

Histopathophysiological of naturally – occurring exudative epidermitis in wildlife swines

A.A.Hassan

Coll.of Sci./ Univ.of Al-Muthna

M.Abdul-ameer

Coll.of Vete.Med./ Univ.of AL-Qadisiya

M.A.Hasson

Abstract

This study showed that XE are an acute,generalized dermatitis, characterized by sudden onset at the 2 months of age mostly.The more obvious clinical findings concentrate on the level of histopathological changes.So,there are apathy,dandrufflike condition, thickening of the skin, vesicles are found. Histopathologically,the microscopical investigations shows formation of clusters of itchy bullae, edematous papillary dermis, eosinophilic abscesses. Microbiologically, the labs investigation confirmed that the staphylococcus sp.,Mostly *S. hyicus* which abraded feet and leg mostly. In acute cases a vesicular – type virus may be the predisposing factor. So, this study confirms that the XE is a common disease in pigs and lead to the death of half of percentage of swine invaded with the agent of this disease.

Introduction

Wildlife Iraquian brown pigs (*Suis sp.*)are referred to the order *Artiodactyla* from the Infraclass *Gutheria* that is returned to class *Mammalia* which is part at the subphylum *Vertebrata* that is lying at the animal kingdom(8).Those animals have four toes each provided with a hoof, many with hornes. Most of swines living within humidified environments, like marshes, swamps, and near water lakes and product farms (9). Clinicophysiological and histological studies are important manners due to its high quality, of changes carried out by serious diseases(11).So, the application of these methods is useful in detection,diagnosis,and treatment of most diseases and syndromes in animals (6).Exudative epidermitis (XE) is one of

the serious naturally-occurring diseases that is affecting young pigs, commonly referred to as greasy pig disease. This syndrome was originally described by Sompolinsky(16),who found the microbe to be a gram positive coccus.Organism was eventually named *Staphylococcus hyicus* by Devriese(5).This disease is correlated mostly with the moistured environment that swine live in it(17).Siegmond *etal.*,(14)mentioned that the greasy texture of (XE) was coming from a phenomena like a thing contaminated with fat or lipid which seems to being moistened.The aim of this report was to describe the clinical and pathological changes that are associated with (XE) naturally occurring in wild pigs in Iraq.

Materials and methods

Blood sample:- fresh blood was collected in EDTA tubes;1gm/ml; and analysed directly by using autoanalyser (Ms-9).Conformational tests done by using Rowmanky stain for eosinophils.

Bacteriological test:-API staph strip gallery was used for identification of staphylococcus sp. responsible of this case. The method of Roxanna *etal.*, (13) was used.

Tissue sample:- pieces of skin from injured region processed routinely for histopathology and sections were stained with hematoxylin and eosin stain.About 21 samples were gained have a relation with this case at mesopotomia& southern of Iraq.Work at this project continued from September, 2001 and completed at March, 2004.



Fig.(1):Shows a leg of 45-day old pig injured with exudative epidermitis.

Results

Bacteriological examinations showed that the main causative agent for, the XE was *Staphylococcus hyicus*. Other types of agents will be as a side infection. The blood examination tests showed that the white blood cell counts increased to more than $22 \times 10^3/\text{mm}^3$, and the blood smear showing an increase in the eosinophils to being $3.5 \times 10^3/\text{mm}^3$, with mild increase in both monocytes and neutrophils to more than $2 \times 10^3/\text{mm}^3$, and $10 \times 10^3/\text{mm}^3$, respectively. Early clinical signs of the disease include listlessness, apathy, and dullness of skin and hair coat, followed by a dandruff-like condition. Later the pig become more depressed and refuses to eat. The body temperature was normal. Reddish-brown spots appear from which serum exudes at the skin. Catarrhal inflammation of the eyes happened. Bursts and Vesicles develop on the skin. Moist, greasy exudate of sebum and serum covered the body later, and became crusty and with noxious odor. Erosion of the feet occurred (fig.-1). In some animals the disease may be milder with lesions developing slowly. The mortality usually is low, but many affected pigs recover slowly and growth was retarded. Microscopical examination showed two obvious conditions the first (fig.-2) showed a mass

of the causative agents in the tissues. Some surrounding tissues were invariably killed (necrosis). An abscess thus forms and containing polymorphonuclears, necrotic tissue elements and serous fluid. This is early stage in formation of a pustule. Large numbers of polymorphonuclear leukocytes have migrated from the small blood vessels in the dermis (bottom) into the stratified squamous epithelium of the skin, that are starting to form a collection within the epithelium (above centre). The second form of the condition (fig.-3) was characterized by the formation of clusters of vesicles (bullae) or abscesses containing leukocytes, degenerate polymorphonuclear cells, and eosinophilic debris (thin arrow). So, many eosinophilic leukocytes seem to shed their granules. Similar eosinophilic fibrillary material is present deeper in the dermis (thick arrow). The dermis was edematous and infiltrated with polymorphonuclear cells and macrophages. With further increase in size of these microabscesses, the inter papillary ridges of the epidermis become detached and a subepidermal blisters form. This case resembling those exemplifying scalded skin syndromes and dermatitis herpetiformis in humans caused by *Staphylococcus aureus*

Discussion

Microscopical results confirms a status of necrosis which is arising from inflammatory agent (lysin enzymes and toxins of bacteria) and enzymes released by polymorphs in a manner to decrease the

distribution of causative agent and this is may be considered as a mucocutaneous reaction happened during postexponential phase of bacterial growth. a pustule forms as a result of aggregation and reaction

results from polymorphs leukocytes which were releasing antibody substances which interact with residues of destroyed tissues as a result of staphylococci. Skin thickening arise from oedema and tumor which are physiological cases considered as a side effect from the immunological reaction between body defence mechanism and bacteria with its products.(1,2,10). There are many agents assist and contributes in causing XE disease in animals around the world. One of the famous agents are the *Staphylococcus aureus*, but XE mostly known to arise from Infection of *Staph. hyicus* or *chromogenes*; which is product from the adventure from weakness occurred at the injured skin. Physiologists thinks that environmental condition help in the distribution of *Staphylococcus hyicus* than other types of microbes. (4,5,18). At the physiological side, the increase in WBCs counts especially eosinophils & neutrophils being a normal condition. This case was correlated with the immunological status of the body. So, when a foreign body enter the animal body, it stimulate body defence mechanism to increase their activities for countering this microbe. So, one of those mechanisms was increase in the number of WBCs, to control fully those enemy. Second, some types of leukocytes, like eosinophils and neutrophils increased in their production

due to its biological constructor which is composed from specific granules that were assist in the neutritization the virulence of bacterial number and products material. This is may be a type of hypersensitivity correlated with eosinophilia, while neutrophilia come in from inflammation with bacteria. (6,7,12). Some first signs of syndrome were related to the dosage of virulence that secreted by the bacterial agent. Those poisons had a lytic, chemical effects shutdown and breakdown skin and its derivatives slowly as a secondary effect of bacterial attack. Other side symptoms results from nervous excitation due to occuracy a painfull, sickness, and disorders like feelings, this will stimulate secretion of some hormones as respond to that excitation to face all those feelings. Most important hormones at this stage was epinephrine and norepinephrine those secreted from adrenal medulla to combat those condition.(15). Some bursts, vesicles, being as a normal physical state arising from exudate serum and fluids out of the blood and lymph vessels as result of damage and nercrosis. (2). Low percentage of mortality was related to the life of (nature) lives of those pigs whom being wildlife. That their immune system was adapted to symbiosis with many parasites along of the life cycle. This is considered as acquired immunity. (3).

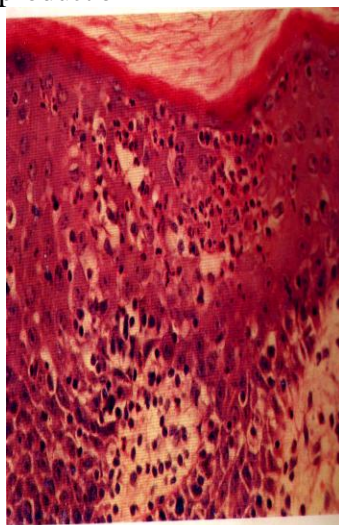


Fig.(2):Shows masses of causative agent in the tissues. Necrosis,pustule seen.Dermis at the bottom showing large numbers of polymorph onuclear leukocytes which migrate and collect at the epithelium(above centre) of the stratified sequamous epithelium.(100X)hematoxylin-eosin

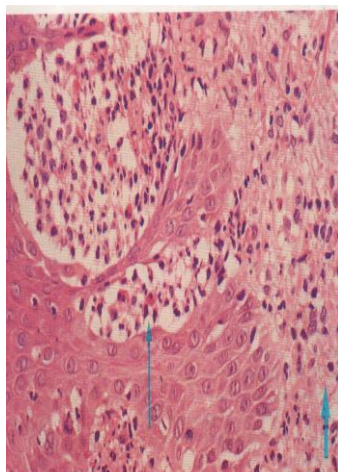


Fig.(3):Shows a case from bullae of eosinophilic leukocytes(thin arrow), and a case of eosinophilic fibrillary material at the dermis (thick arrow).(100X) hematoxylin-eosin stain.

References

- 1.Andersen.L.O.,P. Ahrens,L. Daugaard, and V.Bille-Hansen. (2005).XE in pigs caused by toxigenic *S. chromogenes*. *Veter. Microb.* 105(34):291-300 .
- 2.Bailey, C.J.,B.P.Lockhart, M.B.Redpath, and T.P.Smith. (1995). The epidermolytic (exfoliative) toxins of *S.aureus*. *Med. Microbiol. Immunol.*184: 53-67.
- 3.Breen,C.,M.Golightly. (1998). Immunology. In: Lehmann, C.A. "Saunders's Manual of Clinical Laboratory Science" (1stedn). Philadelphia, W.B. Saunders Co.
- 4.Brun, Y., J. Fleurette, and F. Fey. (1978). Micromethods for biochemical identification of coagulase - negative staphylococci. *J.Clin. Microbiol.* 8:503-508.
- 5.Devriese, L.A. (1977). Isolation and identification of *staphylococcus hyicus*. *Am. J. Vet. Res* 38: 787-792.
- 6.Finnegan, K.(1998). Hematopiosis. In: Lehmann, C.A. "Saunders's Manual of Clinical Laboratory Science" (1stedn). Philadelphia, W.B. Saunders Co.
- 7.Hoffbrand, A.V., and J.E. Pettit. (1984). *Essential Heamatology*. (2nd edn). Singapora, Blackwell Scientific Public- ations.
- 8.Johnson, W. H., C. E. Delaney, L. E. Williams, E.A. Cole. (1969). *Principles of Zoology*. (1stedn). New York. Holt, Rinehart and Winston, Inc.
- 9.Jurd , R.D.(1995).Instant notes on animal biology. (1stedn). Bios. Inter. Publishers. Philadelphia.
10. Ladhani, S.,C,L.Joannou, D.P. Lochrie, R.W.Evans and S.M. Poston. (1999). Exudative epidermitis syndrome. *Amer. Soc. Microbe.* Vol: 12 (2), p: 224-242.
11. Lehmann, C.A. (1998). *Saunders manual of clinical laboratory science*. (1 st end.). W. B. Saunders Company. Philadelphia.
12. Reitano, M. and, R. Malowitz (1998). *Quality control in the clinical microbiology laboratory* C.A. Lehmann. "Saunders's Manual of Clinical Laboratory Science" (1st edn). Philadelphia, W.B. Saunders Co.
13. Roxanna, I. Maddux, and G. Koehne (1982). Identification of *Staphylococcus hyicus* with the API Staph Strip. *J. Clin. Microbial.* Vol: 15 (6), p: 984-986.
14. Siegmund, O. H., C. M. Fraser, and J. Achibold. (1979). *Merck Veterinary Manual*.(5th edn.). Merck & CO.Inc,USA.
15. Snell,R.S. (1984). *Clinical and Functional Histology for Medical Students*. (1st edn.). Boston.

- Little, Brown and Company.
16. Sompolsky,D.(1953). De 1 impetigo contagiosa suis et du *Micrococcus hyicus* n. sp. Schweiz. Arch. Tierheilkd. 95: 302-309.
17. Underdahl, N.R., and M.J. Twichaus. (1975). Exudative epidermitis, pp.965-969. In H. W.Dunne and

- A.D.Leman (ed.), Diseases of swine, 4 th ed.Iowa State University Press . Ames .
18. Zarzour, J. Y. and E. A. Belle. (1978). Evaluation of three test procedures for identification of *Staphylococcus aureus* from clinical sources. J. Clin. Microbiol. 7 : 133

دراسة فسيولوجية نسجية ومرضية لالتهاب البشرة النضحي الحاصل تلقائيا في الخنازير البرية

مكية عبد الجبار حسون
كلية الطب البيطري/جامعة القادسية

ميران عبد الأمير عطية
كلية الطب البيطري/جامعة القادسية

عبد الصمد عليوي حسن
كلية العلوم/جامعة المثنى

الخلاصة

أظهرت الدراسة الحالية أن التهاب البشرة النضحي في الخنازير البرية تميز بكونه التهابا حادا يصيب البشرة ويحصل غالبا في الخنازير بعمر الشهرين. وتميز هذا الالتهاب بعلامات سريرية على المستوى النسجي المرضي بكونه التهابا قشرياً في الجلد يصطحب بتكوين حويصلات جلدية إضافة إلى ظهور تجمعات لبثرات وتحبب استسقائي للبشرة مع وجود خراج لخلايا بيض حمضية. أما على المستوى البكتيري فتأكد أن المكورات العنقودية من فصيلة هايكس كانت هي الغازية لأقدام هذه الخنازير. وقد ظهرت حالات من النوع الفيروسي الذي قد يكون عاملاً مرضياً متداخلاً لهذه الإصابة. وبناءً على هذه النتائج يستنتج أن التهاب البشرة النضحي عد كمرض شائع ينتشر في الخنازير العراقية البرية ويؤدي أحيانا إلى هلاك نصف قطعان الخنازير المغزوة بالعامل المرضي المسبب.