

# Hepato-Nephrosis In Chicks

*Feed quality problems due to contamination with moulds and mycotoxins are believed to be the primary cause of serious liver and kidney disease in broiler and layer chicks in Pakistan. — By Dr A.A. Qureshi*

**D**uring the past year Hepato-nephrosis syndrome in young chicks has killed many hundreds of thousands of birds which, along with other diseases in Pakistan, is responsible for a serious increase in mortality of broiler and layer chicks which has risen from 12 to 30% and 6 to 10% respectively. Consequently, the whole industry has been brought to its knees as high mortality and a depressed market has pushed the sector into a state of crisis, never seen during the last 30 years.

Earlier during 1987 a similar situation was created due to hydropericardium syndrome in broilers which caused and is still causing heavy mortality in young chicks in spite of extensive use of an autogenous vaccine prepared from infected liver tissue. Unfortunately, hydropericardium is now also seen in young layer chicks and is believed to be caused by an adenovirus. Recently, a killed oil emulsion vaccine prepared from the DC-94-HCL strain of group-1 adenovirus from Mexico is being used in field trials with variable success in the control of hydropericardium and inclusion body hepatitis.

The incidence of hepato-nephrosis was first seen during August 1996 with a history of sudden deaths without apparent morbidity. Postmortem lesions

consisted of enlargement of the liver with different degrees of discolouration and even haemorrhages. The gall bladder was invariably distended with pale yellowish green bile water contents and a marked congestion of the duodenum. Kidneys also showed extreme swelling and distention with urates. Hepato-nephrosis was seen in both hydro vaccinated and non-vaccinated flocks.

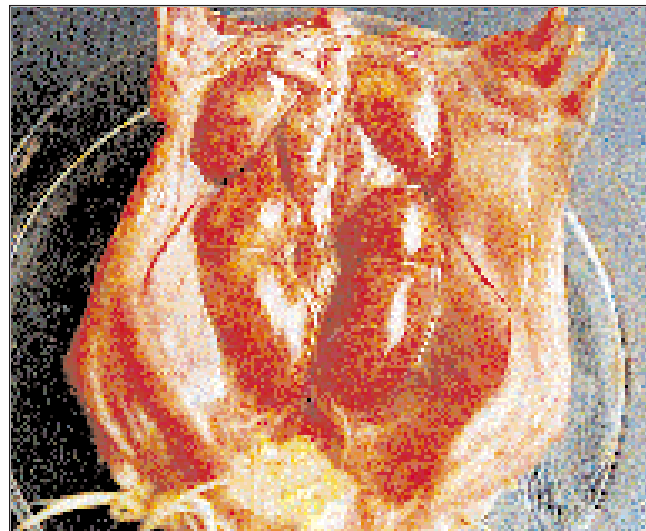
The histopathological examinations of both liver and kidneys showed extensive inclusion bodies in the hepatocytes and the kidney cells.

Attempts to reproduce the disease experimentally in chicks through injection of filter liver homogenate did not succeed nor did chick embryo inoculation show any mortality in 7-10 days post-inoculation. It may be mentioned at this state that the incidence of VV Infectious Bursal Disease is common along with VVVND, during the last two years in the area and its immunosuppression is greatly enhanced.

Since the incidence of hepato-nephrosis was noticed on farms using a particular feed and that change of feed invariably helped to control the mortality rate within 3-5 days, the investigation were diverted to determine the aflatoxin contents of feed. Various feed samples in crumbled form were subjected to ELISA and a high



**Hepato-nephrosis lesions in liver and kidneys.**



**Acute nephrosis in a young broiler chick.**

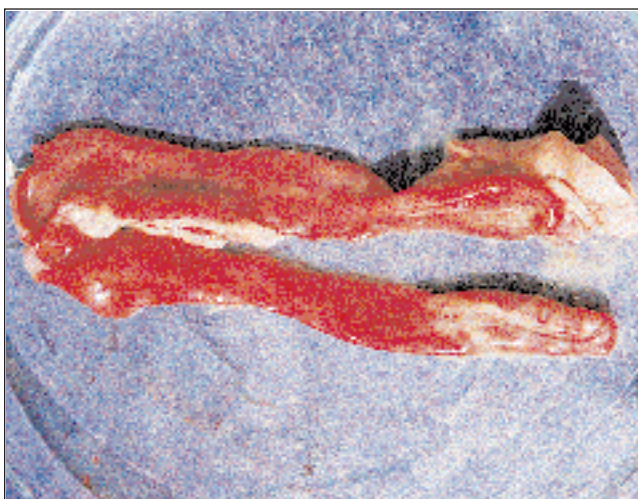
## Hepato-Nephrosis In Chicks



**Enlargement and necrosis lesions of the liver.**



**Discolouration of livers in Hepato-nephrosis.**



**Acute congestion of the duodenum in Hepato-nephrosis.**

level of various aflatoxins were found which apparently caused the lesions of hepato-nephrosis in their target organs and triggered adenovirus intervention.

Various feed samples testing during the period 16.6.96 to 31.12.96 revealed 93% positive for aflatoxins, 59.09% positive for ochratoxin, 34.09% positive for zeralenone and 31% positive for T-2 toxin. More serious was the fact that 54.54% samples contained more than one type of these toxins. However, their maximum, minimum and average levels were as under:

Contents	Maximum	Minimum	Average
<b>Aflatoxin</b>	<b>133 ppb</b>	<b>1 ppb</b>	<b>52.52 ppb</b>
<b>Ochratoxin</b>	<b>97.7 ppb</b>	<b>2 ppb</b>	<b>35.5 ppb</b>
<b>Zeralenone</b>	<b>615.9 ppb</b>	<b>1.6 ppb</b>	<b>153.54 ppb</b>
<b>T-2 Toxin</b>	<b>396.9 ppb</b>	<b>15.8 ppb</b>	<b>150.73 ppb</b>

Inclusion body hepatitis, caused by adenovirus group 1 sero type 8, is said to be responsible for causing inclusion body hepatitis in chicks but in the literature one fails to find any reference for the lesions of nephrosis. However coronavirus IB T-strain has been associated with nephrosis lesions, along with distension of kidney tubules with urates. A somewhat newly recognised Picornavirus agent of AV has also been reported from Japan to cause Infectious Nephritis in chicken with focal lesions in the kidneys cortex. Aflatoxins are known for their immunosuppression and gross lesions in various visceral organs including liver and kidneys. The involvement of aflatoxins in causing hepato-nephrosis and triggering inclusion body hepatitis in this case was fully substantiated as the withdrawal of affected feed greatly improved the birds and mortality started declining within 5-7 days.

Pakistan is situated in a sub-tropical region so its environmental conditions are conducive to aflatoxin production. Since 1973-75 heavy mortality and reduced productivity was often noticed in commercial poultry flocks which showed decreased bodyweight, egg production and egg size including yolk weight. Their gross pathology consisted of pale, enlarged liver and fatty infiltration, regressed bursa, serious infection of pericardium also enlarged spleen and enlarged kidney.

Histopathology of the liver showed periportal fatty infiltration, cell vacuolisation and necrosis, biliary proliferation and increased inflammatory changes. There was also intra mitochondrial inclusion of heart muscle and nuclear segregation of pancreas. Blood examination showed increased blood urea and amino-nitrogen concentration.

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Amongst the various toxins, aflatoxin is known to cause both hepatic and kidney lesions consisting of congestion, enlargement and a firm appearance with a distended gall bladder. Kidneys are swollen, enlarged and congested while the duodenum is distended with catarrhal contents. Similarly ochratoxins also cause pale yellow discolouration and necrotic liver lesions along with nephrotoxicity as the kidneys are enlarged and look pale. Citrinin is also said to cause swollen kidneys and necrosis of liver while Oosporiein too cause nephrotoxicity and high mortality while liver is mottled

having focal necrosis. The gall bladder is distended with a translucent green to yellow bile.

During the investigations in this case, it was noticed that birds receiving more than one type of aflatoxins had mortality increased manyfold. There was also some relief from providing a 2% sugar solution as source of drinking water during the initial stages, besides changing the feed. The problem is still continuing due to a great scarcity of quality feed ingredients. — *Dr A.A. Qureshi, K&N's Poultry Diagnostic & Research Institute, Karachi, Pakistan.* 