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Haematology and Clinical Biochemistry Findings Associated with Equine Diseases - a Review

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Abstract

The course and outcome of a disease process is dependent upon factors such as the disease-causing agent and its cell tropism, defense mechanisms of the host, genetic resistance of the species or breed affected, as well as the age, nutritional status and hormonal levels of the affected animal. When haematology, clinical biochemistry and cytology test results are combined with other laboratory procedures, complete physical examination and also with the history of the patient, a veterinarian is well armed to arrive at a definitive diagnosis, make a certain prognosis (good, poor or guarded) and can also make a concluding statement on the efficacy of the instituted therapy. In clinical biochemistry, demonstration of specific enzyme activity and concentration of analytes in serum/plasma facilitates the disease diagnosis. Also, evaluation of haematology, clinical biochemistry and diagnostic cytology tests can help establish the presence or absence of diseases of internal organs, and by serial performance of these tests, may help to determine whether a disease process remains static, progressive or regressive. This review therefore provides the haematological, serum biochemical and cytological characteristics of diseases caused by the main bacteria, viruses, fungi, protozoa, helminths, arthropods, nutritional deficiencies, endocrine disturbances, neoplasm, allergy, toxins (phytoxins and zootoxins) and inorganic poisons in horses.

Keywords: analytes, diagnostic cytology, disease, disease-causing agents, haematology, serum biochemistry

Introduction

A disease is defined as any alteration in an organism or some of its organs or parts, which interrupts or disturbs the performance of its vital function and constitute a departure from its normal health state (Cheville, 1999; Ihedioha, 2003). In general, diseases can occur as inherited/genetic errors, infections/infestations, nutritional disorders caused by physical and chemical agents, metabolic disorders, endocrine disturbances and/or disordered growth of tissues (Radostits *et al*., 2007; Robbins *et al*., 2010). Inherited/genetic diseases occur as a result of abnormalities in chromosome structure and function mutations, multifactorial or polygonic inheritance (Jones *et al*., 1997; Cheville, 1999). Infections and infestations occur as viral, bacterial, rickettsial, fungal and protozoal diseases, and infestation can also occur with helminths and arthropods (Radostits *et al*., 2007). Nutritional diseases arise as a result of deficiencies, excesses or imbalances in intake and availability of nutrients such as proteins, carbohydrates, fats, vitamins and minerals (Radostits *et al*., 2007). The physical factors that cause diseases include trauma, electrical injury, temperature changes and radiation injury, while the chemical agents associated with diseases include environmental pollutants such as heavy metals, halogenated hydrocarbons and routinely used pesticides (Cheville, 1999; Robbins *et al*., 2010). Metabolic disorders arise as a result of derangement in one or more of the body's normal metabolic processes, while endocrine disturbances include disorders that are caused by hyper-secretion or hypo-secretion of hormones produced by the endocrine glands (Jubb *et al*., 2007; Radostits *et al*., 2007). Growth disturbances range from physiological compensatory responses such as hypertrophy, hyperplasia and metaplasia to uncontrolled proliferation of cells/tissue as it occurs in malignant neoplasms (cancer) (Cheville, 1999; Jubb *et al*., 2007).

Haematological assessments are useful in the clinical diagnosis of infectious, parasitic and metabolic diseases. They are also used in monitoring recovery during treatment and to assess the health status of an entire flock (Lassen and Swardson, 1995; Messer, 1995). Factors that affect the haematological parameters in apparently healthy horses include breed, sex, age, reproductive and training status, time of feeding, diurnal variations, prior exercise, nutritional state and management, subclinical infections/infestations, and also geographical / climatic factors such as temperature, humidity, altitude and day length (Hodgson and Rose, 1994; Lassen and Swardson, 1995).

The haematological parameters of veterinary importance include erythrocyte counts, packed cell volume (PCV), haemoglobin concentration (Hb), erythrocyte sedimentation rate (ESR), mean corpuscular values (MCV), total leukocyte count, differential leukocyte count, platelet count (Schalm *et*

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al., 1975; Coles, 1986). The erythrocytic parameters (erythrocyte count, PCV, Hb and MCV) are a set of haematological indices used to evaluate the state of the erythron and thus determine whether an animal is normal, anemic, or polycythemic (Coles, 1986; Ihedioha and Chineme, 2004). The leukocytes constitute an important part of the defense and immune system of the body and therefore function specifically in initiation, maintenance of inflammation and detoxification. The leukocytic profile and its assessment enable the clinician to evaluate the animal's response to the challenge of infectious agents, toxins and toxic chemicals, physical injury and neoplastic proliferation (Schalm *et al*., 1975; Coles, 1986; Dein, 1986; Ihedioha and Chineme, 2004). Platelets function primarily in haemostasis (blood clotting) and they are involved in the arrest of bleeding following blood vessel injury. Deviations in platelet numbers are clinically significant as indicators of bone marrow stimulation (thrombocytosis) or as the basis for spontaneous and uncontrolled hemorrhages (thrombocytopaenia) (Ihedioha and Chineme, 2004; Stockham and Scott, 2008).

The assessment of the serum biochemistry parameters in animals is important because of its value in predicting pathological changes in vital internal organs of the body such as liver, kidney, pancreas, heart and muscles (Coles, 1986; Harr, 2002). Serum biochemistry assessment is also useful in evaluating the nature and extent of a disease, an animal's response to therapy and to forecast possible outcomes (Coles, 1986; Stockham and Scott, 2008). The serum biochemistry parameters of importance in the clinical assessment of horses include determination of serum activity of enzymes such as aspartate amino transferase (AST) and alkaline phosphatase (ALP), evaluation of synthetic activity of the liver by assessment of total proteins, albumin, serum cholesterol and bilirubin, and measurement of serum indicators of renal function such as creatinine and urea nitrogen (Coles, 1986; Stockham and Scott, 2008).

This review was aimed to provide comprehensive information on the haematology, serum biochemistry and cytological findings associated with commonly observed diseases and disorders in horses.

Diseases of horses

Bacterial diseases

There are several ways by which a bacterium can cause disease. The pathogenic ones are thus classified based on the predominant tissue reaction they cause, and could be said to be pyogenic, toxigenic or intracellular. The pyogenic bacteria produce pus and release factors that are chemotactic for leukocytes*;* toxigenic bacteria elaborate toxins that cause excessive tissue reaction and destruction and also kill specific cells; and intracellular bacteria which replicates inside macrophages and parenchymal cells may produce subacute or chronic diseases. Most bacteria release virulence factors that suppress the host cell's bacteriocidal mechanisms and thus permitting the survival of the bacterium (Cheville, 2006). The following are the most common bacterial diseases that affecthorses.

Brucellosis

Brucellosis is a bacterial disease caused by *Brucella* species. It is a zoonosis of great economic importance, as it affects the productive and reproductive potential of animals, in terms of loss of new born due to abortion, infertility and reduction or cessation of milk production (Corbel, 1997; Radostits *et al*., 2007; CFSPH, 2009). Humans can be infected either by contact or by consumption of contaminated animal products or unsterilized milk or meat. The *Brucella* organism is the cause of undulant fever in humans (Mile Bosilkovski, 2007). Abattoir and laboratory workers, farmers, butchers and veterinarians are at risk of exposure. Their occupation predisposes them to infection with *Brucella abortus* which can penetrate the intact skin, subcutis, wounds, respiratory tract and eye (conjunctiva) (Radostits *et al*., 2007).

Equine brucellosis is caused mainly by *B. abortus* and rarely by *B. mellitensis*. *B. abortus* has a predilection for gravid uterus, udder, testicles and accessory male sex glands, lymph nodes, joint capsules and bursae (Poester *et al*., 2010). The major route of transmission is oral, as animals tend to lick contaminated genital discharges, aborted fetuses, placentas and urine (Ehizibolo *et al*., 2011). It is also transmitted by coitus or when infected semen is used for artificial insemination. Another way of being transmitted is in-utero or during lactation through milk (Bercovich *et al*., 1990; Nicoletti, 2007). Horses commonly infected are those reared/mixed alongside with infected cattle; these horses can develop a positive reaction to the serum agglutination test (SAT) without showing clinical signs.

Equine brucellosis is characterized by a granulomatous supraspinous or supra-atlantal bursa lesion (fistulous withers or poll evil). There is lymphadenopathy, hepatomegaly, arthritis, and splenomegaly, and vertebral osteomyelitis, non-specific lameness due to joint infections, general stiffness and oedema of the limbs, lethargy and late abortion in mares (Cohn *et al*., 1992; Corbel, 1997; Megid *et al*., 2010).

No consistent changes in the haematological and serum biochemistry parameters have been reported in horses infected by *Brucella* species, but an elevated fibrinogen and neutrophilia may be detected (Lavoie and Hinchcliff, 2008). Lymphopenia, normocytic normochromic anemia, hypoproteinemia, hypoglycemia and increases in serum sorbitol dehydrogenase, aspartate aminotransferase and alanine aminotransferase activities had also been reported in camels affected with *B. abortus* (El-Boshy *et al*., 2009).

Mastitis

Mastitis is an inflammation of the parenchyma of the mammary gland characterized by physical and chemical changes in the milk and pathological changes in the glandular tissues. Mastitis is characterized by the presence of a significantly increased somatic cell counts (SCC) in milk, sodium or chloride concentration of the milk (as measured by electrical conductivity) or increased permeability of the blood-milk barrier (as measured by albumin concentration) in affected mammary gland. The increased somatic cell counts are, in almost all cases, due to an increased neutrophil count and represents a reaction of glandular tissue to injury. The most important changes in the milk include discoloration, the presence of clots and large numbers of leukocytes with sloughed off epithelial cells (Radostits *et al*., 2007). Mastitis is a rare condition in both non-lactating and lactating mares and usually occurs during the time of weaning (drying-off period),

affecting one or both glands. *Corynebacterium pseudotuberculosis, Staphylococcus aureus, Pseudomonas aeruginosa, Streptococcus zooepidemicus, Escherichia coli, Neisseria species, Streptococcus equi, Aspergillus* spp., *Coccidioides immitis* and migrating parasite larvae are the common causative agents, with *Streptococcus zooepidemicus* being the most frequent causative agents in horse (Jackson, 1986; McCue and Wilson, 1989). The clinical signs associated with mastitis include severe swelling and soreness of the mammary gland, fever, depression, anorexia, pain, heat in the affected half and ventral edema. Gangrene and sloughing of the ventral floor of a gland may occur. The mammary gland secretion may be sero-flocculent.

Cytological evaluations of milk samples reveal large numbers of neutrophils (McCue and Wilson, 1989). Haematological examination reveals a leukocytosis with neutrophilia, hyperfibrinogenemia, and anemia of chronic disease. Other abnormalities such as azotemia, leukopenia, increased non-segmented white cell count or toxic changes in neutrophils may be seen if systemic endotoxemia, bacteremia or concurrent disease is present (Lavoie and Hinchcliff, 2008).

Tetanus

Tetanus is a very significant disease suffered by horses and it is caused by the neurotoxin, tetanospasmin elaborated by the vegetative stage of *Clostridium tetani* and characterized by spastic paralysis (Popoff, 1995; Tonello *et al*., 1997). The horse is the most susceptible to this disease of all animal species. Foals are more frequently affected than adult horses especially through navel infection, castration and other tissue trauma. The organisms are introduced into the tissue through deep puncture wounds, particularly of the hooves, which provide a suitable anaerobic environment. Another source of contamination can be the sores and wounds arising from ill-fitting saddles (Schiavo *et al*., 2000). The clinical signs of tetanus include an increase in muscle stiffness accompanied by muscle tremor, lock jaw, prolapse of the nictating membrane, and stiffness of the hindlimbs, causing an unsteady straddling gait. There is an anxious alert expression, erect carriage of the ears, retraction of the eyelids, dilatation of the nostrils and exaggerated responses to normal stimuli. Mastication is prevented by tetany of the masseter muscle and drooping of saliva. Swallowing is often followed by regurgitation from the nose. Constipation, urine retention and inability to assume the normal position for urination may also occur. There is a rise in temperature and pulse rate as the muscular tone increases. The animal assumes a 'saw-horse' posture as the disease progresses. There may be curvature of the spine, tail deviation and opistothonos.

There are usually no gross pathological findings or specific abnormalities in cerebrospinal fluid of affected horses (Green *et al*., 1994), but blood picture and serum biochemistry profile of serum producing horses injected with the tetanus toxin reveal a low packed cell volume, low haemoglobin concentration, leukocytosis, decreased mean cell haemoglobin, increased serum activities of aspartate aminotransferase, lactate dehydrogenase and alkaline phosphatase, decreased blood urea nitrogen and increased serum bilirubin and cholesterol levels (Sahal *et al*., 2004).

Botulism

Botulism is an often deadly food poisoning characterized by flaccid paralysis of the voluntary muscles. It is caused by a neurotoxin elaborated during the vegetative growth of *Clostridium botulinum*, types B, C and D (Montecucco, 1995; Prigent *et al*., 2010). Horses are affected by ingesting the pre-formed toxins in feedstuffs, spoiled stored forages and silages (forage botulism) and ingestion of decaying vegetables and potatoes contaminated by *C. botulinum* (Sargison, 1993). Wound botulism occurs in horses with omphalophlebitis, injection abscess and following castration. Toxic-infectious botulism (shaker foal syndrome) occurs in foals of 8 months. This occurs when the toxin is produced by *C. botulinum* in the intestines of foals (Mitten *et al*., 1994). The clinical features of the disease include slow developing flaccid paralysis, restlessness, incoordination, stumbling and ataxia. Skin sensation is retained and the affected horse lies in sternal recumbency. The tongue is paralyzed and in some cases, the horse is unable to chew and swallow. Defecation and urination are usually unaffected.

There had been no reported changes in the haematology of affected animals. hypophosphatemia may be present and muscle enzyme activities may be moderately elevated (Radostits *et al*., 2007).

Glanders

This is a contagious bacterial disease of horses, donkeys and mules caused by *Burkholderia mallei* (Heine *et al*., 2001). It is characterized by the formation of fibrocaseous nodules and ulcers in the nose, skin and lungs (Schlater, 1992). It is a disease of public health importance. The causative agent is transmitted by inhalation or by ingestion of food and water contaminated by nasal and skin discharges. Indirect transmission by fomites such as harnesses and stable utensils can also occur. Horses in a stressed state or fed poorly and kept in dirty environment are more susceptible (Radostits *et al*., 2007). Three clinical forms of the disease are known to occur in equids and they include the pulmonary, nasal and cutaneous (farcy) forms. Acute glanders is characterized by fever, coughing and nasal discharges. There are signs of respiratory distress and death due to bronchopneumonia and septicemia may occur within a few days. This acute form is normally seen in donkeys and mules and it's pretty rare in horses. In chronic form, onset is insidious with general malaise, coughing, unthriftiness and intermittent fever followed in some cases by nasal and cutaneous lesions and enlargement of submaxillary lymph nodes. The pulmonary form is characterized by chronic difficulty in breathing. In the nasal form, the nasal discharges are usually purulent and blood stained. Ulcers develop from ruptured fibrocaseous nodules on the nasal mucosa which may coalesce to form eroded areas on the nasal septum, which in extreme cases may be perforated. In the cutaneous form, nodules are found along the lymphatics under the skin and may rupture, discharging yellowish-gray oily pus and causing ulcers. The nodules, ulcers and neighboring lymph nodes are connected by thickened lymphatic vessels which can be felt under the skin and the affected part becomes markedly swollen and painful. The characteristic gross lesions are ulcers of submaxillary lymph nodes, but the bronchial and thoracic lymph nodes may also be affected (Jones *et al*., 1997).

The disease is characterized by low haemoglobin concentration of the blood, erythrocyte count and packed cell volume. Moderate leukocytosis and neutrophilia are also associated with glanders (Radostits *et al*., 2007).

Dermatophilosis

Dermatophilosis, also known as cutaneous streptotrichosis, rain scald or kirchi, is a moist, exudative dermatitis of horses caused by Actinomycetes, *Dermatophilus congolensis*. The disease had been reported in man, but contamination from livestock is rare (Zaria, 1993). It is characterized by thick scab formation. Prolonged wetting of the skin from rainfall or frequent washing makes its outer layer (stratum corneum) more susceptible to external trauma caused by grooming tools, ectoparasites and biting flies (*Stomoxys calcitrans*) and house fly (*Musca domestica*) (Yeruham *et al*., 1996). Presence of intercurrent disease and pregnancy may weaken the animal's immunity and predispose it to the disease. These abrasions provide an ideal environment for *Dermatophilus congonlesis*. Invasion by the causative organisms through the compromised skin barrier elicits an acute inflammatory response composed primarily of neutrophils. The predominant clinical feature is that of exudative dermatitis which varies with regard to the extent of skin involvement. Lesions appear as tufts of matted hair and crusts over the face, shoulders, back and lower limbs especially around the pastern joint. Scabs are usually covered with gray or cream colored exudates. There are no reports of haematological and serum biochemistry findings associated with the disease in available literature.

Strangles

Strangles is one of the most important diseases of horses, caused by *Streptococcus equi*. Cold weather, poorly ventilated stables, over work, severe weather and grouping of horses together in stables predisposes them to the infection. It is transmitted by inhalation of infectious droplets breathed or coughed out by infected animals, grazing on pastures contaminated by nasal and abscess discharges or indirectly by fomites. Direct transmission from infected animals to susceptible animals occurs through contact. Colt and yearlings are particularly susceptible. The clinical features of the disease include pyrexia, anorexia, increased respiratory rate, serous to mucopurulent discharge, neck and head are outstretched and have difficulty swallowing due to severe pharyngitis. Dysphagia and lymphadenitis are also associated with the disease. Complications have been reported to occur in about 20% of the cases (Sweeney, 1996). The most common complication is the development of suppurative necrotic bronchopneumonia (aspiration pneumonia) consequent upon draining of the abscessed lymph nodes into the trachea (Radostits *et al*., 2007). Other clinical signs include guttural pouch empyema, purpura haemorrhagica and respiratory distress due to tracheal compression by abscessed mesenteric and mediastinal lymph nodes and pleuritis (Radostits *et al*., 2007).

Leukocytosis with a neutrophilia occurs at the early phase of the disease. There is also monocytosis and lymphopenia in mules (Ijaz *et al*., 2011). Serum biochemical abnormalities associated with the disease include hyperfibrinogenemia, hyperproteinemia and hypoalbuminemia. Anemia may be present or absent (Timoney, 1993).

Ulcerative lymphangitis

This is a mildly contagious disease of horses characterized by lymphangitis of the lower limbs, caused by *Corynebacterium pseudotuberculosis*. Horses get infected through contamination of abrasions on the lower limbs and spread the disease to other susceptible horses by contact, especially horses that are crowded together in dirty unhygienic stables. House flies possibly play a role in carrying *C. pseudotuberculosis* from an ulcerating infection of a horse to the wounds or injuries of another susceptible horse (passive transmission). This is followed by invasion of lymphatic vessels and the development of abscesses along the course of these lymphatics. Foals raised on bare ground are more prone to the infection than those raised on pasture. The clinical features of the disease include swelling and pain of the pastern, lameness, presence of nodules on the subcutaneous tissues of the body especially that of the fetlock which may rupture to discharge creamy green pus. The nodules may ulcerate leaving an ulcerative base. There is also enlargement of the lymphatics draining an affected area. *C. pseudotuberculosis* infection has been reported to cause abortion in mares (Poonacha and Donahue, 1995) and unilateral orchitis and epididymitis in stallions (Gonzalez *et al*., 2008).

Haematological and serum biochemical examination reveals a neutrophilia, anemia, increased serum activities of liver enzymes, hyperproteinemia characterized by hypoalbuminemia and marked hyperglobulinemia (Gonzalez *et al*., 2008).

Rhodococcus (Corynebacterium) equi pneumonia of foals

This is a sporadic disease of foals caused by virulent strains of *Rhodococcus equi*. It commonly affects foals of 1-5 months and they are usually infected through inhalation or ingestion during their first weeks of life. It is of public health concern and affects mainly immunocompromised humans (Prescott, 1991). The disease in horses is characterized by pneumonia, fever, respiratory distress, cough, multiple joint distension, occasional diarrhea, septic osteomyelitis and lack of nasal discharge.

The clinicopathological findings in affected foals include leukocytosis with neutrophilia, monocytosis and thrombocytosis (Leadon *et al*., 1992), increased fibrinogenemia and increased inflammatory cells in tracheal aspirates (Radostits *et al*., 2007).

Colibacillosis of foals

The aetiological agents for colibacillosis in foals are the pathogenic serotypes of *Escherichia coli* (enterotoxigenic, enterohemorrhagic and verocytotoxic) (Gyles, 1992). The disease mainly affects newborn foals consequent upon colostrum deprivation and stress (overcrowding), adverse climatic changes and use of inferior milk replacers. The disease is of public health concern in humans. It is characterized by septicemia, diarrhea and dehydration. The clinical chemistry and haematological abnormalities/changes are that of electrolyte imbalance, acidosis, increased packed cell volume (PCV) and elevated blood urea nitrogen in severe cases (Radostits *et al*., 2007).

Equine salmonellosis

Salmonellosis is a common cause of acute enterocolitis in the horse. It is caused by the *Samonella typhimurium* and *Salmonella enteritidis*. It is an important zoonotic disease. Susceptible animals are infected by ingestion or drinking of

contaminated feed and water, respectively. Predisposing factors include food and water deprivation, drought, intensive grazing and housing and mixing of animals from different sources. The clinical signs of the disease include a highly fatal septicemia in foals, acute projectile, profuse and foul smelling diarrhea and dysentery, fever and marked dehydration. Other clinical signs include toxemia, severe acute fulminating enteritis, arthritis, and dry gangrene of extremities, severe diarrhea and dehydration.

There is leucopenia, neutropenia and increases in serum gamma glutamyl transferase and sorbitol dehydrogenase activities and increased serum total bilirubin in affected horses (Palmer, 1987a).

Tuberculosis

Horses are usually highly resistant to the development of this disease. It is caused by *Mycobacterium bovis*. It is a disease of great economic and public health importance especially in developing countries where horses are slaughtered for meat. The mode of transmission is through inhalation or ingestion of contaminated feed and water or when feces contaminate pasture. The principal mode of transmission is by inhalation of infected droplets. The disease is characterized by painful osteomyelitis which causes stiffness of the neck and inability to eat off the ground. Other signs include coughing due to pulmonary lesions, enlarged lymph nodes, nasal discharge and intermittent fever. Necropsy findings include tubercles on the intestinal wall, mesenteric lymph nodes, spleen and cervical vertebrae (Radostits *et al*., 2007).

In available literature, there are no reports of clinicopathological findings associated with the *M. bovis* infection in horses, but it has been reported that *Mycobacterium avium hominissuis* (the causative agent of avian mycobacteriosis) infection in horses led to thrombocytosis, hypoalbuminemia, increased serum activities of alkaline phosphatase and lactate dehydrogenase, decreased packed cell volume and presence of band neutrophil in peripheral blood (Blahutkova *et al*., 2011). *M. ulcerans* infection in horses led to a mild decrease in the serum levels of sodium, creatinine and inorganic phosphate and increased serum activity of gamma glutamyl transferase (Van Zyl *et al*., 2010).

Actinobacillosis of foals (sleepy foal disease)

Actinobacillosis (formerly known as shigellosis) is an acute highly fatal septicemic disease of foals, caused by *Actinobacillus equuli*. It is an important cause of neonatal deaths in foals. Although the disease is limited to foals, septicemia and peritonitis has been reported in older animals. The predilection sites of the organism are the cervix and pharynx of mares. The clinical findings include fever, prostration, diarrhea, dysentery, rapid respiration and cessation of nursing of foals. Foals are usually sleepy or comatose at birth; death follows within 24 hours if not treated. Occasionally, severe abdominal pain, arthritis with swollen joints and lameness are seen as the disease progresses.

Hematological examination may reveal leukopenia with a severe left shift, hyperfibrinogenemia and low plasma immunoglobulin G (IgG). Clinical chemistry findings include hyponatremia and hypoglycemia. Joint fluid cytology may show an increased white blood cell count, and increased protein level in synovial fluid (Radostits *et al*., 2007).

Leptospirosis

This is an acute, subacute and/or chronic bacterial disease of horses characterized by fever, acute hemolytic anemia, still birth, abortion and periodic ophthalmia. It is caused by *Leptospira interrogans* with many distinct serovars (Kmety and Dikken, 1993) and transmitted by contaminated feed, water and pasture by infective urine. The disease is of public health concern. The most important factor for the persistence of the organisms is ground surface moisture. The pathological lesions of the disease are anemia, jaundice, hemoglobinuria, serosal hemorrhages and autolysis of aborted fetuses, fetal hepatitis and nephritis.

Clinico-pathological examination usually reveals hemolytic anemia, increased erythrocytic fragility, hemoglobinuria, mild leukocytosis and albuminuria (Radostits *et al*., 2007).

Viral diseases

The outcome of viral infections depends on factors such as the specific virus and its cell tropism, genetic resistance of the species or breed affected, immune response, age, nutritional status and hormone levels of the affected animal (Jones *et al*., 1997). Viruses are able to gain entry into a susceptible host through wounds on the body or get inoculated by arthropods and penetrate the epithelial lining cells of the skin which serves as barrier. Viruses are classified as being cytolytic or non-cytolytic. Cytolytic viruses cause cellular injury by interfering with nutrient synthesis or redirect host cell organelles to preferentially produce viral DNA. Non-cytolytic viruses encourage production of their antigens on the surface of the host cells while inside the cells. This can therefore lead to lysis of the cells by the antibodies produced against the antigens since the antibodies are not able to recognize the cells as self. Viruses are also classified based on their target organs/tissues and therefore can be said to be epitheliotropic or neutrotropic etc. Most viruses cause disease by replicating inside host cells leading to degeneration and death of the affected cells (Cheville, 2006).

African horse sickness

African horse sickness is an acute or subacute infectious, non-contagious arthropod-borne disease of equids caused by RNA orbivirus of the family Reoviridae and characterized by clinical signs which develop as a result of damage to the circulatory and respiratory systems. It is the most important viral disease of equids; it causes widespread mortality and morbidity especially in horses, as they appear to be more susceptible to the disease than donkeys and mules (Powell, 1987). The disease is mechanically transmitted by the bite of hematophagous insects including culicoides species, mosquitoes (anopheles, culex, and aedes), ticks (brown dog tick and hyalomma spp.), and biting flies such as *Stomoxys calcitrans* and tabanids, infected by the African horse sickness virus (Mellor and Boorman, 1995). The disease has been reported to cause encephalitis and chorioretinitis in humans (Van der Meyden *et al*., 1992). There are four forms of the disease in equids: horse sickness fever (mild form), pulmonary, cardiac/oedematous and mixed forms (pulmonary and cardiac). Horse sickness fever, which is the most common form in African donkeys, Zebra and Nigerian indigenous horses, is characterized by a mild to moderate fever and oedema of the supraorbital fossae. The acute pulmonary form (dunkop) is characterized by acute dyspnea, profuse nasal and oral yellowish serous discharges and froth, severe paroxysms of coughing, and

rapid respiratory rate, which becomes abdominal in character as the disease progresses. An affected animal usually collapses and dies as a result of respiratory distress. The subacute cardiac form (dikkop) is characterized by pyrexia, bulging of the eyeballs, oedematous swellings of the neck, brisket and forelimbs. The skin is stretched, wrinkles disappear, and head and neck assume an unusual rounded appearance. The oedematous lesions are painless on palpation. There is also anorexia and conjunctivitis. Death is preceded by a prolonged recumbency, muscular trembling and sweating. The mixed form has the characteristics of both pulmonary and cardiac forms.

Leukopenia, with lymphopenia, neutropenia and a left shift and mild thrombocytopenia are characteristic of the acute forms of African horse sickness (Mellor, 1994). The biochemical abnormalities include increases in serum creatine kinase, lactate dehydrogenase and alkaline phosphatase activities and creatinine and bilirubin concentrations. There is evidence of activation of the coagulation cascade and the fibrinolysis although disseminated intravascular coagulation is unusual (Mellor, 1994; Mellor and Hamblin, 2004; Radostits *et al*., 2007).

Equine infectious anemia

Equine infectious anemia is an acute or chronic lifelong multi-systemic disease of equidae characterized by intermittent fever, anemia, icterus, oedema, emaciation and death (Warner and Morris, 1987). It is caused by a retrovirus. The virus is usually present in all tissues and body fluids of infected animals. Importation of apparently healthy carriers is the usual means of introduction the virus to clean areas. Blood sucking arthropods (mosquitoes and biting flies) and contaminated instruments spread the disease mechanically. Also, venereal and intra-uterine transmission can occur. The clinical features of the disease include lacrimation, rhinorrhea, epistaxis, congested mucous membrane, petechial hemorrhages under the tongue, anemia, subcutaneous oedema involving the legs and ventral part of the abdomen, emaciation and incoordination. The gross pathological findings are splenomegaly, hepatomegaly, subcutaneous oedema and wide spread serosal and mucosal hemorrhages.

Haematological examination of horses with the acute disease reveals a moderate to marked thrombocytopenia which occurs concurrently with the fever and an anemia that may be severe (Warner and Morris, 1987). Thrombocytopenia also occurs during relapses of the disease, and may be sufficiently low that it allows petechial hemorrhages to develop. The anemia is the normocytic normochromic type and may become unresponsive in horses as the disease becomes chronic. Mild neutropenia, lymphocytosis and monocytosis accompany the clinical disease. The presence of sideroleukocytes (leukocytes containing hemosiderin) are considered highly suggestive of equine infectious anemia (Tornquist and Crawford, 1997). There are no characteristic changes in the white blood cell count. Serum biochemical examination may reveal an increase in bilirubin concentration due to rapid extravascular hemolysis, a decrease in serum iron concentration, increase in serum activity of gamma glutamyl transferase and alkaline phosphatase. Hypergammaglobulinemia results from chronic viral antigenic stimulation (Warner and Morris, 1987; Radostits *et al*., 2007).

Equine viral arteritis

Equine viral arteritis is a viral disease of horses and mules caused by a *Pestivirus* of the family Togaviridae (Jones *et al*., 1997). Horses of all ages are susceptible, but the disease is more severe in new born foals. The disease is transmitted by horizontal or venereal means. Horizontally, horses are infected through contact with infected nasal discharge and body fluids. Infected stallions subsequently excrete the virus in their semen and infect susceptible mares at mating hence this could be a venereal disease. The disease is of great economic importance due to loss of foals through abortion. The clinical signs include fever, nasal discharge (serous to purulent), congestion and petechiations of the nasal mucosa and conjunctiva, urticaria, conjunctivitis, excessive respiratory distress, diarrhea, jaundice, abdominal pain, oedema of the limbs, dehydration, muscle weakness and prostration.

Clinicopathological findings associated with equine viral arteritis include a thrombocytopenia, slight leukocytosis with neutrophilia, lymphopenia and eosinopenia, increase in plasma fibrinogen and a small rise in serum proteins and indirect bilirubin values (Monreal *et al.,* 1995).

Equine influenza

Equine influenza is a respiratory disease characterized by fever, anorexia, depression, dry and hacking cough, dyspnoea, serous/watery nasal discharge and reluctance to move (Jones *et al*., 1997). It is caused by infection with influenza A/equine-1 (H7N7) or influenza A/equine-2 (H3N8) virus which is an orthomyxovirus. The disease is transmitted by direct contact, inhalation of aerosols from infected material and on fomites (veterinary clothing, equipment or vehicles). All age groups of horses are susceptible. Equine influenza is of economic importance in race horses because of the inconvenience it causes to them. The disease is further complicated by stress (transportation, work and adverse climatic changes) and secondary bacterial infection by *Streptococcus zooepidemicus.* The clinical signs of the disease include painful submaxillary lymph nodes on palpation, abnormal lung sounds and reduced endurance of horses when forced to exercise. There are no specific characteristic haematological or serum biochemical changes associated with the disease in available literature (Radostits *et al*., 2007).

Viral encephalomyelitis of horses (Staggers, Borna disease)

Viral encephalomyelitis is an arthropod borne viral disease of horses caused by eastern equine encephalomyelitis virus (EEE), western equine encephalomyelitis virus (WEE) and Venezuelan equine encephalomyelitis virus (VEE) belonging to the family Togaviridae. It is transmitted through the bite of mosquitoes. The disease is of zoonotic importance (Elvinger *et al*., 1994). Affected horses exhibit signs associated with central nervous system derangement such as head pressing, incoordination, opisthotonus, paddling, recumbency and death. Haematological examination usually reveals only leukopenia. The major pathological lesion seen is that of nonsuppurative encephalomyelitis (Radostits *et al*., 2007).

Horse rabies

Rabies is acute viral encephalitis of horses caused by Lyssavirus of the family Rhabdoviridae. It is usually transmitted to the horse through a bite from wild carnivores (skunks,

raccoon and foxes) or infected insectivorous bats or vampire bats. The virus gains entry into the host via contamination of an open wound or abrasion on the skin with infected saliva. The signs of rabies include hyperesthesia at the site of inoculation, lameness, ataxia, paresis, incontinence, dysuria, muscle spasms, dysphagia, uncharacteristic vocalizations, peripheral radial nerve paralysis and colic. Some infected horses may exhibit dullness and depression and appear unresponsive to environmental stimulus followed in a few days by recumbency and death.

The clinicopathological abnormalities associated with the disease include elevated packed cell volume and total protein reflecting dehydration (hyperproteinemia) hypovolemia, increased serum aspartate aminotransferase and creatine phosphorkinase activities (Radostits *et al*., 2007).

Rickettsial disease

Equine monocytic ehrlichiosis (potomac horse fever)

Equine monocytic ehrlichiosis is an infectious, non-contagious sporadic disease of horses caused by *Neorickettsia* (*Ehrlichia*) *risticii* (Goetz *et al*., 1989; CFSPH, 2013). The mode of transmission is unknown, but the disease can be transmitted experimentally to horses by intra-dermal or intra-venous administration of Ehrlichia-infected blood to uninfected horses (Whitlock *et al*., 1984; Perry *et al*., 1985). Equine monocytic ehrlichiosis is the only rickettsial disease that causes enterocolitis and the only enteric disease that is not transmitted by the fecal-oral route (Palmer, 1987b). The clinical signs of the disease include fever, tachycardia, depression, anorexia, enterocolitis, diarrhea, colic, laminitis, congested mucous membrane and abortion. Fluid loss may lead to dehydration and hypovolemic shock. Enlarged mammary gland and retained placenta also occur present in affected female animals.

Haematological examination of affected animals usually reveals leukopenia with neutropenia and a marked left shift, lymphopenia, mild thrombocytopenia and hemoconcentration. Serum biochemical aberrations associated with the disease include hyponatremia, hyperkalemia, hypochloremia, metabolic acidosis and azotemia (Radostits *et al*., 2007).

Protozoan diseases

Pathogenic protozoa are usually associated with diseases of the blood, macrophage-monocyte system, or intestinal tract. Protozoa of veterinary importance include the trypanosomes, toxoplasmas, piroplasmas (babesia, plasmodia and other blood cell parasites) and amoebas. Protozoans usually require a vector (arthropods or flies). They produce disease by killing the host cells when they elaborate a wide range of virulence factors (toxins). They also cause disease when they replicate inside host cells and block the cell's metabolic activity, leading to cell death by lysis. Some protozoa may attach to the epithelial cells of the intestine in large numbers leading to severe cellular damage, malabsorption and diarrhea (Cheville, 2006).

Equine piroplasmosis

Equine piroplasmosis is an infectious disease of horses, donkeys, mules and zebra caused by the haemoprotozoan parasites, *Babesia caballi* or *Theileria equi* transmitted naturally by blood sucking ticks (Knowles, 2010). It may also be transmitted mechanically by unsanitary veterinary practices. *Babesia caballi* is transmitted by the horse tick, *Dermacenter nitens*, while *T. equi* is transmitted by numerous species of ticks, *Rhipicephalous sanguineous* and *Boophilus* spp., being the most likely vector (Stiller *et al*., 2002). The clinical signs of the disease include fever, depression, sudden onset of immobility, anorexia, tachypnea, icteric mucous membrane, ecchymosis of the nictating membrane, oedema of the head, ventral abdomen and fetlocks and colic (Garba *et al*., 2011). In stress conditions (work, racing etc.) the performance of the affected horses or donkey is lowered (Kumar *et al*., 2009). Haemoglobinuria is often present and usually accompanies the severe haemolysis of the parasitized red cells. The clinical signs associated with *T. equi* infection are much more severe. Death of affected horses is usually due to hypoxia during the haemolytic crises. Necropsy findings are splenomegaly, hepatic centrilobular necrosis, excessive fluid in serous cavities and bone marrow hyperplasia.

Clinicopathological findings associated with equine babesiosis include a severe anemia with low RBC count and haemoglobin concentration, leukocytosis, thrombocytopenia, haemoglobinuria, hyperfibrinogenemia and hyperbilirubinemia (Radostits *et al*., 2007; Lavoie and Hinchcliff, 2008; Garba *et al*., 2011). Clinicopathological findings associated with equine theilerosis include an increase in serum urea nitrogen, globulin, lactate and fibrinogen, decrease in serum conjugated bilirubin and iron, increase in serum total protein and bile acid, decrease in serum glucose, increase in sodium electrolyte and a decrease in organic phosphate, macrocytic normochromic anemia and lymphopenia (Takeet *et al*., 2009).

Trypanosomoses

Trypanosomoses is a group of diseases caused by parasitic trypanosomes. It is of great economic importance in animals and of public health importance as it causes human sleeping sickness. Parasitic trypanosomes of animals include *Trypanosoma vivax*, *T. congolense*, *T. brucei brucei*, *T. equinum*, *T. equiperdum* and *T. evansi*. A disease condition known as Nagana (African trypanosomosis) is caused by *T. congolense* and *T. brucei brucei* in equines whose clinical features are anemia, tissue damage and immunosuppression, while Surra is caused by *T. vivax* and *T. evansi.* These trypanosomes are cyclically transmitted by tsetse fly and mechanically transmitted by other biting flies such as *Tabanus spp*. and *Stomoxys calcitrans.* Of all the trypanosomes, *T. brucei brucei* is highly virulent and characterized by development of subcutaneous oedema on the ventral abdominal wall, thorax and limbs, keratoconjuctivitis, ataxia and paralysis. *Trypanosoma vivax* or *T. congolense* usually produce the chronic form and may be cured spontaneously in local breeds (Taylor and L-Authie, 2004).

Horses develop patent infections depending on the strain and species of the infecting trypanosomes, and number of trypanosomes introduced. Clinical features of African trypanosomosis (Nagana) include dullness, long starry hair coat, cachexia and exhaustion. Superficial lymph nodes are enlarged and prominent. There is diarrhea and watery ocular discharge. Infection with *T. vivax* is characterized by photophobia, excessive lacrimation with pale and jaundiced conjunctivae. Horses with *T. brucei* infection may show corneal opacity with accumulated pus in the anterior chamber of the eye, frequent respiration and pulse as well as pounding of

the heart, oedema of the lymph nodes, limbs, ventral surface of the abdomen, scrotum or vulva, staggering and prostration. Donkeys has some degree of resistance to the infection but may be compromised by any form of stress.

Clinicopathological findings associated with African trypanosomosis include anemia of the haemolytic type, leucopenia, thrombocytopenia, increased erythrocyte sedimentation rate, monocytosis, increased serum activities of alanine aminotransferase and aspartate aminotransferase, hypoglycemia, elevated blood urea nitrogen, hypoalbuminemia and hypergammaglobulinemia (Anosa, 1988a,b; Agina and Ihedioha, 2016a).

Coccidiosis

Eimeria leuckarti, a ubiquitous protozoal parasite is the cause of coccidiosis in horses especially foals (Lyons *et al*., 1988). The mode of transmission is by ingestion of food and water contaminated by feces of clinically affected or carrier horses, or by licking the hair coat contaminated by the feces. The nutritional status of the animal and age are the important risk factors that predispose an animal to the disease. Coccidiosis is characterized by diarrhea, acute massive intestinal hemorrhage leading to death in foals and young horses (Lyons *et al*., 1988). There are no reports of clinicopathological findings associated with the disease in available literature.

Fungal diseases

Fungal diseases are usually caused by the pathogenic yeasts and dermatophytes. They are single-celled nucleated plant organisms and are known to cause three types of diseases in animals: mycosis, allergic disease and mycotoxicosis. Mycosis occurs when there is direct invasion of tissue of susceptible animals by pathogenic fungi. Animals infected by pathogenic fungi may develop hypersensitivity to fungal antigens (allergic disease). Animals may also ingest toxic fungal metabolites leading to mycotoxicoxis. Factors that favor the establishment of fungal infections in animals include immunosuppression, inhibition of mechanisms of inflammation, alteration of host protective bacteria microflora and stress (Cheville, 2006).

Dermatophytosis

Dermatophytoses are fungal diseases of superficial keratinized layers of the skin and appendages such as hair and nails caused by *Trichophyton equinum, T. quinckeanum, T. mentagrophytes, T. verrucosum, Microsporum equinum* and *M. gypseum* (Radostits *et al*., 2007). Dermatophytes are spread by close contact with an infected animal, and affects children and adult humans, and the treatment is usually difficult (Pier *et al.,* 1994). The fungi chiefly attack keratinized tissues, particularly the stratum corneum and hair fibres, resulting in autolysis of the fibre structure, breaking off of the hair, and the appearance of characteristic round sharply circumscribed thick elephant skin-like plaque. Exudation from invaded epithelial layers, epithelial debris and fungal hyphae produce the dry crusts which are characteristic of the disease. It mostly affects areas of the skin around the axillary girth area, trunk, rump, eyes, ears, mouth, neck and limbs. There are no reports of haematological and serum biochemistry findings associated with the disease in available literature.

Histoplasmosis

Histoplasmosis is a fungal disease characterized by pneumonia with dyspnoea and nasal discharge, placentitis with abortion, hepatic insufficiency with jaundice and anarsaca, and widespread lesions in neonates (Rezabek *et al*., 1993). It is caused by *Histoplasma capsulatum,* which is transmitted mainly by inhalation of contaminated dust. Necropsy findings about the disease include hepatomegaly with necrotic foci, enlarged splanchnic lymph nodes, and granulomatous pneumonia. Histopathology usually reveals aggregated fungal bodies with large numbers of macrophages in lymphoid tissue, which is characteristic of the disease.

Serum biochemical analysis reveals hypoalbuminemia, hyperglobulinemia, hyperphosphatemia, low ionized calcium, hyponatremia, hyperphosphatemia and hyperchloremia (Nunes *et al*., 2006).

Mycotic pneumonia (pulmonary aspergillosis)

Mycotic pneumonia is a respiratory disease of horses characterized by fever, tachycardia, weight loss, nasal discharge, laminitis and uveitis (Blue *et al*., 1987; Buechner-Maxwell *et al*., 1994). The disease is caused by *Aspergillus* spp. and has been reported in horses on prolonged anti-microbial therapy (Ruoff, 1988). Mycotic pneumonia affects horses on glucocorticoid treatment and those with an overwhelming challenge by Aspergillus species (Ruoff, 1988). It is also seen in cases of hypercortisolemia associated with pituitary adenomas, ulcerative enterocolitis and myeloproliferative neoplasia (King, 1993; Pace *et al*., 1994; Buechner-Maxwell *et al*., 1994). The clinical signs of the disease include fever, lethargy, inappetence, slight tachypnea, increased respiratory rate, moderate increased bronchoalveolar sounds, recumbency and death. Post-mortem examination reveals a large number of nodules disseminated throughout the lungs. Histopathological examination of the lung tissue reveals multifocal accumulation of neutrophils and macrophages with bronchioles and adjacent alveoli. There are large areas of necrosis and infiltration into alveolar spaces by neutrophils and substantial deposits of fibrin.

The haematological abnormalities associated with the disease include low packed cell volume, low haemoglobin concentration, low red blood cell count, leukocytosis with a neutrophilia and lymphopenia (Johnson *et al*., 1999). Serum biochemical findings associated with the disease included increased activities of creatine kinase and lactate dehydrogenase. Further cerebrospinal fluid analysis revealed a slight increase in total protein concentration and a normal nucleated cell count and few erythrocytes (Johnson *et al*., 1999).

Coccidioidomycosis (Valley fever)

Coccidioidomycosis is a non-contagious and benign disease of horses characterized by weight loss, severe emaciation, persistent cough, wheezing, increased breath sounds, muscle pain and superficial abscesses on the pectoral area which often recur. It is a disease of zoonotic importance caused by *Coccidioides immitis* (Ziemer *et al*., 1992). The mode of transmission is by inhalation of fungal spores, by ingestion and also through cutaneous abrasions. Other clinical signs of the disease include oedema of the legs, anemia and intermittent colic due to internal abscess and peritoneal adhesions, and granulomas in the mesenteric lymph nodes.

Clinicopathological examination usually reveals a leukocytosis with neutrophilia, anemia, hyperfibrinogenemia and hyperproteinemia (Lavoie and Hinchcliff, 2008).

Equine phycomycosis (swamp cancer)

Equine phycomycosis is a fungal disease of horses characterized by anemia, pruritis, lameness and oedema. It is caused by *Pithyium insidious*, *Basidiobolus haptosporus* or *Conidiobolus coronatus*. These organisms cause pyogranulomatous lesions on the limbs, ventral abdomen, thorax, neck and head, oral and nasal passages, and pharyngeal mucosae. The ulcerated granulomas contain yellow concretions in the sinus tract of bones causing osteomyelitis, this usually occurs in the chronic pythiosis of the lower limbs (Fadok, 1992).

The haematological and serum biochemistry findings include normocytic normochromic anemia, leukocytosis, hypoproteinemia and hypoalbuminemia (Dowling *et al*., 1999).

Diseases caused by helminth and arthropod parasites

Strongylosis (Red worm infestation)

Strongylosis is a nematode infestation of horses characterized by anemia (due to blood sucking adult worms) and verminous arteritis, thrombosis and thickening of the arterial wall (as a result of strongyle larval migration) (Radostits *et al*., 2007). The disease is caused by *Strongylus vulgaris, S. edentatus* and *S. equinus*. Emboli may break away from the thrombi and lodge in smaller blood vessels leading to partial or complete ischemia in the part of the intestine thus producing colic. Necrosis or gangrene may set in. Intussusceptions, volvolus or torsion may occur occasionally. Some larvae of the strongyle worms cause formation of superficial or submucosal nodules. Affected foals are weak, unthrifty and perform poorly and lose weight. There is fever, depression and diarrhea. Adult mares that are in late pregnancy may have increased heart and respiratory rates, increased intestinal sounds and can even suffer an abortion. They may further become recumbent and die.

Haematological examination reveals a decrease in haemoglobin concentration, erythrocyte count and packed cell volume. The leukocytosis seen is usually a sign of heavy infestation while eosinophilia reflects the presence of migrating larvae. Serum biochemical analysis usually reveals a marked increase in beta-globulins and a decrease in albumin (Radostits *et al*., 2007).

Lungworm infestation

Lungworm infestation in horses is characterized by a chronic irritating cough with no nasal discharge, increased respiratory rate, forced expiration and afebrile symptoms in experimental conditions. It is caused by a nematode parasite, *Dictyocalus arnfieldi*, which is transmitted by ingestion of herbage that contained the infective larvae. Foals may be asymptomatic, but some may show respiratory symptoms. Post-mortem examination reveals discrete areas of over-inflation surrounding blocked bronchi with worms and greenish mucus.

Eosinophils and sometimes egg or larvae may be confirmed in tracheal mucus. Heamatological examination reveals a leukocytosis, neutrophilia with degenerative left shift (Sharifi *et al*., 2010)

Ascarid infestation

Ascarid (roundworm) infestation of horses and donkeys are characterized by digestive disturbances and poor growth in young horses, cough, fever and mucopurulent discharge. It is caused by the nematode parasite, *Parascaris equorum*. Young horses are more seriously affected than adult horses. The clinical signs of the ascariasis include poor hair coat, diarrhea and sometimes colic, convulsions, obstructive jaundice, intestinal obstruction and perforation or rupture which may occur in foals. No clinical signs are observed in affected older horses, but they continue to contaminate the environment.

Clinicopathological examination reveals the presence of large numbers of characteristic eggs in feces and a marked eosinophilia in blood during the larval migration, leukopenia and mild anemia (Lavoie and Hinchcliff, 2008). In severe cases, hypoproteinemia may occur (Lavoie and Hinchcliff, 2008).

Onchocerciasis (worm nodular disease)

Onchocerciasis is a roundworm infestation of horses caused by the filarial worm, *Onchocerca cervicalis* found mostly as convulated, fibrotic and causes lesions in ligamentum nuchae (large ligament that runs from poll to withers). Horses are infested by the bite of an intermediate host, which was infected by the microfilariae (culicoides and simulium, biting midges and blackfly) during their blood meal. The disease is characterized by alopecia, scaliness, pruritis, recurrent uveitis (periodic ophthalmia), hypersensitive reaction on the affected areas of the face, withers, lower abdominal wall, and fore and hind limbs (Foil *et al*., 1990).

The haematological and serum biochemistry findings associated with onchocerciasis (recurrent uveitis) include anemia, leucopenia, thrombocytopenia and hyperproteinemia (Hughes, 2010).

Hepatic fascioliasis

Hepatic fascioliasis is a worm infestation of horses caused by the trematode, *Fasciola gigantica* and *F. hepatica*. The mode of transmission is by ingestion of infective metacercariae on herbage released by the intermediate host, lymnaeid mud snails. Horses are less commonly infected and overcome the migrating flukes at an early stage, such that few reach the liver (Radostits *et al*., 2007). Clinical and necropsy findings of acute hepatic fascioliasis include emaciation, staggering gait, petechial haemorrhage under the tongue, hydrothorax, ascites, peritonitis, enlarged spleen and in some cases, enlarged kidneys (Kralj *et al*., 1960).

Haematological examination reveals a low red blood cell count and accelerated erythrocyte sedimentation rate, leukocytosis, neutrophilia, eosinophilia, and in some cases, lymphopenia and a reduction in serum cholinesterase activity (Kralj *et al*., 1960). Biochemistry abnormalities include an increase in plasma activities of glutamate dehydrogenase and gamma glutamy transferase (Soulé *et al*., 1989).

Tapeworm infestation

Tapeworm infestation is an acute or chronic disease of horses characterized by colic, diarrhea, unthriftiness and weight loss. It is caused by cestodes belonging to Anoplocephalid family. They include *Anoplocephala perfoliata, A. magna* and *Paraoplocephala mamillana*. Horses get infested by accidental ingestion of infected oribatid mites while grazing. Post mortem

examination reveals mild inflammation of intestinal mucosa with small ulcers. There are no reports of haematological and serum biochemistry findings associated with adult tapeworm infestation in horses in the available literature.

Louse infestation

Louse infestation is characterized by skin irritation, intense pruritis with attendant excoriation, and loss of hair coat, rubbing, licking and restlessness. It is caused by the ectoparasites *Werneckiella equi* (*Damalina equi*) (biting lice) and *Haematopinus asini* (sucking lice). *W. equi* is commonly found on the head, mane and base of the tail and feeds on sloughed epidermal tissues and sebaceous secretions on the surface of the skin (Larsen *et al*., 2005), while *H. asini* is most often found on the head, neck, back and inner thighs, and feeds on blood (Wright, 1999). Allergens present in the saliva and feces of the lice cause severe irritation, leading to pruritis and hyperkeratosis (Wright, 1999), and in heavy infestations, weight loss and self-trauma (Gray, 1995). Heavy infestation occurs in horses kept in poor surroundings, or with other diseases (Gawler *et al*., 2005).

The haematological finding associated with severe louse infestation is anemia (Radostits *et al*., 2007).

Tick infestation

Tick infestation may be caused by any of the species of ticks, which may be the soft ticks (*Argasidae*) and/or hard ticks (*Ixodidae*). Ticks act as vectors of diseases such as equine piroplasmosis - *Babesia caballi* and *Theileria equi*, and equine borreliosis, *Borrelia burgdorferi*. Ticks also transmit the causative agent of equine Potomac fever, *E. risticii* and, their bites can be a portal of entry for bacteria and a site of myiasis. The clinical signs associated with tick infestation in horses include anemia, paralysis, loss of production in farm animals, general unthriftiness, susceptibility to disease, tick fever and tick worry, and skin damage due to biting and rubbing (Radostits *et al*., 2007).

Haematological finding associated with severe tick infestation is anemia and leukocytosis.

Mite infestation

Mites of importance in horses include sarcoptic mange mite, *Sarcoptes scabei* var. *equi*; scab mites, *Psoroptes cuniculi* (ear mite), *P. equi* and *P. ovis*, *Chorioptes equi* and Demodectic mites, *Demodex caballi*. These mites are spread from one horse to another through body contact or fomites (grooming instruments, harness). The sarcoptic mites burrow into skin, live in tunnels in the epidermis and feed on serum and cellular components. Their lesions are usually located on the head, neck and ear. Psoroptic lesions are present at the base of long hairs of the mane, tail and hairless areas such as mammary gland prepuce and axilla. There is alopecia and papular formation which become moist, haemorrhagic or crusty. *Psoroptes cuniculi* infestations in horses cause severe irritation in the ear accompanied by discharge, shaking and rubbing of the head, and tenderness of the poll. Chorioptic mites live on the surface of the skin and feed on skin debris. Lesions associated with these mites consist of a scaling, papular eruption usually limited to the lower parts of the limbs and back of pastern (Foil and Foil, 1992; Klei, 1992; Radostits *et al*., 2007). Demodectic mange (follicular mange) is a rare disease in horses characterized by presence of patchy or scaly alopecia. It is caused by *Demodex equi* and *D. caballi*, which infest hair follicles. *D. caballi* occurs in the Meibomian glands of the eyelids and pilosebaceous apparatus of the skin of the muzzle. Demodicosis affects horses of all ages and is usually associated with a debilitating illness or glucocorticoid therapy (Foil and Foil, 1992; Radostits *et al*., 2007).

Nutritional diseases

Energy deficiency

Energy deficiency is the most common nutrient deficiency that limits performance of farm animals due to low availability of food or low quality of food that make the animal unable to meet the energy requirement. It is the presence of carbohydrate in suboptimal quantities in food and it is characterized by underdeveloped growth and delay in the onset of puberty in foals. Mature mares exhibit marked decline in milk production, shortened lactation, marked body weight loss, prolonged periods of anestrus, birth of undersized weak neonates with a high mortality rate, and impaction of the intestines of horses fed poor quality roughage. There is also weakness, recumbency and death.

Hyperlipemia is also evident in fat pregnant or lactating ponies (Radostits *et al*., 2007).

Protein deficiency

Protein deficiency occurs when the food contains suboptimal quantity of proteins. This usually accompanies a deficiency of energy. Deficiency of protein is characterized by reduced appetite, lowered food intake, poor growth rate, lack of muscle development and prolonged time to reach maturity in young horses. In mature mares, there is loss of weight, decreased milk production and oedema.

Clinicopathological analysis reveals decreased haemoglobin concentration, low packed cell volume, low serum total protein and albumin (Stockham and Scott, 2008).

Vitamin deficiencies

Hypothiaminosis

Hypothiaminosis is a vitamin deficiency of horses characterized chiefly by nervous signs. Thiamin deficiency can be primary or secondary. Primary thiamin deficiency occurs as a result of absence of thiamin or presence of very low levels of thiamin in diet. Secondary thiamin deficiency occurs due to ingestion in excess of some poisonous plants such as bracken fern (*Pteridium aquilinum*) and horsetail (*Equisetum arvense*), which contain an enzyme, thiaminase (thiamin antagonist). Horses fed coccidiostat amprolium and large quantities of turnips (*Beta vulgaris*) without grain, may manifest the clinical illness. Secondary thiamin deficiency is characterized by bradycardia, sway gait, pronounced incoordination (i.e. crossing of the forelimbs and wide action in the hindlimbs), muscle tremor, clonic convulsion and opisthotonus, inappetence. Temperature and heart rate are usually normal until the terminal period of the illness. There is also hemiplegia of the vocal cords, bradycardia with dropped heart beats, ataxia, muscle fasciculation, blindness, and diarrhea and weight loss. Necropsy findings indicate a non-specific congestive heart failure, interstitial oedema of the myocardium, liver and intestinal lesions.

Blood chemistry analysis reveals an increase in blood pyruvic acid and decrease in thiamin levels (Radostits *et al*., 2007).

Vitamin E / Selenium deficiency

Vitamin E/Selenium deficiency is associated with the ingestion of inferior quality hay, straw or root crops which are low in vitamin E or conditioning factors such as dietary polyunsaturated fatty acid diet such as cod liver oil, fish meal used as protein concentrate, linseed oil, soybean and corn oils. Vitamin E/Selenium deficiency causes the following syndromes in horses: acute and sub-acute forms of enzootic nutritional muscular dystrophy (NMD), equine degenerative myeloencephalopathy and equine motor disease. Enzootic nutritional muscular dystrophy occurs commonly in foals of about 7 months, born by mares fed for long periods on diets low in vitamin E and Selenium (Step *et al*., 1991). Dystrophic myodegeneration has been reported in adult horses (5-10 years) having vitamin E/Selenium deficiency (Step *et al*., 1991). The acute form of NMD (myocardial dystrophy) occurs mainly in foals. There are widespread lesions of degeneration of myocardial muscles (Kennedy and Rice, 1992). The clinical signs associated with acute NMD include collapse and sudden death after exercise without any obvious signs, lateral recumbency, and loss of neurological reflexes, increased thirst, cardiac arrhythmias, increased respiratory rate with loud lung sounds, and normal or slightly elevated temperature. Affected foals may die within hours after the onset of signs. In subacute muscular dystrophy, foals fail to suck, are recumbent and have difficulty in rising. They are unsteady and tremble when forced to stand. There is polypnea, tachycardia and normal temperature. Adult horses manifest a stiff gait, myoglobinuria, depression, inability to eat, oedema of the head and neck, and colic. At necropsy, cardiac muscles show visible white areas of degeneration, pulmonary congestion and oedema.

Clinical chemistry analysis reveal an increase in plasma creatine kinase (CK) and aspartate aminotransferase activities, and low serum levels of Selenium and vitamin E. Serial measurement of CK activity is commonly used to monitor the recovery of the animals and their response to therapy (Radostits *et al*., 2007).

Mineral deficiencies

Copper deficiency

This occurs principally in young horses and is characterized by unthriftiness, change in hair color, diarrhea, lameness, demyelination of the central nervous system in neonates and anemia as the condition advances. Copper deficiency may be primary or secondary. Primary copper deficiency is caused by an inadequate intake of copper. In secondary copper deficiency, there is adequate intake of copper, but the tissues do not utilize it. Secondary copper deficiency is also seen in horses grazing on forage grown on copper deficient soil. Factors that predisposes animals to copper deficiency include age, demands of pregnancy and lactation, stage of growth, mineral composition of the feed, season of the year (rainy season), breed and concentration of conditioning agents such as Sulphur and molybdenum which interferes with the availability of copper. Adult horses are usually unaffected by copper deficiency, but affected ones may show clinical signs such as ruptured aorta or uterine arteries, anemia, recurrent bacterial infections or depigmentation of skin and hair (Ralston, 1992). Affected foals exhibit unthriftiness and slow growth, stiffness of the limbs and enlarged joints, ataxia, and the central nervous system involvement is absent. Affected foals show these signs at birth or before weaning. Recovered foals are unthrifty up to 2 years of age (Radostits *et al*., 2007). Characteristic post mortem findings are those of anemia, emaciation and extensive deposits of hemosiderin in the liver, spleen and kidney.

Haematological examination reveals a decrease in haemoglobin concentration, and low red blood count (Radostits *et al*., 2007).

Calcium deficiency

This occurs due to consumption of food containing insufficient amount of calcium, or due to high phosphorus intake. This is usually seen in horses in training given artificial diets containing cereal or grass hay, and bran which contain little calcium and grains which have a high content of phosphorus. The clinical signs of the disease are more prominent in young horses. These include abnormal bone and cartilage development, shifting leg lameness, poor growth rate, dental maldevelopment. Other clinical signs of calcium deficiency include tetany, inappetence, stiff gait, fracture of long bones and specific disease syndrome including rickets, osteomalacia and osteodystrophia fibrosa (bran disease). Necropsy findings include severe osteoporosis, parathyroid gland hypertrophy, and thin cortical bone, reduction in size and number of metaphyseal trabeculae (Radostits *et al*., 2007).

A decrease in serum calcium concentration is seen in lactating mares and suckling foals. There is hyperphosphatemia and hypomagnesemia (Lavoie and Hinchcliff, 2008).

Osteodystrophia fibrosa

This is principally a disease of horses and other equids engaged in exercise (training) and racing, placed on artificial diets such as heavy grain, bran, grass or cereal hay with low calcium and high phosphorus content. It is caused by a secondary calcium deficiency due to excessive phosphorus feeding. It affects horses of all ages and affects most especially, those within the range of 2-7 years of age. Imbalance of calcium and phosphorus in blood triggers the parathyroid gland leading to hyperparathyroidism and a defective mineralization occurs (excessive bone resorption). This causes bone weakness and predisposition of such bones to fracture. Osteodystrophia fibrosa is characterized by a shifting lameness, arching of the back, and fracture of the lumbar vertebrae in race horses. Other features of the disease include local swelling of the margins of the mandible followed by a soft symmetrical enlargement and swelling of the facial bones, flattened ribs, swollen joints and curvature of long bones. Articular erosions, separation of muscular and tendinous attachment are also evident in osteodystrophia fibrosa. At post-mortem, the entire animal skeleton is abnormalmost pronounced in mandible, maxilla and nasal bones. Microscopically, there is increased osteoclast activity along bone trabeculae, proliferation of fibrous tissue, and enlarged parathyroid gland (Clarke *et al*., 1996).

Clinicopathological examination reveals a slightly decreased packed cell volume, lymphocytosis, low ionized plasma calcium level, high plasma inorganic phosphorus level, and increased serum alkaline phosphatase, lactate dehydrogenase, gamma glutamyl transferase and aspartate aminotransferase activities, increased serum plasma concentration of urea and

hyperphosphaturia (Benders *et al*., 2001; Stewart *et al*., 2010).

Genetic/inherited diseases

Genetic diseases may develop as a result of inheritance or gene mutation, either a *de novo* mutation or a mutation following environmental factors. A gene may be dominant or recessive; autosomal or sex-linked, therefore a genetic disease can be said to be inherited in an autosomal dominant manner, autosomal recessive manner, sex-linked dominant manner or sex-linked recessive manner. A disease that is inherited in an autosomal dominant pattern, homozygotes and heterozygotes express the mutant allele. Autosomal means that the gene is not situated on the sex chromosomes, thus both males and females are capable of transmitting the genetic disease. Autosomal recessive traits can be transmitted to their offspring by unaffected parents (carriers) or affected parents. In a X-linked dominant disease, the affected male parent can pass the genetic disease only to the female offspring, while an affected female parent can pass the trait to half of the offspring, male or female. Sex-linked recessive traits are usually carried on the X chromosome of males and all female offspring of the affected males will be carriers, and will transmit the recessive gene to one-half of their offspring (Cheville, 2006).

Inherited combined immunodeficiency (CID) in foals of Arabian breeding

This is an immunodeficiency of Arabian foals, inherited as an autosomal recessive defect. Affected foals are normal at birth and remain susceptible to all sorts of infections especially that of the respiratory tract. The disease is usually complicated by adenoviral pneumonia, bacterial and fungal infections. Affected foals are born with a combined immunodeficiency associated with a deficiency in both B-lymphocytes and T-lymphocytes that produce immunoglobulins and provide cellular immunity respectively. Affected foals manifest the clinical illness within 10-35 days from birth. Inherited combined immunodeficiency is characterized by a thick bilateral nasal discharge, unthriftiness and lethargy, deep dry cough, fever, increased heart and respiratory rate, diarrhea, alopecia and dermatitis. They die as a result of acute septicemia and/or recurrent or chronic continuous infection, and poor response to normally effective antibiotic therapy. Post mortem examination reveals a thymic, lymph node and splenic hypoplasia.

Haematological examination reveals a lymphopenia, which is a constant finding. There is a concurrent hypogammaglobulinemia (Radostits *et al*., 2007).

Equine degenerative myeloencephalopathy

This is an idiopathic, diffuse, degenerative disease of the spinal cord and some parts of the brain in foals and horses less than two years of age (Blythe and Craig, 1992). An increased requirement for vitamin E has been reported to be a contributory factor (Dill *et al*., 1990; Blythe *et al*., 1991). It usually affects most breeds of horses. The clinical signs are those of a slowly progressive spinal ataxia that stabilizes when the horse is 2-3 years of age. Affected young horses have symmetrical signs that are most severe in hindlimbs and ataxia characterized by pivoting, truncal sway and difficulty in movement such as backing or walking with the head elevated. They also adopt an abnormal posture when resting and do not spontaneously recover from the disease, but death is unusual. Histopathological examination reveals neuronal atrophy, accumulation of lipofuscin-like pigments, and diffuse gliosis.

No abnormalities are usually reported in the haematology, serum biochemistry and on cytological evaluation of the cerebrospinal fluid. Serum vitamin E concentration may be low or normal. No gross lesions are evident at necropsy (Blythe and Craig, 1992).

Equine hyperkalemic periodic paralysis

Equine hyperkalemic periodic paralysis is a disease caused by an inherited defect in sodium channel of skeletal muscles, characterized by an increase in serum sodium concentration. The disease is inherited in an autosomal codominant manner which means that when a heterozygote and a normal animal are bred, there is a chance that 50% of the offspring will carry the trait. In the case of two heterozygotes breeding, 75% of the offspring will be affected of. Heterozygotes are affected by the disease and manifest the clinical illness such as muscle fasciculation and tremor, weakness, flaccid paralysis and recumbency. They often sweat and thethird eyelid may prolapse. There may be stridor and dysphagia due to laryngeal and pharyngeal dysfunction (Carr *et al*., 1996). These clinical signs are more severe and frequent in affected homozygotes.

Serum biochemistry analysis reveals hyperkalemia (> 5.5 mEq/L; 5.5 mmol/L) during or immediately after episodes (Naylor, 1997).

Metabolic diseases

Lactation tetany of mares (eclampsia, transit tetany)

Lactation tetany of mares is a non-infectious disease of mares characterized chiefly by tetany and incoordination. It is caused by low levels of calcium in blood (hypocalcemia). The disease manifests in mares that graze on lush pasture, few days after foaling or one-two days after weaning. It is also seen in mares engaged in hard labor (physical exertion), and after transportation. The disease has also been reported in male ponies (Richardson, 1991). The clinical signs of the disease include profuse sweating, difficulty in movement due to tetany and incoordination, stiff gait and rapid labored breathing, wide dilated nostrils accompanied by a distinct thumping sound from the thorax. This is thought to be due to spasmodic contraction of the diaphragm, muscular fibrillation and trismus. Temperature may be normal or slightly elevated. Pulse is normal, but it becomes rapid and irregular as the condition advances. There is cessation of urination and defecation. Recumbency and death follows if untreated.

Serum biochemical examination reveals serum calcium level in the range of 4-6 mg/dl (1-1.50 mmol/L) in hypocalcemic condition (Radostits *et al*., 2007).

Equine hyperlipidemia

This is a metabolic disease of horses associated with insulin resistance and a derangement in fat metabolism (Watson and Love, 1994). The disease affects pregnant or lactating fat middle-aged mares; it is uncommon in stallions and geldings, and rare in foals (Mogg and Palmer, 1995). It also affects obese ponies, donkeys and American miniature horses. Transportation, food deprivation and overlying diseases in miniature horses can predispose an animal to the disease (Mogg and Palmer, 1995). Insulin resistance and stress causes an

unchecked mobilization of fatty acids from adipose tissue at a rate that exceeds the gluconeogenesis and ketogenic capacity of the liver. Excess fatty acids are re-esterified in the liver to triglycerides and released into the peripheral circulation as very low-density lipoproteins (VLDL). The clinical findings associated with hyperlipidemia include depression, weight loss, inappetence, muscle fasciculation, ventral oedema, somnolence, mania, mild colic (flank watching, stretching and rolling), jaundice, inability to swallow, normal temperature, increased heart and respiratory rate, and diarrhea, which is usually evident in the terminal stage.

Clinicopathological examination reveals a leukocytosis with neutrophilia, hyperlipidemia (serum triglyceride is 500 mg/dl or 5 mmol/L), elevated serum cholesterol, and free fatty acid concentration may be up to 1,200 mg/dl in ponies, low plasma glucose level and increased serum gamma glutamyl transferase activity (Mogg and Palmer, 1995). Increased serum creatinine and urea nitrogen levels are due to a decline in renal function. There is also increased blood clotting time and metabolic acidosis (Radostits *et al*., 2007). Visual examination of the plasma reveals cloudy, milky, mildly opalescent plasma.

Exertional rhabdomyolysis (Azoturia)

This is a sporadic disease of working horses worldwide caused by inherited anomalies of the muscle metabolism, hypothyroidism, hormonal imbalance, vitamin E and Selenium deficiency, electrolyte abnormalities (sodium and potassium deficiency), high carbohydrate diets, exercise at irregular intervals and viral infections (Valberg *et al*., 1994). The disease is almost always associated with exercise in thoroughbred, standardbred, event and endurance horses (Harris, 1991). The clinical findings include poor performance, stiff gait after exercise, reluctance to move, apprehension and anorexia, hard and painful muscles (gluteal muscle), profuse sweating, tachycardia or tachypnoea, hyperthermia, recumbency and death.

Clinicopathological examination reveals elevated serum activities of creatine phosphokinase, aspartate aminotransferase and lactate dehydrogenase, and increased serum myoglobin and carbonic anhydrase III in affected horses (Valberg *et al*., 1994; Nishita *et al*., 1995). There is decreased blood sodium, increased blood potassium and decreased blood chloride levels. There is also increased serum creatinine and urea nitrogen (azotemia), increased packed cell volume due to hemoconcentration that is attributed to dehydration, which is caused bysweating and increased serum total protein indicative of dehydration. Myoglobinuria is also evident. An important necropsy finding is a widespread degeneration of striated muscle (diaphragm and heart) (Radostits *et al*., 2007).

Equine Cushing's disease

Cushing's disease is a non-sporadic, non-infectious and non-contagious disease of older horses and ponies caused by a non-malignant functional tumor comprised of melanotropes of the pars intermedia of the pituitary gland (Radostits *et al*., 2007). It is characterized by polyuria, polydipsia, hirsutism (long, curly hair coat that is not shed during the warmer months), laminitis, hyperhidrosis, infertility, obesity characterized by excessive fat deposition in the crest of the neck and in supraorbital fossae; and blindness or seizures which occur in rare cases. Hirsutism is a constant and specific finding in affected horses.

Haematological examination reveals a mild neutrophilia and lymphopenia. Serum biochemical analyses reveal hyperglycemia and increase serum alkaline phosphatase activity. Glucosuria may be evident (Radostits *et al*., 2007).

Neoplastic diseases

Neoplastic diseases may occur when tissues grow abnormally and continuously in an uncontrolled manner. These neoplastic cells may closely resemble the host cells from which they arose. Their arrangement differs from epithelial cells or other cells of the body which are orderly structured. This is because their mitotic activity, differentiation and cell contact inhibition are abnormal, therefore they grow rapidly and compromise host tissue structure and function. Neoplasms can be benign or malignant (cancer). A malignant neoplasm is usually metastatic, invasive, anaplastic, undifferentiated and unencapsulated (Ihedioha, 2003; Cheville, 2006).

Lymphosarcoma

Lymphosarcomas are the most common tumor of horses and a common cause of neoplasia-associated deaths in horses. It has been reported that it affects horses between 4 months and 22 years. The aetiology is unknown. Four forms of the tumor exist, and they include generalized, alimentary, mediastinal and cutaneous forms. Common signs associated with the neoplastic disease include anorexia, weight loss, depression and ventral oedema. Lymphadenopathy, pyrexia and concurrent infections may also be present. Lymph nodes, liver, spleen, intestine, kidney and lungs are the most affected tissues in the generalized form of the lymphosarcoma. Other tissues/organs affected in the generalized form are upper respiratory tract, spinal cord, heart, reproductive organs, brain and retrobulbar spaces. The clinical signs seen in this form include icterus, neurological deficits, upper airway obstruction, tachycardia, dyspnea and localized lymphadenopathy. The alimentary form of lymphosarcoma mainly involves the small intestines, and to a lesser extent, the stomach and large colon. The mesenteric lymph nodes are also affected. The clinical signs associated with this form include anorexia, weight loss, and mild colic which predominate. Diarrhea occurs in rare occasions. The mediastinal form involves the mediastianal lymph nodes which enlarge and compress intrathoracic structures. The clinical signs include venous distension with jugular pulses, dyspnoea due to pleural effusion and/or tracheal compression. The cutaneous form comprises of multifocal firm subcutaneous nodules (Lavach, 1992). Histopathological examination of the affected tissue reveals neoplastic cells that closely resemble large, relatively well differentiated lymphocytes, with nuclear chromatin clumping, prominent nucleoli and cytoplasmic basophilia.

Clinicopathological findings include neutrophilia, lymphocytosis, hypoalbuminemia, hypergammaglobulinemia and hyperfibrinogenemia. Anemia of the normocytic normochromic type is also observed (Collatos, 1992).

Plasma cell myeloma

Plasma cell myeloma is a myeloproliferative disorder characterized by uncontrolled proliferation of a clone of plasma cells. It occurs rarely in horses, and affects horses within 3 months and 22 years. These clones of plasma cells produce a paraprotein (immunoglobulin) of no immunological

importance. The clinical manifestation of the neoplasm is associated with infiltration of organs/tissues by neoplastic cells or systemic of the immunoglobulin produced. The clinical signs of the disease include weight loss, anorexia, and renal disease, bleeding diathesis, neurological deficits and lameness. The latter is secondary to osteolysis via an osteoclast-activating factor produced by the myeloma or direct invasion of bones by neoplastic cells. Paraprotein in circulating blood can coat platelets and interfere with clotting factors, resulting in bleeding dyscrasias. Chronic infections usually result from a decrease in the production and function of normal immunoglobulins (Collatos, 1992).

Clinicopathological findings associated with the disease include anemia, macrocytic red blood cells, leukopenia, thrombocytopenia, circulating plasma cells, hyponatremia, hypocholesterolemia, hypercalcemia, azotemia, hyperproteinemia, hypoalbuminemia and hyperglobulinemia, decreased concentration of normal immunoglobulins and proteinuria (Edwards *et al*., 1993).

Squamous cell carcinoma

Squamous cell carcinoma is a common equine neoplasia that usually involves the eye and its adnexa, pharynx, gastrointestinal tract, penis and urinary tract, urethra and prepuce (Lavach, 1992). Squamous cell carcinoma of the eye and adnexa is usually caused by solar irradiation damage to epithelial cells of the skin of horses lacking pigment, melanin. Windy and dusty environment can be contributory stimuli for squamous cell carcinoma formation. Draft horse breeds are mostly affected and the neoplasm can occur at any age, and affects mainly horses older than 9 years. Squamous cell carcinomas are erosive and ulcerative, and can invade deeper soft tissues and bones of the orbit. Metastases can occur to lymph nodes of the head, neck and thorax (Lavach, 1992). Gastric squamous cell carcinoma usually occurs in middle aged or older male horses. Affected horses progressively lose weight, have low grade fever and are anorexic. Rectal examination reveals palpable abdominal masses or adhesions around the cranial mesentery. Squamous cell carcinoma of the equine penis, distal urethra and prepuce can develop from existing papillomas and are usually seen as reddened or pale plaques while in advanced cases, the lesions become granulomatous and cauliflower-like. Histopathologically, the tumor is made of squamous epithelial cells that may or may not have the normal succession of layers (Gatewood, 1992).

Clinical pathology laboratory examination reveals a macrocytic normochromic anemia, leukocytosis, neutrophilia, monocytosis, high erythrocyte sedimentation rate, decreased serum activities of aspartate aminotransferase, alanine aminotransferase, and increased serum activity of alkaline phosphatase and elevated concentration of total proteins and globulins in serum (Agina and Ihedioha, 2016b).

Cutaneous mastocytoma (mast cell tumor)

Equine cutaneous mastocytoma is a non-metastatic tumor of horses. It is a relatively uncommon tumor in horses characterized by formation of varying sizes of cutaneous or subcutaneous nodules located on the hairless or in rare occasions, ulcerated areas of the head, neck or limbs. These nodules are painless, encapsulated and freely movable under the skin. They may also develop soft centers and ulcerate or regresses and heal. Histopathological examination of fine needle aspirates reveals numerous mast cells filled with metachromatic granules, and eosinophils. Biopsy of a regressing nodule shows lesser number of mast cells, and more fibroblasts and collagen.

The haematological parameters of affected foals usually appear normal (Nyrop, 1992).

Melanoma

Melanoma is a common, slow-growing dermal tumor of grey and Arabian horses, which begin as darkening and thickening of skin, and occur singly or simultaneously at multiple sites (Tarrant *et al*., 2001). It may be squamous or wart-like, and has the tendency to metastasize or may invade locally. Melanomas usually involve the eyelids and genital areas (Gatewood, 1992; Lavach, 1992). Older horses are at greater risk of developing the tumor than young horses (Lavach, 1992).

The haematological and serum biochemistry findings associated with malignant melanoma include a mild macrocytic anemia, neutrophilia with a mild left shift, lymphopenia, and severe thrombocytopenia, and decreased serum total protein concentration, azotemia and metabolic acidosis (Tarrant *et al*., 2001).

Diseases caused by allergy

Purpura haemorrhagica

Purpura haemorrhagica is a sporadic, non-infectious and common cutaneous sharply demarcated vasculitis in horses, caused by deposition of immune complexes in the walls of capillaries with subsequent vasculitis and extravasation of blood and plasma. It is characterized by extensive subcutaneous oedematous swelling. It may be seen in horses with a recent history of *Streptococcus equi* infection, strangles or equine influenza (Fadok, 1992) or an adverse reaction to therapeutic drugs (Radostits *et al*., 2007). The disease is immune complex-mediated and due to a type III hypersensitivity reaction. The clinical signs associated with purpura hemorrhagica include subcutaneous oedematous swellings of the face, muzzle, head and limbs. These lesions are usually not painful on palpation and pits with gentle pressure. Necropsy findings include petechial haemorrhages of the mucous membrane of the conjunctiva, and submucousal hemorrhages in nasal cavities and mouth.

The clinicopathological examination of affected horses may reveal a mild anemia with a neutrophilic leukocytosis and hyperfibrinogenemia. Hypergammaglobulinemia may be present. A remarkable serum biochemical abnormality is hyperproteinemia (Kaese *et al*., 2005). Platelet count is usually normal and there is a marked elevated serum activity of creatine kinase and aspartate aminotransferase (Fadok, 1992).

Infarctive purpura hemorrhagica is an uncommon manifestation of the disease characterized by infarction of tissues of the gastrointestinal tract and muscles, dyspnea and death from blood loss (Kaese *et al*., 2005).

The clinicopathological findings associated with infarctive purpura hemorrhagica include elevations in serum activity of creatine kinase (CK) and aspartate aminotransferase, neutrophilia and in severely affected horses, there is evidence of disseminated intravascular coagulation (Kaese *et al*., 2005).

Sweet itch (equine seasonal allergic dermatitis; queensland itch)

Sweet itch is severe pruritic dermatitis of horses characterized by intense pruritis. It is caused by immediate hypersensitivity to salivary antigens introduced into the skin by biting midges (*Culicoides species*) and other biting insects. Distribution of skin lesions are related to the feeding habits of biting midges or their predilection sites. The disease affects horses all over the world and is common in areas where hot and humid weather favors the causative insects. The skin lesions consist of self-inflicted hair loss due to constant scratching, excoriations with crusting, scaling and eventually hyperkeratosis and thickening of the skin. These lesions are usually confined to the base of the tail, rump, back, withers, crest, poll, ears facial areas and ventral midline.

Haematological examination reveals eosinophilia and thrombocytosis (Foil and Foil, 1992). Skin biopsy usually reveals oedema, eosinophilic and mononuclear perivascular infiltration (Quinn *et al*., 1983).

Chronic pulmonary disease of horses (recurrent airway obstruction; heaves)

Heaves is an allergic condition affecting the lungs of older horses and that of ponies kept in poorly ventilated barns and fed poor quality hays containing mold. It is caused by inhalation of moldy spores found in stable/barn dust, bedding and dusty feeds especially hay and straw (allergens) which irritate the lining of respiratory tract and elicit an immune-mediated response in the lungs (Clarke, 1993). A common mold known to cause recurrent airway obstruction is *Aspergillus fumigatus,* although high levels of ammonia in poorly ventilated stables and concurrent infection with respiratory viruses predisposes susceptible horses to full blown respiratory airway obstruction. The immune reaction is usually of the delayed hypersensitivity type. The clinical findings associated with recurrent airway obstruction include a chronic cough, mucopurulent nasal discharge, poor athletic performance, increased respiratory rate, increased expiratory effort, wheezing, crackling sounds on thoracic auscultation, and presence of abundant mucopurulent material in the trachea on endoscopic examination (Naylor *et al*., 1992). The major necropsy findings include pale, voluminous lungs which do not collapse when the thoracic cavity is opened.

Cytological examination of tracheal and bronchoalveolar lavage fluid may reveal a neutrophilia. There is no significant change in the hemogram or serum biochemistry profile of affected horses (Radostits *et al*., 2007).

Alloimmune hemolytic anemia of the newborn (neonatal isoerythrolysis)

Neonatal Isoerythrolysis is an important immunological condition of horse and mule foals that results in a fatal haemolytic crisis if not diagnosed early and treated effectively (Darien and Feldman, 1992). It is caused by ingestion and absorption of colostral antibodies (alloantibodies) directed against the foal's red blood cells resulting in lysis/agglutination. Destruction of neonatal RBCs is immune-mediated (type II hypersensitivity). These alloantibodies are produced by the mare in response to foreign red blood cell antigens, which leak into the maternal circulation during parturition or gestation (alloimmunization). Mares may also be exposed by transfusion of incompatible blood. In subsequent pregnancies, the alloantibodies are produced by mare, concentrated in the colostrum, and absorbed with other immunoglobulins when the foal acquires passive immunity. The clinical findings include an uneventful pregnancy and parturition. Newborn foals are normal for the few hours of life, but develop clinical signs following ingestion and absorption of colostrum, containing anti-red blood cell factor antibody. Affected foals are weak, lethargic, depressed and icteric.

Haemoglobinuria is present. Haematological examination reveals anemia (red blood cell counts, packed cell volume and haemoglobin concentrations are low), increased erythrocyte fragility and sedimentation rate. There may be leukocytosis with neutrophilia and monocytosis; and presence of nucleated red blood cells. Affected mule foals are often thrombocytopenic (Traub-Dargatz *et al*., 1995). Serum biochemical analysis usually reveals an elevated serum concentration of unconjugated bilirubin (Dimmock *et al*., 1982).

Diseases caused by toxins

Phytotoxins

Nitrate poisoning

Nitrate poisoning occurs in horses that consumed a lethal dose of potassium and sodium nitrate in cereal crops, e.g. immature oats and barley and plants such as *Amaranthus species,* rye-grass and sugar beet and rape. It is also seen in horses given water which was polluted by industrial processing plants, or from polluted deep wells. Nitrate poisoning is characterized by salivation, abdominal pain, diarrhea, dyspnea, rapid respiratory, muscle tremor, severe cyanosis, weak pulse, normal or subnormal temperature, brown mucosae, terminal clonic convulsion, frequent urination and abortion. Post-mortem examination usually reveals congested and haemorrhagic gastrointestinal mucosa, dark red to coffee brown blood that clots poorly, petechial hemorrhages in heart muscles and trachea.

Haematological examination reveals an increase in blood levels of methaemoglobin (Radostits *et al*., 2007).

Oxalate-induced equine nutritional secondary hyperparathyroidism

Oxalate-induced equine nutritional secondary hyperparathyroidism is seen in horses grazing on pastures that contain hazardous grasses such as *Setaria sphacelata*, *Panicum maximum* and *Digitaria species*. It affects horses regardless of ages and sex, grazing on such hazardous grasses without other sources of food. Nursing mares and weaned foals are more susceptible. The disease occurs in horses when the calcium oxalate crystals in the plants are consumed and digested only in the large intestine. This will make calcium unavailable for absorption in the small intestine (duodenum), thereby creating a negative calcium balance in the horse leading to hyperparathyroidism. Affected horses have a stiff gait, are reluctant to move and recumbent in severe cases. Exercise usually exacerbates lameness. There is weight loss despite ample food supply, bilateral firm swelling of the maxilla, mandible and over the root of the molar and premolar. Histopathological biopsy of jaw swellings reveals lesions of osteodystrophia fibrosa, osteoporosis in long bones and hyperplasia in parathyroid glands. Necropsy examination reveals a swollen jaw, erosive or pitted joint surface and enlarged parathyroid glands (Radostits *et al*., 2007).

Clinicopathological findings include abnormal increase in blood of parathyroid hormone, and increased alkaline phosphatase activity. There are no consistent abnormalities in serum calcium, phosphorus and magnesium levels (Stewart *et al*., 2010).

Sweet clover poisoning

Sweet clover poisoning is caused by consumption of large quantity of moldy plants, moldy hay or silage containing dicoumarol. These molds in plants convert the normal constituents of some plants (coumarin, melatonin and ferulenol) to dicoumarol. Dicoumarol affects the final carboxylation and activation of clotting factors thereby resulting in extensive haemorrhages into body cavities, visible and palpable haematomas. The infecting fungi are usually of the *Aspergillus* species. Affected horses may be lame, stiff and even recumbent. Temperature, respiratory and heart rates are normal until the terminal stages. Also, horses have pale mucosae, haematuria, epistaxis and dysentery, severe bleeding of accidental and surgical wounds. The carcass is pale. Necropsy findings include subcutaneous haemorrhages and large haematomas seen in flanks and joints. Extravasation is uncommon in lungs, kidney and adrenals (Radostits *et al*., 2007).

Clinicopathological findings include a severe anemia with a marked increase in clotting and prothrombin time and haemorrhagic anemia when loss of whole blood is severe (Radostits *et al*., 2007).

Mycotoxins

Aflatoxicosis

Aflatoxicosis is caused by aflatoxins B1, B2, G1 and G2 produced by *Aspergillus* sp. (*A. flavus, A. parasiticus*), growing on stored food, and spoiled food such as sorghum grain, corn, and moldy bread. The disease is unusual in the horse since they are not likely to be fed damaged (spoiled feeds) (Vesonder *et al*., 1991). No clinical illness has been observed, but anorexia may begin in a few days after the access to contaminated feed. Clinical signs such as depression, inappetence, tremor and prostration with death following in 2-6 weeks, are evident in experimental conditions. Histopathological examination of liver and nervous tissues reveals encephalomalacia, hepatocyte necrosis and fibrosis, bile duct hyperplasia, haemorrhagic enteritis and myocardial degeneration.

Clinicopathological findings include an increase in serum hepatic enzyme activities (Radostits *et al*., 2007).

Citrinin poisoning

Citrinin is a nephrotoxin produced by the fungi, *Penicillium* sp. and *Aspergillus* species characterized by pruritis, hair loss, popular dermatitis, fever, variable appetite, and petechiation of the conjunctiva and mucosae, unthriftiness and occasionally death. In severe cases, there is petechiations in all mucosae and free blood at the anus and other body orifices. Histopathological examination reveals a low-grade, long standing interstitial nephritis

Haematological and serum biochemistry indices are usually normal. Post mortem examination shows petechiations in all organs and tissues (Radostits *et al*., 2007).

Zootoxins

Blister beetle poisoning (cantharidin toxicosis)

Blister beetle poisoning is characterized by anorexia, oral mucosal erosions, frequent urination, colic and diaphragmatic flutter, depression, shock and death. It is caused by consumption of food material (alfalfa hay or weedy meadow hay) infested by blister beetles, *Epicauta* species, the most common being *E. accidentalis* and *E. temexa* (Edwards *et. al*., 1989). Identification of blister beetles in hay and/or gastrointestinal contents, and gross identification of ulcers in the distal esophagus, stomach and urinary bladder, is usually seen at necropsy (Echevarria, 2006).

Clinicopathological findings associated with the disease include haematuria, azotemia, and hemoconcentration, leukocytosis with a neutrophilia, hypocalcemia and hypomagnesemia (Edwards *et. al*., 1989; Lavoie and Hinchcliff, 2008).

Snake bite (snake envenomation)

Snake envenomation caused by a snake bite is established when the venom is injected into the horse through the snake's specially adapted fangs. Horses appear to be much more susceptible to venom than any other species. The toxin in venom is neurotoxic, and causes flaccid paralysis, paralytic respiratory failure and papillary dilatation. Snake venoms also contain cytolysins (which cause tissue necrosis), hemolysins, (a coagulant), thrombace (an anti-coagulant that causes extensive haemorrhage) and myotoxins (that causes muscle necrosis and myoglobinuria). The most common venomous snakes include rattlesnakes, vipers, adder, cobra, tiger snakes and mamba. The clinical signs observed in horses bitten by tiger snakes (venom contains neurotoxins and coagulants) include pronounced pupillary dilatation, lack of response of pupil to light, and muscle tremor (Anlén, 2008). Foals bitten by brown snake exhibits drowsiness, droopy eyelids and lips, partial tongue paralysis, muscle tremor, weakness, and recumbency. Respiration is labored and abdominal in nature. At necropsy, fang marks are usually visible and there are local swellings at the site of the bite due to exudation of serous fluid (Fowler, 1993).

The clinicopathological findings associated with rattlesnake envenomation include leukocytosis with a neutrophilia, thrombocytopenia, hypoproteinemia, hyperlactatemia, hyperglycemia, hypomagnesemia, hypocalcemia, hypokalemia, hypocholeremia and hyperphosphatemia, and increase in serum activities of creatine kinase, aspartate aminotransferase, gamma glutamyl transferase and serum dehydrogenase (Lawler *et al*., 2008; Fielding *et al*., 2011).

Adder snake envenomation is associated with mild anemia, leukocytosis with neutrophilia, left shift and toxic changes, monocytosis, moderate hyperfibrinogenemia and mild hyperproteinemia (Anlén, 2008).

Diseases caused by inorganic poisons

Lead poisoning (plumbism)

Lead poisoning is caused by accidental ingestion of toxic levels of lead compounds or ingestion of food grazed on pastures containing excessive amounts of lead. It may also be as a result of ingestion of paint chips from a fence or pasture (Sojka *et al*., 1996). There is high case of fatality risk if untreated. Lead poisoning in horses is characterized by rough hair coat, pharyngeal dysfunction and weight loss (Sojka et al., 1996). Some horses may not show the clinical illness. Other clinical signs include inspiratory dyspnea caused by paralysis of the recurrent laryngeal nerve, recurrent choke and regurgitation of food and water through the nostril due to

pharyngeal paralysis and aspiration pneumonia. There is also paralysis of the lips, mild to severe abdominal pain, general muscle weakness and stiffness of joint, muscle fasciculations over the triceps, recumbency and clonic convulsion. There is diffuse congestion of the lungs, degeneration of the liver and kidney, epicardial hemorrhages, and congestion of meningeal and cerebral vessels at necropsy. Histopathological examination of renal cortex reveals eosinophilic inclusion bodies, which is of great diagnostic importance.

Haematological examination in lead poisoning may reveal normocytic normochromic anemia in some horses, and microcytic hypochromic anemia in others, basophilic stippling in red blood cells, and marked poikilocytosis and anisocytosis (Liu, 2003). Cytological examination of cerebrospinal fluid reveals a slightly elevation in leukocyte numbers (Radostits *et al*., 2007).

Arsenic poisoning

Poisoning with arsenic is a non-infectious disease of horses characterized by severe colic, marked congestion of the mucosae, and signs of severe and multi-focal ulceration of the caecum (Pace *et al*., 1997). It is caused by accidental ingestion of high doses of arsenic in plants, herbicide contaminated pasture, orchards contaminated by insecticide sprays and pasture contaminated by calcium arsenate. It also may be seen in horses administered drugs such as arsanilic acid. Arsenic can also be absorbed through abraided or hyperemic skin (Bahri and Romdane, 1991).

Haematological and serum biochemistry findings associated with arsenic poisoning include hemoconcentration, leukopenia, degenerative changes in circulating polymorphonuclear neutrophils including cytoplasmic basophilia, foamy cytoplasmic vacuolation and Dohle bodies, azotemia, hypokalemia, hyponatremia, hypochloremia, hyperglycemia, and unconjugated hyperbilirubinemia, increased serum activities of lactate dehydrogenase and creatine kinase (Pace *et al*., 1997). There is also elevated protein concentration in peritoneal fluid, proteinuria and slight hematuria (Pace *et al*., 1997).

Zinc poisoning

Zinc poisoning is caused by ingestion of plants or grazing on pasture located near Zinc mines and Zinc smelting operations. It may also occur in horses given drinking water with high content of Zinc. Foals and yearlings are much more susceptible to Zinc poisoning than adult horses. The clinical features of the disease include an enlarged epiphyseal region of the long bones, stiff gait and lameness. Severely affected foals have an arched back, and are unthrifty and anemic. There is swelling and pain in the fetlock, carpal, hock and stifle joints. There may be a non-specific degenerative arthritis and generalized osteoporosis. Post mortem examination reveals eroded and rough joint surfaces in the facets of the cervical vertebrae or in the articular cartilage of limb joints (Thompson, 1992). Haematological examination revealed a low packed cell volume (Eamens *et al*., 1984).

Conclusions

The reviewed horse diseases have shown that most of them are characterized by significant/specific changes in the haemogram, clinical biochemistry and cytologycal profile of horses.

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