Assiut Vet. Med. J. Vol. 47 No. 94. July 2002

Dept. Of Pathology,
Animal Health Research Institute, Dokki, Giza, Egypt.

ABORTION AND STILLBIRTH IN ARABIAN MARE: CLINICAL, ETIOLOGICAL AND PATHOLOGICAL STUDIES IN ARABIAN MARE

(With 2 Tables and 19 Figures)

By
*DAWLAT AMIN; OMRAN, RAWHI; I. ESSAM and M. AHMED***
*: El-Zahra Arabian horse stud, Cairo, Egypt
(Received at 2/6/2002)

دراسة الأسباب الباثولوجية والإكلينيكية للإجهاض ونفوق الجنين في الخيول العربية

دولت مسعد أمين ، روحية عمران ، ابراهيم عصام ، محمد أحمد

أجريت هذه الدراسة في الفترة ما بين ١٩٩٨ - ٢٠٠١ (لمدة أربع سنوات) وذلك للوقوف على أهم الأسباب التي تسبب الإجهاض في الخيول العربية وقد اشتملت الدراسة على جميع البيانات والأعمار الخاصة بالأمهات . في هذه الدراسة وبناءا على مسببات الأجهاض بالخيول فقد تم تقسيمها إلى ثلاث مجموعات: المجموعة الأولى وهى الإجهاض الناتج عن الأصابة البكتيرية أما المجوعة الثانية وهى الإجهاض الناتج عن الإصابة الفيروسية أما المجموعة الثالثة وهى الإجهاض الناتج عن إصابات رضية أو نقص غذائي أو وجود تواءم أو أسباب غير معروفة لدينا. بالنسبة للعزل البكتيرى فقد أعتمد التشخيص على عزل البكتيريا من محتويات المعدة اما بالنسبة الإجهاض الناتج عن الإصابة الفيروسية فقد اعتمد التشخيص على التغيرات الهستوباثولوجية ووجود الأجسام الأحتوائية بالخلايا وقد اتضح من هذه الدراسة ان الإجهاض قد حدث في ٢١ حالة أى ما يعادل (٣٦ر٦%) وأن نسبة ولادة أجنة ميتة أو نفوق المهور حديثة الولادة كانت ٧٣ر٢% وقد وجد أن الإجهاض الناتج عن الإصابة البكتيرية كانت في ١٦ حالة ووجد أن أهم أنواع البكتيريا المعزولة كانت:
Streptococcus zooepidemicus, Klebstella peumonia, Escherichia coli, Rodococcus Equi , Staphylococcus aurius and Pseuelomonus SPP.
هذا وقد أوضحت النتائج الهستوباثولوجية أن الإجهاض الناتج عن الإصابات الفيروسية قد تم في عدد ٦ حالات بالإضافة عدد ٦ حالات نتج فيها الإجهاض عن النقص الغذائي أو الرضوض وعدد حالتين ولادة توأم.

SUMMARY

A study was conducted to investigate some of the etiological aspects of fetal death and stillborn foals in Arabian mares during a four-year period from 1998 till 2001. Data on the age and breeding history of mares were recorded. The causes of abortion were determined according to the clinical, bacteriological and the histopathological evaluations and were grouped into bacterial abortion, viral abortion, twin abortion or miscellaneous (traumatic abortion and unidentified cause). Isolation of bacterial organisms from fetal stomach contents in pure culture or as the predominant organisms was the basis for a diagnosis of bacteria as a cause of abortion. Viral abortion was diagnosed depending on the histopathological changes and the presence of inclusion bodies within certain cells. The twin and the traumatic abortion were determined clinically. Abortion had occurred to 21 mares (6.36 %). Stillbirth and death of newly born foals, which died within few days postparturient, were estimated to be 9 foals (2.73 %). Bacterial abortions were determined in 16 cases. The isolated bacteria were *Streptococcus zooepidemicus, Klebsiella pneumoniae, Escherichia coli, Rodococcus equi and staphylococcus aurius. Pseudomonas spp.* The histopathological results provided evidence for the viral infection in 6 cases. Twin abortion occurred in 2 and other miscellaneous causes of abortion were found in 6.

Key words:*Abortion, Arabian mare clinical pathological studies.*

INTRODUCTION

Abortion continues to be a problem in equine industry. The subject has been well studied, yet diagnosticians are all too often ignorant of the cause. Normal gestation length in Arabian mare is approximately 340 days. Strictly, abortion means the termination of pregnancy before 300 days. Delivery of a dead foal after 300 days is referred to as stillbirth (McKinnon and Voss, 1993).

Most equine abortions are sporadic. Pregnancy losses are highest during the period of embryo, as gestation progress, the risk of abortion decreases (Kobluck *et al.*, 1995). Reports from many areas of the world involving many mares cite an average incidence of abortion ranging from 7 to 9 percent (Laing and Leech, 1975; Merkt and Gunzel, 1979; Chevalier-Clement, 1989). Abortion has been associated with a

reduction in the likelihood of the mare becoming pregnant again (Kobluck *et al.*, 1995).

Although many specific causes of abortion and stillbirth exist, other factors such mare older than 18 years and foal heat breeding have been associated with increased incidence of abortion but without a clear causal relationship (Platt, 1973; Loy, 1988).

The causes of equine abortion can be infectious (bacterial, viral and fungal), non-infectious (twins, hormones, genetic defects, others) or unknown. Most reports categorize about 40% of abortions as of unknown cause (Rooney, 1970; Platt, 1973; Dimock *et al.*, 1974; Roberts, 1986; McKinnon and Voss, 1993).

Common pathogens isolated in cases of bacterial abortion include those commonly responsible for endometritis in nonpregnant mares such as *Salmonella abortus equi*, *Streptococcus zooepidemicus, Escherichia coli, klebsiella spp* and *pseudomonas spp.* Also *staphylococcus aureus, Listeria monocytogenes and Actinobacillus spp.*are not uncommonly isolated in case of bacterial placenititis (Neely *et al.*, 1983; Roberts, 1986; Mahaffey, 1986; Kobluck *et al.*, 1995).

The present work was conducted to investigate some of the etiological aspects of fetal death and stillborn foals based upon evidence from stud records, clinical, bacteriological and pathological studies.

MATERIALS and METHODS

An analysis was made of the record of pregnancy in Arabian mares of Arabian stud farm during the years of 1998- 2001. All aborted fetuses and stillbirth foals that died shortly after birth were used in this investigation. 21 fetus and 9 dead foals were subjected for autopsy. The aborted fetuses and dead foals were examined externally and internally for gross abnormalities, and specimens were collected for microscopic and bacteriologic evaluation.

Histopathological studies:

Tissue samples from lung, liver, spleen, lymph nodes, kidney, brain and placenta were taken for pathological examination and were fixed in 10% neutral Formalin, and embedded in paraffin blocks. Tissue sections were cut at 4 m and stained with haematoxylin and eosin and phloxin tertrazin were used as specific stain for inclusion bodies and examined microscopically (Drury and Wallington 1980).

Bacteriological studies:

Samples from stomach content were taken aseptically from fetuses and foals and were used for bacteriological investigations. Isolation and identification were carried out according to Quinn *et al.* (1994).

Data on the age and breeding of mares were obtained from the general studbook.

Pregnancy diagnosis had been carried out by the use of ultrasound wave.

The causes of abortion were determined according to the clinical, bacteriological and the histopathological evaluations and were grouped into bacterial or viral abortion, twin abortion, and miscellaneous (traumatic or unidentified cause). Isolation of bacterial organisms from fetal stomach contents in pure culture or as the predominant organisms was the basis for a diagnosis of bacteria as a cause of abortion. Viral abortion was diagnosed depending on the histopathological changes and the presence of intracytoplasmic or intranuclear inclusion bodies within certain cells. The twin and the traumatic abortion were determined clinically.

RESULTS and DISCUSSION

The present investigation showed that the total number of confirmed pregnant mares over the four-year period from 1998 till 2001 were estimated to be 330. Out of those pregnant mares 300 were completed their pregnancy tills full term and delivered healthy foals. Abortion had occurred to 21 mares (6.36 %). Stillbirth and death of newly born foals, which died within few days postparturient, were estimated to be 9 foals (2.73 %). These incidences of fetal loss are in consistent with several previous report of Chevalier-Clement (1989) who found that the incidence of pregnancy loss from abortion in mares from 7 to 9 percent. However Ginther (1992) have reported that the overall fetal loss was about 10% of all mares bred or about 12.5% of all mares diagnosed pregnant.

Clinical symptoms, breeding history of the mares, postmortem observations and the isolated bacteria from the stomach contents of the fetuses and dead foals are presented in Tables (1 & 2).

There was no specific clinical sickness before the onset of abortion. Many of the abortion were sudden and without premonitory signs. Mild fever and depression of some mares were recorded. Abdominal pain and colic were observed in two cases. Four foals were delivered premature 20-40 days before expected time of delivery.

The postmortem changes of aborted fetuses and dead foals are demonstrated in Tables 1 and 2. Congestion, edema and peticheal hemorrhage were seen on most of the internal organ and gastrointestinal tract mucosa, in several cases, in addition to the presence of clear or bloody fluids in the thoracic and abdominal cavities.

This study showed that most of the aborted cases were due to bacterial infection (16 cases out of 30) and many had occurred during the last tri-semester of pregnancy. Similar findings were reported by Platt, 1975; Whitweel, 1987 and Giles et al, 1993, who found that the majority of infectious abortions occurred in the second half of pregnancy and the pathogen can also responsible for septicemia in foals as well as endometritis and sterility in mares.

Isolation of bacterial organisms from fetal stomach contents of 16 cases in the present work indicated that the cause of abortion in those cases are bacterial infection of the fetuses. Bacteria that commonly cause abortion, are isolated most frequently with fewer contaminants from fetal stomach content (Platt, 1975).

The bacteriological results (Tables 1 and 2) demonstrated that *Streptococcus zooepidemicus* were isolated from 3 cases, *Escherichia coli* from 2, *Klebsiella pneumoniae* from 3, *Rodococcus equi* from 3 and *Staphylococcus aurius* from 1 and *Pseudomonas spp* from 1, were isolated as single isolates in pure cultures from the fetal stomach. Mixed infection of *Streptococcus zooepidemicus* and *Escherichia coli* were isolated from 2 cases and *Staphylococcus aurius* and *Escherichia coli* from one case. Most of these bacterial isolates are similar, to a great extent, to those bacterial agents, which had been reported by several authors to cause abortion in mares and infectious placentitis (Neely *el al.*, 1983 and Mahaffey, 1986). Similarly previous reports of Mckinnon and Voss, 1993 have provided evidence that the same pathogens, which have been isolated in case of endometritis in non-pregnant mares, were responsible for abortion in pregnant mares. In some cases, abortion may be attributed to transplacental passage of these organisms and subsequent fetal death (Roberts, 1986). The route of fetal infection were discussed by Jones *et al.* (1996) via the hematogenous route during septicemia in mares, or more commonly occur as ascending infections from the lower genital tract.

The microscopical examinations of the various tissues in cases of bacterial abortion revealed various pathological changes among the various organs.

The histopatholgical examination of the lung of several cases demonstrated different types of pneumonia. Pneumonia due to aspiration of amniotic fluid was seen in cases in which *Streptocoous zooepidimicus* as a single isolate or mixed with *Escherichia coli* or *Rodococcus equi* were isolated. Most of the lung alveoli. bronchi and bronchioles were filled with granular eosinophilic material (amniotic fluid) as demonstrated in Fig. (1B). These findings may be attributed to some difficulty occur during birth, where, the fetus may draw excessive amounts of amniotic fluid into its lung and subsequently fail to expel it completely (Bacha & Wood, 1990). This result was documented by Simpson and Buergelt (1981) who described elongated dense bodies in the alveoli of two aborted foals which was considered to be a concentrations of amniotic fluid by light and electron microscopic.

Other cases showed lung gangrene on which enormous numbers of microorganisms (stained blue) were growing into the bronchiole, and lung tissues, which was necrotic and disintegrating *Fig.* (1C and D). These were associated with sloughing of the bronchial epithelia and filling of the bronchial lumen with sloughed necrotic debris and round cells (*Fig.* 1A). Chronic venous congestion was obvious among many cases: where the lung alveoli were filled with eosinophilic fluid which has leaked from the congested capillaries and free of any inflammatory cells. The pulmonary capillaries were seen swollen and tortuous. Numerous numbers of haemosiderin-laden macrophages tend to congregate around respiratory bronchioles. Thrombosis of the pulmonary vasculature were obvious among different cases.These findings are in accordance to those reported by Rooney & Robertson, (1996). *Streptocoous zooepidimicus, and Rodococcus equi* have been reported as the most pathogenic opportunistic bacteria that colonize the respiratory tract of equine and equine fetus (Fitzgerald & Yamini, 1995; Soedermanto *et al.*, 1996; Rooney & Robertson, 1996) and characterized by widespread dissemination in the various organs (Amin *et al.*, 2001). This may explain the invading of these organisms to the gravid uterus via the hematogenous route during septicemia in mares (Jones *et al.*, 1996).

Examination of the pleura and lung from cases in which *klebsiella pneumoniae or Staphylococcus aurius* were isolated as single or mixed with other organisms showed great thickening of the pleura by oedema, fibrinous exudate containing round cells and congested and/or thrombosed pleural vasculature. The interstitial tissue of the lung was also thickened and infiltrated with round cells. The bronchial lumin was

seen filled with sloughed necrotic tissues and inflamatory cells Lobar pneumonia was seen in most of the cases, where the alveoli were filled with inflammatory exudate containing large numbers of polymorph leukocytes, lymphocytes along with red cells and fibrin. Lung gangarene was seen on some cases (*Klebsiella pneumoniae* was the only isolate) where the lung revealed large areas of liquefactive necrosis invaded by mononuclear and polymorphonuclear cells. These results were documented previously by Quinn *et al.* (1994) who noted that *Klebsiella pneumoniae* resposible for pneumonia and suppurative condition in foals. It has been reported by Koterba, (1990) that gram negative enteric bacteria such as *Escherichia coli, Klebsiella or Salmonella* may cause foetal septicemia and foal pneumonia.

The microscopical examination of the different parts of the intestinal tract and the stomach showed variable degrees of congestion, hemorrhage and mono- and polymorphonuclear cells infiltrating the mucosa and submucosal layers especially in cases of *Escherichia coli* and *Streptocoous zooepidimicus* were isolated. These were associated with thrombosis of submucosal lymphatics and blood vessels. Intestinal gangrene was found in 2 cases, the epithelial lining of the ileum were extensively necrosed and associated with aggregation of large numbers of microorganism and numerous polymorphnuclear leukocytes within the submucosal blood and lymphatic vasculatures (Fig. 2).

The histopathological examination of the kidney revealed variable degrees of tubular degeneration and/or necrosis, and even bacterial aggregation were seen within some renal tubules in several cases of bacterial abortion Glomeular oedema, hemorrhages associated with atrophy of the glomerular tuft were also seen. In addition, some cases showed thrombosis of most of the cortical vasculature. Focal peritubular and perivascular fibrosis was seen. Moreover, interstitial haemorrhages were detected on the renal cortex and medulla of some cases as demonstrated in Fig. (3).

The histopathological examination of the liver showed variable degrees of hepatocytes vacuolar degeneration, extrahepatic cholestasis *(Streptocoous zooepidimicus, Staphylococcus aurius* or *Rodococcus equi)*. Thromboses of the central veins accompanied with tissue emboli were also observed. Other cases in which *klebsiella pneumonia* or *Escherichia coli* were isolated showed subcapsular and interstitial hemorrhages and edema, which caused disorganization and disruption of the hepatic parenchyma, associated with vasculitis (*Fig.* 4).

Microscopically examination of different parts of the brain revealed histopathological alteration on the cerebrum of such cases on which *Streptocoous zooepidimicus* or *klebsiella pneumonia* were isolated. In such cases, the submeningial blood vessels were severely dilated and congested. Trigrelysis and neuronophagia were also seen (Fig. 5).

The heart showed subendocardial and interstitial haemorrhages associated with mononuclear cells. Thromboses of the coronaries were also seen among several cases with bacterial abortion.

The lymph nodes in several cases of bacterial abortion showed severe congestion, interstitial hemorrhages, edema, and cortical and interfollicular areas of necrosis. Moreover, fibrin thrombi and similar microthrombi were present in small blood vessels (thrombotic thrombocytopenic purpura) as demonstrated in Fig. (6). These findings agreed with those of Bacha & Wood, (1990), who attributed the lesions of thrombotic thrombocytopenic purpura to the microangiopathic hemolytic anemia on which the thrombi resulted from intravascular activation of blood clotting mechanism. Vascular damage (thrombotic microangiopathy) and fragmentation of red cells caused the hemolytic anemia. Bacterial emboli were seen in the lymph nodes of some cases (Fig. 7).

The histopathological changes, which have been found in the various organs of bacterial abortion group are in consistent with the previous reports of Bacha & Wood, (1990); Jubb *et al.* (1992); Jones *et al.* (1996). In contrast to the report of Rooney & Robertson, (1996) who reports the presence of fetal diarrhea as a common finding in cases of bacterial septicemia, we found fetal diarrhea in very few cases. The authors attributed that to fetal distress, invariably anoxic in origin and present whenever asphyxia occurred before abortion with no specific etiological significance.

In this study placentitis were also seen among several cases in the group of bacterial abortion. The histopathological changes demonstrated as necrosis and mineralization of chorioallantoic villi associated with round cells and neutrophilic infiltration. Erosions were seen in some other cases (Fig. 8) and fibrin thrombi occurred infrequently in maternal venules and small arteries. Placentitis were reported by Platt, 1975; Swerczek, 1986; Whitwell 1987; and Gills *et al.*, 1993 in abortion in mares They attributed the placentitis and fetal death in these cases to the transplacenteal passage of the organism and subsequently ascending infection via the cervix which affect on gas exchange and nutritional

support of the fetus leading to fetal loss. Acute diffuse placentitis may also be caused by bacteria that gain access to the placenta via the hematogenous route (Prickett, 1970).

Viral abortions in the present study were diagnosed in 6 cases depending on the histopathological changes and the presence of intracytoplasmic or intranuclear inclusion bodies within certain cells. On this group, obvious histopathological alterations were seen on the liver, lung, intestine, spleen, mesenteric lymph nodes, placenta and brain.

Concerning the liver, the hepatocytes were found swollen, vacuolated and / or associated with small areas of necrosis with minimal inflammation and few to many large intranuclear eosinophilic inclusion bodies as demonstrated in Fig. (9). In addition to these changes, variable degrees of sinusoidal congestion, hemorrhage and kuppffer cells activation were seen. The presences of the intranuclear inclusions in the hepatocytes were confirmed in 4 cases.

Another case showed eosinophilic intracytoplasmic inclusion bodies (phloxin-tertrazin stain positive) in vacuolated hepatocytes (Fig. 10 A, B) and on the cytoplasm of the epithelial lining of the mucosa of the dudenum (Fig. 11).

The findings of intranuclear inclusions supported by previous reporters (Platt, 1975; Burek *et al.*, 1975 and Weiblen *et al.*, 1994) who noted its presence in lung and liver as pathognomonic to Equine herpesvirus infection. Rooney & Robertson, (1996) reported that the viral abortion of horse (equine herpsvirus-1) is one of the major causes of acute respiratory disease in horses, with unusual sequel occurs in pregnant mares. The Mares infected at any time during a pregnancy may abort during the last 2 months of gestation irrespective of the time of infection (Allen, 2002).

Examination of the lung of such group showed acute bronchitis, thickening of the pleura and interstitial tissues associated with variable degrees of thrombosis of pulmonary blood vessels were also demonstrated (Fig. 12). These findings were parallel with that described by Whitwell (1982) in which equine herpesvirus induce necrotizing bronchiolitis and interstitial pneumonia with intranuclear viral inclusions.

The microscopical examination of different parts of the brain revealed some changes on the cerebrum as submenengial congestion. In some cases, focal areas of malacia occurred in the cerebral cortex. The tissues in these areas were vacuolated, pale and contained shrunken neurons and necrotic glial cells (Fig. 13 A & B). It has been

demonstrated by Rooney *et al.* 1970, Jackson *et al.*, (1977) that equine herpes virus might be associated with neurological pathologic features chacterized by encephalomylopathy and occasionaly severe vasculitis in young foals.

The respiratory disease, neurological disorders and abortion associated with equine herpes virus have been given considerable attention in the old and recent literatures (Burek *et al.*, 1975; Allen, 2002). However Jubb *et al.* (1992) added that Equine herpesvirus-1 responsible for mild to severe upper respiratory disease complicated by secondary bacterial infection frequently with *Streptococcus zooepidemicus.* The extensive histopathological evidence of multiorgans infection in the present work are in agreement with the information recently published by Allen (2002) regarding equine herpesvirus-1.

Marked lymphocytic depletion was seen on the spleen, where the splenic white pulp was seen filled with huge numbers of large eosinophilc histocytes and epithelioid cells (Fig. 14A). On the mesentric lymph nodes, the lymphocytic depletion as seen microscopically was related to the destruction of the lymphocytes within the lymphoid follicles (Fig. 14B). These observation were also supported the equine herpsvirus in this group. It was explained by Tizard (1992) that equine herpes virus-1 may cause immunosuppressive effect and T-cell lymphopenia in foals and as a result, cell mediated responses are depressed in these animals, also the virus cause drop in T-cells and null cells and depression in the responses to T-cell mitogens. Slaucon & Cooper (1990) reported immunosuppressive effect of equine herpesvirus demonstrated by thymus atrophy and lymphoid depletion.

Variable degrees of vasculitis associated with thrombus formation in different sized arteries of kidney, lung, intestine and placenta were seen in many cases of this group of viral abortion. This vasculitis was characterized by fibroblastic and angioblastic proliferation associated with lymphocytic infiltration within the wall of blood vessels. (Fig. 15 A, B).

The intranuclear inclusion bodies, which have been mentioned earlier, could not be detected in the liver of one fetus which demonstrate vasculitis in several organs. Presence of intranuclear inclusion bodies confirms a diagnosis of equine herpesvirus, however its absence will not exclude such a diagnosis.

The occurrence of the variable degrees of vasculitis in the present study, associated with thrombus formation with different sized arteries which were seen on the blood vessels of kidney, lung, intestine and

placenta in such case drew our attention to the possibilities of equine viral arterities infection. Equine viral arteritis is a global infectious disease of horse and is characterized by panvasculitis including edema, hemorrhage and abortion in pregnant mares. Lesions are uncommon in the aborted fetus; if present, they are mild (Del Piero, 2000). The author added that it is difficult to distinguish histologically equine virusherps-1 or equine viral arteritis in cases when severe vasculitis was demonstrated.

The present findings were similar to those obseved by Coignoul & Cheville, 1984 and Cole *et al.*, 1986) in equine arteritis virus infection. The latter attributed the most common vascular lesions observed in different organs to the endothelial damage of blood vessels caused by the direct cytopathic effect of the virus on the endothelium inducing anoxia or thrombosis. The pathogenesis of the disease has been described by Jubb *et al.*, (1992) who reported that the virus is pathogenic to endothelial cells and causes a panvasculitis following initial replication of the virus in macrophages, endothelial cells are invaded beginning 3 days after experimental aerosal infection. As inflammation progresses and neutrophil damage the internal elastic lamina, media cells are invaded arterial necrosis peaks at day 10. They added that antibody play a little roles in the pathogenesis of the disease, in contrast to the immune-complex component of some other viral vacsulitis.

It is our opinion, based on the above information, the vasculitis which were observed in several organs may correspond to the equine viral arteritis infection at least in one cases which inclusion bodies were not detected. Additional immunohistochemistry needed to confirm the diagnosis but unfortunately it could not be done at our laboratory due to the unavailability of the specific monoclonal antibodies. Further work will be done in the near future.

The histopathological examination of the liver and intestine which revealed the presence of eosinophilic intracytoplasmic inclusion bodies in the vacuolated hepatocytes and on the cytoplasm of the epithelial lining of the mucosa of the small intestine one case (Fig. 9 & 10). We could not confirm the diagnosis of such case to an infection with specific virus. On the other hand Weiland (1984) recorded intracytoplasmic incusions by light and electron microscope, in cases of equine infectious anemia. However, the disease has not been recorded in Egypt (Hosny, 2001). The author had tested 675 serum horse serologically in Egypt for the presence of antibody against equine infectious anemia in various breeds, but they were all negative.

Concerning the placenta of viral abortion group, the severity of the lesions varied among different cases, but most of the cases showed focal to diffuse areas of necrosis and mineralization of the chorioallantoic villi. Moreover, vaiable degrees of vasculitis was seen and characterized by endothelial proliferation and thrombosis, degeneration of the media or adventitia or both. Many placental vessels showed fibrinoid necrosis of the muscular layer associated with leukocytic reaction, edema of the adventitia and cuffing by lymphocytes, histocytes, plasma cells and few macrophages (Fig. 16 A, B).
In some cases, a few placental vessels had discrete foci of necrosis in the media with cuffing invading by lymphocytes, plasma cells and histocytes (Fig. 17 A, B).

A few cases showed placental hemorrhages replacing the chorial villi epithelia as seen in Fig. (18). Coignoul and Cheville (1984) found that pathological findings of arteritis and ischemia progressed to placental separation and abortion of pregnant mares demonstrated similar observations.

As far as the noninfectious abortion, the present investigation showed that two mares out of three pregnant with twins were aborted during the four-year period of study (1998-2001). Only one twin was completed the gestation period till full term pregnancy and gave healthy foals. As presented in Table (2), the abortion of the two cases occurred in last trimester with no clinical sign. Both cases showed that the twins were approximately equal in size. Neither autopsy nor histopathology was performed to the aborted twin's fetuses. In contrast to the present investigation, higher percentage of abortions up to 20 to 30% due to twins' pregnancy noted by Rossdale (1987) in Thoroughbreds. It has been reported by Swerczek (1986) and Acland (1987) that development of twins has long been recognized as one of the most important causes of abortion in mares. The authors attributed the abortion in twin's pregnancy and the inability to carry twins successfully to full term due to placental insufficiency caused by a lack of endometrial surface area.

The cause of fetal loss was traumatic in 2 cases. As presented in Table 2, there was only one foal died 24 hours postparturient due to rupture of the bladder and peritonitis as a result of vigorous handling of enema. Another foal has died immediately after delivery as a result of dystochia because of malposture (caudal presentation). Kobluck *et al.*, (1995) reported that the chances of delivering a viable foal after dystochia would be greatly reduced if the fetus was present in caudal presentation with bilateral hipflextion or transverse presentation.

In our work one fetus also was found dead at the 9th month of pregnancy, it was also presented in malposture and umbilical cord torsion which probably was the cause of death.

Retained placentas were seen in 2 cases postparturient in the present study. The probability of retained placenta increases after dystocia, probably as a result of trauma to the uterus or if severe placentitis was present (Roberts, 1986). Although many specific causes of abortion and stillbirth exist, other factors such mare older than 18 years and foal heat breeding have been associated with increased incidence of abortion but without a clear causal relationship (Platt, 1973; Loy, 1988). Colic has been accused to cause abortion of 14% of medically treated pregnant mares. Qusey and Mcgladdery (2000).

Two mares developed colic before aborted their fetuses and the causes of abortion could not be determined in those cases. Bacteriological cultures of their fetal stomach content did not produce any growth. Pathological studies of the various organs of these two mares were performed. There is no indication that the cause of abortion could be infectious in those cases.

The histopathological examination of the lungs of showed marked thickening of the pleura by oedema. This subpleural vasculature appeared severely dilated, congested and most of them were thrombosed. Thickening of the alveolar wall by congestion and edema associated with bronchial epithelial sloughing was obvious.

Marked interstitial hemorrhages were seen in the cortex and medulla of the kidney accompanied with glomerular hemorrhages, edema and / or atrophy. Subcapsular edema and hemorrhages were also seen. Moreover variable degrees of tubular degeneration and necrosis were obvious.

A subcapsular hemorrhage was seen also on the liver of such cases accompanied with thrombosis of the vasculature. Also marked interstitial congestion and hemorrhages associated with round cells aggregation were found (Fig. 19 A).

Bile duct hyperplasia associated with peribile duct fibrosis was also demonstrated among this group (Fig. 19 B). Examination of the stomach, revealed severe hemorrhages on the mucosa associated with thrombosis of the mucosal and submucosal vasculature.

No obvious histopathological changes could be seen on the heart, intestine, spleen, lymph nodes or the brain of those fetuses.

The histopathological changes of the liver, bile ducts, lung and kidneys are correlated to those changes induced by mycotoxins (Jones *et al.*, 1996).

It is worthwhile to mention that mycotoxins, especially zearlanone were detected in the barely of the ration which have been given to the pregnant mares during 1999 at higher level than the permissible limits (personal communications). Such toxins might contribute to the problem by the immunosuppressant effects of prolonged exposure to mycotoxins. The numbers of fetal loss due to abortion and stillborn were increases during 1999 in the stud under investigations. The following year, the percent of fetal loss were decreases after changing the ration. These findings are supported recently by Kuzma (2001), who reported the occurrence of late term abortion in pregnant mares which (mares' reproductive loss syndrome) due to the presence of zearlanone mycotoxin in their ration in Kentucky.

The present investigation showed that most abortion in Arabian mares were due to bacterial infection of the fetus. Postmortem and histopathological examination could diagnose some cases of viral abortion as demonstrated in herpesvirus infection. However, in those cases where these examination do not give clear cut results, viral isolation and serological examination is a must to ensure accurate diagnosis. Our findings demonstrate the need for specific monoclonal antibodies to confirm the diagnosis of the viral- induced equine abortion.

REFERENCES

Acland, H.M. (1987): Abortion in mares: diagnosis and prevention. Contin, educ. Pract. Vet, 9: 318-324

Allen, G.P. (2002): Respiratory infection by equine herpesvirus type 1 and 4. In: equine Respiratory Diseases, Lekeux, P (Ed). Itheca: Internationat Veterinary Information Service, 2002, B0323.0202

Amin, D.M.; Rahmy, N.A. and Seleim, EL-Nagar, M. (2001): Bacteriological and pathological studies on Streptococcus equi infection in Arabian Horses. J Egypt Vet Med ass

Bacha, W.J. and Wood, L.M. (1990): Color Atlas of Veterinary Histopathology. Lea & Febiger.

Burek, J.; Roos, R. and Narayan, O. (1975): Virus-induced abortion. Studies of equine herpesvirus-1 (abortion virus) in hamesters. Lab Invest. 33 (4): 400-406

Chevalier-Clement, F. (1989): Pregnancy loss in the mare. Animal reprod Sci 20: 231

Coignoul, F.L. and Cheville, N.F. (1984): Pathology of maternal genital tract, placenta, and fetus in equine viral arteritis. Vet Pathol 21: 333.

Cole, J.R.; Hall, R.F and Gosser, H.S. (1986): Transmissibility and abortogenic effect of equine viral arteritis inmares. J Am Vet Med Assoc 189: 769.

Del Piero, F. (2000): Equine viral arteritis. Vet Pathol 37: 287-296.

Dimock, W.W., Edwards, R.P. and bruner, D.W. (1974): Infections observed in equine fetuses and foals. Cornell Vet. 37: 89-99.

Drury, R.A. and Wallington, E.A. (1980): Carleton's histological techniques. 5th. Ed. Oxford Univ. press UK.

Fitzgerad, S.D. and Yamini, B. (1995): Rhodococcal abortion and pneumonia in equine fetus. J. Vet. Diagn Invest Jan; 7 (1): 157-158.

Giles, R.; Donahue, J.; Hong, C.; Tuttle, P.; Petrites-Murphy, M.; Poonache, K.; Roberts, A.; Tramontin, R.; Smith, B. and Sweczek, T. (1993): Causes of abortion, stillbirth, and perinatal death in horses: 3,527 cases (1986-1991). JAVMA, v203 (8): 1170-1175.

Ginther, O. J. (1992): Reproductive biology of the mare 2nd Ed. Cross Plaines, Ginther.

Hosny, W.A. (2001): Studies on Equine Infectious Anaemia and Equine Influenza Viruses MS Thesis, Faculty of Vet Medicine, Beni-Suef, Cairo university.

Jackson, T.A.; Osburn, B.I.; Cordy, D.R. and Kendrick, J.W. (1977): Equine herpesvirus 1 infection of horses: studies on the experimentally induced neurologic disease. Am J Vet Res, 38:709-719.

Jones, T.C.; Hunt, R.D. and King, N.W. (1996): Veterinary Pathology Sex Edition Lippincott Williams & Wilkins. Philadelphia. Baltimore. New York. London. Buenos Aires. Hong kong. Sydney. Tokyo.

Jubb, K.V.F.; Kennedy, P.C. and Palmer, N. (1992): Pathology of domestic animals. Fourth Edition, V.3 Academic Press, INC. Harcourt Brace Jovanovich publishers. San Diego New York Boston London Sydney Tokyo Toronto.

Kobluck, C.; Ames, T.; Geor, R. (1995): Abortion PP 1013-1047 cited in: The Horse: Diseases and Clinical Management. V.2. W.B. Saunders company

Koterba, A.M. (1990): Equine Clinical Neonatology. Philadelphia, Lea & Febiger

Kuzma, Cindy, D. (2001): Kentucky veterenarians investigate mysterious foal death syndrome. JAVMA, 218(12): 1870-1872.

Laing, J.A. and Leech, F.B. (1975): The frequency of infertility in thoroughbred mares. J. reprod. Fertil. Suppl. 23: 307.

Loy, R.G. (1988): The arithmetic of postpartum breeding decision in mares. Proc. Soc. Theriogenol: 13.

Mahaffery, L.W. (1986): Abortion in mares. Vet. Rec. 82: 681.

McKinnon, A.O. and Voss, J.L. (1993): Abortion in mares PP 554-562. Cited in Equine reproduction. Lee and Feibniger.

Merkt, H. and Gunzael, A.R. (1979): A survey of early pregnancy losses in West german thoroughbred mares. Equine Vet. J. 11: 256.

Neely, D.P.; Liu K.M. and Hillman, R.B. (1983): Equine Reproduction. Veterinary Learning System co. princeton, N.J.Philadelphia. London.

Platt, H. (1973): Aetiological aspects of abortion in the thoroughbred mares. J. Comp. Path. 83: 199.

Platt, H. (1975): Infection of the horse fetus. J Reprod Fertil suppl 23: 605-610.

Prickett, M.C. (1970): Abortion and placental lesions in the man. J Am Vet Med Assoc 157: 1465-1470.

Qusey, J. and McGladdery, A. (2000): Clinical diagnosis and treatment of problems in the late pregnant mare. In practice, April: 200-207.

Quinn, P.J.; Carter,M.E.; Markey, B.K. and Carter, G.R. (1994): Clinical veterinary microbiology. Mosby-Year Book Europe Limited, Wolfe Pub.

Roberts, S.J. (1986): Veterinary Obestetrics and Genital Diseases. (Theriogenology). Third Edition.

Rooney, J. and Robertson, J. (1996): Equine Pathology, First Edition Iowa State Univ/ Ames.

Rooney, J.R. (1970): Autopsy of the horse. Baltimore, Williams & Wilkins.

Rossdale, P.D. (1987): Twin pregnancy P.532. In Robinson NE: current therapy in equine medicine. Second edition. W.B. Saunders, Philadelphia.

Simpson, C. and Buergelt, C. (1981): Congealed amniotic fluid in the alveoli of lungs of aborted foals. Equine Vet J 13: 109-111.

Slaucon, D. and Cooper, B.J. (1990): Mechanisms of disease- a textbook of comparative general pathology. Second Edition. Williams & Wilkins Baltimore. Hong Kong London. Sydney.

Soedermanto, I.; Pasaribu, F.H. Wibawan, I.W.T. and Lammler, C. (1996): identification and molecular characterizationof group C *Streptococci* isolated from diseased pigs and monkeys. J. Clinic. Microbiol. 34: 2201-2205.

Swerczek, T.W. (1986): Equine fetal diseases in: Morrow DA, ed. Current therapy in thiriogenology. Second edition. Philadelphia W.B. Saunders Co, 699-704.

Tizard, J. (1992): Veterinary Immunology, An Introduction. Fourth Edition. W.B.Company. Harcourt Brace Jovanovich. Inc. Philadelphia, London, Toronto, Montreal, Sydney, Tokyo.

Weiblen, R.; Rabuske, M.; Rebellato, M.; Nobre, V. and Canabarro, T. (1994): Abortion due to equine herpesvirus in southern Brazil. Braz J Med Biol Res 27 (6): 1317-1320.

Weiland, F.M. (1984): Cytoplasmic inclusion in cells infected with the virus of equine infectious anemia. Eur J Cell Biol 33(2): 294-9.

Whitwell, K.E. (1982): An assessment of the criteria used to diagnose virus abortion: a review of 100 cases. J Reprod Fertil 32 (suppl 1); 110

Whitwell, K.E. (1987): Infective placentitis in the mare. In proceedings 5th Int Conf. Equine Infect Dis 5: 172-180.

LEGEND

Fig. 1: Pneumonic lung, **(A)** showing filling of the bronchial lumen with sloughed necrotic tissues, debris and inflammatory round cells. The pulmonary vasculature are thrombosed. The lung alveoli are filled with lymphocyte and macrophages cells. X 12.5x 1.25. **(B)** The bronchial lumen and some lung alveoli showing eosinophilic coagulated porteinious materials (aspirated amniotic fluid). X 25x 0.8. **(C & D)** showing variable degrees of Lung gangrene on which enormous numbers of bacteria (stained blue) are growing in the bronchi and lung alveoli which is necrosed and disintegrating. These are associated with polymorphs and mononuclear cell aggregation and alveolar edema. C) X 25x 1.25, H & E, D) X 25 x 1.

Fig. 2: Intestine showing gangrene on which there is intense necrosis of the mucosa, diffuse infiltration of polymorph and lymphocytes in the mucosa and submucosa associated with bacterial aggregation (arrow) and thrombosis of the mucosal and submucosal vasculature. H & E stain, X 12.5 x 0.8.

Fig. 3: Kidney showing interstitial and cortical hemorrhages, atrophy of renal tubules, glomerular edema associated with atrophy of glomerular tuft. Also there is mononuclear cells aggregated interstitialy. H & E, X 12.5x 1.

Fig. 4: Liver showing interstitial hemorrhages substitute the hepatic parenchyma in (A) associated with emboli in (B). H & E, A) X 25x 1.25, B) X 25x 0.8.

Fig. 5: Cerebrum showing pericellular and perivascular edema, trigrelysis (arrow a) and neuronophagia (arrow b). H & E, X 25x 1.25.

Fig. 6: Lymph nodes showing areas of cortical and interfollicular necrosis associated with thrombotic thrombocytopenic purpura where there are fibrin thrombi in small vessels. H & E, A)X 12.5x0.8. B) X 12.5x0.8.

Fig. 7: Lymph nodes showing severe congestion of the vasculature associated with bacterial emboli (arrow). H & E, X12.5 x 1.25.

Fig. 8: Placenta showing severe necrosis and erosion of the chorial surface of the allantochorion. H & E stain, X 12.5 x 1.25.

Fig. 9: Liver showing eosinophilic intranuclear inclusion body within the hepatocyte. H & E X, 100x 1.25.

Fig. 10: liver showing A) Vacuolation and swelling of the hepatoctes associated with intracytoplasmic eosinophilic inclusion bodies (arrow). H & E X 100, and in B) Red intracytoplasmic inclusion bodies and interstitial hemorrhages. Phloxin Tetrazin, X 50x1.25

Fig. 11: Duodenum showing homogenous eosinophilic intracytoplasmic inclusion bodies. H & E stain, X 100.

Fig. 12: Lung showing thickness of the pleura and interstitium associated with congestion and inflammatory fluid in the alveoli. H & E stain, X 12.5 x0. 8.

Fig. 13: Cerebrum showing A) Vascular congestion and B) Showing areas of malacia. H & E stain, A -X 12.5 x 0.8, B- 25 x0.8.

Fig. 14: A) spleen showing lymphocytic depletion and nearly an empty white pulp of lymphocytes and presence of huge numbers of histocytes and epitheliod cells. Notice the depletion of the margimal lymphatic. B) Lymph node showing lymphocytic destruction inside the follicles. And depletion of the paracortical zone. H & E A) X 25x 1.25 B) X 25x 0.8.

Fig. 15: Kidney showing variable degrees of vasculitis. A) Most of the cortical vasculture are thrombosed and showing severe thickening of the media. B) showing proliferation of mononuclear, fibroblast and angioblast cells proliferation of the wall of the blood vessels. H & E A) X 12.5 x 1, B X 25 x 0.8.

Fig. 16: Placenta showing necrosis and mineralization of the placental villi associated with infiltration of the interstitium of the chorioallantoic arcade. Vasculitis is obvious; the vascular wall showed fibrinoid necrosis of the media and its infiltration with round and polymorphnuclear cells. Notice the presence of perivascular edema and inflammatory reaction. H & E stain, A -X 12.5 x 1, B- 25x 1.25.

Fig. 17: Placenta showing variable degrees of placental vasculitis, on which there is destruction of the arterial wall and infiltrated with plasma cells, lymphocytes, macrophages and polymorphs. Notice the perivascular edema and also inflammatory cells in A. and B. H & E stain, A -X 25x1.25, B- 50x 0.8.

Fig. 18: placenta adjacent to endometrium showing hemorrhage and congestion of the maternal blood vessels. Notice the necrosis and sloughing of chorioallantoic villi. H & E stain, X 12.5 x 0.8.

Fig. 19: Liver showing A) sever congestion of the centeral vein and hepatic sinusoid with pericenteral vein necrosis and round cells infiltration. B) Showing bile duct hyperplasia. H & E, A) X 25x 0.8 B) X 12.5x1.

Table 1: Abortion: Breeding history, clinical signs, autopsy finding and bacteriological investigation.

	Mare				Fetus		
No	Age Year	Clinical signs	Previous pregnancy	Prev abortion	Fetal age month	Postmortem changes	Isolated bacteria (stomach content)
1	6	None	2	none	3	No gross abnormalities	Negative
2	20	Prev. history of resp infect few weeks before abortion	8	2	5	Bloody peritoneal & thoracic fluid, severe congestion of trachea, lung, liver, kidney & intestine.Red to black spot (1cm) on the lung	*S. zooepidemicus* *E. coli*
3	10	None	5	none	5	Peticheal hemorrhage on kidney, spleen, lung, epi & pericardium, intestincand brain	*K.pneumoniae*
4	4	Colic, abdominal pain	6	none	4	No report	*Negative*
5	11	Retained placenta	6	1	5	Congestion of liver,intestine,kidney, lung, heart & brain. Peticheal hemorrhage on the lung.	*E.coli* *S.aureus*
6	19	Dystochia, malposture of the fetus which died inside the uterus	8	none	9	Not done	Not done
7		General weakness, Dystochia, foal in caudal position.	6	None	9.5	Not done	Not done
8	5	None	1	none	10	Severely congested lung, liver and spleen. Calcified nodules (15cm) on the umblicus	*K.pneumoniae*
9	7	Fever (40C) depression 48hrs before abortion	2	none	8	Lobular congestion and hepatization of lung. Enlarged and congested liver and kidney.	*R.equi*

10	16	Subnormal temperature, depression, general weakness and anorexia	10	1	6	No report	*Pseudomons spp.*
11	5	None	1	none	9.5	Severe congestion of lung, heart and liver, petichial hemorrhage on heart &liver, microabsess on lung.	*S. zooepidemicus*
12	8	None	2	none	10.5	Congestion of intestine which is filled with yellowish mucoid fluid	*Negative*
13	13	Colic 24hrs before abortion	4	None	9	Congested placenta, clotted bloody fluid in abdominal and thoracic cavity, congestion of internal organs and brain	Negative
14	17	None	10	2	7..5	Peticheal hemorrhage on lung and heart. Enlarged liver and kidney.	*S. zooepidemicus*
15	8	None	3	None	10	Clear fluid in pleural cavity. Edema of lung. Fine white foci on liver	Negative
16	5	None	1	None	7	No gross abnormalities	Negative
17	11	Prev. history of resp infect few weeks before abort-ion	3	None	9..5	Enlarged and congested heart. Peticheal hemorrhage on lung and spleen	*S. zooepidemicus*
18		None				Severly congested lung, peticheal hemorrhage on pleura. Congenital opening in the diaphram	*K.pneumoniae*
19	12	Twin preg	7	None	9	No report	Not done
20	14	Twin preg	10	1	8.5	No report	Not done
21	4	None	-	none	7	No gross abnormalities	Negative

Table 2: Stillbirth: Breeding history, clinical signs, autopsy finding and bacteriological investigation.

	Mare				Fetus		
No	Age Year	Clinical symptoms	Prev pregnancy	Prev abortion	Foal Age	Postmortem changes	Isolated bacteria (Stomach content)
1	20	30 days delivery before expected time. Refused milking the foal,	7	None	3 days	Severe congestion of lung, heart, spleen and liver, petichial hemorrhage on heart &liver, microabsess on lung. Hole bet R&L atrium	*R.equi*
2	7	Fever, retained placenta	2	None	2 days (30 days premature)	Bloody pericardial fluid, peticheal hemorrhage on heart, congestion and edema of lung., liver and intestine	*S. zooepidemicus E.coli*
3	19	None	10	2	24hrs	Severely congested serosa of the and small large intestine. Flabby heart, clotted blood in the thoracic cavity	*E.coli*
4	12	30 days delivery before expected time	5	None	1hr (30 days premature)	Edema a d congestion of lung, heart and kidney.	Negative
5	8	None	3	None	6hrs (40 days premature	Congestion and edema of lung, peticheal hemorrhage on liver	Negative
6	11	None	6	None	24hrs	Rectal enema cause traumatic rupture of the bladder, peritonitis, congestion of the intestine. Turbid fluid in abdominal cavity.	Negative

7	6	The mare stop milking the foal after 24hrs.	1	None	2 days (20 days premature	Enlarged and congested heart, lung, liver and spleen. Peticheal hemorrhage on lung and spleen	*E.coli*
8	15	Yellow (purulent) vaginal discharge at time of parturition (18days before expected time) infected necrotic placenta	8	2	2 days	Congestion and enlargment of liver and spleen, Congested intestine filled with yellowish watery fluid.	*S. aurius*
9	12	Subnormal temperature, depression, general weakness and anorexia	6	None	Immediately after birth	No report	*R. equi*

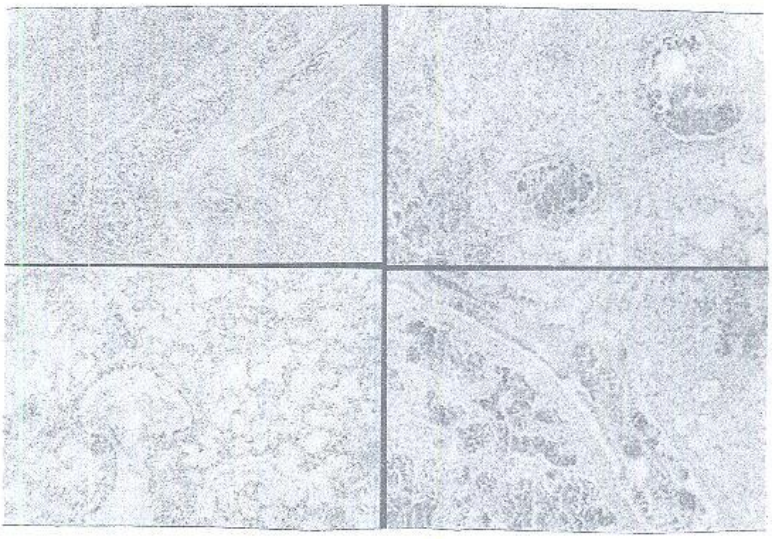

Figure 1: Pneumonic lung.
A: showing filling of the bronchial lumen with sloughed necrotic tissues, debris and inflammatory round cells. The pulmonary vasculature are thrombosed.The lung alveoli are filled with lymphocyte and macrophages cells. X 12.5x 1.25
B: The bronchial lumen and some lung alveoli showing eosinophilic coagulated porteinious materials (aspirated amniotic fluid). X 25x 0.8
C & D: showing variable degrees of Lung gangrene on which enormous numbers of bacteria (stained blue) are growing in the bronchi and lung alveoli which is necrosed and disintegrating. These are associated with polymorphs and mononuclear cell aggregation and alveolar edema. C) X 25x 1.25 , D) X 25 x 1
H & E

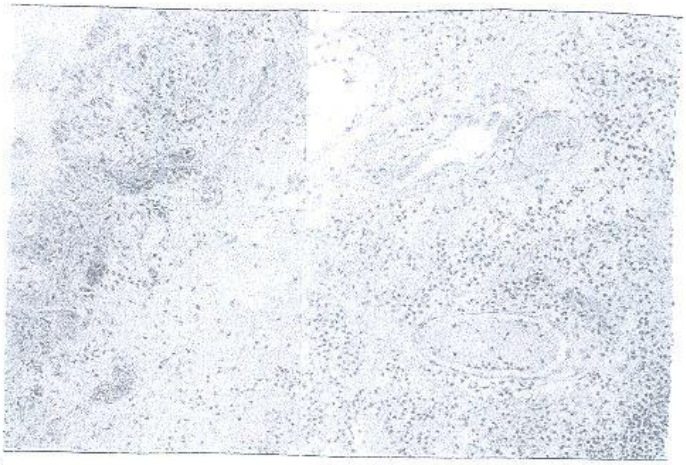

Figure 2: Intestine showing gangrene on which there is intense necrosis of the mucosa, diffuse infiltration of polymorph and lymphocytes in the mucosa and submucosa associated with bacterial aggregation (arrow) and thrombosis of the mucosal and submucosal vasculature. H & E stain. X 12.5 x 0.8

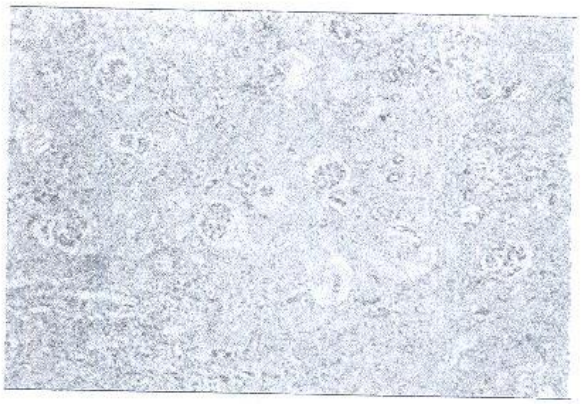

Figure 3: Kidney showing interstitial and cortical hemorrhages , atrophy of renal tubules, glomerular edema associated with atrophy of glomerular tuft. Also there is mononuclear cells aggregated interstitialy. H & E. X 12.5x 1

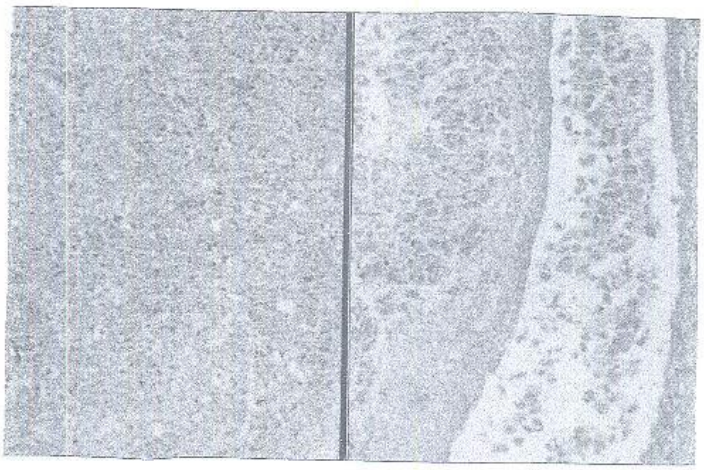

Figure 4: Liver showing interstitial hemorrhages substitute the hepatic parenchyma in (A) associated with emboli in (B). H & E, A) X 25x 1.25, B) X 25x 0.8

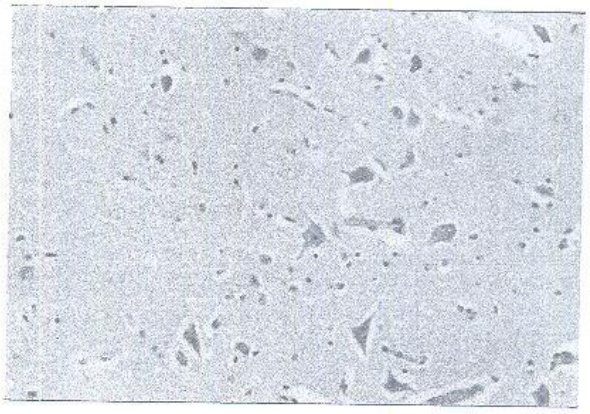

Figure 5: Cerebrum showing pericellular and perivascular edema, trigrelysis (arrow a) and neuronophagia (arrow b). H & E, X 25x 1.25

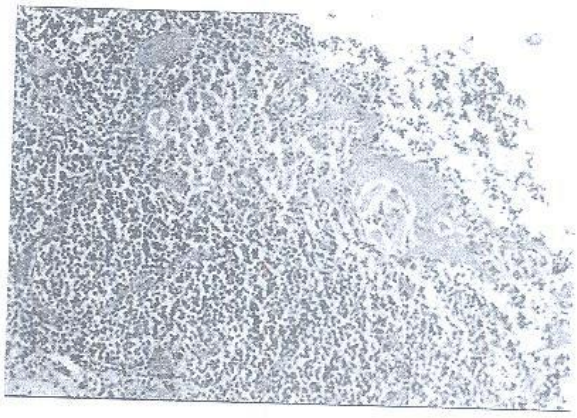

Figure 6: Lymph nodes showing areas of cortical and interfollicular necrosis associated with thrombotic thrombocytopenic purpura where there are fibrin thrombi in small vessels.
H & E, A)X 12.5x0.8 B) X 12.5x0.8

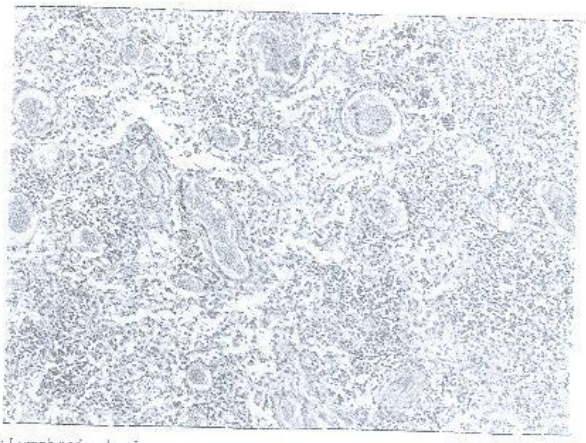

Figure 7: Lymph nodes showing severe congestion of the vasculature associated with bacterial emboli (arrow).
H & E, X12.5 x 1.25

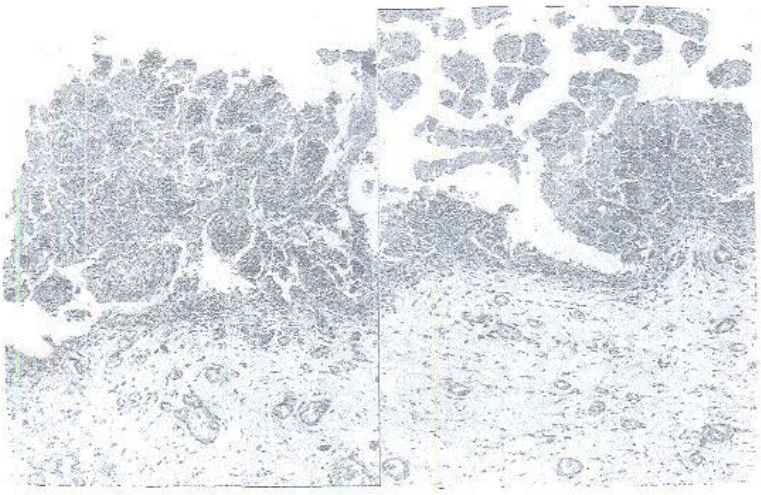

Figure 8: Placenta showing severe necrosis and erosion of the chorial surface of the allantochorion. H & E stain. X 12.5 x 1.25

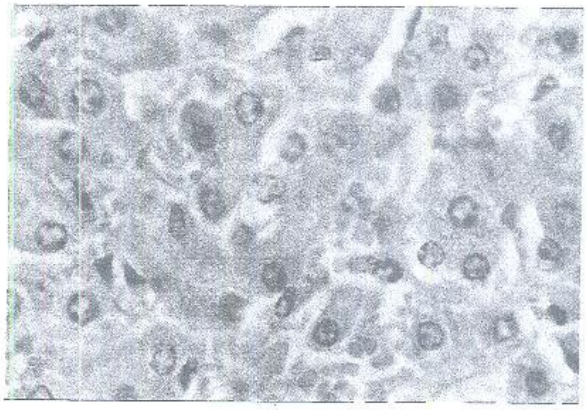

Figure 9: Liver showing eosinophilic intranuclear inclusion body within the hepatocyte. H & E X, 100x 1.25

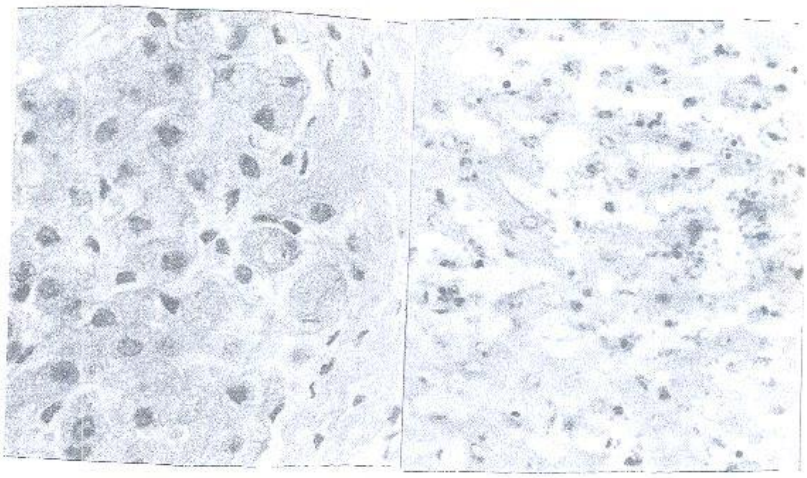

Figure 10: liver showing A) Vacuolation and swelling of the hepatocytes associated with intracytoplasmic eosinophilic inclusion bodies (arrow). H & E X 100, and in B) Red intracytoplasmic inclusion bodies and interstitial hemorrhages. Phloxin Tetrazin, X 50x1.25

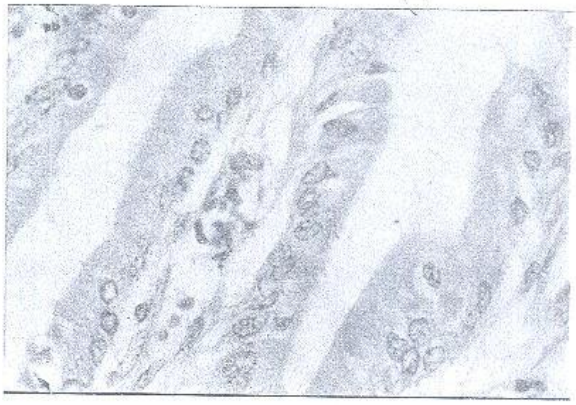

Figure 11: Duodenum showing homogenous eosinophilic intracytoplasmic inclusion bodies. H & E stain, X 100

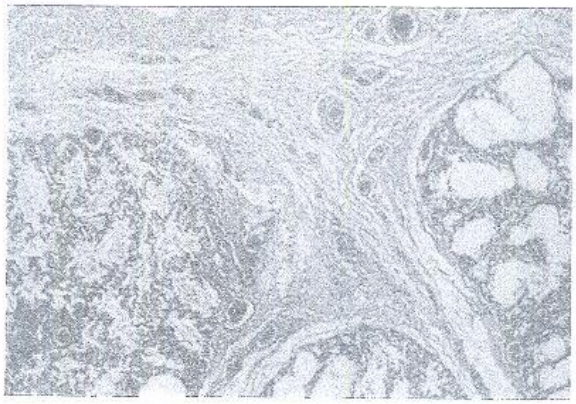

Figure 12: Lung showing thickness of the pleura and interstitium associated with congestion and inflammatory fluid in the alveoli. H & E stain, X 12.5 x0. 8

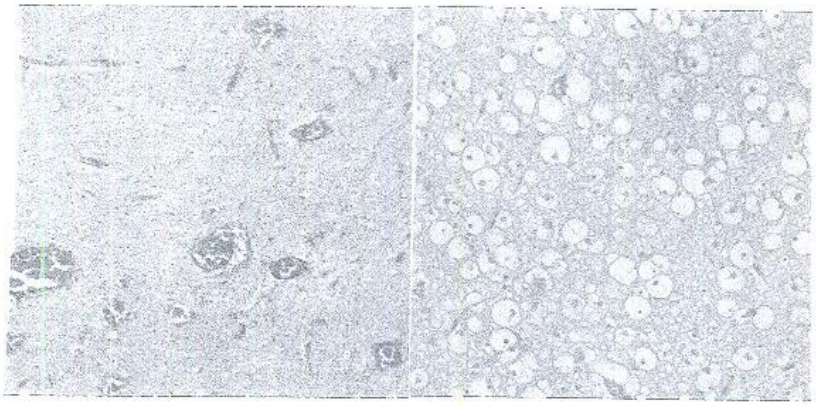

Figure 13: Cerebrum showing A) Vascular congestion and B) Showing areas of malacia . H & E stain, A -X 12.5 x 0.8, B- 25 x0.8

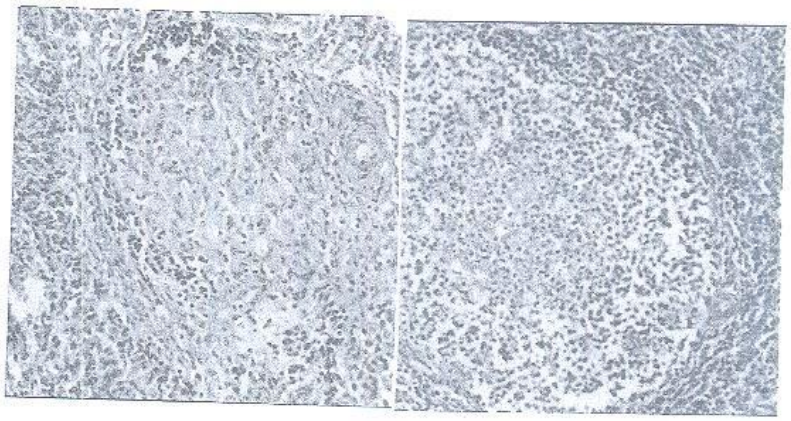

Figure 14: A) spleen showing lymphocytic depletion and nearly an empty white pulp of lymphocytes and presence of huge numbers of histocytes and epithelioid cells.. Notice the depletion of the marginal lymphatic. B) Lymph node showing lymphocytic destruction inside the follicles. And depletion of the paracortical zone. H & E A) X 25x 1.25 B) X 25x 0.8

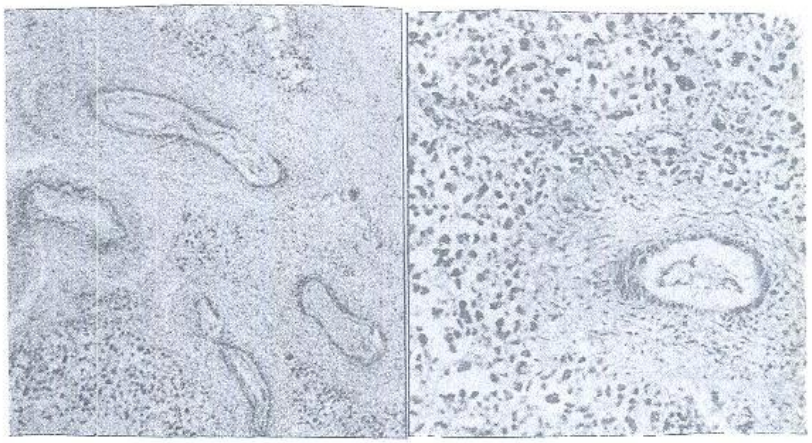

Figure 15: Kidney showing variable degrees of vasculitis. A) Most of the cortical vasculture are thrombosed and showing severe thickening of the media. B) showing proliferation of mononuclear, fibroblast and angioblast cells proliferation of the wall of the blood vessels. H & E A) X 12.5 x 1, B X 25 x 0.8

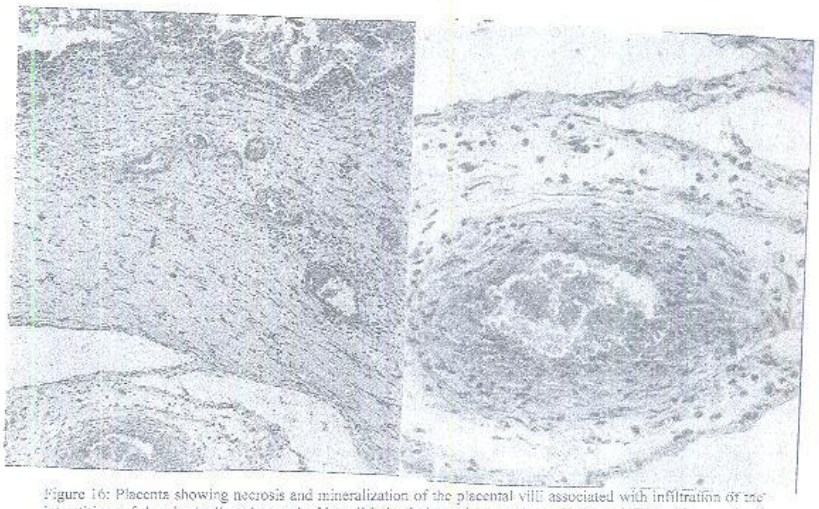

Figure 16: Placenta showing necrosis and mineralization of the placental villi associated with infiltration of the interstitium of the chorioallantoic arcade. Vasculitis is obvious; the vascular wall showed fibrinoid necrosis of the media and its infiltration with round and polymorphnuclear cells. Notice the presence of perivascular edema and inflammatory reaction. H & E stain, A -X 12.5 x 1, B- 25x 1.25

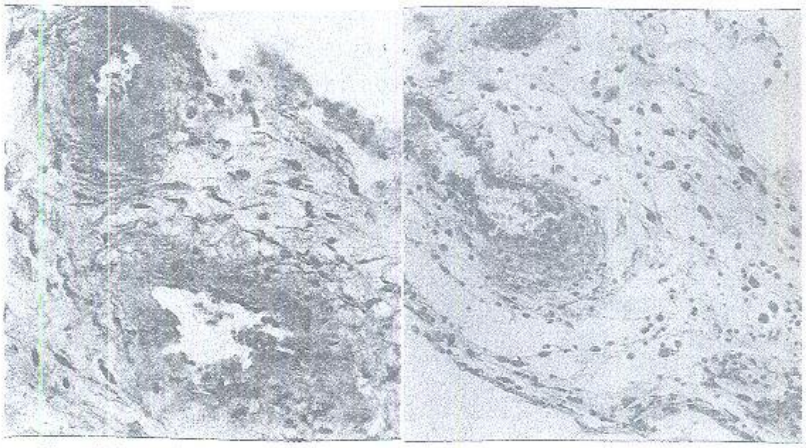

Figure 17: Placenta showing variable degrees of placental vasculitis, on which there is destruction of the arterial wall and infiltrated with plasma cells, lymphocytes, macrophages and polymorphs. Notice the perivascular edema and also inflammatory cells in A. and B H & E stain, A -X 25x1.25, B- 50x 0.8

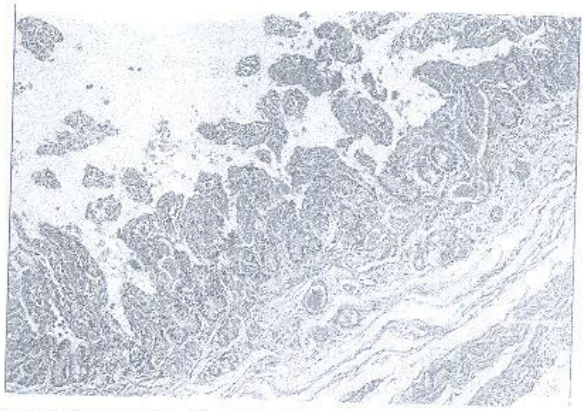

Figure 18: placenta adjacent to endometrium showing hemorrhage and congestion of the maternal blood vessels. Notice the necrosis and sloughing of chorioallantoic villi.
H & E stain, X 12.5 x 0.8

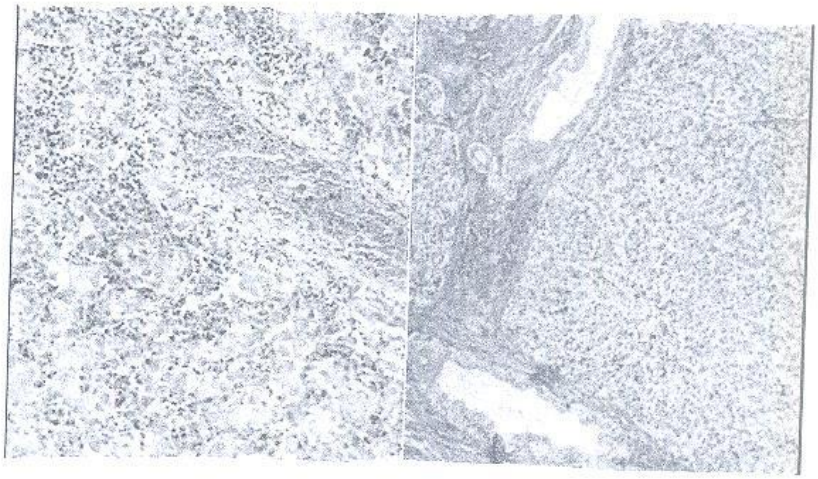

Figure 19: Liver showing A) sever congestion of the centeral vein and hepatic sinusoid with pericenteral vein necrosis and round cells infiltration. B) Showing bile duct hyperplasia.
H & E, A) X 25x 0.8 B) X 12.5x1