



Necropsy findings and histopathological analysis of a terminal stage ewe from a herd with sudden deaths in Mosul

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Abstract

A three-year-old ewe was received as a terminal stage animal for necropsy in the Teaching Veterinary Hospital, College of Veterinary Medicine, the University of Mosul, on 18/12/2018. The animal was carried alive from the Al-Fthilya region in the eastern north of Mosul and expired before hospitalization. Statement of the owner prevailed sudden deaths of healthy animals or a concise course of illness followed by respiratory distress and nervous manifestations and death at 14 mature animals within mortality rate reached 15.5% in the herd. Necropsy findings and histopathological analysis showed that encephalitis included hyperemic cortical blood vessels, severe perineuronal edema, microglial proliferation. The lungs revealed severe pulmonary edema and signs of peracute pneumonia. Liver sections demonstrated congested portal and central veins and lobular sinusoids with centrilobular coagulative degeneration. At the kidneys, we detected both glomerular and interstitial nephritis with severe tubular cell necrosis. We concluded a state of bacterial septic shock, suggesting Pasteurellosis as a probable etiologic factor from the symptoms and pathological examination. The case was reported, and laboratory tests were requested.

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Introduction

Although most recorded causes of sudden death cases in sheep herds are usually related to the enteric form of *Clostridial* infections in enterotoxaemia (1) or mixed with *Fascioliasis* as a black disease (2), other causative agents were reported to induce such unexpected mortalities, including bloat, toxic plants, heavy metals poisoning, and acute salmonellosis. (3). All possibilities mentioned above were previously recorded in our local environment in the Iraqi and Mosul region (4-7). In less frequent occurrence, sheep are also susceptible to other septicemic conditions resulting in sudden or short course illness death like *Hemorrhagic septicemia* caused by *Pasteurella multocida* (8), Anthrax caused by *Bacillus Anthracis*, and Listeriosis by *Listeria monocytogens* (9).

Materials and methods

A three-year-old ewe was received as a terminal stage animal for necropsy in the Teaching Veterinary Hospital, College of Veterinary Medicine, the University of Mosul on 18/12/2018. Necropsy was performed immediately after filling the necropsy submission form by the clinician using a stainless steel set of necropsy with disposable blades, gloves, and gowns. The Primary examination and secondary examination were performed and recorded by photographing. Sampling was selected from brain, lungs, liver, and kidneys and directly preserved in 10% neutral buffered formalin. Later histopathological preparation of tissue sections. (10-13). and light microscopic examination was achieved and photographed by Samsung digital camera.

Results

Case history and clinical signs

The animal was carried alive from the Al-Fthilya region in the eastern north of Mosul and expired before hospitalization. Statement of the owner prevailed sudden deaths of healthy animals or concise course of illness included anorexia, fever, followed by respiratory distress, inhibitory nervous manifestations and death at 14 mature animals from a total 90 animals of the owner within mortality rate reached 15.5% of the herd. Similar mortalities were recorded in other herds in the village.

Necropsy findings

External examination and animal appearance reflected bad hygienic status with skinny cadaver and rough dirty wool, congested mucous membranes. Pinpoint subcutaneous hemorrhages and congested abdominal muscles were noticed. The abdominal cavity contained a tan-colored or sanguineous fluid. Fibrous adhesions between the diaphragm

and anterior abdominal organs with necrotic foci in the spleen. Lungs showed pneumonic appearance with abdomen-caudal gray hepatization, severe pulmonary edema evident by tense pulmonary capsule and separated lobules with frothy white edematous fluid oozing from bronchi at cut section and prominence interlobular septa. Very clear hydropericardium was recognized with a tan color fluid filling the pericardial sac and petechial to ecchymotic hemorrhages on the heart's coronary fat. There were enlarged edematous mesenteric lymph nodes in the abdominal cavity and moderately congested small intestine; the Liver was very dark in color, congested with a solitary necrotic nodule. Kidneys appeared pale with a pinpoint to ecchymotic subcapsular hemorrhages. The brain seemed to be hyperemic with fibrinous exudate at sub arachnoid space, referring to fibrinous meningitis. The cerebral parenchyma was slightly pink with severely hyperemic blood vessels, and whitish necrotic foci were noticed in the gray matter of the cerebral hemisphere (Figures 1 and 2).

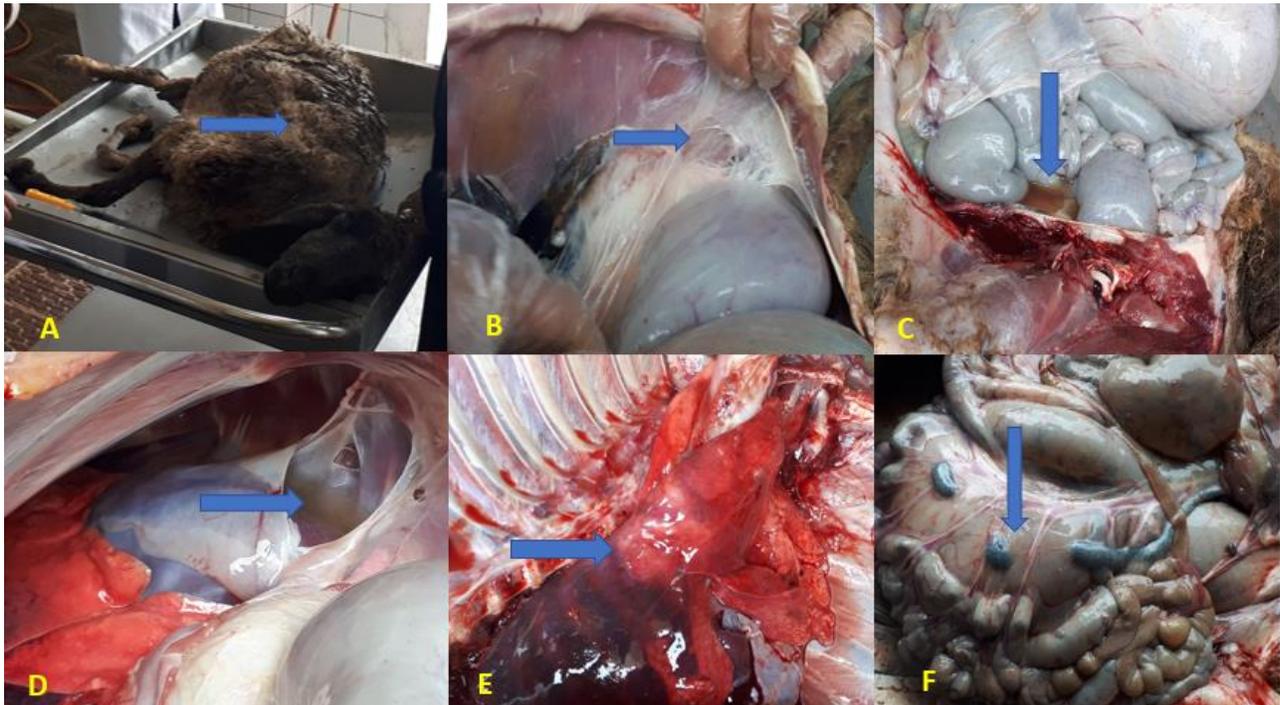


Figure 1: Ewes cadaver reflecting bad condition (A). Fibrous adhesions between the diaphragm and anterior abdominal organs with necrotic foci in the spleen (B). The abdominal cavity contained a tan-colored or sanguineous fluid (C). Tan color fluid fills the pericardial sac (D). Lungs showed pneumonic appearance with abdomen-caudal gray hepatization (E). enlarged edematous mesenteric lymph nodes (F).

Histopathological examination

Microscopic examination of tissue sections revealed noticeable pathological changes in all examined organs. There were severe portal and central veins in the liver, sinusoidal congestion, subcapsular pressure necrosis,

centrilobular and midzonal hepatic cell swelling, and coagulative necrosis. Focal inflammatory infiltrations, perivascular and perisinusoidal hemosiderosis. A solitary hepatic abscess was noticed (Figure 3). The lungs sections prevailed severe pulmonary edema, bronchi filled with

edematous fluid and atelectatic, congested blood vessels, and interstitial perivascular hemorrhages with emphysematous alveoli (Figure 4). There was evidence of acute glomerulonephritis and interstitial nephritis in kidneys manifested by inflammatory infiltrations, degenerative changes to the tubular epithelial cells, and focal inflammatory infiltrations in the medulla surrounding

collecting tubules (Figure 5). Sections from the brain showed evidence of meningoencephalitis with congested blood vessels, a proliferation of microglial cells and neuronal chromatolysis, neurophagy, severe perineuronal edema, and Wallerian degeneration (Figure 6).

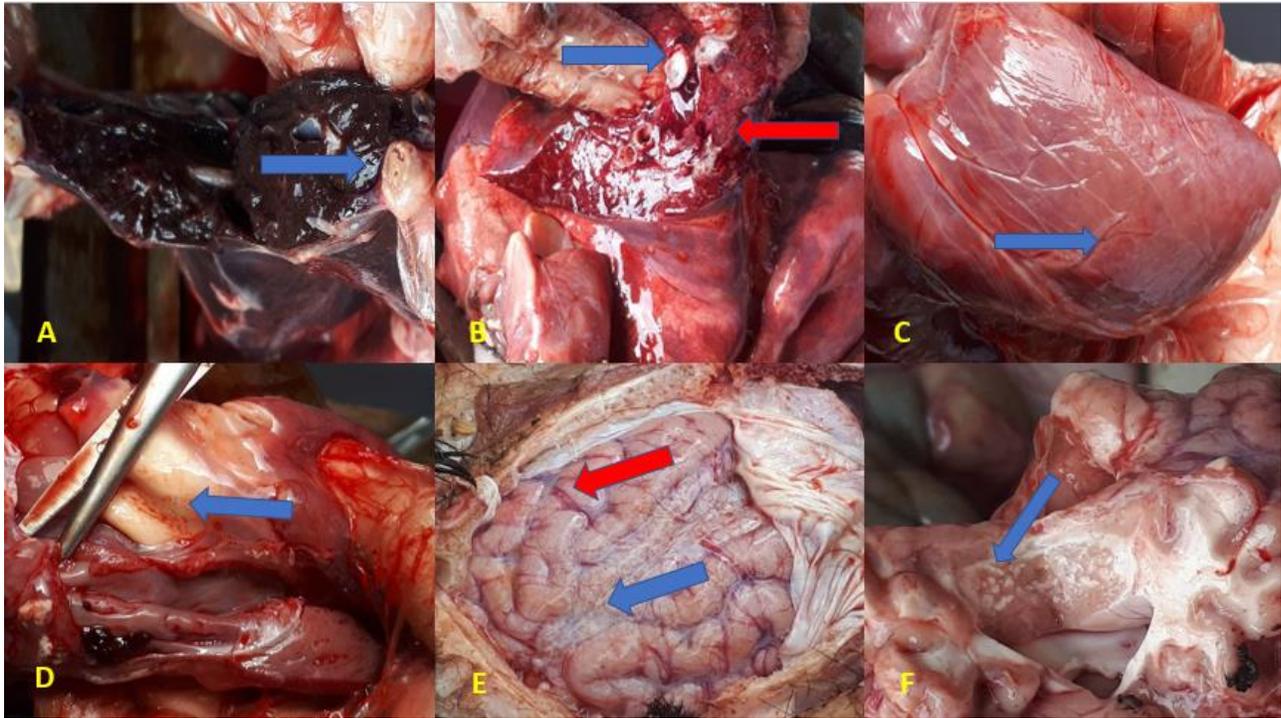


Figure 2: Congested liver with a solitary hepatic abscess (A). Pulmonary edema with frothy fluid from bronchi → and bulged lobes → (B). Petechial hemorrhages in the myocardium (C). Petechial hemorrhages in coronary fat (D). Cerebral hyperemia → and fibrinous meningitis → (E). whitish necrotic foci were noticed in the gray matter of the cerebral hemisphere (F).

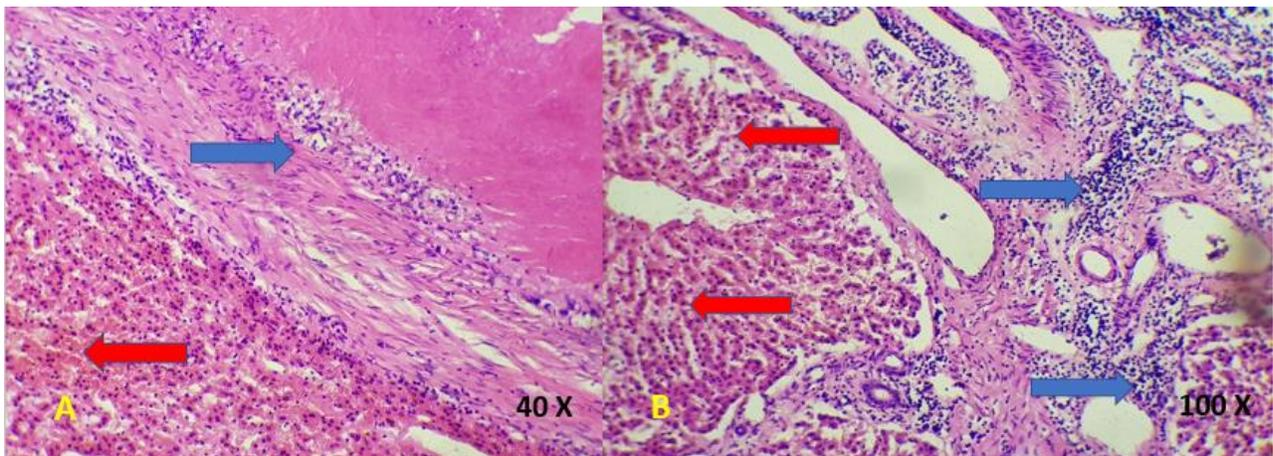


Figure 3: Hepatic abscess with granulomatous reaction → separating the caseous necrotic area from viable hepatic tissue with cloudy swelling → (A). Periportal inflammatory infiltrations → with centrilobular and midzonal cell swelling and coagulative necrosis → (B).

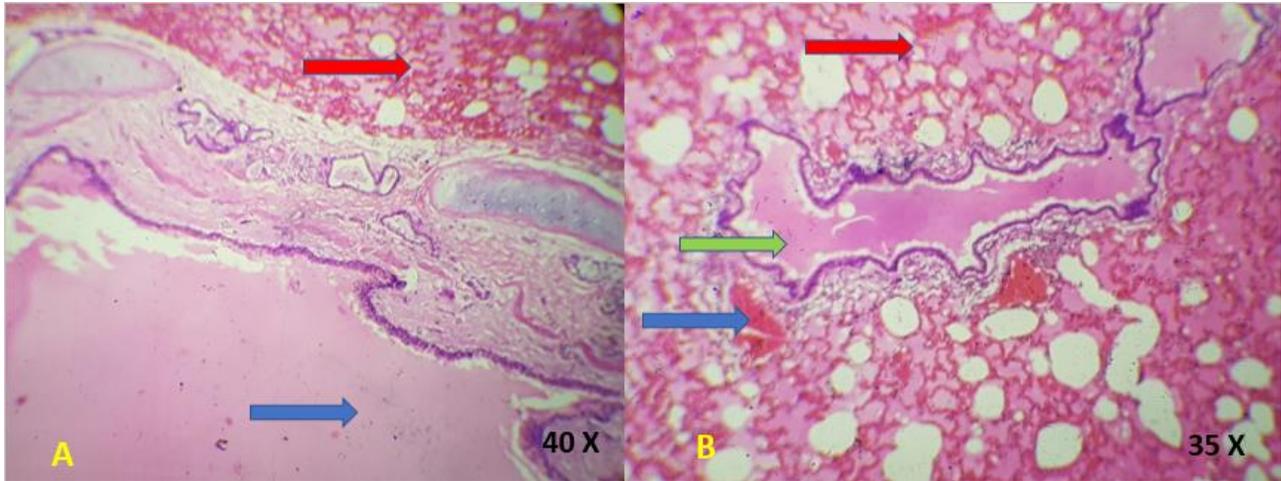


Figure 4: bronchus filled with edematous fluid → and pulmonary edema → (A). Atelectatic bronchus filled with edematous fluid →, congested capillaries →, Pulmonary edema, and emphysematous alveoli → (B).

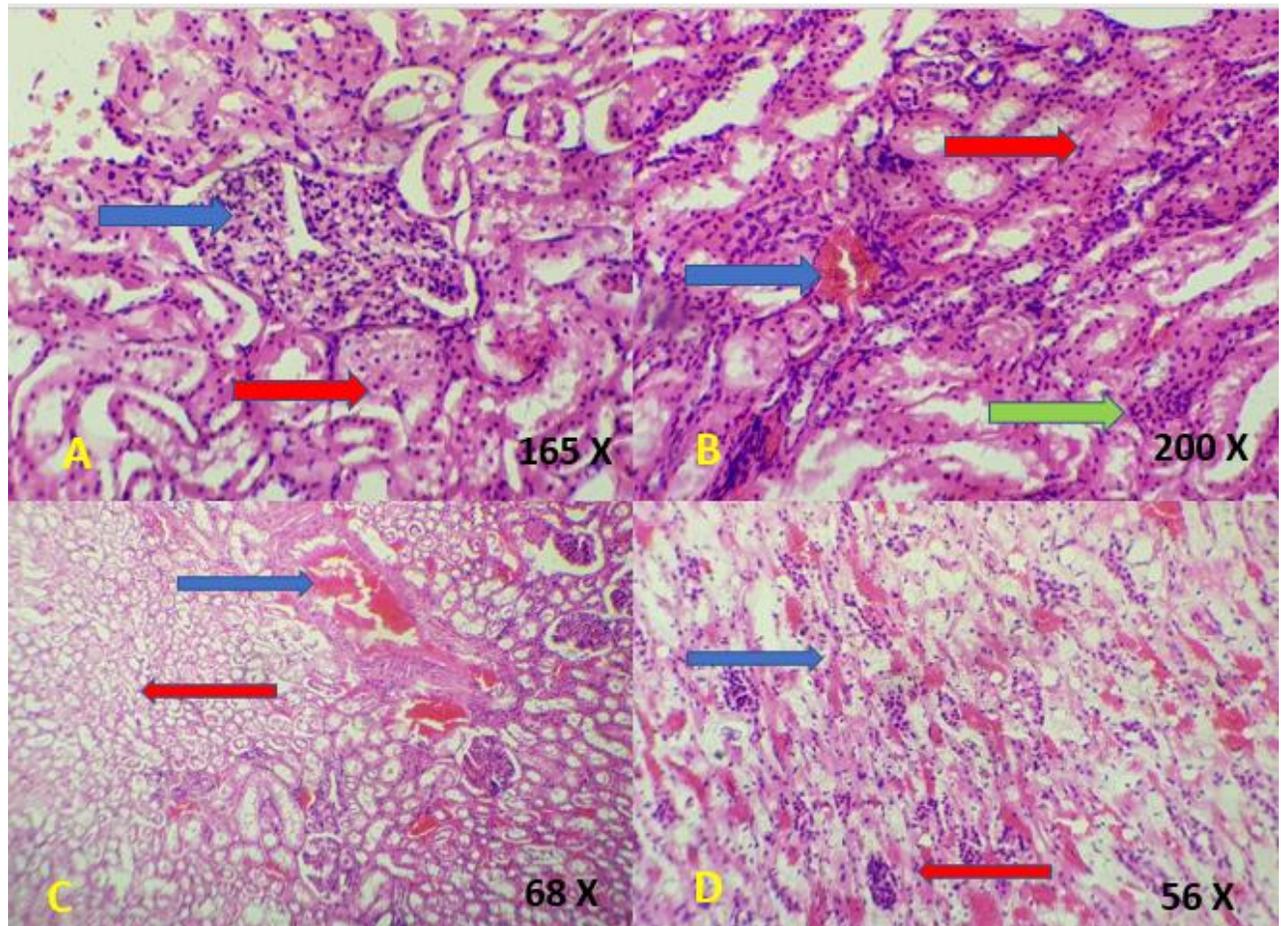


Figure 5: Glomerulus infiltrated with inflammatory cells →, cloudy swelling → (A). Hyperemic blood vessel →, Cloudy swelling →, and inflammatory infiltrations → (B). Hyperemic vessels → and coagulative necrosis → (C). Hyperemic vessels → and focal inflammatory infiltrations in renal medulla → (D).

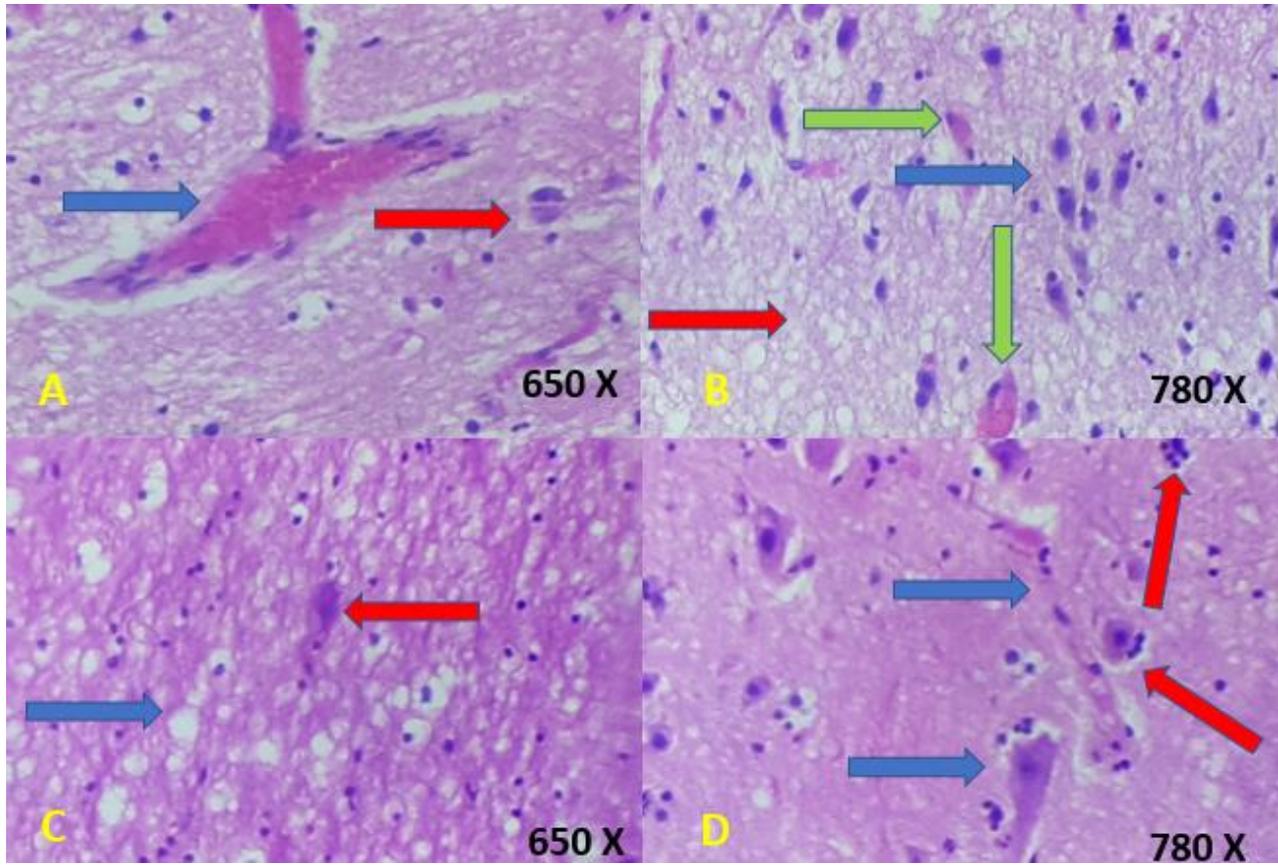


Figure 6: Hyperemic cerebral blood vessel → and neurophagy → (A). Microgliosis→, Wallerian degeneration→ , and degenerative neurons→ (B). Wallerian degeneration→ and degenerating neuron→ (C). Oligodendroglia are surrounding degenerative neurons →and neurophagy→ (D).

Discussion

This condition in early winter, not in spring or harvest season, besides the absence of evidence of severe enteric congestion, may exclude clostridial enterotoxaemia (1), which is considered the most common cause of sudden or short-term illness-deaths in Mosul. The development of respiratory distress in the terminal stage correlated with extensive pulmonary edema, suggesting an etiologic factor affecting endothelial cells and increasing permeability; this hypothesis is aided by noticing a sanguineous serous fluid in body cavities and pericardium besides the petechial hemorrhages in cardiac muscles and coronary fat. Several possible bacterial toxins (14). Viral infections or heavy metals toxicity (3) may perform those effects. Some of these pathogens may induce encephalitis and neuropathy, but the development of fibrinous meningitis may nominate bacterial toxins as the more probable cause as referred by (15) that *Mannheimia hemolytica* infection in sheep and goat was reported to occur as outbreaks and secondary to Parainfluenza type 3, adenovirus type 6, respiratory syncytial virus, also *Mycoplasma ovipneumoniae* infections

(15). Other than the mortal etiologic factor, there was an obvious clue for chronic infections that the animal was suffering from; those include the abdominal and thoracic fibrous adhesions usually accompanies infection with *Mycoplasma spp* besides meningoleukoencephalitis with secondary demyelination (16) and the hepatic abscess that may result from Pyogenic microorganisms such as *Fusobacterium necrophorum*, *Corynebacterium pyogenes*, and *Staphylococcus aureus* (17). these infections mostly thrilled the immune resistance and predisposed the mortal etiologic factor-like *Mannheimia hemolytica* to attach alveolar cells, proliferate, and secrete endotoxin, leukotoxin, and capsular polysaccharide resulting in fibrin deposition in lungs and pleural cavity. The lipopolysaccharide endotoxin contributes to adverse reactions in the lungs and leads to systemic circulatory failure and shock (15). The occurrence of such pathogenic bacteria may depend on activation of the virulence factors through gene expression as mentioned by (18), who referred that specific genes encode to virulence factors can be detected from the highly pathogenic strains of *pseudomonas aeruginosa*, besides these virulence factors usually changed in type and severity

through the passage of bacteria through different hosts as stated by (19) who Found that virulence factors of *Pseudomonas aeruginosa* vary between animal species and even between other organs as infection site from the same individual.

Conclusion

We concluded that Multi pathogenic factors might be involved in the induction of those mortalities, including biological, environmental, and hygienic aspects. The clinical cause of death is septic shock, and the pathological analysis suggested respiratory distress and cardiac failure as a second probable cause of death.

Acknowledgment

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Conflict of interest

We declare that no conflict of interest present with any other published papers.

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نتائج الصفة التشريحية والتحليل المرضي النسيجي لنعجة نافقة من قطع مظهر للنفوق المفاجئ في الموصل

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الخلاصة

استلمت نعجة بعمر ثلاث سنوات كحالة نفوق في المستشفى التعليمي البيطري التابع لكلية الطب البيطري، جامعة الموصل بتاريخ ١٨ / ١٢ / ٢٠١٨ حيث نقلت حية من منطقة الفاضلية شمال شرق الموصل ونفقت قبل وصولها المستشفى وأفاد صاحبها بأن قطيعه يتعرض لهلاكات مفاجئة بعد فترة مرض قصيرة تنتهي بفشل تنفسي واختلاجات عصبية يعقبها الموت، وأنه فقد هذه النعجة وأربعة عشر حيوانا بالغا قبلها بهذه الأعراض بنسبة هلاكات وصلت إلى ١٥,٥% في القطيع، أجري التشريح المرضي للحالة مع توثيق التغيرات المرضية العيانية كما اجري التحليل المرضي النسيجي لأعضاء شملت الدماغ والرئتين والكبد والكلية لتقييم الحالة بالفحص المرضي المجهرى. لقد وضح من نتائج التشريح المرضي والفحص المجهرى للشراخ وجود التهاب الدماغ الحاد تميز بفطر دم شرايين القشرة الدماغية والوذمة الشديدة حول العصبات مع تكاثر الدبقيات الصغيرة، في حين شخص التهاب الرئة فوق الحاد تميز باحتقان الأوعية الشديدة والوذمة الرئوية الشديدة، وأظهرت شرائح الكبد احتقان الأوردة البابية والمركزية مع الحبيبات ووجود النخر التجلطي للخلايا الكبدية في نطاق مركز الفصيص. في الكلى لوحظ التهاب الكلى الكبيبي والخلالي الحاد سوية في القشرة الكلوية مع نخر النبيبات الكلوية الشديدة. من خلال الأعراض والتغيرات المرضية تم استنتاج أن الموت حدث بسبب صدمة إنتانية جرثومية مقترحة جراثيم الباستوريليا كالمسبب الأكثر احتمالاً. تم تسجيل الحالة وطلبت الفحوصات المخبرية التكميلية.