BOTULISUM

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Bacteriology.

It is a rapidly fatal, moral paralysis caused by the ingestion of the toxin of the Clostridium Botulium; organism proliferates in decomposing animal matter and some times in plant material.

It is large sporulated motile bacillus by means of peritrichus flagella, with rounded ends. It is 1µ x 4µ length but many forms are recorded up to 8µ, motile, single or pairs seen. Spores are oval or terminal, it has capsule. This is gram positive and non acid-fast organism. Grow best at 30°C to 35°C Poor at 37 °c. 02 relation - fastidious 35°C good 37°C poor Disease food poisoning, limber neck.

ETIOLOGY:

The causative organism is cl. Botulinum, a spore forming anaerobe, which proliferates only in decaying animal or plant material. There are a number of antigenically distinct types of cl. Botulinum classified as A, B, C, D, And E. The geographical*distribution of these types varies considerably under favorable conditions of warmth and moisture. The spores multiply rapidly elaborating a stable and highly lethal toxin which when injected causes the disease.

DISTRIBUTION:

The spores are very widely distributed all over the world. The organism is supposed to have the following types- type A, B, C, D, and E but type D and E are thought to be identical to type C. Type - A spores are more prevalent in virgin type of soils and in far off the forest areas or on the mountain tops. Type - B is very commonly seen in the soils that are heavily manures. The organisms in it self is not pathogenic, but under the anaerobic conditions it is capable of producing a very powerful toxin, which is contaminated with the spores or the organisms, which liberate this toxin in the foods.

CLASSIFICATION OF TOXINS:
The organisms forming powerful exotoxins almost entirely belong to this group anaerobic spore forming bacteria. It is water-soluble toxin, and not alcohol or ether soluble. Toxin has special affinity towards nerves tissues. The toxin is thermo labile. Its pathogenic activity is determined by the formation of toxin in the food substances prior to their ingestion. Clostridium botulinum 0.0084 μg would have lethal action on man. It kills the animal in smallest dose 0.0001 cc kills the guinea pig in short time.

1. TOXIN FORMING NONINVASIVE GROUP

Clostridium Tetani, CL. Botulinum And CL. Para botulinum.

2. TISSUE INVADING GROUP

Clostridium Chauvoei, CL. Oedematiens, CL. Welchii, CL. Haemolyticum.

Toxin free spores or organisms are non pathogenic.

All the types of toxin produced by it are capable of producing death in guinea pig, rabbit, mice, mink and rats. Toxin Type A and B produces botulism in man.

Toxin Type A AND C produces limber neck in chickens Toxin Type D causes lamziekte in cattle.

EPIDEMIOLOGY:

The spores of Clostridium botulinum are extremely resistant and survive on long periods in most environmental circumstances. The toxin is also capable of serving for long periods particularly in bones or if protected from leaching. In its vegetative form the organism is a common inhabitant of the elementary tract of the herbivores and may be introduced in to new areas in this way. It occurs commonly in soils in affected areas and soil and water contamination occurs from feces and decomposing carcasses. Careful study of soils is necessary to determine the conditions whichever the presence of cl. Botulinum showed regional distribution for the various types in the United States.

Type A was found in neutral or alkaline soils in the west,
Type B and E were in damp or wet soil all over except that B was not found in south.

Type C was found in acid soils in the gulf coast, and Type D was found in alkaline soils in the west.

Microorganisms capable of inhabiting cl. Botulinum were present, with or without the Clostridia in many soils. The source of infection for animals is almost always canyon which includes domestic and wilds animals and birds, where cattle subsist on a phosphorus deficient diet and manifest osteophagia and the ingestion of carrion. The botulism disease likely to occur in outbreak form.

In sheep pica is more usually associated with a dietary deficiency of protein or net energy. Occasional outbreaks occur due to drinking of water contaminated by carcasses of dead animals, a not uncommon occurrence is in livestock drinking lake water contaminated by the carcasses of ducks and other water fowl. Which have died of botulism. The disease has also occurred in horses fed on spoiled vegetables and potatoes contaminated by cl. Botulinum. Dead rodents in hay stocks or ensilage pits may provide a source of toxin chicken manure used as cattle feed or postural fertilizer and chicken house liter used as bedding in cattle loose housing have also caused mortality when the organism was present in the intestinal tract of the birds. Dried poultry waste is a very important source of protein supplement in some countries but poisoning due to botulinum has caused very heavy losses after its use. Factors which affect the toxicity of the product are the efficiency of the heat sterilizing used in making it, and the amount fed. Although decomposing animal carcasses are by far the commonest source of toxin, proliferation of the organism can occur in decaying vegetable material. Decaying grass at the base of old tussocks and in trampled stubble is reputed to suitable sites for growth of cl. Botulinum. Silage and hay may spoil to a stage suitable for the growth of cl. botulinum. This is most likely if the forage is very succulent or is wet by rain.

When it is made. Grass clippings allowed to accumulate and decay in pile have poisoned horses. Grass in silage, provided the Ph is not too low, can be a suitable substrate for the growth of the organism and so provides a large amount of toxin in the diet. The use of wilted grass to make the ensilage may
be a critical factor in the epidemiological of the disease in all species and
Outbreaks in horses have been related to the feeding of big bale silage and
of hay polluted by rodent carcasses. This ability to grow in such material may
be limited to the proteolytic strains of cl. Botulinum type A and B.

INCUBATION PERIOD:

The long incubation is period of two to seven days. In per acute cases a
cow may become paralyze within few hours. In more slow developing cases
paralysis often begin in the four quarters and proceeds to the hind quarter,
the head and the neck.

INFECTIVITY:

The disease is intoxication rather than an infection the disease is not
infectious or contagious. It cannot transmit from one animal to other. The
bacteria can be transmitted from one lot moldy material to another and latter,
if eaten many also cause botulism.

SUSCEPTIBLE ANIMALS:

Poultry, horse, cattle, pigs and sheep are affected. All animals are liable to
the condition if they happen to eat the toxin containing food.

VIABILITY OF BACTERIA:

As the germ itself does not live in the living animals but upon dead organic
matter, it will continue its life cycle indefinitely without the presence of the
domestic animals.

Hence on farm where the infection has once occurred some caution should
be observed.
SEASONAL INCIDENCES:

The germ grows best under muggy conditions, which usually occur in the summer.

SIGN AND SYMPTOMS:

Clinical signs of the botulism are those of symmetrical bulbar paralysis. Dump rabies or curare poisoning Clinical sign usually occur 6 to 96 hours after ingestion of the infected material.

SYMPTOMS INCLUDE:

Stragring gait, extension of head when lying down (limberneck) increasing paralysis. Ocular paralysis (unequal dilatation of pupil) is common. Temp. Normal or subnormal thirst and pharyngeal paralysis with loss of voice there is dyspnea with concurrent flaccidity of back and abdominal muscles. Death from aspexia by paralysis of respiratory muscles may occur within 24 hours Disease is usually fatal once symptoms occur. In some out breaks the main symptom is an increase in flaccid paralysis of the legs first of hindquarter and then extending to involve finally the thorax, neck and throat. It is notable that fodder carrying botulism toxin, which may produce acute cases in horses, may produce only sub acute or mild cases in cattle. The animal may drool saliva from the mouth and have half chewed food or lying in rope like masses out of the gullet due to paralysis of throat and surrounding structure. Tongue may paralyze and protrude between the incisor teeth. Simmons and tammemagi (1964) Note that the earliest and the most common sign of botulism were adipose (absence of thirst and refusal of water) followed by anorexia or vice a versa.
CHRONIC BOTULISM SYMPTOMS:

The water and feed intake is less or longer period often weeks. Restlessness, circling difficulty in rising, arching the back, urinary inconvenience. Salivation, prolapsed of tongue, grinding the teeth, camping the jaws and groaning.

In addition to the usual symptom of phosphorus deficiency, softening of bones and general un thriftiness, cattle also develop pica, deprived of appetite as the result of which they eat various objects that ordinarily they ignore such as clothes, paper, sticks, and especially the carcasses of the small animals.

PATHOGENESIS:

Botulism is produced only after the organism has grown in favorable food material where the toxin has been eliminated although vegetable, fruit and animal foods may be involved. Animal food has found to be most important in USA.

There are no significant lesions produced by outline. General hyperemia is constant and microscopic thrombosis is founds in some blood vessels.

Hemorrhages may be observed in lung. The pathological findings in botulism in cattle are usually few. Marked hyperemia of blood vessels with edematous dilation of perivascular spaces. The medulla Oblongata was most common site of the perivascular hemorrhages. Demyelinization was some tine found.

Pamakcu considered that, the distribution of the pathological changes in the central nervous system corresponded well with the sign of disease, but he concluded that the pathological changes could not be inter prated to completely explain the symptoms.

DIAGNOSIS:

A positive diagnosis depends on isolating the toxin from the bowel content.

This is quite often impossible and following features assist the clinical diagnosis.

1. History of deficiency of phosphate or protein and access to contaminated bones, feed or decaying carcasses or other matter.
   1. Typical symptoms in affected animals.
   2. Absence of lesions of other diseases at post mortem.
Botulism can be confused with after drought weakness, grass tree and zamia poisoning, mechanical injuries and broken bones of the spine, post parturient paralysis and impaction of the stomach, particularly in the third stomach.

Confirmation by laboratory examination a negative lab finding doesn't eliminate botulism as the cause of mortality Diagnosis of botulism confirmed by demonstration of type c toxin in liver and bowel contents of the animal.

**LABORATORY TEST:**

A small portion of the sample should be heated in boiling water or ten minute to give a check on heat labiality. Mixtures of material should be made with various antitoxins and held at room temperature for thirty minutes. Two mice are inoculated IP- intraperitonially with the toxin and antitoxin mixture other mice are inoculated with an equivalent sample of the boiled control sample. Where mouse death is very rapid with potent toxin.

Dilute ten fold and inject in to other mice. Mice may die within 6 to 8 hour but should be held for at least four days. Guinea pigs may be used. In g. pig muscles of back and abdomen become increasingly flaccid and increase salivation noticed. There is paralysis of tongue and g.pig is breathless with coastal breathing.

Local working veterinarian must give his own clear observations in every case A. specific selection of villages from the reported area, map of the village be prepared.

**B. COLLECTION OF MATERIAL FROM SELECTED VILLAGES:**


**C. ANIMAL AILING CASES / CLINICAL CASES :**


**D. DEAD ANIMAL/AUTOPSY MATERIAL:**

1. Heart blood smears 2. Other organ impression smears 3. Intestinal content in sterile vial, 4. Intestinal loop piece in sterile vial, 5. Spinal cord piece in sterile vial, 6. Cultures of heart blood, intestinal tract content and
other organs on rcmn, 7. for histo pathological study collect all tissue pieces 2 to 3 cm size (includes half diseased and half healthy) lung, liver, heart, kidney, spleen, spinal cord, brain, intestine small portion, other necessary organs showing the changes.

**CONTROL:**

1. Improved nutrition either by improved pastures or by supplementary feeding. Diet containing bone meal or phosphate containing material.

   1. Adding soluble phosphate in drinking water.

   4. Phosphorus and calcium suplimate in some area are sufficient but in other areas addition of trace minerals such as cobalt, copper, manganese, zinc and others may be necessary.

   5. Once botulism has occurred moldy feed stuffs should be avoided.

   1. Toxic water should be treated by adding one kg fresh lime to every 200 liters of water.

   2. Control of botulism can be primarily by immunization.

**CONTROL OF DISEASE FOLLOWING VACCINATION:**

Effective vaccine first produced by the South African workers Protection is so reliable that the vaccine is widely used in lamsiekte areas, more than five million doses being used annually in South Africa. Mass vaccination programmees have also been used in Australia where bivalent (c & d) vaccine give best protection. Botulinum vaccine (bivalent) containing both type c and d is in common use. It is a formalize treated product produced from cl. botulinum type c and d. adsorbs on aluminum phosphate it gives an immunity in 14 days, which increases to about four weeks. A booster dose after four to six week will augment. The doses are given s/c. And usually 5 ml for cattle and 2ml for sheep s/c on neck. Do not inject deeper than s/c an annual repeat dose should be given the best time to give the first injection is about six weeks before the anticipated risk periods. Vaccination either single or double with the bivalent C and D type of toxide, produce immunity, which was effective for at least 24 months against the toxin in the field.
DISPOSAL OF THE CARCASS:

Burial or the burning method as far as kokan region is concerned it is rather difficult to bury the carcass as the nature of the soil in the area is concerned. Soil is of rock in nature. It is easier to burn the carcass irrespective of rains during the monsoon. Sufficient grants for burning of the carcass are necessary. Coordination of local peoples, sarpanch, police patil and other concerned are necessary.

TREATMENT:

It is usually of the little value Do not overlook the fact that the animal can not swallow, it is necessary to give water by stomach tube in appropriate amount.

Constipation should be treated with purgative, Epson salt - 750-gram powder, which also assist in, eliminate toxin. A full dose of general poisoning antidote worthwhile. Acid food helps to destroy the toxin in intestinal tract.

Continue with the symptomatic treatment like anti histamine, calcium borogluconate, glucose saline etc. Antitoxin if available is the logical treatment if injected early. If there is animals available that have been vaccinated with toxide, blood transfusion or serum from such animal may be of help, as it will carry certain amount of antitoxin.

IMMUNITY:

Antitoxin can be prepared which neutralizes cl. Botulinum toxin. The antitoxin is active for the homologue strains only, a fact, which must be considered in treatment. Experimentally it has been found that antitoxin will prevent death when given immediately after injection of the toxin. No out standing results has been obtained, by using antitoxin in the field cases. Botulism antitoxin has been used extensively in all types of forages poisoning in horses, particularly in cornstalk poisoning.
## DETAILS ABOUT DIFFERENTIAL DIAGNOSIS WITH FOOD POISONING

<table>
<thead>
<tr>
<th>Sr. No</th>
<th>PARTICULARS</th>
<th>BOTULISM</th>
<th>FOOD INFECTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Cause</td>
<td>Botulinum toxin</td>
<td>Bacilli of the paratyphoid enteritidis group</td>
</tr>
<tr>
<td>2.</td>
<td>Fever</td>
<td>Not characteristic, temp.</td>
<td>Characteristic, acute</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Usually subnormal</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Occurring</td>
<td>Mainly in the winter</td>
<td>Mainly in the summer</td>
</tr>
<tr>
<td>4.</td>
<td>Associated</td>
<td>Preserved foods</td>
<td>Fresh foods or the freshly contaminated foods, usually meat or milk.</td>
</tr>
<tr>
<td></td>
<td>With</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Condition of the Bowel</td>
<td>Constipation, rarely diarrhea</td>
<td>Diarrhea offensive</td>
</tr>
<tr>
<td>6.</td>
<td>Visual Disturbances</td>
<td>Double vision, ptosis of lids</td>
<td>Absent</td>
</tr>
<tr>
<td>7.</td>
<td>Abdominal Pain</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>8.</td>
<td>Onset</td>
<td>Usually gradual</td>
<td>Sudden</td>
</tr>
<tr>
<td>9.</td>
<td>Incubation Period</td>
<td>Variable, usually from twelve hours to several days</td>
<td>Short, usually from six twelve hours.</td>
</tr>
<tr>
<td>10.</td>
<td>Throat</td>
<td>Swallowing difficult</td>
<td>Normal</td>
</tr>
<tr>
<td>11.</td>
<td>Treatment</td>
<td>Antitoxin</td>
<td>Systemic</td>
</tr>
<tr>
<td>12.</td>
<td>Mortality</td>
<td>60 to 70 %</td>
<td>1 to 2 %</td>
</tr>
</tbody>
</table>

## REFERENCES:

1. VETERINARY MEDICINE - BLOOD ADERSON  
2. VETERINARY BACTERIOLOGY - MERCHANT AND PACKER  
3. DISEASES OF LIVESTOCK - HUNGER FORD  
4. DEPARTMENT CIRCULARS.