Some Clinical And Pathological Features Of Enteritis In Broilers — Observations On Treatment In The UK — Mark Pattison, Ph.D., D.P.M.P., M.R.C.V.S.

“Biography”

Qualified as a Veterinary Surgeon in 1968 from Bristol University Veterinary School.

1970 - 1974 Research and diagnostic work in Poultry Department, Central Veterinary Laboratory, Weybridge.
1972 Awarded MSc Veterinary Pathology from London University.
1974 - 1982 Head of Veterinary Services for Ross Breeders, Newbridge, Midlothian, Scotland.
1976 Awarded Ph.D for thesis “Studies on the Pathology of Infectious Bursal Disease”.
1982 Head of Veterinary Services for Sun Valley Poultry, Hereford.
1984 President of British Veterinary Poultry Association.
<table>
<thead>
<tr>
<th>Year</th>
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<tr>
<td>1991</td>
<td>Group Technical Manager for Sun Valley Poultry, Hereford with responsibility for veterinary, technical and laboratory services for the Company.</td>
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<tr>
<td>1996</td>
<td>Veterinary Services Director, Sun Valley Poultry, Hereford.</td>
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Some Clinical And Pathological Features Of Enteritis In Broilers -- Observations On Treatment In The UK

“Abstract”

Mark Pattison, D.V.M., MP Consulting

Introduction

The appearance of wet litter with birds producing abnormally wet droppings has become a regular feature of UK broiler production. It is important to recognise the problem quickly, make an appropriate diagnosis and treat as quickly as possible. It may also be possible to anticipate when the problem may arise and devise a preventive strategy to minimise the effects of the disease.

Clinical signs

The signs of enteritis are seen at any time from about 15 days. The disease is characterised by diarrhoea with the production of abnormally wet droppings. The faeces usually have a larger volume, are pale or viscous with an excess of fluid around them. The use of litter boxes (described elsewhere) can be helpful in recognising the early stages of the condition. At the onset of enteritis there is normally an increase in water intake but sometimes there may actually be a reduction in water consumption. Sometimes birds are huddled with ruffled feathers but this is not a consistent feature. Often selective feeding is reported where birds ‘flick’ feed or whole grains out of the pans onto the litter.
The end result is a reduction in growth and an adverse effect on feed conversion. There are often secondary effects such as an increase in leg problems due to osteomyelitis.

**Post mortem features**

The signs described above are similar for quite a broad range of pathological lesions. The most severe lesions are seen in necrotic enteritis, but these occur less frequently now, as most cases of enteritis occur without mortality.

Where death occurs, the birds are usually in good condition with food in the crop and gizzard indicating that death has been sudden. There is often a greenish tinge on the abdomen and the intestinal tract is swollen and flaccid, with a thin wall. There are usually focal areas of ulceration or larger patches of necrosis, which ultimately coalesce resulting in sloughing of the intestinal lining.

It is much more common to investigate cases of wet litter or enteritis where no mortality has occurred. In this situation it is convenient to examine six freshly culled birds per house. They should be average birds neither the biggest nor the smallest. The intestines are incised at three points to include the duodenum, mid intestine and lower intestine. The aim is to follow the digestive process and examine the contents to see if they become progressively more solid.

Where the enteritis is mild the wall of the intestine maybe thinned, ballooned or slightly inflamed. The gut contents may be full of bubbles, watery or contain viscous orange-coloured mucus. Occasionally the contents appear yoghurt-like and the presence of whole grains of wheat is indicative that digestion is not occurring properly. This condition is often referred to as dysbacteriosis.

In many cases, there is no enteritis and the intestine contains an abnormal amount of fluid, which is clear and like water. The caecae may be dilated.

It is important to distinguish this condition from malabsorption syndrome which can produce similar clinical signs, in particular selective feeding behaviour. Also the possibility of coccidiosis either at a clinical or sub-clinical level must be considered.

**Prevention and Treatment**

Various strategies have been employed within one integration with varying degrees of success. These regimes have to be changed regularly because, not surprisingly, the antibiotics become less effective over time.

Tylosin tartrate (Tylan®, Elanco Animal Health) was used in a prevention programme for three crop cycles as follows:

Treatment was given for two days at 14 and 15 days, repeated at 21 and 22 days at a dose of 100mg/5000 kg bodyweight. The farms were categorised according to the effectiveness of the treatment. Of 78 farms, 55 completed three cycles of production without any clinical signs of enteritis. On 15 farms,
Some or all the houses had to be treated and the product used was amoxycillin. Eight farms did not receive the Tylan programme and all had to be treated and amoxycillin was used.

After three cycles, in order to prevent the response to Tylan becoming less satisfactory, it was decided to rest this product.

Amoxycillin has been used as the standard for treatment but proved not to be effective in a prevention programme. There are signs, too, that this product became less effective over time and several flocks have had to be treated twice or even three times. Other prevention programmes have been tried:

- Penicillin in starter crumb at 1.2 Kg/tonne for 10 days was not effective, compared with two years ago when it worked well.

- Lincospectin at 50mg/Kg in water for 5 days at the start followed by chlortetracycline at 20-25 days in feed was reasonably effective. If the chlortetracycline was given earlier at 10-20 days, it was less effective.

In relatively mild cases it can be very difficult to decide whether to treat. There is always the possibility that if you decide not to treat, feed conversion will suddenly be lost with disastrous financial consequences.

**Conclusion**

Some farms are regularly affected by enteritis whereas others only see the problem intermittently. So there must be important factors of management and farm conditions which influence this syndrome.

It is quite clear that treating this condition with therapeutic medicines is not a sustainable long-term proposition.

A solution needs to be found so that these important medicines are being used sparingly and resistance does not become a problem.

The answers may differ between organisations and farms but will involve a combination of more optimal management, nutrition and satisfactory control of intercurrent disease.
Some Clinical And Pathological Features Of Enteritis In Broilers — Observations On Treatment In The UK

“Slide Presentation”

Figure C-1.

Figure C-2.

Figure C-3.

Figure C-4.

Some clinical and pathological features of enteritis in broilers - observations on treatment in the UK

Dr Mark Pattison
MP Consulting

HISTORY
March 2000: Removal of AGP’s
November 2000: Appearance of wet litter @ around 21 days
December 2000: Use of Amoxicillin for treatment/prevention
June 2001: Use of Tylan as preventive

GROWING REGIME

<table>
<thead>
<tr>
<th>No. of birds killed/week</th>
<th>970,000</th>
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<tbody>
<tr>
<td>No. of farms</td>
<td>78</td>
</tr>
<tr>
<td>Killing ages</td>
<td></td>
</tr>
<tr>
<td>37 days pullets</td>
<td></td>
</tr>
<tr>
<td>47 days pullets</td>
<td></td>
</tr>
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<td>52 days cockerels</td>
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| Coccidiosis to May 2001| Nicobarin 0-20 days
| Coccidiosis after June 2001| Nicobarin 0-12 days

Clinical Signs

- Droppings - larger volume with excess fluid around; maybe pale and viscous
- Water intake usually increases, but may decrease
- Mortality may rise
- Selective feeding - flicking of wheat or feed
- Ruffled feathers - sometimes
- Huddling - sometimes
- Secondary disease - leg problems
Post mortem procedure
- Examine duodenum/ mid intestine/ terminal intestine
- Follow the digestive process
- Cull 6 birds/house

Figure C-5.

Clostridiosis

Figure C-6.

Post mortem findings- three conditions
- Necrotic enteritis
- Dysbacteriosis/ mild enteritis
- Watery gut contents

Figure C-7.

Necrotic enteritis
- Birds in good condition- food in crop
- Greenish tinge on abdomen
- Intestine swollen and flaccid
- Gut lining ulcerated or necrotic and sloughing

Figure C-8.

Severe and extensive necrosis typical of field cases of necrotic enteritis

Figure C-9.

Dysbacteriosis/mild enteritis
- Intestine thinned, balloonied or slightly inflamed
- Gut contents
- Full of bubbles
- Watery
- Viscous, orange mucus
- Yoghurt-like
- Whole grains of wheat

Figure C-10.
Watery gut

- No enteritis
- gut wall slightly thin with watery contents
- caeca may be dilated

Mild enteritis and ballooning

Mild enteritis and ballooning

Differential diagnosis

- Malabsorption syndrome
- Coccidiosis

Enterotoxema acutum
- Enterotoxema necrotica
Some Clinical and Pathological Features

Figure C-17.

Figure C-18.

Figure C-19.

Figure C-20.

Figure C-21.

Other prevention programmes

- Penicillin in starter crumb 0-10 days, 1.2Kg/tonne
- Linconspectin 50mg/Kg - 5 days in water
- Chlortetracycline 4Kg/tonne-10-20 days or 20-25 days