Kasetsart J. (Nat. Sci.) Vol.22:371-375 (1988)

Pathological Findings of Swine Lymphosarcoma

Jarernsak Salakij[1], Chaleow Salakij and Chareon Thongma[2]

ABSTRACT

A lymphocytic lymphosarcoma poorly differentiated with multicentric form was found in a 4-week old female mixed breed piglet. The neoplastic cells showed high-grade malignancy with many mitotic figures and infiltrated in many organs including all lymph nodes. The other affected organs were the liver, spleen, kidneys, lungs, heart, gastrointestinal tracts, brain, trigeminal ganglion, dorsal root ganglion of thoracic spinal cord, longissimus lumborum muscle and bone marrow.

INTRODUCTION

Lymphosarcoma is the most common neoplasm in swine (Jubb *et al.*, 1985 ; Moulton and Dungworth, 1978 ; Fisher and Olander, 1978) and affects both sexs of pigs, primarily in the young animal before maturity and also in mature animal (Bostock and Owen, 1973). Anatomical classification includes multicentric and thymic lymphosarcoma (Jarret and Mackey, 1974). Histological classification includes poorly differentiated, lymphoblastic, lymphocytic and prolymphocytic, and histiocytic, histioblastic and histiolymphocytic lymphosarcoma (Jarrett and Mackey, 1974).

A possible viral etiology has been considered, the viruses transmit to the fetus via placenta (Bostock and Owen, 1973) and the serological investigations (Busse *et al.*,1978) have been unsuccessful. Head *et al.*,(1974) and McTaggart *et al.*, (1971) suggested this disease associated with an autosomal recessive gene. Saito *et al.* (1982) reported familial leukemia in swine.

MATERIALS AND METHODS

A 4-week old female mixed breed piglet showed emaciation, depression and pneumonia. It was the smallest among the littermates. The animal died during transportation to the Animal Hospital on March 5, 1987.

General appearances before post-mortem examination were emaciated anemic carcass, palpeble enlarged kidney and purulent nasal discharge. All post-mortem examination specimens were fixed in 10% neutral buffered formalin for routine histological examination. The sections were cut at 5 to 6 μm in thickness and stained by hematoxylin-eosin.

RESULTS

Gross Pathology. All lymph nodes (superficial, deep and viseral lymph nodes) were enlarged to about five-times of normal size (Figure 1). On the cut surface showed soft greyish white bulgy nodes, no cortex or medulla recognizable. There were many white foci easily seen throughout the

[1] Dept. of Medicine, Faculty of Veterinary Science, Kasetsart University, Bangkok 10900, Thailand.
[2] Dept. of Pathology, Faculty of Veterinary Science, Kasetsart University, Bangkok 10900, Thailand.

liver surface. On the cut surface white foci accentuated around blood vessels.

The spleen looked normal although spleenic lymph nodes were enlarged markedly. The Kidneys showed severe cortical hemorrhage, on cut surface showed cortical hemorrhage and pressure atrophy of renal medulla by virtue of renal nodes enlargement. Throughout the cortices on the cut surface had many poorly demarcated streaks which were greyish-white in color. The urinary bladder contained 30 ml urine and urinalysis is shown on Table 1. From the urinalysis indicated that renal failure.

The stomach showed many white nodules throughout stomach mucosa and on serosal surface. The duodenum had prominent white nodules which obliterated normal mucosa. The jejunum and ileum had white nodules on both mucosal and serosal surfaces. In the large intestine, there were less white nodules than in the small intestine.

The lungs showed lobar pneumonia. The heart showed generalized cardiac enlargement with white foci on epicardial fat. The trigeminal ganglion showed marked enlargement where as the brain looked normal. Thoracic spinal cord showed the abnormal enlargement of dorsal root ganglion. The involvement of longissimus lumborum muscle showed dull white fish-meat appearance and the cut surface was homogeneous grey with total loss of muscular architecture and petechial hemorrhages. The bone marrow from left femur showed greyish white in color.

Table 1 *Urinalysis*

Color	Pale yellow
Transparency	Clear
Sp. gr.	1.018
pH	5
Protein	trace
Glucose	Neg
Ketone	Neg
Bilirubin	Neg
Hb/blood	Neg
RBC	< 1/hpf
WBC	< 1/hpf
Casts/Crystals/Cells	
	Wbc casts
	tubular epithelium

Histopathology. In paraffin sections of lymph nodes with hematoxylin-eosin stained, nodal architecture was replaced by infiltration of neoplastic cells that obliterated both cortex and medulla of all nodes. The neoplastic cells were round or oval shape and had round opening nuclei with a large prominent haloed nucleolus. The cytoplasm staining were ranged from eosinophillic to mildly basophillic and had indistinct cell membrane (Figure 3). Mitotic figures were about 10-20 per 40 × field. In some nodes, there were capsule infiltrations. In the liver, many neoplastic cells were crowded at the perilobular connective tissues, which formed broad peri-

lobular marginations with sinusoidal colonization that caused hepatocellular degenerations and hepatic cord destructions (Figure 2).

In the spleen, neoplastic cells invaded both spleenic hilar fat and the red pulps, no white pulp was recognizable. In the kidneys, neoplastic cells invaded both cortex and medulla that destructed normal cells of the kidney. Also seen neoplastic cells in congested blood vessels in the cortex. The neoplastic cells were not different from those found in the lymph nodes and mitotic figures were easily seen.

The stomach sections showed heavy infiltration of neoplastic cells in all layers but still looked normal. The duodenal sections infiltrated with severe invasion of neoplastic cells and total columnar epithelial cells were slaughed out. The jejunal sections showed heavy invasion only on serosal surface. In the ileum, there were hyperplasia of Payer's patches with normal lymphocytic series and reticulum cells.

Infiltrating peribronchiolar and interalveolar septum with neoplastic cells were found in the lung, caused sign of pneumonia. The neoplastic cells were found in epicardial fat of the heart which showed myocardial degenerations.

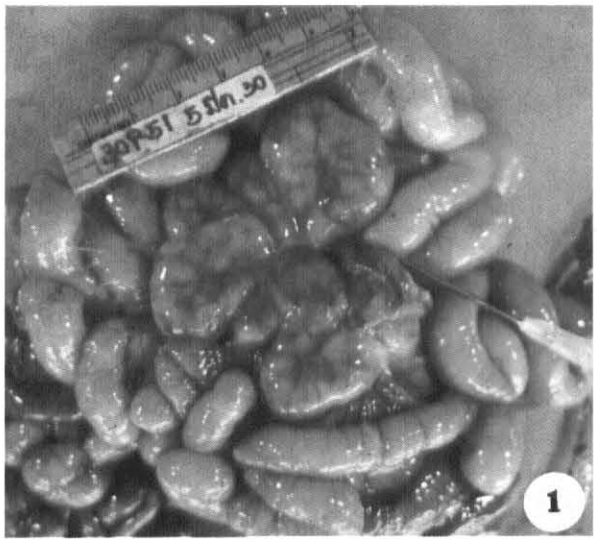
The infiltrated neoplastic cells were noted in the trigeminal ganglion (Figure 4) and dorsal root ganglion that caused pressure atrophy to thoracic spinal cord. Fews neoplastic cells invaded in pia mater of cerebral cortex and choroid plexus of 4^{th} ventricle of cerebellum. The muscle section showed marked infiltration of neoplastic cells with many mitotic figures and the fibers disrupted with a considerable degree of variation in size (Figure 5). The bone marrow imprint showed only lymphocytic series, no myeloid series appeared.

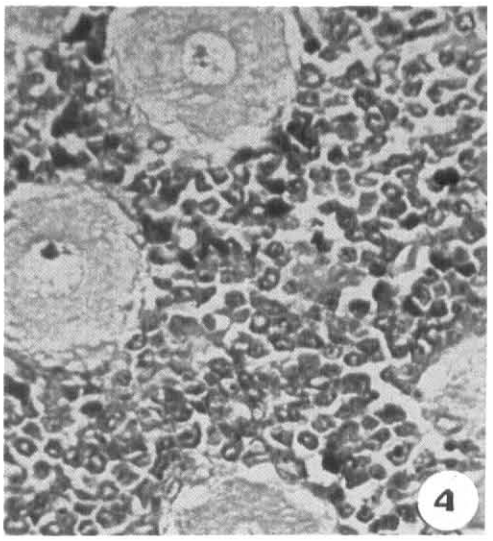
DISCUSSION

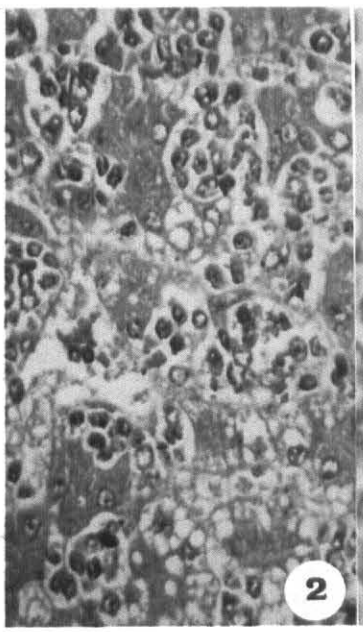
In this suckling pig, there was a generalized massive bilateral enlargement of all lymph nodes and the spleen. The neoplasm showed a high-grade malignancy by many mitotic figures and severe organ destruction. The other affected organs were the liver, spleen, kidneys, lungs, heart, gastrointestinal tracts, cerebrum, cerebellum, trigeminal ganglion, dorsal root ganglion of thoracic spinal cord, longissimus lumborum muscle and bone marrow. According to anatomical classification, it is multicentric form of lymphosarcoma (Moulton and Dungworth, 1978; Jarrett and Mackey, 1974; Anderson and Jarrett, 1968).

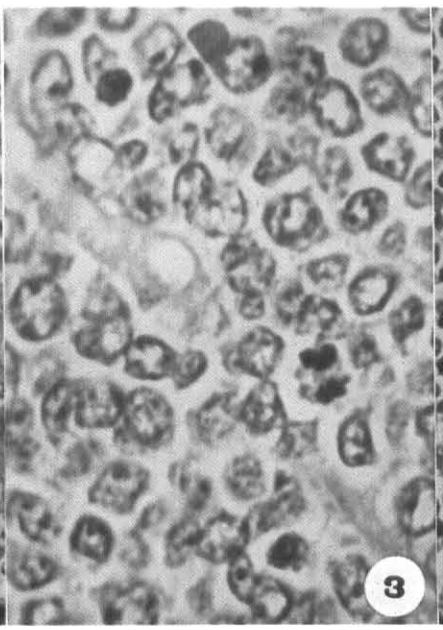
Many neopastic cells in organs were round or oval shape and had round open nuclei with large prominent haloed nucleoli. The cytoplasm staining were ranged from eosinophillic to mildly basophillic and were irregular in outline. According to the international histological classification of tumors in domestic animal (Jarrett and Mackey, 1974), it is poorly differentiated lymphocytic lymphosarcoma. But in Moulton and Dungworth's classification include poorly differentiated lymphocytic lymphoma in lymphoblastic lymphosaroma (Moulton and Dungworth, 1978). The working formulation of National Cancer Institute classification of human lymphoma justified to diagnose the present piglet as a highgrade malignancy lymphoblastic lymphosarcoma (Jubb *et al.*, 1985).

Head *et al.*,(1974) and McTaggard *et al.*, (1971) reported many cases of lymphosarcoma in a large-white herd, apparently associated with an autosomal recessive gene, so the authors followed up this case littermates by monthly complete blood examinations until they were slaughtered. At staughtered examinations, no gilts and barrows showed pathological findings of lymphosarcoma. In the next litter of the dam, no piglets showed both clinical signs and pathological findings of lymphosarcoma. So this case is a sporadic disease, occurring only as isolated case (Anderson and Jarrett, 1968) and cannot demonstrate familial lymphosarcoma condition.









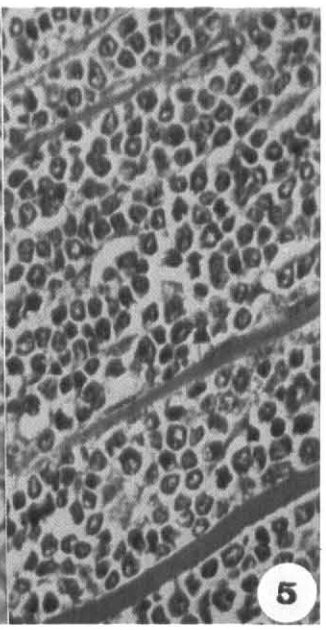


Figure 1 Massive lymph nodes enlargement in the mesentery.

Figure 2 Liver, sinusoidal colonization of neoplastic cells causing hepatocellular degenerations and hepatic cord destructions. (Hematoxylin-eosin stain; × 400)

Figure 3 Lymph node, pooly differentiated lymphocytic lymphosarcoma. (Hematoxylin-eosin stain; × 1,000)

Figure 4 Trigeminal ganglion, marked infiltration of neoplastic cells. (Hematoxylin-eosin stain; × 400)

Figure 5 Longissimus lumborum muscle, marked infiltration and disrupted muscle fiber. (Hematoxylin-eosin stain; × 400)

ACKNOWLEDGEMENT

We wish to thank Dr. Prawat Nithiyanant for histopathological consults, Mrs. Sumalee Tassanawat and Miss Orawan Bootdee for histopathological preparations.

LITERATURE CITED

Anderson, L.J. and W.F.H. Jarrett. 1968. Lymphosarcoma (leukemia) in cattle, sheep and pigs in Great Britain. Cancer 22:398-405.

Bostock, D.E. and L.N. Owen. 1973. Porcine and ovine lymphosarcoma : A review. J. natl Cancer Inst. 50 : 933-939.

Busse, C., H.J. Marschall and V. Moenning. 1978. Further investigations on the porcine lymphoma C-type particle (PLCP) and the possible biological significance of the virus in pigs. Annales de Recherches Veterinaires. 9(4): 651-658.

Fisher, L.F. and H.J. Olander. 1978. Spontaneous neoplasms of pigs : a study of 31 cases. J. Comp. Path. 88 : 505-517.

Head, K.W., J.G. Campbell, P. Imlah, A.H. Laing, K.A. Linklater and H.S. McTaggart. 1974. Hereditary lymphosarcoma in a herd of pigs. Vet Res 95 : 523-526.

Jarrett, W.F.H. and L.J. Mackey. 1974. Neoplastic diseases of the haematopoietic and lymphoid tissues. Bull. Wld Hlth Org. 50 : 21-34.

Jubb, K.V.F., P.C. Kenedy and N. Palmer. 1985. Pathology of domestic animals, pp 105-114 and 181-197. 3rd ed., Vol. 3 Academic Press, Orlando, Fla.

McTaggart, H.S., K.W. Head and A.H. Laing. 1971. Evidence for a genetic factor in the transmission of spontaneous lymphosarcoma (leukemia) of young pigs. Nature. 232 : 557-558.

Moulton, J.E. and D.L. Dungworth. 1978. Tumors in domestic animals. 2nd Ed. Univ. of Calf. Press, Barkeloy, Los Angeles and London: 150-156, 186-190.

Saito Y., Y. Nomura, K. Shiroto, J. Yamakoshi, H. Hizawa, T. Kashima, I. Hara, M. Shinoda and I. Miyashita. 1982. Familial leukemia of swine a report on two cases in consecutive two generations and one related case. Bull Azabu. Univ., Vet. Med. 3(2) : 201-202.