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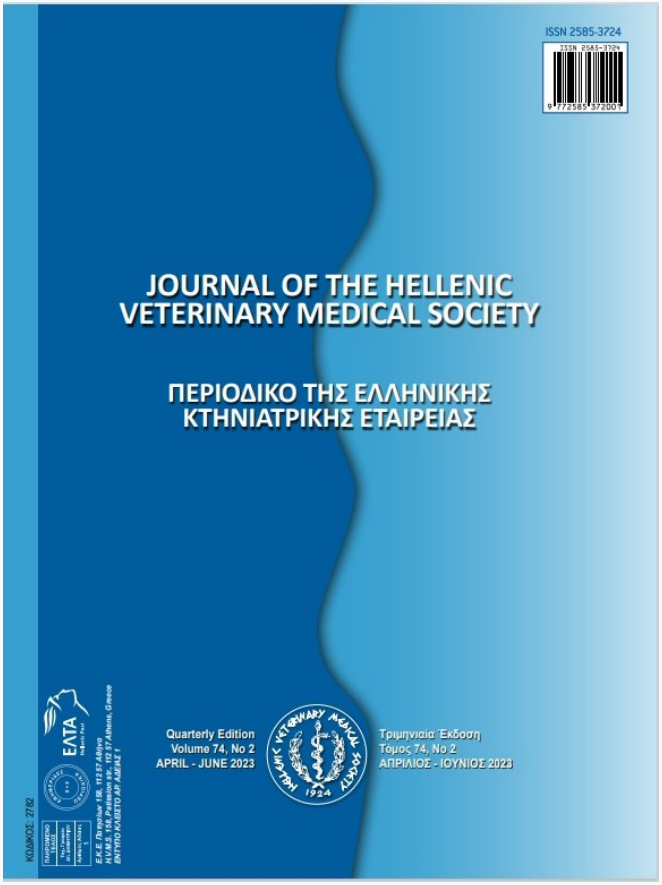

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Case report
Κλινικό περιστατικό

Strangles in a stallion in southeast Nigeria: clinical and pathological report

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ABSTRACT. *Streptococcus equi* subspecies *equi* is the causative agent of strangles, a highly contagious mucopurulent upper respiratory tract infection of horses. This report describes the clinicopathologic findings of a fatal case of pulmonary form of strangles in a two-and-half-year-old stallion presented at a Veterinary Teaching Hospital in Southeast Nigeria. Treatment was attempted in quarantine using high doses of procaine penicillin; but it was unsuccessful. The clinical signs observed in the affected horse include a left unilateral supra-orbital swelling containing purulent matter, which progressed to a chronic intractable exudative pus. Anaemia with leukocytosis (due to neutrophilia) were observed in the haematologic parameters. Macroscopic and histologic lesions were multifocally extensive abscessing necrotic lymphadenitis and bronchopneumonia with inflammatory oedema and infiltrate of polymorphonuclear leukocytes.

***Keywords*:** horse; strangles; clinicopathologic findings.

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CASE HISTORY

Strangles, caused by infection with the bacterium *Streptococcus equi* subspecies *equi* (*S. equi* subsp. *equi*), is the most common, highly contagious and suppurative upper respiratory tract infectious disease of horses worldwide. Although the official name of the causative agent is *S. equi* subsp. *equi*, we have decided to use the descriptive term *S. equi* throughout this report based on its widespread usage in the scientific literature. The bacterium is encapsulated and may vary in pathogenicity from strain to strain (Taylor and Wilson, 2006). Transmission of the disease occurs by direct contact with the mucopurulent discharges from infected horses or from contaminated fomites (Laus et al., 2007; Lindahl et al., 2011). *Streptococcus equi* is highly host-adapted and causes disease only in horses, mules, and donkeys (Timoney, 1993).

The first clinical signs of strangles are sudden onset of fever, anorexia, depression, and serous nasal discharge that rapidly becomes mucopurulent, which appear after an incubation period of approximately 3 to 8 days, and generally last for 3 or 4 weeks (Sweeney, 1996; Timoney and Kumar, 2008). Sometimes, a moist cough may be present (Reile and Genetzky, 1983). These symptoms are followed by acute swelling and subsequent abscess formation in the submaxillary, submandibular, and retropharyngeal lymph nodes. Retropharyngeal lymph node enlargement may cause obstruction of the oro- and nasopharynx with subsequent dyspnoea (Hardy and Léveilé, 2003). Subsequently, the submandibular lymph nodes may become enlarged, firm, painful, and result in dysphagia (Timoney and Mukhtar, 1993). The name strangles was coined because affected horses often suffocate as the lymph nodes became enlarged and obstruct the pharynx or airways (Sweeney et al., 2005). Different presentations of the disease, from a mild nasal discharge to "bastard strangles" - a metastatic form of strangles which spreads to lymph nodes, chest, abdomen and other parts - may be seen in infected horses (Sweeney, 1996; Slater, 2003; Robinson, 2009; Arias, 2013). Documented complications associated with *S. equi* infection are numerous and, generally are the result of local, haematogenous or lymphatic spread of bacteria to other sites (Ford and Lokai 1980; Barratt-Boyes et al., 1991; Sweeney, 1996; Slater, 2003; Robinson, 2009; Arias, 2013). Diagnosis of strangles is usually based on the presence of classic clinical signs of lymphadenopathy with subsequent abscessation and rupture. A definitive diagnosis of *S. equi* infection is usually based on isolation of *S. equi* from either a nasal or lymph node discharge. A polymerase chain reaction (PCR) method allowing detection of *S. equi* in horses can be achieved by performing real-time PCR on DNA extracted from bacterial cultures (Lindahl et al., 2011), or on DNA extracted directly from nasopharyngeal washes or nasal swab samples (Timoney and Mukhtar, 1993). A complete blood count and plasma fibrinogen concentration are useful to support the diagnosis and may also differentiate horses with acute *S. equi* infection from horses with *S. equi zooepidemicus* infection (Knowles et al., 2010) and acute viral processes (Timoney and Mukhtar, 1993).

In this report, we describe the clinical signs, haematological, biochemical, microbiological, macroscopic, and histopathologic findings of a suspected case of strangles in a stallion. To the best of our knowledge, this is the first report of strangles in Southeast Nigeria in particular and the entire country in general. What is known is that strangles occurs in Nigeria in that it is worldwide in distribution (Radostits et al., 2007). In all likelihood, however, the disease may have been under-diagnosed and unreported. A supporting fact is the December, 2018 strangles outbreak which killed about 4000 equids in Niger republic - a West African country which trades and shares border with Nigeria (Horse Media group, 2019).

A two-and-half-year-old male horse procured primarily for teaching and learning purposes in the Faculty of Veterinary Medicine, University of Nigeria, Nsukka, with the history of free-grazing and supplementation with grain-based concentrate, routine deworming but no immunoprophylaxis, was presented at the Veterinary Teaching Hospital with a primary complaint of a swelling above the left eye and mild depression with loss of appetite. At first presentation, the weight was 200kg and clinical observation confirmed the left unilateral supra-orbital swelling which was soft to touch and moderately warm, with blepharitis and corneal opacity (Fig. 1). The third and the lower eyelids were severely inflamed. Hyper-lacrimation and photophobia were also observed, and score of 2.25/5 was the estimated body condition.

Assessment of the heart rate showed tachycardia (heart rate 40 - 56 beat per minute [bpm], reference range 28 - 40 bpm), respiratory rate showed tachypnoea (16 - 44 cycles per minute [cpm], reference range 10 - 14 cpm), and temperature readings showed fluctuating pyrexia (temperature 37.2°C - 40.1°C, reference range 37.2°C - 38.1°C) (Mercks, 2015)

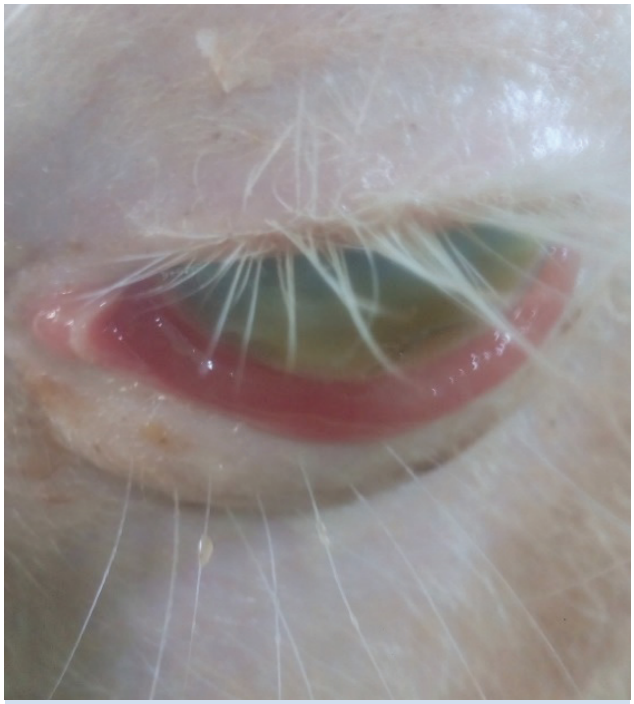

Figure 1: The stallion showing blepharitis corneal opacity

throughout treatment period. However, periods when normal body temperatures were recorded coincided with few hours after treatment with antipyretics. A timeline chart depicting the main events of this case report is presented in Fig. 2.

Two ml of blood were collected via jugular venipuncture into a clean sample bottle containing 2 mg of ethylenediamine tetraacetic acid (EDTA) powder as an anticoagulant for haematological analysis, and another 4 ml into a clean plain tube for serum biochemical analysis. The horse was restrained in a chute; the area around the proposed site of incision was washed, shaved, and scrubbed, with chlorhexidine solution. Thereafter the site was infiltrated with about 10 ml of lidocaine (2%). The swelling was surgically lanced (Fig. 3), and about 40 ml of grayish to yellow purulent material was expressed. The wound was packed with gauze soaked in tincture of iodine. A sample of the purulent material was aseptically collected from the lesion and sent for microbial culture and sensitivity test. A tentative diagnosis of clinical strangles was made based on the presenting signs. The differential diagnoses were glanders, melloidosis/pseudoglanders, ulcerative lymphangitis, photosensitization, actinomycosis, actinobacillosis, African horse sickness and brucellosis.

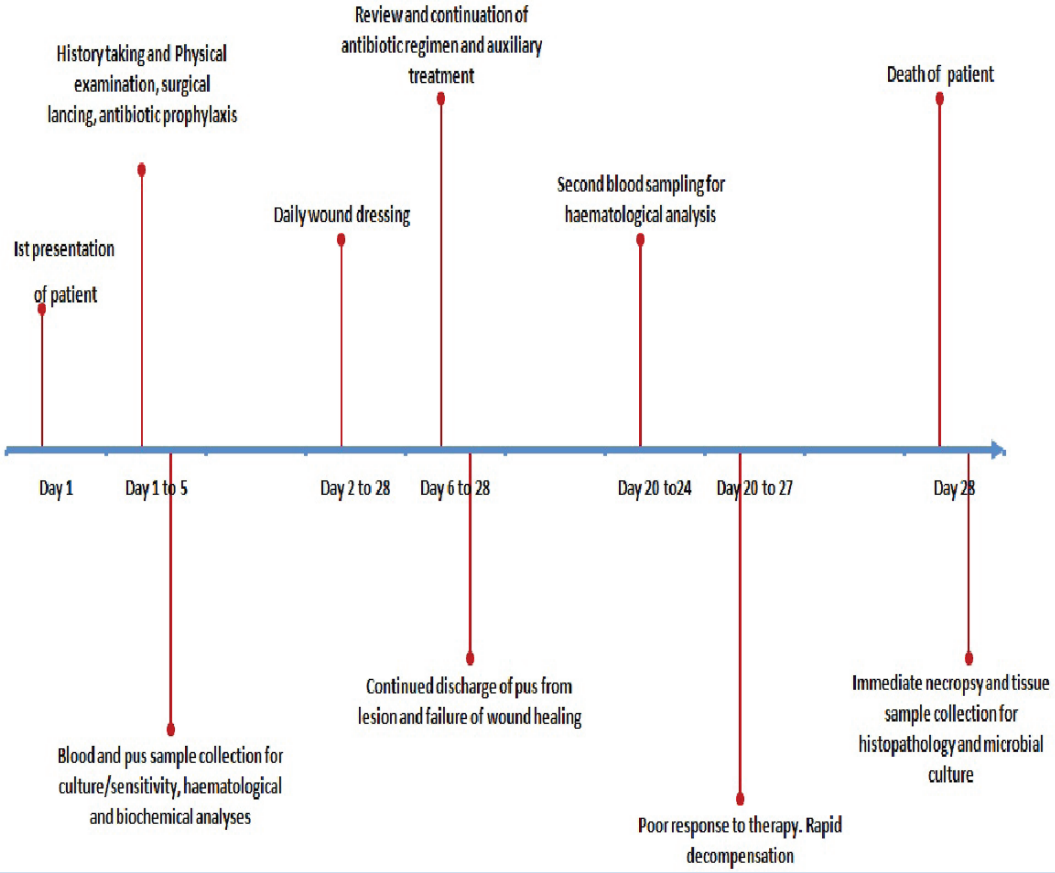

Figure 2: Timeline chart showing the case progression and outcome of intervention in a strangles patient in Southeast Nigeria

Figure 3: Patient (the stallion), during lancing of swelling (cutaneous abscessation), at first presentation

While awaiting the result of culture and sensitivity results, the following treatments regimen was instituted:

1. Injectable procaine penicillin (22,000 iu/kg bwt, IM, q12h for 7 days).
2. Injectable gentamicin (6mg/kg bwt, IV, daily for 7 days).
3. Injectable tetanus antitoxin (6000 iu, IM, Stat)
4. Injectable acetaminophen (10mg/kg bwt, IM, Stat)
5. Injectable multivitamin (10ml, IM, daily for 7 days).
6. Eye-drop autogenous antiproteinase (BID, daily for 7 days).

The surgical wound showed initial signs of healing before aggravating and failing to heal. About 20-40 ml of the grayish to yellow purulent matter was expressed daily during wound dressing, in addition to the volume discharged unto the horse-stable outside treatment periods. Daily physical examination of the horse revealed an expanding purulent lesion affecting the left maxillary, left temporal and left facial regions. The lesion was deep seated and constantly discharged pus to the exterior. The corneal lesion only showed a marginal improvement. The wound dressing was changed daily and packed with gauze and tincture of iodine.

The haematology and serum biochemistry results at first presentation of this case are shown on Table 1. The aseptically collected pus sample was cultured on 5% blood enriched nutrient agar and Sabouraud dextrose agar (SDA), for a minimum of 48 hours. Culture yielded beta-hemolytic mucoid (smooth) and matt (wrinkled) colonies (1-3 mm in diameter) of *S. equi*, with gram-positive spherical or ovoid cocci in pairs or chains, that were catalase-negative, with no growth in 6.5% NaCl. The identification of *S. equi* was also done through biochemical method: there was production of acid from glucose, maltose sucrose and galactose and no production of acid from lactose, sorbitol, or ribose. Antimicrobial susceptibility testing was performed following Clinical and Laboratory Standards Institute criteria. The growth was highly sensitive to penicillin G, bacitracin, cephalothin, nitrofurantoin, followed by erythromycin and gentamicin, and resistant to ofloxacin, norfloxacin and tetracycline. No growth was detected on fungal culture on SDA even after 7 days.

Following culture and sensitivity result, injectable procaine penicillin (30,000 iu/kg bwt, IM, q12h for 7 days) was the primary antibiotic administered, while the earlier supportive care, including injectable multivitamin (10ml, IM, daily for 7 days), injectable acetaminophen (IM, Stat), daily wound dressing and packing with tincture of iodine was continued.

By the third week after initial presentation, venous blood was collected for a second haematological analysis. The haemogram recorded is presented on Table 2. However, the prognosis became poor as the condition of the horse took a downward turn for the worse. The horse died on the 28th day after the onset of the treatment. The carcass was immediately presented for postmortem examination to the Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka.

The dead stallion was necropsied and examined for

Table 1: Haematology and serum biochemistry findings at initial presentation of a stallion naturally infected with ***Streptococcus equi***

Parameters	Patients Profile	*Reference Ranges	Remark
Total Erythrocyte Count (x10^6/uL)	3.56	6 - 10.4	Anaemia
Haemoglobin (g/dl)	9.6	10 - 16	Anaemia
Packed Cell Volume (%)	26	27 - 43	Anaemia
Total Leukocyte Count (x10^3/uL)	14.8	5.6 - 12.1	Leukocytosis
Mean Corpuscular Volume (fL)	73.0	37 - 49	Macrocytosis
Mean Corpuscular Haemoglobin Concentration (g/dL)	36.9	35.3 - 39.3	
Differential leukocyte count			
Lymphocytes (x10^3/uL)	4.4	1.2 - 5.1	
Neutrophils (x10^3/uL)	10.4	2.9 - 8.5	Neutrophilia
Band Neutrophils (x10^3/uL)	0	0 - 0.1	
Eosinophil (x10^3/uL)	0	0-0.8	
Basophil (x10^3/uL)	0	0-0.3	
Monocyte (x10^3/uL)	0	0-0.7	
Blood Cellular Morphology	Normal		
Serum biochemistry results			
Urea (mg/dl)	31	11-27	
Creatinine (mg/dl)	1.2	0.4-2.2	
Aspartate Amino Transferase (u/L)	59	160-412	
Alanine Aminotransferase (u/L)	12	2.7-21	

*The Merck Veterinary Manual (2016)

Table 2: Haematological findings by week three of treatment in a stallion naturally infected with ***Streptococcus equi***

Parameters	Patients Profile	*Reference Range	Remark
Total Erythrocyte Count (x10^6/uL)	3.87	6-10.4	Anaemia
Haemoglobin (g/dl)	9.99	10-16	
Packed Cell Volume (%)	28	27-43	
Total Leukocyte Count (x10^3/uL)	13.1	5.6-12.1	Leukocytosis
Mean Corpuscular Volume (fL)	72.35	37-49	Macrocytosis
Mean Corpuscular Haemoglobin Concentration (g/dL)	39.09	35.3-39.3	
Differential Leukocyte Count			
Lymphocytes (x10^3/uL)	4.2	1.2-5.1	
Neutrophils (x10^3/uL)	8.9	2.9-8.5	Mild Neutrophilia
Band Neutrophils (x10^3/uL)	0	0-0.1	
Eosinophil (x10^3/uL)	0	0-0.8	
Basophil (x10^3/uL)	0	0-0.3	
Monocyte (x10^3/uL)	0	0-0.7	
Blood Cellular Morphology	Normal		

* The Merck Veterinary Manual (2016)

macroscopic lesions. The lymph nodes, lungs, spleen, and kidney were collected, fixed in 10% buffered formalin, and embedded in paraffin. 5 µm sections were cut and stained with haematoxylin and eosin (HE) by routine method as described by Suvarna et al. (2018). Macroscopically, the carcass was dehydrated with poor general body condition. There was swelling in the submandibular area with thick purulent discharge (abscessation) from the sites of the swelling and rupture (Fig. 4A). On incision, the submandibular abscess (lymphadenopathy) of the carcass showed a thick, homogeneous white-yellow material (Fig. 4B), which was collected for culture. There was severe, diffuse lung tissue abscessation consisting of multifocal extensive yellow to dark-red or gray areas in the lungs and in the bronchi extending to the parenchyma (Fig.

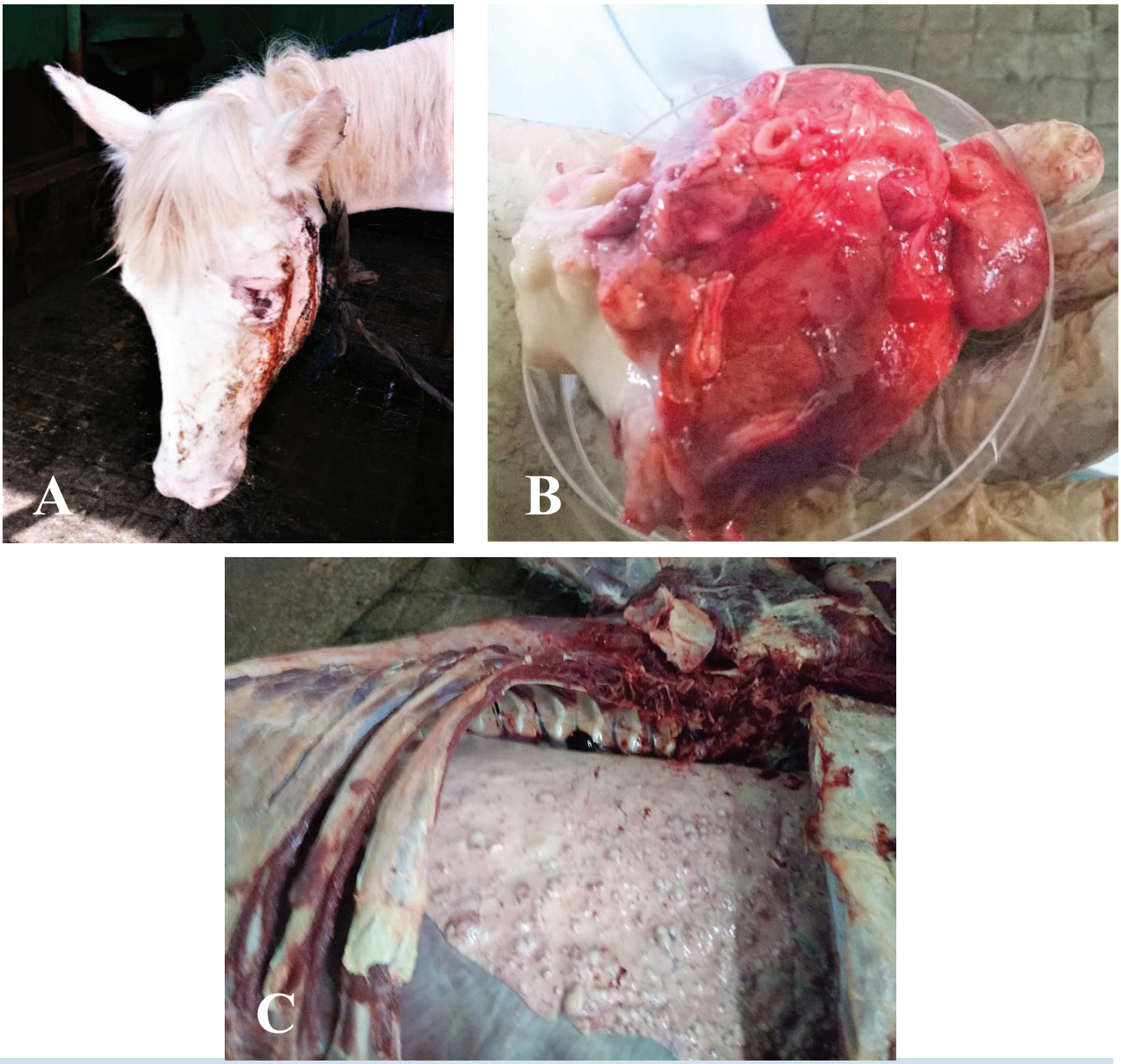

Figure 4: (A). Guttural pouch and lymph node abscessation and lancing with epilation and draining abscess mixed with oozing of serum in the affected horse with strangles. (B). A thick, homogeneous purulent discharge (white-yellow material [arrow]) from (A). (C). Marked diffuse lung tissue abscessation consisting of multifocal yellow to dark-red or gray areas in the lung parenchyma of the affected horse

4C). Histopathologically, the lymph node showed severe purulent, haemorrhagic and necrotic lymphadenitis with inflammatory oedema and infiltrate of polymorphonuclear leukocytes into the lymphoid follicles of the retropharyngeal node (Fig.5A, B). The lungs showed multifocally extensive neutrophilic exudate of an acute abscessing necrotic bronchopneumonia involving the bronchi, bronchioles, and alveolar spaces (Fig. 5C, D). In addition, moderate hyperaemia, collapsed alveoli and severe polymorphonuclear cells infiltration of the area accompanied by fibrin deposition characterized these lesions (Fig. 6A). The spleen showed moderately reactive white pulp and severe infiltration of the red pulp by polymorphonuclear leukocytes (Fig. 6B). The liver showed moderate necrosis of hepatocytes in the peri-portal area with mild mononuclear leukocytes infiltration (Fig. 6C).

Streptococcus equi was isolated from culture of the purulent material collected from the edge of the lesion pre-mortem and thick purulent material post-mortem.

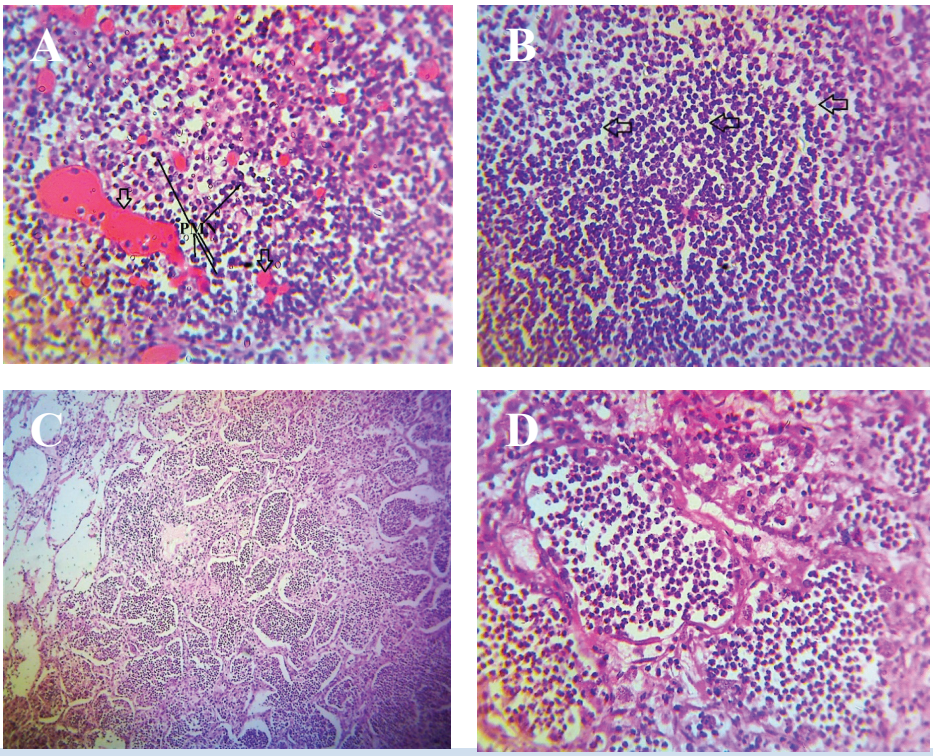

Figure 5: (A). Photomicrograph of lymph node of the affected horse showing haemorrhages (short arrows), inflammatory oedema and infiltrate of polymorphonuclear leukocytes into the lymphoid follicles (PMN). H & E, X400. (B). Lymph node of the affected horse showing mild oedema (short arrows) of non-reactive follicles. H & E, X400. (C). Lung showing severe multifocal to coalescing extensive infiltrate of neutrophils involving the alveolar spaces. H & E, X100. (D). Higher magnification of severe infiltrate of neutrophils in the alveolar spaces accompanied by fibrin deposition from (C). H & E, X400.

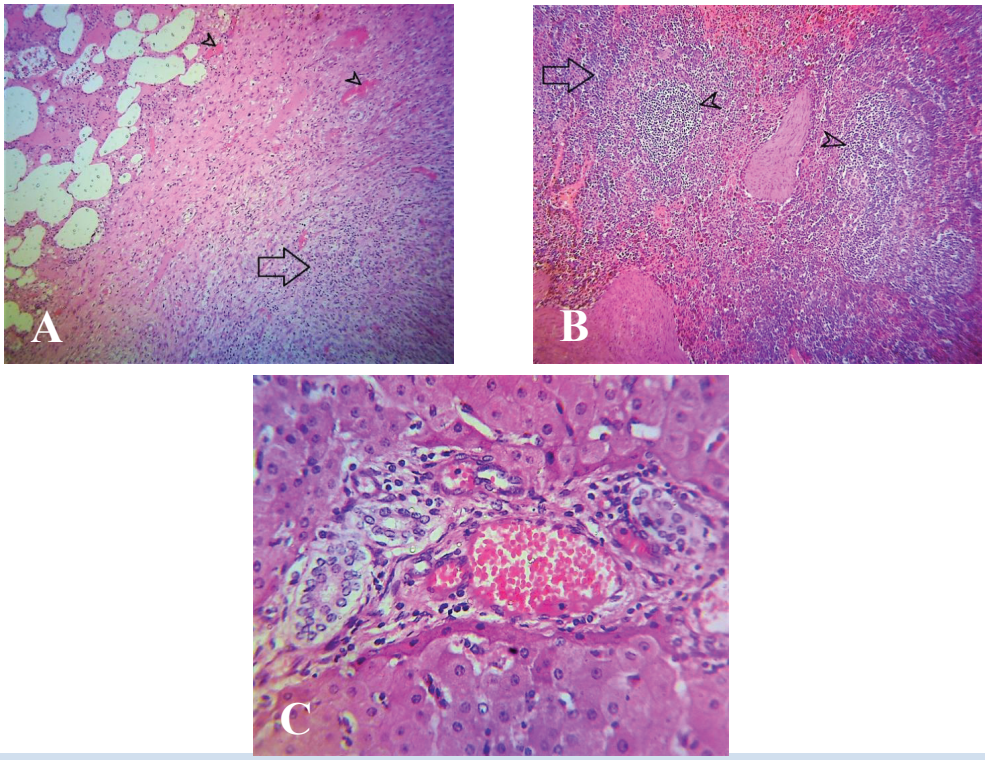

Figure 6: (A). Lung section showing moderate hyperaemia (arrow heads), collapsed alveoli and severe polymorphonuclear cells infiltration of the area (arrow). H & E, X100. (B). Spleen section showing moderately reactive white pulp (arrow heads) and severe infiltration of the red pulp by polymorphonuclear leukocytes (arrow). H & E, X100. (C). Liver section showing moderate necrosis of hepatocytes in the peri-portal area with mild mononuclear leukocytes infiltration. H & E, X400.

DISCUSSION

The case reported here is a presentation of *S. equi* and metastatic *S. equi* ("bastard strangles"). The diagnosis was obtained by the association of the presenting clinical symptoms, the location of the abscesses, macroscopic, microscopic, haematological and bacteriological features. The case is a presentation of *S. equi* in a susceptible two-and-half-year-old stallion with no immunoprophylaxis that showed the common clinical signs and pathological findings associated with strangles and with metastatic *S. equi*. This finding supports previous reports that strangles can occur in horses of any age, but 1-5-year age group is most predisposed, with clinical signs being more severe in the immunologically naive (Sweeney, 1996; Boyle, 2016). A tentative diagnosis of clinical strangles in the present report was made based on the signs of fever, anorexia, depression and catarrhal nasal discharge and the cutaneous swelling of the submaxillary lymph node, which was liquefactive and moderately warm on palpation and suppurative on lancing and the latter preceded by epilation and oozing of serum. In this case report, it is probable that the patient, being young and unvaccinated against strangles, was highly susceptible to the infection. Occasionally, the disease breaks out in stables which have had no recent arrival and no known contact with infected horses. In this situation, the organism has apparently persisted in the tissues of the nasopharynx/guttural pouches of recovered or carrier animals (outwardly healthy horse) and contaminated fomites for long periods (months or years) and is subsequently transmitted to susceptible in-contact animals (Newton et al., 1997; Boyle, 2016). The tachypnoea is evident of the extensive abscessation expressed daily during treatment periods, suggesting that there may have been respiratory obstruction as a result of aspiration of pus into the lungs and distress. Probably, pneumonia was the initial source of the infection, as the respiratory tract is its most common route of entry. Although, these observed clinical signs agree with previous reports by various workers in young horses (Sweeney, 1996; Pusterla et al., 2011; Waller, 2014; 2016; Boyle et al., 2018); unilateral panophthalmitis and persistent tachycardia which are complications of strangles were also observed in the present case study. Panophthalmitis was reported by Barratt-Boyes et al. (1991) and Kaplan and Moore (1996), however, the horses used in the cited case reports were an eight-year-old mixed breed mare that had previously participated in a day-long ride with 150 other horses and a 16-year-old Thoroughbred gelding, respectively and the condition likely resulted from haematogenous spread of bacteria to the eye. Boyle (2016) reported that *S. equi* abscessation can occur at the site of any subcutaneous lymph node and have been seen in unusual locations such as ventral to the eye with *S. equi* positive purulent drainage from the medial canthus. It is unknown whether the unilateral panophthalmitis resulted from haematogenous embolism or extension of bacterial lymphadenitis. However, the unilateral panophthalmitis/corneal lesion in the present case was remarkable and showed mild improvement in response to daily cleaning with antiseptic solution, and topical application of an autogenous antiproteinase. The persistent tachycardia in the present case reflects a malfunctioning of the heart which is present in cases of myocarditis and endocarditis (Ford and Lokai, 1980; Kaplan and Moore, 1996). Young horses (less than 4 years) appear to be predisposed and *S. equi* may produce adherence factors allowing colonisation of previously undamaged vascular endothelium (Dedrick et al., 1988). The prognosis is grave and successful treatment is rare.

The rectal temperature remained predominantly high; in fact, short periods of normothermia coincided with periods after analgesic therapy was given to the patient. Strangles is characterized by pyrexia then the abscessation of the lymph nodes of the head and neck (Waller, 2014; 2016). The disease can occur in horses of any age. *S. equi* was the most common agent identified in horses of 6-10 years age (Pusterla et al., 2011). Younger horses seem to exhibit more severe clinical signs with lymph node abscess formation and rupture, whereas older horses are often less severely affected and recover more rapidly. While most horses display classic clinical signs, not every horse presents the same way (Boyle et al., 2018). Pyrexia with lethargy become typically the first signs occurring 3-14 days after exposure and before most horses are contagious. The pyrexia is persistent and may exceed 42.8ºC (107.68ºF) in some cases. Fever may persist until lymph node abscesses rupture (Waller, 2014; Boyle et al., 2018). The patient's poor body condition (body condition score of 2.25 on a scale of 5) is a marker of both nutritional and management associated stress on the horse. Poor nutrition, strenuous exercise beyond level of training, inclement weather etc. have been identified as stressors that predispose horses to strangles (Bianchi et al., 2018).

A key differential diagnosis of the disease described in this case is glanders and melloidosis

(pseudoglanders), which is caused by *Burkholderia psuedomallei,* presents with visceral abscessations, and almost clinically indistinguishable from glanders. The horse described in this report was a sedentary horse from a closed population with no known exposure to *S. equi*. The source of infection and duration of pulmonary disease is unknown. This type of multiple organ system involvement has been described for end stage bacterial endocarditis and glanders (Kaplan and Moore, 1996; OIE, https://www.oie.int/en/disease/glanders/). Treatment is rarely successful in cases of bacterial endocarditis and panophthalmitis with fulminant clinical progression. However, aspirates of the abscess cultured were positive for *S. equi* and a definitive aetiological diagnosis of the strangles was *S. equi* was confirmed, one of the oldest recognised infectious diseases of horses and continues to cause significant welfare and economic cost throughout the world (Waller, 2016).

The initial haematological picture was that of a macrocytic normochromic anaemia, with mild to moderate leukocytosis dominated by neutrophilia. The findings in this case were consistent with previous reports of *S. equi* and metastatic *S. equi* in experimental and natural infections (Hamlen et al., 1992; Whelchel et al., 2009; Arias, 2013). Complete blood counts are often characterised by leukocytosis with a mature neutrophilia and anaemia and can provide suggestion for *S. equi* testing in an index febrile horse (Ijaz et al., 2011; Duffee et al., 2015). Haemolysis due to the in vivo release of *S. equi* haemolysins remains a possible explanation for the observed haematologic changes seen (Hamlen et al., 1992). Blood chemistry is generally unremarkable (Mallicote, 2015). A mild improvement in the haemogram was seen on second laboratory analysis on day 14 of treatment. This improvement in the haemogram represents only a rather transient positive response to medical therapy before relapse.

Our antibiotic therapy that commenced with procaine penicillin and gentamicin, was administered while awaiting the results of bacterial culture and sensitivity test. The result indicated that the isolated organism was susceptible to penicillin. Various workers have reported widespread high susceptibility of *S. equi* isolates to penicillin (Erol et al., 2012; Boyle et al., 2018). Therefore, antimicrobial failure in the present case seems to be more likely due to the disseminated and severe nature of the presenting disease.

The macroscopic and microscopic lesions were characteristic of strangles and “bastard strangles”. Macroscopically the location of the abscesses, lymphadenopathy with subsequent abscessation and rupture observed in the present case support the possible explanation that successful infection begins with bacterial entry to the oral or nasal passage of the horse and associated lymph nodes with abscess formation. Bacteria access pharyngeal tonsillar tissue. Bacterial cell surface antigens mediate the entry of *S. equi* into tonsillar epithelial cells. Within a few hours of initial colonization, *S. equi* is no longer evident on the epithelial tissue but can be found within subepithelial cells and the lymph nodes responsible for draining the pharyngeal region resulting in lymphadenopathy with surrounding oedema (Timoney and Kumar, 2008). Bacterial (*S. equi*) arrival into the local lymph nodes stimulates an influx of neutrophils, but because of evasion of phagocytosis due to its hyaluronase capsule and elaboration of antiphagocytic enzymes and toxins, these cells are generally unable to prevent bacterial multiplication and colonization of the node. These accumulated neutrophils eventually contribute to the typical lymph node abscesses seen with the disease. Streptolysin S and streptokinase also seem to contribute to cell membrane damage and the ultimate formation of abscesses (Sweeney et al., 2005). Mortality is most often associated with secondary development of pneumonia.

The macroscopic and histopathologic findings also, indicated that suppurative bronchopneumonia was the main lung lesion with multiple organ system involvement of the spleen and liver in the case reported here. Common sites for metastatic strangles include lung, mesentery, liver, spleen, kidneys and brain (Sweeney et al., 2005; Bianchi et al., 2018). The occurrence of this condition seems to be related to animals with an inadequate immune response to the initial infection (Reile and Genetzky, 1983). The main lung lesion indicates the suppurative and metastatic nature of strangles and correlates with the presenting clinical signs of tachypnoea recorded in the present case in the respiratory tract. Clinical signs depend on the organ system involved. As previously mentioned, aspiration of mucopurulent discharge or haematogenous or lymphatic spread to the lungs can cause pneumonia (Boyle, 2016). *S. equi*, the aetiologic agent of strangles, predominantly involves the upper airways and associated lymph nodes with abscess formation but spread to other organs may occur in metastatic abscessation (also known as “bastard strangles”) through blood, lymphatic or aspiration of pus into the

lungs (Timoney, 2000; Sweeney et al., 2005; Duffee et al., 2015; Boyle, 2016), with the development of necrotic suppurative bronchopneumonia. This lesion distribution is consistent with inhalation of aerolised bacteria and has been referred as acentric-acinar, corresponding, macroscopiclly, to well-defined areas of dark red consolidation (Oikawa et al., 1995, Racklyeft and Love, 2000; Bianchi et al., 2018).

In conclusion, pyrexia, chronic intractable exudative suppuration, anaemia, neutrophilia**,** inflammatory oedema and infiltrate of polymorphonuclear leukocytes as were seen in this report are features of strangles in young horses. Also, we hereby report yet a failed attempt on isolated treatment of a clinical case of strangles in a horse even with high doses of penicillin over a course of about twenty eight days.

ETHICAL CONSIDERATION

All interventions on the patient were based on recommended practice by the Faculty Institutional Animal Care and Use Committee (IACUC) of Faculty of Veterinary Medicine, University of Nigeria, Nsukka.

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