Diseases of the Rumen, Reticulum, Omasum, Abomasum.

Simple indigestion

Occur commonly in hand feed ruminant and it is characterized clinically by inappetance to anorexia, decrease ruminal movement and abnormal feces (dry, scant or diarrhea) .

Etiology:

1- It is common in dairy cattle & stall fed beef cattle because of variability in quality & the large amount of food legumes and this disease not commonly in pasture beef cattle &sheep.
2- Minor degree including indigestible raphage particularly when the protein intake is low.
3- Moldy over heated and frosted feed & pica.
4- Sudden change to a new source of grain & concentrated food intake.
5- Limitation of the available drinking water.
6- Prolonged or heavy oral dosing with antimicrobial lead to inhibition of ruminant flora.

Clinical findings:

1- Reduction in appetite in the first signs followed by the decrease in milk production both occur suddenly.
2- Anorexia may be partial or complete.
3- Mild depression &dullness.
4- Ruminal a tony & ruminal movement depress or stasis.
5- Feces reduced in quantity & then later diarrhea feces.
6- No systemic reaction (H.R, R.R, Temp.) all of this normal.
7- Most cases recover spontaneously or with simple treatment in about 24 hours.

Differential diagnosis :

1- Ketosis 2- Vagus indigestion . 3- Abomasum displacement.
4- Acute carbohydrate engorgement. 5- Traumatic reticulo pericarditis .(T. R. P.)

Treatment :

1- Rumenatorics drugs :
- Tarter emetic at dose 10-12 gm orally in cattle.
- Neostigmine at dose 2.5 mg/ 45 kg of B.W.

2- Magnesium sulfate at dose 500-1000 gm. orally (purgative & relax of muscle).
3- Magnesium bicarbonate 400 gm./450 kg (Alkalizer) .
4- Administration of fresh flora by stomach tube (Ruminal fluid) & give normal saline orally if the rumen contents are dry..
5- Give the Acids such as acetic acid or vinegar 5-10 liter/animal if the reaction is alkalinity.

Acute Carbohydrate Engorgement

(Ruminal Lactic Acidosis or Rumen Overload)

The disease occur due to ingestion of large amount of highly fermentable carbohydrate and lead to in acute cases to excessive of production lactic acid in lumen and cause sever toxemia , dehydration , ruminal stasis and weakness, recumbency and high mortality rate .

Etiology:

The sudden ingestion toxic doses of carbohydrate-rich feed such as grain, apples, grapes, bread, sugar beet, rice.

Pathogenesis:

The excessive quantities ingestion of highly fermentable feed is followed within 2-6 hours by a marked change in the microbial population in rumen, there is increase in the number of streptococcus bovis which utilize the carbohydrate to produce large quantities of lactic acid lead to decrease the rumen PH to 5 or less. which result the destruction of the cellulolytic bacteria and protozoa. And the concentration of the volatile fatty acid increases initially and this lead to fall in ruminal PH. The low PH allows lactobacilli to use the large quantities of carbohydrate in the rumen to produce excessive quantity of lactic acid resulting ruminal lactic acidosis. .

Both D & L forms of the acid are produced which markedly increase ruminal osmolality and water is drawn in from the systemic circulation, causing hemoconcentration and dehydration, and when the ruminal PH decreased at about 5 causing ruminal a tony and this lead to complete ruminal stasis.

Systemically the blood pressure declines causing a decrease in oxygen supply to peripheral tissue. The renal blood flow and glomerular filtration rate also decreased resulting in anuria eventually there is shock and death. All of these events can occur within 24 hours.

Clinical findings:

The speed of severity the disease varies with the nature of the feed being more rapid with ground feed than whole grain.

1- Depression, dehydration, inactivity, weakness and abnormal abdominal distention.
2- The temperature is usually below normal 36.5 – 38.5 C^{o}.
3- The heart rate increased to the 120 – 140 beat/minute.
4- The respiratory rate is usually increased to 60 – 90 /minute.
5- Diarrhea is almost present and usually profuse and light in color.
6- The dehydration is sever and progressive & anuria occur.
7- Ruminal is firm & doughy and present of gurgling sound .
8- Acute laminitis may occur.
9- Recumbency and death occur through 72 hours.

Clinical pathology :

1- The PH of the ruminal fluid is acidic (↓5).
2- Absence of ruminal protozoa.
3- Predominantly gram –positive bacterial flora in the rumen.
4- Hemoconcentration reaches to the 50 – 60 from normal 30-32.
5- The urine PH falls to about 5 & terminally there is anuria.

Differential diagnosis:

1- Parturient paresis.
2- Simple indigestion.
3- Acute diffuse peritonitis.
4- Tampany.

Treatment:

The principles of treatment are:

1- Correct the ruminal and systemic acidosis and prevent production of lactic acid.
2- Restore fluid and electrolyte losses and maintain circulation blood volume.
3- Restore of stomach and intestinal motility to normal.

* All of this process completed from through: -

- prevent the animal excess to feed.
- Do not provide any water for 12 – 24 hours.
- Supply a good feed.
- Exercise all animals every hours for 12-24 hours.
- Give sodium bicarbonate 2-5 litters to cows in 30 minute .
- Fluid therapy at dose in mild (10-20 ml/kg), moderate (40-50ml/kg), sever (100-110 ml/kg).
- Give the Anti-histamine to return of rumen motility.
- Give the corticosteroids (Dexamethasone) in shock cases & calcium boroglocornate.
- Oral administration of antibiotic (Tetracycline, Penicillin, Sulphamycine).
- Rumenatomy in sever cases.