

## **Pregnancy Toxaemia**

Pregnancy toxaemia, although primarily considered a disease of sheep, does also affect cattle, particularly cattle in late pregnancy. The problem is best described as starvation, but the aetiology and pathogenesis are similar to acetonaemia in that an energy deficit in the diet leads to massive mobilization of fat reserves, resulting in hypoglycaemia and hyperketonaemia.

The problem is most common in beef cattle grazing marginal land, but has been seen in dairy cattle in late winter in seasons where there has been a shortage of conserved forage. Cows of all ages are affected, but over fat animals and those carrying twins are the most susceptible. Cows often have access to good pastures in the summer months and can get overfat. If the same cows do not have access to good quality forage during the winter months, when they are in late pregnancy they will succumb to ketosis because of the deficit in energy intake. In dairy cows the problem can occur at or around calving and is again the result of insufficient energy intake in excessively fat animals.

### **Signs**

The severity of the clinical signs and their speed of onset are associated with the stage of pregnancy and the degree of nutritional stress. Affected cows are usually seven to nine months pregnant and show the same clinical signs as cows with acetonaemia. They become increasingly dull and depressed and the smell of acetone can be detected on the breath. Many cows become recumbent fairly quickly, within a few days of the onset of hyperketonaemia. Often in poorly supervised herds recumbency is the first sign noticed by the stockworker. Recumbent cows are severely depressed, have an increased respiratory rate and faeces are scanty, hard and covered in mucus. Some cows develop bloodstained or fetid diarrhoea in the terminal stages. Most cows die three to fourteen days after recumbency, having fallen into lateral recumbency. This often occurs two to five days after sternal recumbency. Cows affected close to parturition often die during parturition.

## Clinical pathology

Hypoglycaemia, hyperketonaemia and ketonuria are consistent findings. In recumbent cases the blood levels of bHB are much higher than in acetonaemia; levels up to 22 mmol/l (125 mg/100 ml) may be found. Cows affected close to parturition have hypocalcaemia and occasionally hypomagnesaemia. Recumbent cows in the terminal stages have hyperphosphataemia (up to 6.5 mmol/l; 20 mg/100 ml), hyperglycaemia (up to 9.0 mmol/l; 160 mg/100 ml) and raised AST levels. At post mortem the most consistent findings are an enlarged, yellow, fatty liver with fatty changes in the kidney and adrenal cortex.

## Diagnosis

The history, stage of pregnancy and the nutritional status will usually be enough to enable a tentative diagnosis. Raised blood or urine ketone levels and low blood glucose (plus low calcium in cows close to calving) will usually confirm the diagnosis.

## Treatment

Treatment as described under acetonaemia would normally be indicated. However, so severely affected are the majority of these cows that medical treatments almost invariably fail to succeed. Immediate removal of the calf by Caesarean section may save a valuable cow. This should be followed by the full course of treatment described under acetonaemia.

## Prevention

Although the problem is more common in fat cows it is essentially the result of starvation and is predominant in years when insufficient conserved fodder has been made. To prevent further cases developing and becoming recumbent a supply of good quality forage is essential.