Neurological syndrome in “black and white” birds in Australia

Fact sheet

Introductory statement

A syndrome of unknown aetiology has been reported in “black and white” birds (mainly in Australian magpies, and pied currawongs) on the east coast of Australia, primarily in the Sydney region. Birds present with signs of paralysis and paresis and, in most cases, death follows within 24 hours of presentation. Outbreaks of the syndrome occurred in 2003 and in 2005-2006, with as many as several hundred birds reportedly affected. Actual numbers may have been significantly higher. Small numbers of cases with a similar presentation continue to be reported on a sporadic basis, including a cluster of presentations in magpies and Australian ravens on the South Coast of NSW in 2015.

Aetiology

The syndrome is thought to have a viral or parasitic aetiology, but no agent has been able to be grown in culture from swabs and tissue samples of affected birds (Australian Registry of Wildlife Health 2014).

Natural hosts

The syndrome is primarily seen in:

- Australian magpies *Gymnorhina tibicen* (about 70% of reported cases in 2005-2006 outbreak)
- Pied currawongs *Strepera versicolour* (about 15% of reported cases in 2005-2006 outbreak)
- Australian ravens *Corvus coronoides*
- Small numbers of other related species; common koel *Eudynamys scolopacea* and magpie lark *Gallina cyanoleuca* also fit the case definition (see below) at the time of the 2005-2006 outbreak.

World distribution

There are no known reports of a similar syndrome overseas.
Occurrence in Australia

The syndrome has only been reported on the east coast of Australia, primarily in and around the Sydney region. Outbreaks were reported in 2003 and 2005-2006. Sporadic cases meeting the case definition continue to occur in the greater Sydney region and adjacent areas. A cluster of cases of similar presentation was reported in magpies and a small number of Australian ravens on the South Coast of NSW in May 2015, from a very specific location in the suburb of Oak Flats (Jarratt and Rose 2016). A small number of reported cases from Healesville, Victoria and Central Queensland also meet the case definition.

Epidemiology

In the 2005-2006 outbreak, reported cases appeared to follow a temporal and spatial spread consistent with an infectious agent. Reported cases progressed spatially in a south and westerly direction from the Central Coast of NSW to various regions of Sydney. Cases were more frequently reported from coastal suburbs than inland suburbs. The affected area within the Central Coast was bound by Mannering Park (north), Ettalong/Hawkesbury River (south), Bateau Bay/coast (east), Central Mangrove/Somersby (west). Within Sydney, the affected area was bound by Matraville/Kingsford (south), Eastern Suburbs (east), northern beaches (north) and Blue Mountains (west). No suspicious deaths were reported west of the Great Dividing Range.

The number of cases reported weekly during the 2005-2006 outbreak followed a typical epidemic curve, from December 2005 when recording began. It is unclear whether the observed ‘peak’ in case numbers represented a true peak in clinical cases or if it was reflective of a change in reporting rate (mainly from wildlife carers) due to increased awareness of the outbreak.

Over a five-week period during February and March 2006, a large number of wild bird deaths in Australian magpies and pied currawongs were reported in the Sydney and Central Coast areas of eastern NSW. Cases were first reported in December 2005, however anecdotal reports suggest cases may have occurred from September 2005. From early December 2005, around three to six reports were received each day until the last week of April 2006. The reporting rate then dropped to about three reports each week until the middle of May 2006.

Male and female, adult and sub adult birds were all represented. More male birds were necropsied than females. Deaths typically occurred in clusters (from one to 15 birds; most clusters two to six birds). Only Australian magpies, pied currawongs, Australian ravens, magpie larks and possibly common koels showed signs consistent with the case definition. It is likely that the deaths observed in the other species were unrelated to the syndrome seen in the “black and white” birds.

Deaths were also reported during this period in:

- Kingfishers (*Todiramphus* spp.)
- Laughing kookaburra (*Dacelo novaeguineae*)
- Magpie lark (*Gallina cyanoleuca*)
- Crested pigeon (*Ocyphaps lophotes*)
- Silver gull (*Larus novaehollandiae*)
- Australian raven (*Corvus* spp.)
- Channel-billed cuckoo (*Scythrops novaehollandiae*)
- Common koel (*Eudynamys scolopacea*).
There were isolated, anecdotal reports of bird deaths with similar presenting signs from other regions of NSW including Ballina, Lismore, Coffs Harbour, the Illawarra region, Batemans Bay, Young and Scone, however none of these individuals met the case definition for the syndrome. Only reports from Healesville, Victoria (Australian ravens, n=3) and Central Queensland (common koels, n=2) met the case definition.

A similar outbreak is believed to have occurred in July and August 2003 with the first cases reported at Budgewoi, NSW. Cases were subsequently reported from the Central Coast to the Northern Beaches of Sydney over the following 2-3 week period.

The apparent outbreak in Oak Flats in May 2015 affected at least 17 birds, all found in or around one tree, over the course of two days (Jarratt and Rose 2016).

A suggested case definition for the syndrome is:

“Birds of the species Australian magpie (Gymnorhina tibicen), pied currawong (Strepera versicolour) or Australian raven (Corvus coronoides) from the coastal areas of NSW (and possibly other regions) with neurological signs (inability to fly, paresis, retaining mental alertness) progressing rapidly to death and with histopathological findings of non-suppurative encephalitis.”

**Clinical signs**

Birds are found dead (alone or in groups), or alive with neurological signs including severe paresis. Affected birds lack a righting reflex, but have normal cloacal tone, peripheral light reflexes and withdrawal reflexes, and are otherwise bright and alert (Figure 1). Many birds can flap their wings and stand for brief periods, or move about awkwardly using their legs when stimulated. Some birds are dyspnoeic, showing open-mouth breathing, tail bobbing and tachypnoea, and a small number have diarrhoea.

![Figure 1. An Australian magpie unable to right itself (left). The second bird (right) had neurological signs and was unable to raise its head (Images courtesy of Australian Registry of Wildlife Health).](image)

Clinical progression appears to progress through the following steps:

- Inability to fly
- Inability to stand (paresis, ventral recumbency), but maintain alertness
- Acute respiratory problems
- Death.

Most cases progress to death within 12 to 24 hours, once identified, although there some cases have been reported to survive 10 days or more and some cases reportedly recover after intensive treatment. Four cases
in the 2015 Oak Flats event were treated with supportive care, with rapid improvement in clinical signs, being released to the wild 7 days after rehabilitation began (Jarratt and Rose 2016).

**Diagnosis**

Clinical signs are supportive of the diagnosis, as are the characteristic histopathological changes. As there is no confirmed aetiology, diagnosis cannot currently be confirmed. We are not aware of any reports of clinical pathology changes associated with this syndrome.

**Pathology**

**Gross pathology:** Most birds are dehydrated, in either slightly poor or normal body condition, with empty gastrointestinal tracts. Some birds show gross evidence of epicardial and gastrointestinal tract haemorrhage. A smaller number of birds show evidence of hydropericardium.

**Histological changes** vary, but most commonly consist of multisystemic perivascular inflammation, particularly within the coelomic membranes, heart and skeletal muscle. Non-suppurative encephalitis is a feature of some cases, especially in corvids, where the lesions can be very similar to those seen in West Nile virus infection.

Details of histological findings in a typical case are available in the Australian Registry of Wildlife Health Case Blog TARZ-9586.1 ([http://arwh.org/node/188](http://arwh.org/node/188)) (Australian Registry of Wildlife Health 2014).

**Differential diagnoses**

Laboratory investigation has ruled out the following diseases and intoxications as being involved in the syndrome:

- Avian influenza
- Avian paramyxovirus
- West Nile virus and/or Kunjin virus
- Murray Valley encephalitis
- Japanese encephalitis
- Other flaviviruses
- Enteroviruses
- Organophosphate, carbamate, pyrethroid, neonicotinoid or organochlorine intoxication.

**Laboratory diagnostic specimens**

Representative samples of tissues should be submitted for histopathological investigation, including heart, brain, liver, spleen, bursa of Fabricius, thymus and intestine. Duplicate tissue samples should be frozen for potential viral investigation.

**Laboratory procedures**

Laboratory investigation includes histopathological investigation and exclusion of other disease and intoxications (listed under Differential diagnoses). Additional attempts to determine an aetiological agent may include viral culture, pan-family viral PCR and other molecular techniques.
Prevention and control

As there is no confirmed aetiologicaal agent and no information about potential transmission pathways, disease prevention and control is not possible at this stage. The epidemiological curve suggests an infectious agent.

Surveillance and management

Wildlife disease surveillance in Australia is coordinated by 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. Cases of “black and white bird neurological disease” are reported in eWHIS.

Treatment

Most cases progress to death within 12 to 24 hours despite attempts at treatment. A small number of cases may recover after intensive care including the use of thiamine, activated charcoal and anti-inflammatory doses of corticosteroids.

Research

Further work is recommended in the following areas:

- Aetiology of the syndrome
- Basic epidemiology, including risk factors, causation and characteristics of the outbreaks
- Risk assessment, and if indicated, mitigation measures for other wildlife, human and domestic animal health.

Human health implications

Due to the unknown aetiology, appropriate biosecurity precautions should be adopted when treatment or post mortem investigation of affected birds is undertaken. The syndrome does not appear to be associated with human health risk, however, until an aetiology can be determined, this cannot be ruled out.

Conclusions

Further work is required to determine the aetiology and improve understanding of the risk factors contributing to this syndrome. Suspect cases should be reported to your state or territory WHA Coordinator.

References and other information


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To provide feedback on this fact sheet

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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