Hyndman and Shilton 2011; Doneley et al. 2014; Hyndman and Shilton 2016). There are a small number of reports of the virus in apparently healthy free-living dragons (see Statistics).

Epidemiology

Transmission is by the faecal/oral route (Doneley et al. 2014). There is anecdotal evidence of vertical transmission, but this has not been confirmed (Parkin et al. 2009). One study failed to detect evidence of the virus by PCR in the ovaries of adenovirus positive females (Wagner et al. 2007).

Affected dragons are generally less than 12 weeks old (Doneley et al. 2014), but disease can occasionally occur in older animals (Moormann et al. 2009; Parkin et al. 2009). Concurrent dependovirus (a virus in the parvovirus family that cannot replicate without the assistance of a helper virus) and coccidial infections have also been diagnosed (Jacobson et al. 1996; Kim et al. 2002). There appears to be no sex-linked predisposition.

The virus can be carried asymptotically. A UK study of clinically healthy captive bearded dragons found a viral prevalence, based on PCR of cloacal swabs, of 40% in the 13–18 month age group and an 8% prevalence in animals over 24 months of age (Kubiak 2013).

Clinical signs

Clinical signs are often non-specific, including loss of appetite, weight loss, diarrhoea and weakness (Jacobson et al. 1996; Doneley et al. 2014). Neurological signs, such as head tilt, opisthotonos, circling and limb paresis may be observed (Kim et al. 2002; Doneley et al. 2014). Sudden death is a common occurrence (Kübbler-Heiss et al. 2006; Hyndman and Shilton 2011; Doneley et al. 2014).

Two separate outbreaks occurred in captive colonies of eastern bearded dragons. The only premonitory sign reported in the first case was haemorrhagic diarrhoea (Australian Registry of Wildlife Health [ARWH] case report 2756/1). The second case involved hatchlings from the same clutch that presented with severe dehydration and watery faeces and died soon after (ARWH case reports 5316/1, 5322/1). Another unrelated lizard was submitted that died after presenting with abdominal distension (ARWH case number 5858/1).

Diagnosis

Diagnosis is by signalment, histology and PCR of cloacal swabs.

Clinical pathology

Clinical pathology of adenoviral infection has not been described.

Pathology

Gross pathology is often unremarkable. The liver may be mottled, pale or reddish tan in colour, and friable. Histologically, acute diffuse hepatic necrosis is usually present (ARWH case reports 5316/1, 5858/1). Hepatocytes and cholangiolar cells may contain large amphophilic intranuclear inclusion bodies (ARWH case report 5316/1). Intranuclear inclusion bodies may also be found within the epithelial cells of the gastrointestinal tract, kidneys and exocrine pancreas (ARWH case report 5316/1) (Ladds 2009; Moormann et al. 2009; Doneley et al. 2014).

Differential diagnosis

- Dehydration
- Weakness secondary to suboptimal husbandry
- Hypocalcaemia
- Concurrent dependovirus and coccidial infection have been observed in neonatal bearded dragons infected with adenovirus (Jacobson et al. 1996; Kim et al. 2002).

Laboratory diagnostic specimens

Procedures for specimen collection should follow those presented by Rose (2007). A full range of tissue samples should be taken. Neonates and hatchlings may be submitted whole (coelomic cavity opened) in 10% neutral buffered formalin for histopathological examination. Cloacal swabs can be submitted for PCR testing on live animals.

Laboratory procedures

Laboratory testing includes histopathology and electron microscopy. Agamid adenovirus 1 and pan-adenovirus PCRs are available through Tim Hyndman (t.hyndman@murdoch.edu.au) at the School of Veterinary and Life Sciences, Murdoch University, WA, Australia.

Treatment

None.

Prevention and control

Affected animals should be culled from breeding populations. In contact dragons should be tested by cloacal PCR (Hyndman and Shilton 2016). All new lizards should be quarantined.

Adenoviruses are resistant to inactivation when outside a host and can remain infectious for long periods in soil, substrate, water and contaminated faeces. They can be inactivated by treatment for more than one hour with formalin, aldehydes or iodophors (Doneley et al. 2014).

Surveillance and management

Wildlife disease surveillance in Australia is coordinated by the Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia's States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. Please contact admin@wildlifehealthaustralia.com.au.

There is no targeted surveillance program AgAdv-1 and reptile adenoviruses are not on the OIE wildlife disease list.

Statistics

There are a small number of cases of AgAdv-1 infection in free living lizards in the National Wildlife Health Surveillance (eWHIS) database. In a small survey of free-ranging Australian bearded dragons in 2014-2017, agamid adenovirus-1 was detected by PCR in an eastern bearded dragon from south-east Queensland and a number of inland bearded dragons from western New South Wales. In addition, *Lizard atadenovirus A* (formerly helodermatid adenovirus 2) was detected by PCR in one of the inland bearded dragons from western NSW. All dragons in this survey were apparently well. Adenoviruses were not detected from very small numbers of inland bearded dragons from central Australia and western bearded dragons from Western Australia.

Research

Adenoviral infections in bearded dragons are yet to be fully characterised in Australian outbreaks. Surveys to determine the prevalence of adenovirus in populations of free living reptiles in Australia are required.

Human health implications

None.

Conclusions

Agamid adenovirus 1 is a common pathogen of neonate captive bearded dragons in the USA. Its presence has been detected in captive collections in Australia. Given the popularity of the *Pogona* genus as a pet species it is important to monitor the presence of this virus both in captive and free- living populations.

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To provide feedback on this fact sheet

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be very grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for the network (or update current sheets). A small amount of funding is available to facilitate this.

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