# Post parturient metabolic diseases of cattle

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Extensive use of exotic germplasm and cross breeding programmes has, definitely improved the production potential of dairy cattle. The elevation in the milk produ ction potential demanded an alteration in the feeding pattern also. Feeding irregul arities and malnutrition during pregnancy, followed by sudden demand of metab olites, immediately after parturition results in negative metabolism and high occurrence of metabolic disorders in crossbred cattle.

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**1. Milk fever (Hypocalcaemic parturient paresis)**

Usually occur with in 48 hours of calving

It is important to identify and treat the affected animals during the first stage of the disease itself. This stage is characterized by the clinical signs like anorexia, agalactia, rumen stasis, scanty faeces, excitement, protrusion of tongue, grinding of teeth and muscle tremors of head and limbs.

Give the correct dose of calcium borogluconate (25 per cent solution) 1.5 ml/ kg body weight as slow intra venous injection. Hefty animals may require two bottles of calcium. Sub normal doses can lead to downer cow syndrome.

Ascertain whether any previous subcutaneous calcium therapy was administered to the animal during the course of illness. If calcium is already available subcutaneously, subsequent administration of I/V calcium will result in increased blood circulation, absorption of the depot calcium and finally calcium toxicity.

If signs of excitement and hyperaesthesia are persisting beyond the first stage of disease, give magnesium along with calcium.

Auscultate the heart during the I/V administration of calcium and magnesium. If any arrhythmia is noted, temporarily stop the administration.

Calcium toxicity can be counteracted by using atropine sulphate, I/V or S/C.

Test the urine for ketone bodies. If concurrent ketosis is noted, it should also be treated.

Temporary response to calcium and recurrent recumbency after repeated calcium injections are indications to use inorganic phosphorous preparation (eg Alphos-40) along with calcium. However it is not wise to administer more than three doses of calcium consecutively during the course of milk fever, due to the risk of developing myocardial aesthenia and death.

In recurrent milk fever cases as a last resort, udder insufflation technique can also be employed to decrease the milk production.

Even after correcting the calcium deficit, if the animal is reluctant to stand by its own, rectal enema using warm water will help to enhance the circulation to the pelvis and there is increased chance for the animal to get up.

**2. Downer cow syndrome**

Usually occur following milk fever and it is characterized by prolonged recumbency even after two successive treatments with calcium.

Collect a detailed history to identify the cause.

Enquire regarding a difficult parturition, delayed calcium therapy, slippery floors, chronic carbohydrate feeding, recent epidural injection etc.

Collect all the available clinical materials like citrated blood (for haemogram and leukogram), serum (for calcium, magnesium, phosphorous, potassium, glucose estimations and liver function tests) and urine (for ketone bodies, albumin and glucose).

Animal should be given proper bedding/inflatable bags. Frequent turning of the animal, massaging, flexion and extension of the limbs etc., should be practiced. Use proper hip slings, or body slings to hoist the animal.

Identify inflammatory conditions if any like peritonitis, mastitis, metritis etc., and treat them using proper antibiotics.

Correct the metabolic deficits. Glucose, magnesium, phosphorous, potassium (kcl orally) etc., can be supplemented.

Fluid therapy – Balanced electrolytes (eg. Ringers lactate) and 5 per cent DNS I/V.

Analgesics and anti-inflammatory agents can be given. One of the important non-steroidal anti-inflammatory agent is Flunixine-meglumine (eg. Finadyne) 1.1 mg/kg body weight, I/V or I/M.

The only therapeutic agent with specific indication for use in downer cow is Tripelenamin hydrochloride (eg. Vetibenzamine). However it is not available in India.

Once the animal is able to stand, put an '8' knot on the hind limbs to prevent straddling of legs and further falling.

**Predicting the prognosis in downer cows**

Favourable prognosis in – sternal recumbency, creeper cows, frog legged posture and alert downers.

Unfavourable in - non-alert downers, lateral recumbency, recumbent for more than 10 days, hind-legs extended behind the animal, hind limbs extended rostrally up to the elbow of the front limbs and increased rate and decreased strength of pulse.

**3. Hypomagnesaemic tetany**

Hypomagnaesaemia, usually coupled with hypocalcaemia is the cause for tetanic convulsions in this condition.

First aid – Magnesium chloride 60g in 200 ml water as enema. This will result in an immediate relief.

Hyperaesthaesia and tetany should be controlled by using sedatives.

Solutions containing magnesium and calcium (eg. Mifex) should be given as slow I/V injection.

**Or** 25 per cent calcium borogluconate I/V and 20 per cent magnesium sulphate 200 ml S/C.

Parentral therapy should be followed by oral magnesium supplementation to avoid recurrence. Calcinated magnesite 50-60g daily for 10-14 days is ideal. Otherwise use $MgSo_4$ orally.

**4. Ketosis**

This is a disease of negative energy metabolism after calving, characterized by wasting or nervous forms.

Acertain whether the condition is primary or secondary ketosis.

Conditions causing secondary ketosis such as left side displacement of abomasum, metritis, pneumonia etc., should be diagnosed and treated along with glucose supplementation intravenously.

Dextrose 25 per cent 540 ml I/V daily for 2-3 days is curative.

In case of dehydrated animals with ketosis, use 5 per cent dextrose solution I/V.

Dextrose parentally should be followed by oral hyperglgcaemic agents, like propylene glycol (eg. Glucafed, ketonil etc.,) or glycerol 125-250 ml BID.

For animals with recurrent ketosis, 25 per cent dextrose solution 540 ml + Bovine plain insulin (0.5 units/kg body wt) intravenously can be given. Keep a spare 25 per cent dextrose solution in order to use it, if there is any hypoglycaemic signs like convulsions.

Use of corticosteroid is controversial. Cortico steroids will cause an initial reduction in blood glucose and exacerbate the signs in nervous ketosis. In fatty animals coricosteroid therapy can aggravate the ketone body production due to its property to enhance the gluconeogenesis from the depot fat.

Even though Jaggery can be given in under feeding ketosis, it is not indicated in alimentary ketosis because it contain higher quantities of butyric acid and can aggravate the condition.

**4. Hepatic lipidosis**

Hepatic lipidosis is associated with the following disease conditions.

Fatty liver syndrome or chronic fat mobilization syndrome (CFM) – occur in dairy cows after parturition.

Ketosis of pregnant cows (Pregnancy toxaemia of cattle).

Fat cow syndrome – only occurring in fatty cows before or after parturition.

The common feature in all these conditions are hypoglycaemia, ketonaemia, ketonuria and fat accumulation in the internal organs especially in the liver. In some severe cases, hyperglycaemia and glycosuria can be seen.

The clinical signs commonly encountered are anorexia, reduced milk yield in lactating cows, progressive debility, inco-ordination, nervous signs and

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