**ANTHRAX**

Anthrax is originated from Greek word Anthrox means coal. Other names for this disease are spleenic fever, woolsorter’s disease. Locally it is called as golle or sut. It is an acute, contagious and septicemic disease. Highly fatal and affecting a wide range of mammalian species including human beings.

Before the availability of an effective vaccine, anthrax was one of the most important causes of death in livestock throughout the world. The results of national epidemiological survey of important diseases of livestock in Pakistan has indicated that anthrax is one of the leading causes of death among sheep, goat cattle in hilly and desert areas.

**Causative Agent:**

Anthrax is caused by a bacterium known as *Bacillus anthracis*. The organism is G +ve, non motile, aerobic, facultative anaerobe and spore forming. There are two forms of this organism: vegetative and spore forming. Vegetative form occurs inside the body of affected animals and is responsible for producing clinical signs and pathological lesions. The spore formation occurs outside the body of host and is the result of exposure of vegetative form to oxygen.

**Transmission:**

Mostly animals are infected while grazing in areas that have previously experienced anthrax.

The spores are also transmitted through the consumption of contaminated water, hay, and fodder.

Eating of bone meal and blood meal of infected animals also cause transmission.

Eating of dry fodder or spiky grass produces lesions in gastrointestinal mucosa, and the chances of infection are increased.

Flies are also a source of transmission.

**Clinical Findings:**

Its incubation period is 1-2 weeks, some says 7 weeks. Most common sign of disease is sudden death. There are three forms of disease:

**Peracute:**

It is most common at the beginning of outbreak. Animals are found dead without signs. Course of disease is only two hours. Signs may be fever, dyspnia, congestion of mucosa and muscle tremor and animal dies after convulsion. After death there is discharge of blood from natural orifices (mouth, nostrils, anus, vulva etc.).

**Acute:**

Course of disease is 48 hours. There is severe depression, increased body temperature upto 107 oF, rapid and deep respiration, and congested mucosal lining.

Pathogenic signs are congestion of mucous membrane, hemorrhage from natural orifices, increased heart rate, animal off feed, ruminal stasis, abortion in pregnant cows, blood stained or deep yellow milk, diarrhea, dysentery, and local edema of tongue.

**Chronic:**

Chronic infection is characterized by localized, subcutaneous, edematous swelling that can be quite extensive. Areas most frequently involved are ventral neck, thorax, and shoulders.

**Diagnosis:**

It is based on the history of the occurrence of disease in an area, clinical signs, and necropsy findings. Sudden death in an animal without prior symptoms should lead to suspicion of anthrax and bloody fluid exuding from the nose and mouth or anus of living or dead animal is particularly suggestive of anthrax.

Postmortem is not allowed in case of anthrax. To prepare blood smear, blood is obtained from ear by giving incision. Blood film should be dried and fixed by heat or immersion for one minute in absolute methanol and stained with polychrome methylene blue. Then it is washed after thirty seconds into hypochlorite solution. After drying the slide, it is examined under microscope for reddish purple capsular material and deep blue Bacilli. This reaction is termed as M- Fadyean reaction.

**Treatment:**

Because this disease is rapid in onset and with large mortality rate (90%), this is insufficient to initiate treatment before death. If anthrax is suspected, segregation of animal should be done. Early supportive and antimicrobial therapy is useful and Bacillus anthracis is highly susceptible to a wide range of antibiotics including benzylpenicillin, tetracycline, and ciprofloxacin. First dose of antibiotic should be administered intravenously and then intramuscularly for 5 days. Prognosis is not favourable and no time to treat the animal.

**Differential Diagnosis:**

In cattle and buffalo differentiate it from acute fatal blot, per acute babesiasis, gross tetany, black quarter, acute poisoning, and enteritis. Anthrax should be considered in differential diagnosis when an animal dies after having observed apparently good health during the preceding 24 hours.

**Control Strategy for Anthrax:**

Control measure is aim to break the cycle of infection. The important thing is to correct the disposal of carcasses. When an animal dies inside a shed, paddle or barn, its carcass should be received for burial or incineration. Plug all the natural orifices properly before disposing carcass. Burial should be away from water supply and pasture. The pit should be at least 180 cm deep. The top layer after burying should be covered with unslacked lime. Decontaminate the area, bedding, unconsumed feed, and room. Dip the equipments in 4 % formaldehyde solution for 12 hours.

**Vaccination:**

Veterinary Research Institute (VRI), Lahore has developed anthrax spore vaccine. It imparts solid immunity for one year. Its dosage in cattle and buffalo is 1 ml subcutaenously. Vaccination should not be done in area where disease does not occur. During vaccination one should not be exposed to vaccine by needle prick.

**Hemorrhagic septicemia**

Hemorrhagic septicemia is one of the most important diseases of cattle and buffalo in Pakistan and causes heavy losses in Livestock. It is considered number one killer of buffalo. HS is caused by two serotypes of G –ve, non motile, coccobacillus bacteria named as ***Pasteurella multocida***.

Disease is associated with humid weather; increase incidence of disease is reported in wet season. It is evident that out breaks do occur in all times of the year but those occurring during wet season spread rapidly because of the longer survival of organism in the moist conditions. The disease is spread by direct or indirect contact. The source of infection is infected animals or carriers. The causative agent does not survive for more than 2-3 weeks in soil or pastures.

**Signs & Symptoms:**

Majority of cases are acute or peracute in nature with death occurring from 6-24 hours in cattle and buffalo after the appearance of signs. Signs include:

* Dullness
* Reluctant to move
* High body temperature
* Serous nasal discharge
* Salivation
* Edematous swelling (starts in throat region and then spread to parotid region and to the neck)
* Mucous membranes are congested
* Difficult respiration and animal dies within few hours

**Diagnosis:**

Field or clinical diagnosis is usually based on history, clinical signs, pathological lesions observed on postmortem, previous occurrence of HS in the herd, endemicity of the area, species affected, and vaccination history.

For lab diagnosis best tissues for smear and cultures are blood (from heart), liver, lungs and spleen. Staining can be done with Giemsa stain and methylene blue stain. Pasteurella grows on common laboratory media like blood agar. Haemagglutination test, detecting somatic Ag, or mouse protection tests, detecting capsular antigen, are more reliable.

**Treatment:**

It is caused by G -ve bacteria. So antibiotics affected for G –ve should be administered. Bactericidal antibiotics should not be administered. In acute HS endotoxins is the main cause of pathological changes so non steroidal anti-inflammatory (dichlophanic Na) and steroids are given which have beneficial effects for stoppage of endotoxins.

**Vaccination program:**

Vaccination is available in Pakistan prepared by NIAB and VRI.

**Mastitis**

Mastitis is a major problem in dairy industry throughout the world. This disease causes huge economic losses to farmers in terms of production and price of milk. Price of dairy animal is based in its milk production. In case of cow front teats produce more milk (60%) than rear teats (40%) and vice versa in case of buffalo. If cow loses its front left or front right teat then there will be a huge economic loss. According to National Mastitis Council (NMC), USA:

*“Mastitis is an inflammation of mammary glands that happens in response to the injury for purpose of neutralizing infectious agent to prepare way for healing and turned to normal function.”*

Normal somatic cell count in milk is 200,000 and in case of mastitis it goes to several thousands. Mastitic milk has 90 % neutrophils and 10% somatic cells. If 40 % neutrophils, there will be mild mastitis while in case of 90 % neutrophils; there will be severe mastitis.

**Effect of Mastitis on Milk**

Mastitis affects the quality of milk and there is increase in the number of bacteria. Common sources of bacteria are inadequate cleaning of milking utensils, hands not properly washed, skin of udder not properly cleaned and contamination of the teat skin. There is a direct relationship between skin and mastitis. If dirty skin then more chances of mastitis. By providing hygienic conditions mastitis can be controlled and quality of milk and its byproducts can be improved. Increased somatic cell count and increased neutrophils and macrophages also deteriorate the quality of milk. Increased somatic cell count claims mastitis. So there is direct relationship between mastitis and somatic cell count. But in late lactation and newly parturating animals, there is also increase in somatic cells. Whenever there is rise in somatic cell count, casein content falls down (which is very important protein). Similarly leakage of certain proteins from serum like albumin, immunoglobulin and transferrin into milk also occurs. There is also an increase in sodium and potassium ions. Calcium level decreases. Normal pH of milk is 6.6 but it may raise upto 6-9 or more in the milk collected from subclinically mastitic animal and even more in clinically mastitic animals. There is release of proteolytic enzyme from blood like plasmin. Plasmin is excessive in blood but low in milk. It cannot be destroyed at 140 oC. If it is high in milk then deteriorate the quality of milk. Watery milk shows chronic type of inflammation. If watery secretion present in first few streaks (about 10 streaks) then normal but if more than it then chronic mastitis. Plaques present in milk show severe infection.

**Types of Mastitis**

**Latent Mastitis:**

Pathological organisms present in milk but no swelling of udder and normal cell count.

**Sub Clinical Mastitis:**

Bacteria and somatic cells present in milk and change in composition of milk but no gross lesions.

**Clinical Mastitis:**

It is divided into three categories depending upon severity:

**(a): Acute:** There are obvious symptoms of inflammation present on udder, change in colour and composition of milk and increased temperature.

**(b): Subacute:** No obvious change in udder but clots and plaques present in the milk.

**(d): Chronic:** Every acute infection develops into chronic infection if not treated. In this phase major changes are fibrosis of udder. There may be fibrotic mass particularly in teat canal.

**Aseptic/Non Specific Mastitis:**

It is due to trauma or injury to the udder.

**Pathogens Involved in Mastitis**

**Contagious Pathogens:**

They spread from quarter to quarter through contamination by hands, flies, wounds etc. They always require host e.g. *S. aureus*, Mycoplasma, Pasteurella. They have very limited life in environment. In Pakistan mastitis caused by *S. aureus* and *Streptococcus agalectia* is 70-80 % of mastitis and rest of it is caused by environment (*E. coli*)

**Environmental Opportunist:**

The primary source is the environment in which the animal lives. They spread by direct contact of the teats to the bedding or mud, dirt and manure. Examples are Coliform species like *E.coli*, Klebsiella, *Streptococcus uberis*, *Streptococcus agalactae*, and *Streptococcus faecalis* etc.

**Opportunist Pathogens:**

This group of mastitis pathogens includes around 30 different species of the genus Staphylococcus (other than *S. aureus*) and *Corynebacterium bovis*. They are normally present on the teat skin and streak canal. Therefore they are in an opportunistic position to colonize the teat canal and penetrate the udder.

**Endogenous Pathogens:**

Etiological agents of systemic diseases with mammary gland involvement like Leptospira, *Mycobacterium bovis* etc.

**Sources of Mastitis**

Hands of milker. Pathogens present on skin, naries of human if no proper bath taken. He will shift from one herd to other. Lack of proper management i.e. proper teat dipping is not carried out, no antiseptic solution is used and no sanitation measures are taken. Trauma during sitting posture or due to kicking udder leads to mastitis. Folded thumb milking particularly in villages damages the teat and causes adhesion and increases the chances of mastitis. In old animals teat canal is fragile and immune system is weak. So there are more chances of infection in old animals.

**Clinical Findings of Mastitis**

There is change in udder size; size increases in acute cases while in chronic cases it decreases due to fibrosis and atrophy. Consistency of udder is soft and hot in acute but hard in chronic due to fibrosis. In case of endogenous spread (like *E.coli*) systemic reaction may occur and cause temperature, anorexia, depression and whenever increase in fever animal is off feed.

**Diagnosis of Mastitis**

**Somatic cell count:**

By counting the number of somatic cells in the milk sample.

**NAGase Assay:**

NAGase (N-acetyl glucosamide) is a lysosomal enzyme. Its level increases due to mastitis which can be detected for the diagnosis of mastitis. Kits to detect are available.

**California Mastitis Test:**

A reagent is used in California Mastitis Test which is alkaline in nature. Whenever mastitis occurs, there will be destruction of leukocytes due to phagocytosis. As a result DNA content increases in milk which is acidic in nature and causes the increase in the acidity of milk. Any alkaline reagent if added, it will neutralize the milk. The reagent added in California mastits has alkyl aryl sulfoxide which will cause the precipitation or gel formation in milk.

**Surf Field Mastitis Test:**

A test discovered by Prof. Dr. Ghulam Muhammad, Department of Clinical Medicine and Surgery, Faculty of Veterinary Science, University of Agriculture, Faisalabad. Make 3 % surf field solution: add 6 teaspoons of surf in half litre water, mix it, filter the solution and heat it. Take milk and add equal volume of 3% solution, swirl this mixture for half minute and then examine for precipitation or gel formation (In case of mastitis). The test solution is stable for 6 months at room temperature. The solution should be shaken well before use.

**Strip Cup Method:**

It is the simplest method. Take few streaks in cups with black background and observe any abnormality e.g. clots.

**Ground Test:**

Take few streaks on ground. If the absorbance of streak is quick in ground then animal is –ve for mastitis but if the absorbance is slow then milk is mastitic. Late absorbance is due to pus as mastitic milk is pus containing milk.

**Measuring Electrical Conductivity of Milk:**

The concentration of sodium and chloride increases in milk as a result of mastitis. These ionic changes together with increase in milk pH and decrease of milk fat lead to increased electrical conductivity of milk. Electrical conductivity measuring can be converted into computer readable signal. Therefore, this method is easily applicable to online automatic monitoring of udder health and can be installed in milking machines. The method however, is not very specific for mastitis.

**Treatment of Mastitis**

We have to target three things:

1. Specific treatment
2. Symptomatic treatment
3. Supportive treatment

First of all determine the nature of mastitis and on the basis of nature of mastitis and its etiological agent select antibiotics. Antibiotics can be administered through intramuscular or intramammary route. Whenever given through intramuscular route the best drugs to be given are macrolides (erythromycin, tylosin), oxytetracyclin, cephlosporin, chlorofluracin, and quinolines (norfloxacin). Tribrissen is also good. The best approach is to give antibiotics through intramammary route. Commercial. Intramammary tubes are also available in market. For subsiding inflammation steroids may be used in acute inflammation otherwise NSAIDS. Vitamins may be used to increase immunity. To enhance immunity, trace elements like zinc, copper, and iodine may also be used. Biotechnological products like Interleuken-1, Interleuken-2, and lysostaphin are also used.

**Udder Toilet:**

It refers to infusing larger quantity of weak antiseptic solution into quarter and withdrawing it. For this purpose acriflavin solution (1:10000 boiled in water) is generally used. Remove milk from the udder and infuse the solution, remain there for 5 minutes and then remove out with the help of syringe.

**Permanent Drying/Blocking of Affected Quarter:**

If quarter does not respond to antibiotic, infuse tincture iodine into that quarter; it will cause irritation and block that quarter permanently.

**Control of Mastitis**

Two main objectives of control:

1. Prevention of new infection in the herd

2. Reduction of duration of existing infection

There are five different plans to control mastitis which were devised by NMC (National Mastitis Council), USA in 1990.

Pre milking teat dipping

Post milking teat dipping

Dry cow therapy

Prompt treatment of clinical cases

Culling of chronic mastitic animals from the herd

**Pre Milking Teat Dipping:**

Dip the teats before milking with the antiseptic. Dry the teats after pre dipping by towel or tissue. Do not use same towel for more than one animal. Teat cups are available having antiseptic in it like iodofores (0.1-1 % iodine). Dip the teats one by one for 2-3 seconds. Quaternary ammonium compounds, chlorhexidine, and sodium hypochlorite may also be used as dip solution.

**Post Milking Teat Dipping:**

Organism is present in environment and teat skin. In order to avoid it we go for post milking dipping. After milking teat sphincter remains open for 30 minutes to 2 hours. It is ideal time for entry of organism to teat canal. So perform teat dipping after milking. Solutions for post milking teat dipping are same as for pre milking teat dipping.

**Dry Cow Therapy:**

The rate of new udder infections increases dramatically shortly after drying off and remains elevated during the first 3 weeks of mammary involution. During the first few days after drying off, the animal goes through a period of stress that may predispose her to infections. Up to 40% of all new intramammary infections are established during the first two weeks of the dry period and without dry cow therapy, 10 to 15% of the quarters will become infected during the dry period. Dry cow treatment is aimed at preventing new infection from occurring during this period of increased susceptibility as well as curing existing infection and is beneficial against both contagious and environmental pathogens. Advantages of dry cow treatment include the following:

The cure rate is higher than during lactation

Higher concentrations of drugs can be used

New infections during the dry period are reduced except first 3 weeks after drying off

Drug residues in milk are avoided

**Prompt Treatment of Clinical Cases:**

Despite implementation of effective mastitis control measures, clinical cases still occur. These cases should be treated promptly to maximize the chances of recovery. Treatment of clinical cases involves intramammary and parenteral administration of antibiotics.

**Culling of Mastitic Animals:**

Cull mastitic animals from herd.

**Managemental Control**

Segregation of healthy and infected animals and milking of healthy animals ahead of infected animals. Cull chronically infected animals. Purchase mastitis-free animals. Mastitis control in heifers. Proper treatment of teat and udder wounds. Fly control. General cleanliness of farm. Proper disposal of mastitic milk of clinical cases. Proper nutrition.

**Black Leg Disease**

This disease is also called Chlostridial myonecrosis, black quarter and quarter ill. It is an important disease of cattle and sheep. It affects skeletal muscles. This condition starts when tissue damage occurs. It is acute bacterial, emphysematous, myonecrotic, highly fatal disease. Wide range of ruminants is susceptible to this condition but cattle and sheep are more susceptible at age of 6 months – 2 years. This disease is more in summer and fall.

**Epidemiology:**

It is widely distributed all over the world occurring in all the countries mostly in hilly areas and sandy regions. This is one of the most important causes of cattle mortality in sandy and hilly regions. According to epidemiological survey it is an economically important disease of livestock. When pasture or grazing grounds once become affected, the disease will reappear regularly in susceptible animals year after year.

**Etiology:**

This disease is caused by *Cl. Chouvei* which is G +ve, spore forming, anaerobic bacillus. The spores of this organism are highly resistant to environment influences and disinfection.

**Transmission:**

First entry is through alimentary mucosa after ingestion of contaminated feed or eruption of teeth. Contamination of soil and pasture occurs from infected feacal material or decomposition of carcasses of animals.

**Clinical Signs:**

High fever

Gaseous swelling under skin mainly on hind quarter and shoulder.

There is stiffness or limping of one leg.

Gaseous swelling may develop in other parts of the body as well like neck, chest and flanks.

At the beginning swellings are hot, painful and limited later they become larger, cold and painless.

Skin over the swellings becomes dry, dark in colour and on palpation crepitation is felt.

Other symptoms include less appetite, cessation of rumination, rapid breathing, depression and death of animal within 24-48 hours. Affected animal may die without showing signs.

**Diagnosis:**

Diagnosis is on the basis of age, season, and swelling on specific area. Make smear, gram staining and observe clostridium.

**Treatment:**

Treatment must be in time within 24-48 hours. For specific treatment, provide oxytetracyclin (without xylocain or lignocain) 10 mg/kg body weight intravenously for several days. For supportive treatment give multivitamins.

**Vaccination:**

Vaccine prepared by Veterinary Research Institute (VRI) is available.

**Enterotoxemia**

**Etiology:**
Cl. perfringens is the causative agent of enterotoxemia. Its types B and C mainly cause disease in calves. It mostly occurs in highly fed calves.

**Sign and Symptoms:**In calves, there is acute diarrhea, dysentery, abdominal pain, convulsions, and opisthotonos. Death may occur in a few hours, but in less severe cases survive for a few days, and recovery over a period of several days is possible.

**Treatment:**
Treatment is usually ineffective because of the severity of the disease, but if available, specific hyperimmune serum is indicated, and oral administration of antibiotics may be helpful.

**Control:**Vaccinate the calves against enterotoxemia