Short Communication

PRELIMINARY OBSERVATIONS ON THE EFFICACY OF AN IODOPHOR IN REDUCING THE MORTALITY IN CHICKENS EXPERIMENTALLY AFFECTED BY THE 'HYDROPERICARDIUM SYNDROME'

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Abbreviations: HPS, hydropericardium syndrome

INTRODUCTION

Hydropericardium syndrome (HPS) is a newly emerging disease affecting broiler chickens; it occurs mainly in 3- to 6-week-old birds and results in up to 60% mortality (Hasan, 1989; Abdul-Aziz and Al-Attar, 1991). The most prominent and consistent lesions in affected birds are the accumulation of excessive amounts of a clear yellowish fluid in the pericardial sac, and enlargement and discoloration of the liver, which may contain haemorrhagic and/or necrotic foci (Khawaja *et al.*, 1988; Anjum *et al.*, 1989; Cheema *et al.*, 1989). The disease could be reproduced in broilers by inoculating a bacteria-free liver homogenate prepared from naturally affected chickens either subcutaneously (Cheema *et al.*, 1989; Chishti *et al.*, 1989; Afzal *et al.*, 1991) or orally (Abdul-Aziz and Hasan, 1995). Adenovirus particles have been visualized in (Cheema *et al.*, 1989) or isolated from (Khawaja *et al.*, 1989) livers of affected chickens. It has been reported that iodophors, when used in the drinking water, were effective in reducing the severity of field outbreaks of HPS (Abdul-Aziz and Al-Attar, 1991).

The purpose of this study was to determine whether the use of iodophor in drinking water is effective in reducing the mortality in chickens experimentally affected by HPS.

MATERIALS AND METHODS

Experimental birds

One-day-old chicks were obtained from a commercial hatchery. They were raised on a litter floor and given tap water and unmedicated commercial broiler ration *ad libitum*.

Preparation of liver homogenate

Livers from chickens naturally affected by HPS were ground with sterile sand and diluted with normal saline to make a 25% (w/v) suspension. The suspension was centrifuged at 3000g for 30 min and the supernatant was removed and used for inoculation.

Experimental design

At 30 days of age, the chickens were divided into three groups of 30 birds each. Each group was housed in a separate room on woodshaving litter. The chickens in group 1 were inoculated orally with 0.2 ml of the liver suspension; those in group 2 were inoculated intramuscularly with 0.1 ml of the liver suspension; the chickens in group 3 were uninoculated controls. After inoculation, each group was further divided into two subgroups (A and B) of 15 birds each. The two subgroups of each group were kept in the same room but were separated from one another by a 1.5-m-high netting partition.

Immediately after intramuscular inoculation, an iodophor (nonylpolyethoxyethanol-iodine complex 18.35%, (equivalent to 1.8% free iodine), phosphoric acid 3% and inert ingredients 78.65%) was added to the drinking water of subgroups 2A and 3A at a concentration of 0.75 ml/L (equivalent to 13.5 ppm free iodine). Twelve hours after oral inoculation, the same concentration of the same iodophor was added to the drinking water of subgroup 1A. No medication was given to the chickens in the other three subgroups (B). The iodophor was given continuously for 8 successive days, during which time the iodophor-containing water was the only source of drinking water available to the chickens in the A subgroups. All the birds were observed closely for clinical signs and mortality. Dead birds were necropsied and examined for gross lesions. Eight days after inoculation, the surviving chickens in groups 1 and 2 and all those in group 3 were killed, necropsied and examined for gross lesions. The results from subgroups 1A and 1B were compared by the χ^2 test.

RESULTS

Chickens in both the subgroups of group 3 remained healthy throughout the 8-day period of observation. Deaths occurred in all the other subgroups, but the lowest mortality was in subgroup 1A (orally inoculated, iodophor-treated subgroup) (Table I).

TABLE I

Subgroup	Treatment	Number of dead birds
1A	Orally inoculated, ^a iodophor-treated ^b	2/15 (13.3%)
1 B	Orally inoculated, untreated	8/15 (53.3%)
2A	Intramuscularly inoculated, iodophor-treated	13/15 (86.6%)
2B	Intramuscularly inoculated, untreated	14/15 (93.3%)
3A	Uninoculated, iodophor-treated	0/15
3B	Uninoculated untreated	0/15

The effect of an iodophor in the drinking water on the mortality rate in chickens experimentally affected by the hydropericardium syndrome

^aThe inoculum was a liver extract prepared from chickens naturally affected by the hydropericardium syndrome. The oral dose was 0.2 ml and the intramuscular dose was 0.1 ml of the supernatant from a 25% (w/v) homogenate

^bThe iodophor was given in the drinking water at a concentration equivalent to 13.5 ppm free iodine

The probability that the results in subgroups 1A and 1B did not differ was <0.05 ($\chi^2 = 5.4$).

Gross lesions were found in all the birds that died during the 8 days of the experiment; the most consistent and prominent lesions being hydropericardium and an enlarged discoloured liver, which in many cases was mottled with foci or areas of necrosis. There were no gross lesions in the surviving chickens in the inoculated groups, or in any of those in the uninoculated control group.

DISCUSSION

The results of this study strongly suggest that the iodophor, when used in the drinking water at a concentration equivalent to 13.5 ppm free iodine, was effective in reducing the mortality in the orally inoculated chickens but not in the intramuscularly inoculated ones. These results support the previous finding that iodophors given in the drinking water reduced the severity of field outbreaks of HPS (Abdul-Aziz and Al-

Attar, 1991). It seems that the causative agent of HPS is sensitive to the iodine present in the iodophor. Avian adenoviruses, one of which has been incriminated in this condition, lose their viability when treated with iodine (McFerren and Adair, 1977). The effectiveness of the iodophor in the orally inoculated group suggests that the iodine probably attacks the causative agent of HPS in the digestive tract. It has been assumed that, under field conditions, infection occurs by oral route and that infected birds shed the causative agent, which is spread laterally to other birds in the flock (Abdul-Aziz and Hasan, 1995). Contact transmission has been shown experimentally to occur in HPS (Abdul-Aziz and Hasan, 1995) and, therefore, it is possible that, in field infections, the iodophors may slow the spread of the virus in drinking water and hence reduce the severity of an outbreak.

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