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INCIDENCE AND PATHOLOGY OF COLIBACILLOSIS ON LUNGS OF BUFFALO CALVES

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Colibacillosis occurs as an acute fatal septicaemic disease of young buffalo calves and is seen worldwide. The main determinant of the disease is decreased immune status of the young one along with improper managemental conditions and stress factors, which also enhanced the growth of opportunistic bacteria and therefore infection flares up. Colisepticemic form of colibacillosis usually results in the rapid death of the calf and is associated with an *E. coli* bacteraemia. Present paper highlighted the incidence and pathology of colibacillosis on lungs of buffalo calves. Incidence of colibacillosis affecting lung was recorded as 20.73 per cent. Grossly, the affected lungs showed moderate to marked areas of congestion, haemorrhages and presence of white necrotic foci on parenchyma. Microscopically, lung sections showed marked vascular congestion along with hyperplasia of bronchiolar epithelium, moderate to marked haemorrhages in between alveoli, marked cellular infiltration of neutrophils, macrophages and few lymphocytes in lung parenchyma, perivascular and peribronchiolar cellular infiltration, pulmonary emphysema and thickening of alveolar septa due to congestion of septal blood vessels and leucocytic infiltration along with presence of oedema in some alveoli.

Key Words: Colibacillosis, Buffalo calves, Septicaemia, Lung.

To exploit dairy industry for maximum gains, greater attention needs to be given to the disease problems of buffalo in general and buffalo calves in particular. Diseases of buffalo calves are known to cause heavy financial losses due to mortality along with decrease in productivity of recovered calves and limited scope for selection of breeding animals. Buffalo calves suffer from high mortality than cow calves (Tomar and Tripathi, 1991).

Diarrhoea is a major problem in livestock production in Egypt and throughout the world (Farid *et al.*, 2001 and Ibrahim, 2007). A key role in the aetiology of bacterial infectious diarrhea in buffalo calves has been attributed to *E. coli* infection (Zaman *et al.*, 2006). Diarrhoea due to *Escherichia coli* is one of the most common diseases of young buffalo calves, despite vaccination programs, management measures and necessitating treatment with antibiotics and fluid therapy.

Colibacillosis is one of the most important bacterial diseases and is a major cause of morbidity and mortality in buffalo calves. Mortality due to *E. coli* infection in buffalo calves was 25.49 per cent (Abd-Elrahman, 2011). *E. coli* produces enterotoxic and septicaemic colibacillosis in young buffalo calves. Calves that are deficient in immunoglobulins are most susceptible to colisepticaemic form (White *et al.*, 1986). Systemic infection occurs when a large no. of *Escherichia coli* gain access to the blood stream from the respiratory tract or intestine. There is a period of subclinical bacteremia that progresses to septicemia and death. By invading the wall of small intestine, *E. coli* destroys the epithelium and causes bacteremia and localizes in other organs including lungs. Therefore, the present report deals with the incidence and pathology of colibacillosis on lungs of buffalo calves.

MATERIALS AND METHODS

For present study, a total of 610 buffalo calves of below 6 weeks of age, irrespective

of sex and breeds were examined from different slaughter houses of Rajasthan. Out of these, 68 buffalo calves suspected for colibacillosis were further processed for bacteriological and histopathological examination. Samples were collected in duplicate, one for bacteriological and another for histopathological examination.

Isolation and identification of *E. coli* organism from lungs of buffalo calves:

For bacteriological examination, sterile cotton swabs were used for aseptic collection of the samples from the sites showing visible abnormality at necropsy for the bacterial isolation and streaked on Mac-Conkey agar petriplates within two hours of sampling. Inoculated plates were incubated at 37 ^{0}C for 24 hours. After this, single pink colony from the Mac-Conkey lactose agar showing lactose fermentation was fished out and streaked on Eosine Methylene Blue agar petriplates. The inoculated plates were then incubated at 37 ^{0}C for 24 hours. From EMB agar the colonies showing metallic lusture, were further subjected to primary and secondary identification tests as per the method described by Cowan and Steel (1965) and Carter (1975).

Histopathological Study:

After confirmation of bacteriological examination, lung samples were processed for histopathological examination. Samples were examined grossly for alteration in morphology in terms of shape, size, colour, consistency, odour, location and type of the lesions. As far as possible, the colour of lung was noted immediately after collection and prior to fixation. Samples were promptly preserved in 10 per cent formal saline and processed mechanically for paraffin embedding by Acetone and Benzene technique (Lillie, 1965). The sections of 4-6 micron thickness were cut and stained with routine Haematoxylin and Eosin staining method.

RESULTS AND DISCUSSION

In the present study, total 217 *E. coli* isolates were obtained from different organs, out of which, 45 were isolated from lungs. Therefore, the incidence of colibacillosis affecting lung was recorded as 20.73 per cent. Similar incidence was recorded by Seema (2007) as 22.05 per cent. A relatively higher incidence was recorded by Verma and Kalra (1975) as 33.33 per cent and Herbach *et al.* (1998) as 36 per cent.

Bacteriological Study:

The isolation and identification of bacteria (*Escherichia coli*) was carried out from 45 samples of lung. The 45 tissue samples inoculated on Mac-Conkey agar plates revealed the presence of lactose fermenting pink colonies (Fig. 1).

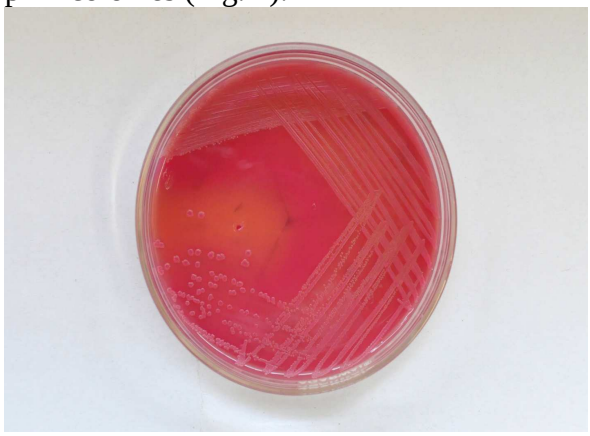

Fig.1 Photograph of MacConkey lactose agar petriplate showing pink colonies of bacteria.

The bacteria subcultured on Eosine Methylene Blue (EMB) agar plates resulted into colonies exhibiting metallic sheen, a characteristic feature of *Escherichia coli* (Fig. 2). These organisms when subjected to primary identification tests revealed following properties:

Gram Reaction	G(negative) (Fig. 3)
Morphology	Bacilli
Catalase	+
Oxidase	-
O/F Test	Fermentative
Growth in TSI slant	A/A Gas
Indole	Positive
M.R.	Positive
V.P.	Negative
Citrate	Neg

Histopathological Study:

Grossly, the affected lungs showed marked congestion and haemorrhagic foci on lung parenchyma. Lungs were pale and also revealed presence of white necrotic foci on lung parenchyma. These morphological manifestations reflects acute nature of the disease and such type of pathological

alterations have also been recorded by Wray *et al.* (1992), Singh *et al.* (1996), Herbach *et al.* (1998), Jubb *et al.* (2007) and Seema (2007).

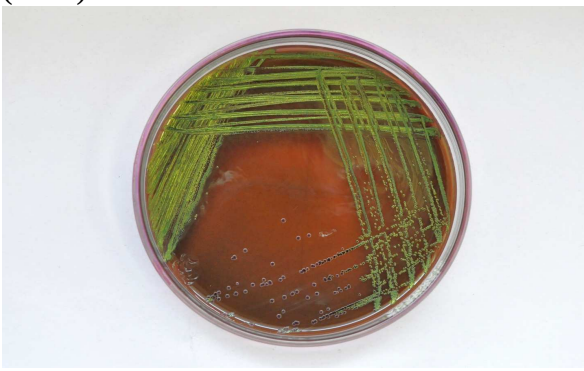

Fig.2 Photograph of *Escherichia coli* colonies showing metallic sheen on EMB petriplates.

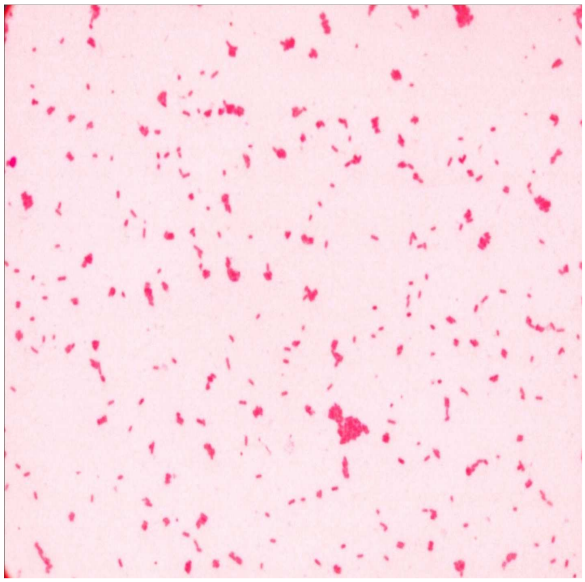

Fig.3 Photograph of Gram staining showing pink coloured Gram-negative rods *Escherichia coli* isolated from lung samples.

Microscopically, the histopathological alterations in the lungs comprised of marked vascular congestion along with hyperplasia of bronchiolar epithelium (Fig. 4). Some sections revealed moderate to marked haemorrhages in between alveoli, marked cellular infiltration of neutrophils, macrophages and few lymphocytes in lung parenchyma, perivascular and peribronchiolar cellular infiltration and pulmonary emphysema. Thickening of alveolar septa due to congestion of septal blood vessels and leucocytic infiltration along with presence of oedema in some alveoli was also present in few cases (Fig. 5).

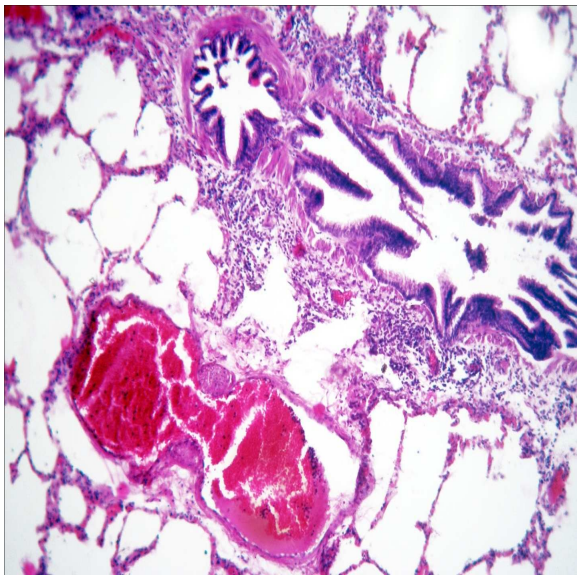

Fig.4 Microphotograph of lung showing marked vascular congestion along with hyperplasia of bronchiolar epithelium and peribronchiolar cellular infiltration. H&E, 10

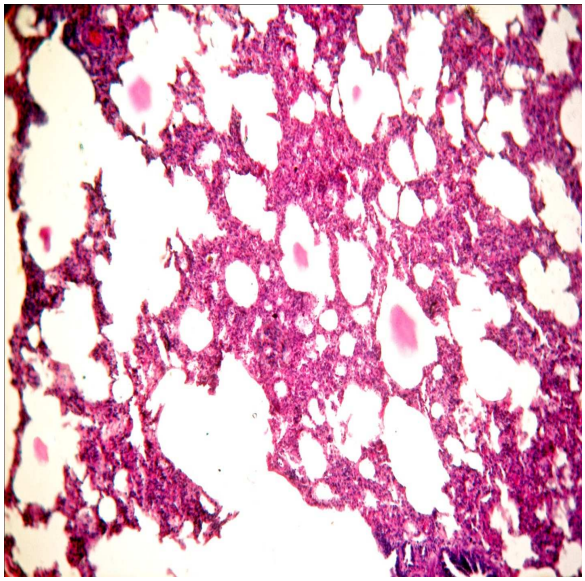

Fig.5 Microphotograph of lung showing thickening of alveolar septa due to congested septal blood vessels and leucocytic infiltration along with presence of oedema in some alveoli. H&E, 100 X

Similar findings were observed by Singh and Singh (1983), Singh *et al.* (1996), Jubb *et al.* (2007) and Sayyari and Sharma (2011). The acute inflammatory reaction might be due to damage of minute blood vessels *viz.* capillaries which resulted into oedema and congestion.

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