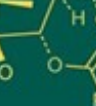
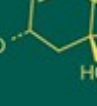
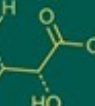
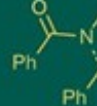
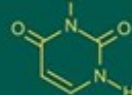


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Pathological manifestations of T2 toxin in commercial poultry

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ABSTRACT

Mycotoxins are important constraint to poultry health and productivity. They cause economic losses to poultry. Mycotoxins are produced as secondary metabolites by several species of filamentous fungi. Aflatoxins, ochratoxin A and trichothecene toxins such as T-2 toxin are the major mycotoxins affecting poultry. This study investigates the presence of T2 toxin in poultry feed and its pathological effects in poultry. This study was conducted at the Department of Pathology, Mamnoon, Warangal. Feed samples and carcass of affected birds from the suspected poultry were collected. Birds were examined for the clinical signs, gross pathology and histopathology. Clinical signs noticed were feed refusal, depression, reduced activity, watery droppings, abnormal feathering and characteristic oral lesions. Gross lesions noticed were oral and beak ulceration, gizzard erosion, pale friable liver, swollen kidneys and atrophy of lymphoid organs. Histologically, epithelial degeneration and necrosis was noticed in alimentary tract, hepatic degeneration, renal tubular injury and lymphoid depletion were prominent. Presence of T-2 toxin in the feed was confirmed by Thin layer chromatography.

Keywords: Mycotoxins, T2-Toxin, Poultry, Signs, Gross lesions, Histopathology

Introduction

Mycotoxins are secondary metabolites produced by several genera of fungi. They contaminate, feed ingredients in compounded poultry feeds. Poultry are particularly susceptible to several mycotoxins. Small quantities of mycotoxin in feed cannot be identified by texture of feed and smell. Their occurrence in feed is of considerable importance because, the chronic exposure results in feed refusal, poor growth, organ damage, immunosuppression and increased susceptibility to infectious diseases (Alkheraie, 2026)^[1]. Trichothecenes such as T-2 toxin produced by *Fusarium* species is also having considerable importance in poultry industry (Meneely *et al.*, 2023)^[2]

T-2 toxin belongs to the type A trichothecene with a tetracyclic sesquiterpene and typical spiro-epoxy ring structure which is the main toxic group (Sokolović *et al.*, 2008)^[3]. The trichothecenes are non-volatile low molecular weight compounds (MW 250–500). T-2 toxin is readily soluble in ethyl acetate, acetone and diethyl ether. The metabolites are remarkably stable to heat and acidic conditions and therefore are not destroyed during normal food processing or digestion (McCormick *et al.*, 2011)^[4] and their presence in feed cause toxicity.

The T-2 toxin inhibits DNA and protein synthesis by reacting with the thiol groups of sulfhydryl enzymes, impair the antibody production and membrane functions, and reduce lymphocyte proliferation. Oxidative damage is the main mechanism of damage in different tissues (Wu *et al.*, 2020 and Song *et al.*, 2023)^[5, 6].

The pathological effects of trichothecenes can be particularly important because the clinical presentation may range from reduced productivity to severe mucosal lesions and mortality. In poultry, T-2 toxin is capable of producing lesions in the oral cavity, alimentary tract, lymphoid organs and haematopoietic tissues. It also effects liver, nervous system, kidneys and feathering (D'Mello *et al.*, 1997; Čonková *et al.*, 2003, Pande *et al.*, 2011)^[7, 8, 9].

There are very few reports on t2 toxin toxicity in the field due to difficulty in the diagnosis and its confirmation in the feed. This study is aimed to describe the pathological manifestations of T2 toxicity in natural field conditions and its confirmation, which collectively provides a more comprehensive assessment of toxin-associated disease.

Materials and Methods

The present study was carried out in the department of pathology, College of Veterinary Science, Warangal, Telangana from January 2026 to September 2026. For poultry health care, birds were brought for the post-mortem examination. The flocks suspected for the T2 toxicosis were observed for clinical signs, gross

pathology and sample were collected for hisopathology in neutral buffered formalin and fixed. These tissues were processed by paraffin embedding techniques and sections were stained with Haemotoxyllin and eosin method according to standard histological procedures. Presence of T2 toxin in the feed was confirmed by the thin layer chromatography.

Results

Poultry were highly sensitive to the T2 toxin. The affected birds were dull and depressed. Reduced feed intake was observed. Few birds were showing the watery droopings. Birds showed caseous plaques on the beak, hard palate, corners of mouth (Fig. 1), hard palate and tongue. Oral lesions observed were erosions and ulcers in oral mucosa (Fig. 2). The results were in confirmation with experimental study carried by Pande *et al* (2011)^[9] who reported caseous plaques to necrotic foci at beak, hard palate, angle of mouth and on tongue in experimental study.

In earlier T-2 toxin studies feed refusal, reduced activity, depression, watery droppings, abnormal feathering and oral lesions as characteristic clinical manifestations (Čonková *et al.*, 2003; D'Mello *et al.*, 1999; Sousa *et al.*, 2020) [8, 7, 10]. The severity was related to the dietary concentration of T-2 toxin. Feed refusal was particularly prominent in birds receiving higher concentrations (Singh *et al.*, 2020)^[11]. Abnormal feathering like sparse, short and disorganized feathers was observed (Hoerr, 2020)^[12]. In laying hens there was decrease in egg production and egg quality (Puvača *et al.*, 2024)^[13]. In a study, T-2 toxin contamination in laying birds is associated with lesions in the buccal cavity, which contributes to reduced feed consumption and weight gain (Dazuk *et al.*, 2020)^[14].

Carcass of effected birds was pale and dehydrated. The caseous to necrotic lesions in the oral cavity, crop and gizzard were observed (Fig. 3). The liver was pale, enlarged and friable. Kidneys were swollen and congested (Fig.4). Mesenteric blood was congested and intestines contained scanty yellowish feed. Mild atrophy of thymus, spleen and bursa was noticed in few birds. These results were in agreement with the previous studies, authors noticed grey- yellow exudate on buccal mucosa, liver was friable, swollen kidneys, congested mesentery and atrophy of lymphoid organs (Pande *et al.*, 2011)^[9].

In the present study T-2 toxin produced prominent epithelial lesions in the upper alimentary tract. The tongue showed loss of papillae and degeneration of the superficial epithelium and inflammatory cell infiltration. The oesophagus, crop showed superficial epithelium degeneration and ulceration. In Proventriculus ulceration and desquamation of glands were observed (Fig.5) In the gizzard shortening of gastric glands and desquamation of epithelium were observed (Fig 6). Duodenal lesions included shortening of villi, necrosis of mucosal epithelium and lumen containing debris with inflammatory cell infiltration (Fig.7)

Hepatic lesions noticed were congestion, degeneration of hepatocytes, focal necrosis, biliary hyperplasia, Kupffer-cell proliferation, inflam-

matory-cell infiltration (Fig.8). Kidneys showed congestion, interstitial haemorrhage, granular degeneration (Fig.9).

The thymus, bursa (Fig.10) showed depletion of lymphocytes from the medullary and cortical regions. The spleen showed depletion of lymphocytes in the white pulp. Caecal tonsils showed depletion of lymphoid cells.

These results are in-line with the previous studies. In earlier studies the crop showed hydropic degeneration, focal necrosis and of the superficial epithelium with fibrin and inflammatory cell infiltration At higher toxin concentrations, microvesicle formation and necrosis of glands in the lamina propria was also observed (Pande *et al.*, 2011)^[9]. In experimental study t2 toxin affected birds showed, necroses in the glandular stomach and intestines. Bursa showed lymphoid depletion, cystic spaces, fibrosis. Spleen revealed lymphoid depletion and recticular cell hyperplasia. Lymphoid cell depletion and lymphocytolysis was observed in thymus. This leads to immunosuppression and predisposes birds to the infectious diseases (Kamalavenkatesh *et al.*, 2005) [15].

T-2 toxin-induced small intestine injury in 1-day-old male broilers by feeding them diets supplemented with T-2 toxin at 1.0, 3.0, and 6.0 mg/kg for 2 weeks, leads to severe degeneration, necrosis, and desquamation of villous epithelial cells, and increased inflammatory cells in the intestinal mucosa (Liu *et al.*, 2023)^[16]. T-2 toxin destroys the intestinal mucosa and intestinal barrier function; and kills intestinal epidermal cells by inducing oxidative stress, apoptosis and inflammatory responses, further it disrupts the intestinal microbiota, causes intestinal flora disorders, affects normal intestinal metabolic function, (Pinton *et al.*, 2012 and Zhang *et al.*, 2024)^[17, 18]

Liver is major detoxification organ hence it is main target of T-2 toxin toxicity. It can cause histological alterations in cell structure, the loss of live function, energy metabolism disorder and bile acids metabolism disorder that involve the activation of oxidative stress, mitochondrial damage, abnormal DNA methylation, autophagy and apoptosis (Yang *et al.*, 2016 and Song 2023)^[19, 5].

In a natural trichothecene poisoning, pale, slightly enlarged liver, severe depletion and necrosis of lymphocytes in the bursa of Fabricius, disseminated mucosal necrosis in the glandular stomach and severe necrotic typhilitis with pseudomembrane were observed (Konjević *et al.*, 2024)^[20].

The feed samples of suspected flocks were sent to mycotoxins lab and were confirmed positive for presence of T2 Toxin through thin layer chromatography. This was considered as gold standard test for confirmation of mycotoxins in poultry feed samples (Raju *et al.*, 2000; Romer *et al.*, 1978) [21, 22].

Conclusion

The combination of field history, clinical signs, gross and histopathological examination and laboratory detection of T-2 toxin provides a more reliable approach for the diagnosis and investigation of T-2 toxicosis in poultry. Early recognition is essential for reducing production losses and preventing severe mycotoxicosis.



Fig.1: Bird showing caseous plaques at angle of mouth



Fig.2: bird showing ulcer in oral mucosa



Fig.3. Ulcers on the gizzard mucosa

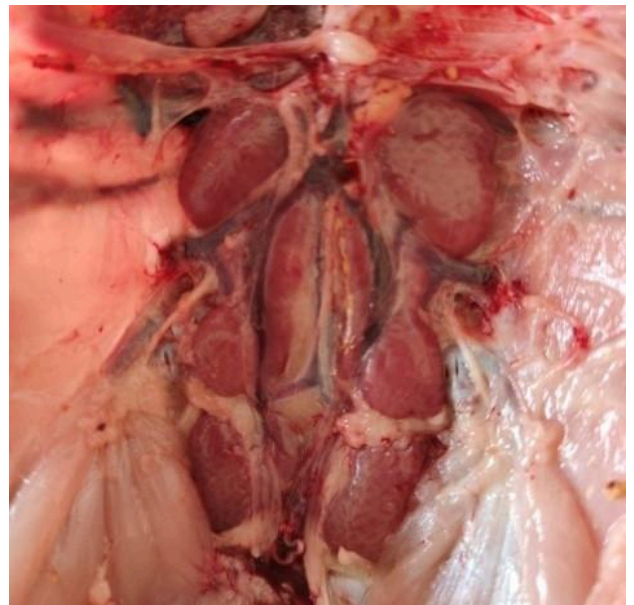


Fig.4: Swollen and congested kidneys

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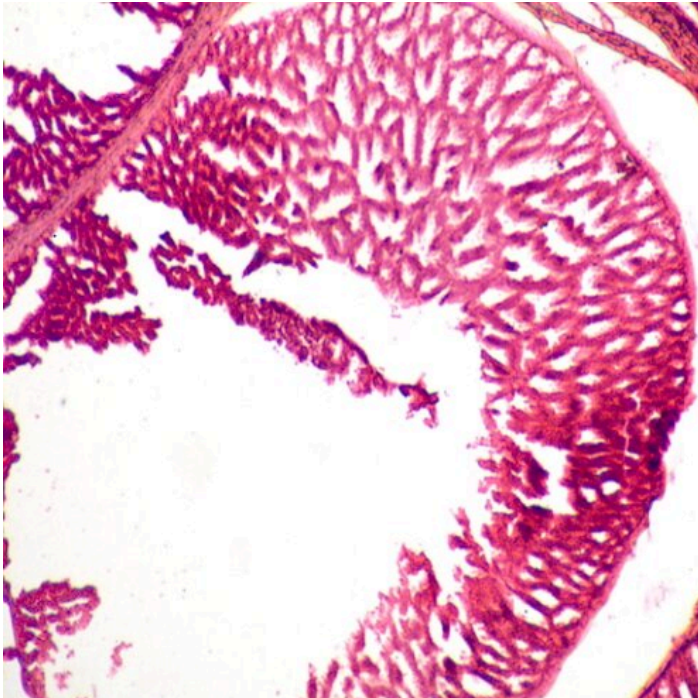


Fig 5: Proventriculus showing ulceration and desquamation of epithelium 100x

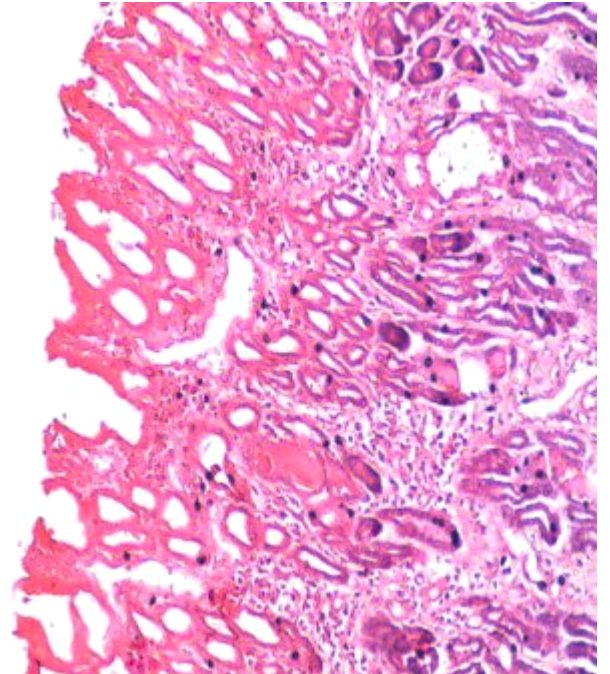


Fig6. Gizzard showing desquamation of epithelium 100x
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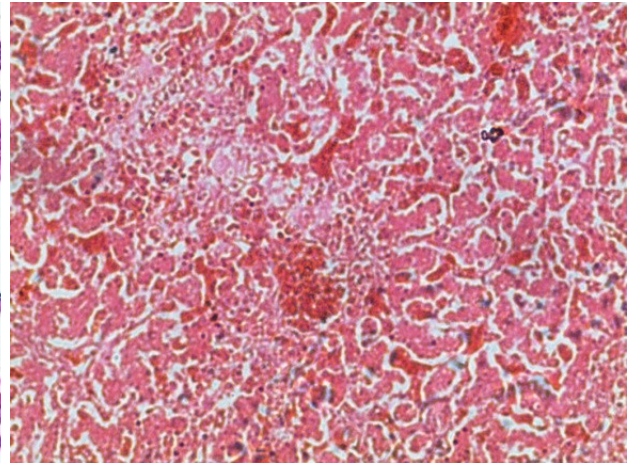
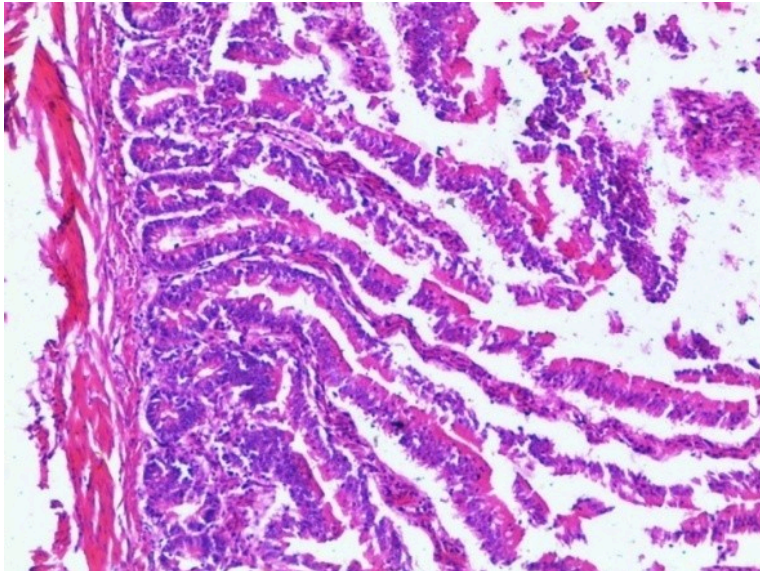


Fig 7. Intestine showing necrosis of epithelium and debris in the lumen 100x

Fig 8. Liver showing focal areas of necrosis, congestion and cellular infiltration 200x

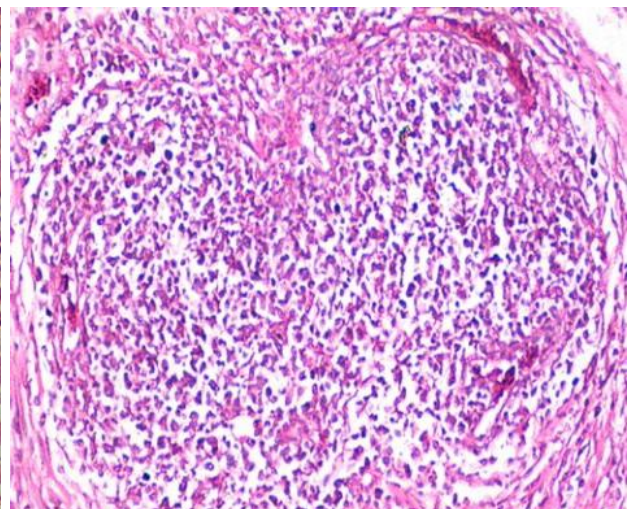
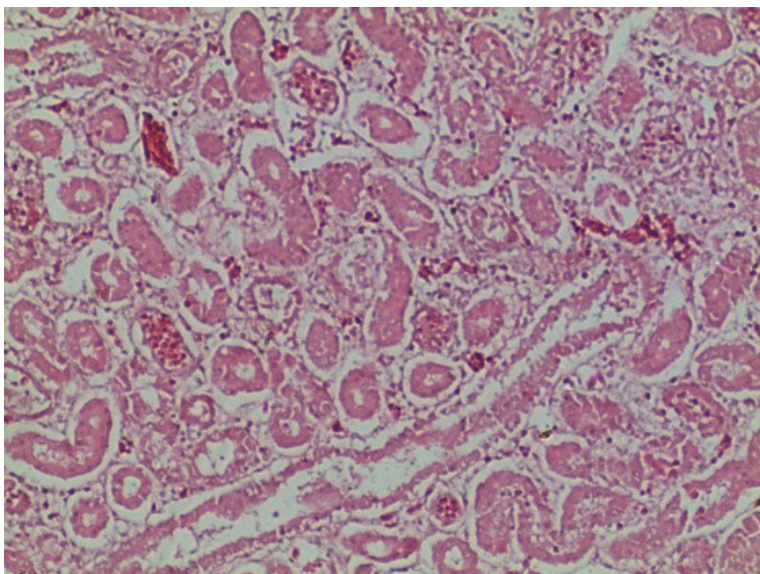


Fig 9. Kidneys showing tubular degeneration and necrosis 100x

Fig 10. Bursa showing showing depletion of lymphocytes 200x

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Conflict of interest

All authors declare that they have no conflict of interest

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