

**CLINICAL STUDY OF STRANGLES AND ITS  
COMPLICATIONS (PURPURA HEMORRHAGICA)  
ON A HORSE FARM AT SOHAG  
GOVERNORATE, EGYPT**  
(With 9 Figures and 7 Tables)

By  
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دراسة اكلينيكية عن مرض خناق الخيل ومضاعفاته (الزفرية النزفية)  
في مزرعة خيول بمحافظة سوهاج - مصر

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الهدف من البحث هو محاولة معرفة السبب الذى أدى الى اصابة بعض الخيل - بمزرعة خيول بسوهاج - بورم أوديمي فى رؤوسها مع عدم القدرة الكافية على الحركة او بلع الطعام. وقد اتضح بالفحص الاكلينيكي لجميع خيول المزرعة - والبالغ عددها ٢٣ جوادا - والتحليل البكتريولوجى للعينات المجمعّة من ذات الخيول اصابة ١٢ حالة بمرض خناق الخيل منهم ٥ حالات تطورت الى احدى مضاعفات هذا المرض (الزفرية النزفية) وتم الوصف الاكلينيكي لمضاعفات المرض (الزفرية النزفية) ومناقشته بأسباب. وقد اتضح ايضا أنه ليست كل الحالات التى كانت ايجابية بالعزل البكتريولوجى كانت ايجابية اكلينيكيًا مشيرًا الى وجود حالات حاملة للعدوى دون ظهور أعراض مرضية عليها. وأن نزول (افراز) الميكروب المسبب لمرض الخناق غير مستديم. وقد تم معرفة مصدر العدوى بالمزرعة وكذلك تم حساب معدل الانتشار التراكمى لمرض الخناق بالمزرعة خلال فترة الفحص وأشار الى وجود عوامل مساعدة ساعدت على انتقال العدوى من حيوان الى آخر. وقد تم تحديد هذه العوامل ومناقشتها. وأظهرت نتائج تحليل عينات دم الخيل المريضة بالخناق ارتفاع عدد خلايا الدم البيضاء مع زيادة فى الخلايا المتعادلة (neutrophils) نتيجة للطبيعة الصديدية للميكروب بينما أظهرت نتائج تحليل عينات دم الخيل المريضة بالزفرية النزفية (الخالية من الطفيليات) وجود فقر دم مع ارتفاع عدد خلايا الدم البيضاء وزيادة واضحة فى كلا من الخلايا الحامضية (eosinophils) والمتعادلة وربما يدل هذا على وجود حساسية للحيوان المصاب كرد فعل مناعى ضد الميكروب. تمت محاولات علاجية ناجحة لكل الحيوانات المريضة بالمزرعة. أوضحت هذه الدراسة الأتى: أولاً- الخيول المصابة بخناق الخيل الغير مصحوب بالمضاعفات والتي تربي فى بيئة صحية مقبولة

ليست في حاجة ماسة للعلاج بالمضادات الحيوية العامة لأن الجرعات العالية من المضادات الحيوية قد تؤدي إلى تأخير النضوج الكامل لخراج الغدة الليمفاوية في الخيل المصابة الذي قد يساعد استمراره في سرعة ظهور المضاعفات أو يؤدي إلى حدوث حساسية للحيوان المصاب كرد فعل مناعي غير مرغوب فيه ضد الميكروب المتواجد. ثانياً- استخدام الأدوية المضادة للحساسية كأحدى خطوات علاج مرض خناق الخيل قد يساعد في علاج وربما منع ظهور مرض الزفرية النزفية. ثالثاً- النتائج السلبية للعزل الميكروبي عن طريق المسحات الأنفية للخيل مرتين متتاليتين بينهما اسبوع لم تكن كافية للتصريح بأن الحيوان خالي من العدوى. وبناءاً عليه يوصى بأن لا تقل محاولات العزل عن ٣ مرات بواقع أسبوع بين كل مرة والأخرى.

## SUMMARY

Twelve cases out of 23 examined horses showed typical form of strangles. Out of the diseased horses, 5 cases developed signs of purpura hemorrhagica. Detailed clinical signs of this complicating form were described and discussed. Not all bacteriologically positive horses were clinically positive referring to asymptomatic carriers. Shedding of *Streptococcus equi subsp. equi* was intermittent. Source of infection in the investigated farm was determined. The cumulative incidence of the disease (strangles) of the investigated horses (which were bred in a wide-open yard) during the period of investigation was increased gradually and referred to the presence of assisting factor assisted in transmission of the infection from horse to another. This factor was monitored. Hemogram of the diseased horses with uncomplicated strangles showed leukocytosis with neutrophilia, while in cases that had signs of purpura hemorrhagica (parasitic free) showed anemia (microcytic hypochromic) and leukocytosis with neutrophilia and eosinophilia suggesting allergic responses to the bacterial infection led to blood losses. Therapeutic trials were successfully achieved. It is concluded that, the uncomplicated form of strangles in horses that bred in acceptable hygienic environment possibly has no systemic anti-microbial drugs. The undesirable therapeutic entrance of this classical form by intensive systemic antibiotics delayed the full maturation of the abscessed lymph nodes (inadequate immune response). This persisted non-maturated abscessed node may produced a favorable chance for accelerating the complications, or probably adversely increased the sensitization of the horses to streptococcal infection and/or its toxic products. Anti-allergic drug as one of the therapeutic lines of strangles may have a beneficial value in treatment and probably preventing purpura

hemorrhagica. The negative results of 2 bacteriological nasal swabs with one-week interval were not sufficient for full declaration that horses become *Streptococcus equi subsp. equi* free. Three swabbing at weekly intervals is therefore recommended.

*Keywords: Strangles-Purpura hemorrhagica-Incidence-Clinical-Hematology-Therapy*

## INTRODUCTION

Strangles of the family equidae was reported many decades ago, yet it remains a common and important disease worldwide. This disease characterized chiefly by pathological alterations (mucopurulent inflammation) in the upper respiratory tract with regional lymph node involvement (nasopharyngeal region) of horses (Olsson *et al.*, 1994 and Newton *et al.*, 1997).

Previously Bryans *et al.* (1964) reported that *Streptococcus equi* was the pathogenic etiologic agent responsible for strangles in horses without any aiding agent like viruses or others. However Gerber (1986) concluded that the incidence of strangles in horses decreased since equine influenza disease had been effectively controlled via vaccination. This may reveal that the equine influenza virus or probably other predisposing factors plays a pivotal role in the development of strangles through minimizing the immunological condition of the infected host.

Newton *et al.* (1997) reported that transmission of *Streptococcus equi* was not fully cleared but it survived for varying lengths of time either in the animal hosts (Olsson *et al.*, 1994) or in the environment (Jorm, 1991). However, Ames (1995) reported that direct and indirect contact through fomites that had been contaminated by the nasal discharges or saliva of the infected animals was commonly incriminated in transmission of the disease.

Bastard form of strangles, which was systemic spread of *Streptococcus equi* infection in the various organs of the infected host causing many serious problems, guttural pouch empyema, purpura hemorrhagica and deaths were encountered as complication of strangles in horses (Sweeny *et al.*, 1987). Purpura hemorrhagica was a largely sporadic, non-contagious disease of horses, characterized by symmetrical

or non-symmetrical, cold, localized subcutaneous edema of the infected hosts in association with petechiation (Blood and Studdert, 1990).

The aim of the following work was carried out to describe the clinical picture of some diseased horses on a farm and to determine the probable etiological agent(s) responsible for such cases. Hematological and parasitological examinations of the diseased cases were done. Therapeutic trial of the diseased animals was also achieved.

## **MATERIAL and METHODS**

### **ANIMALS:**

A horse farm located in El-Baliana, Sohag governorate-Upper Egypt consisted of 23 adult horses (aged from 4-10 years old) were clinically and bacteriologically examined. Two of them were recently purchased from the commercial market since 3 weeks before the first time of clinical examination (27/03/1989) and they were previously suffered from mandibular lymphadenitis. All horses were reared in a wide-open yard at day and at night they were kept in a semi-closed stable. The hygienic measures of that stable and yard were apparently acceptable. Feeding of those horses was principally on roughage with suitable amount of concentrates and green fodder. Drinking of the horses occurred through one source, a wide capacity plastic barrel filled with underground water (hand pump). This barrel's water was changed every day or two days. The owner with massive doses of streptomycin mixed with penicillin (streptopencid-Cid, Egypt) and/or oxytetracycline (panterramycin-Pfizer, Egypt) treated any horse showed nasal discharge with the first signs of mandibular lymphadenitis. The investigated horses were routinely injected by thiabendazole (Equizole suspension, MSD) every 3 or 4 months (two doses with 11-21 days apart) for control of helminthiasis particularly red worms. Owner's complaint and detailed history were taken and discussed.

### **Cumulative incidence of the disease:**

The cumulative incidence of the present disease (strangles) in the investigated farm during the period of investigation was calculated according to the criteria of Thrusfield (1986) who reported that cumulative incidence was an indication of the average risk of developing disease during particular time.

## **COLLECTION OF THE SAMPLES AND LABORATORY TECHNIQUES:**

### **Bacteriological examinations:**

Forty-six deep nasal swabs were collected from all examined horses (one nasal swab per each nasal opening). These collected swabs were immediately immersed into sterile centrifuge tubes containing brain-heart infusion broth (BHI) (Gibco) supplemented with 10 % sterile inactivated horse serum. On the other side, the abscessed lymph nodes of the diseased horses were also sampled in BHI's tubes. The inoculated tubes were incubated at 37° C for 24 hours. The incubated tubes were centrifuged at 3000 xg for 10 minutes and the sediments were plated onto plates containing brain-heart infusion broth (Gibco) supplemented with 1.5 % agar (Oxoid) and 5 % citrated sheep blood. The inoculated plates were incubated at 37° C for 24–72 hours. The characteristic colonies of family streptococci, particularly the hemolytic ones, were picked up and purified, and thereafter subjected to Lancefield serological grouping by using latex agglutination test-kits (Oxoid). The beta hemolytic lancefield group C streptococci were biochemically tested. These biochemical tests were based on the fermentation of sugars; lactose, ribose, sorbitol and trehalose according to the methods described by Grant *et al.* (1993).

Water samples from the drinking barrel were collected in sterile 100-ml. flask immediately after drinking of the horses. As soon as possible, the collected water sample was filtrated through filter paper. Thereafter, the filtrate were distributed in sterile centrifuge tubes and centrifuged at 5000 xg for half-hour and the sediments were streaked onto serum-BHI's agar plates and incubated as described above. The examined horses were bacteriologically sampled more than once over the following three weeks.

### **Hematological examinations:**

Blood samples of the examined horses were collected in vials containing anti-coagulant, ethylene diamine tetra acetic acid, disodium salt, and were subjected to some hematological examinations (Table 6) according to the methods described by Coles (1980).

### **Parasitological examinations:**

Fecal samples from all examined horses were collected in separate nylon bags. Flotation-sedimentation technique by using saturated salt

solution was done for fecal analysis according to the methods described by Smyth (1994).

**Therapeutic trial:**

The examined horses were clinically classified into 5 groups according to the occurrence of the disease (Table 7) and each group was treated according to the results of clinical and laboratory examinations.

**Statistical analysis:**

The statistical analysis of the obtained data was done by means of a Microstat Computer Program (1984).

## RESULTS

**Clinical examinations:**

According to the observed clinical signs, the clinically examined horses were classified into five groups as illustrated in Table 1.

***The first and second groups:***

Seven horses were suffered from clearly systemic reactions including elevation of the body temperature ( $40.3 \pm 0.1$  °C), congested ocular mucous membranes and the sclera vessels were engorged with blood, hurried respiration in association with bilateral or unilateral mucopurulent nasal discharge. Their heads and necks were clearly extended and the mandibular lymph nodes were either swollen or spontaneously ruptured discharging whitish pussy material. These horses had no ability to swallowing the ingested food.

***The third group:***

Five horses had a drowsy appearance whereas their movements were markedly reduced and their heads and necks were in low level near the ground (Fig. A). However, they were respond to the external stimuli. Closed examinations of these horses showed well-defined edematous swellings localized in their heads (Fig. B). This swelling was unsymmetrical in distribution and included all regions of the head including the intermandibular space with difference in the degree of edema. Edema in the both supra-orbital fossae was clearly remarkable (Fig. C). This edematous swelling was increased gradually in size causing distention on the external covering (skin) particularly above the supra-orbital fossae resulting lesion similar to sun brunt or eczema (Fig. C). This lesion was oozed a serum like fluid. Signs of pain (nervousness) were noticed during hand palpation on the edematous lesions of these horses

particularly on the inter-supra-orbital fossae (forehead) and above the cheeks.

The eyes of the diseased horses of the third group were approximately closed due to the edematous swelling of the both eye-lids, and the ocular mucous membranes were markedly red in color (Fig. D). The mucous membranes of the buccal cavities of those horses had petechial hemorrhages and the tongues were edematous, slightly immovable and bluish reds in color. Erosions on the dorsal surface and the lateral borders of the tip of this bluish edematous tongue were observed (Fig. E and F). The nasal cavities were also red in color without discharging secretions.

The third group horses could pick the applied food, with slightly mastication and thereafter they regurgitated the masticated food from their mouths and nostrils and the fecal matters were dark green or blackish in color and firm in consistency. Their body temperature were around  $38.7 \pm 0.2$  °C while the heart rates were  $87 \pm 8$  / min.

#### ***The fourth and fifth groups:***

Eleven horses appeared to be normal. Two of them were purchased since 3 weeks before the first clinical visiting. The mandibular lymph nodes of these purchased cases appeared slightly enlarged without abscess formation. Systemic reactions could not be detected in the these grouped horses.

#### **Bacteriological examinations:**

Bacteriological examinations of the collected samples adverted to the presence of *Streptococcus equi subsp. equi* either alone or mixed with other Gram's positive bacteria of genus streptococci (Table 5). *Streptococcus equi subsp. equi* coupled with unidentified Gram's positive and negative bacteria were isolated from the collected water samples.

#### **Hematological examinations:**

The hematological examinations of the diseased horses with typical uncomplicated strangles showed significant ( $P < 0.05$ ) leukocytosis with neutrophilia. Hemogram of horses that had signs of purpura hemorrhagica showed significantly decreased ( $P < 0.05$ ) values of erythrocytes, hemoglobin, packed cell volume, mean corpuscular volume and mean corpuscular hemoglobin in association with highly significant increased ( $P < 0.01$ ) in leukocytic counts with eosinophilia and neutrophilia.

#### **Parasitological examinations:**

Parasitological examinations of the collected fecal samples revealed that the examined horses were significantly parasitic free.

#### **Therapeutic Trial:**

The therapeutic trials of the diseased cases with typical uncomplicated form of strangles were respond well to the applied treatment. Both anti-allergic disorders drug (Clemastine fumarate) in association with non-steroidal anti-inflammatory drugs (NSAID) (Acetylsalicylic acid and Diclofenac sodium) had a beneficial value in treatment of the complicating form of strangles (purpura hemorrhagica).

## **DISCUSSION**

Pyrexia, mandibular lymphadenitis and bilateral or unilateral nasal discharges in the diseased horses in association with isolation of *Streptococcus equi subsp. equi* microorganism may prove that the present disease in the investigated horse's farm was strangles.

Beside the existence of typical form of strangles, some cases showed unsymmetrical, painful edematous swelling localized in their heads with petechiation of the ocular and oral mucosae, and without marked systemic involvement, except tachycardia, may refer to the occurrence of purpura hemorrhagica as a complication. Similar clinical findings in horses had purpura hemorrhagica were reported by Blood and Studdert (1990) with exception of signs of pain, where the latter authors concluded that the edematous swelling was (cold) painless. The observed signs of pain during hand palpation of the edematous head particularly on the intra-orbital fossae and cheeks was probably related to the progressive increasing of the edematous swellings, which may induced pressure on the cutaneous sensory branches of the trigeminal nerve (supraorbital, Infraorbital and mental nerves). The latter superficial nerve (trigeminal) was sensory to the skin and the deeper tissues of the face and was motor to the first pharyngeal (mandibular) arch origin (Dyce *et al.*, 1996). This account may also give an explanation to the difficulty of swallowing in those horses. Moreover, pharyngeal compression due to edematous swelling in the intermandibular space (around the throat) of the edematous heads-horses should not also be neglected as a possible reason for regurgitation of food from their mouth and nostrils. The bluish coloration and the erosions of the tongues of the edematous heads-horses were



probably due to pharyngeal edema resulting decreases of the blood flow to the tongue.

Although the bacteriological examinations of the recently purchased horses that introduced directly to the old horses in the farm without hygienic precautions were positive to *Streptococcus equi subsp. equi*, there were no clinical abnormalities observed by the owner or the authors for 40 days post purchasing. This may prove that the new introduced cases were the main source of infection to the other old horses in the farm (indoor infection). Moreover, isolation of *Streptococcus equi subsp. equi* organism from the nostrils of purchased cases and other horses (Table 4) without clinical abnormalities may also advert to the presence of asymptomatic-carrier status. Such conditions were reported by Olsson *et al.* (1994) and Newton *et al.* (1997) who found that the average duration of the carrying period of *Streptococcus equi subsp. equi* infection was  $23 \pm 16$  months. Therefore, culling of the symptomless carrier-animals is a paramount idea to exclude the major source of infection in the farm. Otherwise, the occurrence of strangles is possibly stilling problem.

Table 2 indicated that the cumulative incidence of the present disease (strangles) of the investigated horses was increased gradually during the period of investigation (Fig. 1) referring to the presence of assisting factor assisted in transmission the infection from animal to another. Horses contracted *Streptococcus equi subsp. equi* infection through inhalation of droplet infection or through ingestion of contaminated material (Ames, 1995). The latter mode of infection was more strongly suspected than the former, because of the examined horses were free-lanced in a wide-open yard during the periods of clinical examinations and the follow up. This suspicion was supported by isolation of the causative agent of strangles from the collected water samples. Consequently the method of spreading of infection from horse to horse was possibly explained and subsequently the ascending increases in the values of cumulative incidence of the disease of the investigated farm were elucidated.

In the present farm, because of the running water was not available, changing of drinking water twice, or may be more, daily has probably a beneficial value. Apparently, it is preferable that, in the infected small farm with the absence of running water, each horse has a separate suitable water container and after drinking these containers should

carefully be washed or immersed in effective disinfectant's bath. Jorm (1991) concluded that povidone iodine, chlorhexidine gluconate and glutaraldehyde disinfectants at recommended concentration had strong inhibitory effects on *Streptococcus equi subsp. equi* destroying it within 1.5 hour. It is suggested that further investigations should be carried out to declare the survival period of *Streptococcus equi subsp. equi* microorganism in the stagnant and semi-stagnant water is therefore valuable.

The obtained results in Table 4 showed that some horses yielded *Streptococcus equi subsp. equi* from their nostrils on the first trial of isolation and they became bacteriologically negative on the second trial. On the third trial, these cases returned to the positive status. This may reveal that shedding of *Streptococcus equi subsp. equi* microorganism through the nostrils of bacteriologically or clinically positive cases was intermittent. Table 4 also showed that some cases were clinically and bacteriologically negative after 2 occasions of isolation. However the authors could not emphasized that these cases were completely free of infection because of case N<sup>o</sup>. 22 (clinically negative case) shed *Streptococcus equi subsp. equi* from it nose only on the third trial of isolation. This may indicated that two trials of isolation with one week apart were probably not sufficient for declaration that horses become infection free. Three occasions of isolation is therefore recommended.

Table 5 and Fig. 2 showed that 85.71 % of the clinically diseased horses with strangles shed *Streptococcus equi subsp. equi* coupled with either *Streptococcus equisimilis* or *Streptococcus zooepidemicus*. This may refer to synergistic situation between these types of bacteria in the occurrence of strangles.

From hematological point of view, Coles (1980) and Smyth (1994) concluded that neutrophilia was commonly attributed to pyogenic bacterial infection while eosinophilic responses were usually ascribed to parasitic infestation or allergic reaction. The obtained results of the hematological examinations of the diseased horses with typical uncomplicated form of strangles showed leukocytosis with neutrophilia indicating pyogenic bacterial infection. *Streptococcus equi subsp. equi* was one of the pus producing microorganisms (Grant *et al.*, 1993 and Ames, 1995). This may give account for failing of the applied treatment by the owner where pus was a protective agent. On the other hand, hemogram of the horses that had signs of purpura hemorrhagica showed anemia (microcytic - hypochromic) and leukocytosis with neutrophilia and

marked eosinophilia (with the absence of parasitic infestation) suggesting undesirable allergic response to the streptococcal infection or probably its toxic products led finally to blood losses. This suggestion was supported by the successful treatment of those cases by anti-allergic disorders drug (clemastine fumarate). Galan and Timoney (1985) concluded that horse's purpura hemorrhagica was undesirable unions (immune complexes) between antigen (M protein of *Streptococcus equi subsp. equi*) and antibody (IgA). This complexes was circulated in the blood and deposited in the small cutaneous blood vessels causing vasculitis leading to escaping of the plasma and blood constituents from the vessels into the surrounding tissues. Consequently the observed signs of edema, petechiation, serum exudation, tachycardia and anemia of the edematous heads-horses were probably explained. The resulted anemia may be responsible for the dark colored stool of the edematous heads horses.

Increasing the degree of body resistance of horses to overcome *Streptococcus equi subsp. equi* infection could be stimulated by bathing and poulticing the abscessed nodes to accelerate the full maturation of these abscesses and ruptures (Hartwigk and Gerber, 1986; Rooney and Robertson, 1996). Conversely, delayed maturation of the abscesses by intensive doses of systemic antibiotics may lead to decreasing the body resistance to the infection (inadequate immune response). The persisted non-maturated abscessed node in horses may produced a favorable chances for developing of the complications of the disease (Ames, 1995) or may accelerating the sensitization of the body to infection. This conclusion may be explain the high prevalence of purpura hemorrhagica in the respective horses (Table 3) whereas, according to the history taking, the owner treated the infected horses in the early stages of the disease with massive doses of antibiotics without waiting till full maturation of the abscesses.

Declining of the leukocytes (Fig. 6) of the horses had classical uncomplicated form of strangles after treatment by careful draining and irrigation of the full-maturated mandibular abscesses with betadine lotion, and without systemic antibiotic therapy, suggested that the role of antibiotics was not greatly essential. Such suggestion was coincided with the opinion of Gerber (1986) who reported that prognosis of the classical form of strangles in horses without any complication was generally good and required no chemotherapy.

It is concluded that, the typical uncomplicated form of strangles in horses that bred in acceptable hygienic environment possibly has no

systemic anti-microbial drugs. The undesirable therapeutic entrance of this form by intensive systemic antibiotics delayed the full maturation of the abscessed lymph nodes (inadequate immune response), and may produced a favorable chance for accelerating the complications, or probably adversely increased the sensitization of the horses to streptococcal infection and its toxic products. Administration of anti-allergic drug as one of the therapeutic lines of the uncomplicated strangles may have a beneficial value in treatment and probably preventing the complications (purpura hemorrhagica). The negative results of 2 successive bacteriological nasal swabs with one week apart were not sufficient for declaration that horses become *S. equi* free. Three swabbing at weekly intervals is possibly recommended.

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**Table 1: Classification of the examined horses.**

Group	Nr. of animal	Clinical signs
I	4	Typical form of strangles including : - Enlarged and painful mandibular lymphadenitis (unopened) - Bilateral nasal discharges (3 cases, 42.86 %) - Unilateral nasal discharge ( 1 case + 3 cases of group II, 57.14%) - Marked systemic reactions
II	3	Typical form of strangles : with opened (spontaneously ruptured) lymph nodes
III	5	Horses showed painful edematous swellings in their heads without marked systemic reaction excepting tachycardia
IV	2	Horses were apparently healthy but their mandibular lymph nodes were slightly enlarged with no systemic involvement
V	9	Horses appeared to be normal

**Table 2: Cumulative incidence (CI) of the clinical form of strangles  
of the investigated horses.**

Date	Nr. of the examined horses	Diseased cases Nr. (%)	CI of the classical form of strangles
27/03/1998 3/04/1998	23	5 (21.17)	0.22 horse for the 1-week period
4/04/1998 11/04/1998	23	3 (13.04)	0.35 horse for the 2-week period
12/04/1998 19/04/1998	23	4 (17.39)	0.48 horse for the 3-week period

**Table 3: showed that the percentage of purpura hemorrhagica (P.H.)  
of the clinical diseased horses (strangles)**

Date	Nr. of the diseased cases	Nr. of horses showed the complicating form (P.H.)	% of the complicating form
27/03/1998 3/04/1998	5	2	40.0
4/04/1998 11/04/1998	3	2	66.7
12/04/1998 19/04/1998	4	1	25.0
<b>Total</b>	<b>12</b>	<b>5</b>	<b>41.7</b>

**Table 4: Results of bacteriological analysis of the collected nasal swabs (NS) of the examined horses (23).**

No. of case	Date	NS	C	Date	NS	C	Date	NS	C
1	27/3/98	2	+	6/4/98	2	+	12/4/98	2	-
2	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
3	27/3/98	2	-	6/4/98	2	+	12/4/98	2	+
4	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
5	27/3/98	2	-	6/4/98	2	+	12/4/98	2	+
6	27/3/98	2	+	6/4/98	2	+	12/4/98	2	-
7	27/3/98	2	+	6/4/98	2	+	12/4/98	2	+
8	27/3/98	2	+	6/4/98	2	+	12/4/98	2	+
9	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
10	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
11*	27/3/98	2	+	6/4/98	2	-	12/4/98	2	+
12*	27/3/98	2	+	6/4/98	2	-	12/4/98	2	+
13	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
14	27/3/98	2	-	6/4/98	2	+	12/4/98	2	+
15	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
16	27/3/98	2	-	6/4/98	2	+	12/4/98	2	-
17	27/3/98	2	+	6/4/98	2	-	12/4/98	2	+
18	27/3/98	2	-	6/4/98	2	+	12/4/98	2	-
19	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
20	27/3/98	2	-	6/4/98	2	-	12/4/98	2	-
21	27/3/98	2	+	6/4/98	2	+	12/4/98	2	-
22	27/3/98	2	-	6/4/98	2	-	12/4/98	2	+
23	27/3/98	2	+	6/4/98	2	+	12/4/98	2	-
<b>Total</b>		<b>46</b>	<b>9</b>		<b>46</b>	<b>11</b>		<b>46</b>	<b>9</b>

C: Culture

+: *Streptococcus equi* was isolated either alone or coupled with other bacteria (Table 5)

-: *Streptococcus equi* could not be isolated

\*: Recently purchased cases.

N.B. Not all culturally positive cases were clinically diseased.

**Table 5: Frequent distribution of the isolated bacteria from the clinically diseased horses (7)**

Nr. of cases	Isolates	%
1	<i>S. equi subsp. equi</i>	14.29
4	<i>S. equi subsp. equi</i> <i>S. equismils</i>	57.14
2	<i>S. equi subsp. equi</i> <i>S. zooepidemicus</i>	28.57

**Table 6: Hematological examinations of the investigated horses.**

Blood—Parameters	Unit	A (n=5) $\bar{X} \pm SD$	B (n=8) $\bar{X} \pm SD$	C (n= 7) $\bar{X} \pm SD$
Erythrocytes	( $\times 10^6 \mu\text{l}$ )	5.8 $\pm$ 3.1*	6.5 $\pm$ 2.9	7.10 $\pm$ 2.6
Hemoglobin	(g/dl)	6.1 $\pm$ 1.2**	11.8 $\pm$ 2.0	13.71 $\pm$ 1.5
Hematocrit	(%)	19.5 $\pm$ 1.5**	31.5 $\pm$ 1.8	39.8 $\pm$ 3.0
MCV	(fl)	33.62 $\pm$ 1.0*	48.5 $\pm$ 3.4	56.14 $\pm$ 2.4
MCH	(pg)	10.52 $\pm$ 0.5*	18.15 $\pm$ 2.7	19.31 $\pm$ 1.5
Leukocytes	( $\times 10^3 \mu\text{l}$ )	19.1 $\pm$ 4.2**	16.8 $\pm$ 2.3**	8.90 $\pm$ 3.1
Neutrophils	(%)	53.0 $\pm$ 3.1*	61.2 $\pm$ 4.2**	49.6 $\pm$ 3.5
Eosinophils	(%)	13.0 $\pm$ 1.8**	5.2 $\pm$ 2.3	6.1 $\pm$ 2.8
Basophils	(%)	0.2 $\pm$ 1.0	0.1 $\pm$ 0.2	0.3 $\pm$ 0.49
Lymphocytes	(%)	35.1 $\pm$ 2.3	32.8 $\pm$ 2.3	40 $\pm$ 7.1
Monocytes	(%)	1.0 $\pm$ 1.2	1.0 $\pm$ 2.3	3.5 $\pm$ 2.0

A : Horses showed signs of purpura hemorrhagica

B : Horses showed signs of classical form of strangles

C : Apparently healthy horses

$\bar{X}$ : Mean

SD : Standard deviation.

MCV: Mean corpuscular volume

MCH: Mean corpuscular hemoglobin

\* : Significant ( $p > 0.05$ ).

\*\* : Highly significant ( $p > 0.01$ ).



**Table 7: Therapeutic trial of the examined horses**

Group	Clinical picture	Treatment
I	Horses showed signs of purpura hemorrhagica (5 cases)	1-Anti-allergic disorders drug (Clemastine fumerate, 2 mg /head twice daily, I/M) 2-Diclofenac sodium (NSAID) 75 mg daily I/M. 3-Acetylsalicylic acid (NSAID) 1.8 gm daily (I/M). 4-Dextrose-saline I/V infusion. 5-Penicillin G. Sodium, Five million units per head twice dose on the first day followed by procaine penicillin 20000 IU / Kg BW. daily (I/M).
II	Horses showed signs of the typical form of strangles with swelled unopened lymph nodes. (3 cases)	1-Ripening the abscess till point of full maturation by using Icthyol 10% cream and thereafter draining and irrigating the nodes by using 10 % Povidone-Iodine lotion. 2-Dextrose-saline I/V infusion 3-Course of penicillin therapy as described for 10-15 days till disappearance of the systemic reactions. 4-Clemastin fumerate 2mg/head daily (I/M)
III	Horses showed signs of the typical form of strangles with swelled unopened lymph nodes (2 cases)	1-Ripening the abscess till point of full maturation by using Ecthyol 10% cream and thereafter draining and irrigating the nodes by using 10 % Povidone-Iodine lotion. 2 - Dextrose-saline I/V infusion
IV	Horses showed opened submandibular lymph nodes without marked systemic reaction. (2 cases).	The swelled nodes were surgically squeezed, cleaned and irrigated with 10 % betadine lotion
V	Apparently healthy cases	Had no therapy

Clemastine fumerate = Tavegyle ampoule (Novartis pharma, Cairo)

Diclofenac sodium = Voltaren ampoule (Novartis pharma, Cairo)

Acetylsalicylic acid = Aspegic vial. (Amriya/Synthelabo, Egypt)

Povidone-Iodine =Betadine (Nile Co., A.R.E.)

NSAID: Non-steroidal anti-inflammatory drug

\*: The leukocytic counts of these cases were monitored (Fig. 3).

Fig. 1: Trend of the cumulative incidence of the disease (strangles) of the examined horses in the investigated farm.

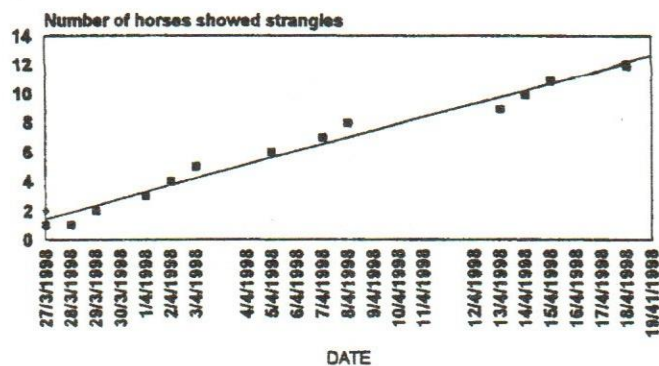


Fig. 2: shows that 85.7 % of the clinically diseased cases shed *Streptococcus equi subsp. equi* coupled with other cocci (*S. equisimilis* and *S. zooepidemicus*) (B) while 14.3 % of these case shed *Streptococcus equi subsp. equi* alone.

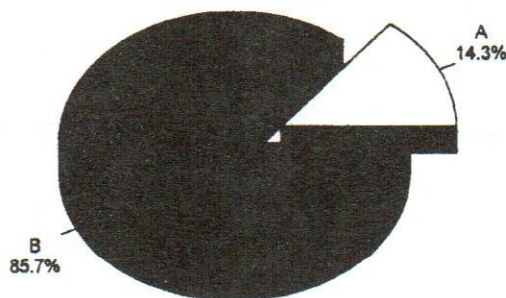


Fig. 3: Average Leukocytic counts before and after abscessation in 2 horses had classical uncomplicated form of strangles.

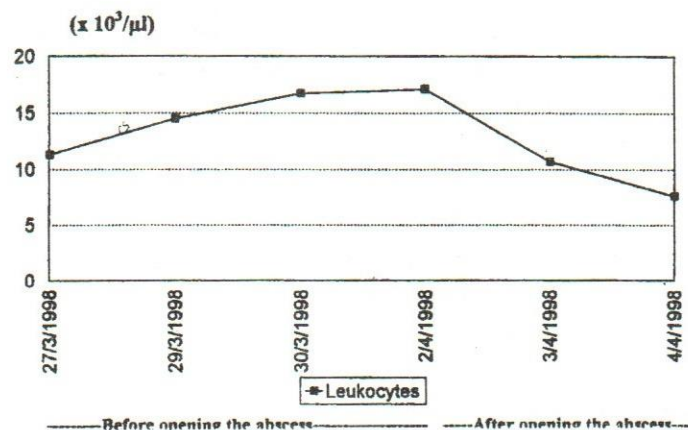


Fig. A: A drowsy appearance of the diseased horse with purpura hemorrhagica. Note the position of head and neck, are usually near the ground.

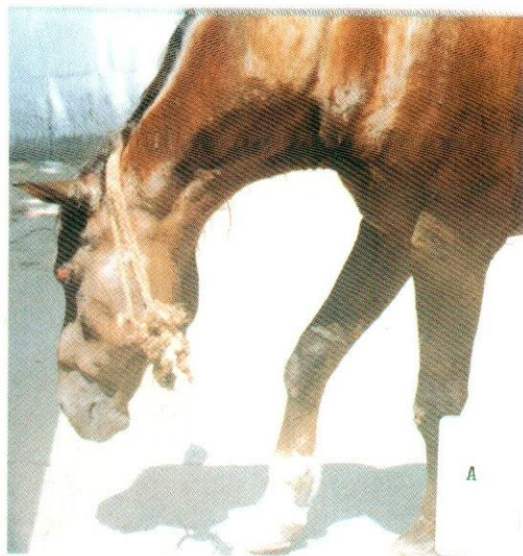


Fig. B: Unsymmetrical well-defined edematous swelling in the head of the infected horse with purpura hemorrhagica

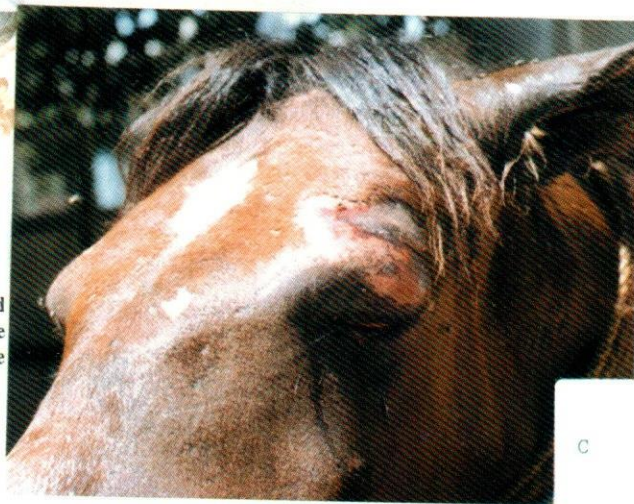


Fig. C: Severe edematous swelling of the supra-orbital fossa and the covering skin above this swelling has a lesion similar to sun-brunt or eczema.

Fig. D: Severe edematous swelling in the both eye-lids with congested ocular mucosa and pin-point petechial hemorrhages.



Fig. E: Small erosions on the lateral borders of the tip of the tongue of the diseased horse with purpura hemorrhagica.



Fig. F: large erosion on the tip of the dorsal surface of the tongue of the diseased horse with purpura hemorrhagica.

