

中華民國比較病理學會
第十三次比較病理學（神經病理專題 I）研討
會

主辦單位：台北市立動物園
臺灣養豬科學研究所
新光吳火獅紀念醫院
行政院農業委員會
臺灣省政府農林廳
中華民國比較病理學會

時 間：中華民國八十七年六月十四日（星期日）

地 點：台北市立動物園教育中心演講廳

研討會籌

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中華民國比較病理學會

第十三次比較病理學（神經病理專題 I）研討會

議 程 表

時間：中華民國八十七年六月十四日（星期日）上午08:40~下午16:20

地點：台北市立動物園教育中心演講廳

主辦單位：台北市立動物園 臺灣養豬科學研究所 新光吳火獅紀念醫院

行政院農業委員會 臺灣省政府農林廳 中華民國比較病理學會

時 間	議 程	主 講 者
08:40- 09:30	報到	
09:30- 09:40	開幕致詞	
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09:40- 10:00	Case 103 新光吳火獅紀念醫院	李進成 醫師
10:00- 10:20	Case 104 國家實驗動物繁殖及研究中心	梁鍾鼎 獸醫師
10:20- 10:40	Case 105 花蓮佛教慈濟綜合醫院	鄭建睿 醫師
10:40- 11:10	茶 點	
11:10- 11:30	Case 106 臺灣養豬科學研究所	劉振軒 博士
11:30- 11:50	Case 107 台北市立仁愛醫院	施麗順 主任
11:50- 13:30	午餐（中華民國比較病理學會理監事聯席會議）	
	Section 【2】	
13:30- 13:50	Case 108 屏東縣家畜疾病防治所	蔡睦宗 獸醫師
13:50- 14:10	Case 109 高雄醫學院	林相如 主任
14:10- 14:30	Case 110 國立屏東科技大學	張聰洲 獸醫師
14:30- 14:50	Case 111 三軍總醫院	李偉華 主任
14:50- 15:05	休 息	
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15:05- 15:25	Case 112 國立中興大學	林正忠 獸醫師
15:25- 15:45	Case 113 羅東聖母醫院	祝志平 主任
15:45- 16:05	Case 114 美國紐約動物醫學中心	劉振軒 博士

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病 歷 摘 要

CP Case 103 新光吳火獅紀念醫院 (S96-17260)

A 33-year-old, previously healthy man suffered from progressive headache, unstable gait and dizziness after a traffic accident for 10⁺ days. Brain CT revealed severe hydrocephalus and a large mass of 7.0×5.0×5.0 cm in size with calcification over suprasellar region mainly occupying third and lateral ventricles. The data of laboratory examination and biochemical studies were all within normal limits. He received frontal vertex craniotomy and transcallosal approach. A gray and well-vascularized soft tumor was seen and encircled the internal cerebral vein. The right ventricle showed nearly total obstruction by the tumor. Partial resection of the tumor was done.

CP Case 104 國家實驗動物繁殖及研究中心 (NLAC84-64)

A 52-week-old, female Sprague Dawley CD(crl: CD[®] [SD] BR) rat showed dyspnea and weight loss.

CP Case 105 花蓮佛教慈濟綜合醫院 (98-373B)

This 55-year-old farmer was a victim of diabetes for years without regular control. He got upper respiratory tract infection 2 weeks prior to this admission with rhinorrhea, cough and fever. Painful swelling at right orbital area and droop of eyelid were found one week later. The acute right paranasal sinusitis and right orbital cellulitis were suspected and treated. Sudden conscious change was noted one day later. The brain CT revealed multiple cerebral infarction. Septic embolization was suspected. The patient's condition worsened and developed multiple new brain infarctions. The patient expired on 36th day of admission.

CP Case 106 臺灣養豬科學研究所 (P97-320K)

A 1 to 2-month-old male, mixed dog was admitted to the hospital with coma on 11/30/1997. After emergency treatment, he got improvement and was discharged with

continuing medicine administration. On 12/4, the dog was submitted again with the clinical signs of coma, corneal edema, meiosis, and frequent urination. Routine urinalysis did not reveal increased level of sugar. He had hematuria and anemia. Blood transfusion was performed on 12/7, but poor response to the treatment. On abdominal palpation, he felt pain. The dog expired on 12/9.

CP Case 107 台北市立仁愛醫院 (J96-4625)

A 31-year-old man, suffered from fever and headache off and on for 5 months. The patient was homosexual and known to be HIV(+) for 5 years. CT scan of brain revealed a progressively enlarging hyperdense tumor, 7.0×6.2 cm, with peripheral rim enhancement of right frontal lobe. Stereotactic needle biopsy in June '96, under the impression of "right frontal lesion, R/O Toxoplasmosis", revealed reactive gliosis and mild perivascular lymphocytic infiltrate. No specific organisms were found. Tumor excision was performed in Sept. '96. The slides presented were from right frontal tumor excision.

CP Case 108 屏東縣家畜疾病防治所 (Q84-517)

A dairy goat farm reared about 85 ewes, 35 rams and 20 2-month-old lambs in Pingtung prefecture. One of the lambs showed signs of depression, and decreased appetite for ten days in December. These prodromal signs were followed by incoordination and hemiparesis with a tendency for the lamb to lean, stumble, or move in one direction only. This tendency progressed to obvious torticollis and circling to the right side direction repeatedly when forced to move. Later, the lamb became exhausted and recumbent with the head pulled tightly into the flank, unable to straighten the neck voluntarily. The lamb also showed facial nerve paralysis with drooped ear, swelling of the left cheek, and drooling. The owner treated the lamb with Procaine Penicillin G and Dexamethasone, but poor responses. The owner sent the affected lamb to our laboratories for pathological diagnosis after our visit. Three lambs, 2-3 months old, also occurred the circling signs in early summer time two years ago and died within one week.

CP Case 109 高雄醫學院 (KMC-96-8886-A7)

A 2-year-old girl suffered from seizure attack on 85/7/21. Brain CT and MRI

revealed a brain tumor with cystic change over right temporal lobe. Pilocytic astrocytoma was impressed. So she was referred to our hospital and received operation on 85/8/13. A grayish-white and elastic nodule, measuring 6.8×6×3.8 cm was submitted.

CP Case 110 國立屏東科技大學 (D82-7799)

A 50-day-old weanling piglet was from a pig farm with 500 sows. Abortion appeared in the sows and nervous signs were seen in the suckling piglets and spread to weanling pigs within a few days. The piglets with nervous signs were submitted for pathological diagnosis. Grossly, there were multiple whitish spots (0.5×1 cm) scattered on the surface and cutting surface of the liver. An ulceration (0.3×1 cm to 1×2 cm) of tonsil was observed. A total loss in one month was 200, mainly weanling piglets.

CP Case 111 三軍總醫院 (263537A)

A 31-year-old man suffered from severe low back pain for 2 years. The symptom was progressively worse several weeks before the admission. CT scan and MRI revealed an intradural and extramedullary tumor. The laboratory examination showed Hgb: 17 gm/dl, WBC 15,500, others: within normal limits. Urinalysis showed no significant finding. The neurological examination revealed consciousness: alert, cranial nerves: intact, sensory function: well preserved, motor function: muscle power 5+ of lower limbs, DTR: 2+ of four limbs, Babinski's reflex: absent, bil.

CP Case 112 國立中興大學 (CP98-036)

Several 14-day-old chicks were submitted from a farm raising 4000 broilers for necropsy. The affected chicks were ataxic, sitting on their hock joints or lying on their sides. According to the owner, the disease was found about a week ago and the morbidity was 12.5%. The chicks had no significant gross lesion.

CP Case 113 羅東聖母醫院 (A028-31)

A 3-year-old boy suffered from a motorcycle traffic accident on sep.22.1993. Consciousness became drowsy & vomitting for several times were noted, so he was sent to LMD, then transferred to Tzu-Chi general hospital (emergency room). At there, physical examination showed neck stiffness, anisocornia and a back hematoma (5×3

cm), Cranial CT revealed hydrocephalus. Under the impression of head injury with hydrocephalus, he was admitted SICU on Oct 3.1993. The conscious state got worse 3 days later, therefore, brain CT was performed again and revealed a communicating hydrocephalus. The arterial blood gas revealed respiratory acidosis, so endotracheostomy was performed with ventilator connected. Because of increased intracranial pressure (20-30mm Hg) on Oct. 12, CSF cytology was examined, but no positive finding was noted. The clinical condition downhill and BP dropped since Oct. 15, and the medical treatment seemed to be not effective, so he was asked discharged by his family and expired on Oct.19.1993. Autopsy was performed with systemic survey of the pathologic changes.

CP Case 114 美國紐約動物醫學中心 (A37721)

This is an eight-year-old, male Rottweiler dog. He has a history of progressive growth of midline cranial mass for 3 months. The dog has progressively deteriorated and now it is unable to eat, has become blind. The owner declined to work-up and treat. Skull radiographs showed severe lysis of skull.

Comparative Pathology Case 103

Contributors: Chin-Cheng Lee (李進成), MD, Ph.D.; Yuh-Yu Chou (周玉瑜), MD; Chih-Peng Wei (魏志鵬), MD.

Department of Pathology and Laboratory Medicine, Shin Kong Wu Ho-Su Memorial Hospital, Taipei, Taiwan (新光吳火獅紀念醫院病理檢驗部)

Clinical history: A 33-year-old, previously healthy man suffered from progressive headache, unstable gait and dizziness after a traffic accident for 10⁺ days. Brain CT revealed severe hydrocephalus and a large mass of 7.0 × 5.0 × 5.0 cm in size with calcification over suprasellar region mainly occupying third and lateral ventricles. The dates of laboratory examination and biochemical studies were all within normal limits. He received frontal vertex craniotomy and transcallosal approach. A gray and well-vascularized soft tumor was seen and encircled the internal cerebral vein. The right ventricle showed nearly total obstruction by the tumor. Partial resection of the tumor is done. Unfortunately, the patient died next day due to acute hydrocephalus and brain edema.

Diagnosis: Atypical Central Neurocytoma

Pathological findings: The specimens were divided into two parts: one part was fixed in 10% buffered neutral formalin, routinely processed and stained with hematoxylin and eosin. The other part, used for electron microscopy, was fixed in 2.5% glutaldehyde. Paraffin sections were also stained for the immunoperoxidase studies using the avidin-biotin-peroxidase (ABC) method, with the following antibodies: neuron-specific enolase (1:100 dilution; DAKO, Kyoto, Japan), synaptophysin (1:30 dilution; DAKO, Kyoto, Japan), Glial fibrillary acidic protein (1:100 dilution; DAKO, Kyoto, Japan), Chromogranin A (1:100 dilution; DAKO, Kyoto, Japan), and Ki-67 (1:30 dilution; DAKO, Kyoto, Japan).

The specimens submitted were fragmented and measured up to 0.7 × 0.5 × 0.4 cm. They are gray and fragile. Microscopically, the tumor composed of monotonous

cells with round to slightly oval nuclei and finely stippled chromatin. The cells were tightly packed in a delicate fibrillated matrix with abundant vasculature. Mitoses were frequent and measured up to 3 mitoses/10 HPF. Focal perinuclear halos, microcalcification, and formation of perivascular pseudorosettes are also seen. Immunohistochemical study shows that the tumor cells were reactive against Synaptophysin, chromogranin A and NSE antibodies and not reactive against GFAP antibody and the labeling index of Ki-67 staining of tumor cells were 5. Electron microscopic study showed presence of synapse-like structure in the tumor cell membrane and neurosecretory granules in the cytoplasm of tumor cells.

Discussion: Central neurocytomas were first described by Hassoun et al [1] in 1982. They are characterized by their intraventricular location, predominant occurrence in young adult and histologic features of uniform round cells with neuronal differentiation. Over 50 serials of reports have appeared in the literatures [2], and their rate of occurrence is 0.1%. Central neurocytomas usually follow a benign course. Only a few cases with malignant histopathology [3] or malignant behavior [4] have been described. In 1997, Soylemezoglu et al proposed a distinct term “atypical central neurocytoma” for those exhibiting a Ki-67/MIB-1 index of $\geq 2\%$ and/or vascular proliferation, as these are associated with a somewhat less favorable clinical course [5]. We reported an additional case of atypical central neurocytoma that has never reported in our country.

Central neurocytoma is a rare and a relative benign intracranial neoplasm, but little is known about the biological behavior of this tumor. Proliferative activity of central neurocytoma was investigated in recent years and revealed a variable Ki-67 labeling index. “Atypical central neurocytoma” was just described in the literature in 1997 and has been noted to exhibit a Ki-67 labeling index $\geq 2\%$ and/or vascular proliferation, corresponding to WHO grade II. Atypical or malignant central neurocytoma is never reported in Asia. In the present report we document an additional case of 33-year old man with a huge intraventricular central neurocytoma having high Ki-67 staining index and distinct mitotic figures. On light microscopy, the tumor composed of small monotonous cells with uniform round nuclei, inconspicuous nucleoli and a fine chromatin pattern in a well-vascularized stroma. Occasionally perivascular pseudorosettes and perinuclear halos with areas of

calcification are seen. The characterized immunoreactive for both synaptophysin and neuron specific enolase and the ultrastructural features of neuronal differentiation distinguished it from ependymoma and oligodendroglioma. The mitotic activity (up to 3 mitoses/10HPF) and high percentage of Ki-67 staining tumor cells (labeling index 5) of our case fitted the atypical variant of central neurocytoma. The literature and the clinicopathological manifestations are reviewed and discussed.

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Comparative Pathology Case 104

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Clinical history: A 12-month-old female specific pathogen free (SPF) Sprague-Dawley, CD (Crj:[SD] BR) rat showed abdominal respiration, dyspnea, and finally death.

Diagnosis: Cardiac schwannoma, malignant, heart, SD rat.

Synonyms: Endocardial schwannoma, neurofibroma, neurinoma, neurilemmoma, Antischkow cell sarcoma, polymorphous sarcoma, endocardial fibromatous proliferation, endomyocardial disease, endocardial mesenchymal tumor, cardiac fibrosarcoma, cardiac spindle cell tumor, neurosarcoma.

Gross findings: When necropsied, a large ovoid firm, grayish-yellow tissues that partially fill one or more cardiac chambers, diffusely invade the myocardium, and expand as an exophytic mass on the external surface of the heart. Other organs, including lung, liver, kidney, spleen, uterus et. al. are grossly normal.

Histopathological findings: The tumors are nonencapsulated, poorly circumscribed,

composed of spindle shaped and polyhedral cells with elongated to large bizarre, pleomorphic plump ovoid nuclei.

These cells are arranged in sheets, wavy bands, whorls, streams and separated by abundant eosinophilic fibrous matrix and intermingled with adipose tissues. The original myocardial architectures are almost missing.

In areas, glandular growth patterns are characterized by cells occurring in nests, tubules, and papillary formations within a variable amount of eosinophilic fibrous stroma. The lining neoplastic cells are simple squamous to large, vesicular, plump cuboidal and lumen containing granular necrotic debris.

Other organs, including lung, liver, kidney, spleen, cerebrum, hippocampus show no significant lesions.

Immunocytochemistry results: Neoplastic cells did not stain for NF-200, MBP, Factor-8, Laminin, CD-68, Desmin; strong staining for vimentin, moderate staining for S-100 and neuron-specific enolase (NSE).

Histochemistry results: Variable degrees of Trichrome staining was observed in the neoplastic fibrous stroma, in which Alcian blue staining was negative.

Electron microscopic findings: In the present case, the neoplastic cells show prominent irregular, interdigitating nuclei and variable cytoplasm containing mitochondria, RER, and occasional filamentous body.

Bundle of collagen fibers and thin cell processes are demonstrable between cells, a few of which are enveloped by a single layer of basal lamina with desmosomes.

Discussion: Cardiac (endocardial vs intramural) schwannoma in rat had been called a variety of names. One reason for this plethora of names is the lack of agreement on the histogenesis of the tumor. Another reason is several pathognomonic features of schwannomas which are absent (Verocay bodies, pigmented areas etc).

This tumor is the most common tumor in most rat strain, is very uncommon in man and other domestic animals. Tumors diagnosed as cardiac neurofibromas are very rare in man and are generally associated with von Recklinghausen's disease.

In human, primary tumor of the heart are rare. The most common tumors are myxomas, fibromas, lipomas, papillary fibroelastomas, rhabdomyomas,

angiosarcomas and other sarcoma.

However, in the present case, the typical Antoni type A (schwann cells arranged in rhythmical structures, sometimes forming Verocay bodies) and Antoni type B patterns (schwann cells arranged in a reticular pattern) with cysts are not apparent.

The tumor appear to have a dual or triple character, sharing some features with fibromatous proliferations (neurofibroma), glandular patterns lined by lightly stained cuboidal cells and filled with necrotic debris (mesothelioma), bizarre pleomorphic nuclei in myxoid matrix (similar to Antoni B pattern). These features along with basal lamina, desmosomes and presence of S-100 and NSE immunoreactivity are in favor of the diagnosis of cardiac schwannoma.

The immunohistochemical study of canine schwannoma show that staining positive percentage in decreasing order was vimentin (85.7%), NSE (35.7%), S-100 (21.4%) and GFAP (21.4%), MBP and MAK-6 (0%). Also in rats, anaplastic schwannoma, consist of little or no Antoni type A tissue and contain less S-100 protein.

The most distinctive ultrastructural features of schwannoma are the long cell processes and pericytoplasmic basal laminae. However, these features are often not well developed in malignant form. The differential diagnosis in this case showed consider:

- a) Peripheral nerve sheath tumor (PNST) in rat: The majority of PNST occur in subcutaneous tissues, especially of the ear, neck, head, and shoulder, sometimes eye, spinal nerve roots, adrenal capsule, salivary gland and uterus.
The PNST in rat delineate four main types: neurinoma, neurofibroma, plexiform neurofibroma, neurofibrosarcoma. Neurinoma (schwannoma) are tumors of the PNS characterized by the exclusive proliferation of the schwann cells. In contrast, neurofibromas are tumors of the PNS characterized by the combined proliferation of all of the cellular elements of a peripheral nerve: schwann cells, fibroblasts and perineurial cells. These two have different histogenetic derivation.
- b) Mesothelioma: Atriocaval and pericardial mesothelioma are characterized by tubular and alveolar structures with debris in the lumen. The former is found in the right atrium, more frequently in its outer wall, sometimes in the inferior vena cava. The latter occurs as pedunculated nodules, and thicken the pericardium.
- c) Endocardial and Intramural schwannoma: The prevalence is about 0.07% vs 0.03% in F344 rat. It is a firm white tumor that thicken the endocardium and may form

discrete masses. Cells with Anitschkow nuclear pattern are commonly observed. In contrast, intramural schwannoma usually diagnosed as fibroma or neurilemoma. This neoplasm is described only in the rat. It occurs in the intraventricular septum and right and left ventricle.

The natural history of this lesion are not fully understood.

Diagnostic criteria:

1. Compact cellular growth (Antoni type A)
2. Less loose myxoid tissues (Antoni type B)
3. Palisading nuclei of the neoplastic spindle cells
4. Verocay bodies
5. Hyalinization of blood vessels (Human, dog)
6. Ultrastructural features: with continuous basal lamina, cytoplasmic processes, pinocytotic vesicles, long-spacing collagens
7. Immunohistochemical staining-S-100 (alpha unit), Vimentin, Laminin, NSE, GFAP, MBP.

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Comparative Pathology Case 105

Contributors: Chien-Jui Cheng (鄭建睿), MD; Min-Shin Kuo (郭明勳), MD;

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Clinical history: This 55-year-old farmer was a victim of diabetes for years without regular control. He got upper respiratory tract infection 2 weeks prior to this admission with rhinorrhea, cough and fever. No obvious necrotizing mucosa is noted within the nasal cavity. Painful swelling at right orbital area and droop of eyelid were found one week later. The acute right paranasal sinusitis and right orbital cellulitis were suspected and treated. Sudden conscious change was noted one day later. The brain CT revealed multiple cerebral infarction. Septic embolization was suspected. The patient's condition worsened and developed multiple new brain infarctions with subsequent days. The patient expired on 36th day of admission.

Diagnosis: 1. Mucormycosis, rhinocerebral type with multiple cerebral septic infarction.
2. Diabetes mellitus

Gross finding: The brain taken from the autopsy reveals multiple adhesion area at right parieto-temporal area, including the temporal tip and right cerebellum area. The cribriform plate and skull base are not destructive without local abscess formation grossly. The sella turcica is markedly adhesive with fibrinopurulent exudate. The pituitary gland is embedded within the fibrotic tissue within the sella turcica. On the cut surface, one large cystic lesion containing necrotic tissue, measuring up to 5.0 × 4.0 cm. in largest dimension, extending from the right frontal area into the temporal area. Some thrombi are found within the basilar artery and middle cerebral arteries.

Histopathological findings: The specimen from the right nasal cavity reveals fibrosis

with mononuclear cells infiltration. Within it, there are some separated, broad, nonseptate hyphae within fibrotic stroma. The Grocott's Methenamine silver (GMS) stain demonstrates the fungus hyphae within the fibrotic stroma. Subsequent specimen from the right parietal lobe of brain reveals some fungus hyphae within the necrotic area. The fungus hyphae are also seen within the necrotic area of right brain tissue and sphenoid bone in the autopsy specimen. Some fungus hyphae are also found within the blood clot of middle cerebral artery.

Histochemistry results: The Grocott's Methenamine silver (GMS) stain shows broad, nonseptate hyphae invading tissue and within the thrombus of middle cerebral arteries.

Discussion: The fungus responsible for mucormycosis belongs to the genera *Mucor*, *Rhizops* and *Absidia*, which are members of the family Mucoraceae, which belongs to the class Phycomycetes. Therefore, infections from these organisms can be termed phycomycoses.

These are four major types of mucormycosis: rhinocerebral, pulmonary, gastrointestinal and disseminated. The most common form is rhinocerebral, and this is most commonly associated with uncontrolled diabetes. Other disease, such as acute leukemia and lymphoma could predispose the infection.

On the rhinocerebral form, the initial presentation reveals sudden onset of facial pain, headache, rhinitis with epistaxis, lid edema, ptosis, chemosis and unilateral blurr vision. Later, the common initial findings of rhinocerebral mucormycosis have been lethargy, headache and visual loss. Loss of ocular movement and periorbital cellulitis frequently follows. Abramson and co-workers postulates that the early visual loss and evidence of retinal artery occlusion of orbital mucormycosis are not found in pyogenic cavernous sinus thrombosis, and therefore help differentiate the two syndromes. In fact, cavernous sinus thrombosis and internal carotid artery thrombosis are frequent complication in rhinocerebral mucormycosis. Then orbital involvement progresses, loss of function of the second, third, fourth and six cranial nerves may occur with resultant proptosis, ptosis, pupillary dilatation and visual loss. The fifth and seventh nerves may become involved later.

The infection begins in the nose and spreads to the paranasal sinuses. The organism presumably enters the nasal cavity and paranasal sinusitis in inhaled dust particles. It sporulated in tissue that has lost their natural resistance to this fungus.

Hyphae are produced and blood vessels are invaded. The organism proliferates within the muscular walls of arteries, and to lesser extent vein and lymphatics, producing purulent arteritis, thrombosis and tissue necrosis. Black necrosis of a turbinate is a diagnostic clinical sign, but may not be present until late in course of the disease. From the sinuses, the disease spreads to the medial orbit and the orbital apex through the intravascular, perineural and direct soft tissue invasion through the nasolacrimal duct, resulting in local infarction and suppuration. The regional anesthesia and orbital cellulitis are resulted. The clinical and pathological pictures could help to differentiate the fungus infection from the local malignancy or orbital cellulitis.

The brain is involved often presenting as multiple cerebral infarctions. Coagulation necrosis may also occur, most frequently in frontal lobes. Pituitary involvement is not infrequent. Invasion of the cavernous sinus may lead to thrombosis or hemorrhage of and to cavernous sinus thrombus. Even in the presence of brain involvement, the cerebrospinal fluid findings are usually non-specific. In some patients, the cerebrospinal fluid has been normal. In others, cerebrospinal fluid abnormalities have included slightly increased cerebrospinal fluid pressure, modest pleocytosis with about 50% polymorphonuclear cells and slight protein elevation.

The diagnosis of rhinocerebral mucormycosis is dependent on the biopsy specimen showing broad, nonseptate hyphae invading tissue. The pathologic trademark of mucormycosis is invasion of the walls of vessels, particularly arteries. Lowe and Hudson described hyphae in the walls of occluded internal carotid arteries in all cases they examined. Thrombosis probably results from endothelial damage caused by the invading hyphae, but the growth of the hyphae into the lumina of vessels may also contribute to thrombosis. Definitive diagnosis could be delayed in some cases in which the CT findings were suggestive of mucormycosis, but the reluctance to biopsy resulted because the typical clinical signs, such as the black mucosal lesion, were lacking, or because the patient did not seem "sick enough" to have this disease.

The importance of early diagnosis and treatment of the disease cannot be overemphasized. Death occurs within one to several weeks in nearly all untreated cases or cases treated late.

Differential diagnosis:

1. Local malignancy

2. Orbital cellulitis

Treatment:

1. Control the blood sugar
2. Intravascular use of Amphotericin B for mucormycosis

Diagnostic criteria:

1. Presence of fungus hyphae within the brain parenchyma and supplying arteries
2. Liquefaction necrosis of brain parenchyma in area of thrombotic arteries supply
3. Presence of same fungus hyphae within the sinus mucosa

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Comparative Pathology Case 106

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Clinical history: A 1 to 2-month-old male, mixed dog was admitted to the hospital with coma on 11/30/1997. After emergency treatment, he got improvement and was discharged with continuing medicine administration. On 12/4, the dog was submitted again with coma, corneal edema, meiosis, and frequent urination. Routine urinalysis did not reveal increased level of sugar. He had hematuria and anemia. Blood transfusion was performed on 12/7, but poor response to the treatment. On palpation, he felt pain. The dog expired on 12/9.

Diagnosis: Parasitic meningoencephalitis, caused by *Toxocara canis* larvae migration

Gross findings: Both eyes had corneal edema. In the right side of neck, there was a 3 × 3 cm hematoma in the subcutis. Hemorrhage was observed in the retroperitoneum, lungs, stomach, and joints of the right rear leg. The stomach and small intestines contained dark-red watery fluid. Locally extensive hemorrhages were markedly present in the serosa of urinary bladder. Congestion and hemorrhage were seen in the brain.

Histopathological findings: There were several parasitic sections in the leptomeninges and brain parenchyma, the former was congested and had focal hemorrhages. The characteristic features of the parasite, including lateral alae and colemyrian muscle, suggested they were *Toxocara canis* larvae. Some larvae were arrested with association of granulomatous inflammation or hemorrhage around them. The ependymal cells around the lateral ventricle were destroyed and mixed with a few

inflammatory cells. Microglial nodules and hemorrhagic foci were occasionally detected throughout the cerebellum and medulla oblongata. Besides, bacterial colonies within the blood vessels and postmortem change were noticed. The lesions described above were variably seen in submitted sections.

Discussion: *Toxocara canis* is the most prevalent parasite of dogs worldwide. It usually occurs in the small intestine of dogs and foxes and other Canidae. Heavy infection is most often seen in young puppies, in which the adult ascarids may cause abdominal discomfort, whimpering and groaning, potbellied appearance, dull haircoat, unthriftiness, stunted growth, and diarrhea. Occasionally, large tangled mass of ascarids occludes the intestinal lumen and cause death from intestinal obstruction, intussusception, or perforation. Dogs become infected with *Toxocara canis* by ingestion of infective eggs, ingestion of larvae in tissues of paratenic hosts (mice, birds, pigs, earthworms, and others), transplacental migration, or transmammary passage through milk. After infection, the life histories may follow the following diaphragm:

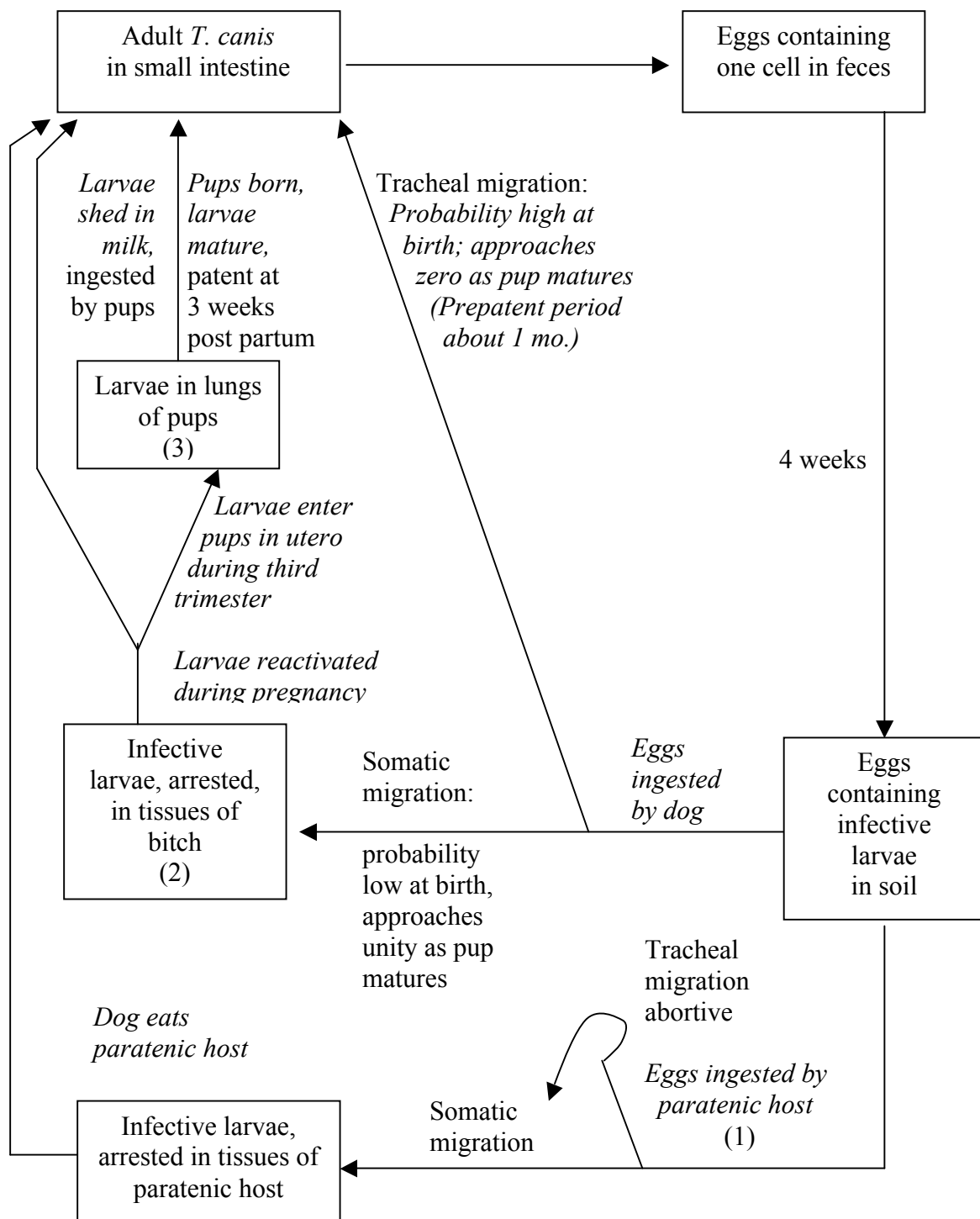


Figure 3-114. Alternative life histories of *Toxocara canis*. (1) A *paratenic host* is any in which a larval parasite may survive and remain infective for its definitive host without undergoing development. Any of a wide range of animal species including rodents, sheep, pig, monkey, man, earthworm, and adult dog may serve as paratenic host for *T. canis* larvae. (2) Arrested infective larvae are also found in the tissues of male dogs, but these are supposed to be of little if any epidemiological importance. (3) The larvae that have entered the pups through the placenta molt once in the fetuses but defer further development until after birth (Sprent, 1958).

Ascarid larvae may wander throughout the body and produce granulomatous inflammation in many sites. They are most commonly encountered in the kidney, but may localize in any tissues, including the liver, lung, heart, brain, eye, and lymph nodes. Since widespread distribution of dog feces and the prevalence of *Toxocara canis* eggs, people (particularly the children) may become infected when they ingest infective *Toxocara canis* eggs in the soil contaminated by feces from infected animals. Two distinct forms of disease in human produced by *Toxocara canis* larval migration are visceral larva migrans (VLM) and ocular larva migrans (OLM). The few human fatal cases have been attributed from extensive involvement of the myocardium or central nervous system or from an exaggerated immunologic response. Therefore, human toxocariasis can be prevented by careful personal hygiene, eliminating intestinal parasites of dogs and cats, and not allowing the children to play in potentially contaminated environment.

Clinical signs of Toxocaral larva migrans in human and dogs

	Human	Dogs
Clinical signs	<ul style="list-style-type: none"> - asymptotic - fever - leukocytosis, persistent eosinophila - hypergammaglobulinemia - bronchiolitis, asthma, pneumonia - myositis, arthralgia - hepatomegaly - splenomegaly - abdominal and chest pain - nervous system signs - uveitis, endophthalmitis 	<ul style="list-style-type: none"> - asymptomatic - symptoms with affected visceral organs

Diagnostic criteria:

1. Histologic sections reveal characteristic features of *Toxocara canis* larva
2. History of ascarid infection

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Comparative Pathology Case 107

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Abstract of clinical history: A 31-year-old man, suffered from fever and headache off and on for 5 months. The patient was homosexual and known to be HIV (+) for 5 years. CT scan of brain revealed a progressively enlarging hyperdense tumor in the right frontal lobe, 7.0x6.2 cm, with peripheral rim enhancement. Under the impression of “right frontal lesion, R/O Toxoplasmosis”, stereotactic needle biopsy was performed in June '96, which revealed reactive gliosis and mild perivascular lymphocytic infiltrate. No specific organisms were found. Tumor excision was performed in Sept. '96. The slides presented are from right frontal tumor excision.

Clinical history: The 31-year-old male patient suffered from fever and headache off and on for 5 months. The patient was homosexual since high school age and known to be HIV (+) for 5 years. Antiretroviral therapy with AZT was maintained at 性病防治所. Episodes of fever, cough or diarrhea bothered him frequently in recent one year. The patient complained of fever and headache since 5 months before this admission. CT scan in June 1996 revealed a right frontal lobe hyperdense tumor with peripheral rim enhancement and central low density, measuring 5.8x4.0 cm. Stereotactic needle biopsy under the impression of “right frontal lesion, R/O Toxoplasmosis” revealed reactive gliosis and mild perivascular lymphocytic infiltrate. No specific organisms were found. Drowsy consciousness and mutism developed in September 1996. CT scan revealed that the tumor progressed in size, up to 7.3x5.5 cm. Tumor excision was performed in Sept. '96. Radiation therapy of a total dosage of 5400cGy was delivered 3 weeks after surgery. From December 1996, the patient suffered from blurred vision due to CMV retinitis, which responded very poorly to aggressive therapy with Gancyclovir. Cocktail therapy for AIDS began from June 1997. Acute onset of seizure and loss of consciousness without focal sign occurred in July 1997. CT scan revealed tissue loss with fluid accumulation of right frontal area, and a suspicious

hypodense lesion of white matter in the right frontal lobe, R/O infarction, R/O tumor recurrence. The patient expired 5 days after the last admission.

Diagnosis: 1. Primary cerebral malignant lymphoma, diffuse, large cell type, of B-cell lineage.
2. Acquired immune deficiency syndrome.

Gross findings: The specimen of right frontal tumor excision consisted of multiple grayish tan and elastic soft tissue fragments, weighing 74.2 gm. Cut surface revealed grayish tan solid tissue with focal whitish to yellowish discoloration.

Histopathological findings: The cerebral tumor is composed of markedly pleomorphic polygonal to oval cancer cells in sheets, which are characterized by markedly pleomorphic and hyperchromatic nuclei with coarse chromatin clumping, frequently convoluted nuclear profile, and prominent nucleoli. Uninucleated or multinucleated bizarre tumor giant cells are encountered frequently. Multiple irregular-shaped foci of tumor necrosis and frequent mitotic figures are noted.

Histochemistry result:

1. Reticulin stain: abundant and dense reticulin meshwork distributed rather evenly, focally leaving islands of cancer cells free of reticulin.
2. Acid-fast, periodic-acid-Schiff (PAS), and Giemsa stains: all non-revealing.

Immunohistochemistry result:

1. The majority of cancer cells are positive for vimentin, CD20 (L26), and CD45 (LCA).
2. Scattered cells positive for glial fibrillary acid protein (GFAP), S-100, epithelial membrane antigen (EMA) and CD45 RO (UCHL-1) are also noted.

Discussion: Primary central nervous system lymphoma (PCNSL) is a rare tumor, forming about 1% of all the intracranial tumors and only 2% of all lymphomas before the introduction of transplant surgery and the beginning of AIDS epidemic. Recently, the figures ascend to 2.2% of all intracranial neoplasms. It seems that PCNSLs are frequent complications of HIV infections, ranging from 3.4% to 8.0% in the USA.

Before the epidemic of AIDS, PCNSLs are tumors of adults, most commonly occurring in the sixth decade of life. But the mean age declines to 38 years in HIV (+) population.

Clinically, the presenting signs or symptoms, in order of decreasing frequency, include: personality change, cerebellar signs, headache, seizures, motor dysfunction and visual changes. A typical CT scan reveals hyperdense masses in precontrast study, and diffuse enhancement on administration of contrast media. Sometimes, extensive central necrosis, which is most commonly encountered in AIDS-related cases, may lead to a ring-enhancing radiologic appearance indistinguishable from *Toxoplasma* abscesses.

PCNSLs may occur anywhere in the CNS. Roughly, 75-80% are supratentorial and 42% involves frontal lobes. Most of the remainder involve the cerebellum or brainstem, only rare examples being isolated to the spinal cord. The localization of PCNSLs may include four patterns: (1) discrete nodules, solitary or multiple; (2) diffuse meningeal or periventricular; (3) uveal or vitreous deposits; (4) localized intradural spinal growth. In contrast to secondary intracranial involvement of systemic lymphoma, which frequently involves leptomeninges, PCNSLs usually arise within brain parenchyma, especially in the subcortical white matter. Only about 5% PCNSLs present as diffuse leptomeningeal infiltrates without demonstrable intraparenchymal lesions. While more than 60% of PCNSLs are solitary in sporadic cases, the vast majority of AIDS-associated cases are multicentric.

Microscopically, although not apparent in all cases, a vasocentric growth pattern with infiltration of vessel walls and Virchow-Robin spaces is common to PCNSLs. The vast majority of PCNSLs are of B cell origin, and usually corresponding to diffuse large B-cell lymphoma of REAL classification, although all major cytologic variants have been reported. Primary T cell lymphomas of CNS are extremely rare, though have been reported, and have a propensity to affect the leptomeninges. For the 99 PCNSLs of Massachusetts General Hospital from 1958 to 1989, 89% were high grade, all being diffuse in architecture, and 77% were large cell subtype. Of 41 tumors studied immunohistochemically, all except one are B cell types.

Presence of Epstein-Barr virus (EBV) nucleic acid sequences in PCNSLs has been detected in more than 80%. In one study of 85 HIV-infected patient with neurological disorders, EBV nucleic acid sequences, detected by nested PCR method, are found in CSF of all 17 cases of AIDS-related PCNSL (100%), while only in 1 of 68 (1.5%)

HIV-infected patients without histologically detectable lymphoma at necropsy. Besides, HIV was rarely detected in tissue of AIDS-related PCNSLs. Therefore, EBV may play a very important role in pathogenesis of PCNSLs, and may be useful as a diagnostic tumor marker.

The clinical course of PCNSLs is usually very rapid and aggressive. The median survival time for AIDS-associated cases is only 2 to 3 months, in comparison to 10-18 months in non-HIV patients. Radiotherapy complemented with chemotherapy may prolong the survival, although the prognosis is still dismal in most cases.

Differential diagnosis:

1. undifferentiated carcinoma.
2. anaplastic oligodendroglioma.
3. metastatic amelanotic melanomas.
4. neuroectodermal tumor.
5. astrocytoma or small cell astrocytoma.
6. medulloblastoma.
7. Toxoplasma or Aspergillus encephalitis.
8. autoimmune arteritides, eg. Polyarteritis nodosa, CNS lupus erythematosus, granulomatous angiitis, etc.

Treatment:

1. Irradiation therapy complemented with chemotherapy (intravenous or intra-arterial methotrexate) is the first choice.
2. Surgical intervention is usually restricted to biopsy.
3. Aggressive treatment is not recommended for AIDS-associated lymphomas, though whole brain irradiation seems to be beneficial.

Diagnostic criteria:

1. The histopathological features of PCNSLs are quite similar to those of systemic non-Hodgkin's lymphomas, except that follicular growth pattern is rare in PCNSLs.
2. The most striking feature is the angiocentric growth pattern: neoplastic lymphoid cells infiltrating the brain around blood vessels, esp. small arteries, arterioles or venules, with concentric layers of reticulin fibers in the perivascular infiltrate.
3. Immunohistochemistry may be a great aid to the diagnosis.

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Comparative Pathology Case 108

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Clinical history: A dairy goat farm reared about 85 ewes, 35 rams and 20 2-month-old lambs in Pingtung prefecture. One of the lambs showed signs of depression, and decreased appetite for ten days in December. These prodromal signs were followed by incoordination and hemiparesis with a tendency for the lamb to lean, stumble, or move in one direction only. This tendency progressed to obvious torticollis and circling to the right side direction repeatedly when forced to move. Later, the lamb became exhausted and recumbent with the head pulled tightly into the flank, unable to straighten the neck voluntarily. The lamb also showed facial nerve paralysis with drooped ear, swelling of the left cheek, and drooling. The owner treated the lamb with Procaine Penicillin G and Dexamethasone, but poor responses. The owner sent the affected lamb to our laboratories for pathological diagnosis after our visit. Three lambs, 2-3 months old, also occurred the circling signs in early summer time two years ago and died within one week.

Diagnosis: Listeric encephalitis in lamb

Gross findings: The lamb was emaciated with rough hair and showed nervous signs with circling movement. No gross visible lesions were found.

Histopathological findings: Microscopically, the pathognomonic histological lesions which were predominantly most severe in brain stem, particularly in pons and medulla oblongata. The characteristic lesion was a focus of inflammatory cells with adjacent perivascular cuffs consisting predominantly of lymphocytes with histiocytes, and plasma cells. Large areas of inflammation with glia nodules, liquefied

neuroparenchyma filled with gitter cells and neuronal degeneration were evident. Cerebrum and cerebellum revealed a mild lymphocytic meningitis. No histopathological lesions in cervical, thoracic, lumbar spinal cords were noticed.

Microbiological examinations: Smears from brain stem lesions revealed Gram-positive rods. A “cold-enrichment” procedure was necessary for brain tissue from neural listeriosis. Small pieces of medulla oblongata were homogenized and a 10 percent suspension was made in brain heart infusion broth (Difco). The broth suspension was placed in the refrigerator at 4°C and subcultured onto McBride listeria agar (Difco) with 5% sheep blood once weekly. Small greyish-white colonies with narrow zones of beta-haemolysis appeared on agar in 24 hours. The colonies subcultured onto the Tryptic soy agar (Difco) with 5% sheep blood and Horse blood overlay medium. Small and round colonies with beta-haemolysis also appeared on agar in 24 hours. The colonies again subcultured onto the Oxford medium base (Difco). Small and round colonies with black ring zone in rim also appeared on agar in 24 hours. The biochemical and other tests of the *Listeria* spp. were as followed: positive reaction for catalase, positive reaction for umbrella motility, CAMP test positive with *Staphylococcus aureus* and negative with *Rhodococcus equi*, fermentation of Rhamnose, negative reaction for nitrate reduction, positive reaction for esculin hydrolysis, positive reaction for VP test, positive reaction for Listeria latex slide agglutination test. API-*Listeria* (bioM'erieux) and Micro-ID *Listeria* (Medvet) identification system identified the *Listeria monocytogenes* spp.

Animal inoculation: Anton test: inoculation of the conjunctiva of a rabbit or guinea-pig. *L. monocytogenes* caused a purulent keratoconjunctivitis within 24-48 hours of inoculation.

Discussion: Listeric infections, caused by microorganisms of the genus *Listeria*, occurred worldwide and in a variety of animals including man. Listeriosis was first recognized as a disease of animals and although the link between silage feeding and infection in farm animals has been known for decades it was the recognition in the 1980s of listeriosis as a food-borne human disease. Two pathogenic species exist in the genus *Listeria*, *L. monocytogenes* and *L. ivanovii*, and 16 serovars. Most clinical

infections are caused by *L. monocytogenes* serovar 1/2a, 1/2b and 4b. Phage typing, pyrolysis mass spectrometry, multilocus enzyme electrophoresis, randomly amplified polymorphic DNA, and automated ribotyping system have been used to type *L. monocytogenes* strains. It is an exquisitely adaptable environmental bacterium capable of existing both as an animal pathogen and plant saprophyte with a powerful array of regulated virulence factors. Most cases of listeriosis arise from the ingestion of contaminated food and in the UK the disease is particularly common in ruminants fed on silage. Although a number of forms of listeriosis are easily recognized, such as encephalitis, abortion, and septicemia, the epidemiological aspect and pathogenesis of infection in ruminants remain poorly understood. A number of conditions are associated with infection with encephalitis and uterine infections being most frequently identified. However, it is the exception for different listeric conditions to occur in the same flock or herd.

The first description of listeric encephalitis was as a widespread neurological disease of sheep in New Zealand known locally as “circling disease”. *L. monocytogenes* was isolated from the lesions and listeric encephalitis of sheep, cattle, and goat, almost invariable associated with *L. monocytogenes* infection, has since been described throughout the world. The clinical signs of infection are a consequence of the lesions in the brain stem. In sheep and goats recumbency and death occur within 2 or 3 days but in cattle the duration of illness is often longer. Gross pathological lesions of the brain are rarely observed but there are pathognomonic histological lesions which are predominantly unilateral and consistently most severe in medulla oblongata and pons. Lesions are less frequent in the cerebellum, cervical spinal cord and diencephalon, and when present in these sites are of lesser severity. The characteristic lesion is a focus of inflammatory cell with adjacent perivascular cuffs consisting predominantly of lymphocytes with histiocytes, plasma cells and occasional neutrophils in severe cases lesions may coalesce to affect large areas of brain tissue. Meningitis is often present, developing secondary to the parenchymal lesions, but the ependyma and choroid plexuses are rarely affected. Neurologic diseases that can produce localizing signs typical of listeriosis include the neurologic form of CAE, focal brain abscesses, parasitic encephalitis, middle ear infection, bacterial meningitis, early rabies, and trauma to the facial nerve. In the present case, according to clinical signs and histopathological lesions, Listeric encephalitis is the first priority of diagnosis. No experimental study has conclusively

defined the pathogenesis of listeric encephalitis and it must be appreciated that the experimental direct injection of *L. monocytogenes* into the tooth pulp. The facial or trigeminal nerve of sheep does not exclude the possibility of infection arising via haematogenous spread.

Listeric abortion caused by *L. monocytogenes* occurs in ruminants and many other species of domesticated animal. *L. ivannovii* is also recorded as a cause of abortion in sheep and cattle but occurs less frequently than *L. monocytogenes*. Placental lesions are pin-point, yellowish, necrotic foci involving the tips of the cotyledonary villi with a focal to diffuse intercotyledonary placentitis covered in a red/brown exudate. The foetus is usually autolytic and with miliary necrotic foci occasionally visible throughout the liver and spleen. Histologically, these foci show coagulative necrosis and infiltration to variable degrees by macrophages and neutrophils.

Septicemia is relatively uncommon and generally, although not invariable, occurs in the neonate as an extension of intrauterine infection. The most consistent lesion is focal hepatic necrosis with pinpoint greyish, white nodules throughout the liver. Lesions are present also in the spleen but rarely in other tissues. The histological appearance is of multiple, focal area of necrosis with invasion by polymorphs and mononuclear cells. Occasional massive outbreaks of septicemia involving pregnant ewes have been described, with clinically affected animal being pyrexia and showing profuse diarrhea. In adult sheep, a marked enteritis with extensive haemorrhage affecting the abomasal folds, ulceration of the abomasal and intestinal mucosa and abscessation of Peyer's patches as a result of infection by *L. monocytogenes* has been recognized. The differential diagnosis for septicemic listeriosis, particularly when diarrhea is present, includes salmonellosis, yersiniosis, and enterotoxemia. When weakness predominates and diarrhea is absent, milk fever, and pregnancy toxemia should be ruled out.

Iritis and keratoconjunctivitis caused by *L. monocytogenes* have been recorded occasionally in both cattle and sheep. Bovine mastitis has been reported but with few documented cases. In monogastric animals, listeriosis is uncommon although septicemia and meningoencephalitis have been reported. Infection in birds causes a septicemia and myocardial necrosis. Infection of rabbits with sublethal doses characteristically induces a marked monocytosis and laboratory animals and rodents a septicemia.

L. monocytogenes was first described as a human pathogen in the 1920s. Although relatively rare, human listeriosis is often severe and mortality rates may approach 50%. Infection may present as meningitis or, more rarely, encephalitis. It can also cause a generalized infection or in pregnant women can result in spontaneous abortion, stillbirth or infection of the newborn. Minor skin infections, particularly affecting farmer or veterinarians after handling bovine calvings or abortions, are also recognized. The infection dose is unknown but is likely to be high, and host susceptibility is an important factor in infection since most cases of invasive disease involve the immunosuppressed, the elderly or pregnant. Other, less frequent, predisposing factors include alcoholism, cirrhosis, hemochromatosis, ulcerative colitis, asthma, AIDS, and other immunologic conditions. Neonates acquire infection *in utero* from the mother, resulting in stillbirth, septicemia, or meningitis. During pregnancy, infections have occurred most often in the third trimester. Patients usually have a transient illness with nonspecific, mild-to moderate symptoms, including fever, headache, vomiting, possible other GI complaints, and back pain. Other documented focal infections have included purulent conjunctivitis, lymphadenitis, endocarditis, arthritis, osteomyelitis, brain abscess, peritonitis, and cholecystitis. Pneumonitis has been reported in immunosuppressed patients. During the 1980s and early 1990s a number of listeriosis outbreak were linked with consumption of contaminated foodstuffs. Nosocomial infections and person-to-person spread are recognized but uncommon and there is general agreement that food-borne transmission is the predominant means of infection for sporadic cases which form the majority of listeriosis cases. The World Health Organization reports that animals are not important as direct sources of human infection. Recent studies have confirmed the presence of *L. monocytogenes* in a wide variety of foodstuffs. Milk, soft cheese, ice cream, cook-chill foods, raw meats, read-to-eat poultry, pates, unprocessed vegetables, salads, raw fish, fish products, sandwiches and fried rice were amongst those foodstuffs shown to be contaminated. Pates and soft cheese were particularly likely to contain more than 1000 organism per gram. Many of the problems of contaminated food products are due to post-processing contamination.

Diagnostic criteria:

1. In listeric encephalitis, There were pathognomonic histological lesions which were

predominantly most severe in brain stem, particularly in pons and medulla oblongata. The characteristic lesion was a focus of inflammatory cells with adjacent perivascular cuffs consisting predominantly of lymphocytes with histiocytes, and plasma cells. Large areas of inflammation with glia nodules were evident. Clinically, the affected goats with nervous signs, especially circling movement should be highly suspected.

2. Culture of *L. monocytogenes* should be attempted for definitive diagnosis, recognizing that the organism may need special enrichment methods to isolate. Different forms of listeriosis need suitable specimens submitted for culture.
3. Demonstration of the infectious agent or its products in tissues or body fluids.
4. Detection of a specific immune response.

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Comparative Pathology Case 109

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Clinical history: A 2-year-old girl suffered from focal seizure attack on 85/7/21. Brain CT and MRI revealed a brain tumor with cystic change over right temporal lobe. Pilocytic astrocytoma was impressed. So she was referred to neurosurgical department and received operation on 85/8/13.

Diagnosis: Desmoplastic infantile ganglioglioma

Gross findings: The resected mass measuring $6.8 \times 6 \times 3.8$ cm. in size. The superficial surface is smooth. It appears well circumscribed without extension to the adjacent dura. On section, this grayish-tan, solid tumor shows cystic change.

Histopathological findings: The lobular tumor is separated by dense collagenous tissue. The neoplastic neuroepithelial cells in varying stage of differentiation exhibit heterogeneous morphology. The predominant cells have round nuclei with perinuclear halo simulating oligodendroglioma. There are clusters of ganglion-like cells intermingled with scattered bizarre or multinucleated giant cells with smudged nuclei. Foci of necrosis and increase of mitotic activity are noted. The astrocytes are mainly present in the margin within the dense fibrous matrix.

Immunohistochemical staining: The tumor is diffusely stained by neuron specific enolase. Double immunoperoxidase stains disclose filamentous staining of the neoplastic ganglion cells by neuron filament protein and a few GFAP positive astrocytes in the margin.

Discussion: The term of desmoplastic infantile ganglioglioma was first proposed by VadenBerg et al. in 1987 and is included in the revised WHO classification in 1993,

The divergent differentiation of neuronal and glial tumor cells suggests an origin from bipotential precursor cells which may be situated in the subpial granular layer of the cerebral hemisphere. Few identical cases were reported as superficial cerebral astrocytoma due to absence of ganglion cell. Therefore a general designation as desmoplastic supratentorial neuroepithelial tumor of infancy was suggested. Atypia in the form of large, bizarre, or hyperchromatic nuclei is not infrequent in the ganglion cell tumors and often appears to be degenerative in nature. Malignant transformation of the glial cells in ganglion cell tumor is rare. The histopathological diagnosis is important because most patients have favorable prognosis after surgery in spite of mitotic activity in the hypercellular tumor.

Diagnostic criteria:

1. In infancy
2. Massive size, superficial location
3. Mixed neuron-glial component with desmoplasia

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Comparative Pathology Case 110

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Clinical history: A 50-day-old weanling piglet was from a pig farm with 500 sows. Abortion appeared in the sows and nervous signs were seen in the suckling piglets and spread to weanling pigs within a few days. The piglets with nervous signs were submitted for pathological diagnosis. Grossly, there were multiple whitish spots (0.5×1 cm) scattered on the surface and cutting surface of the liver. An ulceration (0.3×1 cm to 1×2 cm) of tonsil was observed. A total loss in one month was 200, mainly weanling piglets.

Diagnosis: Pseudorabies

Gross finding: The piglets affected with pseudorabies, had lesions on the tonsil, spleen, and liver. The liver and spleen were characterized by multiple whitish, 1-2 mm necrotic foci. There were some yellowish spots with a flat greenish surface about 0.5-1.5 cm in diameter in the tonsil.

Histopathological findings:

Cerebrum: The large clumps of mononuclear cells in the pia mater consists mainly of lymphocytes, monocytes and plasma cells.

Perivascular cuffs are thick and composed of as many as six to eight layers of cells. The cuffs are chiefly made up of lymphocytes with a few neutrophils and macrophages. The endothelial cells of cuffed vessel were swollen. The neuronal change and gliosis are widely disseminated.

Liver: There were discrete foci of coagulation necrosis, randomly distributed in most lobules. Eosinophilic intranuclear inclusion body were found nearby by the necrotic foci.

Tonsil: The initial changes occurred in the subepithelial areas between the lymphoid nodule and the crypt epithelium, showing a characteristic pattern of necrosis. Some necrotic lesion also extended into the lymphoid nodule and crypt epithelium. Numerous eosinophilic intranuclear inclusion bodies within the degenerating crypt epithelium were observed.

Discussion: Pseudorabies is an acute, frequently fatal disease affecting most species of domestic and wild animals; however, man and certain apes are resistant to it. The disease is caused by a herpes virus and is characterized by a variety of clinical signs: the most prominent involve the nervous and respiratory systems. The virus (PrV) is a neurotropic and easily spreads from a peripheral viral replication site to the central nervous system (CNS) by axonal and hematogenous transmission, producing nonsuppurative trigeminal ganglionitis and meningoencephalitis.

The first outbreak of pseudorabies infection in swine was recorded in Taiwan in 1971, since then, the disease became sporadic malady in the pig farms throughout the Island. In this outbreak, the virus mainly attacked the suckling and weanling pigs which were characterized by presence of nervous disorder. Until 1974, reproductive failure of abortion and stillbirth was noticed significantly in the pregnant sows. Recent Researches indicated that viscerotropic, pneumotropic, genitotropic, neurotropic, dermatotropic strain of PrV also existed.

Histopathological lesions caused by PrV are often characterized by nonsuppurative meningoencephalomyelitis and focal areas of necrosis in a variety of tissues with formation of intranuclear inclusion bodies. Foci of necrosis were mainly found in the tonsil, liver, adrenal gland, pharynx, spleen, lymph nodes, lung, turbinate trachea and placenta.

Herpes viruses such as the herpes simplex, pseudorabies and bovine rhinotracheitis are well known for their propensity to cause recurrent infection and induce latent ganglionic infection. Pigs with natural and experimental infections of PrV might result in partial immunosuppressive effect. Therefore, the PrV infected pigs provided a

favorable condition to be complicated with Hog cholera, Actinobacillus pleuropneumonia and salmonellosis.

Clinical signs and histopathological lesions of nervous system resemble other viral diseases; therefore, laboratory confirmation is imperative when pseudorabies is suspected. Diagnostic procedures used in laboratories are usually needed, including animal inoculation, virological examination, electron microscopic examination, and fluorescent antibody techniques.

Diagnostic criteria:

1. Non-suppurative meningoencephalitis
2. Necrotic foci with intranuclear inclusion body in visceral organs
3. E.M., F.A., animal inoculation & virus isolation_

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7. Diseases of swine

Comparative Pathology Case 111

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Clinical history: A 31-year-old man suffered from severe low back pain for 2 years. The symptom was progressively worse several weeks before the admission. CT scan and MRI revealed an intradural and extramedullary tumor. The laboratory examination showed Hgb: 17 gm/dl, WBC: 15,500, and others were within normal limits. Urinalysis showed no significant finding. The neurological examination revealed consciousness: alert, cranial nerves: intact, sensory function: well preserved, motor function: muscle power 5+ of lower limbs, DTR: 2+ of four limbs, Babinski's reflex: absent, bil.

Diagnosis: Schwannoma, L1-L2 (intradural and extramedullary).

Gross findings: The specimen submitted consists of one opened cyst measured $4.5 \times 2 \times 1.8$ cm in size. Grossly, it was encapsulated, soft and gray-white. The lining of the cyst was rough, and there were focal areas of hemorrhage and myxoid change.

Histological findings:

1. Characteristic Antoni A and B structure and nuclear palisading (Verocay bodies)
2. Infiltration by foamy macrophages, vascular hyalinization and cystic change.

Immunohistochemical staining results:

1. S-100 protein: +
2. Neuron Specific Enolase (NSE): +
3. Glial Filament Acid Protein (GFAP): -
4. Epithelial Membrane Antigen (EMA): -

Discussion: Schwannomas are the most frequent variant to abut the central neuraxis,

usually presenting in adulthood as tumors of the cerebellopontine angle or lumbosacral spinal extramedullary space. Only exceptionally do these benign neoplasms afflict children. Nearly all cerebellopontine angle tumors originate in the vestibular branch of cranial nerve VIII (acoustic schwannoma or neuroma) and produce hearing loss. Schwannomas arising at spinal levels exhibit a similar predilection for sensory divisions of the neuroaxis, typically involving the posterior roots. These often assume a “dumbbell” configuration as they squeeze through adjacent intervertebral foramina and expand into the paravertebral soft tissues. Schwannomas rarely lie within the substance of the brain, can be embedded within the spinal cord proper, and may involve cranial nerves other than the acoustic. Bilateral eighth nerve examples are a defining feature of NF-2, an autosomal dominant disorder transmitted by allelic loss involving chromosome 22. Affected kindreds are prone to an assortment of neoplasms, all typified in this setting by multiplicity, that includes craniospinal schwannomas, meningiomas, and intramedullary ependymomas. Loss of heterozygosity involving chromosome 22 is also a feature of acoustic schwannomas occurring in sporadic fashion.

The schwannoma's characteristic Antoni A and B structure, nuclear palisading (Verocay bodies), infiltration by foamy macrophages, and vascular hyalinization usually suffice for its recognition; but meningiomas on occasion exhibit similar features and are the former's most frequent counterfeit. Immunohistochemical techniques and electron microscopic study may both be usefully applied to this problem. Schwannomas are characterized by diffuse cytoplasmic S-100 protein expression and pericellular immunolabeling for laminin and type IV collagen, the latter reflecting investment of their elongated cellular processes by a continuous basal lamina foreign to the typical meningiomas. Cytoplasmic expression of EMA a regular feature of meningothelial tumors-is absent from the schwannoma or restricted to normal perineurial cells incorporated into the latter's capsule and thus limited to its periphery. Admittedly, however, other researchers have described EMA-reactive neoplastic elements in the substance of some schwannomas. These are said to exhibit diffuse cytoplasmic reactivity without the cell membrane accentuation characteristic of meningothelial tumors. It is worth remembering that meningiomas only rarely present in the lumbosacral regions favored by schwannomas of the spinal roots.

Differential diagnosis:

1. Meningioma
2. Ependymoma
3. Astrocytoma

Treatment: Total laminectomy with removal of tumor.

Diagnostic criteria:

1. Characteristic Antoni A and B structure and nuclear palisading (Verocay bodies)
2. Infiltration by foamy macrophages, vascular hyalinization and cystic change.
3. Tumor cells positive for S-100 protein.

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Comparative Pathology Case 112

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Clinical history: There was a chicken farm in the central of Taiwan. The farm raised about 40,000 broilers, divided into 4 groups. 10,000 3-day-old chicks had showed signs of nervous symptoms and the morbidity was 200 per day. The owner sent 10 two-week-old chicks to NCHUVTH for necropsy and serological diagnosis at March 23. The owner said the total eliminated chicks were 1,500 and they were treated with antibiotics and vitamins but didn't work. The total eliminated chicks were 3,000 and the survived or healed still showed walked on hocks and mild ataxia at April 9 (the chickens were four-week-old).

Diagnosis: Avian encephalomyelitis of chicks

Gross findings: There was no remarkable gross finding.

Histopathological findings: Perivascular cuffing was evident within the CNS. Most cells in perivascular cuff were lymphocytes. Central chromatolysis of the neurons were found in the medulla oblongata and spinal cord. Hyperplasia of the lymphocytes aggregates scattered in the pancreas, proventricular and myocardium.

Discussion:

1. Avian encephalomyelitis virus (AEV): genus enteroviridae (family picornaviridae).
2. The gross lesions: The only gross lesions associated with AE in chicks are whitish areas (due to masses of infiltrating lymphocytes) in the muscularis of the ventriculus. These are subtle changes and require favorable conditions to be discerned. No changes have been described for infected adult birds.

3. Histopathological lesions: An important point in differential diagnosis--The peripheral nervous system is not involved.

In the CNS the lesions are those of a disseminated, nonpurulent encephalomyelitis and a ganglionitis of the dorsal root ganglia. The most frequently encountered addition is a striking perivascular infiltrate seeming to occur in all portions of the brain and spinal cord except the cerebellum, where it is confined to the nucleus (n.) cerebellaris. Infiltrating small lymphocytes may pile up several layers to form an impressive cuff.

Microgliosis occurs as diffuse and nodular aggregates. The glial lesion is seen chiefly in the cerebellar molecular layer, where it tends to be compact. A loose gliosis is usually found in the n. cerebellaris, brain stem, midbrain, and optic lobes and less often in the corpus striata. In the midbrain, two nuclei—n. rotundus and n. ovoidalis—are invariably affected with a loose microgliosis that can be considered pathognomonic. Another lesion of pathognomonic significance is central chromatolysis (axonal reaction) of the neurons in the nuclei of the brain stem, particularly those of the medulla oblongata. If several sagittal sections are made, one can always find the alteration. The dying neuron is surrounded by satellite oligodendroglia and, later, microglia phagocytize the remains; the central chromatolysis is never seen without an attending cellular reaction.

4. Lesions in the spinal cord are identical to those of the brain. Although no detailed study of the spinal cord has been made, random sampling of various levels suggests that all were involved.

The dorsal root ganglia often contain rather tight aggregates of small lymphocytes amid the neurons. The lesion is always confined to the ganglion and never enters the nerves.

In general, signs cannot be correlated with severity of lesions or distribution in the CNS.

Visceral lesions appear to be hyperplasia of the lymphocytic aggregates scattered in a random fashion throughout the bird. In the proventriculus there are normally a few small lymphocytes in the muscular wall; in AE these are obvious dense aggregates that are certainly pathognomonic. Similar lesions occur in the ventriculus muscle, but unfortunately they also occur in Marek's disease. In the pancreas, circumscribed lymphocytic follicles are normal, but in AE the number increases several times. In the myocardium and particularly the atrium there are

aggregates of lymphocytes considered to be the result of AE. However, lymphocytes in the myocardium of young chicks are not unusual; one may consider them a lesion only if they are widespread and accompanied by previously noted alterations.

There appears to be an excellent correlation between clinical signs and histopathological lesions. In one study 11% had signs but no lesions, while 8% had lesions but no signs. Later, Jungherr believed that all birds with clinical signs had histological lesions. This was based on more intensive research that in turn was based on multiple sections of brain and viscera. Experimentally inoculated chicks killed in sequential fashion invariably yield lesions 1-2 days before clinical signs. Recovered birds free from signs have CNS lesions for at least 1 wk and probably much longer.

5. Central chromatolysis: Central chromatolysis is characterized morphologically by swelling of the cell body, disappearance of Nissl bodies--which persist only at the periphery of the cell--and flattening and displacement of the nucleus to the periphery. It is seen usually in lower motor neurons (anterior horns of the spinal cord, cranial nerve nuclei), where it represents a reaction of the cell body to a lesion of the axon (axonal reaction or retrograde degeneration). Subsequent recovery of normal cell morphology or, conversely, further progression to nerve cell degeneration depends on the reversibility of the axonal lesion. Central chromatolysis maybe seen in upper motor neurons, but is then more difficult to interpret. On the one hand, the axonal lesions within the central nervous system either do not produce changes in cell body morphology or result in a simple type of atrophy (Gudden's atrophy); on the other, some disorders that do not a priori involve axonal lesions are accompanied by central chromatolysis (e.g., Wernicke's encephalopathy, pellagra encephalopathy). Neuronal chromatolysis may be so diagnosed only after comparison with the normal morphology of the appropriate nerve cell. Some nuclei (e.g., the mesencephalic nucleus of the fifth cranial nerve, Clarke's column) normally possess rounded neurons with marginated Nissl bodies.
6. Peripheral chromatolysis: Peripheral chromatolysis can be differentiated from central chromatolysis by the persistence of the Nissl bodies in the central portion, as opposed to the periphery, of the cell body. Peripheral chromatolysis is an exceptional occurrence and is usually considered to be a stage of recovery from central chromatolysis.

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Comparative Pathology Case 113

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Clinical history: A 3-year-old boy suffered from a motorcycle traffic accident on sep.22.1993. Because his Consciousness became drowsy & vomitting for several times were noted, so he was sent to LMD, then transfered to Tzu-Chi general hospital (emergency room). At there, physical examination showed neck stiffness, anisocornia and a back hematoma (5×3 cm), Cranial CT revealed hydrocephalus. Under the impression of head injury with hydrocephalus, he was admitted SICU on Oct 3.1993. The conscious state got worse 3 days later, therefore, brain CT was performed again and revealed a communicating hydrocephalus. The arterial blood gas analysis revealed respiratory acidosis, so endotracheostomy was performed with ventilator connected. Because of increased intracranial preasure (20—30mm Hg) on Oct. 12, CSF cytology was examined, but no positvie finding was noted. The clinical condition downhilled and BP dropped since Oct. 15, and the medical treatment had no obvious effect, so he was asked discharged by his family and expired on Oct.19.1993. Autopsy was performed with systemic survey of the pathologic changes.

- Diagnosis:**
1. Tuberculous meningitis, basilar, exudative, marked;
 2. Hydrocephalus and tuberculous ependymitis, post-ventriculostomy status.
 3. Encephalomalacia, basal ganglion, focal, old
 4. Hematoma, subdural and subarachnoid, bilateral, fresh.
 5. Herniation, uncal, bilateral, mild.

Gross findings: The brain showed bilateral subdural hematomas with massive

swelling. On the brain base, diffuse whitish gelatinous exudate coating was noted and suggestive of tuberculous meningitis. On serial sectioning, mild hydrocephalus with tuberculons ependymitis, periventricular and cortical swelling were noted.

Histopathological findings: Acute fibrinous exudate accompanied with neutrophil infiltration was noted in the meninx. Endarteritis obliterans was noted in the meningeal arteries with picture findings of fibrinous exudate occluding the lumen of vessels & diffuse neutrophil infiltration in the vessel walls. The complications of tuberculous meningitis also included prominent hydrocephalus (post-ventriculostomy induced periventricular edema), tuberculous ependymitis (secondary to CSF infection) and encephalomalacia of basal ganglion (due to endarteritis obliterans occluding branch of striae artery).

Histochemistry results: Acid fast stain showed evidence of tuberculosis.

Diagnosis criteria:

1. Granulomatous inflammation with prominent central caseous necrosis & Langhan's giant cell formation.
2. acid fast stain shows positive bacilli.
3. family history (patient's father is a victim of open TB)

Discussion: Tuberculosis (TB) is a chronic communicable granulomatous disease that affects the lungs & may disseminate to involve other organs. The most severe form of TB is that involving the CNS, and TB meningitis (TBM) is the most common form of TB of CNS. In countries where TB is common (Taiwan, for example), TBM can occasionally run an acute fulminating course, particularly frequent in children due to spread of infection from a primary focus. The autopsy cases in NTUH (1947~1986) revealed TBM were common during 1947~1976, but were very rare since 1977. The mycobacterium tuberculosis is highly virulent and the primary TB affects healthy (unexposed, unsensitized) persons, while reactivation of TB is a consequence of immune-depression (alcoholism, silicosis, old age, cancer patients etc.). This 3-year-old boy, owing to long-termed contact with his father (an open TB case), suffered from primary TB, but without early diagnosis, he lost the opportunity of the good response of TBM to anti-TB drugs.

The gross pathology of TBM shows a much thicker green exudate fill the basal cisterns and slight grey-green opacity of meninges over cerebral convexity. In early disease, the gelatinous exudate is most evident in the basal cisterns anteriorly. Tubercles are not easily found in the exudate and must not be confused with arachnoid granulations. On cutting the brain, hydrocephalus, swelling of white matter and infarction may be found. Histology is required to confirm a suspected diagnosis. Typical changes include granulomas with central caseous necrosis rimmed by epithelioid cells with a peripheral ring of lymphocytes. Langhans' giant cells may be noted. Endarteritis obliterans is a common complication of the healing process of TBM and results in hydrocephalus.

Rich attributed TBM to the discharge of the microorganisms from small tuberculous lesions (Rich's foci) in the meninges, brain, Spinal cord or less often in choroid plexus. Most Rich's foci arise as a consequence of bacillaemia that always follows primary infection & may also follow late reactivation of TB elsewhere in the body and activated even years later so that TB bacilli are released into the CSF to cause TBM. Rapid diagnosis of TBM is difficult and the proportions of cases in which bacilli can be seen in CSF using either Ziehl-Neelsen or auramine staining varies with the technique. Detection of specific antigens yield rapid & simple diagnostic tests of high specificity. PCR could be a rapid & sensitive method to detect MTb genome in CSF & allow a quick & specific diagnosis for TBM. PET scan in the diagnosis & localization of TBM is also reported.

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Comparative Pathology Case 114

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Clinical history: An eight-year-old, castrated male Rottweiler dog had a 3 month history of progressive growth of a midline cranial mass. The dog had progressively deteriorated and had become unable to eat and blind. The owner declined a work-up and treatment.

Radiological findings: Skull radiographs showed a large, infiltrating frontal and nasal lesion arising from the trabecular bone and eroding through the cortex. The intramedullary and soft tissue components of the mass contained foci of mineralized tumor, osteoid-producing, fluffy cumulus cloud densities, and rarefaction of underlying cranial bones. The adjacent periosteal bones were stimulated to form Codman triangles. Computal tomogram revealed rarefaction of the frontal and nasal bones. The dog was euthanatized and necropsied.

Diagnosis: Osteosarcoma of the skull with hemorrhage and necrosis in the brain

Gross findings: Blood exuded from the nostrils and a large, soft, flocculent swelling was seen over the cranial vault. An 8×11×14 cm, raised mass rose from the top of the skull. The mass was encased in a sheet of extremely vascular fibrous tissue. The peripheral portions of the mass were soft, cystic, and flocculant with multiple blood filled areas. The central and caudal portions of this mass invaded adjacent frontal and nasal bones and nasal cavity, affecting all normal architecture in these areas.

Histopathologic findings: The tumor consisted of sarcomatous spindle cells with neoplastic osteoid or osseous production. The original osseous trabeculae and dura mater were destroyed by the tumor tissue. Areas of necrotic and hemorrhagic debris

were seen in the adjacent neoplastic osseous or osteoid structure. The adjacent cerebral cortex was characterized by focal and diffusely necrosis and hemorrhage.

Discussion: Clinical, radiologic and pathologic findings of this tumor are similar to those in dogs (1), cats (2) and human patients (3) with osteosarcoma. Osteosarcoma is rare in humans (4.6 per one million population) (3). In dogs, osteosarcoma was diagnosed in 0.23% of a clinical population of dogs at The Animal Medical Center (4).

Osteosarcoma has been reported to account for 46% (1), 65% (5) and 85% (6) of all canine bone tumors. Locations include appendicular skeleton 73%, axial skeleton 27%, and skull, 8% (7). The biologic behavior of osteosarcoma of the skull is locally aggressive, but it seldom metastasizes in humans (3), dogs (7), in cats (2). Osteosarcoma of the skull in laboratory rodents is highly malignant, usually with multiple metastases in a short course (8).

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中華民國比較病理學會第一次至第十三次比較病理學研討會 病例一覽表

第一次比較病理學研討會病例（83年10月30日於台灣養豬科學研究所舉行）：

<u>動物別</u>	<u>診斷</u>	<u>提供單位</u>
1.Dog	Myxoma	美國紐約動物醫學中心
2.Ferret	Chordoma	美國紐約動物醫學中心
3.Human	Ependymoblastoma	長庚紀念醫院
4.Goat	Cryptosporidiosis	台灣養豬科學研究所
5. <i>Lemur fulvus</i>	Amoebiasis	台灣養豬科學研究所
6.Monkey	Tuberculosis	台灣大學獸醫學系
7.Human	Tuberculosis	省立新竹醫院

第二次比較病理學研討會病例（84年4月9日於台北病理中心舉行）：

8.Pigeon	Synovial sarcoma	美國紐約動物醫學中心
9.Cat	Perinephric pseudocyst	台灣大學獸醫學系
10.Human	Choledochocyst	長庚紀念醫院
11.Rat	Bile duct ligation	中興大學獸醫學系
12.Human	<i>H. pylori</i> -induced gastritis	台北病理中心
13.Human	Pseudomembranous colitis	省立新竹醫院
14.Dog	Dirofilariasis	台灣省家畜衛生試驗所
15.Human	Pulmonary dirofilariasis	台北榮民總醫院
16.Squirrel	Toxoplasmosis	台灣養豬科學研究所
17.Pig	Toxoplasmosis	屏東技術學院獸醫學系

第三次比較病理學研討會病例（84年8月27日於國立台灣大學舉行）：

18.Human	Malignant lymphoma	長庚紀念醫院
19.Wistar rat	Malignant lymphoma	國家實驗動物繁殖及研究中心
20.Human	Sparganosis	台北榮民總醫院
21.Chickens	Newcastle disease	國立台灣大學獸醫學系
22.Goldfish	Herpesvirus infection	國立台灣大學獸醫學系
23.Human	Chromomycosis	台北病理中心
24.Human	Metastatic thyroid carcinoma	省立新竹醫院
25.Human	Chordoma	新光吳火獅紀念醫院
26.Pig	Swine salmonellosis	國立中興大學獸醫學系
27.Pig	Vegetative valvular endocarditis	台灣養豬科學研究所

第四次比較病理學研討會病例（84年11月19日於新光吳火獅紀念醫院舉行）：

28. Human	Nocardiosis	台灣省立新竹醫院
29. Largemouth bass	Nocardiosis	屏東縣家畜疾病防治所
30. Dog	Demyelinating canine distemper encephalitis	台灣養豬科學研究所
31. Malayan sun bears	Adenovirus infection	國立台灣大學獸醫學系
32. Human	Actinomycosis	台灣省立豐原醫院
33. Human	Tuberculosis	苗栗頭份為恭紀念醫院
34. Dog	Interstitial cell tumor	國立中興大學獸醫學系
35. Human	Carcinoid tumor	長庚紀念醫院
36. Siamese cat	Hepatic carcinoid	美國紐約動物醫學中心
37. Human	Myositis ossificans	台北醫學院

第五次比較病理學研討會（85年 2月 4日於台北市立仁愛醫院舉行）：

中華民國比較病理學會成立大會暨專題演講

第六次比較病理學研討會（85年 6月 9日於台中榮民總醫院舉行）：

38. Ferret	Pheochromocytoma	美國紐約動物醫學中心
39. Human	Extra adrenal pheochromocytoma	新光吳火獅紀念醫院
40. Spragur-Dawley CD-1	Mammary gland fibroadenoma	國家實驗動物繁殖及研究中心
41. Human	Fibroadenoma	省立豐原醫院
42. Pointer bitch	Canine benign mixed type gland tumor	國立中興大學獸醫學系
43. Human	Phyllodes tumor	台中榮民總醫院
44. Dog	Canine oral papilloma	國立台灣大學獸醫學系
45. Human	Squamous cell papilloma	中國醫藥學院

第七次比較病理學研討會（85年11月10日於國立屏東技術學院獸醫系舉行）：

46. Cat	Feline dirofilariasis	美國紐約動物醫學中心
47. Human	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	三軍總醫院
48. wild rodents	Adiaspiromycosis	國立台灣大學獸醫學系
49. Human	Echinococcosis	台北榮民總醫院
50. Piglet	Porcine cytomegalovirus infection	台灣省家畜衛生試驗所
51. Human	Pneumocystis carinii pneumonia	台北病理中心

52. Goslings	Aspergillosis	屏東縣家畜疾病防治所
53. Human	Intracavitary aspergilloma and cavitary tuberculosis, lung.	羅東聖母醫院
54. Human	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	林口長庚紀念醫院
55. Broilers	Infectious laryngo-tracheitis (Herpesvirus infection)	國立屏東技術學院獸醫學系

第八次比較病理學研討會（86年3月2日於台中榮民總醫院第一會議廳舉行）：

56. Human	Gastrointestinal stromal tumor	台中榮民總醫院
57. Chicken	Cecal coccidiosis	國立中興大學獸醫學系
58. Human	Tuberculous enteritis with perforation	佛教慈濟綜合醫院
59. Dog	Colonic adenocarcinoma	美國紐約動物醫學中心
60. Human	Intestinal capillariasis	台北馬偕醫院
61. Goose	Spirochetosis	國立嘉義農專獸醫科
62. Human	Submucosal leiomyoma of stomach	頭份為恭紀念醫院
63. Porcine	Proliferative enteritis (<i>Lawsonia</i> <i>Intracellularis</i> infection)	屏東縣家畜疾病防治所
64. Human	1. Adenocarcinoma of sigmoid colon 2. Old schistosomiasis of rectum	省立新竹醫院
65. Carprine	Cryptosporidiosis	台灣養豬科學研究所

第九次比較病理學研討會（86年7月20日於新光吳火獅紀念醫院B1大會議室舉行）：

66. Chapman's zebra	Echinococcosis	國立台灣大學獸醫學系
67. Human	Hepatic ascariasis and cholelithiasis	彰化基督教醫院
68. Human	Liver abscess (<i>Klebsillae pneumoniae</i>)	台北醫學院
69. Pig	Pseudorabies (Herpesvirus infection)	台灣養豬科學研究所
70. Human	Acute Q fever hepatitis	佛教慈濟綜合醫院
71. Human	Myelolipoma	台北耕莘醫院
72. Mouse	Reticulum cell sarcoma	國家實驗動物繁殖及研究中心
73. Human	Hepatocellular carcinoma	新光吳火獅紀念醫院
74. Wistar strain rats	Hepatocellular carcinoma induced by aflatoxin B1	台灣省農業藥物毒物試驗所
75. Rabbits	Acute yellow phosphorus intoxication	國立中興大學獸醫學系

第十次比較病理學研討會（86年11月2日於三軍總醫院研究大樓一樓視聽教室舉行）：

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|---------------------------|---|-----------------------|
| 76. Cat | Polycystic kidney bilateral and renal failure | 美國紐約動物醫學中心 |
| 77. Human | 1.Xanthogranulomatous inflammation with nephrolithiasis, kidney, right.
2.Ureteral stone, right. | 羅東聖母醫院 |
| 78. Chicken | Marek's disease in native chicken | 屏東縣家畜疾病防治所 |
| 79. Human | Emphysematous pyelonephritis | 彰化基督教醫院 |
| 80. SHR rat | 1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate
2.Benign hypertension | 國防醫學院 & 國家實驗動物繁殖及研究中心 |
| 81. Human | Angiomyolipoma | 羅東博愛醫院 |
| 82. Human | Inverted papilloma of prostatic urethra | 省立新竹醫院 |
| 83. SD rats | Phagolysosome-overload nephropathy | 國科會國家實驗動物繁殖及研究中心 |
| 84. Human | Nephrogenic adenoma | 國泰醫院 |
| 85. Dog | Renal amyloidosis | 台灣養豬科學研究所 |
| 86. Human | Multiple myeloma with systemic Amyloidosis | 佛教慈濟綜合醫院 |
| 87. Human | Squamous cell carcinoma of renal pelvis and calyces with extension to the ureter | 台北病理中心 |
| 88. Human | Fibroepithelial polyp of the ureter | 台北耕莘醫院 |
| 89. Goose | 1.Severe visceral gout due to kidney damaged
2.Infectious serositis | 國立中興大學獸醫學系 |
| 90. Human | Clear cell sarcoma of kidney | 台北醫學院 |
| 91. orange-rumped agoutis | Hypervitaminosis D | 國立台灣大學獸醫學系 |

第十一次比較病理學研討會（87年3月1日於佛教慈濟綜合醫院舉行）：

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| 92. Pig | Foot-and-mouth disease (FMD) | 屏東縣家畜疾病防治所 |
| 93. Dog | Mammary gland adenocarcinoma, complex type, with chondromucinous differentiation | 國立台灣大學獸醫學系 |
| 94. Human | 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, carcinoma, metastatic | 羅東聖母醫院 |
| 95. Dog | Transmissible venereal tumor | 國立中興大學獸醫學系 |

96.	Human	Malignant lymphoma, large cell type, diffuse, B-cell phenotype	彰化基督教醫院
97.	Tiger	Carcinosarcomas	台灣養豬科學研究所
98.	Human	Mucinous carcinoma with intraductal carcinoma	省立豐原醫院
99.	Mouse	Mammary gland adenocarcinoma, type pulmonary metastasis, BALB/cBYJ mouse	國家實驗動物繁殖及研究中心
100.	Human	Malignant fibrous histiocytoma and paraffinoma	中國醫藥學院
101.	Pig	Swine pox	國立屏東科技大學獸醫學系
102.	Human	Pleomorphic adenoma (benign mixed tumor)	佛教慈濟綜合醫院

**第十二次比較病理學研討會（87年4月19日於臺灣養豬科學研究所舉行）：心臟血管
專題 演講**

第十三次比較病理學研討會（87年6月14日於台北市立動物園舉行）：

103	Human	Atypical central neurocytoma	新光吳火獅紀念醫院
104	SD rat	Cardiac schwannoma	國家實驗動物繁殖及研究中心
105	Human	1. Mucormycosis 2. Diabetes mellitus	花蓮佛教慈濟綜合醫院
106	Dog	Parasitic meningoencephalitis, caused by <i>Toxocara canis</i> larvae migration	臺灣養豬科學研究所
107	Human	1. Primary cerebral malignant lymphoma 2. Acquired immune deficiency syndrome	台北市立仁愛醫院
108	Lamb	Listeric encephalitis	屏東縣家畜疾病防治所
109	Human	Desmoplastic infantile ganglioglioma	高雄醫學院
110	Piglet	Pseudorabies	國立屏東科技大學
111	Human	Schwannoma	三軍總醫院
112	Chicken	Avian encephalomyelitis	國立中興大學
113	Human	Tuberculous meningitis	羅東聖母醫院
114	Dog	Osteosarcoma	美國紐約動物醫學中心

中華民國比較病理學會第一次至第十三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	國立中興大學獸醫學系
	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	Spragur-Dawley CD 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	國立中興大學獸醫學系
	43	Phyllodes tumor	Human	台中榮民總醫院
	44	Canine oral papilloma	Dog	國立台灣大學獸醫學系
	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	台北耕莘醫院
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
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	台北耕莘醫院
90.	Clear cell sarcoma of kidney	Human	台北醫學院
93.	Mammary gland adenocarcinoma, complex type , with chondromucinous differentiation	Dog	國立台灣大學獸醫學系
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, papillary carcinoma, metastatic	Human	羅東聖母醫院
95.	Transmissible venereal tumor	Dog	國立中興大學獸醫學系
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 B, with pulmonary metastasis, BALB/cBYJ mouse	Mouse	國家實驗動物繁殖及研究中心
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	2.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
細菌	6	Tuberculosis	Monkey	台灣大學獸醫學系
	7	Tuberculosis	Human	省立新竹醫院
	12	<i>H. pylori</i> -induced gastritis	Human	台北病理中心
	13	Pseudomembranous colitis	Human	省立新竹醫院
	26	Swine salmonellosis	Pig	國立中興大學獸醫學系
	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 (<i>Lawsonia intracellularis</i> infection)	Porcine	屏東縣家畜疾病防治所
	68	Liver abscess (<i>Klebsillae pneumoniae</i>)	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	國立中興大學獸醫學系
	108	Listeric encephalitis	Lamb	屏東縣家畜疾病防治所
	113	Tuberculous meningitis	Human	羅東聖母醫院
病毒	21	Newcastle disease	Chickens	國立台灣大學獸醫學系
	22	Herpesvirus infection	Goldfish	國立台灣大學獸醫學系
	30	Demyelinating canine distemper encephalitis	Dog	台灣養豬科學研究所
	31	Adenovirus infection	Malayan sun bears	國立台灣大學獸醫學系
	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	Pseudorabies	Piglet	國立屏東科技大學
	112	Avian encephalomyelitis	Chicken	國立中興大學
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	國立台灣大學獸醫學系
	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	花蓮佛教慈濟綜合醫院
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	國立台灣大學獸醫學系
	67	Hepatic ascariasis and cholelithiasis	Human	彰化基督教醫院
	106	Parasitic meningoencephalitis, caused <i>Toxocara canis</i> larvae migration	Dog	臺灣養豬科學研究所
原蟲	4	Cryptosporidiosis	Goat	台灣養豬科學研究所
	15	Amoebiasis	<i>Lemur fulvus</i>	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	<i>Pneumocystis carinii</i> pneumonia	Human	台北病理中心
	57	Cecal coccidiosis	chicken	國立中興大學獸醫學系
	65	Cryptosporidiosis	Carprine	台灣養豬科學研究所
立克次體	70	Human	Acute Q fever hepatitis	佛教慈濟綜合醫院
其它	9	Perinephric pseudocyst	Cat	台灣大學獸醫學系
	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	國立中興大學獸醫學系
	76.	Polycystic kidney bilateral and renal Failure	Cat	美國紐約動物醫學中心
	80.	1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate 2.Benign hypertension	SHR rat	國防醫學院 & 國家實驗動物繁殖及研究中心
	83.	Phagolysosome-overload nephropathy	SD rats	國家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	國立中興大學獸醫學系
	91.	Hypervitaminosis D	orange-rumped agoutis	國立台灣大學獸醫學系

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

苗栗縣竹南郵政信箱 23 號

病理生物系 邱慧英 小姐

Tel: (037) 672352轉505

Fax: (037) 687803

e-mail address: hic01@mail.prit.org.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：_____