

DISEASES OF THE NEWBORN

This includes the principles of diseases which occur during the first month of life in animals born alive at term.

1) Prenatal disease:

1) **Fetal diseases:** Diseases of the fetus during intrauterine life, e.g. prolonged gestation, congenital defects, abortion, fetal deaths with resorption or mummification.

2) **Parturient diseases:** diseases associated with dystokia causing cerebral anoxia, injuries of skeleton and soft tissues.

3) **Postnatal diseases:**

(A) *Early postnatal diseases:* within 48 hours e.g. malnutrition due to poor mothering, hypothermia due to exposure to cold, low vigor in neonates due to malnutrition, special disease (Navil ill and Colibacillosis).

(B) *delayed postnatal diseases:* within 2 -7 days after parturition e.g. mammary incompetence resulting in starvation, increased susceptibility to infection due to hypoglobulinemia such as lamb dysentery, colibacillosis and foal septicemia.

(C) *late postnatal disease:* within 1 - 4 week of life e.g. white muscle disease, enetrotoxemia.

In summary, the causes of mortality in newborn lambs are largely physical and environmental and occur immediately at birth or shortly thereafter.

In calves, the major causes of mortality are with dystokia and with neonatal diarrhea in postnatal life. Meanwhile, lambs are susceptible to physical and environmental influences.

11) Congenital defects:

Abnormalities of structure or function which are present at birth. They may be or may not inherited, the inherited defects may not be present at birth. The effects of noxious agents in the pregnant female during the three major periods of gestation are going as following:

- **Period of the ovum:** causes deaths of the ovum and resorption e.g. vibriosis.
- **Period of the embryo and organogenesis:** depending on dosage and duration of insult and may lead either to:
 - Structural abnormality or functional deficit caused by vitamin A deficiency, toxic agent, infection.
 - Death of the embryo and resorption or abortion (vibriosis).
- **Period of the fetus and fetal growth:** illness of fetus resulting in:

Abortion → *Brucella abortus*, IBR .

Mummification → inherited, infectious and unknown.

Stillbirth → Dystokia, hypoxia.

Weak neonate → infection in last week of gestation.

Causes:

1) Virus infection:

Only seven viruses are known to cause congenital defects in man and animals condition. They include the viruses of :

- Swine fever and Bovine viral diarrhoea → in animals
- Blue tongue and Acabane disease → in animals

- **Hog cholera virus:**

Vaccination of sows with modified vaccine virus between 15 – 25 days of pregnancy produces piglets with edema, deformed nose and kidney. Meanwhile natural infection causes cerebral hypoplasia in piglets.

- **Blue tongue virus:**

Vaccination of ewes with attenuated vaccine virus between 35 – 45 days of pregnancy causes porencephaly in lamb.

- **Bovine viral diarrhoea virus:**

Natural infection causes cerebral hypoplasia in calves. Meanwhile experimental infection of pregnant heifers causes cerebral hypoplasia, optic including cataract, retinal degeneration, hypoplasia and neuritis of nerve.

- **Acabana virus:**

Infection of pregnant cows produces arthrogryposis and hydroencephaly.

2) Nutritional deficiency:

There are numbers of congenital defects in animals which are known to be caused by deficiency of specific nutrients in the diet of dam; such conditions include the following.

- Goiter in all species due to iodine deficiency.
- Enzootic ataxic in lambs due to copper deficiency.
- Manganese deficiency which causes limb deformity in calves.
- Vit. D causes neonatal rickets.
- Vit. A deficiency which causes eye defect, hairless in piglet.
- Simple inanition or nutritional deficiency of protein do not cause congenital defects, but can increase prevalence of stillbirth and abortions.

3) Chemical poisons:

a- Poisonous plants.

b- Farm chemical.

Parbendazole and cambendazole in sheep.

Organophosphorus compound (may be).

c- Miscellaneous chemical and drugs.

- Cortisone in early pregnant.
- Estrogen derivatives.
- Bismuth, selenium, nitrogen mustard and tetanus toxin.

d- Physical insults.

Severe exposure to beta or gamma irradiation.

Hyperthermia.

4) Environmental influences.

The fetus is a sensitive biological indicator of the presence of some noxious influences in the environment.

Diagnosis of these congenital defects:

1. Clinical signs and P.M. lesions.
2. Clinical laboratory findings.
3. Epidemiological investigation including:
 - a. Pedigree analysis.
 - b. Nutritional history of dams of affected neonates.
 - c. Disease history of dams of affected neonates.
 - d. History of drugs of affected neonates.
 - e. Seasons and translocation.
 - f. Introduction of new comers to the herd.

Intrauterine growth retardation:

It is failure to grow properly and is distinct from failure to gain body weight. It is usually measured by the crown to rump length. Dwarfism in cattle and ranting in pigs are most common disorders of retarded intrauterine growths.

111) Diseases caused by physical and environmental influences:

1- Particular injury and intrapartum death:

As a result of dystokia, which may not be assisted by the farmers, there is a chance of physical trauma to limb or rib cage. Damage of the brain by way of intracranial hemorrhage which causes death of about (70%) of neonates within days of life.

- In causes of prolonged birth which leads edema of many parts of the body, especially head region (tongue) may develop.
- Intrauterine deaths may results in cause of prolonged birth from hypoxia of the fetus due to impaired circulation through placenta.

2) Fetal hypoxia:

Normally, foals are born in a primary apnea state, but gasping respiration begins with 30 – 60 seconds.

Placental dysfunction or occlusion of the umbilicus during second stage of labor result in a state of terminal apnea. It will be still born unless urgent and vigorous resuscitation is initiated immediately including the following:

- Extending the head and clearing the nostrils from mucous.
- Sealing one nostril by hand and breathing forcibly into the other, the chest wall to be moved only slightly with each breathe.
- I/V administration of 200 ml of 5% sodium. Bicarbonate solution to overcome acidosis.
- Neonatal hypoxia usually occur in foals use than 325 days of fetal age, they are small size, reduced body weight unable to rise and to suck and, suffering from respiratory distress.
- Stomach tube feeding with mare's milk or reconstituted by milk at dose rate of 80 ml/kg. B. wt. In 10 divided dose day.

Differential diagnosis:

1. Intrauterine infection in late gestation.
2. Exposure of neonate to cold environment.
3. Maternal deficiencies of the dam.

1V) Environmental and nutritional factors:

There are two environmental factors which may predispose to infection, besides causing mortality themselves, which are low: environmental temperature and deprivation of carbohydrate leading to hypoglycemia.

• *Effects of temperature:*

Piglets are more susceptible to hypothermia and hypoglycemia. The thermoregulation mechanism in piglets is efficient in first 9 days of life after which it is not fully functional until 20 days of life.

The major causes of hypothermia in lambs are excessive heat loss, depressed heat production caused by intrapartum hypoxia and immaturity, and starvation.

Administration of glucose is necessary in such lambs at a dose rate of 2 gm / kg BWt. as 20% solution injected intraperitoneally in addition to rewarming animal at 40 c and feeding of 100 –200 ml colostrum.

• *Effect of nutrition:*

Nutrition of dams is so important in relation to the resistance of offspring. Newborn lambs, calves and foals are much more capable of maintaining their blood glucose level than piglets when starved.

Hypoglycemic coma may be observed in lambs while calves are highly resistance to insulin deficiency where produced hypoglycemia during first 48 hrs of life.

Malnutrition of lambs caused by:

- Antipartum malnutrition of ewe which causes reducing of milk flow.
- Inclement weather preventing lamb sucking or ewe mothering.
- Inadequate maternal behavior.
- Too old ewes → low milk flow.
- Too tired ewes → low milk flow.

Inadequate mothering by ewe causes:

- Malnutrition of lamb.
- Lack of warmth and protection of lamb.

Reduced vigor of the lambs caused by:

- Small lamb, especially during cold weather.
- Intrauterine and postnatal infection.
- Malnutrition.
- Very cold or very hot weather.
- Being one of multiple birth.

V) Neonatal infection:

Attention is currently devoted to both the virulence of the pathogens and the resistance of the host, because of some of non-pathogenic microorganisms can cause the diseases if the immunological status of the animal is not at an optimum level.

Etiology:

The common neonatal infections among the domestic farm animals are:

Cattle:

- Bacteremia or septicemia caused by *E. coli*, *listeria monocytogenes*, *pasteurella spp.*, *Streptococcus.*, *salmonella spp.*
- Enteritis caused by pathogenic *E. coli*, *salmonella spp*, rotavirus, corona virus, cryptosporidia, clostridium.
- Respiratory tract diseases caused by IBR, PI3 viruses.

Horses:

- Septicemia with localization especially in joints, caused by *E. coli*, *Salmonella typhimurium*, *Sterpt. Pyogenes*.
- Septicemia with localization particularly in lungs caused by *C. equi*.
- Enteritis caused by *Clostridium perfringens* and rotavirus.

Sheep:

- Bacteremia with localization in joints caused by *Strept. micrococci*, *E. insidiosa*.
- Gas gangrene of the navel caused by *CI. septicum*, *CI. edematus*.
- Lamb dysentery caused by *Cl. Perfringens* type B.

Epidemiology:

1. Portal of infection:

It could be intrauterine or postnatal. If it is intrauterine the infection gains entrance via the placenta, and probably by means of placentitis due to a blood born infection or an existing endometritis.

Thus disinfections of the uterus is so important and disinfections of the environment may have little effect on the incident of the disease.

If the disease is postnatal the portal of infection may be through the navel or by ingestion. Contamination of the environment can occur from soiling of the udder or bedding by uterine discharges from the dam, from previous parturition, or from discharge of other affected neonates.

2. Resistance to infection:

All newborn farm animals are more susceptible to infection than adults due to:

- They are a gammaglobulinemic and possess no resistance to infection until after they have ingested colostrums. The immune system of the newborn animal is less mature than adult and dose not respond effectively to antigens as the adult.
- The fetal lamb produce large quantity of corticosteroides beginning 8–10 days before birth (also fetal calves) → result in lymphopenia → decrease phayocytic activity → decreased cellular immunity and decreased resistance.

- There are four recognizable immunological deficiency states in newborn foal which are:

(1) Combined immunodeficiency (CID):

Which is inherited as an autosomal recessive trait in Arabian horses. There is marked lymphopenia, absence of lymphocytes from all lymphoid organs, hypo- or a gammaglobulinemia and death at 2-20 weeks of age.

(2) Primary a gammaglobulinemia:

Animals are usually affected with chronic infection. Serum concentration of IgG and IgM are low and may not be detectable. There is failure of antibody production following injection of foreign antigens, but tests for cell mediated immunity may be positive.

There is complete absence of B-lymphocytes and immunoglobulins but normal T-lymphocyte counts.

(3) Hypogammaglobulinemia:

Following inadequate transfer of colostrum immunoglobulins from dam to foal; it is most common in foals and associated with high incidence of infection.

(4) Selective immunoglobulin M deficiency (IgM):

It is associated with infection 4-8 months of age.

(5) Passive immunity:

There are many important factors which influence the level of serum immunoglobulins achieved by the newborn and they are:

(1) Insufficient ingestion of immunoglobulins due to:

- Insufficient amount of colostrums produced by dam for many reasons as poor husbandry, malnutrition.
- Low concentration of immunoglobulin in the colostrums.
- Insufficient amount of colostrums ingested by the newborn due to:
 - i. Poor mothering behavior which may prevent newborn from sucking.
 - ii. Poor udder or teat conformation.
 - iii. Weak and traumatized calf.
 - iv. Failure to allow the newborn to ingest.

(2) Insufficient absorption of immunoglobulin from colostrums:

- Delayed ingestion of colostrums.
- Interference with the efficiency of absorption of immunoglobulins from colostrums.

Clinical finding of newborn infection:

The clinical finding depend on the rapidity of infection.

Slow spreading:

Omphalophlebitis, fever, depression, anorexia, leucocytosis and signs referable to the localization as :

- a. Endocarditis with heart murmur.
- b. Panophthalmitis with pus in the anterior chamber of the eye, meningitis with

Principles of control and prevention of Infectious diseases of newborn animals

(1) Removal of the cause of diseases from the environment:

- The newborn should be born in an environment which is clean, dry and conducive for the animal to get up after birth and such the dam.
- Then swabbing of the navel with tincture iodine to prevent the enter of infection.
- Disinfections of the uterus before conception is necessary also. Examination of swabs from the uterine contents before and after treatment in suspected animals.

(2) Removal of the newborn from the infected environment:

- Transfer the newborn to a non-infected environment either temporary or permanently in cases of over crowded barn.
- Removal of the newborn away from the main calving ground.
- Diseased calf should be transferred with his dam to hospital pasture during the period of treatment and convalescence.

(3) Increasing and maintaining the non – specific resistance of the newborn:

- Ingestion of colostrums from dam is so important as the only one source of immunoglobulin to newborn.
- Calf fed about 80 ml / kg body weight of colostrums at 6 hours of age.
- Special nutritional and housing requirements.
- Isolation of newborn calf in calf – rearing unit within few days after birth.
- Provision of suitable environment.

(4) Increasing the specific resistance of the newborn:

Vaccination of dam before parturition to stimulate the production of specific antibodies which are then transferred to the newborn via the colostrums.

Navel-ill

Infection of the umbilicus and its associated structures occurs commonly in newborn farm animals especially in calves. Normally the umbilical cord dries up within one week after birth. There is usually a mixed bacterial flora including: *E. coli*, *Proteus spp*; *Staph. Spp.* and *Coryn. Pyogenes*.

This infection may result in omphalitis, omphalophlebitis, omphaloarteritis, infection the urachus extended to the bladder causing cystitis.

OMPHALITIS/OMPHALOPHLEBITIS:

Definition and etiology. Omphalitis is inflammation of umbilical structures that may include the umbilical arteries, umbilical vein, urachus, or tissues immediately surrounding the umbilicus. The umbilicus consists of their types of structures and undergoes functional and anatomic changes at birth. Two umbilical arteries connect internal iliac arteries to the placenta. These later regress and become the round ligaments of the bladder. One umbilical vein connecting the placenta to the liver and porta cava regresses to become the round ligament of the liver within the calceiform ligament. The urachus connects the fetal bladder to the allantoic cavity.

Umbilical abscess or infection Of any of the three components of the umbilicus may produce local infection or be a source of septicemia. The source of infection is most commonly the external environment, coupled with failure of passive transfer. Organisms isolated in foals include *Escherichia coli* and proteus and *streptococcus spp*. Bacteria isolated from calf umbilical cord remnant infection include *actinomyces pyogenes*, *E.coli*, and proteus and *enterococcus spp*. The urachus is the most commonly affected structure in calves and the umbilical arteries the least. Omphalophlebitis may extend the length of the umbilical vein into the liver and result in liver abscess formation.

Clinical signs and differential diagnosis: When the umbilicus is enlarged and draining purulent material, infection is easily noted. In other cases the umbilicus may be dry and larger in diameter than expected. In addition, neonates may have a completely normal – appearing, dry external navel and severely ill from infection of the urachus, umbilical arteries, or vein. In a septic neonate without external signs of infections, involvement of the umbilicus can be difficult to determine.

The presence of pain on palpation of the umbilicus indicates inflammation. Ultrasound has recently been shown to aid the detection of involvement of urachus or arteries and vein by detection of size abnormalities. The umbilical area of neonates less than 20 days of age with fever of unknown origin should be scanned. Hematoma developing after umbilical rupture may produce distention of the umbilical stump shortly after birth.

Overt signs of infection are heat, swelling, purulent discharge, or pain. Concurrent signs of systemic infection such as joint infection, pneumonia, diarrhea, meningitis, or uveitis may be noted.

Diagnostic methods: In addition to detection of overt umbilical inflammation as described, ultrasonography may help in evaluating a normal – appearing navel. The umbilical vein, arteries. and urachus may be imaged in the newborn. The umbilical

arteries leave the umbilical stalk and cause on the other edges of the urachus in a parallel fashion. In the foal the urachus connects the apex of the bladder with the umbilicus and is located along the midline immediately adjacent to the body wall.

Persistent dilation of the umbilical vein or arteries with a hypoechoic echogenic fluid is seen with infection. In calves the urachus normally retracts up into the abdomen at birth, and ultrasonographic identification of a urachal remnant is abnormal. In the hands of a skilled ultrasonographer familiar with normal ultrasonographic findings of the umbilicus, there is an excellent correlation between surgical and ultrasound findings.

Treatment and prognosis: Early treatment with antibiotics and supportive care as described for the septicemic foal may allow resolution before development of abscessation and distention of the urachus or the umbilical arteries and vein. Established infection, which may occur within 24 hours, usually requires surgical removal of involved structures in addition to medical therapy. When omphalophlebitis extends into the liver, the umbilical vein may be facilitating drainage and flushing.

Surgical removal of urachal abscesses in calves often requires resection of the apex of colostral immunoglobulins has occurred and when joints or other structures are not involved. Sequelae such as renal abscessation, joint inflammation, septicemia may develop if therapy is started too late or discontinued.

1st) Omphalitis:

Inflammation of the external aspect of the umbilicus and occurs commonly in calves 2 – 5 days after birth. The umbilicus is enlarged, painful, may be closed or draining purulent discharge. The calf is depressed, febrile and does not suck normally.

2nd) Omphalophlebitis:

Inflammation of umbilical veins. Large abscess may develop along the course of umbilical vein and spread to the liver forming liver abscess. It usually occurs in calves 1 – 3 month age. Umbilicus is enlarged and containing purulent material. Inappetence, unthrifty, mild fever.

3rd) Omphaloarteritis:

Inflammation of umbilical arteries (less common) the Clinical signs and treatment as omphalophlebitis.

SPECIFIC CONDITIONS

Meconium impaction:

Meconium impaction is the most common cause of colic in the newborn foal. Many foals show some degree of straining and discomfort while passing meconium, but in most instances it is passed uneventfully by 24 to 48 hours of age. The meconium most commonly becomes impacted in the rectum or small colon.

The clinical signs associated with meconium impaction in the otherwise normal foal include repeated attempts to defecate, straining with the back arched, swishing of the tail, and restlessness. Digital examination often reveals a rectum packed with hard fecal balls. Occasionally the impaction is located more proximally (large or small colon) and radiography or ultrasonography is required for diagnosis.

An enema with mild soap and warm water, a commercial enema, a small amount (10 ml of a 5% solution diluted in warm water) of diocyl sodium sulfosuccinate (DSS), or acetylcysteine usually result in prompt evacuation of the meconium. Care must be taken to avoid traumatizing the rectal mucosa by stiff tubing or multiple enemas with harsh detergents.

Clinical signs associated with meconium impaction in the compromised foal may be absent. In asphyxiated or premature individuals that are receiving litter or no enteral feeding, meconium may remain in the large colon for days, gradually forming into hard concretion that are diagnosed by palpation or radiographs or at postmortem examination. In these cases the routine administration of an enema is often ineffective in mobilizing the impaction because it is high in the large colon. DSS and water or mineral oil administered through a stomach tube may be more effective in breaking up the impaction. Intravenous fluids and analgesics also may be helpful. Although most meconium impactions can be treated successfully with aggressive medical therapy, those few foals that are refractory to treatment or display uncontrollable pain are candidates for surgical intervention. Meconium impaction is rare in neonatal ruminants.

Uroperitoneum:

Uroperitoneum is a relatively common cause of abdominal distention and depression in the neonatal foal. The condition predominates in males, but may occur in females. The most common cause of uroperitoneum is a ruptured urinary bladder, but other sites in the urinary tract may also leak, including the ureters, urachus, and urethra. Most cases of ruptured bladder are presumed to occur during parturition because of external pressure on a distended bladder, but a developmental defect also has been proposed as a possible cause. The recent development of neonatal critical care has resulted in an increasing number of reports of uroperitoneum secondary to the necrotic or infected urinary bladder or urachus in the compromised foal.

Clinical signs of uroperitoneum are rarely noticed before 48 to 72 hours of age, particularly if the foal is not being watched closely. The first signs may be urinary incontinence or frequent attempts to urinate, with only small amounts voided. Sometimes, particularly in those animals that rupture sometime after birth, there is a history of a period of normal urination, with at some point stopped or become abnormal.

Increasing abdominal distention is usually accompanied by worsening depression and increasing heart and respiratory rate. If the condition is allowed to persist, foals become increasingly weak and dyspneic and may present in cardiovascular collapse. Fillies with ruptured ureters have a characteristic protruding perineum, presumably as a result of retroperitoneal accumulation of fluid.

Laboratory findings commonly associated with uroperitoneum are elevated serum creatinine and blood urea nitrogen (BUN), hyperkalemia, hyponchloremia, and metabolic acidosis. These changes are probably a result of the normal diet of the foal (milk being relatively high in potassium [25 meq/l] and low in sodium [12 mEq/L]) and the third spacing of urine in the peritoneal cavity. With urine potassium concentration relatively higher than serum and urine sodium concentration lower than serum levels, the net effect of partial equilibration of serum with peritoneal fluid across a semipermeable membrane is hyponatremia and hyperkalemia, along with an inability to excrete the waste products of metabolism.

In hospitalized foals that developed uroperitoneum as a secondary complication, these typical electrolyte abnormalities were not consistently observed. Because most of those foals were receiving replacement intravenous fluids (high in sodium, low in potassium) and little milk, it was theorized the intake has a great influence on the electrolyte abnormalities associated with uroperitoneum. Foals with renal failure, blocked urethra, and enteritis have shown the same electrolyte changes.

Diagnosis of uroperitoneum is usually made by detection of urine in the peritoneal cavity of a foal with a distended abdomen. In the foal with uroperitoneum, the creatinine in the peritoneal fluid should be at least double the serum level. If the creatinine is the same in both serum and peritoneal fluid, other explanations for the clinical signs should be investigated. The WBC count, total protein, and cytology of the fluid also should be determined.

Most uncomplicated cases of ruptured bladder have fairly normal values for peritoneal fluid. In some cases, however, an increased WBC count and total protein and the presence of bacteria may suggest peritonitis. This may be a result of the urine in the abdomen, but more commonly there is a primary ongoing infectious problem (necrotic urachus or bladder, enteritis), and the prognosis becomes worse. If laboratory facilities are not available, new methylene blue can be injected into the bladder using a urinary catheter, and a few minutes later a sample of peritoneal fluid should have a blue discoloration if a ruptured bladder is present. This technique, however, may not detect other causes of uroperitoneum such as a ruptured ureter or distal urachus. Positive contrast cystography using a 10% solution of water-soluble media may be helpful in detecting the location of the urinary tract leakage. The ability to obtain urine on catheterization of the urinary bladder does not rule out uroperitoneum. Hematology and blood cultures should be performed to detect primary or secondary sepsis.

Treatment of uroperitoneum is surgical repair; however, the foal with uroperitoneum should not be rushed to surgery without first carefully stabilizing it. Serum electrolytes and blood gases should be run to determine the extent of hyperkalemia, hyponatremia, and acidosis present. Although the total amount of

water in the body is usually grossly increased by the peritoneal accumulation of urine, effective circulation volume may be drastically reduced. If the eyeballs are sunken and pulse quality and capillary refill time are poor, aggressive fluid therapy is indicated to support the circulation.

This therapy is best performed by concurrently removing as much fluid as possible from the abdomen with a treat cannula or peritoneal dialysis catheter to avoid worsening fluid overload and respiratory distress. The fluids of choice to treat the typical electrolyte alterations associated with uroperitoneum are saline, dextrose, and possibly sodium bicarbonate solutions, depending on the degree of acidosis.

The prognosis for uncomplicated ruptured urinary bladders is usually good (>80% survival), provided the animal is stabilized before anesthesia. The presence of concurrent generalized infection carries with it a considerably worse prognosis.

Gas or Fluid Accumulation in the Gastrointestinal Tract: Heus:

Abdominal distention and colic secondary to excessive gas and / or fluid accumulation in all or a portion of the gastrointestinal tract are common complications in the compromised neonate undergoing intensive care. The exact mechanisms responsible for the presumably altered gastrointestinal motility are not well defined. In the foal neuralgia, particularly in the struggling or hypoxic neonate, often results in gas distention that is not easily removed through a nasogastric tube, because gas tends to move quickly through the gastrointestinal tract.

Abdominal distention is also commonly observed during mechanical ventilation in the foal, as a result of overfeeding in the calf, or secondary to bacterial overgrowth during rewarming after hypothermia and shock. In may severely asphyxiated or very immature foals, even mare's milk is poorly tolerated, necessitating the discontinuation of oral feeding. Foals with botulism are often intolerant to enteral feeding, probably because of altered gastrointestinal motility. Use of certain milk replacer can result in bloat colic, and diarrhoea, even in the apparently healthy orphan neonate. Discontinuation of or a decrease in the amount of enteral feeding and, if possible, increased activity of the patient usually result in resolution of the problem.

Patent urachus:

Definition: Patent urachus is a persistence after birth of the tubular connection between the bladder and umbilicus. The urachus drains the bladder into the allantoic sac during gestation. Urine flow should gradually changes, with some urine entering the amniotic sac through the urethra in later gestation. At birth, with umbilical cord rupture the urachus should be closed, and urine should be voided through the urethra. Foals with a patent urachus may dribble urine from the urachus during or after urination or may simply present with a constantly wet umbilical stump.

Etiology: A variety of causes have been suggested for failure of the urachus to close and completely involutes. Early closure or ligation of the umbilical cord, inflammation, infection, and excessive physical handling of the neonate have been implicated. Rather than being the original cause?? Hospital admission, patent urachus develops as a complication of hospitalization in a significant percentage of foals in neonatal intensive care.

Clinical signs and differential diagnosis: Differential diagnosis includes concurrent infection of the navel (omphalophlebitis) by:-
Ultrasound may assist the diagnosis and determine the involvement of umbilical arteries or vein. Moist hairs around the umbilicus and visualization of fluid coming for the navel are diagnostic.

Clinical pathology: Identification of concurrent infections essential. A complete physical examination should be performed. If abnormalities are noted, serum IgG, complete blood count, and urinalysis are helpful for detecting susceptibility to infection and presence of systemic or urinary tract infection.

Pathophysiology: Congenital patent urachus caused by excessive torsion on the umbilical cord in utero occurs in 6% of normal foals. The obstruction of the urachus caused by the torsion cause retention of urine in the bladder and overdistends the proximal urechus, which interferes with normal involution. Infection of umbilical structures or the urachus itself may result in inflammation and failure to completely involute. In a review of 16 cases of umbilical cord infections in foals, 13 had patent urachus. The majority of these foals had acquired patent urachus after birth, with the youngest age of onset 3 days and the mean age of onset 12 days.

Excessive manipulation and improper lifting of the foal's abdomen in the presence of high urethral sphincter tone may force urine within the bladder out into the involuting urachus. In our experience farms have experienced outbreaks of patent urachus when procedures (such as tests for failure of passive transfer) have been implemented that require handling of foals in the first 12 to 24 hours of life. A similar cause may be responsible for the increased incidence of patent urachus in hand – reared calves.

Treatment and prognosis: Therapy consists of either conservative management though monitoring or medical treatment for infection and cauterization of the urachus with iodine, phenol, or silver nitrate sticks applied into the urachus. Persistence of urine dribbling after 2 to 3 days of cauterization, the detection of involvement of other umbilical structures through ultrasound, and a rent in the urachus that produces subcutaneous swelling are indications for surgery. Not all foals that have persistent patent urachus have an infected umbilicus.

Use of general anesthesia and removal of the entire urachus to the tip of the bladder are performed in foal or ruminants with an infected or enlarged urachus. Associated arteries and veins should be legated and removed if they are infected or necrotic. Merely legating the exterior stump can trap organisms and cause infection. In our neonatal unit the majority of patients with acquired patent urachus respond to conservative therapy. Late-onset patent urachus (>5 days of age) may be more refractory to conservative therapy. Complications of delaying surgery relate to development of bladder necrosis and uoperitoneum caused by extension of infection and inflammation of the urachus.

Prevention: Allowing the umbilical cord to rupture without legation or the careful use of specific umbilical clamps after birth has been suggested to decrease the incidence of patent urachus. Minimum handling of neonates and careful restraint may prevent pressure build-up in the bladder and subsequent patent urachus.

FAILURE TO THRIVE CACHEXIA AND WEAK CALF

SYNDROME

Definition and etiology : Neonates that are born weak or fail to grow as anticipated pose important problems. In the foal, twins, prematurity, hypothyroidism, and congenital heart or other organ defects may produce failure to thrive. Infections acquired shortly after birth that produce chronic pneumonia, nephritis, endocarditis, arthritis, or gastric ulcers are a cause of morbidity in the neonatal period. Nutritional problems in sick and convalescing foals have been identified as being previously unrecognized factors in stunting. daily consumption of 25 % to 30 % of their body weight as milk in convalescing foals has been reported. This is not surprising, as normal foals gaining 1 kg / day consume 20 % of their body weight in milk.

In calves the weak calf syndrome has been reproduced by feeding low- protein diets to prepartum cows that subsequently calved in environments in which the temperature was well below the thermoneutral zone for calves. The dietary recommendation for crude protein intake for third-trimester pregnant cows and heifers is 0.9 kg (2 lb) of total crude protein / day. This particularly important for heifers and cows calving early in the spring calving season when temperatures well below freezing can occur cold rains, however, also can produce the hypothermic conditions that aid in precipitating this syndrome.

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