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A REVIEW

Necrotic enteritis in layer chicken

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Abstract : Necrotic enteritis (NE) is one of the economically important disease of young chicken causing high mortality and morbidity. The disease is caused mainly by toxin of *Clostridium perfringens* type A and C. This review describes in brief about necrotic enteritis of layer chicken.

Key words : Necrotic enteritis, Layer chicken

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INTRODUCTION

Clinical necrotic enteritis can be defined as a disease of primarily young chickens, caused by infection with and toxin production by, *Clostridium perfringens* type A and C. The clinical infection is characterised by sudden onset, high mortality and necrosis of the mucous membrane of the intestine. The disease is also known as clostridial enteritis, enterotoxaemia and rot gut (Opengart, 2008).

Occurrence :

Global scenario:

Necrotic enteritis was first described by Bennetts (1930) in Australia and fully characterised by Parish (1961) in the United Kingdom. Soon afterwards, NE emerged as a worldwide problem (Williams, 2005). It was reported in various countries of the world including Bangladesh (Islam *et al*., 2006), Belgium (Timbermont *et al*., 2011), Denmark (Pedersen *et al*., 2008), Norway (Kaldhusdal and Skjerve, 1996), South Korea (Lee *et al*., 2011), Turkey (Kalender and Ertas, 2005), United Kingdom (Hermans and Morgan, 2007) and United States (Dhillon *et al*., 2004).

Indian scenario:

Necrotic enteritis was first reported by Chakraborty *et al*. (1984) in West Bengal. It was also reported in Jabalpur, Madhya Pradesh in layers at 7-8 weeks of age (Shukla *et al*., 2007) and in caged layer chickens at 22 weeks of age (Sawale *et al*., 2010) in Maharashtra. Necrotic enteritis in commercial broilers of two to six weeks of age and in commercial layers of 9 to 15 weeks of age groups was reported by Malmarugan *et al.* (2013).

Epidemiology :

Agent:

Clostridium perfringens type A and type C is a gram-positive, spore forming anaerobe causes necrotic enteritis in layer chicken (Seedy, 1990). Both of these strains are capable of producing various toxins and enzymes responsible for the associated lesions and clinical signs. Specifically, alpha-toxin produced by *C. perfringens* types A and C and beta-toxin produced by *C. perfringens* type C are responsible for the production of the intestinal mucosal necrosis commonly associated with NE (Songer and Meer, 1996). *Clostridium perfringens* type A or C leading to gaseous extension of the small intestine, the production of one or more exotoxins and enteric toxicosis (McDevitt *et al*., 2006).

Host:

Necrotic enteritis is encountered in both layers and broilers (Shukla *et al*., 2007). Outbreaks of NE have been reported in chickens from two weeks to six months of age (Opengart, 2008). Necrotic enteritis has been reproduced experimentally in turkeys (Fagerberg *et al*., 1984) and Japanese quail (Opengart, 2008).

Epidemiological measures of causal association:

Age:

Naturally occurring outbreaks of NE have been reported in chickens from two weeks to six months of age (Opengart, 2008). A majority of outbreaks of NE have been reported in two to five weeks old broiler chickens raised on litter (Tsai and Tung, 1981).

Predisposing factors:

Consumption of diets high in energy, protein, fish meal as well as the consumption of wheat based diet predispose the birds to NE. Bird fed diets based on wheat, rye, oats, barley suffer with more severe NE than birds fed with maize based diets (Kaldhusdal and Skjerve, 1996).

Dysfunctions of the alimentary tract are necessary predisposing factors of NE infection in poultry. Intestinal stasis, intestinal distension, coccidiosis, salmonellosis, crop mycosis and haemorrhagic enteritis may predispose the birds to NE infection (Williams, 2005).

Immunosuppression in birds caused by IBD, chicken infectious anaemia virus and Marek's disease (MD) as well as non-specific stress may predispose the birds to NE (Schuring and Gils, 2001).

Clinical signs:

The birds affected with NE showed ruffled feathers, marked depression, inappetence, tendency to huddle, watery droppings and diarrhoea (Porter, 1998). Clinical illness of NE was very short; often birds were just found acutely dead without any outward signs of disease (Opengart, 2008).

Gross lesions:

Gross lesions found in birds affected with naturally occurring NE outbreaks usually confined to the small intestine, primarily the jejunum and ileum (Tsai and Tung, 1981). Intestines are often friable and distended with gas. The mucosa was lined by a loosely to tightly adherent yellowish to greenish pseudomembrane that is often described as having "Turkish towel" appearance. Flecks of blood have been reported but haemorrhage is not a prominent feature (Opengart, 2008 and Malmarugan *et al*., 2013).

Diagnosis:

A presumptive diagnosis may be made from the case history, clinical signs, lesions and staining fresh smears of upper part of the intestinal tract with Gram staining showing an abundant number of Clostridial organisms (Ficken and Wages, 1997).

Collection of samples:

Clostridium perfringens can be isolated readily from intestinal contents, scrapings of intestinal wall or haemorrhagic lymphoid nodules (Opengart, 2008).

Isolation and identification:

Isolation of *C. perfringens* is by enrichment of intestinal contents and intestinal scrapings in Robertson's cooked meat medium and subsequent culturing in blood agar supplemented with 5 per cent defibrinated sheep blood or egg yolk agar or clostridial agar or perfringens agar (Malmarugan *et al*., 2013). Identification of *C. perfringens* can be done by using biochemical tests such as fermentation of lactose, glucose, maltose, hydrolyze gelatin and reduce nitrate and by Gram staining (Hafez, 2011).

Economic losses:

Globally, the economic loss attributable to avian NE is estimated to the cost of $ 2 billion annually, largely because of medical treatments and impaired growth performance (Cooper and Songer, 2009). Subclinical NE causes 12 per cent reduction in body weight and a 10.9 per cent increase in feed conversion ratio compared with healthy birds (Skinner *et al.*, 2010). For the commercial poultry industry, controlling the levels of *C. perfringens* is an important issue because of the economic cost of infected flocks (Hafez, 2011).

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