J. Egypt. Vet. Med. Ass. 48, No. 165-173 (1988)

EDITOREAL

COLIBACILLOSIS IN ANIMALS, POULTRY AND MAN

Prof. Dr. M.REFAI

Department of Microbiology, Faculty of Veterinary Medicine, Cairo University, Giza, Egypt.

CONTENTS

1. INTRODUCTION

Colibacillosis is the most common disease of newborn animals caused by Escherichia coli which leads to a high mortality rate

and hence great ecomonic losses. E.coli, isolated for the first time by Escherich in 1885, is in general a normal commensal in the intestinal tract of mammals and birds. Some strains under certain conditions can however cause enteritis or septicaemia in young animals. These strains are called enteropathogenic (enterotoxic) Escherichia coli (EEC). They are mostly host-specific and a limited number of well-defined serotypes are closely associated with specific diseases in each animal host.

2. AETIOLOGY

2.1 Morphology and antigenic structure

E.coli is a small rod-shaped, gram-negative organism, may be motile or non-motile. It grows readily on all ordinary media through a wide range of temperatures with an optimum of 37°C. It is fairly resistant to drying and to the action of many chemical disinfectants. It is destroyed by pasteurization.

The surface structures of E.coli are expressed as 0 (somatic), K (capsular) and H (flagellar) antigens. There are at least 150 recognized 0 antigens, 90 K, and 50 H. The 0 antigens are lipopolysaccharide and heat-stable. The K antigen are polysaccharide and/or protein. On the base of their sensitivity to heat, they are classified to L, B, and A. The H antigens are protein. Some strains of E.coli develop pili or fimbriae. Certain pilus-antigens, e.g. K88 and K99, act as adhesion or colonization factors. These factors are glycoprotein, heat-labile, and plasmid-controlled.

2.2 Resistance to chemotherapeutics

Originally, wild strains are sensitive in-vitro to most antibiotics and sulfonamides. However, the massive application of such substances in the treatment and prophylaxis, in addition to their use as feed additives exert a selection pressure on the intestinal flora so that high percentage of E.coli cells in the intestine acquire a multiple resistance against such drugs, The resistance to antibiotics and sulfonamides is often induced by extrachromosomal genes in plasmids and can be transferred within seconds through certain pili to antibiotic sensitive cells, not only of E.coli, but also of salmonella and other members of the family Enterobacteriaceae.

2.3. Toxins

2.3.a. Endotoxins

Endotoxins are a part of the cell-wall of E.coli composed of polysaccharides which determine the antigenic specificity of the antigens and phospholipids which are responsible for the toxic activity.

2.3.b. Exotoxins (enterotoxins)

The exotoxins act directly on the cells of the small intestine and they are therefore termed enterotoxins. These toxins are either heat-stable (ST) or heat-labile(LT) based on their resistance to heating at 60°C for 10 minutes. The ST is not antigenic, but it is hapten-like, whereas the LT is antigenic and can be neutralized by specific antisera. Enterotoxin production is plasmid-controlled. The capability to produce enterotoxins (Ent +) is transmissible to apathogenic recipient cell of the same type.

The enterotoxins cause no damage to the intestine but they stimulate guanylate cyclase activity of the ileal epithelium (ST) or adenyl cylase activity of intestinal and capillary epithelium (LT) and this results in a hypersecretion of electrolytes particulary Na^+ and HCO_3 and an increased diffusion of water into the lumen of the intestine. This results in acidosis and dehydration.

3. COLIBACILLOSIS IN CALVES

3.1.Colisepticaemia

3.1.a. Aetiology

It is a peracute or acute systemic disease of newborn calves that occurs in the first few days of life of calves with hypo or agammaglobulinaemia. The high mortality is the result of the severe damage caused by the endotoxins of certain serovars , namely 08, 09, 015, 078, 055, 0115, and 0117. Most of the strains possess Kantigens. It is not commonly an intestinal infection, the nasopharynx and its lymphatic tissues are most probably the main route of infection. However, in case of delayed infection the organisms may reach the intestine via the blood and cause enteritis. Sometimes, the infection may occur orally and diarrhoea takes place at first.

3.1.b. Clinical picture

There are no typical diagnostic symptoms. Calves become soon off-food, weak, and sleepy. Diarrhoea may precede or follow these symptoms and death is sudden.

3.1.c. Immune mechanism

The bovine placenta does not allow the passage of maternal immunoglobulins to the foetus. Accordingly, the immunity in newborn calves depends on the postnatal intake and resorption of sufficient amounts of colostral immunoglobulins. The IgG and IgA are the important immunoglobulins that protect the calf from infection, while the IgM and IgG act against colisepticaemia. The colostral protein can be resorped in the intestine of the newborn calves only during the first 24 hours after birth. The permeability of the intestinal mucosa to the immunoglobulins is highest in the first 6 hours of life, then it is reduced.

3.1.d. Therapy

A. Antibiotics as Colistin, Ampicillin, Amoxycillin, Gentamicin, Neomycin, Trimethoprim-Sulphonamide compound, Furazolidon or nitro-furazone. and Chloramphenicol.
B. Intravenous administration of gammaglobulin preparations.
C. Anti-shock treatment.

3.1.f. Prophylaxis

A. Application of general hygiene measures.
B. Feeding newborn calves sufficient colostrum in the first hours after birth.
C. Vaccination of the mothers with coli-vaccines 6-8 weeks before delivery.

3.2. Coli-diarrhoea

(Enteric colibacillosis, enterotoxaemic colibacillosis, scours).

3.2.a. Aetiology

Coli-diarrhoea is a disease of newborn calves that occurs during the first few hours and up to 3 weeks after birth. The causative E.coli strains possess the pili antigens that enable the adhesion of E.coli cells to the intestinal mucosa of the calves. They also have the capability to produce the thermostable entertoxins (ST). Such

strains belong mostly to the serovars : 8, 9, 20 and 101. It is to be noted that both pili-development and enterotoxin-production are plasmid-controlled. They are not a serotype-specific character.

3.2.b. Clinical picture

The main symptom is diarrhoea. The faecal matters are offensive, greyish white or yellowish, sometimes bloody, or watery.

3.2.c. Pathogenesis

The newborn calves ingest the E.coli from the contaminated environment, which multiply rapidly in the intestine particularly in the absence of colostral immunoglobulins. With the help of K99 they adhere to the mucosal surface and secrete the heat-stable enterotoxin which stimulates the secretion of water and electrolytes by the mucosal epithelial cells into the lumen of the intestine which causes the diarrhoea. The loss of body fluid and electrolytes including sodium hydrogen carbonate leads to acidosis and in severe cases to cholera-like dehydration-shock and death.

3.3.a. Immune mechanism

As mentioned before (3.1 a.), the newborn calves are at birth immunologically unprotected and depend on the intake of colostral antibodies for protection. The protection mechanism is not yet fully clear. It seems however, that antibodies against K99 prevent the adhesion of the E.coli strains containing the colonizing factor K99 to the intestinal mucosa. On the other hand, the resorption of the colostral antibodies and the presnce of the IgG in the blood protect the calves from the sequellae of an entertoxamia.

3.3.b. Therapy

A. Antibiotic treatment e.g. Colistin, Neomycin, Ampicillin, Amoxycillin, Gentamicin, Trimetroprim-Sulphonamide compound, chloramphenicol and Furazolidon.
B. Replacement of lost water and electrolytes.

3.3.c. Prophylaxis

A. Application of hygienic measures.
B. Intake of sufficient colostrum in the first hours of life.
C.Vaccination of the mother.

4. COLIBACILLOSIS IN LAMBS AND KIDS

4.1. Colisepticoemia (systemic colibaciloosis)

The systemic form of colibacillosis is the most frequent E.coli infection in lambs 1 day to 14 weeks old. The disease is characterized by a peracute course and sudden death. The common serovars involved are 015, 020, 035, 075, 078, 0115, 0125,and 0137. In less severe cases, the infection may be localized in the meninges. In chronic cases, the infection is localized in the joints.

4.2. Coli-diarrhoea (enteric colibacillosis)

The enteric form of colibacillosis is rare. It occurs at the age of 2-8 days and is caused by the same enterotoxaemic strains of calves. These strains possess the K99 antigen and produce ST enterotoxins. The morbidity is mostly very high. The main symptom is the severe watery diarrhoea. Pathogenesis, therapy and prophylaxis as in calves.

5. COLIBACILLOSIS IN PIGLETS

5.1. Piglet diarrhoea (piglet scours)

It is an acute disease of newborn piglets characterized by severe diarrhoea, caused by E.coli strains that belong to the 0 groups 8, 9, 20, 45, 101, 138, 139, 141, 147, 149 and 157. Most strains are haemolytic and possess the pilus-antigen (colonizing factor) K88. Very few strains possess the pilus-antigen K99 or 987P. Most of the piglet enteropathogenic strains can produce the thermolabile (LT) and thermostable (ST) enterotoxins.
Antibiotics such as chloramphenicol, chlortetracycline and oxytetracycline are successfully used in treatment. They are also used prophylactically in sows before delivery to obtain sufficient concentration of the drugs in colostrum and milk. Vaccination of the sow and intake of colostrum by the newborn are of paramount importance.

5.2. Enterotoxaemia (Odema disease)

It is a disease that affects piglets in the first week after weaning where a massive multiplication of certain E.coli serovars take place in the intestine which produce toxins that cause the diarrhoea and oedema. The most frequently reported serovars are 0139 and 0141, however other serovars are also involved as 02, 08, 018,

020, 045, 075, 078, 086, 0111, 0115, 0117, 0121, 0133, 0145 and 0147, Most of strains involved are haemolytic. The pathogenesis of the disease is not fully clear. The nervous manifestations have been attributed to a neurotoxin, the oedema to a sort of hypersensitivity and anaphylactic shock caused by resorbed endotoxins and the diarrhoea to the enterotoxins.

6. COLIBACILLOSIS IN FOALS

In newborn foals E.coli can cause also colisepticaemia and coli-diarrhoea. It is also involved in joint ill or navel ill.

7. COLIBACILLOSIS IN PUPPIES

E.coli, particularly hoemolytic strains of the serovars 042, have been associated with acute generalized infections.

8. COLIBACILLOSIS IN POULTRY

9.1 Colisepticaemia

This disease occurs mostly in broilers and is characterized by fever, loss of appetite, respiratory distress, weakness and often also diarrhoea. The most frequently recovered strain belong to the 0 groups 02:KL, 01:K1 and 078:K80. The infection occurs through inhalation of the E.coli in the faecal dust which reaches the air sacs where it multiplies and causes local inflammation. It then extends to the surrounding tissues and reaches the blood. The disease occurs frequently in combination with other infections as mycoplasmosis, infectious bronchitis and New Castle disease, with the development of severe lesions. The most common lesions are fibrinous pericarditis, peritonitis, perihepatitis, inflammation of the air-sacs, salpingitis, synovitis and panophthalmitis. The inflammatory reactions in the internal organs are caused probably by the endotoxins released following the lysis of the bacterial cells. The disease can be controlled by antibiotic therapy and application of hygienic measures. Excessive over crowding should be avoided and adequate ventilation should be provided.

9.2. Coligranuloma

It is a relatively rare and sporadic disease of chicken caused by various E.coli serovars and is characterized by the development of tuberculosis or leucosis-like nodules in the caecum, liver,

lung and kidneys. The cut surface of the nodules is dry. The clinical symptoms are not characteristic and death may be sudden.

10. COLIBACILLOSIS IN ADULT ANIMALS

10.1 . Mastitis

E.coli mastitis is rare in cattle but frequent in sows. The disease is more common in housed cattle and heavily fed sows, soon after calving and farrowing,respectively. The disease may be peracute, acute or chronic. The peracute form is most common in cattle and is the only form in sows. The onset is sudden and the systematic reaction is severe. The secretion is thin, yellow, and contains flakes. In the acute form there is a mild inflammation and clots appear in the milk but there is no systematic reaction. In chronic form the symptoms are less severe and recurs intermittently. The causative E.coli strains possess a distinct capsule. The inflammation and systemic reactions are probably caused by the endotoxins.

10.2 Metritis in mares and bitches and infection of the **urinary system** in different animals have been also attributed in some cases to E.coli infection.

11. COLIBACILLOSIS IN MAN

In man, E.coli causes infantile gastro-enteritis, traveller's diarrhoea, infection of the urinary system (cystitis and pyelopephritis) as well as cholangitis, sinusitis, otitis etc.

The infantile gastro-enteritis is caused by certain serovars e.g. 025, 026, 055, 0111, 0119, 0126, 0128 and 0.142. The disease occurs mostly in infants less than one year. It seems that the enterpathogenic strains possess a colonizing factor analogous to K88 and K99 of animals and produce LT and ST enterotoxins. The pathogenesis of diarrhoea is the same as in calves. In adults the enterotoxins are the cause of what is known as traveller's diarrhoea.

The urinary tract infections are caused by relatively few serovars of E.coli(02, 04, 06). They colonize the peri-urethral area or the vagina by certain adhesion-fimbrae Once the bacteria have invaded the urinary tract, they can ascend to cause infection of the upper urinary system.

12. DIAGNOSIS

Final diagnosis depends on the isolation of E.coli from various organs in case of septicaemic infections and from the faeces in profuse amounts in case of enteritis

12.1. Isolation

For isolation MacConkey's agar is satisfactory on which E.coli appear as pink coloured colonies after incubation at 37°C for 18 hours. Blood agar plates can be useful additional medium for the detection of the haemolytic strains. There are many other media which can be used as Endo and EMB media.

12.2. Biochemical identification

It is known that all strains of E.coli ferment glucose and lactose with production of acid and gas. The majority of strains ferment mannitol, form indole but fail to produce H_2S and do not grow in citrate medium. Most strains do not develop urease, give a negative Voges-Proskauer reaction and are positive to the methyl red test. In the routine diagnosis IMVIC tests would be sufficient for pink colonies recovered from MacConkey's agar.

12.3. Serological identification

The biochemically identified pure cultures of E.coli are then subjected to serotyping with poly and monovalent antisera for the different 0,K and H antigens using slide agglutination test. The commercially available E.coli antisera are OK-antisera which are useful in the routine diagnosis. The presence of the pilus antigens K88 and K99 can be detected by slide agglutination test using monospecific absorbed antisera.

12.4. Rapid diagnosis

The biochemical identification can be done using APT, Enterotube or Micro ID systems.

The flourescence antibody technique (FAT) is used routinely in many laboratories for the direct detection of the E.coli in the specimens. The dirct method is mostly applied using poly and monovalent E.coli antisera. Recently the India-ink immune-reaction has been recommended for rapid detection of the organisms. For the detection of enterotoxins, the ligated ileal lube test, suckling mouse test, ELISA or SPIRA tests can be used.