Discovery, The Student Journal of Dale Bumpers College of Agricultural, Food and Life Sciences

Volume 3 Article 10

Fall 2002

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ABSTRACT

Two cases of organophosphate toxicity were diagnosed at the University of Arkansas Poultry Science Department poultry research farm in the Spring of 2002. In both cases the birds were being treated with the organophosphate RaVap® for Northern Fowl Mites (*Ornithonyssus sylvarium*) infestations. A total of 61 birds died and 13 were treated successfully with atropine sulphate.

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INTRODUCTION

Organophosphate compounds are commonly used as contact insecticides and acaracides for many animals and plants. The major classes of organophosphates are phosphates, phosphorothionates, phosphorothioloth-ionates, phosphorothiolates, phosphoramidates, pyrophosphates, phosphonates, and phosphoramides (Hatch 1978).

Since these chemicals are widely used, they may be a source of animal poisoning in a variety of ways. Feeds may be contaminated at the processing plant or during shipping, storage, or handling on the farm. Feed crops may be dusted with insecticide, or the harvested crop may become accidentally contaminated. Animals may be sprayed or dusted with too much insecticide and may become overdosed. Also, water sources may be contaminated by spillage or by rinsing out spray tanks and hoses. Occasionally, the source of contamination cannot be determined (Hatch, 1978).

In most animals, acute toxicosis from organophosphates is due to an irreversible inhibition of acetylcholinesterase (ACh-E) wherever acetylcholine (ACh) functions as a transmitter (Reigart and Roberts, 1999. Fraser and Mays, 1986). The result of ACh-E inhibition is accumulation of ACh in neuromuscular junctions; parasympathetic postganglionic terminals in smooth muscle, cardiac muscle, and glands; in all autonomic ganglia; and in cholinergic synapses within the central nervous system. This causes over-stimulation of the cholinergic receptors by ACh. If the overstimulation is intense enough, the ACh receptors may become blocked. However, complete blockage of all cholinergic receptors is usually not produced, and early clinical signs reflect hyperfunction of these receptors. Lethal amounts of organophosphates cause death from a number of effects of nicotinic, muscarinic, and central cholinergic receptor over-stimulation and/or paralysis. The animal eventually dies of asphyxia (Hatch, 1978).

If the exposure is not too severe, then the animal may not die. In fact animals have been known to spontaneously recover from organophosphate poisoning. ACh-E is usually not 100% blocked since the enzyme is constantly being produced by neurons. Even though the animal can recover, it may still take several days or weeks for an animal to recover completely from a sublethal dosage (Hatch, 1978).

Female chickens with organophosphate toxicity show significant alterations in the brain, spinal cord and peripheral nerves. Axons in the brain stem, ventral and lateral tracts of the spinal cord, in gray matter of the spinal cord, and in the sciatic nerve become swollen and

MEET THE STUDENT-AUTHOR

Robert Hubbard

I graduated from Harrison High School in 1999 and began college at the University of Arkansas the following fall semester as an undecided major. The next summer I became aware of the Poultry Science Department and decided to look into becoming a poultry science major. I quickly decided to begin pursuing a degree in poultry science. Since then I have been introduced to different types of research in the poultry field and have had opportunities to do some research of my own. I am now a senior and plan to graduate in May of 2003 with a B.S.—in poultry science with a minor in music. I am an active member in the Razorbacks for Christ and the Poultry Science Club.

I chose this project based on my fascination with diseases and microorganisms. I thank Dr. Wayne Kuenzel for introducing me to research and Dr. F. Dustan Clark for his guidance throughout this project.

fragmented with the heaviest damage occurring in the spinal cord. Cell bodies in these areas also sustain significant damage. Organophosphate toxicity also causes an effect known as organophosphorus-induced delayed neurotoxicity (OPIDN), which is characterized as a "dying back" of axons. The degeneration of the myelin and axonal portions of the long axons in the spinal cord, peripheral nerves, and medulla usually accompany OPIDN. Syndromes causing nerve damage other than OPIDN have also been known to occur (Carrington et al., 1988).

During a study on the effects of massive oral dosages of Rabon®, a popular organophosphate insecticide, six groups of chicken hens were given dosages of Rabon® daily. Hens given more than 188 mg/kg of Rabon® became inactive and lethargic by the third day of treatment. Birds given higher amounts of 752 mg/kg and 1504 mg/kg had a decrease in weight and food intake, and developed loose and sometimes bloody droppings. Birds given 1504 mg/kg were unable to stand, had whitish dry combs, and sat trembling with their heads down and eyes closed. All birds given 1504 mg/kg died by the seventh day of treatment and sixty percent of the birds given 752 mg/kg died by the fourteenth day (Yadava et al, 1970).

In an experiment using Cyanophenphos (0-ethyl-0-cyanophenyl phenyl phosphonothionate), which causes delayed neurotoxicity similar to that of neurotoxic organophosphorus compounds, chicken hens with high doses (80-540 mg/kg) became paralyzed, deteriorated rapidly in body weight, and eventually died of ataxia. It was observed that hens receiving higher doses became fatigued and unwilling to walk. Clumsiness and unsteadiness of gait followed with muscle tone continuing to diminish and flaccidity progressing within 12-24 days. Eventually complete flaccid paralysis developed and the birds showed no sign of recovery (El-Sabae et al, 1980).

MATERIALS AND METHODS

Our article includes two case reports describing instances of organophosphate toxicity in chickens associated with the treatment of Northern Fowl Mites (*Ornithonyssus sylvarium*) with the product RaVap®. Clinical symptoms, lesions, associated mortality, and therapy are described.

CASE REPORTS

Case 1. In March 2002 approximately 500 birds housed at the University of Arkansas Center of Excellence for Poultry Science research farm, Fayetteville, were treated with the organophosphate. RaVap® (tetrachlorvinphos 23%, dichlorvos 5.7%. Boehringer Ingelheim Vetmedica, Inc. St. Joseph, MO.) for Northern fowl mites. The birds were of various genetic crosses and purebred standard poultry used to study poultry genetics. The birds were housed in floor pens (approximately 1.53 meters by 3.84 meters) with a total of 28 pens in the house. The birds were fed a standard poultry ration with the younger birds on a developer ration. All birds were watered from a standard bell type (Plasson waterer) with one waterer per pen. The birds had been diagnosed earlier with Northern fowl mites. a poultry parasite which can cause anemia, feather loss, and skin damage. The treatment for the mites consisted of RaVap® solution applied to each bird in the house. The poultry farm uses a rotating chemical external parasite control program consisting of RaVap®, Sevin® 80% WP, and a 10% permethrin compound. The schedule was such that RaVap® was the compound to be used at this time. The RaVap® solution was prepared according to label instructions by mixing 1/2 gallon of the product in 25 gallons of water. This solution is a 0.6% solution. The birds were caught and submerged (up to the head) in the RaVap® solution. The house was kept warm (approximately 27-30 degrees Centigrade). Standard ventilation was used in the house during dipping of the birds. The birds were treated on a Friday morning. Clinical symptoms appeared within 4-6 hours in a few birds being described as listless. By Monday morning a total of 56 birds were dead and another 7 birds had varying degrees of clinical symptoms and lesions. Ataxia, depression, anorexia, and closed eyes were the most common symptoms; two of the seven birds were almost comatose. It was noted that of the 56 birds that died over the weekend; 27 died Friday night. Treatment consisted of daily injections of 1/120 grain of atropine sulphate (Amtech, Phoenix Scientific Inc St. Joseph, MO) subcutaneously. A dosage of 0.5-1.0 ml was used and body weight was estimated. The dosage used corresponds to a dosage of 1 ml per 7.5 pounds of body weight. Injections were given for 3 days and birds were examined for a total of 5 days. Affected birds recovered in 3 days with the two most severely affected birds recovering in 5 days. The most severe mortality was seen in the purebred Australorp (three males) and Australorp-Smyth line crosses (27 birds). The three Australorp males lost represented 50% of the Australorp males treated for mites.

Case 2. In May 2002 approximately 288 female chickens and 96 male chickens housed at the Center of Excellence for Poultry Science research farm, Fayetteville, were treated with the organophosphate Ravap®. for Northern Fowl Mites (Ornithonyssus sylvarium) infestation. These chickens were from the Ascites

and random-bred genetic lines maintained for genetic studies. The female chickens were housed in standard-size Leghorn wire cages and the males were housed in broiler breeder male wire cages. Nipple-type drinkers were used in the male cages and cup-type drinkers were used in the female cages. The birds were fed standard breeder ration. The RaVap® solution that had been used was prepared according to label instructions by mixing 1/2 gallon of the product in 25 gallons of water producing a 0.6% solution. The birds were treated for mites by spraying them around the vent area until that area was thoroughly wet. Overnight four of the birds sprayed died and another seven developed clinical symptoms. The symptoms were similar to those seen in the other chickens in that the affected birds had varying degrees of depression, closed eyes, ataxia, and appetite loss. However, some of the affected chickens had a reddish discoloration of the shanks of the legs. The treatment of these surviving birds consisted of daily subcutaneous injections of 1/120 grain of atropine sulphate. The dosage was 1.0-1.5 ml and again body weight of the chickens was estimated. The dosage used corresponds to a dosage of 1 ml per 7.5 pounds of body weight. Injections were given for three days and birds were examined for a total of five days. One of the seven affected birds died but the remaining six recovered. The five total birds that died were all males. No females developed clinical symptoms of organophosphate toxicity.

RESULTS AND DISCUSSION

The exact reason for the toxic effects in the birds in these two case reports could not be determined. The Ravap® was mixed in accordance with the label instructions according to personnel involved in treating the chickens with the insecticide. If the product was mixed incorrectly it can be speculated that more birds would have been affected.

The Australorp males and Australorp-Smyth line crosses suffered the highest mortality of all Ravap® treated birds in the first case report. Three of the six purebred Australorp males died from the Ravap® treatment (representing a 50% loss of purebred males treated). This suggests that the Australorp breed of chicken may have a greater sensitivity to the Ravap®. This latent sensitivity to organophosphates may be related to genetics, stress, or other variables. In the practice of veterinary medicine, it is reported that some pets have a skin reaction to flea collars (Fraser and Mays, 1986) and occasionally certain breeds of dogs have other reactions to some types of flea collars. This reaction often is manifest by the dog appearing tired, sluggish, and having periods of vomiting; the dog may recover uneventfully when the collar is removed (Dustan Clark, personal communication).

In the second case report, five male birds treated with Ravap® died and two other males showed symptoms of organophosphate toxicity and then recovered. These five birds were random-bred broiler-breeder males and were the first birds of the males sprayed for mites. It had been speculated earlier that these birds may have received a higher level of Ravap® since they were sprayed first and as such the product may not have been thoroughly mixed. However, it was determined that the Ravap® was mixed correctly and, in fact, the hens in the second case report were sprayed before the males. Therefore, these first five males would in actuality have been birds 289 through 293 sprayed from the spray tank since 288 hens were sprayed prior. Since only males died in the second case report, it is possible that there may be a sex-specific sensitivity to the ingredients in Ravap®.

A total of 61 chickens died in both case reports following either dipping or spraying with Ravap®. In the first case report, 27 of the chickens died the first night and 29 more died over the remainder of the weekend. In the second case report four birds died overnight. A total of 14 chickens in both case reports were treated with atropine sulphate for clinical symptoms consistent with organophosphate toxicity. Atropine sulphate was an effective treatment since only one of the 14 birds treated failed to respond to the atropine sulphate injections. Fig.1 depicts a bird with clinical symptoms of Ravap® toxicity; fig. 2 depicts a chicken that has responded to atropine sulphate therapy. It is possible that more of the chickens could have been saved (especially in case report one) if the adverse reactions had been reported to the Poultry Science Department veterinarian immediately so atropine therapy could have been started sooner.

The exact reasons for the animal's toxicity from the Ravap® may never be determined from these two cases. Since organophosphate products can be toxic it is important to mix the products in accordance with label instructions and use them appropriately. It is also important to report any adverse symptoms in animals after product use to a veterinarian immediately so treatment can be administered.

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Fig. 1. A male chicken exhibiting clinical symptoms of organophosphate (RaVap®) toxicity.

Fig. 2. A recovered male chicken (top left) after atropine sulfate therapy in a pen with another male and two females.