

Malicious Phosphamidon Poisoning in Zoo Birds

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Birds are one of the diverse and evolutionary successful groups and occur in large numbers in the tropics. The threats leading to the decline in their worldwide population are many and varied and it is a matter of serious concern (BLI, 2008). Pesticide poisoning is known to have killed birds and is difficult to estimate accurately (Mitra et al., 2011). Phosphomidon (Parathion : 2-chloro-2-diethylcarbamoyl-1-methylvinyl dimethyl phosphate), an organophosphorous insecticide was widely used in agriculture, industry, gardening and veterinary medicine. It is capable of disrupting the avian nervous system by inhibiting cholinesterase activity (Ludke et al., 1975; Hill and Fleming, 1982; Quick, 1982). Phosphamidon was the most frequently identified cause of poisoning for 23 mortality events in Korea (Kwon et al., 2004). Carbofuran, phosphamidon, fenthion and fanphur were responsible for 11 of the mortalities in red tailed hawks collected from 1987 through 1992 and were detected based on brain cholinestrace activity (Franson et al., 1996). Farmers intentionally killed 65,000 birds with Parathion (Carson, 1962). In New York State, Red-winged Blackbirds (*Agelaius phoeniceus*) and Common Grackles (*Quiscalus quiscula*) were also killed by farmers with Parathion (Stone, 1980). This report describes malicious phosphomidon poisoning in zoo birds in view of its rare occurrence in an aviary.

CASE REPORT

The Arignar Anna Zoological Park in Chennai housed 480 birds namely Spot-billed Pelicans (*Pelecanus philippensis*), Little Egrets (*Egretta garzetta*), Painted Storks (*Myceteria leucocephala*), Eurasian Spoon bills (*Platalea leucorodia*), Black-headed Ibis (*Threskiornis melanocephalus*) and Black-crowned Night Herons (*Nycticorax nycticorax*) in a closed aviary. As part of their daily routine, the birds were fed fish in the afternoon. Within half an hour of consuming the fish, the birds exhibited neurological signs and died.

Clinical signs included dullness, drooling (Fig.1), regurgitation, inability to fly, diarrhoea, ataxia, hock-

sitting (Fig. 2), prostration (Fig. 3), torticollis and death. Thirty three birds died within three hours. All attempts to treat affected birds failed.

Thirty three carcasses (Fig. 4) were necropsied: one spot-billed pelican, a painted stork, five Eurasian spoon bills, eight little egrets, eight black-headed ibis, and 10 black-crowned night herons. External examination revealed that the birds were in good nutritional condition. Some had torticollis (Fig. 5), fish within the oral cavity (Fig. 6) and oesophagus (Fig.7). Fish (whole to semi pulpy) were found impacted up to the gizzard (Fig. 8), accompanied by a foul odour. The small intestines were empty, with bloody liquid contents mixed with catarrh in a few birds. Some birds had multifocal areas of petechiae and congestion of the small intestine (Fig. 9). Most of the other organs namely liver, kidney and brain showed mild congestion. However, the lungs showed severe congestion and multifocal haemorrhages (Fig. 10). Heart blood was collected for bacterial culture. Cloacal swabs and samples of spleen, intestine and brain were collected for Newcastle disease virus virology. Intestinal contents were collected for parasitological examination. Heart blood smears and impression smears from the liver and lungs were collected and stained with Leishman - Giemsa. Materials collected for bacteriological and virological examination were negative. Parasitological examination revealed a few *Ascarid* spp. eggs. Impression smears from liver and lungs were negative for bipolars. Left over fish and fish recovered from the gizzard of carcasses were frozen and forwarded to the toxicological laboratory of Salim Ali Centre for Ornithology and Natural History, Coimbatore. The analysis reported phosphamidon in high levels. The analysis was done by QuEChERS (Quick, Easy, Cheap, Effective, Rugged and Safe) multi residue extraction method with suitable solvents and reagents. Representative organs such as liver, lungs, intestine, kidneys, spleen and brain were collected in 10% formalin. Histopathological sections of 5µm were prepared on paraffin embedded tissues and stained with haematoxylin and eosin (H&E).

Histopathological examination of proventriculus, gizzard, liver, kidney, intestine, lungs (Fig.11), testis and brain revealed mild to diffuse congestion and haemorrhage.

DISCUSSION

The Anna Zoological Aviary lost 33 (6.9 %) birds within a period of three hours after being fed fish. In this report, clinical neurological signs with fish in impacted gizzards, multifocal areas of petechiae and congestion of small intestine were evident. Proventriculus, gizzard, liver, kidney, testis and brain showed mild congestion, the lungs showed severe congestion and multifocal haemorrhages. Histopathological examination of different organs revealed mild to diffuse congestion and haemorrhage. These observations are in accordance with an earlier report of similar neurological signs with no specific gross or histopathological changes except for the presence of ingested grains in the upper digestive tract of water fowls and cranes deliberately poisoned with phosphamidon (Kwon et al., 2004). Ingested fish removed from the gizzard were positive for high levels of phosphamidon. Quantification of phosphamidon was not performed. The acute oral LD₅₀ for mallard ducks and pigeons have been reported as 3.81 mg/kg BW and 2-3 mg/kg BW respectively (WHO, 2007). There was no literature for the acute LD₅₀ for the birds necropsied in this study.

Birds in the aviary died as a result of acute phosphamidon poisoning. Organophosphate insecticides are more acutely toxic to birds than organochlorine compounds and many bird mortalities have been reported (Stone, 1980; Fleming, 1982). Phosphamidon was readily available, cheap and toxic and hence it was used intentionally to poison the birds mixed with fish. This was in agreement with a previous report (Kwon et al., 2004) where phosphamidon was preferred by people who poisoned wild

birds. Zoo authorities on further enquiries found out that rivalry among fish vendors resulted in the malicious poisoning of birds. This was intentionally done by the fish vendor who was previously requested not to supply fish because of poor quality. Phosphamidon poisoning in waterfowls of Korea was reported due to its use in pharmaceutical treatment (Kwon et al., 2004). Most of the incidents were due to deliberate poisoning with rice seed laced with phosphamidon. Phosphamidon is readily absorbed through the gastrointestinal tract and hence the birds in this report quickly developed clinical signs and died within three hours. Death was mainly due to tissue anoxia (WHO, 2007).

In this report phosphamidon was identified as the cause of the mortality. The clinical signs that followed immediately after consumption of fish helped in suspecting an acute toxicity. Immediate removal of the remaining fish prevented further mortality.

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Figure 1: Black-crowned Night Heron - ptyalism



Figure 2: Painted Stork - hock-sitting



Figure 3: Black-crowned Night Heron - prostrate



Figure 4: Thirty three dead birds



Figure 5: Black-headed Ibis - torticollis



Figure 6: Black-crowned Night Heron - fish in oral cavity



Figure 7: Black-headed Ibis - oesophageal impaction



Figure 8: Spot-billed Pelican - Gizzard impaction



Figure 9: Eurasian Spoon bill intestine - multifocal areas of congestion, petechiae and catarrah

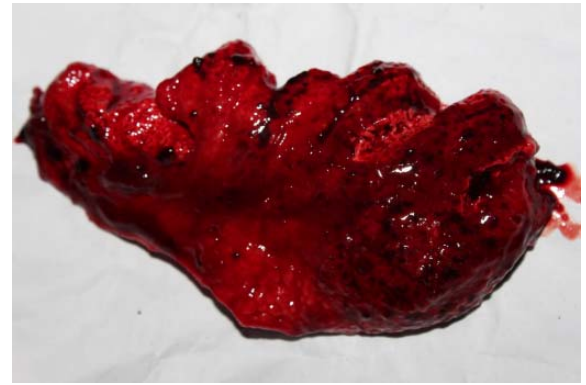


Figure 10: Spot-billed Pelican lung - multifocal areas of congestion and haemorrhage

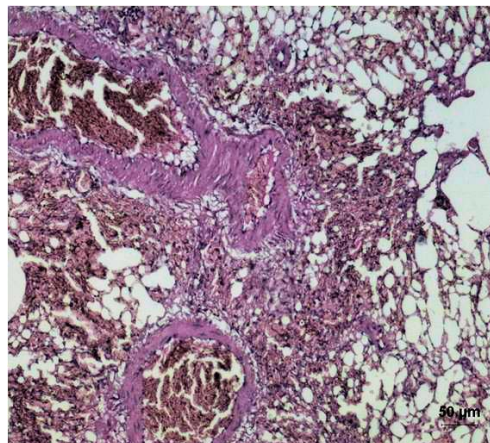


Figure11: H&E. Spot-billed Pelican lung - pulmonary congestion and haemorrhage

