BACILLUS CEREUS

Definition:

It`s a sporadic highly fetal bacterial disease affect mainly racing camels characterized by endotoxemic signs.

Synonyms:

Endotoxemia, bacillus cereus, hemorrhagic diathesis or hemorrhagic disease. (Wernery and Kaaden, 1995).

Cause:

Gram negative bacteria constituting the normal flora of gastro intestinal tract of the camel provide a potential pool of endotoxins.

Endotoxins are (lipopolysaccharides) consist of 3 parts:

1- Lipid A: in cell wall it made most of toxic effect.
2- O region: give antigenic specificity and it`s highly variable between species.
3- Core region: act as a link between the inner A lipid and outer (O) region.

This endotoxins are highly toxic and lethal even though in very low concentration (10^{-9})g/ml. it`s a chemical stable, also boiling can`t destroy it and not altered by gastric acids or enzymes.

Predisposing factors:

High temperature and humidity in the summer season and changes of the diet from low quality fiber(that adapted with camel`s stomach) to high concentrate ration specially at the beginning of the camel racing season, that lead to impairment of normal rumen fermentation and destroy the flora and so high drop of rumen PH and formation of lactic acid acidosis, that is a good environment for rapid proliferation of bacillus cereus to leaks of endotoxins lead to endotoxemia and sometimes shock due to destroy of high number of bacilli

Pathogenesis:

After absorption of endotoxin from small intestine circulate in the blood and reach to liver to detoxify so if the liver efficacy not good or the endotoxins with high concentration so can`t neutralize all endotoxin and so lead to (endotoxemia).

With sever consequences of the disease the endotoxin cause wide spread of vascular endothelial damage and lead to hemorrhage.

Also endotoxins activate the clotting factor and cause DIC (disseminated intravascular coagulation).

First (24 – 48 hr) of disease characterized by dramatic drop of WBC about (2.1×10^3/L) then begin to rise up to (26×10^3/L) 2 – 4 days later.

Susceptibility:

Racing camel 2 – 4 years old (80%)and may be younger in age.

Clinical signs:

- Fever may be reach 41°C.
- Depression, dullness and in appetence.
- Some camels develop a cough and swelling of the throat accompanied by a marked uni or bi lateral enlargement of lymph node for this reason derived the name of (Abu El nohor) for this disease between Bedouins in Arab peninsula.
- Injected mucous membrane this is so obvious in conjunctiva of the eye.
- Complete atonia of first compartment of stomach with abdominal pain and regurgitation observed.
- Rectal examination reveals normally formed balls of stool that are covered in fresh or tar-like blood (melena). Only few camels develop diarrhea (Manefield and Tinson, 1996).
- The affect camel die between 3rd and 7th day. Two or 3 days before death, the animal become recumbent. Some camels develop central nervous system disturbances at end of the disease, lacrimation and hyper salivation.

Postmortem diagnosis:

- Bleeding into organs and intestinal tract specially in colon.
- Ecchymotic hemorrhages of varying severity are seen in pharynx and trachea and some camels show ulcer in trachea, epicardium and subendocardium, abomasums with ulcer formation, intestinal tract and renal pelvis of the kidney.
- Interstitial hemorrhage of the lung.

Laboratory diagnosis:

Histopathology:

- Bacteriomas of different bacteria types due to damage of immune system.
- Micro thrombosis in kidney lead to renal failure.
- Fatty degeneration of liver.
- Necrosis of lymph nodes

Clinical pathology:

Table show blood parameter and serum enzymes of camels with endotoxicosis, blood taking (1 – 2 days) and (3 – 4 days) after onset of disease, (Wernery et al. , 1999).

Parameters	Unit	Normal value	1 – 2 days average	3 – 4 days average
WBC	$\times 10^3$ /L	6 – 13.5	2.1	21.2
Neutphils	%	50 – 60	67	80.6
Lymphocytes	%	30 – 45	26	14.6
Monocytes	%	2 – 8	6	4.8
Eosinophils	%	0 – 6	1	0
Basophils	%	0 – 2	0.5	0
RBC	$\times 10^6$ /L	7.5 – 12	8.56	8.54
Hb	g/dL	12 – 15	11.54	11.24
Platlets	$\times 10^3$ /L	350 – 450	168.8	286.6
CK	Iu/L	40 – 120	70.4	465.2
AST	Iu/L	60 – 120	102.8	337.6
LDH	Iu/L	400 – 775	338.4	1197.8
Glucose	mg/dL	70 – 110	49.2	98
Urea	mg/dL	3 – 21	21.8	104
creatinine	mg/dL	0 – 2.2	2	6.26

From this table we will see that dramatic drop of WBC (leucopenia) is a typical for endotoxemia due to decrease of neutrophils and lymphocytes, this persist for 1 – 2 days and then reversed with overshooting reaction after 3rd day reach up to (26×10^3 /L).

Sharp rise of serum enzymes specially with developing of the disease due to toxic damages to organs and that is seen histologically.

This table show blood parameters and serum enzymes of a dromedary that survived endotoxemia.

Parameters	Unit	Normal value	Day1	Day2	Day3	Day6	Day11	Day25
WBC	$\times 10^3$ /L	6 – 13.5	1	1.5	5.2	22.4	20.8	9.9
Neutphils	%	50 – 60	82	84	92	84	78	61
Lymphocytes	%	30 – 45	12	10	7	10	16	32
Monocytes	%	2 – 8	4	5	1	4	5	4
Eosinophils	%	0 – 6	2	0	0	2	1	3
Basophils	%	0 – 2	0	1	0	0	0	0
RBC	$\times 10^6$ /L	7.5 – 12	8.3	9.1	7.6	8.3	7.2	7.6
Hb	g/dL	12 – 15	12.2	13.5	12.2	11.5	10.1	11.1
Platlets	$\times 10^3$ /L	350 – 450	176	140	193	251	301	483
CK	Iu/L	40 – 120	67	112	721	882	324	140
AST	Iu/L	60 – 120	196	380	475	680	232	160
LDH	Iu/L	400 – 775	338	790	819	1083	660	379
Glucose	mg/dL	70 – 110	46	38	44	70	78	108
Urea	mg/dL	3 – 21	13	23	51	58	34	22
creatinine	mg/dL	0 – 2.2	1.7	3.1	3.8	4.1	2.6	1.6

Treatment:

Treatment and prognosis depending mainly on the rapid discovery of the disease and it concentrate on 6 basic line:

1- Bacteriostatic antibiotic + antipyretic, e.g (R/ Hexazol).
2- Antiacid + probiotic, e.g (R/Laxavet, Bykodigest)(R/interlac paste)
3- Vitamin B1 + Bcomplex, e.g (R/T500B1 + compivet)
4- Fluid theraby, e.g (R/ringer lactate).
5- Liver treatment , e.g (R/methioB12).
6- Anti ulcers, e.g (R/gastrozol paste).

Control and prevention:

- Avoiding predisposing factors like changes in diet and using of large amount of crushed barley , honey, cow milk and dates.
- Avoiding excessive training of young racing camels specially within one years old.

- **Vaccination:**

The best way to control this dangerous disease is vaccination against endotoxins of gram negative bacteria, and can use vaccine against many types of endotoxin to get wide range of protection.

R/endovac-Bovi

This is vaccination against endotoxins of E.coli mastitis and has also been tried against endotoxicosis in camel. It enhance both of B and T lymphocytes.

Also can use with combination with:

R/Re-17 bacterin

Its vaccine against many types of endotoxins of gram negative bacteria, it`s seem to protect against other endotoxin mediated diseases although its efficacy has not broven in camel yet.

References:

1- Infectious diseases in camelids, Ulrich Wernery, O.-R. Kaaden, 2nd edition.
2- Camels a compendium, Geoffrey W. Manefield, Alexander H. Tinson, university of Sydney.
3- Medicine and surgery of camelids, 3rd edition, Murray E. Fowler.