Systematic Review on Equine Gastric Ulceration: Predisposing Factor, Epidemiology and Treatment

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Abstract: The aim of this paper is to describe the epidemiology, clinical sign, diagnosis, treatment, control and prevention of equine gastric ulceration. In many countries, endoscope examination of equine gastric ulceration has made a significant impact of gastric ulceration in equine. However, in same this countries technique have not been used as effective and improved disease control strategy on bases of disease control technique. The studies of the disease in naturally diseased animal of equine gastric ulceration have provided valuable information; detail knowledge can also be gained through studies of risk factors. The pathogenesis of equine gastric ulceration has been evolved by destructive effect of risk factors for effect of HCl & pepsin, and defensive effects of protective factors, such as mucosal layer & bicarbonate product of stomach. Experimental studies have been adapted to study risk factor for equine gastric ulceration, the mechanisms of risk factors and natural disease pathogenesis. In general, in this review it has been attempted to give a highlight in understanding mechanisms of risk factors and pathogenesis of disease in relation to its effect on the health of host, which have paramount importance in designing intervention methods in the control of equine gastric ulceration in equine. Finally, relevant recommendations are forwarded. [Getinet A, Maradona B, Melesse G. **Systematic Review on Equine Gastric Ulceration: Predisposing Factor, Epidemiology and Treatment.** *Biomedicine and Nursing* 2019;5(2): 92-98]. ISSN 2379-8211 (print); ISSN 2379-8203 (online). http://www.nbmedicine.org. 9. doi:10.7537/marsbnj050219.09.

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1. Introduction

According to Radostits *et al.,* (2000), gastric ulceration disease in equine is recently recognized disease, with most reports originating after 1990 and considering with the wide spread availability of endoscopes of sufficient length to permit examination of the stomach of adult horse, gastric ulceration in equine may occurs as primary problem, or it may occurs secondarily to another intestinal disorders. Gastric ulceration affect large number of foals, yearlings, and adult horses, and different clinical syndromes and lesions distributions occur in each group (Radostits *et al.,* 2000).

Equine gastric ulcer syndrome continues to be a conundrum for horse owners, trainers and veterinarians. The high prevalence of gastric ulceration in horses, vague clinical signs and negative effect on performance make it a significant clinical and economic problem in horses.

Current pharmaceutical agents (Omeprazole, ranitidine) are effective in treatment of equine gastric ulceration but expensive and there is a high rate of recurrence after treatment. Preventive measures, such as pasture turnout, *ad libtum* hay feeding, reducing training levels and stress reduction are essential to prevent recurrence but may not be possible or effective in some horse (Radostits *et al.,* 2007). Equine gastric ulceration is a syndrome in which veterinarians could not take into consideration. There for this review is to induce veterinarian in the direction of predisposing factors and treatment with epidemiological situation.

2. Equine Gastric Ulceration

2.1. Etiology

The etiology of most common occurrences of gastric ulcer in the horse is unknown but several risk factors have been identified. The disease is common in horses under taking regular exercise and might be related to decreased stomach volume and subsequent exposure of squamous mucosa of proximal part of the stomach to acid during exercise (Radostits *et al.,* 2007). Individual cases of gastric ulcers are may be associated with parasitic gastritis, such as in horses affected with *Gastrophilus* spp, *Habronema megastoma* larvae, *trichostrongylus axei* and *Drashia megastoma* (Taylor, 1999). Tumors of stomach, such as gastric squamous cell carcinoma or lymph sarcoma may cause ulceration of gastric mucosa. Gastric phytobezoars persimmon seeds *(D.virginian*) have been associated with gastric impaction, ulceration and perforation of the glandular portion of the stomach in horses. There is no evidence of infectious etiology, for instance *H.*spp (Radostits *et al,* 2000). Many infectious diseases with major systemic or alimentary tract signs else were produce gastric lesions. Systemic states, such as uremia and endotoxemia. Blister beetle /*Epicauta*. spp/ intoxication in horse, induced by

cantharid contained in these insects, may cause necrosis and ulceration of the distal esophagus and pars esophagi and intense hyperemia of the glandular mucosa of the stomach. In some cases it is associated with ulcers or gastric raptures (Goodman.1985). *Helicobacter*.spp, (other than *H. pylori*) were isolated from man and varieties of animals suffering from gastric ulcer and gastritis. *Helicobacter*. spp, *Helicobacter equirum* were isolated from fecal samples of clinically healthy horses. *Helicobacter* like DNA was detected in the stomach of thorough breed horses in Venezuela (Goodman. 1985). Once gastric ulcers present other bacteria have been implicated in inhibiting ulcer healing bacteria including *E.coli*, were cultured from stomach of horses (Garris. (1989).

2.2. Epidemiology

2.2.1. Prevalence

Equine gastric ulceration is a very common disease of stomach of foals and adult horses, and reported prevalence of up to 50%in foals and 75% of in adult horses, this percentage do heavily depend on habitat breed performance age of population. The horse's stomach is divided in to two distinct areas, the non-glandular/squamous and glandular region separating by a Sharpe demarcation called the *margo plicatus*. The prevalence of erosion or ulcer of the gastric glandular and non-glandular mucosa, detected by gastroscopy examination, over ages 50% in foals less than 2-monthes of ages that do not have signs of gastric ulcer disease. Lesions of the squamous mucosa are present in 45% of foals, were lesions in the glandular mucosa occur in fewer than 10% of foals less than 4-monthes of age. Diseases attributable to duodenal ulcer and gastric ulcer occur in approximately 0.5% of foals although the prevalence is greater in foals with other diseases, such as pneumonia, septicemia (Radostits *et al*., 2000). Despite wide spread use of anti-ulcer medication the prevalence of EGUS remains high. This is due to the cost of anti-ulcer agents, which results in shorter than prescribed treatment course, the administration of sub therapeutic doses or substitution of compounded medications or feed supplements that are ineffective (Anon, 1999).

The prevalence of gastric ulceration in competitive horses varies from 11% in riding horses to 93% in race horses (Andrews *et al*., 2008). Also the prevalence of horses gastric ulceration is high in horses with bowel, liver and esophageal lesion (Sandin *et al.*, 2000). The prevalence of equine gastric ulcers in brood mares' pregnant, non-pregnant broad mares, under similar pasture management condition, had a prevalence of 66.6% and 75.9% respectively. Race horses have a high prevalence of EGUs, 56.5% of horses in endurance competitions show jump, dressage or western performance and travel, had gastric ulcers after competition (Chameroy *et al*., 2006).

2.2.2. Mechanisms of disease

HCl and sustained gastric P^H less than 4 are probably the most important causes of gastric ulceration. Ulcers are more prevalent in the non-glandular mucosa because these are lack resistance to acid injury. HCl induces injury in this region by damaging the outer cell barrier, followed later by diffusion in to the squamous cells of *stratum spinosum* resulting in inhibition of cellular Na^+ transport, cell swelling and eventual ulceration (Svendsen *et al.,* 2008). Volatile fatty acids (acetic acid, propionic acid, butyric acid and valeric acid) induce damage by rapidly diffusing in to the squamous mucosal cells of *stratum spinosum* layer and immediately inhibit Na^+ transport which results in cell swelling and ulceration (Andrews *et al*., 2006). Lactic acid was exposed to the non-glandular mucosa; it did not affect Na ion transport as with VFAs. Instead, lactic acid (PH 1.5 & 40mmol/L) exposed to the glandular mucosa increased tissue permeability, as indicated by Transepithelial conductance (Andrews *et al*., 2008).

2.2.3. Risk factors associate

The risk of equine gastric ulceration is multi factorial, and some of the **risk** factors have been identified. Acute gastric ulcer mostly occurs in the stomach and may be associated with stress (e.g., extensive surgery, shock, sever burn, major medical illness) and by certain drugs (e.g. asprin and other NSAIDs, corticosteroids, anti-neoplastics, and potassium tablets) (Abrams. 1992).

2.2.3.1. Influence of Exercise

Intense exercise is common risk factors for equine gastric ulceration. The mechanism for this is that compression of the stomach by abdominal viscera and diaphragm leads to delivery of acid contents in to the proximal region of stomach. This mechanism is thought to deliver the acid to the non-glandular mucosa. Also time in work, crib biting, difficulty maintaining body weight and playing the radio in the barn were identified as risk factors (Hansten, 1989)

2.2.3.2. *Non-steroidal Anti-inflammatory drug*

According to Garris *et al.,* (1989), when prostaglandin synthesis is inhibited, erosion and ulceration of gastric mucosa may occur. This is apparently the mechanism by which, asprin and other NSAIDs cause acute gastric ulcers. NSAIDs have been shown to cause gastric ulcers in horses. This is usually related to the use of high doses have been known to cause ulcers in horses. However a more recent study in horses given. Suxibutazone or Phenylbutazone at therapeutic doses for 14 days showed no significant difference in gastric ulcers when compared to each other and control horses receiving no treatment. According to Reed *et al*., 2006

the study evaluated the use of a combination of NSAIDs and gastric ulcer in horses. Phenylbutazone 2.2mg/bwt kg Po; 9. 12h for 5 days or phenylbutazone (1.1mg/kg bwt, i.v., 9. 12h for 5 days) were administered to adult horses. In this study, total plasma protein & albumin decreased in NSAIDs treated horses and non glandular gastric ulcer scores were significantly higher in horses treated with the 2NSAIDs (Andrews *et al*., 2006).

2.2.3.3. Animal risk factors

According to Radostits *et al*. (2007), age is an important risk factor for ulceration of the squamous epithelium, with 88% of foals less than 9 years of age affected compared to 30% of foals more than 70 days of age. Gastric lesions occur in fewer than 10% of foals over 90 days of age 4. There is no sex influence on the prevalence of ulcers in horses. According to Abrams *et a*l. (1992), but in human beings men have a higher incidence of both duodenal (4 times) and gastric (2.5 times) ulcer compared to women. And also have its influence, in man with blood type O are more likely to have duodenal ulcers; those with blood type A are more likely to have gastric ulcers. Among adult horses age and sex are only weak risk factors, if at all for presence of gastric ulcers. Gastric ulcers tend to be more severe in older horses (Radostits *et al* 2007).

2.2.3.4. Stress/Diseases

Stress and Disease are important risk factors for development of ulcer s of the glandular mucosa. Lesions of the gastric glandular mucosa occur in 27% of foals with other diseases but in 3% of other with healthy foals (Radostits *et al.,* 2007).

2.2.3.5. Management/Environmental Risk factors

Race horses in training have a higher prevalence of ulcers than do race horses, that are speeling (not in active training), And horses that are racing regularly have a higher prevalence than resting horses or horses in training but not racing. Exercise is strongly associated with development of gastric ulcers in horses; this is through the increase intragastric pressure According to Radostits et al. (2007).

2.2.3.6. Diet

Diet is suggested to be a risk factor for development of gastric ulcerations, but definitive studies lacking. A high grain low roughage diet is a risk factor of equine gastric ulceration. High roughage diet provides of chewing and the production of salivary bicarbonate that bathes the stomach and protects against gastric ulcers (Radostits *et al*., 2007). Small low starch meals empty from the stomach significantly faster than large high starch meals. Since high starch diets are fermented to VFAs, and lactic acid, large high starch meals should be avoided in horses prone to gastric ulcerations. Feeding a diet that contains 0.5 kg of grain per 100kg bwt no more frequently than 6 hrs apart can reduce the risk of equine gastric ulceration (Andrews *et al.,* 2008). Alfalfa hay was shown to protect horses against EGUs, by increasing stomach pH. Gastric juice pH and ulcer scores were lower in horses fed a diet containing alfalfa hay compared to the same horses fed dietary brome or costal Bermuda hay (Jubbs *et al*., 1995).

2.2.3.7. Feed with holding

Feed with holding causes gastric ulcers in horses probably because of the lack of buffering of acids produced during periods when the stomach is empty. The loss of buffering is due to lack of feed material in the stomach and too decrease production of saliva, which normally buffers gastric acids. Horses grazing at pasture eat frequently and have feed in the stomach almost all the time (Rodastits *et al.,* 2007).

2.3. Pathophysiology

2.3.1. Anatomy & Physiology of equine stomach

Horses have a simple stomach that can be sub divided anatomically into the non-glandular squamous mucosa lined proximal area (approximately one-half) and the glandular area which covers the remainder of the stomach through the *antrum* down to the pyloric and, consists of 3 distinct histological regions cardiac, fundic and pyloric. The *margplicatus* define the lower border of squamous mucosa with the glandular mucosa. The inlet of stomach is cardiac sphincter at the end of esophagus, and the out let is the pyloric sphincter at the beginning of duodenum (Abram. *et al*., 1992). It churns and mixes the feed with digestive juice, secrets mucus and enzymes starts protein digestion, and secrets intrinsic factors, which is necessary for absorption of vitamin B_{12} from the intestine. Some carbohydrates, and amino acids are also poorly absorbed and only few highly lipid soluble substances, such as alcoholic compounds and some drugs, are absorbed in moderate quantities (Abrams *et al*., 1992). Gastric juice consists of mucus, digestive enzyme HCl & electrolyte. This mucus is secreted by mucus glands for the function of to protect the lining of the tract from digestive juice lubricate feeds, promote adherence of the fecal mass and neutralize acids & bases (Abrams *et al*., 1992). The pH of stomach is highly acidic (pH 1-3). The secretion is highly estimated by the parasympathetic nervous system, by hormone gastrin, by presences of food in mouth by smelling, seeing of feed. Once produced, gastric acid is released by activation of any enzyme system (H^+, K^+_{ATP}ase) at the surfaces of parietal cells. These enzyme system acts as a gastric acid /proton/pump to move gastric acid from parietal cells in the mucosal lining of the stomach into the stomach lumen. Autodigestion of the stomach wall and ulcer formation are normally prevented by cell-protective effects of mucus secretion, dilution of gastric acid by

food and secretion, prevention of diffusion of HCl from the stomach lumen back in to the gastric mucosal lining. The presences of certain prostaglandin and perhaps other mechanisms (Abrams *et al.,* 1992).

2.3.2. Pathogenesis of EGU

According Contreras *et al.* 2007), idea as, although the dictum is "no acid-no ulcer" is still valid in the majority of peptic ulcer cases. Now a day much more is known about their complex etiology and pathology. Inconsideration of possible pathogenic mechanisms, the anatomic location must be taken in to account. Lesions in the gastric squamous mucosa result primarily from excessive acidity were as gastric glandular lesions result primarily from defective mucosal protection. In general ulceration is considered to result from an imbalance of aggressive and protective factors. The relevant aggressive factors are HCl, Pepsin, where as relevant protective factors are include mucus-bicarbonate barrier PGE_2, mucosal blood flow, cellular restitution, and growth factors that promote angiogenesis and mucosal proliferation. Gastric motility also is important, because delayed gastric emptying and prolonged gastric contractions have been implicated in the pathogenesis of ulcers. The squamous mucosa of equine stomach locks a mucus-bicarbonate layer, and it has minimal resistance to exposure to HCl. The gastric glandular mucosa comprises multiple cell types and functions and has elaborate mechanisms for protecting against peptic injury. 24hrs gastric acidity was significantly less in horses with hay available compared with horses deprived of feed. Intermittent periods of feed deprivation can induce damages to the gastric squamous mucosal ulcers within two days because of gastric acidity. Concentrate feeding may contribute to ulcers by increasing serum gastrin levels by increasing the horse's roughage intake, and, most importantly by reducing the amount of time the horses spends eating. This feeding management play a pivotal role in the pathophysiology of gastric ulcers in horses. In fact, we have to found that horses that are turned out on to pasture full time typically have no gastric ulcers. The high prevalence (50%) of gastric ulcers, particularly in the squamous mucosa, in young foals may be associated with gastric developmental changes that occur at the first day and week of life (Goodman *et al.,* 1985). At birth, the equine gastric mucosal epithelium is thin and not highly keratinized. Within days, the mucosa becomes hyperplasic and parakeratotic-Desquamation of the squamous epithelium can be observed endoscopically in the first month of life in. Increasing gastric acidity temporally parallel the proliferation of gastric squamous epithelium with minimal acidity during the first few days of life and marked acidity present by 7-14 days. It is possible that the developing epithelium is less resistant to acid than more mature gastric squamous epithelium, thus predisposing it to peptic ulcers. Illness appears to be a risk factors for foals developing glandular mucosal ulcers, because foals that were sick or had pain full musculoskeletal condition had a greater prevalence of glandular lesion as compared with normal foals (Radostits *et al*., 2007). *Trichostrogylus axei*, the change induced in the gastric mucosa within an alteration in pH and increases permeability of mucosa the worm penetrates between glands rather than in to glands and forms coalescence of the subsequent nodular lesions often results in plaques or ring–like gastric ulcers. *Gastrophilus* /bot flies/attaches to the gastric epithelium and this larvae provokes an inflammatory reactions with the formation of funnel shaped ulcers surrounded by arim of hyperplasic epithelium (Jubbs *et al.,* 1995).

2.3.3. Anatomical Distribution and Scoring

Gastric ulcers have been identified throughout the stomach with the non-glandular stratified squamous mucosa along the *margoplicatus* most commonly affected. The location of the ulcer could be either glandular only, non-glandular only, or both. In addition, the location in each horse were the highest severity (severity location) were located was recorded (i.e., dorsal part of the non glandular region, *margoplicatus*, at the lesser curvanture, cardiac or fundic region of the glandular region or pyloric area). Hyperkeratosis in the non-glandular part of the stomach was not registered as ulcers since the Mac Allister scoring system does not include Hyperkeratosis as a lesion (Taylor, 1999). In other studies and scoring systems Hyperkeratosis is included in equine gastric ulcerations as cited by Andrews *et al., (2002*). In the current study, there was no association between age and likelihood of EGUs >two. Although, with age ulcers appears to be come more anatomically widespread (that is more likely to be found in both glandular and non-glandular regions of the stomach) in older horses. In contrast, found no non-glandular ulcers among the 34 horse's age 8-17years, in particular 3 years old horses as cited by Chameroy *et al*. (2006).

Table: 1- Gastric ulcers scoring system (Radostits *et al*., 2007)

Lesion score No	**Description of lesions**
0	No lesion
1	1-2 localized lesions.
2	3-5 localized lesions.
3	6-10 localized lesions.
4	>10 very large lesions.

Table 1 indicates as the lesions are localized and it can be scored for varied degree.

Table: 2- Description of Gastric ulcers lesion scores. (Radostits *et al*., 2007).

Lesion	Description on lesions
0	No lesion.
1	Appears superficially mucosal missing.
2	Deeper structures involved.
3	Multiple lesions and variable severity.
4	Same as two and has active appearance /hyperemia/or darkened lesion crater/.
5	Same as four plus active hemorrhage of adherent blood clot.

Table: 3- Anatomical location of most severe ulcers and their level of severity (Radostits *et al*., 2007)

Location	Most Severe Ulcers (%)	Severity (%)				
		1	2	3	4	5
Non glandular	136 (81)	46	38	28	19	5
Dorsal part	4 (2)	4	0	0	0	0
Margoplicatus:						
At the greater curvature	107 (64)	31	30	24	17	5
At the lesser curvature	25 (15)	11	8	4	0	0
Glandular	32 (19)	15	10	5	0	2
Cardiac region	2 (1)	0	1	1	0	0
Fundic region	4 (2)	1	1	0	0	2
Pyloric region	26 (15)	14	8	4	0	0
Total	168 (100)	61	48	33	19	7

2.4. Clinical Signs

According to Radostits *et al.* (2007), vast majority of horses with gastric ulcer, lesions do not have clinical signs. The clinical signs of equine gastric ulcers may include abdominal discomfort/colic, reduced appetite, weight and body condition loss, rough hair coat, reduced performance, loss of vitality, dorsal recumbency and grinding of teeth. The severity of signs did not related to the severity of EGUs and this was supported by the fact that there was no association between body condition score and the incidence of egus (Andrews *et al*., 2008).

2.5. Diagnosis

Information regarding the details of the individual horse was collected from the person who kept and looked after the horse for diagnosis. This includes detail of: breed types, age, gender, life stage (young, breeding working, retired /not working) and behavior details with respect to work, over load, management and nutrition, any health problems resent administration of NSAIDs any problems with lameness, previously diagnosed EGUS or recurrent air way diseases and, if so, what medication had been given were collected and described (Radostits *et al.,* 2007).

2.5.1. Tentative diagnosis

If an endoscope of sufficient length is not available clinical signs (diarrhea, inappetence, colic, teeth grinding, excessive salivations, weight loss, rough hair coat, gastric dilation, gastroesophageal reflux & unexpected death, shock, dehydration, sweating, and an increase in respiratory rate) and responses to treatment can be used in horses suspected of having egus and /or per acute peritonitis secondary to gastric perforation (Anon, 1999). There may be pain evidenced by deep palpation of cranial abdomen but this not reliable diagnostic signs. Measuring urine and plasma sucrose concentration /sucrose permeability test/. Fecal occult blood test was found to be help full in diagnosis of equine gastric ulceration. Horses with a positive FOBT is likely to have gastric ulcers. Serum *a*1-antitrypsin was present in 44/47 foals, *a*1-antitrypsin may be released from damaged gastric tissue (Radostits *et al*., 2007).

2.5.2. Definitive diagnosis

The definitive diagnosis of equine gastric ulcerations can only made after visualization of the stomach either *antemortum* using endoscope or at *postmortem* (Radostits *et al.,* 2007).

2.6. Treatment

Principles of treatment of equine gastric ulcer disease in foals are promotion of healing by reducing gastric acidity and enhancing mucosal proliferation the; enhancement of gastric emptying, provision of nutritional and metabolic support and treatment of other diseases (Radostits *et al.,* 2007).

2.6.1. Anti-ulcer Drugs.

Drugs used in the prevention and treatment of gastric ulcer diseases act mainly to decrease cell-distractive effects, increase cell-protective effects or both (Abrams, 1992).

2.6.1.1. Gastric acid inhibitors

These anticholinergic agents block the action of acetylcholine on the parasympathetic nervous system (e. g; atropine). These are weak inhibitors of gastric acid secretion even in maximum doses (Abrams, 1992).

2.6.1.2. Proton Pump inhibitors.

These drugs binds with $H,^{+}$ K^{+}_{ATPase} to prevent the pumping or release of gastric acid into the stomach lumen (Lansoprazole, omeprazole, pantoprazole and pariprazole /rabenprazole (Abrams, 1992). $H\text{-}_2$RAS are structural analogues of histamine that at least those in clinical use competitively and surmountably block the $H\text{-}_2$ receptors located on the basolateral membrane of the parietal cell. (E.g; Cimetidine, Famotidine, Rantidine and Roxatidine). According to Garris and Kirkwood. (1989), antacids are alkaline substances that neutralize acids. Non systemic gastric antacids are Almunium, Magnesium, and Calcium compounds (e.g; Almunium hydroxide, Almunium phosphate, Magnesium hydroxide, magaldrate) react with HCl in the stomach to produce neutral, less acidic, or poorly absorbed salts and to arise the PH of stomach secretion (Contreras *et al.,* 2007).

2.6.1.5. Miscellaneous drugs

2.6.1.5.1. Prostaglandin derivatives

Prostaglandin (PG), such as *misoprostol* and *prostaglandin* inhibits gastric acids secretion and performs protective function on gastric mucosal cells as well. protective function are attributed to their ability to increase secretion of mucus and bicarbonate, mucosal blood flow and perhaps mucosal repair (Garris *et al*., 1989).

2.6.1.5.2. Prokinetic drugs

Prokinetic drugs (e.g. Domperidone, metoclopramide) which facilitate the oral to aboral clearing of the esophagus and emptying of the stomach (Andrews *et al.,* 2008).

2.6.1.5.3. Anti-muscarinic drugs

Anti-muscarinic drugs (eg; pirenzepine) have selectivity for M1-receptors present on intramural neurons and paracrine, histamine releasing cells (i.e. ECL-cells) of gastric mucosa (Andrews *et al.,* 2008)

2.6.1.5.4. Sucralfate/ Cytoprotective

These are locally active agents help to heal gastric and duodenal ulcers by forming a protective barrier between the ulcer and gastric acid, and bile salts. But they do not alter secretion of gastric acid (Abrams, 1992).

2.6.2. Drugs for Underlining Causes

2.6.2.1. Drugs against Helicobacter. Species

Company's worldwide try to develop antimicrobial both specific for *H.* spp and effective as a monotherapy, corrent therapy still takes advantage of the available repertoire of antibiotics. Although it has been claimed that antibiotics alone can eradicate *H.* spp infection still under debate. One-week triple therapies consists of a PPI in combination with two of the three antibiotic amoxicillin, clarithromycin, and metromidazole are the therapy of choice (Sande and Mandell, 1985).

2.6.2.2. Drugs against ulcerating parasites

The goal of anthelmintic drug therapy may be for complete eradication of the parasite or eradication of the "worm burn" (Abrams *et al*., 1992).

Table-4: Treatment of *gasrophilus* species. (Hansten, 1989)

Drugs	Doses	Administration Rout
Carbondisulfide	5.3mg/kg	PO
Dchlorvas	10 mg/kg	PO
Ivermectine	0.2 mg/kg	PO
Moxiddectin	0.4 mg/kg	PO
Trichlorton	40mg/kg	PO

2.7. Prevention and Control.

Due to high prevalence rate of equine gastric ulcers once treatment is discontinued, preventive measures must be implemented to prevent recurrence; omeprazole paste (1mg/kgbwt, PO, 9. 24hrs) was found to prevent gastric ulcers in horses maintained under ulcerogenic conditions (White *et al*., 2003; White *et al*., 2007). Horses could develop gastric ulcers in as little as 8 days after initiation of heavy to light training and omeprazole paste administration decrease the incidence. Management changes are essential to prevent gastric ulcers recurrence after treatment is discontinued. Free access to good quality pasture and feeding alfalfa or other Ca^{++} or high protein forages may help to prevent gastric ulcers (Garris et al., 1989).

Since a large quality of VFAs are produced in the stomach of horses feed high concentrate diets it has been suggested that concentrates should be at <0.5kg/100kgbwt not more frequently than other every 6 hrs and horses that are prone to egus should be feed concentrates with caution. Prevention of gastric ulceration in the athletic horse centers up on minimizing the effect of factors that promote ulcer developments. Ideally, horses at risk would be kept at pasture, but this is not feasible under most management or husbandry systems. However, the efficacy of pharmacological prophylaxis in prevention of disease or death due to gastric ulceration has not been administered. Suppression of gastric acidity in either sick or normal horses may be unwise because of the protective effect of low gastric PH on gastric colonization of bacteria (Anon, 1999)

3. Conclusion and Recommendation

There has been an ongoing guest for knowledge of the risk factors, pathogenesis of equine gastric ulceration, with the ultimate aim of improving diagnosis, treatment and developing new control methods used to induce this. Over recent decades, the methods used to induce this disease experimentally have mainly involved exposing to the risk factors of equine gastric ulceration. Nonetheless, this work has facilitated significant progress in our standing of disease, and of improved option for disease control. The generation of new infection protocols, including an ability to expose animals to controlled and potentially very low risk factors of equine gastric ulceration, will allow further understanding of the occurrence of the disease and allow greater progress to combat the ongoing treat poised by equine gastric ulceration.

There for, by taking the fore going facts, the following points are recommended: Strict hygienic measures should be practiced in the farm to reduce contamination from feces, urine and other contaminates in order to cut the cycle of gastric parasites. Newborn foals should be kept in good management that should not have stress and avoid administration of NSAIDs. Racehorses should never have with holding of feed. Racehorses having race and work load (hard working) should take Omeprazole or antacids to prevent autodigestion of stomach wall by HCl and pepsin. Further researches should be conducted on the etiology, risk factors and pathogenesis of egus in natural host in order to understand the establishment and progress of the disease for designing effective control method.

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