中華民國比較病理學會

Chinese Society of Comparative Pathology

第 46 次比較病理學研討會

國軍桃園總醫院

桃園縣・臺灣

中華民國 98 年 7 月 11 日

46th Meeting of Comparative Pathology

Taoyuan Armed Forces General Hospital

Taoyuan, Taiwan

July 11, 2009

中華民國比較病理學會第 46 次比較病理學研討會議程表

Schedule, 46th Meeting of the Chinese Society of Comparative Pathology

時間：98 年 7 月 11 日(星期六) 08:30~17:00　　Date: July 11, 2009 (Sat) 08:30~17:00
地點：國軍桃園總醫院　　Location: Taoyuan Armed Forces General Hospital
地址：桃園縣龍潭鄉中興路 168 號　　Address: No. 168 Chong Shin Road, Lungtan, Taoyuan
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Time 時間	Schedule 議程		Moderator 主持
08:30~08:50	Registration 報到		
08:50~09:00	Welcome Ceremony 致詞		Dr. C.H. Liu 劉振軒 理事長
09:00~09:15	Speech 專題演講	**Dr. T.H. Wang 王東弘 醫師** Taoyuan Armed Forces General Hospital 國軍桃園總醫院	Dr. J.L. Chang 張俊梁 主任
09:15~09:30		**Dr. M.D. Lin 林孟德 醫師** Taoyuan Armed Forces General Hospital 國軍桃園總醫院	
09:30~09:45		**Dr. K.T. Liu 劉光庭 醫師** Taoyuan Armed Forces General Hospital 國軍桃園總醫院	
09:45~10:15	Case 319 病例討論	**Dr. H.Y. Chiou 邱慧英 獸醫師** Animal Technology Institute Taiwan 台灣動物科技研究所	
10:15~10:30	Coffee Break		
10:30~11:00	Case 320 病例討論	**Dr. C.H. Chang 張正皇 醫師** Buddhist Tzu-Chi General Hospital 佛教慈濟綜合醫院	Dr. F.J. Leu 呂福江 主任
11:00~11:30	Case 321 病例討論	**Dr. M.T. Tsai 蔡睦宗 獸醫師** Pingtung Livestock Disease Control Center 屏東家畜疾病防治所	
11:30~12:00	Case 322 病例討論	**Dr. C.W. Shih 施洽雯 醫師** Lotung Poh-Ai Hospital 羅東博愛醫院	
12:00~13:30	Lunch 午餐暨「中華民國比較病理學會理監事會議」		
13:30~14:00	Case 323 病例討論	**Dr. J.L. Chang 張俊梁 醫師** Taoyuan Armed Forces General Hospital 國軍桃園總醫院	Dr. Y.H. Hsu 許永祥 主任
14:00~14:30	Case 324 病例討論	**Dr. Y.L. Chen 陳燕麟 醫師** Cardinal Tien Hospital 天主教耕莘醫院	
14:30~15:00	Case 325 病例討論	**Drs. C.C. Jeng, V.F. Pnag 鄭純純、龐飛 獸醫師** National Taiwan University 國立臺灣大學獸醫專業學院	
15:00~15:10	Coffee Break		
15:10~15:45	Case 326 病例討論	**Drs. C.N. Hsiao, C.R. Jeng 蕭君倪、鄭謙仁 獸醫師** National Taiwan University 國立臺灣大學獸醫專業學院	Dr. C.H. Liu 劉振軒 理事長
15:45~16:15	Case 327 病例討論	**Drs. Y.H. Wu, J.W. Liao 吳依璇、廖俊旺 獸醫師** National Chung Hsing University 國立中興大學獸醫學院	
16:15~16:45	Case 328 病例討論	**Dr. C.T. Liang 梁鍾鼎 獸醫師** National Laboratory Animal Center 國家實驗動物中心	
16:45~17:00	General Discussion 綜合討論		

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Case Signalment
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Case No.	Presenter	Institution	Slide No.	Signalment
Case 319	Dr. H.Y. Chiou 邱慧英 獸醫師	Animal Technology Institute Taiwan 台灣動物科技研究所	R05-43	adult female goat
Case 320	Dr. C.H. Chang 張正皇 醫師	Buddhist Tzu-Chi General Hospital 佛教慈濟綜合醫院	07-4713	35-year-old man
Case 321	Dr. M.T. Tsai 蔡睦宗 獸醫師	Pingtung County Livestock Disease Control Center 屏東縣家畜疾病防治所	Q95-133B	10-month-old crossbred goat
Case 322	Dr. C.W. Shih 施洽雯 醫師	Lotung Poh-Ai Hospital 羅東博愛醫院	LP08-7346	52-year-old woman
Case 323	Dr. J.L. Chang 張俊梁 醫師	Taoyuan Armed Forces General Hospital 國軍桃園總醫院	90581	69-year-old woman
Case 324	Dr. Y.L. Chen 陳燕麟 醫師	Cardinal Tien Hospital 天主教耕莘醫院	CTH 297047-2	70-year-old man
Case 325	Dr. C.C. Jeng 鄭純純 獸醫師	National Taiwan University 國立臺灣大學獸醫專業學院	NTU08-718A	9-month-old male *Macropus rufogriseus*
Case 326	Dr. C.N. Hsiao 蕭君倪 獸醫師	National Taiwan University 國立臺灣大學獸醫專業學院	NTU09-103B	4-year-old spayed female Persian cat
Case 327	Dr. Y.H. Wu 吳依璇 獸醫師	National Chung Hsing University 國立中興大學獸醫學院	CW09-001G	adult female Formosan serow
Case 328	Dr. C.T. Liang 梁鍾鼎 獸醫師	National Laboratory Animal Center 國家實驗動物中心	NLAC 080192	wild adult male rat

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Case Diagnosis
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Case No.	Presenter	Institution	Slide No.	Diagnosis
Case 319	Dr. H.Y. Chiou 邱慧英 獸醫師	Animal Technology Institute Taiwan 台灣動物科技研究所	R05-43	Placentitis, *Coxiella burnetii*
Case 320	Dr. C.H. Chang 張正皇 醫師	Buddhist Tzu-Chi General Hospital 佛教慈濟綜合醫院	07-4713	Cutaneous leishmaniasis
Case 321	Dr. M.T. Tsai 蔡睦宗 獸醫師	Pingtung County Livestock Disease Control Center 屏東縣家畜疾病防治所	Q95-133B	Pneumonia, *Burkholderia pseudomallei*
Case 322	Dr. C.W. Shih 施洽雯 醫師	Lotung Poh-Ai Hospital 羅東博愛醫院	LP08-7346	Allergic fungal sinusitis
Case 323	Dr. J.L. Chang 張俊梁 醫師	Taoyuan Armed Forces General Hospital 國軍桃園總醫院	90581	Metastatic papillary serous cystadenocarcinoma, abdomen
Case 324	Dr. Y.L. Chen 陳燕麟 醫師	Cardinal Tien Hospital 天主教耕莘醫院	CTH 297047-2	Malignant gastrointestinal stromal tumor
Case 325	Dr. C.C. Jeng 鄭純純 獸醫師	National Taiwan University 國立臺灣大學獸醫專業學院	NTU08-718A	Myocarditis/encephalitis, *Toxoplasma gondii*
Case 326	Dr. C.N. Hsiao 蕭君倪 獸醫師	National Taiwan University 國立臺灣大學獸醫專業學院	NTU09-103B	Meningoencephalitis, *Aspergillus flavus*
Case 327	Dr. Y.H. Wu 吳依璇 獸醫師	National Chung Hsing University 國立中興大學獸醫學院	CW09-001G	Dermatitis, mange infestation
Case 328	Dr. C.T. Liang 梁鍾鼎 獸醫師	National Laboratory Animal Center 國家實驗動物中心	NLAC 080192	*Trichosomoides crassicauda*, urinary bladder

EphrinA5 acts as a tumor suppressor in colon cancer by down regulating EGFR expression

Tong-Hong Wang (王東弘), Wan-Hua Yang (楊婉華), Kuang-Ting Liu (劉光庭), Junn-Liang Chang (張俊梁)
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EphrinA5, a member of Eph/Ephrin family, are known to overexpress in numerous human cancers and has been demonstrated to strong involvement in tumorigenesis. In this study, we showed that the expression of eprinA5 was dramatically downregulated in colon cancer compared with normal tissue. The tumor grade was significantly correlated with ephrinA5 levels. In addition, the expression pattern of ephrinA5 was mutually exclusive and negative correlated with epidermal growth factor receptor (EGFR), which frequently acts as an oncoprotein in colon cancer. Furthermore, forced expression of ephrinA5 in human colorectal carcinoma cell line SW480 significantly reduced EGFR expression and cell proliferation. These findings suggested that ephrinA5 acted as a tumor suppressor by down regulating EGFR expression in colon cancer. In this study, we identified a novel tumor suppressor mechanism of ephrinA5 in colon cancer and it may provide the assets for developing improved therapeutic strategies.

Key words: EphrinA5, tumorigenesis, colon cancer, EGFR

Expression of CDCA8, as a Potential Biomarker, in Renal Cell Carcinoma

Meng-De Lin (林孟德),Tong-Hong Wang (王東弘), Wan-Hua Yang (楊婉華), Mei-Lin Li (黎美玲), Junn-Liang Chang (張俊梁)
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Cell division cycle associated 8 (CDCA8), also called borealin, is a construction of Chromosomal passenger complex associated with cell division cycle and helping localization during mitosis. Thus, significant evaluation of CDCA8 expression would be found in active cell division. Patient with over expression of CDCA8 would have worse prognostic in colon, stomach, brain, and lung cancers. However, CDCA8 expression in renal cell carcinoma (RCC) is seldom discussed. In this study, we applied tissue microarray (TMA) by immunohistochemical (IHC) method to recognize the relationship of CDCA8 and RCC and trying to find potential prognostic marker. There 60 renal cell carcinoma tissues, included 10 transitional cell carcinomas (TCCs), 32 clear cell carcinomas, 6 chrophobe cell carcinomas, 9 papillary cell carcinomas, and 3 squamous cell carcinomas (SCCs), were founded 23% of 0 IHC score, 25% of 1, 35% of 2 and 10% of 3. Comparison of normal tissue, 77% of RCC expression of CDCA8 and 52% showed strongly IHC stain for CDCA8 in the RCC. Base on pilot result, we proposed that highly CDCA8 expression correlation with RCC. Herein, the finding suggested that inhibition of CDCA8 and cell division may be a target therapy marker or prognostic marker in renal cell carcinoma.

Key words: CDCA8, tissue microarray (TMA), renal cell carcinoma (RCC), immunohistochemistry (IHC)

EphrinA5 acts as a tumor suppressor in colon cancer by down regulating EGFR expression

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Institute of Medical Technology, National Chung-Hsing University[b]

Basal cell carcinoma (BCC) is the most frequent malignant skin tumor in human, usually involving the sun-exposed skin of head and neck area. The tumor growth of basal cell carcinoma is relatively slow and locally invasive, exits a low metastatic potential. BCC displays a considerable diversity of appearance under the microscopie. In the clinical histopathology, BCC can be classified into superficial, infiltrative, nodular, micronodular, morpheaform, pigmented, and mixed BCC forms. The subtypes of BCC morphological presentations not only exhibit different behavior and etiology but also an important distinction for prognosis and treatment.
Myeloid cell leukemia-1(Mcl-1, also named Mcl-1L), an anti-apoptosis protein of Bcl-2 family, acts as a critical molecule in apoptosis control, preventing cell death by inhibiting cytochrome-C release and caspase activation. Many reports demonstrate Mcl-1 plays an important role in many cancer cell and has been also reported to enhance the apoptosis by silencing in some tumor cell lines. Thus, we purpose that differential expression of Mcl-1 may exit between transformed BCC and primary keratinocytes, more, in subtypes of BCC. In this study, we showed that the mRNA and protein expression level of Mcl-1 in BCC cell line was higher three folds than primary keratinocytes. In addition, we demonstrate the histological sebaceous, infiltrative subtype of BCC may exit dominant expression of Mcl-1. This study provides that a potential morphological etiology and treatment role of Mcl-1 in Basal cell carcinoma.

Key words: B cell leukemia protein family (Bcl-2), Basal cell carcinoma (BCC), Myeloid cell leukemia-1 (Mcl-1L)

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School of Veterinary Medicine, National Taiwan University (國立台灣大學獸醫專業學院)[b]

CASE HISTORY:

Signalment: adult, female, goat

Clinical History:

During 2004 and 2005, a goat farm in Northern Taiwan accounted episodes of severe abortion. Two flocks of goats had been introduced from central and Southern Taiwan about half a year prior to the incidence.

Gross Pathology:

At necropsy, the lungs of fetus were mottled, patchy, dark purple to dark grayish red. The placenta was diffusely dark-red with the presence of some yellow to red turbid exudates on the surface of the cotyledonary and intercotyledonary areas. No other gross lesions were found.

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School of Veterinary Medicine, National Taiwan University (國立台灣大學獸醫專業學院)[b]

CASE RESULT:

Histopathologic Description:

Microscopically, the placenta had multifocal to locally extensive necrosis in the superficial epithelium in both cotyledons and intercotyledonary areas. The trophoblasts were distended by small, approximately 1 μm diameter, basophilic, intracytoplasmic organisms. The underlying stroma had mild to moderate lymphoplasmacytic infiltrates with evident perivascular lymphoplasmacytic aggregates. Macchiavello's staining revealed heavy intracytoplasmic load of positive microorganisms that were negative for Gram stain. In the fetus, mild to moderate inflammation consisting of lymphocytes, plasma cells, and epithelioid macrophages are frequently seen within in the parenchyma of liver, lungs, and kidney. Occasionally, non-suppurative meningoencephalitis and lymphocytic perivascular cuffing could also be observed.

Morphologic Diagnosis:

Placenta: Placentitis, necrotizing, acute, multifocal, moderate, with focal vasculitis and intracellular organisms

Laboratory Results:

1. The detection of *Coxiella burnetii* by PCR with the primer pair of Trans1/Trans2 (687 bp) (Houver et al., 1992) and by nested PCR with the primer pairs of OMP1/OMP2 (501 bp) and OMP3/OMP4 (438 bp) (Zhang GQ et al., 1998) was employed for the final diagnosis of this case.
2. Other laboratory tests including chlamydiosis by PCR, foot & mouth disease by ELISA, and bluetongue by ELISA were all negative. Bacterial culture of the fetal tissues was also negative.

Comments:

Coxiella burnetii is a ubiquitous zoonotic pathogen of Q fever, initially identified in Queensland, Australia, in 1935, after an outbreak of febrile illness among slaughterhouse workers. The *Coxiella* was historically considered as a Rickettsia, but gene-sequence analysis now classifies it in the order *Legionellales*, family *Coxiellaceae*, genus *Coxiella*. It is an intracellular, small pleomorphic Gram-negative bacterium, which completes its life cycle within the phagosomes of

infected cells. Although possessing a membrane similar to that of the gram-negative bacteria, it is usually not stained by the Gram technique. According to Raoult et al. (2005), the survival and multiplication of *C. burnetii* in the acidophilic phagosomes prevent antibiotics from killing the bacteria. Increasing pH with lysosomotropic agents such as chloroquine restores the bactericidal activity of doxycycline. The agent has 2 distinct life cycle stages known as the large-cell variant (LCV) and small cell variant. The large-cell variant is the vegetative form of the bacteria seen in infected cells. The small-cell variant (SCV) may be metabolically inactive and is the extracellular and presumably with infectious form of the organism. The SCV form of C. burnetii is likely to be long-lived in the environment because of its resistance to osmotic stress, physical disruption, and chemical agents. Two phases of the bacterium have been described: the highly virulent phase I organisms are found in the infected hosts and insect vectors. The phase II organisms are less virulent or devoid of virulence for mammalian hosts and are obtained through multiple passages of chicken embryos.

Coxiella burnetii is a potential bioterrorism and occupational hazardous agent. Q fever has been described worldwide except in Antarctica and New Zealand. Through the air-borne, it can be inhaled by humans. A single organism of C. burnetii may cause disease in a susceptible person. In animals, *C. burnetii* can infect many animal species, including domestic animals, birds, reptile, wildlife, and arthropods such as ticks. Cats and dogs may represent reservoirs of *C. burnetii*. Dogs may be infected by tick biting, by consumption of placentas or milk from infected ruminants and by aerosol. The possibility of human Q fever acquired from infected dogs and cats had been reported. The infected animals are generally asymptomatic, but in mammals they may induce pneumonia, abortion, stillbirth, and delivery of weak lambs, calves or kids. The Coxiella burnetii-infected herds of cows have showed shedding the organisms within the milk for 13 months. The ewe can shed the organisms within the vaginal mucus for 71 days. People who may contact with infected animals are at the greatest risk, including farmers, slaughterhouse workers, laboratory workers, and veterinarians. In human, C. burnetii causes highly variable clinical manifestations, ranging from acute to fatal chronic infections. However, about 60% of the human infections are asymptomatic seroconversions. Acute Q fever in humans displays mainly flu-like symptoms, atypical pneumonia or granulomatous hepatitis. Various rare clinical signs of meningoencephalitis, endocarditis, pericarditis, pancreatitis, and abortion have also been described. It is prudent for pregnant women to limit the contact with infected animals, especially with fetal fluids and unpasteurized milk.

In Taiwan, the first case of acute human *C. burnetii* infection was reported in 1993. Since 2005, samples have been routinely collected from goats, sheep, cattle, and wildlife for the study of the seroprevalence and histopathological changes of Q fever and the DNA sequence of *C. burnetii*. The results indicate that Q fever should be considered as a possible pathogen in association with the commonly observed abortion in goats, cattle, and wildlife in Taiwan. To our knowledge, this is the first diagnosis of Coxiella burnetii infection in Taiwan livestock.

Acknowledgments:

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CASE HISTORY:

Signalment: 35 year-old man

History :
A 35-year-old man volunteered to rescue sufferers in Sri Lanka after the tsunami in South Asia in December 2004. An erythematous papule appeared on the ventral side of his right wrist 3 weeks later after he returned to Taiwan. Neither pain nor itching was noted. Gradually it became an erythematous nodule with a central ulceration. The nodule was treated with liquid nitrogen with poor response at a clinic. So he was referred to the Department of Dermatology at the Buddhist Tzu Chi General Hospital. Physical examination did not reveal any systemic symptoms or signs and the skin lesion was totally excised.

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CASE RESULT:

Histopathologic Findings:

Histopathologic examination revealed dense and diffuse infiltrate of histiocytes and lymphocyte in the dermis. On high magnification, the cytoplasma of the histiocytes were filled with numerous bluish to grayish colored oval amastigotes .

Diagnosis: Cutaneous leishmaniasis.

Comments:

The World Health Organization (WHO) has estimated the prevalence of cutaneous leishmaniasis to be 12 million cases per year. Cutaneous leishmaniasis is classified into two different clinical syndromes: New World when it is acquired in the Americas, which is infected by L. mexicana complex (L. mexicana mexicana, L. mexicana amazonensis, L. mexicana venezuelensis); and Old World when it is acquired in Asia, Africa, the Middle East, or Europe, which is infected by L. major, L. tropica, or L. aethiopica. Both the Old World and New World forms of CL present as a spectrum of diseases ranging from single, chronic ulcerative lesions ('oriental sores') as in our case to disseminated nodular lesions ('diffuse' CL). The typical lesion appears as an erythematous papule at the site of inoculation which increases in size and ulcerates. The lesions are round with raised borders and appear 2 to 8 weeks after the patient is being bitten by an infected sandfly. Outbreaks of CL may occur in war-torn countries or in areas after disasters such as the tsunami in the affected regions of Sri Lanka. It is not considered to be endemic in Taiwan and physicians in industrialized countries have few experiences in this type of diagnosis.

The first autochthonous case of CL in Sri Lanka was reported in 1992. The case incidence has increased and more than 600 cases of CL were reported during the past 4 years. The pathogen of CL in Sri Lanka is Teishmania donovani which is usually associated with visceral leishmaniasis in all Asian and Eastern countries. This may require epidemiological study to identify genetic susceptible patients.

Leishmanial infections are transmitted via the bite of infected female sandflies. The parasite lives as an extracellular, flagellated promastigote in the gut of the insect. After multiplication and differentiation in the sandfly gut, in approximately 1 week the infectious promastigotes migrates to the proboscis. Following inoculation into the skin of a mammalian host,

macrophages engulf promastigotes into the lysosomes and the parasites are transformed to obligate intracellular life, amastigotes. Free amastigotes are released from the infected macrophages and infect dendritic cells. Interleukin –12 is released from infected dendritic cells and the infected hosts generate antigen-specific T cell-dependent immunity, the IFNγ-producing Th1 cells. Leishmaniasis is known to induce excellent protection against a secondary challenge, and yet vaccines for Leishmaniasis are non-existent at this moment. An important characteristic of Leishmania infection is the presence of persistent parasites following resolution of the infection. It appears that these parasites are maintained long-term at a very low level by the immune response. As infection with Leishmania induces a strong cell-mediated immune response, it is unclear why the parasites are not completely eliminated. Recent studies indicated that one contributing factor may be the generation of regulatory T cells during the infection, which limit the immune response sufficiently to maintain persistent parasites. These regulatory T cells function in part by the production of IL-10.

The clinical and histologic appearance of the CL skin lesions is related to an interaction between the host's immune response and the virulence factors of the different Leishmania species. The indication for systemic treatment is the presence of mucosal lesions, lymph node metastasis or lesions unresponsive to local treatment. The therapeutic cornerstone is Pentavalent antimonials (Pentostam, Glaxo-Wellcome, Research Triangle Park, North Carolina, USA) and meglumine antimoniate (Glucantime, Rhone-Poulenc, Paris, France) remain the mainstay of therapy for most forms of leishmaniasis. However, those drugs are not available in Taiwan. Other major treatments include physical modalities (heat, cryosurgery), local or intralesional injections, various anti-infective agents (Dapsone, metronidazole, trimethoprim-sulfamethoxazole), amphotericin B, pentamidine, allopurinol, azoles, immunotherapy (i.e. interferon-γ), and a variety of agents . In our patient, CL presented as a local recurrent ulcerative nodule. He had tried cryotherapy first with no effect, then received surgical excision. Although recurrent, the amount of amastigote in pathology was reduced. After the second excision, he took Itraconazole as a combination therapy. Until this writing, no local recurrence or remote infection has been noted.

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CASE HISTORY:

Signalment: 10-month-old, goat, crossbred type, Caprine

Clinical History:

During September 4th and 5th, 2006, four and two dead goats, respectively, were presented to our lab from a goat farm. These goats showed signs of fever, dullness, anorexia, rough hair coat, cough, nasal discharge, respiratory distress, lameness of the hindlimbs, swollen testis, reduced milk production and emaciation in August, 2006 and didn't respond well to OTC, penicillin and licomycin antibiotic treatment. The disease occurred from Jun to September, which was a rainy wet season with occasional typhoons in Pingtung county, southern Taiwan. The susceptible age of the goats was around 10-month-old to 4-year-old with various goat types. The morbidity and fatality was 2.43% (28/1150) and 100% (28/28), respectively. This lung tissue slide was made from a 10-month-old, crossbred type goat sent on September 5th, 2006.

Gross Lesions:

At necropsy, Single or multiple yellowish-white and creamy purulent nodules/abscesses, ranging from 0.2-15 mm in diameter, were found on various organs including lung, spleen, liver, lymph nodes, mammary gland and kidneys of the goats. Abdominal aortic aneurysm was also noticed.

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CASE RESULT:

Histopathologic Description:

Multiple small pyogranulomatous lesions scattered on the parenchyma of the lung. The pyogranulomatous lesions are characterized by central aggregation of necrotic cell debris, neutrophils and bacterial clumps, which surrounded by a thin layer of epithelioid macrophages or giant cells. Then, mostly lymophoctes, few plasma cells and fibroblasts infiltrate at the periphery of this thin layer and finally surrounded by a thick layer of fibrous connective tissue in more chronic lesions. These small lesions may coalesce to become a larger abscess. The pyogranulomatous lesions were also seen on various organs including the liver, spleen, mammary gland and kidney with some haemorrhage. Lots of neutrophils and few plasma cells infiltration were also found on the meninges of cerebellum and formed purulent leptomeningitis.

Morphologic Diagnosis: Lung: pneumonia, pyogranulomatous, multifocal, moderate to severe, goat, alpine type, caprine.

Etiology: *Burkholderia pseudomallei* (formerly named *Pseudomonas pseudomallei*) caused melioidosis in goats.

Comments:

Melioidosis, also known as Whitemore disease, is taken from the Greek word 'melis' (distemper of asses) and 'eidos' (resemblance) by Stanton and Fletcher in 1932. The pathologist Alfred Whitmore and his assistant C. S. Krishnaswami first described melioidosis as a "glanders-like" disease among morphia addicts in Rangoon, Burma, in 1911 (Chen et al. 2005). Melioidosis is a zoonotic disease causes by Pseudomonas Pseudomallei (now known as Burkholderia pseudomallei), a facultively anaerobic gram-negative bipolar-staining bacillus. Infection is acquired from contaminated soil or water, by inhalation, ingestion, or inoculation of cutaneous wounds. Acute disease, most common in young animals, may initially affect the lungs with later spread to other organs, or be primarily septicemic from the site of inoculation. Chronic disease is the more frequent manifestation, in which abscesses in multiple organs cause chronic nonsepecific illness or are an incidental finding of public health importance at slaughter. Melioidosis is a systemic infectious disease affecting a wide range of animal species in tropical

climates and most common in goats, sheep, pigs, and rodents, and is an emerging tropical disease of human, Horses, deer, camel, and laboratory animals are less commonly affected, while dogs, cats, cattle, water buffalo, and fowl are resistant to the disease unless they are immunosuppressed. The pathogen is endemic to regions which typically border 200 north and south of equator, the incidence of disease is particular high in Southeast Asia (most cases in Northeast Thailand, Singapore, parts of Malaysia, Vietnam, Combodia, Laos, and Myanmar) and Northern Australia. Following the 2004 Asian tsunami there were increasing numbers of melioidosis cases in the region, including a cluster of cases from Aceh, Indonesia. There are some reports of its presence in other parts of the world such as Central America, the Caribbean, Indian subcontinent, southern China, Hong Kong, Taiwan, Africa, The Middle East and South Asian countries. The prevalence is highest in wet seasons, and caused acute outbreaks or chronic endemic disease. Clinical signs are variable and reflect the range of organs affected, and case fatality rate is high. Melioidosis causes abscess in multiple organs. The lungs are most consistently affected, with disseminated coalescing nodules, or locally extensive areas of consolidation. Abscesses also affect the spleen, liver, lymph nodes, kidney, or joints and may occur in any visceral tissue. Lesions are particular widespread in goats, and may include mastitis or aortic aneurysm. Neurologic disease is most common in goats; but multifocal aggregates of neutrophils and lymphocytic perivascular cuffs are present in the brain stem and spinal cord. Nodular lesions in the nasal mucosa may be mistaken for glanders. Endometritis and placentitis are important manifestations in cattle. The abscesses of melioidosis are not distinctive in their appearance. The chronic lesions are encapsulated nodular masses, up to 5 cm diameter, with creamy or caseous yellow center. Multifocal aggregates of neutrophils form within 12 hours of infection. Granulomas, present by 3 days, form nodular aggregates of epithelioid macrophages and lymphocytes, and these develop central areas of caseous necrosis and neutrophils infiltration. The gross and histopathologic appearance of melioidosis is not pathognomonic. Caseous lymphadenitis, glanders, and abscesses caused by other bacteria may have a similar appearance, and the brain lesions may be mistaken for listeriosis. A tentative diagnosis may be based on identifying gram-negative bipolar-staining bacilli in sections or tissue smear, but definitive diagnosis requires culture (Caswell et.al. 2007).

Although Taiwan (between the latitudes of 220 and 250 North) is geographically close to this endemic area, Human infection of melioidosis has been reported in the country only sporadically. However, in recent years, the number of sporadic cases has increased substantially in Taiwan. From 2001 to 2005, there are 118 cases of B. pseudomallei infections confirmed by the Taiwan CDC. The majority (104 cases, 88%) resided in southern Taiwan, located between latitudes 220 and 230 North. An outbreak of melioidosis cases developed after typhoon Haitang in southern Taiwan from July 2005 to September 2005. Southern Taiwan can therefore be regarded as an endemic area (Chou et al. 2007). An increase in melioidsis cases compared to other areas in Taiwan was observed in the Er-Ren River Basin, southwestern Taiwan, from November 2001 to August 2006. Findings indicated that the Er-Ren River Basin

in Taiwan has the potential to become a high-prevalence area for melioidosis (Su et al. 2007).

This is a case report of animal melioidosis occurred in the goat farm at Pingtung county, southern Taiwan, near the latitudes of 220 North. The gross lesions, which are similar to the report of animal melioidosis in Australia (Choy et al. 2000), are characterized by single or multiple yellowish-white and creamy purulent nodules/abscesses, ranging from 0.2-15mm in diameter, and were found on various organs including lung, spleen, liver, lymph nodes, mammary gland and kidneys. Abdominal aortic aneurysm was also noticed. The histopathological lesions are characterized by pyogranulomatous lesions surrounded by various thickness of fibrous tissue. Purulent meningitis was found in cerebellum. The pathological lesions are also similar to the reports of human clinically chronic infection of melioidosis with multiple abscesses on various organs. The disease of this goat farm occurred in Taiwan from Jun to September, which was a rainy wet season and occasional typhoons at Pingtung county, southern Taiwan. The occurrence seasons and climate of this goat farm are quite similar to the human medicine reports of melioidosis in the Northern territory of Australia, Thailand and Taiwan, but the goat farm geographically occurred at Pingtung county, which is located at further southern Taiwan, compared to the human reports at Kaoshiung and Tainan counties of southwestern Taiwan. Although there were already some reports on human infection of melioidosis in Taiwan, research studies of animal melioidosis in Taiwan just had been undergoing since 2006. Major advances have been made in molecular studies of Burkholderia pseudomallei and immunology of melioidosis in the world reports. However, there remain large gaps in understanding of the epidemiology of this enigmatic disease and need to be further elucidated.

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CASE HISTORY:

Signalment: 52-year-old woman.

Clinical History:

The 52-year-old woman had a past history of chronic paranasal sinusitis with polyposis and received FESS (functional endoscopic sinus surgery) on 90-12-4. She has suffered from purulent rhinorrhea , nasal obstruction and post-nasal dripping for 2 months. She came to our ENT OPD for help. The functional endoscope showed congestion and swelling of mucosa with abundant mucus in the paranasal sinuses especially the right maxillary sinus. Sinus x-rays showed opaque of right maxillary, ethmoidal and sphenoidal sinuses. CT scan showed radio-opaque masses in the right paranasal sinus. FESS was performed, the hypertrophic sinus tissue and mucinous concretions were removed.

Clinical Pathology:

RBC: 4.53x106/uL (0-5 x106/uL), Hb: 13.9 gm/dL (12.0-16.0 gm/dL), Hct: 41.9 % (37-47%), WBC: 6900/uL (4500-11000/uL), Plt: 18.9 x104/dL (15-40 x104/dL), Lymphocyte: 38.1% (20.0-45.0%), Neutrophil: 48.4% (45.0-75.0%), Monocyte:4.7% (0.0-9.0%), Eosinophil:7.7% (1.0-3.0%), Basophil:1.1% (0.0-1.0%). BUN:18 mg/dL (7-22 mg/dL), Creatinine:0.7 mg/dL (0.6-1.3 mg/dL), Glucose:105 mg/dL (70-110 mg/dL), AST: 47 U/L (5-40 U/L), ALT: 51 U/L (5-40 U/L), Na:143.3 mmol/L (133-145 mmol/L), K:3.56 mmol/L (3.3-5.1 mmol/L), Total IgE: 896 (<140). D Pteronyssinus: <0.35 (class 0), Cat dander: <0.35 (class 0), Dog dander: <0.35 (class 0), Cockroach: <0.35 (class 0), Aspergillus: 6.06 (class 3), Candida: <0.35 (class 0).

Gross Findings:

The specimen submitted consisted of a large amount of mucus and about 20 small tissue fragments from the right maxillary sinus and measuring up to 3.8 x 2.3 x 1.5 cm in size. Six small tissue fragments were removed from the left maxillary sinus and measuring up to 1.2 x 1.0 x 1.0 cm in size. The specimens were soft elastic or hard in consistency and grayish-brown in color.

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CASE RESULT:

Histopathologic Findings:

The tissue fragments obtained from the right and left maxillary sinuses show edema, congestion of blood vessels, proliferation of mucous glands with increased secretory activity and irregularly enlarged with cystic dilatation containing inspissated mucous materials, chronic inflammatory cells infiltrate with many eosinophils. The mucin obtained from the right maxillary sinus shows eosinophils infiltrate, scattered fungal hyphal elements, and Charcot-Leyden crystals. The fungal hyphal element were noted within the mucin without tissue invasion.

Histochemistry:

Fungal hyphal elements were observed in mucin materials by periodic acid-Schiff (PAS) stain.

Differential Diagnosis:

1. Invasive fungal sinusitis.
2. Saprophytic fungal growth.
3. Allergic fungal sinusitis.
4. Fungus ball of the sinuses.
5. Eosinophilic mucin sinusitis.

Diagnosis: Allergic fungal sinusitis

Comments:

In 1976, Safirstein noted that the combination of polyposis, crust formation and sinus cultures yielding aspergillus was similar to the constellation of findings observed in allergic bronchopulmonary aspergillosis. Subsequently allergic fungal sinusitis (AFS) was initially described by Millar in 1981. Over the past two decades AFS has been increasingly identified. It is probably the most frequent rhinosinus disease caused by fungus. AFS is a term used for an infection of the sinus caused by fungal organisms that results in the formation of so-called allergic mucin, made up of pools of mucin-containing eosinophils, numerous Charcot-Leyden crystals, and fungal hyphae.

AFS is a non-invasive disease and often missed in cases of unexplained chronic sinusitis. About 6-8% of chronic sinusitis requiring surgery are caused by AFS. The incidence of AFS appears to

be impacted by geographic factors. Most reported cases of AFS are located in temperate regions of relatively high humidity. AFS is most common among adolescents and young adults. The ages ranging from 5-75 years. The mean age is 21.9 years. The M/F ratio may be age dependent and different in children and adults. In children, males dominated (M/F ratio 2.1:1; average age, 13 y), and in adults, females dominated (M/F ratio 1:1.4; average age, 36 y)

AFS is thought to be an allergic reaction to fungi. Aspergillus is the most common species of AFS. In 1989, Robson et al reported that this condition could be caused by a number of different fungi such as Curvularia, Exserohilum, Alternaria, Drechslera, Helminthosporium, and Fusarium, not only Aspergillus. Since fungal hyphae and elements are often rare, scattered, and fragmented within allergic mucin, rendering identification difficult unless specific histologic stains are used and multiple sections from different areas of the nose and paranasal cavities are formed. Torres et al. believed that in some of the AFS cases, the lack of fungal hyphae may have been resulted from inadequate sampling in the presence of sparse or degenerated fungal hyphae. A positive fungal culture does not confirm the diagnosis of AFS, nor does a negative culture exclude it. For example, fungi may proliferate as saprophytic growth in diseased sinuses. Furthermore, mycology laboratories vary in capability, and specimen handling significantly influences the rate of positive fungal cultures in a clinical setting.

Allergic mucin remains the most reliable indicator of AFS. Recognition of allergic fungal mucin is the initial step to make an accurate diagnosis of AFS. Examination of mucosa and polyps obtained from involved paranasal sinuses reveals pictures consistent with the inflammation of a chronic inflammatory process and should be performed to exclude fungal invasion

Bent and Kuhn proposed five criteria for the diagnosis of AFS (1) type I hypersensitivity diagnosed by history, positive skin test, or serology (2) nasal polyposis (3) characteristic CT scan findings (4) positive fungal smear and (5) allergic mucin. These are now referred to as major criteria. The other six minor criteria are (1) asthma, (2) unilateral predominance, (3) radiographic bone erosion, (4) fungal culture, (5) Charcot-Leyden crystals and (6) serum eosinophilia. These are probably the most widely accepted criteria for diagnosis of AFS.

AFS has a highly specific radiographic appearance based on CT scan and MRI. CT scan shows central area of hyperattenuation in the sinus cavity which represents the allergic mucin. The triad of nasal polyps, characteristic CT scan findings and specific immunoglobulin E titers has been shown to have a sensitivity of 70% and a specificity of 100% for the preoperative diagnosis of AFS. The expanding allergic mucin may cause thinning and erosion of the bone.

The symptoms and signs of AFS may be similar to those of chronic sinusitis including nasal blockage, purulent rhinorrhea, postnasal discharge or headache. The incidence of polyposis in AFS is almost 100%. Most patients with AFS have a history of allergic rhinitis, and the exact

timing of AFS development can be difficult to discern.

Recent evidence supports the theory that AFS represents an immunologic rather than infectious disease. An improved understanding of this underlying disease process has led certain changes in treatment concepts. Medical therapy has begun to shift from an emphasis on systemic antifungal therapy to various forms of topical treatment and immunomodulation including local and systemic corticosteroids, antifungal agents and immunotherapy. Likewise, surgical treatment of AFS has shifted from radical to a more conservative but complete including polypectomy, remove the hypertrophic tissue and mucinous concretions, usually by endoscopic approach. A comprehensive management plan incorporating medical, surgical and immunologic care remains the most likely means of providing long-term disease control for AFS.

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CASE HISTORY:

Signalment: 69-year-old woman

Clinical History:
The 69-year-old female was admitted to our hospital due to intermittent epigastric pain and abdominal fullness off and on for 2 months. She had been well until two months earlier, when intermittent epigastric pain and abdominal extension developed, and worse on the right side than on the left. She denied general weakness, poor appetite and body weight loss. She had suffered from progressive fullness of right abdomen. There was no nausea, vomiting or thin stool. Because the symptoms were persisted, she visited at our OPD for help. Physical examination, there presented a palpable hard and fixed mass about Bcm in greatest diameter over the right lower abdomen without tenderness. Malignancy was highly suspected. Thus, she was admitted for further evaluation and management. On admission, she showed ill-looking status with stable vital sign. A diagnostic procedure by gastroendoscopy with gastric mucosa biopsy was performed.

Clinical Pathology:
CBC abnormality:

Hgb : 9.3g/dl (Ref: 12-16), Hct: 29.3% (Ref: 37-47), WBC: 8.01 x 10^3/μl (4.8-10.8 x 10^3/μl), others were within normal limits.

Clinical chemistry: Within normal limits.
Tumor markers:

CA125: 3188.6 IU/ml (Ref: <35), serum CEA, AFP, and β-HCG showed within normal limits.

Other test: None made.

KUB Findings:
- Spondylosis of L-spine with compression fracture, L2.
- Stool impaction with local ileus.
- IUD *in situ*.

Image Findings of the Abdomen and Pelvic CT scan:
- Thickening of wall of gastric antrum region.

- Minimal ascites.
- Huge intraperitoneal tumor with heterogeneous density (fluid and soft tissue and calcified density HU : 20-200).
- Small lower density nodule noted at right lobe of the liver adjacent to right kidney region with hypo density in arterial phase.
- Heterogeneous tumor in pelvis with ovary and uterus involvement.

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CASE RESULT:

Histopathologic Findings:
The specimen submitted consisted of four tiny pieces of gastric mucosa tissue fragments with gray in color, measured 0.2 x 0.2 x 0.1 cm in the largest one. Microscopically, the sections showed pictures composed of small cysts, acmar, papillary, or dilated tubuloglandular architectures lined with high columnar and cuboidal epithelia with infiltrating growth patterns, and directly invaded to the gastric mucosa. However, there also presented marked ulceration and focally calcification or scattered psammomatous bodies-like components were also found. Subsequently, the fine needle biopsy of the subcutaneous soft tissue mass of the abdominal wall was also performed. The microscopic characteristic was similar to the findings of the gastric mucosa.

Morphologic Diagnosis: Stomach, abdomen : Metastatic papillary serous cystadenocarcinoma with disseminate carcinomatosis (peritoneum, uterus, liver and kidney), suggestive of originating from the malignant papillary serous tumor of the ovary.

Clinical Progression: She was transferred to the Medical Center for further adjuvant chemotherapy application under the final diagnosis.

Comments:
Metastatic involvement of the stomach is unusual. The most common sources of gastric metastases are systemic lymphomas. Metastases of malignant melanoma and carcinomas tend to be multiple and may develop central ulceration. Breast and lung or esophageal carcinomas may mimic diffuse gastric carcinoma by diffusely infiltrating the gastric wall to generate linitis plastica, as described .earlier for primary gastric carcinoma. In previous study, primary cancers were frequently found in lung (22%), breast (20%), malignant melanoma (19%) and esophagus (18%). Multiple lesions were seen in only 43% of these cases solitary metastases were more common than multiple metastases. Most of the metastatic tumors were located in the middle and upper gastric body, mainly on the greater curvature.

Endoscopic features of metastatic tumors of the stomach:
In previous serial studies, endoscopic appearance was broadly classified into 3 categories, which were (1) suhmucosal tumor-like formation (50%), (2) cancer-like fOffilation (33%) and (3)

others (17%). In category (1), 70% of them had a central depression. Concerning category (2), 23 out of 29 cases resembled primary advanced cancer of the stomach.

Endoscopically diagnosed cases, the metastases presented as solitary (65%) or multiple lesions (35 %), and were more frequently located in the middle or upper third of the stomach. It is often resembled that of submucosal tumor (51 %) or primary gastric cancer (39%), the final diagnosis was easily obtained in over 90% of cases from endoscopic biopsies. In two cases of lung cancer and breast cancer, gastric metastases were found before the primary tumors. In the autopsy cases with solid malignancies, metastatic lesions to the stomach were found in 5.4%, and the lung, breast, and esophagus were common primary sites. Malignant melanoma was the most frequent tumor to metastasize to the stomach (29.6%).

Psammoma body in the tumors: A psammoma body is a round collection of calcium, seen microscopically. The term is derived from the Greek word psammomas meaning "sand". Psammoma bodies usually have a laminar appearance, are circular, acellular and eosinophilic. The etiologies of the psammoma bodies are associated with the papillary (nipple-like) histomorphology and are thought to arise from (1) the infarction and calcification of papillae tips, (2) calcification of intralympbatic tumor thrombi.

Psammoma body is Associated with malignant lesions:
Psammoma bodies are commonly seen in certain tumors such as papillary thyroid carcinoma, papillary renal cell carcinoma, serous papillary ovarian adenocarcinoma (cystadenocarcinoma), endometrial adenocarcinomas (papillary serous carcinoma ~3%-4%), meningiomas (benignant and/or malignant), methotheliomas (benignant and/or malignant) etc. Ovarian Tumors are the most common forms of neoplasia in women. The incidence of ovarian cancer ranks below only carcinoma of the cervix and the endometrium. Ovarian cancer accounts for 6% of all cancers in the female and is the fifth most common form of cancer in women in the United States (excluding skin cancer). About 80% are benign, and these occur mostly,in young women between the ages of 20 and 45 years. The malignant tumors are more common in older women between the ages of 40 and 65 years.

Pathogenesis of the ovarian tumors:
Risk factors include nulliparity, family history, and heritable mutations, unmarried women and in married women with low parity, and gonadal dysgenesis in children. Women 40 to 59 years of age who have taken oral contraceptives or undergone tubal ligation have a reduced risk of developing ovarian cancer. The most intriguing risk factors are genetic mutations in both BRCAI and BRCA2 genes. BRCAI mutations occur in about 5% of patients younger than 70 years of age with ovarian cancer. BRCAI or BRCA2 mutations is 20% to 60% by the age of 70 years. Most of these cancers are serous cystadenocarcinomas. About 30% of ovarian adenocarcinomas express high levels of HER2/neu (ERB-B2) oncogene with a poor prognosis.

Mutations inp53 are found in 50% of ovarian carcinomas.

Ovarian Serous Tumors:

These common cystic neoplasms are lined by tall, columnar, ciliated epithelial cells and are filled with clear serous fluid. Together the benign, borderline, and malignant types account for about 30% of all ovarian tumors. About 75% of ovarian tumors are benign or of borderline malignancy, and 25% are malignant. Serous cystadenocarcinomas account for approximately 40% of all cancers of the ovary and are the most common malignant ovarian tumors. Cystadenocarcinomas occur later in life on average, although somewhat earlier in familial cases. The biologic behavior of serous tumors depends on degree of differentiation, distribution, and characteristics of the peritoneal implants, if present. Peritoneal spread may manifest as noninvasive or invasive implants, the latter signifying malignancy. Implants of carcinomas invade the adjacent stroma inducing desmoplasia (invasive implants) and may form large intra-abdominal masses with rapid clinical deterioration. The 5-year survival rate for borderline and malignant tumors confined within the ovarian mass is, respectively, 100% and 70%, whereas the 5-year survival rate for the same tumors involving the peritoneum is about 90% and 25%, respectively. Because of their protracted course, borderline tumors may recur after many years, and 5-year survival is not synonymous with cure.

Since metastatic lesions to the stomach are rare, the above characteristics of the lesions should be borne in mind, and biopsies should be taken for precise diagnosis during endoscopic examinations. The mean time between diagnosis of the primary tumor and diagnosis of gastric metastasis was 16 months (range, 0 to 56 months). Only seven patients were given some form of treatment after diagnosis of the gastric metastasis. The median survival was 4.75 months. Overall survival during the first year was 20% and survival was nil at 2 years. In conclusion, the gastric metastasis marks advanced disease and the prognosis is poor.

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CASE HISTORY:

Signalment: 70-year-old man.

Clinical history:
This is a 70 years old male patient had left flank pain for 2 months and progressive in recently 3 days. Poor appetite, general malaise were also noted. He had no fever, SOB, nausea, vomiting, change of bowl habit, urinary discomfort or trauma history. Because symptoms were persisted, he came to our hospital for management.

At admission he had left abdominal pain and palpable mass was noted on physical examination. Abdominal sonography was done and showed huge mass lesion at the left upper abdomen, R/O malignant tumor with peritoneal seeding. CT was arranged and a huge mass was found, also r/o peritoneal metastasis. Exploratory laparotomy with Debulking remove tumor, splenectomy and cholecystectomy were performed on 98-05-26. There is a huge mass at left abdomen connected with spleen and some small mass at small intestinal, peritoneum and abdominal wall. Glivac 400mg po QD was prescribed for GIST treatment. Now his condition is well without recurrence with follow up for 2 months.

Gross Findings:
The specimen submitted consisted of 3 pieces of soft tissue measuring about 17 x 15 x 6 cm, 6 x 5 x 5 cm and 7 x 4 x 3 cm in size, fixed in formalin. Grossly, they showed large firm gray tumor mass with tumor necrosis and myxoid change on cut sections. Representative sections were taken.

Laboratory Results:
CBC/DC: WNL
Biochemistry:
Sugar, Ca, BUN, Cr, Na, K, Cl, AST, ALT: WNL
Albumin: 2.4
Serum:
HBsAg , Anti HBs , Anti-HCV Ab: all negative

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CASE RESULT:

Histopathological Finding:

Microscopically, the sections show picture of malignant gastrointestinal stromal tumor with proliferation of thin wavy tumor cells, storiform, palisading, or herringbone pattern. Hemorrhage and necrosis can be seen. The nuclei are blunt ends and are bullet or cigar shaped with atypia and mitotic activities are found.

Immnohistochemical Stains:

1. SMA: focal weakly (+)
2. S-100: (-)
3. CD34: (+)
4. CD117: (+)
5. F-13a: (+)
6. PAS stain: Focally +.

Diagnosis:

Omentum ?, Exp. Lap. --- Malignant gastrointestinal stromal tumor.

Diagnostic Criteria:

1. Heterogeneous with evidence of smooth muscle, neural, fibroblastic or fibrohistiocystic differentiation
2. Typically GISTs are immunohistochemically positive for KIT tyrosine kinase receptor (CD117), which is perhaps their single best defining feature
3. Malignant criteria please see discussion

Discussion:

The gastrointestinal stromal tumor (GIST) is a tumor with nonepithelial spindled and epithelioid cells, most often showing smooth muscle and/or neural differentiation, with varied benign and malignant behavior. GIST accounts for 2.2% of malignant gastric tumors with no gender preference. The most common location of GIST is stomach (60-70%), followed by small intestine (20-30%) and colorectum / oesophagus (together < 10%). Clinical manifestation of GIST is vague abdominal discomfort at majority. Obstructive symptoms and GI bleeding are also common. GIST tumors will project into the lumen with variable ulceration then become the

source of bleeding.

The tumors vary from slightly firm to soft, tan, often with foci of haemorrhage on gross examination. Larger tumors may have massive haemorrhagic necrosis and cyst formation with narrow rim of peripheral viable tissue left. Malignant GISTs are often large, well-circumscribed, heterogeneous with complex cystic masses. Multinodular peritoneal seeding is the typical abdominal metastasis pictures. Microscopic features are heterogeneous tumors composed of the smooth muscle, neural, fibroblastic or fibrohistiocystic differentiation. Typically GISTs are KIT tyrosine kinase receptor positive. It is the single best defining feature so origin from Cajal cells has been proposed.

Most GSTs are positive for KIT (CD117) with membrane, diffuse cytoplasmic or a perinuclear accentuation pattern. About 70-80% are positive for CD34 with membrane pattern and 30-40% positive for SMA with focal or diffuse pattern. There are < 5% positive for desmin and S100.

The criteria for asscessing malignant GISTs is variable. Tumor size and mitotic activities are the most important factors.

	Intestinal GIST	Gastric GIST
Probably benign	< 2 cm, < 5/50 HPF	< 5 cm, < 5/50 HPF
Low malignant potential	2-5 cm, < 5/50 HPF	5-10 cm, < 5/50 HPF
Malignant	> 5 cm or > 5/50 HPF	> 10 cm or > 5/50 HPF

Prognosis will depend on mitotic rate, size, necrosis, depth of invasion, and presence or absence of metastasis. Race and gender did not play a role in survival rates The typical malignant GISTs at all locations is intra-abdominal spread as multiple tumour nodules, and distant metastases most commonly to liver followed by lung and bone in decreasing frequency.

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CASE HISTORY:

Signalment: 9-month-old, male, *Macropus rufogriseus* (紅頸袋鼠)

Clinical History:
The animal showed signs of depression on September 9 and supportive treatment was given. He was found dead at 8:30 am on the next day.

Gross Finding:
The mucosa of the right nostril was red and a small amount of blood was noted in the oral cavity. All lung lobes appeared wet and mottled red with many 1-2 mm white foci throughout the caudal lobe. The left medial lobe of the liver, near the diaphragm, had an about 1.5 x 2.5 cm irregular whit lesion on it. On cross section, the discolored portion involved only the superficial 0.2 cm of the liver parenchyma. The content of the stomach and proximal intestine was red-brown. The mucosa of the gastric body near pylorus had a 7 x 7 cm, dark red, erosive to ulcerative region. The mesenteric fat was hard. The right ventricle of the heart was dilated; its wall was thinner than normal and the moderator bands were more prominent. The left ventricle was moderately hypertrophied. In the region of inter-ventricular septa and the adjacent free wall of the right ventricle of the heart, there was a 3 x 5 cm, red patch in the epicardium. There was a white-yellow area, about 1.2 x 0.7 x 0.2 cm, in the right ventricular wall near the apex of heart. The pancreatic lymph node was enlarged, about 3 x 4 x 2 cm, and a 1 x 1.5 cm, dark red region with yellow to white margin was noted on the cross section.

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CASE RESULT:

Microscopic Findings:

The affected regions seen grossly in the heart are characterized by multifocal, variably sized infiltrates of lymphocytes, macrophages, and plasma cells with necrosis and mineralization in the myocardium. The affected myofibers become homogeneous and atrophy with loss of striations and cytoplasmic mineralization. Within the necrotic regions, intra- and extra-cellular spheroid cysts, ranging from 5 to 30 μm in diameter, morphologically compatible with the cysts of Toxoplasma gondii are often observed. The cysts enclose varying numbers of 1.5 μm, basophilic bradyzoites. Within the brain, there are areas of mild gliosis and perivascular cuffs of lymphocytes and macrophages along with scattered similar cysts of toxoplasma gondii.

Morphologic diagnosis:

Heart: Myocarditis, severe, multifocal, subacute, necrotizing, with intralesional cysts compatible with *Toxoplasma gondii* and myofiber mineralization.
Brain: Encephalitis, mild, multifocal, subacute, with scattered intralesional cysts compatible with *Toxoplasma gondii*

Immunohistochemistry:

Immunohistochemical staining of the heart by using antibodies against *T. gondii* revealed positive result for those intralesional cysts morphologically compatible with those of *T. gondii*.

Molecular Diagnosis:

The DNA extracted from the formalin-fixed tissue subjected to PCR using the primer pair specific for the target B1 locus of *T. gondii* followed by gel electrophoresis revealed a 193 bp amplicon on poliacrylamide gel.

Final Diagnosis:

Toxoplasmosis in a *Macropus rufogriseus*

Comments:

Toxoplasma gondii is a ubiquitous protozoan parasite with a broad intermediate host range, although felids are the only known definitive hosts. In infected cats and most intermediate hosts, clinical disease is generally limited to immature or immunosuppressed animals. However, wallabies and other macropods are known to be highly susceptible to this protozoan and

toxoplasmosis has been reported in these species with high mortality rates through several parts of the world.

Why wallabies are highly susceptible to clinical toxoplasmosis is not known. Morphologically and biologically there is only 1species, T. gondii in the genus. Howe and Sibley (1995) genetically grouped T. gondii isolates from animals and humans into 3 genetic types (I, II, and III). Until that time, T. gondii isolates had been distinguished as mouse virulent or avirulent. Howe et al. (1997) suggested that type I isolates were highly pathogenic for mice, irrespective of the dose, and types II and III were relatively avirulent for mice. However, mouse virulence of T. gondii may have no correlates with clinical toxoplasmosis in higher animals and pathogenicity can vary with the host, the stage ingested, and the dose.

It has been also proposed that all strains of T. gondii should be regarded pathogenic, irrespective of the mouse virulence. The dose, stage of the parasite ingested, and the immune status of the host influence the final clinical outcome of toxoplasmosis. Wallabies inoculated with a nonpathogenic, nonpersistent vaccine strain of *T. gondii* (S48) died of acute toxoplasmosis. In Dubey's study, tissue stages (tachyzoites and bradyzoites) of the T. gondii strains from wallabies caused asymptomatic infection in mice, whereas less than 10 oocysts of 1 isolate either killed the mice or made them ill. The isolates of T. gondii from all wallabies that had clinical toxoplasmosis were genotype III. Nothing is known of the genetic diversity of *T. gondii* isolates circulating in various species of marsupials in Australia. However, other studies suggest that seropositive macropods may be resistant to clinical toxoplasmosis. Not all infected macropods were ill, showing that the infection may be also asymptomatic. Most Australian marsupials are herbivores and presumably acquire *T. gondii* by ingestion of oocysts from cat feces. Transplacental or transmammary infection may occur in joeys.

Treatment with agents aimed at halting tachyzoite replication may alleviate clinical disease, but will not eliminate infection. There are no available drugs that will kill the encysted bradyzoites, and clinical disease may recur during times of stress or immunosuppression. Clindamycin is the drug of choice for treating felids. Pyrimethamine and sulfonamides are often used for treatment in a variety of species, including humans. Treatment of peracute cases in macropods is generally unsuccessful, but prophylactic treatment of other animals in a group may be beneficial. No controlled treatment studies in macropods have been performed. Recent reports on treatment with atovaquone in wallabies have appeared promising.

A serologic survey of antibodies to *Toxoplasma gondii* in Taipei Zoo Animals revealing that overall seroprevalence being 38.75%. The seroprevalence of Macropodidae in Marsupialia is 63.04%(n=46) in particular. The outbreak of acute toxoplasmosis in the red-necked wallabies had occurred in 1993 and 1998. It is hard to eliminate infection that the environment has been contaminated.

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CASE HISTORY:

Signalment: 4-year-old, spayed female, Persian feline

Clinical History:

The animal showed anorexia and poor spirit on 1/24; opisthotonus and stiffness of forelimbs with screaming and sneezing on 1/29; hindlimb weakness and ataxia on 1/30; falling on 2/1; weakness of the left body, staggers and seizure-like episodes occurred on 2/3 and 2/6; stiffness of whole body was noted on 2/6 as well.

Physical examination revealed poor spirit with whole body weakness, 5% dehydration, rigidity when measuring the body temperature and wheeze in auscultation. The heart beat rate was 192 bpm, respiratory rate was 24 bpm, and blood pressue was 120 mmHg. The neurological examination displayed stuporous, Proprioception abscent, tetraparesis and muscle tone increased of all limbs. The exams of cranial nerves were all normal; tail movement was presented as well.

Gross Pathology:

There are no significant abnormalities externally. There are some erosive lesions on the edges of the apex of tongue. The cerebellum shows more swollen than normal because of the suclus become flattened. On the cut surface, the segment of mesencephalic aquaeduct which adjacent to the cerebellum was dilated. Both kidneys have pale, mottled and rough surface. The left lobe of the pancreas contain a round and dark-red material, which about 0.3 × 0.2 cm in size and firm in texture. The liver shows moderate swollen and nut-meg apparently on its surfaces. The spleen displayed moderately swollen. All lobes of right lung show diffuse reddish and swollen. When exposures the thoracic cavity, few volumes of clear and reddish fluids was found, which were submitted for PCR to detect corona virus, the result was positive.

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CASE RESULT:

Histopathologic Description:

Cerebellum: Lesions are located at the white matter majority. In addition to the lymphoplasmocytic and eosinophilic cuffing, there are extreme large numbers of macrophages, lymphocytes, plasma cells, eosinophils and multinuclear giant cells infiltrations within the parenchyma of white matter. There are a lot of opaque and un-staining structures within the cytoplasm of the macrophages and multinuclear giant cells, which are about one to two folds larger than erythrocytes in diameter, and have a halo surrounding and around the eosinophilic vacuolated center. Fungal hyphi are found within the parenchyma of white matter as well, which are branched and segmented and the walls display parallel and have the same diameter compare to the erythrocytes. The meninges and gray matter also have the similar pathological finding aforementioned but have less degree of severity. In GMS stain, multiple branched and segmented fungal hyphi are found within the white matter. The degree of the branch is about 45°, the diameter of wall is about 1.5 ~ 2 fold larger than erythrocyte.

Cerebrum: Multiple lymphoplasmocytic cuffings were found within the white matter. In the parenchyma of white matter, there are large numbers of macrophages, lymphocytes, plasma cells and multinuclear giant cells infiltration. The mesencephalic aquaeduct is dilated, that the ependymal cells within it become cuboid in shape.

Nasal sinus: All the mucosa has been sloughed, which are replaced by large numbers of neutrophils and eosinophiles with some macrophages, lymphocytes and plasma cells.

Morphologic Diagnosis:

1. Meningoencephalitis, granulomatous, locally-extensive, severe, chronic, white matter, with branched and segmented hyphi infiltrate, cerebellum
2. Encephalitis, granulomatous, multifocal to locally-extensive, moderate, white matter, with mesencephalic aquaeduct dilation, cerebrum
3. Nasal sinusitis, diffuse, severe, chronic active, mucosa, nasal sinus

Microbiological Examination:

Samples submitted from brain tissues showed *Aspergillus flavus* by PCR.

Discussion:

Based on the history and pathological findings, it is originally considered that the disease cause the animal illness may be phaeohyphomycosis and Aspergilosis. But the Fontana stain and PCR examination showed negative to Cladosporium, and positive to Aspergillus. Fungi of the genus Aspergillus is a common fungus that lives in soil and decaying vegetation and is ubiquitous throughout the world. The diseases vary in severity and clinical course, depending upon the organds affected. According to the papers published, it can cause high mortality and morbidity if the pathogens invade to the brain. Most cases of aspergillosis are caused by A. fumgiatus, and others are A. flavus, A. niger and A. terreus. These fungi are considered opportunistic pathogens and most patients of neuroaspergillosis are associated about the immunocompetent, organ transplantion, concomitant diabetes mellitus and bacterial infection. The most common neurological symptoms are alteration of mental status, seizures, focal neurological deficits and meningeal signs. The prognosis for CNS Aspergillosis is guarded based on the infected genus and phase of clinical diagnosis. Treatment with both flucytosine and amphotericin B has been recommended for nonresectable lesions in humans and experimental model. Use of corticosteroids has obvious concerns regarding dissemination and worsening of the infection.

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CASE HISTORY:

Signalment: Formosan Serow (*Capricornis Swinhoei*), adult, 12kg, female, goat.

Clinical History:

This animal presented a pitiful body condition, with 90% body surface affected by alopecia, cutaneous crusts, hyperkeratosis, and was found dead at Ji-Ji mountain area.

Gross Finings:

It appeared extremely emaciation, orbit cloudy, alopecia, and obvious cutaneous crusts with cracks, creases and fissures. The lesions could be found overall the body, but the face, ears, limbs, chest, abdomen, flank, and inguinal were the most severe affected regions. In coat exam, there were a large number of mites and its eggs. At necropsy, there were some dark red plaques, different in sizes and disseminated on the cardiac lobe and diaphragmatic lobe of left lung. Some of white plaques with firmness in palpation were found on the bilateral diaphragmatic lobes of lungs.

Microbiological Examination:

Skin specimens from face, forelimb, and abdominal areas were cultured with blood agar and MacConkey agar under 37℃ incubator for 24 hrs. *Staphylococcus aureus* was isolated and identified by using API system.

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CASE RESULT:

Histopathologic Description: Microscopically, the epidermis was thickened by compact hyperkeratosis, hypokeratosis and acanthosis. There was spongiotic, hyperplastic superficial dermatitis with eosinophils and presence of large numbers of mites in the intracorneal tunnels and superficial layers. Two strains of mites were identified as *Sarcoptes spp*. of Sarcoptidae and *Chorioptes spp* of Psoroptidae.

Morphologic Diagnosis:
Hyperkeratosis, hypokeratosis, acanthosis and dermatitis with mange infestation, locally extensive, severe, chronic, skin.

Final Diagnosis: Mange infestation in a Formosan Serow (*Capricornis Swinhoei*)

Comments:
Recently, a case of manage infestation in wild Formosan Serows in Taiwan was first reported by Chen and Pei (2007) at National Pingtung Unerversity of Science and Technology. We also found another case obtained form Ji-Ji mountain area at Nanto County on 2008. This Formosan Serow presented a pitiful body condition, with 90% body surface affected by alopecia, cutaneous crusts, hyperkeratosis, and was found dead, indicated that Formosan Serow might be faced a high risk on its survive in the wild field.

The varieties of mites are parasitic on a number of different hosts, causing a cutaneous disease known as mange. The *Sarcoptes spp.* had lead to severe death in wild animal populations, and even contributed to the extinction of the fox (Vulpes vulpes) on Bornholm island, Denmark. It also became the main reason in the death of chamois (Rupicapra rupicapra) and goats (Capra pyrenaica) at mountain area in south of Europe. Besides, over 80% mortality in chamois in the Alpes, over 95% infection rate and over 90% mortality in goats (Capra pyrenaica) in Cazorla National Park in north of Spain were relate to Sarcoptes spp. Sarcoptes spp. are primarily host

specific, with little evidence of interbreeding between strains (Oleaga et al., 2008).

On the other hand, Chorioptes spp. infects ruminants mainly, but there were few related reports focus on the wild animals. The Japanese serow (Capricornis crispus) and ariel (Gazella gazella) distributed in the Israel were the two species been published (Shibata et al., 2003). Contrary to the Sarcoptes spp, Chorioptes spp. had little host specificity and rarely caused serious damage to the skin, it might subclinically infest animals. Generally, serious mange tends to occur in patients with malnutrition or immunosuppression. These two mites lead to intense pruritus, with consequent scratching, excoriation, and skin inflammation. If left untreated, alopecia, scaling, and crusting of the skin with dried exudates of serum are observed. It may block the movement of the patients which is important in wild animals to hunt and to escape and secondary pyoderma may occur (Pence et al., 2002). Untreated scabies is often associated with pyoderma from secondary infection with group A streptococcus and S. aureus (Walton et al., 2007). In this case, S. aureus was isolated from skin that would enhance the severity of infection.

Occasional cases of human scabies have been reported following exposure to animal scabies, but these infestation are generally self-limiting, with no evidence of long-term reproduction occurring on the unnatural host. Human scabies acquired from a captive wombat was first reported in the early 19th century. Since then, numerous human infections acquired from lots of wild and domestic animals have been reported. Human infections of animal origin differ from typical human scabies in duration of infection, distribution of lesion, and other symptoms. Infections, as mentioned, are usually short-term and self-limited. Lesions often begin on exposed parts of the body where mites enter a hair follicle. A small pustule or erythematous papule develops as in human manges and the infection often reduces after a few weeks. The syndrome is characterized by a scaly rash, slight itching and thickened crusts of skin containing thousands of mites. Although it is generally recognized to be a single heterogenous species, host specificity varies among different strains of S. scabiei (Walton et al., 2007).

Cross-transmission is possible between certain hosts with some strains. Thus, cross-transmission of some strains of S. scabiei is possible between their wild and domestic hosts. In many tropical and subtropical areas, scabies is endemic. In industrialized countries, scabies is observed primarily in sporadic individual cases and institutional outbreaks. The primary contributing factors in contracting scabies seem to be poverty and overcrowded living conditions (Walton et al., 2007).

Scabies is most commonly observed in very young, followed by older children. In individuals never exposed to scabies, the onset of clinical signs and symptoms is 4-6 weeks after infestation, in previously exposed individuals; onset can be as soon as 1-4 days after infestation. People with compromised immune systems, such as HIV, cancer or transplant patients may be

susceptible to crusted or Nerwegian scabies (Walton et al., 2007). In this case the scabies go unregulated by cytotoxic cells and spread over the whole body, except the face.

In stable populations, mange epizootics are generally allowed to run their course and seem to have no long-term effect on population abundance. Alternatively, mange can have devastating effects on small remnant populations, genetically compromised populations, or fragmented populations of threatened or endangered species. In such cases, captured and treatment of individual cases may be warranted. The avermectins, especially ivermectin, have been used to treat many different domestic and wild species with success (Mercier et al., 2002), and are the drugs of choice for treatment of sarcoptic mange.

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CASE HISTORY:

Signalment: wild male rat, adult

Gross Lesions: No significant lesion (NSL) was noted.

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CASE RESULT:

Histopathologic Description
Histologically, the presence of the parasite was confirmed in the urinary bladder. The parasite was located in the lumen or intraepithelially. Infected bladders showed papillomatous epithelial hyperplasia ranging from moderate to severe. Variable degrees of hyperplasia and inflammation in the mucosa were not associated to urolithiasis.

Morphologic Diagnosis
Papillomatous epithelial hyperplasia, with nematodes infestation, moderate, urinary bladder.

Etiology:
Trichosomoides crassicauda infestation, urinary bladder, wild rat.

Comments:
Trichosomoides crassicauda (female 10-19 mm long x 200 um in diameter; males 1.5-3.5 mm long; eggs oval, 60-70 u x 30-35 u and brown, with a thick shell and bipolar plugs), a nematode of the urinary bladder of rats, first described by Bellingham (1840), is a nonpathogenic hair-like worm which has been reported in laboratory and wild rats (Zubaidy and Majeed, 1981). The life cycle requires 8 to 9 weeks, but eggs are not usually present in the urine of rats infected as neonate until 8 to 12 weeks of age. The eggs are passed with the urine and ingested by the next host where they hatch in the stomach. The larvae migrate freely to the lungs, through the blood stream to the kidneys and pass down the ureter to lodge in the bladder (Wahl and Chapman, 1967). The male worm resides within the female worm's genital tract while the larger female worm burrows into the bladder mucosa, leaving her posterior end extending into the lumen.

The diagnostic methods in current use for *T. crassicauda* are inadequate. For example, diagnosis using a dissecting microscope is difficult, as these parasites are not visible to the naked eye unless separated from the bladder and suspended in a clear solution. Even in heavily infected bladders it may be difficult to confirm worm infestation using paraffin wax sections, unless care is taken to prevent the loss of worms during fixation and embedding (Chapman,

1964). The preferred method has been urinalysis, but this is not always reliable as the ova are expelled irregularly and then only by mature worms.

Histopathologically, the aphasmid nematodes (Trichurids, Anatrichosomatids, Trichinellids) differ from the phasmid nematodes in that: (1) They lack a tiny pair of sensory papillae on the caudal end. Aphasmid nematodes do not have lateral chords, instead of hypodermal bands with associated nuclei called bacillary bands. (2) The adult females have only one genital tract. In contrast, most phasmids have two tracts. (3) They contain a row of esophageal gland cells (stichocytes) that form a stichosome. This structure surrounds the esophagus are quite basophilic. (4) Eggs are bipolar plugged but may either be unembryonated or contain a larva.

Laboratory diagnosis of *T. crassicauda* infestation has been a problem for the following reasons: (1) The nematode cannot be adequately diagnosed in live rats using urine examination, as the ova are irregularly expelled and only by mature worms. Immature worms can be present in the bladders of 2 month old rats, whereas mature worms may not be recognized until the rats are over 3 months of age (Weisbroth and Scher, 1971).(2) The worms are transparent and often buried within the mucus. Therefore, the parasite cannot be reliably diagnosed by examination of the bladder mucosal surface, even when viewed under a dissecting microscope. (3) In paraffin wax sections it is difficult to confirm worm infestation even in heavily infected bladders, as the worms may be lost during fixation, dehydration and embedding (Chapman, 1964).

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中華民國比較病理學會
第一次至第四十六次比較病理學研討會病例分類一覽表

分類	病例編號	診斷	動物別	提供單位
腫瘤	1.	Myxoma	Dog	美國紐約動物醫學中心
	2.	Chordoma	Ferret	美國紐約動物醫學中心
	3.	Ependymoblastoma	Human	長庚紀念醫院
	8.	Synovial sarcoma	Pigeon	美國紐約動物醫學中心
	18.	Malignant lymphoma	Human	長庚紀念醫院
	19.	Malignant lymphoma	Wistar rat	國家實驗動物繁殖及研究中心
	24.	Metastatic thyroid carcinoma	Human	省立新竹醫院
	25.	Chordoma	Human	新光吳火獅紀念醫院
	34.	Interstitial cell tumor	Dog	中興大學獸醫學系
	35.	Carcinoid tumor	Human	長庚紀念醫院
	36.	Hepatic carcinoid	Siamese cat	美國紐約動物醫學中心
	38.	Pheochromocytoma	Ferret	美國紐約動物醫學中心
	39.	Extra adrenal pheochromocytoma	Human	新光吳火獅紀念醫院
	40.	Mammary gland fibroadenoma	Rat	國家實驗動物繁殖及研究中心
	41.	Fibroadenoma	Human	省立豐原醫院
	42.	Canine benign mixed type mammary gland tumor	Pointer bitch	中興大學獸醫學系
	43.	Phyllodes tumor	Human	台中榮民總醫院
	44.	Canine oral papilloma	Dog	國立臺灣大學獸醫專業學院
	45.	Squamous cell papilloma	Human	中國醫藥學院
	47.	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	Human	三軍總醫院
	56.	Gastrointestinal stromal tumor	Human	台中榮民總醫院

59.	Colonic adenocarcinoma	Dog	美國紐約動物醫學中心
62.	Submucosal leiomyoma of stomach	Human	頭份為恭紀念醫院
64.	1.Adenocarcinoma of sigmoid colon 2.Old schistosomiasis of rectum	Human	省立新竹醫院
71.	Myelolipoma	Human	天主教耕莘醫院
72.	Reticulum cell sarcoma	Mouse	國家實驗動物繁殖及研究中心
73.	Hepatocellular carcinoma	Human	新光吳火獅紀念醫院
74.	Hepatocellular carcinoma induced by aflatoxin B1	Wistar strain rats	台灣省農業藥物毒物試驗所
81.	Angiomyolipoma	Human	羅東博愛醫院病理科
82.	Inverted papilloma of prostatic urethra	Human	省立新竹醫院
84.	Nephrogenic adenoma	Human	國泰醫院
86.	Multiple myeloma with systemic amyloidosis	Human	佛教慈濟綜合醫院
87.	Squamous cell carcinoma of renal pelvis and calyces with extension to the ureter	Human	台北病理中心
88.	Fibroepithelial polyp of the ureter	Human	天主教耕莘醫院
90.	Clear cell sarcoma of kidney	Human	台北醫學院
93.	Mammary gland adenocarcinoma, complex type , with chondromucinous differentiation	Dog	國立臺灣大學獸醫專
94.	1.Breast, left, modified radical mastectomy, showing papillary carcinoma, invasive 2.Nipple, left, modified radical mastectomy, papillary carcinoma, invasive 3.Lymph node, axillary, left, lymphadenectomy, palillary carcinoma, metaststic	Human	羅東聖母醫院
95.	Transmissible venereal tumor	Dog	中興大學獸醫學系
96.	Malignant lymphoma, large cell type, diffuse, B-cell phenotype	Human	彰化基督教醫院
97.	Carcinosarcomas	Tiger	台灣養豬科學研究所
98.	Mucinous carcinoma with intraductal carcinoma	Human	省立豐原醫院
99.	Mammary gland adenocarcinoma, type	Mouse	國家實驗動物繁殖及研

	B, with pulmonary metastasis, BALB/cBYJ mouse		中心
100.	Malignant fibrous histiocytoma and paraffinoma	Human	中國醫藥學院
102.	Pleomorphic adenoma (benign mixed tumor)	Human	佛教慈濟綜合醫院
103.	Atypical central neurocytoma	Human	新光吳火獅紀念醫院
104.	Cardiac schwannoma	SD rat	國家實驗動物繁殖及研究中心
109.	Desmoplastic infantile ganglioglioma	Human	高雄醫學院
107.	1.Primary cerebral malignant lymphoma 2.Acquired immune deficiency syndrome	Human	台北市立仁愛醫院
111.	Schwannoma	Human	三軍總醫院
114.	Osteosarcoma	Dog	美國紐約動物醫學中心
115.	Mixed germ-cell stromal tumor, mixed sertoli cell and seminoma-like cell tumor	Dog	美國紐約動物醫學中心
116.	Krukenberg's Tumor	Human	台北病理中心
117.	Primary insular carcinoid tumor arising from cystic teratoma of ovary.	Human	佛教慈濟綜合醫院
119.	Polypoid adenomyoma	Human	大甲李綜合醫院
120.	Gonadal stromal tumor	Human	天主教耕莘醫院
122.	Gestational choriocarcinoma	Human	彰化基督教醫院
123.	Ovarian granulosa cell tumor	Horse	中興大學獸醫學系
129.	Kaposi' s sarcoma	Human	華濟醫院
131.	Basal cell carcinoma (BCC)	Human	羅東聖母醫院
132.	Transmissible venereal tumor	Dog	國立臺灣大學獸醫專業學院
137	Canine Glioblastoma Multiforme in Cerebellopontine Angle	Dog	中興大學獸醫病理研究所
143	Osteosarcoma associated with metallic implants	Dog	紐約動物醫學中心
144	Radiation-induced osteogenic sarcoma	Human	佛教慈濟綜合醫院
145	Osteosarcoma, osteogenic	Dog	國立臺灣大學獸醫專業學院
146	Pleomorphic rhabdomyosarcoma	Human	行政院衛生署新竹醫院

147	Papillary Mesothelioma of pericardium	Leopard	屏東科大學獸醫學系
148	Cystic ameloblastoma	Human	台北醫學院
149	Giant cell tumor of bone	Canine	中興大學獸醫學院
150	Desmoplastic small round cell tumor (DSRCT)	Human	華濟醫院
152	Hepatocellular carcinoma	Human	羅東聖母醫院
158	Hemangiopericytoma	Human	羅東聖母醫院
160	Cardiac fibroma	Human	高雄醫學大學病理學科
166	Nephroblastoma	Rabbit	紐約動物醫學中心
168	Nephroblastoma	Pig	台灣動物科技研究所
169	Nephroblastoma with rhabdomyoblastic differentiation	Human	高雄醫學大學病理科
172	Spindle cell sarcoma	Human	羅東聖母醫院
174	Juxtaglomerular cell tumor	Human	新光醫院病理檢驗科
190	Angiosarcoma	Human	高雄醫學大學病理學科
192	Cardiac myxoma	Human	彰化基督教醫院病理科
194	Kasabach-Merrit syndrome	Human	佛教慈濟綜合醫院
195	Metastatic hepatocellular carcinoma, right atrium	Human	新光醫院病理科
197	Papillary fibroelastoma of aortic valve	Human	新光醫院病理科
198	Extraplacental chorioangioma	Human	天主教耕莘醫院
208	Granulocytic sarcoma (Chloroma) of uterine cervix	Human	高雄醫學大學病理學科
210	Primary non-Hodgkin’ s lymphoma of bone, diffuse large B cell, right humerus	Lymphoma	彰化基督教醫院病理科
213	Lymphoma, multi-centric type	Dog	中興大學獸醫系
214	CD30 (Ki-1)-postitive anaplastic large cell lymphoma (ALCL)	Human	新光醫院病理科
215	Lymphoma, mixed type	Koala	國立臺灣大學獸醫專業學院
217	Mucosal associated lymphoid tissue (MALT) lymphoma, small intestine	Cat	國立臺灣大學獸醫專業學院
218	Nasal type NK/T cell lymphoma	Human	高雄醫學大學病理科
222	Acquired immunodeficiency syndrome (AIDS)with disseminated Kaposi’ s sarcoma	Human	佛教慈濟綜合醫院
224	Epithelioid sarcoma	Human	彰化基督教醫院病理

			科
226	Cutaneous B cell lymphoma , eyelid , bilateral	Human	羅東聖母醫院病理科
227	Extramammary Paget’ s disease (EMPD) of the scrotum	Human	萬芳北醫皮膚科,病理科
228	Skin, back, excision, CD30+diffuse large B cell lymphoma, Soft tissue, leg , side not stated, excision, vascular leiomyoma	Human	高雄醫學大學附設醫院病理科
231	Malignant melanoma, metastasis to intra-abdominal cavity	Human	天主教耕莘醫院
232	Vaccine-associated rhabdomyosarcoma	Cat	國立臺灣大學獸醫專業學院
233	1. Pleura: fibrous plaque, 2. Lung: adenocarcinoma, 3. Brain: metastatic adenocarcinoma	Human	高雄醫學大學附設中和醫院病理科
235	1. Neurofibromatosis, type I 2. Malignant peripheral nerve sheath tumor (MPNST)	Human	佛教慈濟綜合醫院
239	Glioblastoma multiforme	Human	羅東聖母醫院
240	Pineoblastoma	Wistar rat	綠色四季
241	Chordoid meningioma	Human	高醫病理科
243	Infiltrating lobular carcinoma of left breast with meningeal carcinomatosis and brain metastasis	Human	佛教慈濟綜合醫院
245	Microcystic Meningioma.	Human	天主教耕莘醫院
247	Well-differentiated fetal adenocarcinoma without lymph node metastasis	Human	新光吳火獅紀念醫院
249	Adenocarcinoma of lung.	Human	羅東聖母醫院
252	Renal cell carcinoma	Canine	國立臺灣大學獸醫專業學院
253	Clear cell variant of squamous cell carcinoma, lung	Human	高雄醫學大學附設中和醫院病理科
256	Metastatic adrenal cortical carcinoma	Human	天主教耕莘醫院
258	Hashimoto’ s thyroiditis with diffuse large B cell lymphoma and papillary carcinoma	Human	高雄醫學大學附設中和醫院病理科
262	Medullar thyroid carcinoma	Canine	國立臺灣大學獸醫專

			業學院
264	Merkel cell carcinoma	Human	羅東博愛醫院
266	Cholangiocarcinoma	Human	天主教耕莘醫院
268	Sarcomatoid carcinoma of renal pelvis	Human	佛教慈濟綜合醫院
269	Mammary Carcinoma	Canine	中興大學獸醫學系
270	Metastatic prostatic adenocarcinoma	Human	天主教耕莘醫院
271	Malignant canine peripheral nerve sheath tumors	Canine	國立臺灣大學獸醫專業學院
272	Sarcomatoid carcinoma, lung	Human	羅東聖母醫院
273	Vertebra,T12,laminectomy, metastatic adenoid cystic carcinoma	Human	彰化基督教醫院
274	rhabdomyosarcoma	Canine	國立臺灣大學獸醫專業學院
275	Fetal rhabdomyosarcoma	SD Rat	中興大學獸醫學系
276	Adenocarcinoma, metastatic, iris, eye	Human	高雄醫學大學
277	Axillary lymph node metastasis from an occult breast cancer	Human	羅東博愛醫院病理科
278	Hepatocellular carcinoma	Human	國軍桃園總醫院
279	Feline diffuse iris melanoma	Faline	中興大學獸醫學系
280	Metastatic malignant melanoma in the brain and inguinal lymph node	Human	佛教慈濟綜合醫院
281	Tonsil Angiosarcoma	Human	羅東博愛醫院病理科
282	Malignant mixed mullerian tumor	Human	天主教耕莘醫院
283	Renal cell tumor	Rat	中興大學獸醫學系
284	Multiple Myeloma	Human	佛教慈濟綜合醫院
285	Myopericytoma	Human	新光吳火獅紀念醫院
287	Extramedullary plasmacytoma with amyloidosis	Canine	國立臺灣大學獸醫專業學院
288	Metastatic follicular carcinoma	Human	羅東聖母醫院病理科
289	Primitive neuroectodermal tumor (PNET), T-spine.	Human	羅東博愛醫院病理科
292	Hemangioendothelioma of bone	Human	佛教慈濟綜合醫院
293	Malignant tumor with perivascular epithelioid differentiation, favored malignant PEComa	Human	彰化基督教醫院
297	Mucin-producing cholangiocarcinoma	Human	基隆長庚醫院
300	Cutaneous epitheliotropic lymphoma	Canine	國立臺灣大學獸醫專業學院
301	Cholangiocarcinoma	Felis Lynx	國立臺灣大學獸醫專業學院

	302	Lymphoma	Canine	國立臺灣大學獸醫專業學院
	303	Solitary fibrous tumor	Human	彰化基督教醫院
	304	Multiple sarcoma	Canine	國立臺灣大學獸醫專業學院
	306	Malignant solitary firous tumor of pleura	Human	佛教慈濟綜合醫院
	307	Carcinoma with thymus-like element	Human	彰濱秀傳紀念醫院
	308	Medullary carcinoma of right lobe of thyroid	Human	彰化基督教醫院
	309	Thyroid carcinosarcoma with cartilage and osteoid formation	Canine	國立臺灣大學獸醫專業學院
	312	Systemic T- lymphocytic leukemia/lymphoma	Koala	國立臺灣大學獸醫專業學院
	313	Neuroendocrine carcinoma of liver	Human	佛教慈濟綜合醫院
	314	Parachordoma	Human	羅東博愛醫院病理科
	315	Carcinoma ex pleomorphic adenoma, submandibular gland	Human	天主教耕莘醫院
	316	Melanoma, tongue	Canine	國立臺灣大學獸醫專業學院
	317	Renal cell carcinoma, papillary type	Canine	國立臺灣大學獸醫專業學院
	323	Metastatic papillary serous cystadenocarcinoma, abdomen	Human	國軍桃園總醫院
	324	Malignant gastrointestinal stromal tumor	Human	天主教耕莘醫院
細菌	6.	Tuberculosis	Monkey	國立臺灣大學獸醫專業學院
	7.	Tuberculosis	Human	省立新竹醫院
	12.	H. pylori-induced gastritis	Human	台北病理中心
	13.	Pseudomembranous colitis	Human	省立新竹醫院
	26.	Swine salmonellosis	Pig	中興大學獸醫學系
	27.	Vegetative valvular endocarditis	Pig	台灣養豬科學研究所
	28.	Nocardiosis	Human	台灣省立新竹醫院
	29.	Nocardiosis	Largemouth bass	屏東縣家畜疾病防治所
	32.	Actinomycosis	Human	台灣省立豐原醫院
	33.	Tuberculosis	Human	苗栗頭份為恭紀念醫院
	53.	Intracavitary aspergilloma and cavitary tuberculosis, lung.	Human	羅東聖母醫院

54.	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	Human	林口長庚紀念醫院
58.	Tuberculous enteritis with perforation	Human	佛教慈濟綜合醫院
61.	Spirochetosis	Goose	國立嘉義農專獸醫科
63.	Proliferative enteritis (*Lawsonia intracellularis* infection)	Porcine	屏東縣家畜疾病防治所
68.	Liver abscess (Klebsillae pneumoniae)	Human	台北醫學院
77.	1. Xanthogranulomatous inflammation with nephrolithiasis, kidney, right. 2. Ureteral stone, right.	Human	羅東聖母醫院
79.	Emphysematous pyelonephritis	Human	彰化基督教醫院
89.	1. Severe visceral gout due to kidney damaged 2. Infectious serositis	Goose	中興大學獸醫學系
108.	Listeric encephalitis	Lamb	屏東縣家畜疾病防治所
113.	Tuberculous meningitis	Human	羅東聖母醫院
134.	Swine salmonellosis with meningitis	Swine	中興大學獸醫學系
135.	Meningoencephalitis, fibrinopurulent and lymphocytic, diffuse, subacute, moderate, cerebrum, cerebellum and brain stem, caused by Streptococcus spp. infection	Swine	國家實驗動物繁殖及研究中心
140	Coliform septicemia of newborn calf	Calf	屏東縣家畜疾病防治所
161	Porcine polyserositis and arthritis （Glasser’ s disease）	Pig	中興大學獸醫學院
162	Mycotic aneurysm of jejunal artery secondary to infective endocarditis	Human	佛教慈濟綜合醫院
170	Chronic nephritis caused by Leptospira spp	Pig	中興大學獸醫學院
173	Ureteropyelitis and cystitis	Pig	中國化學製藥公司
254	Pulmonary actinomycosis.	Human	天主教耕莘醫院
259	Tuberculous peritonitis	Human	彰化基督教醫院病理科
260	Septicemic salmonellosis	Piglet	屏東科技大學獸醫系
261	Leptospirosis	Human	佛教慈濟綜合醫院

	267	Mycobacteriosis	Soft turtles	屏東科技大學獸醫系
	290	*Staphylococcus* spp. infection	Formosa Macaque	中興大學獸醫病理學研究所
	291	Leptospirosis	Dog	國立臺灣大學獸醫專業學院
	296	Leptospirosis	Human	佛教慈濟綜合醫院
	305	Cryptococcus and Tuberculosis	Human	彰濱秀傳紀念醫院
	319	Placentitis, *Coxiella burnetii*	Goat	台灣動物科技研究所
	321	Pneumonia, *Buirkholderia pseudomallei*	Goat	屏東縣家畜疾病防治所
病毒	21.	Newcastle disease	Chickens	國立臺灣大學獸醫專業學院
	22.	Herpesvirus infection	Goldfish	國立臺灣大學獸醫專業學院
	30.	Demyelinating canine distemper encephalitis	Dog	台灣養豬科學研究所
	31.	Adenovirus infection	Malayan sun bears	國立臺灣大學獸醫專業學院
	50.	Porcine cytomegalovirus infection	Piglet	台灣省家畜衛生試驗所
	55.	Infectious laryngo-tracheitis (Herpesvirus infection)	Broilers	國立屏東技術學院獸醫學系
	69.	Pseudorabies (Herpesvirus infection)	Pig	台灣養豬科學研究所
	78.	Marek’ s disease in native chicken	Chicken	屏東縣家畜疾病防治所
	92.	Foot- and- mouth disease (FMD)	Pig	屏東縣家畜疾病防治所
	101.	Swine pox	Pig	屏東科技大學獸醫學系
	110.	Pseduorabies	Piglet	國立屏東科技大學
	112.	Avian encephalomyelitis	Chicken	國立中興大學
	128.	Contagious pustular dermatitis	Goat	屏東縣G台東縣家畜疾病防治所
	130.	Fowl pox and Marek’ s disease	Chicken	中興大學獸醫學系
	133.	Japanese encephalitis	Human	佛教慈濟綜合醫院
	136	Viral encephalitis, polymavirus infection	Lory	美國紐約動物醫學中心
	138	1.Aspergillus spp. encephalitis and myocarditis 2.Demyelinating canine distemper	Dog	國立臺灣大學獸醫專業學院

	encephalitis		
153	Enterovirus 71 infection	Human	彰化基督教醫院
154	Ebola virus infection	African Green monkey	行政院國家科學委員會實驗動物中心
155	Rabies	Longhorn Steer	國立臺灣大學獸醫專業學院
163	Parvoviral myocarditis	Goose	屏東科技大學獸醫學系
199	SARS	Human	台大醫院病理科
200	TGE virus	swine	臺灣動物科技研究所
201	Feline infectious peritonitis(FIP)	Feline	國立臺灣大學獸醫專業學院
209	Chicken Infectious Anemia (CIA)	Layer	屏東防治所
219	1.Lymph node:Lymphdenitis, with lymphocytic depletion and intrahistiocytic basophilic cytoplasmic inclusion bodies. Etiology consistent with Porcine Circovirus(PCV)infection. 2.Lung: Bronchointerstitial pneumonia,moderate, lymphoplasmacytic, subacute.	Pig	臺灣動物科技研究所
220	Cytomegalovirus colitis	Human	彰化基督教醫院病理科
221	Canine distemper virus Canine adenovirus type ll co-infection	Canine	國家實驗動物繁殖及研究中心
223	1. Skin, mucocutaneous junction (lip): Cheilitis, subacute, diffuse, sever, with epidermal pustules, ballooning degeneration, proliferation, and eosinophilic intracytoplasmic inclusion bodies, Saanen goat. 2. Haired skin: Dermatitis, proliferative, lymphoplasmacytic, subacute, diffuse, sever, with marked epidermal pustules, ballooning degeneration, acanthosis, hyperkeratosis, and eosinophilic intracytoplasmic inclusion bodies.	Goat	台灣動物科技研究所
238	Hydranencephaly	Cattle	國立屏東科技大學獸醫學系
248	Porcine Cytomegalovirus (PCMV)	Swine	國立屏東科技大學獸

		infection		醫學系
	250	Porcine respiratory disease complex (PRDC) and polyserositis, caused by co-infection with pseudorabies (PR) virus, porcine circovirus type 2 (PCV 2), porcine reproductive and respiratory syndrome (PRRS) virus and *Salmonella typhimurium*.	Swine	屏東縣家畜疾病防所
	255	Vaccine-induced canine distemper	gray foxes	國立臺灣大學獸醫專業學院
	265	Bronchointerstitial pneumonia (PCV II infection)	Swine	國立臺灣大學獸醫專業學院
	295	Feline infectious peritonitis (FIP)	Cat	中興大學獸醫病理所
黴菌	23.	Chromomycosis	Human	台北病理中心
	47.	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	Human	三軍總醫院
	48.	Adiaspiromycosis	Wild rodents	國立臺灣大學獸醫專業學院
	52.	Aspergillosis	Goslings	屏東縣家畜疾病防治所
	53.	Intracavitary aspergilloma and cavitary tuberculosis, lung.	Human	羅東聖母醫院
	54.	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	Human	林口長庚紀念醫院
	105.	Mucormycosis Diabetes mellitus	Human	佛教慈濟綜合醫院
	127.	Eumycotic mycetoma	Human	佛教慈濟綜合醫院
	138	1.Aspergillus spp. encephalitis and myocarditis 2.Demyelinating canine distemper encephalitis	Dog	國立臺灣大學獸醫專業學院
	298	Systemic Candidiasis	Tortoise	中興大學獸醫學院
	322	Allergic fungal sinusitis	Human	羅東博愛醫院
	326	Meningoencephalitis, *Aspergillus flavus*	Cat	國立臺灣大學獸醫專業學院

寄生蟲	14.	Dirofilariasis	Dog	台灣省家畜衛生試驗所
	15.	Pulmonary dirofilariasis	Human	台北榮民總醫院
	20.	Sparganosis	Human	台北榮民總醫院
	46.	Feline dirofilariasis	Cat	美國紐約動物醫學中心
	49.	Echinococcosis	Human	台北榮民總醫院
	60.	Intestinal capillariasis	Human	台北馬偕醫院
	64.	1.Adenocarcinoma of sigmoid colon 2.Old schistosomiasis of rectum	Human	省立新竹醫院
	66.	Echinococcosis	Chapman's zebra	國立臺灣大學獸醫專業學院
	67.	Hepatic ascariasis and cholelithiasis	Human	彰化基督教醫院
	106.	Parasitic meningoencephalitis, caused by Toxocara canis larvae migration	Dog	臺灣養豬科學研究所
	139	Disseminated strongyloidiasis	Human	佛教慈濟綜合醫院
	141	Eosinophilic meningitis caused by Angiostrongylus cantonensis	Human	台北榮民總醫院病理檢驗部
	156	Parastrongylus cantonensis infection	Formosan gem-faced civet	中興大學獸醫學院
	157	Capillaria hepatica, Angiostongylus cantonensis	Norway Rat	行政院農業委員會農業藥物毒物試驗所
	202	Colnorchiasis	Human	高雄醫學院附設醫院
	203	Trichuriasis	Human	彰化基督教醫院
	204	Psoroptes cuniculi infection (Ear mite)	Rabbit	農業藥物毒物試驗所
	205	Pulmonary dirofilariasis	Human	和信治癌中心醫院
	206	Capillaries philippinesis	Human	和信治癌中心醫院
	207	Adenocarcinoma with schistosomiasis	Human	佛教慈濟綜合醫院
	286	Etiology- consistent with *Spironucleus* (*Hexamita*) *muris*	Rat	國家實驗動物中心
	327	Dermatitis, mange infestation	Serow	中興大學獸醫學院
	328	*Trichosomoides crassicauda*, urinary bladder	Rat	國家實驗動物中心
原蟲	4.	Cryptosporidiosis	Goat	台灣養豬科學研究所
	15.	Amoebiasis	Lemur fulvus	台灣養豬科學研究所
	16.	Toxoplasmosis	Squirrel	台灣養豬科學研究所
	17.	Toxoplasmosis	Pig	屏東技術學院獸醫學系

	51.	Pneumocystis carinii pneumonia	Human	台北病理中心
	57.	Cecal coccidiosis	Chicken	中興大學獸醫學系
	65.	Cryptosporidiosis	Carprine	台灣養豬科學研究所
	211	Avian malaria, African black-footed penguin	Avian	臺灣動物科技研究所
	242	Neosporosis	Cow	國立屏東科技大學獸醫學系
	263	Intestinal amebiasis	Human	彰化基督教醫院病理科
	320	Cutaneous leishmaniasis	Human	佛教慈濟綜合醫院
	325	Myocarditis/encephalitis, *Toxoplasma gondii*	Wallaby	國立臺灣大學獸醫專業學院
立克次體	229	Necrotizing inflammation due to scrub typhus	Human	佛教慈濟綜合醫院
	251	Scrub typhus with diffuse alveolar damage in bilateral lungs.	Human	佛教慈濟綜合醫院
皮膚	216	Cytophagic histiocytic panniculitis with terminal hemophagocytic syndrome	Human	佛教慈濟綜合醫院
其它	9.	Perinephric pseudocyst	Cat	國立臺灣大學獸醫專業學院
	10.	Choledochocyst	Human	長庚紀念醫院
	11.	Bile duct ligation	Rat	中興大學獸醫學系
	37.	Myositis ossificans	Human	台北醫學院
	75.	Acute yellow phosphorus intoxication	Rabbits	中興大學獸醫學系
	76.	Polycystic kidney bilateral and renal failure	Cat	美國紐約動物醫學中心
	151	Osteodystrophia fibrosa	Goat	台灣養豬科學研究所 G台東縣家畜疾病防治所
	80.	1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate 2.Benign hypertension	SHR rat	國防醫學院 G 國家實驗動物繁殖及研究中心
	83.	Phagolysosome-overload nephropathy	SD rats	實驗動物繁殖及研究中心
	85.	Renal amyloidosis	Dog	台灣養豬科學研究所
	89.	1.Severe visceral gout due to kidney damaged 2.Infectious serositis	Goose	中興大學獸醫學系
	91.	Hypervitaminosis D	Orange-rumped agoutis	國立臺灣大學獸醫專業學院

118.	Cystic endometrical hyperplasia	Dog	臺灣養豬科學研究所
121.	Cystic subsurface epithelial structure (SES)	Dog	國科會實驗動物中心
124.	Superficial necrolytic dermatitis	Dog	美國紐約動物醫學中心
125.	Solitary congenital self-healing histiocytosis	Human	羅東博愛醫院病理科
126.	Alopecia areata	Mouse	實驗動物繁殖及研究中心
142	Avian encephalomalacia (Vitamin E deficiency)	Chicken	國立屏東科技大學獸醫學系
159	Hypertrophic cardiomyopathy	Pig	國立臺灣大學獸醫專業學院
165	Chinese herb nephropathy	Human	三軍總醫院病理部及腎臟科
167	Acute pancreatitis with rhabdomyolysis	Human	佛教慈濟綜合醫院
171	Malakoplakia	Human	彰化基督教醫院
183	Darier’ s disease	Human	高雄醫學大學病理科
191	1. Polyarteritis nodosa 2. Hypertrophic Cardiomyopathy	Feline	國立臺灣大學獸醫專業學院
193	Norepinephrin cardiotoxicity	Cat	台中榮總
196	Cardiomyopathy (Experimental)	Mice	綠色四季
212	Kikuchi disease (histiocytic necrotizing lymphandenitis)	Lymphandenitis	天主教耕莘醫院
225	Calcinosis circumscripta, soft tissue of the right thigh, dog	Dog	國立臺灣大學獸醫專業學院
230	Hemochromatosis, liver, bird	Bird	國立臺灣大學獸醫專業學院
234	Congenital hyperplastic goiter	Holstein calves	屏東縣家畜疾病防治所
236	Hepatic lipidosis (fatty liver)	Rats	中興大學獸醫學病理學研究所
237	Arteriovenous malformation (AVM) of cerebrum	Human	天主教耕莘醫院
244	Organophosphate induced delayed neurotoxicity	Hens	中興大學獸醫學病理學研究所
257	Severe lung fibrosis after chemotherapy in a child with Ataxia- Telangiectasia	Human	佛教慈濟綜合醫院
294	Arteriovenous malformation of the left hindlimb	Dog	國立臺灣大學獸醫專業學院

	299	Polioencephalomalacia	Caprine	屏東家畜疾病防治所
	310	Thyroid Follicular Hyperplasia (hyperplastic goiter)	Porcine	屏東縣家畜疾病防治所
	311	Melamine and cyanuric acid contaminated pet food induced nephrotoxicity	Rat	國立中興大學獸醫學院
	318	Alfatoxicosis	Canine	國立臺灣大學獸醫專業學院

中華民國比較病理學會章程

第一章　總則

第一條　本會定名為中華民國比較病理學會，英文名稱為 Chinese Society of Comparative Pathology (CSCP) (以下簡稱本會)

第二條　本會依內政部人民團體法設立，為非營利目的之社會團體，以結合人類醫學與動物醫學資源，提倡比較病理學之研究與發展，交換研究教學心得，聯絡會員友誼及促進國際間比較醫學之交流為宗旨。

第三條　本會以全國行政區域為組織區域，會址設於主管機關所在地區，並得報經主管機關核准設主分支機構。前項分支機構組織簡則由理事會擬訂，報請主管機關核准後行之。會址及分支機構之地址於設置及變更時應報請主管機關核備。

第四條　本會之任務如左：

一、　提倡比較病理學之研究與發展。

二、　舉辦學術演講會、研討會及相關訓練課程。

三、　建立國內比較醫學相關資料庫。

四、　發行比較病理學相關刊物。

五、　促進國內、外比較醫學之交流。

六、　其他有關比較病埋學術發展之事項。

第五條　本會之主管機關為內政部。目的事業主管機關依章程所訂之宗旨與任務，主要為行政院衛生署及農業委員會，其目的事業應受各該事業主管機關之指導與監督。

第二章　會員

第六條　本會會員申請資格如下：

一、　一般會員：贊同本會宗旨，年滿二十歲，具有國內外大專院校(或同等學歷)生命科學及其它相關科系畢業資格或高職畢業從事生命科學相關工作滿兩年者。

二、　學生會員：贊同本會宗旨，在國內、外大專院校生命科學或其它相關科系肄業者 (檢附學生身份證明)。

三、　贊助會員：贊助本會工作之團體或個人。

四、　榮譽會員：凡對比較病理學術或會務之推展有特殊貢獻，經理事會提名並經會員大會通過者。

前項一、二、三項會員申請時應填具入會申請書，經一般會員二人之推薦，經理事會通過，並繳納會費。學生會員身份改變成一般會員時，得再補繳一般會員入會費之差額後，即成為一般會員，榮譽會員免繳入會費與常年會費。

第七條　一般會員有表決權、選舉權、被選舉與罷免權，每一會員為一權。贊助會員、

學生會員與榮譽會員無前項權利。

第八條　會員有遵守本會章程、決議及繳納會費之義務。

第九條　會員有違反法令、章程或不遵守會員大會決議時，得經理事會決議，予以警告或停權處分，其危害團體情節重大者，得經會員大會決議予以除名。

第十條　會員喪失會員資格或經會員大會決議除名者，即為出會。

第十一條　會員得以書面敘明理由向本會聲明退會。但入會費與當年所應繳納的常年會費不得申請退費。

第三章　組織及職員

第十二條　本會以會員大會為最高權力機構。

第十三條　會員大會之職權如下：

一、　訂定與變更章程。

二、　選舉及罷免理事、監事。

三、　議決入會費、常年會費、事業費及會員捐款之方式。

四、　議決年度工作計畫、報告、預算及決算。

五、　議決會員之除名處置。

六、　議決財產之處分。

七、　議決本會之解散。

八、　議決與會員權利義務有關之其他重大事項。

前項第八款重大事項之範圍由理事會訂定之。

第十四條　本會置理事十五人，監事五人，由會員選舉之，分別成立理事會、監事會。

選舉前項理事、監事時，依計票情形得同時選出候補理事五人，候補監事一人，遇理事或監事出缺時，分別依序遞補之。

本屆理事會得提出下屆理事及監事候選人參考名單。

第十五條　理事會之職權如下：

一、　審定會員之資格。

二、　選舉及罷免常務理事及理事長。

三、　議決理事、常務理事及理事長之辭職。

四、　聘免工作人員。

五、　擬訂年度工作計畫、報告、預算及決算。

六、　其他應執行事項。

第十六條　理監事置常務理事五人，由理事互選之，並由理事就常務理事中選舉一人為理事長。

理事長對內綜理監督會議，對外代表本會，並擔任會員大會、理事會主席。

理事長因事不能執行職務時，應指定常務理事一人代理之，未指定或不能指定時，由常務理事互推一人代理之。

理事長或常務理事出缺時，應於一個月內補選之。

第十七條　監事會之職權如左：

一、監察理事會工作之執行。
二、審核年度決算。
三、選舉及罷免常務監事。
四、議決監事及常務監事之辭職。
五、其他應監察事項。

第十八條　監事會置常務監事一人，由監事互選之，監察日常會務，並擔任監事會主席。
常務監事因事不能執行職務時，應指定監事一人代理之，未指定或不能指定時，由監事互推一人代理之。監事會主席（常務監事）出缺時，應於一個月內補選之。

第十九條　理事、監事均為無給職，任期三年，連選得連任。理事長之連任以一次為限。

第二十條　理事、監事有下列情事之一者，應即解任：
一、喪失會員資格。
二、因故辭職經理事會或監事會決議通過者。
三、被罷免或撤免者。
四、受停權處分期間逾任期二分之一者。

第二十一條　本會置祕書長一人，承理事長之命處理本會事務，令置其他工作人員若干人，由理事長提名經理事會通過後聘免之，並報主管機關備查。但祕書長之解聘應先報主管機關核備。
前項工作人員不得由選任之職員（理監事）擔任。
工作人員權責及分層負責事項由理事會令另定之。

第二十二條　本會得設各種委員會、小組或其它內部作業組織，其組織簡則由理事會擬定，報經主機關核備後施行，變更時亦同。

第二十三條　本會得由理事會聘請無給顧問若干人，其聘期與理事、監事之任期同。

第四章　會議

第二十四條　會員大會分定期會議與臨時會議兩種，由理事長召集，召集時除緊急事故之臨時會議外應於十五日前以書面通知之。定期會議每年召開一次，臨時會議於理事會過半數認為必要，或經會員五分之一以上之請，或監事會半數函請召集時召開之。

第二十五條　會員不能親自出席會員大會時，得以書面委託其他會員代理，每一會員以代理一人為限。

第二十六條　會員大會之決議，以出席人數過半之同意行之。但章程之訂定與變更、會員之除名、理事及監事之罷免、財產之處置、本會之解散及其他與會權利義務有關之重大事項應有出席人數三分之二以上同意。但本會如果辦理法人登記後，章

程之變更應以出席人數四分之三以上之同或全體會員三分之二以上書面之同意行之。

第二十七條　理事會及監事會至少每六個月各舉行會議一次，必要時得召開聯席會議或臨時會議。

前項會議召集時除臨時會議外。應於七日以以書面通知，會議之決議各以理事、監事過半數之出席，出席人較多數之同意行之。

第二十八條　理事應出席理事會議，監事應出席監事會議，不得委託出席；理事、監事連續二次無故缺席理事會、監事會者，視同辭職。

第五章　經費及會計

第二十九條　本會經費來源如下：

一、入會費：一般會員新台幣壹仟元，學生會員壹佰元，贊助會員伍仟元，於入會時繳納．

二、常年會費：一般會員新台幣五佰元，學生會員壹佰元．

三、事業費．

四、會員捐款．

五、委託收益．

六、基金及其孳息．

七、其他收入．

第三十條　本會會計年度以國曆年為準，自每年一月一日起至十二月三十一日止。

第三十一條　本會每年於會計年度開始前二個月由理事會編造年度工作計劃、收支預算表、員工待遇表，提會員大會通過（會員大會因故未能如期召開者，先提理監事聯席會議通過)，於會計年度開始前報主管機關核備．並於會計年度終了後二個月內由理事會編造年度工作報告、收支決算表、現金出納表、資產負債表、財產目錄及基金收支表，送監事會審核後，造具審核意見書送還理事會，提會員大會通過，於三月底前報主管機關核備（會員大會未能如期召開者，需先報主管機關備查)．

第三十二條　本會解散後，剩餘財產歸屬所在地之地方自治團體或主管機關指定之機關團體所有．

第三十三條　本章程未規定事項，悉依有關法令規定辦理。

第三十四條　本章程經大會通過，報經主管機關核備後施行，變更時亦同。

第三十五條　本章程經本會民國八十五年二月四日第一屆第一次會員大會通過，並報經內政部 85 年 3 月 14 日台(85)內社字第 8507009 號函准予備查。

會 員 資 料 更 新 服 務

各位會員：

您好！如果您的會員資料有更新或誤刊情形，麻煩您填妥表格後寄回學會秘書處或電話連絡：

中華民國比較病理學會秘書處
10617 臺北市大安區羅斯福路四段 1 號
國立臺灣大學獸醫系三館 106 室 蕭世烜秘書長 收
Tel: (02) 33663858
Fax: (02) 23682423
e-mail address: shsiao1@ntu.edu.tw

----------中華民國比較病理學會----------

會員資料更改卡

姓　　名：＿＿＿＿＿＿＿＿　　會員類別：　☐一般會員
☐學生會員
☐贊助會員

最高學歷：＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿

服務單位：＿＿＿＿＿＿＿＿＿＿職　　稱：＿＿＿＿＿＿

永久地址：＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿

通訊地址：＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿

電　　話：＿＿＿＿＿＿＿＿＿＿傳　　真：＿＿＿＿＿＿

E-Mail Address：＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿＿

中華民國比較病理學會
誠摯邀請您加入

入　會　辦　法

一、本會會員申請資格為：

（一）一般會員：贊同本會宗旨，年滿二十歲，具有國內外大專院校（或同等學歷）生命科學及其它相關科系畢業資格或高職畢業從事生命科學相關工作滿兩年者。

（二）學生會員：贊同本會宗旨，在國內、外大專院校生命科學或其他相關科系肄業者（請檢附學生身份證明）。

（三）贊助會員：贊助本會工作之團體或個人。

（四）榮譽會員：凡對比較病理學術或會務之推廣有特殊貢獻，經理事會提名並經會員大會通過者。

二、會員：

（一）入 會 費：一般會員新台幣一仟元，學生會員一佰元，贊助會員伍仟元，於入會時繳納。

（二）常年會費：一般會員新台幣伍佰元，學生會員一佰元。

【註：學生會員身份變更為一般會員時，只需繳交一般會員之常年會費】

三、請填妥入會申請表郵寄或傳真方式寄回中華民國比較病理學會秘書處收。

地址：10617 臺北市大安區羅斯福路四段 1 號 國立臺灣大學獸醫系三館 106 室
蕭世烜秘書長 收

電話：02-33663858、傳真 02-23682423。

中華民國比較病理學會入會申請及會員卡

會籍電腦編號：

<table>
<tr><td rowspan="2">姓名</td><td>中文</td><td></td><td rowspan="2">性別</td><td>□男</td><td>出生日期</td><td colspan="2">民國　年　月　日</td><td rowspan="2">出生地</td><td rowspan="2">省
縣/市</td></tr>
<tr><td>英文</td><td></td><td>□女</td><td>身份字號</td><td colspan="2"></td></tr>
<tr><td colspan="2"></td><td colspan="4">會員身份：□一般；□學生；□贊助</td><td colspan="2"></td><td></td></tr>
<tr><td rowspan="4">學歷</td><td colspan="4">1.</td><td colspan="4">稱謂：□醫師；□獸醫師；□先生；□小姐；□教授；
□主任；□研究員；□</td></tr>
<tr><td colspan="4">2.</td><td rowspan="3">研究興趣</td><td colspan="4">1.</td></tr>
<tr><td colspan="4">3.</td><td colspan="4">2.</td></tr>
<tr><td colspan="4">4.</td><td colspan="4">3.</td></tr>
<tr><td rowspan="4">主要經歷</td><td colspan="4">機關名稱</td><td colspan="2">職稱</td><td>起</td><td>止</td></tr>
<tr><td colspan="4"></td><td colspan="2"></td><td>年　月</td><td>年　月</td></tr>
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<tr><td>現職</td><td colspan="4"></td><td colspan="2"></td><td>年　月</td><td>年　月</td></tr>
<tr><td rowspan="3">地址</td><td colspan="9">通訊：</td></tr>
<tr><td colspan="9">戶籍：</td></tr>
<tr><td colspan="5">Email：</td><td colspan="4">電話：</td></tr>
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<tr><td colspan="9">茲贊同

貴會宗旨妳加入為會員嗣後並願遵守一切規章共圖發展

此致

中華民國比較病理學會

申請人：　　簽章

介紹人：　　簽章

介紹人：　　簽章

中華民國　　年　　月　　日</td><td>審核結果</td></tr>
</table>