FURAZOLIDONE AND CYCLOPIAZONIC ACID TOXICITY IN THE CHICKEN*

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Cyclopiazonic acid (CPA) is a mycotoxin produced by Penicillium and Aspergillus sp. of the fungi. CPA co-exists frequently with other common mycotoxins such as aflatoxin (AF), Ochratoxin (OA) and T2 toxin in the poultry feed. (Widiastuti et al., 1988). The combined toxicity was found to be additive in nature (Smith et al., 1992). Occurrence of CPA upto 20 ppm in the compounded feed and groundnut cake has been reported under field conditions in India (Balachandran and Parthasarathy, 1996). This study was designed to find the interaction between CPA and Furazolidone (FZ) a common feed additive in the poultry feed.

Materials and Methods

Day old single comb White Leghorn cockerels were used for the study. CPA was produced by culturing Penicillium griseofulvum NRRL 3523 on the rice and the toxin was estimated (Rathinavel and Shanmugasundaram, 1984). Commercial chick mash obtained locally was tested and confirmed to be free from CPA and AF. Powdered rice containing known amounts of CPA was incorporated into the chick mash to contain 20 and 40 ppm of CPA.

Weighed day old male chicks were randomly allotted to four groups namely A, B, C and D (n=6 per group). From the day 8 onwards chicks were reared in individual cages with continuous feed, water and lighting upto day 35.

Two trials (1 and 2) were conducted between day 8 and day 35. In both the trials Group A (A1 and A2) served as zero control receiving neither toxin nor drug. Group B served as toxin control with B1 receiving 20 ppm and B2 40 ppm of CPA respectively. Groups C and D received FZ at 100 and 400 ppm respectively along with 20 ppm of CPA in trial-1 (C1 and D1) and 40ppm in trial-2 (C2 and D2). Bodyweight gain and feed intake were recorded at weekly intervals for individual birds and feed efficiency was calculated. On day 35 the birds were sacrificed and sera collected. Glucose, cholesterol, urea, uric acid, total proteins and albumin contents of the sera were estimated adopting standard procedures. The carcasses were examined for pathological changes. The results were statistically analyzed by completely randomized block design (Snedecor and Cochran, 1989).

Results and Discussion

Toxin treated birds (B1 and B2) showed dose related reduction in the feed intake and body weight gain compared to

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**Toxicity in the Chicken**

Table 1: Furazolidone (FZ) on growth and serum biochemical values in cockerels fed CPA at 20 ppm (Mean ± SE)

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Body weight Gms**</th>
<th>Feed Intake Gms**</th>
<th>FCR ***</th>
<th>Glucose mg/100 ml**</th>
<th>Cholesterol mg/100 ml**</th>
<th>Total protein Gms/100ml**</th>
<th>Albumin Gms/100ml**</th>
<th>Urea mg/100ml</th>
<th>Uric acid mg/100ml**</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>0 + 0</td>
<td>410.0 ± 7.69*</td>
<td>1115.5 ± 22.0*</td>
<td>2.72</td>
<td>190.42 ± 6.30*</td>
<td>186.45 ± 4.51*</td>
<td>3.06 ± 0.14*</td>
<td>1.42 ± 0.06*</td>
<td>1.65 ± 0.07*</td>
<td>6.37 ± 0.41*</td>
</tr>
<tr>
<td>B1</td>
<td>20 + 0</td>
<td>353.33 ± 9.71*</td>
<td>952.67 ± 23.19*</td>
<td>2.8</td>
<td>222.00 ± 4.90*</td>
<td>212.15 ± 4.15*</td>
<td>2.40 ± 0.11*</td>
<td>1.05 ± 0.05*</td>
<td>1.94 ± 0.13*</td>
<td>7.91 ± 0.25*</td>
</tr>
<tr>
<td>C1</td>
<td>20 + 100</td>
<td>397.67 ± 6.26*</td>
<td>1081.17 ± 18.65*</td>
<td>2.71</td>
<td>207.78 ± 2.14*</td>
<td>199.08 ± 1.73*</td>
<td>2.71 ± 0.09*</td>
<td>1.14 ± 0.07*</td>
<td>1.77 ± 0.07*</td>
<td>6.68 ± 0.21*</td>
</tr>
<tr>
<td>D1</td>
<td>20 + 400</td>
<td>333.17 ± 9.23*</td>
<td>962.33 ± 20.15*</td>
<td>2.98</td>
<td>231.82 ± 3.35*</td>
<td>223.72 ± 3.26*</td>
<td>2.25 ± 0.13*</td>
<td>0.88 ± 0.07*</td>
<td>1.92 ± 0.04*</td>
<td>8.85 ± 0.26*</td>
</tr>
</tbody>
</table>

**Table 2: Furazolidone (FZ) on growth and serum biochemical values in cockerels fed CPA at 40 ppm (Mean ± SE)**

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Body weight Gms**</th>
<th>Feed Intake Gms**</th>
<th>FCR</th>
<th>Glucose mg/100 ml**</th>
<th>Cholesterol mg/100 ml**</th>
<th>Total protein Gms/100ml**</th>
<th>Albumin Gms/100ml**</th>
<th>Urea mg/100ml</th>
<th>Uric acid mg/100ml**</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2</td>
<td>0 + 0</td>
<td>444.67 ± 22.7*</td>
<td>1110.5 ± 26.00*</td>
<td>2.51</td>
<td>188.42 ± 7.21*</td>
<td>185.08 ± 2.42*</td>
<td>3.26 ± 0.09*</td>
<td>1.47 ± 0.05*</td>
<td>1.86 ± 0.09*</td>
<td>5.28 ± 0.26*</td>
</tr>
<tr>
<td>B2</td>
<td>40 + 0</td>
<td>335.33 ± 11.02*</td>
<td>851.83 ± 12.71*</td>
<td>2.57</td>
<td>248.00 ± 4.77*</td>
<td>257.97 ± 4.77*</td>
<td>2.15 ± 0.05*</td>
<td>1.04 ± 0.04*</td>
<td>2.12 ± 0.04*</td>
<td>9.39 ± 0.49*</td>
</tr>
<tr>
<td>C2</td>
<td>40 + 100</td>
<td>384.67 ± 18.53*</td>
<td>976.0 ± 12.93*</td>
<td>2.56</td>
<td>229.92 ± 2.63*</td>
<td>237.08 ± 2.52*</td>
<td>2.26 ± 0.06*</td>
<td>1.08 ± 0.04*</td>
<td>1.99 ± 0.03*</td>
<td>8.31 ± 0.27*</td>
</tr>
<tr>
<td>D2</td>
<td>40 + 400</td>
<td>315.67 ± 7.31*</td>
<td>858.0 ± 8.7*</td>
<td>2.74</td>
<td>253.62 ± 4.21*</td>
<td>269.63 ± 3.36*</td>
<td>1.65 ± 0.06*</td>
<td>0.68 ± 0.04*</td>
<td>2.44 ± 0.04*</td>
<td>11.83 ± 0.31*</td>
</tr>
</tbody>
</table>

**Significant at one percent level**

Figures with common superscripts do not differ significantly

zero control (Table 1): At 20 ppm CPA, 100ppm furazolidone (C1) showed a highly significant increase in body weight gain (P<0.01) compared to the toxin control (Table 1). There was a marginal increase in the feed efficiency. However, at 40 ppm CPA, (C2) the increase was highly significant (P<0.01) only with reference to feed intake (Table 2). All the above parameters showed an insignificant decrease with 400 ppm FZ at both 20 and 40 ppm of CPA, (D1 and D2) compared to toxin controls. However, the reduction in feed efficiency at 20 ppm CPA was highly significant (P<0.01).

CPA is well proven to cause reduction in the feed intake and body weight gain (Balachandran and Parthasarathy, 1998). FZ is reported to produce the same effects at a dose range of 200 to 800 ppm in quails (Arbid et al., 1990). CPA at 20 and 40 ppm (B1 and B2)
caused increase in the serum glucose, cholesterol, urea and uric acid and reduction in total protein and albumin levels (tables 1 and 2).

FZ at 100 ppm reversed the toxic effects of CPA and the reversal was highly significant (P<0.01) with 40 ppm CPA (C2) for glucose and cholesterol (Table 2). At 400 ppm FZ aggravated the toxic effects over and above the toxin control and the effects were highly significant (P<0.01) with 40 ppm CPA (D2).

CPA is known to cause reduction in the plasma protein levels FZ on the other hand is proteolytic in nature in addition to being an inhibitor of protein synthesis (Nafizi and Asasi, 2001).

In the toxin treated birds (Group B) pale liver was observed at 20 ppm CPA (B1) and both kidney and liver were pale at 40 ppm (B2). Much variations in the gross changes of visceral organs could not be observed in group C (100 ppm FZ) compared to the toxin control. Yellowish liver with pale kidney were observed in D1 (20 ppm CPA + 400 ppm FZ), whereas mottling and focal necrosis of the kidney along with yellowish liver were observed in D2 (40 ppm CPA + 400 ppm FZ). The results are in accordance with Giridhar et al., (2000).

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REFERENCES


