

病例摘要

Case 237 財團法人天主教耕莘醫院病理科：CTH-195743R1

A 36 year-old lady suffered from severe headache with vomiting since the afternoon was sent to our ER in the evening. The patient had past history of head injury 10 years ago. She had received craniotomy for removal of epidural hematoma on her right head in other hospital and recovered quite well.

She experienced sudden onset of explosive headache, located mainly over bilateral temporal and occipital region, accompanying with severe stiffness and tightening sensation of posterior nuchal muscles. She then felt nauseous, and vomit all the foods she had eaten at lunch. Then she collapsed on the ground. There was no lapse of consciousness, no limb weakness or numbness, and no vertigo or convulsion. She also had headaches occasionally in the past, but all of them were only of mild severity, and never happened with vomiting.

Laboratory data (CBC, electrolyte, liver and renal function), physical examinations and neurological examinations (muscle power, DTR, gait, etc.) were all normal except that neck rigidity were noted. Brain CT with contrast were performed and showed intraventricular hemorrhage (IVH) with brain edema and mild hydrocephalus, subarachnoid hemorrhage (SAH) in Rt T-P region, and a large vascular tuft, about 5 x 4 x 3.5 cm, in the Rt T-P lobes. Unfortunately, she was found to have depressed consciousness (GCS: E3V1M3) in the following morning. Dense paralysis of four limbs with left side predominance was also noted. Urgent brain CT revealed recurrent massive hemorrhage with obstructive hydrocephalus and marked brain edema. Craniotomy was performed immediately. She was then recovered gradually after the surgery (GCS: E4VTM6; RA/LA=4/3; RL/LL=3/3).

Case 238 屏東科技大學獸醫學系：D94-17294-15

A 30-day-old female calf, showed clinical signs of propulsive, circling, lethargy and ataxia.

Case 239 羅東聖母醫院：0505904

A 71 year old female suffered from right side limb weakness for weeks. C'Y scan showed cerebral mass and operation finding showed ill-defined tumor which

was not easily separated from surrounding brain tissues. Multiple pieces of brain tumor up to 1.3 x 0.89 x 0.5 cm were submitted for pathologic diagnosis.

Case 240 綠色四季生物科技公司：0202

An untreated female Wistar rat was killed *in extremis* at 683 day-on-exposure (106-week of age). The rat had been thinner and weak for a few days prior to the extermination. External gross findings included two masses, 1.8 x 1.7 x 1.5 cm and 3.5 x 2.8 x 2.7 cm in size observed on the thoracic and abdominal wall, respectively. Internal findings were the kidneys with coarse and pitted surface beneath the capsule, and the brain with a nodule, approximately 1 x 1 cm protruding in between the cerebral hemisphere and cerebellum.

Case 241 高雄醫學大學附設醫院病理科：KMU-05-3889 A4

The 45 year-old woman was a case of depression disorder diagnosed on last June with regular Psychiatry OPD follow-up and medication control in our hospital. Insidious onset of diplopia, headache, right limbs resting tremor, memory impairment, and mild slurred speech was noted since this January. Intermittent visual loss was also noted. Due to the above problems, she visited our Neurology OPD for further help. After series of examination, brain tumor in left frontotemporal area with involvement of left parasellar region was noted. Craniotomy with tumor removal was performed on March 7, 2005.

Case 242 屏東科技大學獸醫學系：D94-17176-12

An aborted fetus from a dairy cow, multiple necrotic lesions were noticed in the muscle, heart, liver and lungs; this slide came from brain tissue.

Case 243 花蓮慈濟醫院病理科：A227-40

A 51-year-old woman had been diagnosed as a case of infiltrating lobular carcinoma of left breast with bilateral lymph nodes and lungs metastasis at Tao-Yuan General Hospital in Dec, 2003, and had received radiotherapy and chemotherapy since then. She came to our hospital for further treatment in May, 2004. Bone scan showed multiple spines, ribs and iliac bone metastasis and chest CT scan showed suspicious lesion of lymphangitis carcinomatosa without definite metastatic lesion. Unfortunately, headache, nausea and vomiting had occurred since late December, 2004. Brain CT scan didn't show any definite lesion. However,

brain MRI showed abnormal findings in bilateral occipital lobes and cerebellar tonsils. Dyspnea with pneumonia developed. Her condition got worse and worse. Finally, she expired due to respiratory failure in Jan, 2005. Autopsy was performed. This section was taken from pons.

Case 244 中興大學獸醫病理研究所：TOCP

White Leghorn Hens (*Gallus gallus domesticus*), adult (14 months of age) were bought from a farm at Nanto county in Taiwan. Hens had been vaccinated, including Marek's disease, Newcastle disease, bronchitis and avian encephalomyelitis. A commercial diet for laying hens and drinking water supplied *ad libitum*. Each hen was individually housed in a wire mesh cage, and were acclimatized for approximated 2 weeks before study. Hens were raised in air conditioned room with temperature 22-25°C, relative humidity 50-70% and 12L/12D photoperiod. Body weight ranging from 1.3 to 1.5 kg of hens was selected for this test. All hens were fasted overnight before treatment. Thirty minutes prior to administration, hens were subjected to an intramuscular injection of an atropine sulfate solution (purity >98%, Fluka, Tokyo) in deionized water at dosage of 20 mg/kg, and 1,000 mg/kg of TOCP were single gavaged by using a syringe fitted with a 16 gauge snub-tipped dosing needle, at a constant dose volume of 10 ml/kg. Control hens were received an equivalent volume of vehicle. Clinical signs showed that TOCP-treated hens exhibited moderate to severe ataxia and reluctant moving after 14 days of treatment. Hens were sacrificed at the end of experiment.

Case 245 財團法人天主教耕莘醫院病理科：CTH-226944B

A 78 years old female was sent to our ER since she had sudden onset of conscious change and bizarre posture last for few seconds. She had past history of hypertension, arrhythmia, and bipolar disorder. She had received regular medication from the cardiologist and psychiatrist and the disease control is fair. The physical examination and the laboratory data are both normal. Her conscious is clear and no sign of episode of bipolar disorder is found. After consultation with neurologist and psychiatrist, brain CT was suggested and performed which showed a sharply demarcated brain tumor in her right parietal area. Craniotomy with tumor removal was performed smoothly and she recovered after the operation.

Case 246 國立台灣大學獸醫學系：NTU2005-832 K

The patient was referred to the emergency with sudden onset of seizure like episode, hemiparesis of right limbs, anisocoria and ptialism on Jan 22. Results of a

complete blood count and serum biochemical analyses were within reference ranges. Sampling whole blood for canine distemper PCR was negative. During hospitalization, fever (40-41°C) sometimes happened. The dog was treated with prednisolone, doxycycline and diazepam and gradually improved. The dog was discharged on the 7th day. The dog returned on June 14 because of seizures and ataxia. After treatment with prednisolone and phenobarbital, the dog improved for a few days then the symptoms gradually worsened. On July 10, the most conspicuous findings were a head tilt and circle to the right, tetraparesis, and nystagmus. Pupil light reflexes were normal bilaterally. Menace responses were absent. The dog had normal appetite but crying when she was awake. Cerebrospinal fluid evaluation revealed normal protein content and the predominant cell type was the small lymphocyte. Despite therapy, the dog's neurological status worsened. Based on poor prognosis the owner requested euthanasia.

Comparative Pathology Case 237

Contributors: Jeng-Hung Suen (孫政宏), Fur-Jiang Leu (呂福江), Chia-Ing Jan (詹佳穎)

Department of Pathology, Cardinal Tien's Hospital

Clinical history: A 36 year-old lady suffered from severe headache with vomiting since the afternoon was sent to our ER in the evening.

The patient had past history of head injury 10 years ago. She had received craniotomy for removal of epidural hematoma on her right head in other hospital and recovered quite well.

She experienced sudden onset of explosive headache, located mainly over bilateral temporal and occipital region, accompanying with severe stiffness and tightening sensation of posterior nuchal muscles. She then felt nauseous, and vomit all the foods she had eaten at lunch. Then she collapsed on the ground. There was no lapse of consciousness, no limb weakness or numbness, and no vertigo or convulsion. She also had headaches occasionally in the past, but all of them were only of mild severity, and never happened with vomiting.

Laboratory data (CBC, electrolyte, liver and renal function), physical examinations and neurological examinations (muscle power, DTR, gait, etc.) were all normal except that neck rigidity were noted. Brain CT with contrast were performed and showed intraventricular hemorrhage (IVH) with brain edema and mild hydrocephalus, subarachnoid hemorrhage (SAH) in Rt T-P region, and a large vascular tuft, about 5 x 4 x 3.5 cm, in the Rt T-P lobes. Unfortunately, she was found to have depressed consciousness (GCS: E3V1M3) in the following morning. Dense paralysis of four limbs with left side predominance was also noted. Urgent brain CT revealed recurrent massive hemorrhage with obstructive hydrocephalus and marked brain edema. Craniotomy was performed immediately. She was then recovered gradually after the surgery (GCS: E4VTM6; RA/LA=4/3; RL/LL=3/3).

Diagnosis: Arteriovenous malformation (AVM) of cerebrum.

Gross findings: The specimen submitted consists of a piece of brain tissue, measuring 7.5 x 6.5 x 3 cm in size and 68 gm in weight, fixed in formalin. Grossly it showed an angry, tangled mass of vessels with variable diameter, measuring 4.8 x 3.6 x 2.8 cm. Subarachnoid hemorrhage is also present.

Histopathological findings: Microscopically, sections show a vascular lesion with irregularity in size, shape, and degree of muscularization characterizing the vessels of an AVM. Structural hybrids of both vein and artery are noticed in many areas. Interposed cerebral parenchyma between the abnormal vessels is present. Subarachnoid hemorrhage is obvious.

Discussion: AVMs most commonly originate above the tentorium and enlarge insidiously, generally becoming symptomatic during the 2nd to 5th decades of life. Hemorrhage, headaches, seizures, and focal neurological deficits are common. In infants and children, the effluent may be sufficient to dilate the great vein of Galen and produce cardiac failure. The most plausible explanation of the pathogenesis of AVMs incriminates congenital absence of the regional capillary bed. This theory is in agreement with in vivo and in vitro arteriographic studies that clearly demonstrate arteriovenous shunting. Shunting elevates intraluminal venous pressure and produces ectasia and muscularization so that hybrid vessels with both venous and arterial characteristics are created. The radiologic features are manifested by serpiginous profiles of abnormal vessels with dark lumen (flow voids) readily evident on MR imaging. Arteriovenous shunting seen during angiography established the diagnosis. The gross and histological features of AVMs are mentioned above. The prognosis of patients with untreated AVMs is contingent upon the unpredictable occurrence of hemorrhage, an event estimated to occur at the rate of 2% to 4% per patient year.^{3, 4} Aggressive treatment with complete excision precludes hemorrhage and mortality.² Conservative treatment with embolization is effective temporarily in reducing vasculature but associated with higher incidence of AVM-related mortality.²

The vascular malformations of brain include capillary telangiectasis, venous angioma, cavernous angioma, AVM, and vascular malformations in dysgenetic syndromes. The capillary telangiectasis is the most innocent lesion and is encountered most often as an incidental finding. It is composed of aggregates of thin-walled, ecstatic vessels resembling dilated capillaries. Venous angioma appear as loose or compact conglomerates on the venous limb of the cerebral circulation. It bleed only rarely and are generally incidental MR findings. Cavernous angioma is a compact mass of malformed vessels often surrounded by a rim of hemosiderin-stained brain parenchyma. Although many cavernous angiomas remain clinically silent, seizures, focal neurologic deficits, or signs of increased intracranial pressure may herald their presence. Hemorrhages are often smaller and less life threatening than those

from AVMs. Vascular malformation in dysgenetic dyndromes includes Sturge-Weber disease, Wyburn-Mason syndrome, and Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber syndrome).

Diagnostic criteria:

1. vascular lesion with irregularity in size, shape, and degree of muscularization.
2. Structural hybrids of both vein and artery.
3. Interposed cerebral parenchyma between the abnormal vessels.
4. Subarachnoid hemorrhage if ruptured.

References:

1. Peter C. Burger, et al. Surgical Pathology of the Nervous System and its Coverings. 4th ed. Churchill Livingstone 2002.
2. ApSimon; Reef, H, et al. A Population-Based Study of Brain Arteriovenous Malformation: Long-Term Treatment Outcomes. Stroke. 2002; 33(12):2794-2800
3. Brown RD Jr, Wieber DO, Forbes G, et al. The Natural History of Unruptured Intracranial Arteriovenous Malformations. J Neurosurg 1988;68:352-357
4. Ondra SL, Troupp H, George ED, et al. The Natural History of Symptomatic Arteriovenous Malformation of the Brain: a 24 Year Follow-Up Assessment. J Neurosurg 1990;73:387-391
5. Hartmann Andreas, Mast Henning, Mohr JP, et al. Mobidity of Intracranial Hemorrhage in Patients with Cerebral Arteriovenous Malformation. Stroke 1998; 29(5): 931-934
6. Hofmeister C, Stapf C, Hartmann A, et al. Demographic, Morphological, and Clinical Characteristics of 1289 Patients with Brain Arteriovenous Malformation. Stroke 2000; 31(6):1307-1310

Comparative Pathology Case 238

Contributors: Shih-Ping Lo(羅世秉); T.C. Chang (張聰洲)

National Pingtung University of Science and Technology (國立屏東科技大學獸醫學系)

Clinical history: A 30-day-old female calf, showed clinical signs of propulsive, circling, lethargy and ataxia.

Diagnosis: Hydranencephaly of cattle

Gross findings: The brain showed almost complete absence of cerebral hemispheres only a thin wall sac filled with cerebrospinal fluid and enclosed by leptomeninges. The cerebellum was of mild atrophy. However, brain stem and spinal cord were normal in size and shape.

Histopathological findings: The slide of cerebrum showed typical hydranencephaly characterized by cystic cavity in the part of the marginal layer of the cerebral cortex attached to a fibrotic leptomeninges.

Discussion: Hydranencephaly is a rare and extreme form of porencephaly. The hemispheres of the cerebrum are absent and replaced by sacs filled with cerebrospinal fluid (CSF). In most cases, the cerebellum and brain stem are formed normally. There is complete or almost complete destructure and lack development of the cerebral hemispheres, a large cavity filled with cerebrospinal fluid and covered by thin-wall could be found. The most common cause is a virus infection of the fetus during gestation, includes the Akabane virus in ruminants in Australia, Japan, and Israel; the Bluetongue virus in sheep and cattle in North America; the Rift Valley fever virus and the virus of Wesselsbron disease in sheep and cattle in Africa; and the Cache Valley virus in sheep in the United States. In Japan, the Chuzan virus (genus *Orbivirus*) has been implicated in the production of hydranencephaly and cerebellar hypoplasia in calves. In addition, Akabane disease is the most popular in the world. An important distinction should be made between maximal hydrocephalus and hydranencephaly. Hydranencephaly is characterized by complete absence of cerebrum which is supplied by the anterior cerebral artery and middle cerebral artery bilaterally. The only intact brain tissue is the basal ganglia, brain stem, and a small amount of occipital lobe, supplied by the posterior cerebral artery. Hydranencephaly is presumably caused by an

intrauterine stroke in the distribution of both internal carotid arteries. In case of hydrocephalus is enlarged due to pressure from an excessive volume of cerebrospinal fluid within the ventricular system. Most cases are congenital in calves, but a rare acquired condition in adult cattles, due to infection or trauma. Congenital alterations in calvarial shape and margination are commonly associated with hydrocephalus. Hydrocephalus is characterized by an abnormal accumulation of fluid in the cranial cavities. In internal hydrocephalus, the fluid is within the ventricular system; in external hydrocephalus, the fluid is in the arachnoid space; and in communicating hydrocephalus, the excess fluid is present in both location. The communicating and external types of hydrocephalus, are distinguished from cerebral atrophy, quite rarely in animals. Internal hydrocephalus, which is denoted by variably dilated ventricular cavities lined by ependyma, is quite common, and may be congenital or acquired, but both forms may be considered here for convenience. In hydranencephaly there can be complete or almost complete absence of the cerebral hemispheres, leaving only membranous sacs filled with cerebrospinal fluid and enclosed by leptomeninges. In animals, the lesion develops in early fetal stages and before the mature arrangements of the cortex are present. Hydranencephaly occurs in all species but is the most common in calves. It occurs either sporadically or as minor epizootics.

References:

1. Blowey, R.W, Weaver, A. D. Color atlas of disease and disorders of cattle. St.Louis, MI:Mosby, 10-11, 2003.
2. Brian a. Summers, John F. Cummings, Alexander de Lanunta. Veterinary neuropathology. Mosby, 72-77, 1995.
3. DeLunta, Alexander. Veterinary neuroanatomy and clinical neurology. Philadelphia: Saunders, 30-52, 1983.
4. K. V. F. Jubb, Peter C. Kennedy, Nigel Palmer. Pathology of domestic animals. Volume 1 : bones, muscles, nervous system, eye and ear, skin. Academic press, inc. New York, 277-281, 1992.
5. Mayhew, Ian G. Large animal neurology: a handbook for veterinary clinicians. Philadelphia: Lea&Febiger, 75-76, 1989.
6. Y. Noda, H. Yokoyama, T.Katsuki, S.kurashige, Y.Uchinuno and M. Navita. Demonstration of Akabane Virus Antigen Using Immunohistochemistry in Naturally Infected Newborn Calves. Vet Pathol 38:216-218, 2001.

Comparative Pathology Case 239

Contributor: Chu Chih Ping (祝志平), MD.MS. Lotung St. Mary's Hospital.

Clinical history: A 71 year old female suffered from dizziness off & on for half month and headache was noted. Physical examination showed muscle power: 5/5 CT scan showed cerebral mass. So craniotomy with removal of tumor (under sono-guide) was performed.

Diagnosis: Glioblastoma multiforme

Gross findings: An ill-defined tumor which was not easily separated from the surrounding brain tissue during operation. Then, multiple pieces of brain tissues up to 1.3 x 0.8 x 0.5 cm. were submitted.

Histopathological findings: Tumor with hemorrhage and necrosis was found. Endothelial proliferation was marked. The tumor cells showed pleomorphism with nuclear atypia and mitoses. There was also infiltrating margins in the tumor.

Discussion: The key features in differentiation diagnosis of CNS tumors (GBM)

1. Age/Sex: GBM: 50-60 years old.
2. Location:
 - A. Intra-axial (parenchymal) vs extra-axial (dural)
 - B. superficial (cortex) vs deep
 - C. Intraventricular vs paraventricular
 - D. Supratentorial vs infratentorial
 - E. Sella, suprasella, hypothalamic
 - F. Cerebellar vs cerebellopontine angle vs brain stem
 - G. Spinal cord: extradural vs intradural, extramedullary vs intramedullary.
3. Image appearance (Gross)
 - A. Site of origin/epicentric
 - B. Mass effect, edema etc.
 - C. Infiltrating vs discrete
 - D. Enhancing: uniform or irregular (ring-enhancing)
 - E. cystic vs solid
 - F. single vs multiple
 - G. Seeding vs nonseeding

Immunohistochemistry:

1. S-100, GFAP: (+) (GFAP shows equally spaced astrocytes with radiating processes. MIB)
2. CK, LCA, SYN, HMB- 45: (-)

Differential diagnosis:

1. Metastatic carcinoma: CAM512, EMA vs GFAP
2. Cerebral lymphoma: Perivascular tumor cells (CD20: +)
3. Abscess-abundant macrophages (CD68) & periphery of granulation tissue with reactive gliosis.

Diagnostic criteria:

A. nuclear atypia

M: Mitosis (scored by MIB-1 staining)

E: Endothelial proliferation= multiple layers

N: Necrosis: usually but not invariably associated with palisading

I: Infiltrating margin (relative circumscription in “primary/de novo GBM”)

Grading Schemes for astrocytoma

Three-tiered

1. Ringertz

2. Modified Ringertz (Burger)

3. WHO

Four-tiered

1. Kernohan

2. St. Anne-Mayo

St. Anne-Mayo (functionally 3-tiered)(AMEN)

Criteria: A: Atypia (nuclear)

M: Mitoses (1 is enough)

E: Endothelial Proliferation

N: Necrosis

Scoring: Grade 1= 0 criteria (very rare)

Grade 2= 1 criteria (atypia)

Grade 3= 2 criteria (atypia + mitoses)

Grade 4= 3 or 4 criteria

Equivalents in terms of WHO grade

Grade 1 has no WHO equivalent

Grade 2: Differentiated astrocytoma

Grade 3: Anaplastic astrocytoma

Grade 4: GBM

References:

1. Daumas-Duport C, Scheithauer BW. Grading of astrocytomas. A simple and reproducible method. *Cancer* 62: 2152-2165. 1998.

2. Burser P, Vogel FS, Green S, et al. Glioblastoma multiforme and anaplastic astrocytoma: Pathologic criteria and prognostic implications. *Cancer* 56:1106-1111, 1985.
3. Stemmer-Rachaminov A O, Louis DN. Histopathologic and immunohistochemical prognostic factors in malignant gliomas. *Curr Opin oncol* 9: 230-234, 1997.

Comparative Pathology Case 240

Contributors: Hans Chen and Koejing Cheung

Greenseasons Biotech Company, Tanshui, Taipei County 251

Clinical history: An untreated female Wistar rat was killed *in extremis* at 683 day-on-exposure (106-week of age). The rat had been thinner and weak for a few days prior to the extermination. No laboratory testing was performed.

Diagnosis: Pineoblastoma

Gross findings: External gross findings included two masses, 1.8 x 1.7 x 1.5cm and 3.5 x 2.8 x 2.7cm in size, observed on the thoracic and abdominal wall, respectively. Internal findings were the kidneys with coarse and pitted surface beneath the capsule, and the brain with a pale, mottled nodular mass, approximately 0.9 x 0.9 cm, protruding in between the occipital pole of cerebral hemispheres and cerebellum.

Histopathological findings: The histopathological observations of the brain mass was a discrete, high cellular, densely packed, basophilic mass occupying in the cerebrum and cerebellum spaces of the brain section. (Fig. 1) The proliferative cells were highly infiltrative and aggressive to the adjacent brain parenchyma. These cells were large, pleomorphic, dark staining basophilic with some undefined pale cytoplasm. (Fig. 2)

There were some neoplastic cells much larger and bizarre. There were occasional smaller, lymphoid-like cells intermingled with the majority cells of large pleomorphic cells. Mitotic figures were common. Small areas of tumor necrosis were seen. The neoplastic tissue had occasional connective tissues and vascular components forming somewhat incomplete acinar partition or pseudolobulation pattern of the neoplastic tissue.

Discussion: This is a neoplasm originated and proliferated from the pineal gland infiltrating to the surrounding spaces and into the peripheral brain tissues of cerebrum hemispheres and cerebellum.

Pineal gland is on the midline, caudal to the cerebral hemispheres and anterior to the cerebellum. It is on the long slender stalk, near the skull, and covered by a leptomeninges. It is approximately 1- 2mm in diameter and weighs 0.5 - 1mg in adult rats. It is larger in males. The gland is covered by a delicate capsule and a dense capillary network, and composed of pinealocytes, specialized cells that secrete melatonin and have features of neuronal

differentiation. The cells are arranged in clusters and rows. There are small number of smaller cells and some astrocytes in the gland.

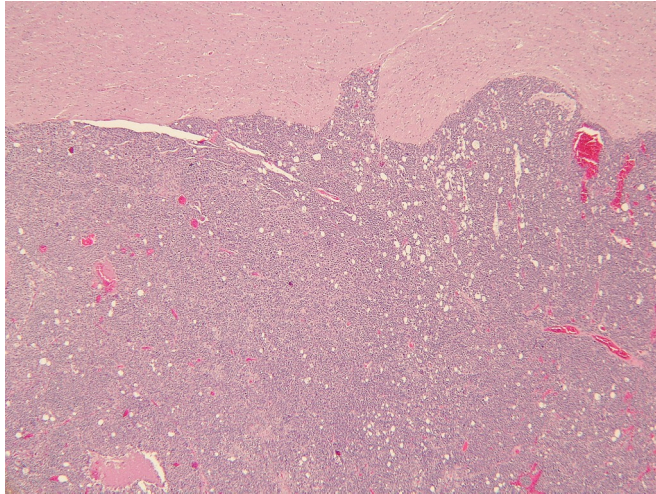
The pineal gland tumors arise from the specialized cells of the pineal gland cells ----- pineocytes. The tumors range in histologic appearance from well differentiated lesions of pineocytomas that consist of two cell types: large pale staining parenchyma cells (pinealocytes) and smaller, round, dark-staining interstitial cells with scant cytoplasm to high-grade tumors of pineoblastomas. The pineoblastomas consist of densely packed small cells with necrosis, frequent mitotic figures, and little evidence of neuronal differentiation. Pineoblastoma is highly aggressive, spread can be throughout the CSF.

Diagnostic criteria:

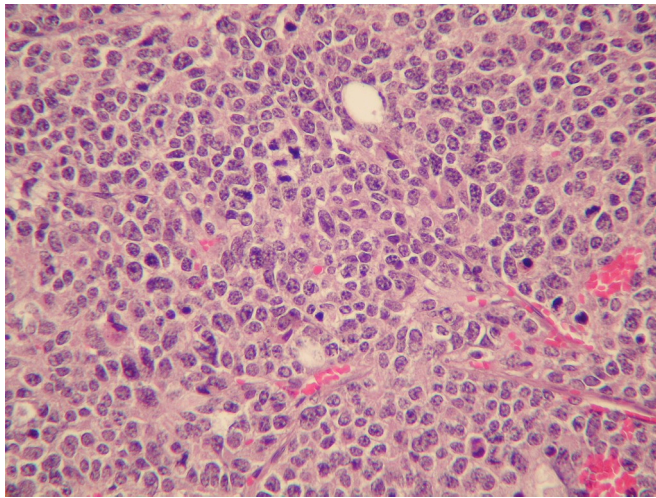
1. Pineocytoma (malignant but any benign?) and Pineoblastoma
2. Astrocytoma and glioma
3. Neuroblastoma

References:

1. Boormam GA, Eustis SL, Elwell MR, Montgomery Jr. CA, Mackenjie WF: Pathology of the Fisher Rat---Reference and Atlas. Academic Press 1990, San Diego.
2. Kumar U, abbas Ak, Fausto N (eds.): Robbins and Cotran Pathologic Basis of Disease (7th ed.). Elsevier Saunders 2005 , Philadelphia.



Pineoblastoma, brain base. X4



Pineoblastoma, brain base. X40

Comparative Pathology Case 241

Contributors: I-Yu Chen (陳怡妤) MD; Sheng-Lan Wang (王勝嵐) MD
Pathology Department of Kaohsiung Medical University Hospital (高醫病理科)

Clinical history: The 45 year-old woman was a case of depression disorder diagnosed on last June with regular Psychiatry OPD follow-up and medication control in our hospital. Insidious onset of diplopia, headache, right limbs resting tremor, memory impairment, and mild slurred speech was noted since this January. Intermittent visual loss was also noted. Due to the above problems, she visited our Neurology OPD for further help. After series of examination, brain tumor in left frontotemporal area with involvement of left parasellar region was noted. Craniotomy with tumor removal was performed on March 7, 2005. (KMU-05-3889 A4)

Diagnosis: Chordoid meningioma

Gross finding: The specimen submitted are tissue fragments measuring up to 6.0 x 4.0 x 3.5 cm in size. They are grayish and elastic.

Histopathological findings: The tumor is composed of proliferated meningotheial cells with sheets, lobular or whorl arrangement. The tumor cells have round to oval or spindle nuclei, eosinophilic cytoplasm and indistinct cellular border. Regions that are histologically similar to chordoma with trabeculae of eosinophilic vacuolated cells in a myxoid background are also noted.

Discussion: Meningiomas are thought to arise from arachnoidal cap cells, which reside in the arachnoid layer of the meninx covering the brain surface. They may occur intra- cranially or within the spinal canal. The surface of the brain, either over the convexity or at the skull base, is the common location. Rarely, meningiomas may also arise in an intraventricular or intraosseous location.

Meningiomas may cause symptoms by irritating the underlying cortex, compressing the brain or the cranial nerves, producing hyperostosis and/or invading the overlying soft tissues, or inducing vascular injuries to the brain. They also may give rise to the stereotyped symptoms in specific locations. In

the cases with subfrontal involvement, changes of mental state, apathy, disinhibited behavior, or urinary incontinence may be encountered. Compression of different location of the visual pathways may cause various visual field defects. Decreased vision and diplopia may be resulted from cavernous sinus involvement.

First reported by Kepes et al. in 1987, they described a meningeal tumor in young patients associated with microcytic anemia and/or dysgammaglobulinemia and angiofollicular lymphoid hyperplasia (Castleman Disease). Such tumors were composed of spindle or epithelioid cells arranged in clusters or cords in a myxoid matrix. They used the term “chordoid meningioma” to such meningeal tumor under the great similarity of their histopathologic fetures to that of chordoma.

Kepes et al. also noticed that the tumors were surrounded by massive polyclonal lymphoplasmacellular infiltrates with follicles and germinal centers formation. They proposed that the characteristic type of systemic manifestations were the consequence of these peritumoral lymphoplasmacellular infiltrates. In a larger study conducted by Couce et al. in 2000, enrolling 42 chordoid meningiomas operated at Mayo Clinic during 1975 to 1997, they found that most of the cases showed only mild or even no lymphoplasmacytic infiltrates (35.7% and 40.5%, respectively) within the tumor, and no systemic manifestation was noted. In their experience, chordoid meningiomas are primarily tumors of adults unassociated with systemic manifestations.

Though the histopathological features of chordoid meningioma are distinctive, they should be distinguished from chordoma, chondrosarcoma and metastatic mucinous carcinoma, especially when in the sacrococcygeal location. The identification of the typical meningioma component and the demonstration of epithelial membrane antigen (EMA) and vimentin but not cytokeratin (CK) by immunohistochemical study in these tumors will help to establish the diagnosis of chordoid meningioma.

If subtotally resected, the recurrence rate in chordoid meningioma is high. Couce et al. observed recurrence in all those tumors being subtotally resected. They assumed that the high recurrence rate may be related to the mucoid stroma, which facilitates the spread of the neoplastic cells.

Recently reported by Arima et al., cytokine production, including interleukin (IL)-6, IL-1 β , and vascular endothelial growth factor (VEGF), plays a role in the pathogenesis of chordoid meningioma associated with Castleman disease. Similarly, Denaro et al. found that the tumor cells showed focal

positivity for the pyrogenic cytokine interleukin-6 by immunohistochemical stain. The capacity of the tumor to produce this pyrogenic cytokine could explain both the patient's clinical presentation of fever, headache, and a serological inflammatory syndrome, as well as the disappearance of these symptoms/signs after surgical management.

Surgical resection remains the treatment of choice of chordoid meningioma, as for other meningioma subtypes. Adjuvant radiotherapy is mainly used for incompletely resected, high grade and/or recurrent tumors. It can also be used as primary treatment in the unresectable cases (e.g., optic nerve meningiomas).

Diagnostic criteria:

1. Cords or clusters of epithelioid or spindle cells in a myxoid background.
2. Presences of classical meningioma component.
3. Positive epithelial membrane antigen (EMA) and vimentin staining by immunohistochemical study.

References:

1. Varma DR, Rao BR, Parameswaran S, Gupta AK, et al.: Chordoid meningioma: a report of two cases. *Neurol India*. 2003; 51(4): 522-4
2. Couce ME, Aker FV, Scheithauer BW: Chordoid meningioma: a clinico-pathologic study of 42 cases. *Am J Surg Pathol* 2000; 24(7): 899-905
3. Arima T, Natsume A, Hatano H, Nakahara N, et al.: Intraventricular chordoid meningioma presenting with Castleman disease due to overproduction of interleukin-6. Case report. *J Neurosurg*. 2005;102(4):733-7
4. Denaro L, Di Rocco F, Gessi M, Lauriola L, et al.: Pyrogenic cytokine interleukin -6 expression by a chordoid meningioma in an adult with a systemic inflammatory syndrome. Case report and review of the literature. *J Neurosurg*. 2005;103(3): 555-8

Comparative Pathology Case 242

Contributors: Hung-Tzau Pan (潘宏造); T.C. Chang (張聰洲)

National Pingtung University of Science and Technology (國立屏東科技大學獸醫學系)

Clinical history: An aborted fetus was submitted for necropsy from a 420 dairy cow farm in Pingtung.

Diagnosis: Neosporosis

Gross findings: The prominent lesions were found involving both aborted fetus and placenta. Whitish spots scattered on the surface of placenta, lung, liver, heart and muscle, especially the skeletal muscle of hind leg and heart having additional pale streaks.

Histopathological findings: The histopathological lesions included necrotizing placentitis, myocarditis, myositis, hepatitis and pneumonitis. Multiple foci of necrosis associated with formation of microglial nodules and monocyctic perivascular cuffings were the remarkable changes in the brain, particularly in the cerebrum. Scattered foci of mixing inflammation with associated segmental myofiber necrosis were often seen in the skeletal muscles.

Discussion: *Neospora caninum*, a *Toxoplasma*-like protozoan has been misdiagnosed until 1988, when it was first isolated from puppies having congenital encephalomyelitis and confirmed to be a new protozoan. *Neospora caninum* is an intracellular parasite, which causes neuromuscular illness and paralysis in the dog, cattle, sheep and goat, It also causes abortion and stillbirths in the ruminants. Isolation of *N. caninum* is difficult because of the autolysis of the aborted fetus and the small number of parasites within the nerve tissues. Both domesticated and strayed dogs and wild rats can serve as a reservoir host for *N. caninum* in cattle farm. In Taiwan, it also causes a comparatively high percentage of bovine abortion based on detection of antibodies in the body fluids such as milk, vaginal secretion and saliva. Two rhesus monkey (*Macaca mulata*) have been experimentally and successfully infected with *N. caninum*. This gives us a hind that it may be a zoonotic pathogen potential for human, despite there is no evidence for human infection

at present.

Diagnostic criteria:

1. Abortion
2. Necrotizing encephalitis ,myositis.
3. Polymerase Chain Reaction

References:

1. B.C.Barr, P.A.Conrad, K.W.Sverlow, A.F.Tarantal, A.G.Hendrickx: Experimental fetal and transplacental *Neospora* infection in the nonhuman primate: Laboratory Investigation. 1994; 71:236-242.
2. C. A. Speer, J. P. Dubey, M. M. McAllister, J.A. Blixt: Comparative ultrastructure of tachyzoites, bradyzoites, and tissue cysts of *Neospora caninum* and *Toxoplasma gondii*: International Journal for Parasitology; 1999; 29:1509-1519
3. C. C. Huang: Study on the epidemiology, life-cycle and diagnosis of *Neospora caninum*. 2004
4. H.K. Ooia, C.C. Huang, C.H. Yangb, S.H. Leec: Serological survey and first finding of *Neospora caninum* in Taiwan, and the detection of its antibodies in various body fluids of cattle: Veterinary Parasitology. 2000;90: 47–55
5. J. P. Dubey: Neosprosis-the first decade of research International Journal for Parasitology; 1999; 29:1485-1488
6. Suzanne Payne and John Ellis: Detection of *Neospora caninum* DNA by the polymerase chain reaction: International Journal for Parasitology. 1996; 26(4):347-351.
7. William C.Rebhum: Diseases of Dairy Cattle: *Neospora* sp. 1995;339.

Comparative Pathology Case 243

Contributors: Bo-Yuan Tseng (曾柏元) MD; Yung-Hsiang Hsu (許永祥) MD
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Clinical history:

A 51-year-old woman had been diagnosed as a case of infiltrating lobular carcinoma of left breast with bilateral lymph nodes and lungs metastasis at Tao-Yuan General Hospital in Dec, 2003, and had received radiotherapy and chemotherapy since then. She came to our hospital for further treatment in May, 2004. Bone scan showed multiple spines, ribs and iliac bone metastasis and chest CT scan showed suspicious lesion of lymphangitis carcinomatosa without definite metastatic lesion. Unfortunately, headache, nausea and vomiting had occurred since late December, 2004. Brain CT scan didn't show any definite lesion. However, brain MRI showed abnormal findings in bilateral occipital lobes and cerebellar tonsils. Dyspnea with pneumonia developed. Her condition got worse and worse. Finally, she expired due to respiratory failure in Jan, 2005. Autopsy was performed. This section was taken from pons.

Diagnosis: Infiltrating lobular carcinoma of left breast with meningeal carcinomatosis and brain metastasis

Gross findings: A gray-whitish fibrotic plaque occupying the site of the left nipple was seen. Several firm elastic nodular lesions over left breast and right upper quadrant of abdominal wall were palpated. The brain was taken by the neurosurgeon and bilateral temporal lobes were removed. The remaining brain weighted 1050 gm. Grossly, no definite tumor nodules or plaques were seen on the surface of dura and brain. On serial section, a necrotic focus suspicious of tumor metastasis is seen in right basal ganglia and right occipital lobe respectively.

Histopathological findings: The left breast shows infiltrating lobular carcinoma with anaplastic tumor cells growing in an Indian file fashion and occasionally scattered signet ring cells in the fibrous stroma. Foci of lymphovascular permeation and perineural involvement are also noted. Immunohistochemical stain shows ER (-), PR (-), p53 (+++) and HER-2/neu (++). Sections from right basal ganglia and right occipital lobe show

intraparenchymal metastatic foci with adjacent reactive gliosis.

Leptomeningeal involvement of tumor was found in the subarachnoid spaces of midbrain, pons, medulla, right cerebellum, bilateral frontal lobes and right occipital lobe, and occasionally descending into involvement of the perivascular Virchow-Robin spaces. The morphological features are the same as the primary breast cancer and the immunohistochemical profile doesn't conflict with it.

Discussion: In addition to lymph nodes, the favorite sites of distant metastases of breast carcinoma are the lungs, bones, liver, adrenal glands, brain and meninges. However, metastases to the peritoneum/retroperitoneum, leptomeninges, GI tract, ovaries and uterus are more frequently observed in lobular carcinoma of breast. As a general rule, metastases are the most common tumors of the CNS. The most common primary sites of brain metastatic carcinoma are lung, breast, skin (melanoma), kidney, GI tract and unknown origin. Diffuse infiltration of the leptomeninges is most frequently associated with lymphoproliferative disorders, breast cancer, melanoma, lung cancer (particularly small cell carcinoma and adenocarcinoma) and carcinoma of GI tract.

The frequency of primary breast carcinoma metastasized to the brain is 15%-25%; on the contrary, about 20% of brain metastases come from breast carcinoma. The accurate frequency of leptomeningeal metastasis from breast cancer is uncertain. 80% of brain metastases are located in the arterial border zones of cerebral hemispheres, 3% in the basal ganglia, and 15% in the cerebellum. Besides, meningeal carcinomatosis is commonly associated with hematogenous metastasis.

Clinical features are dependent on location with variable presentation. Gd-DTPA enhanced MRI is the most sensitive method for detecting leptomeningeal metastasis as the continuous, thin and lineal high signal intensity on the brain surface that could be descend into the sulci.

Several gene expression patterns have been proposed involvement in pathogenesis of metastasis. For example, expression of metastasis-related genes such as EGFR, MMP-2, MDR-1 and KAI-1 is regulated by p53 tumor suppressor gene. Expression of c-erbB-2 (HER-2/neu) is associated with increased metastatic potential. Besides, chemokines and their receptors (CXCR4 and CCR7) are also involved in breast cancer metastasis.

Survival of meningeal carcinomatosis is relative poor. Median survival for patients with breast carcinoma who received intravenous chemotherapy is 20 weeks, but longer than those with other primary neoplasms.

Diagnosis criteria:

1. Infiltration of the subarachnoid space by small and relatively uniform tumor cells demonstrating features of primary breast cancer
2. The immunohistochemical profile consistent with the primary breast lesion

References:

1. Stacey E. Mills, et al. Sternberg's diagnostic surgical pathology, 4th ed. Lippincott Williams & Wilkins, 2004: 482-485.
2. Paul Kleihues, Webster K. Cavenee. Pathology and genetics of tumours of the nervous system. IARC Press, 2000: 250-253.
3. Vinay Kumar, Abul K. Abbas, Nelson Fausto. Robbins and Cotran pathologic basis of disease, 7th ed. Elsevier Saunders, 2005: 1410
4. David I. Graham, Peter L. Lantos. Greenfield's neuropathology, 7th ed. Arnold, 2002: II 971-975
5. Richard A. Prayson, John R. Goldblum. Neuropathology (A volume in the series foundations in diagnostic pathology). Elsevier Churchill Livingstone, 2005: 532-534
6. Sridhar Ramaswamy, et al. A molecular signature of metastasis in primary solid tumors. Nature Genetics. 2003; 33: 49-53
7. Anja Müller, et al. Involvement of chemokine receptors in breast cancer metastasis. Nature. 2001; 410: 50-56
8. Yi Sun, et al. Regulation of metastasis-related gene expression by p53: A potential clinical implication. Molecular Carcinogenesis. 1999; 24: 25-28
9. Antje Roetger, et al. Selection of potentially metastatic subpopulations expression c-erbB-2 from breast cancer tissue by use of an extravasation model. American Journal of Pathology. 1998; 153: 1797-1806
10. Robert Grant, et al. Clinical outcome in aggressively treated meningeal carcinomatosis. Arch Neurol. 1994; 51: 457-461

Comparative Pathology Case 244

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History: Adult (14 months of age) White Leghorn Hens (*Gallus gallus domesticus*) were bought from a farm at Nanto County in Taiwan. Hens have been vaccinated for Marek's disease, Newcastle disease, bronchitis and avian encephalomyelitis. A commercial diet for laying hens and drinking water were supplied *ad libitum*. Each hen was individually housed in a wire mesh cage, and was acclimatized for approximate 2 weeks before study. Hens were raised in air conditioned room with temperature 22-25°C, relative humidity 50-70% and 12L/12D photoperiod. Body weights ranging from 1.3 to 1.5 kg of hens were selected for this test. All hens were fasted overnight before treatment. Thirty minutes prior to administration, hens were subjected to an intramuscular injection of an atropine sulfate solution (purity > 98%, Fluka, Chemical, Tokyo, Japan) in deionized water at a dosage of 20 mg/kg. A single dose of 1,000 mg/kg Tri-orth-cresyl-phosphate (TOCP, practical grades, Fluka Chemical, Tokyo, Japan) in corn oil were gavaged at a constant volume of 10 ml/kg bw. Control hens received an equivalent volume of corn oil.

Clinical signs exhibited that of TOCP-treated hens showed salivation, depression, weakness and watery feces after 1 hour, and became cyanosis in comb after 24 hours. No mortality was noted and hens were completely recovered during 3 to 4 days after treatment. Hens exhibited mild to severe ataxia, decreased limb tone, impaired righting reflex, wing drop and reluctant to move starting from 14 days of treatment. An ataxia progressing with time and incoordination, inability to balance properly without the aid of the wings, paralysis of the legs, and the birds being unable to stand were noticed during 21-28 days of treatment.

Hens were sacrificed at the end of experiment on days 28. Firstly, hens were anesthetized by intravenous injection of sodium pentobarbital. Following anesthesia, perfusion was introduced with the amount of 250 ml of 10% buffered formaldehyde. Representative tissues include brain (cerebellum, cerebrum and medulla oblongata), spinal cord (cervical, thoracic and lumbosacral), sciatic and tibial nerve (left and right) were fixed in 10% buffered

formaldehyde solution for one week. Transverse and longitudinal sections of the central and peripheral nervous systems were processed for the preparation and microscopic examination of hematoxylin & eosin stained paraffin sections.

Diagnosis: Organophosphate induced delayed neurotoxicity in hens

Gross findings: No significant lesions in all organs were found at necropsy.

Histopathological findings: Hens in TOCP-treated group showed mild to severe axonal swelling, fragmentation and vacuolization that related to the axonal demyelination in the spinal cord, sciatic and tibia nerves. Transverse sections of the medulla had lesions restricted to the lateral white matter. In the cervical, thoracic and lumbosacral spinal cord sections were regularly noted in the dorsal lateral and lateral white matters. Changes in the sciatic nerve showed swelling, ballooning and fragmentation of axis cylinders and axonal lyses with accompanying vacuolization of the myelin sheath. A four step grading system: (-), no change absent; (+), equivocal histological changes, rare swollen axon without fragmentation, phagocytosis, or loss of myelin staining; (++) , mild to moderate degeneration of axon and myelin; (+++) , severe degeneration of axon and myelin, was used to define gradable lesions for comparison between dosage groups as mentioned by Abou-Donia et al. (1979). Result revealed that the incidence of axonal demyelination in TOCP-treated hens was 100%.

Discussion: Neurotoxicants, including organophosphorus (OP) insecticides, solvents, antibiotics, and lubricants, cause delayed neurotoxic effects that are a potential safety hazard and public health concern associated with their manufacture packaging, formulation, application, and field use. Most of the OP compounds show highly acute toxicity, with acute cholinergic effects. OPs can inhibit acetyl- cholinesterase (AChE) to degrade the neurotransmitter acetylcholine (ACh). Therefore, excessive ACh causes complete muscle paralysis and respiratory failure in acute intoxication. Cause of death by OP toxicosis is usually due to respiratory failure and consequent anoxia but may be cardiovascular in origin. Factors may contribute to respiratory failure including increased respiratory tract secretions, bronchiolar constriction, weakness of the muscles of respiration (hypoxia), and failure of the respiratory center (respiratory depression from nicotinic stimulation to the point of paralysis) (Ecobichon, 1994).

In addition to the acute neurotoxicity, exposure to some specific OPs is

also associated with the development of organophosphate-induced delayed neurotoxicity (OPIDN). Pathologically, OPIDN is characterized by axonal degeneration and demyelination of the central and peripheral nervous system axons. Typical signs of OPIDN include weakness, ataxia, paralysis and foot-drop of the extremities appearing 2-3 weeks and death after poison (Robberts, 1983). It is believed that some relative structures of OP insecticides, including phosphate, phosphoramidate and phosphonate, initiate from their ability to phosphorylate a protein in the nervous system, called neuropathy target esterase (NTE) (Ecobichon, 1994; Johnson, 1970; Pope et al., 1993). NTE, a nonspecific carboxylesterase (aliesterase or carboxylic acid ester hydrolase (EC, 3.1.1) with a molecular mass of 148 to 172, exhibited as membrane-bound protein to brain microsomes and smooth endoplasmic reticulum (Ecobichon, 1994). Some OPs inhibit NTE that is associated with OP-induced delayed neurotoxicity. Although the underlying mechanism remained unclear, a high degree of inhibition (70%) of NTE in brain and spinal cords after single exposure of some OPs resulting in ataxia 10 to 14 days later had been reported (Ecobichon, 1994).

To assess the potential of OP to induce OPIDN, its ability to inhibit AChE as an acute toxicity or to inhibit NTE as an indicator of neuropathic has been compared (Tain et al., 1998; Wilson et al., 1990). A severe AChE inhibition can result in lethality, while the degree of inhibition of NTE could be used to predict whether animals will survive long enough to develop delayed neuropathy. Therefore, the ratios of AChE and NTE in treated animals may be a better indicator of OPIDN (Tain et al., 1998; Wilson et al., 1990). Current guidelines for testing OPIDN are dose-related inhibition of AChE and NTE in brain and spinal cords of hens (OECD, 1995; USEPA, 19998). Inhibition of at least 70% of NTE activity by the irreversible binding is referred to as “aging reaction” of the insecticide. Some of OPs showed delayed neurotoxic effects, such as Mipafos, EPN, and Leptophos; all had been banned. Recent recommendations encouraged researchers to use *in vitro* methods for testing OPIDN instead of animals. Both primary cultures of neural cells and neuroblastoma cells have been suggested. These cells possess both AChE and NTE activity. Several investigators indicated that neuropathic OPs could be identified by NTE inhibition in human neuroblastoma cells (Ehrich et al., 1997). However, some OPs must be metabolized *in vivo* to much more potent metabolites before they can affect NTE activity (Barber et al., 1999). The alternative cellular study might provide a useful method to rapidly assess the OPIDN potency by using the ratios of AChE/NTE in human neuroblastoma cells (*in vitro*) comparing with the hen intoxication model (*in vivo*) for testing the potency of OPIDN substances in the future.

Diagnostic criteria: Scattered, patchy, degenerative changes of the myelin sheaths, axonal swelling, fragmentation and demyelination in the spinal cord and sciatic nerves.

References:

1. Abou-Donia, M.B., Graham D.G. and Komeil A.A. 1979. Delayed neurotoxicity of O-Ethyl O-4-Nitrophenyl-phenylphosphonothioate: Subchronic (90 days) oral administration in hens. *Toxicol Appl Pharmacol.* 45, 685-700.
2. Abou-Donia, M.B., Graham D.G. and Komeil A.A. 1979. Delayed neurotoxicity of O-Ethyl O-2,4-dichlorophenylphenylphosphonothioate: Effects of a single oral dose on hens. *Toxicol Appl Pharmacol.* 49, 293-303.
3. Barber, D., Correll, L., and Ehrich, M. 1999. Comparison of two in vitro activation systems for protoxicant organophosphorous esterase inhibitors. *Toxicol. Sci.* 47: 16-22.
4. Bickford, A.A., and Sprague, G.L. 1983. The significance of background neurologic lesions in acute delayed neurotoxicity studies: A comparison of neurohistopathologic lesions induced in commercial hens by ROCP with those observed in negative control hens. *NeuroToxicology* 4: 283-310.
5. Ecobichon, D. J. 1994. Organophosphorus ester insecticides. In *Pesticides and neurological disease*. D. J. Ecobichon, 2nd Edition. Chapter 4, pp. 171-249. CRC Press, USA.
6. Environmental Protection Agency Office of Pesticide & Toxic Substances. 1998. Acute and 28-Day Delayed Neurotoxicity of Organophosphorus Substances. In: *Health Effects Test Guidelines*, OPPTS 870.6100, EPA 712-C-98-237, August 1998. pp. 1-10. Washington DC.
7. Ehrich, M., Correll, L., and Veronesi. 1997. Acetylcholinesterase and neuropathy Tamaronget esterase inhibitions in neuroblastoma cells to distinguish organophosphorus compounds causing acute and delayed neurotoxicity. *Fund. Appl. Toxicol.* 35: 55-63.
8. Johnson, M. K., and Barnes, J. M. 1970. Short communications: Age and sensitivity of chicks to the delayed neurotoxic effects on some organophosphorus compounds. *Biochem. Pharmacol.* 19: 3045-3047.
9. Organization for Economic Cooperation and Development. 1995. Delayed Neurotoxicity of Organophosphorus Substances Following Acute Exposure. In: *OECD Guideline for Testing of Chemicals*. Section 4: Health Effects: No. 418. pp. 1-8. Adopted by the council on 24 July 1995, Paris, France.
10. Pope, C.N., Tanaka, D. Jr., and Padilla, S. 1993. The role of neurotoxic esterase (NTE) in the prevention and potentiation of

organophosphorus-induced delayed neurotoxicity (OPIDN). *Chem Biol Interact.* 87, 395-406.

11. Robberts, N. L., Farley, C., and Phillips, C. 1983. Screening, acute delayed and subchronic neurotoxicity studies in the hen: measurements and evaluations of clinical signs following administration of TOCP. *NeuroToxicology.* 4: 263-270.
12. Tian, Y., Xis, X., Piao, F., and Yamauchi, T. 1998. Delayed neuropathy and inhibition of soluble neuropathy target esterase following the administration of organophosphorus compounds to hens. *Tohoku J. Exp. Med.* 185: 161-171.
13. Wilson, B. W., Henderson, J. D., Kellner, T. P., McEuen, S. F., Griffis, L. C., Lai, J. C. 1990. Acetylcholinesterase and neuropathy target esterase in chickens and treated with acephate. *NeuroToxicology* 11: 483-492.

Comparative Pathology Case 245

Contributors: Jeng-Hung Suen (孫政宏), Fur-Jiang Leu (呂福江), Chia-Ing Jan (詹佳穎) Department of Pathology, Cardinal Tien's Hospital

Clinical history: A 78 years old female was sent to our ER since she had sudden onset of conscious change and bizarre posture last for few seconds. She had past history of hypertension, arrhythmia, and bipolar disorder. She had received regular medication from the cardiologist and psychiatrist and the disease control is fair. The physical examination and the laboratory data are both normal. Her conscious is clear and no sign of episode of bipolar disorder is found. After consultation with neurologist and psychiatrist, brain CT was suggested and performed which showed a sharply demarcated brain tumor in her right parietal area. Craniotomy with tumor removal was performed smoothly and she recovered after the operation.

Diagnosis: Microcystic Meningioma.

Gross findings: The specimen submitted consists of pieces of meningeal tissue attached with dura, measuring totally 4 x 3 x 2.5 cm, fixed in formalin. Grossly it showed a cystic tumor with solid component, which is spongy in consistency. The tumor is sharply demarcated and attached to the dura. No brain invasion is found grossly.

Histopathological finding: Microscopically, the tumor is composed of multiple intercellular spaces (microcyst), some of which containing mucin, admixed with vacuolated, and scattered pleomorphic nuclei. Many hyalinized vessels are also present. Whorls, well-form lobules, and psammoma bodies are absent. One large cyst is also noticed. The tumor cells are immunoreactive for EMA (epithelial membrane antigen).

Discussion: Microcystic meningioma is the most common variant of “non-classical” meningiomas, occurs both in pure form and as an admixture with other meningioma patterns. More often it is pure. Its non-meningothelial appearance creates diagnostic difficulty to those unfamiliar with its distinctive features. There is no special prognostic significance to the microcystic pattern. Microcystic meningiomas, especially in pure form, frequently cause diagnostic

confusion. Because there may be little in their appearance to suggest a meningotheial derivation, a pathologist may consider a diagnosis of hemangioblastoma or angioblastic meningioma (hemangiopericytoma). The resemblance to hemangioblastoma is created by the frequent presence of scattered pleomorphic nuclei as well as cells resembling lipid-laden “stromal” cells.¹ Microcystic meningioma contains innumerable, pale mucin-containing intercellular microcysts encircled by thin tumor cell processes compared with xanthoma cells, which is predominant in hemangioblastoma. Particularly in frozen sections or smears preparation, microcystic meningiomas may mimic hemangioblastoma as well as microcyst-rich pilocytic astrocytomas. Awareness of radiologic and operative data is essential (radiologic features, location, age, arise from leptomeninges, etc). Uncertainty about the nature of an individual lesion can be resolved simply by immunohistochemistry; the meningotheial lineage of a microcystic neoplasm is affirmed by EMA immunoreactivity and absence of GFAP stainability,

Meningiomas are histologically heterogeneous, with new subtypes added on what seems almost an annual basis. Most common subtypes are meningotheial, fibrous, and transitional. Most subtypes share a common clinical behavior (benign course with low recurrence, grade I), including meningotheial, fibrous, transitional,,psammomatous, angiomatous, microcystic, secretory, lymphoplasmacyte-rich, and metaplastic meningioma. While some subtypes are more likely to recur and follow a more aggressive clinical course, including atypical, clear cell and chordoid meningioma (grade II), and anaplastic, rhabdoid and papillary meningioma (grade III).

Grading of meningioma is now as much a part of specimen analysis as it is for gliomas. The procedure must consider both classical histologic features, and the tumor subtypes. Numerous studies have identified the negative prognostic factors including tumor location, incomplete resection, and histologic and immunohistochemical features with prognostic significance, such as invasion of the brain, increeeased mitotic rate ($>4/10\text{HPF}$), hypercellularity, small cell change, cytologic atypia with macronucleoli, loss of pattern (sheeting), tumor necrosis, elevated MIB-1 labeling index ($>4\%$), cellular anaplasia, histologic subtype (rhabdoid, papillary, chordoid, clearr cell), and absence of immunoreactivity for progesterone receptors. Careful gross and microscopic examination and adequate sampling especially on the surface (for brain invasion) are essential for diagnosis and grading of meningioma.

Diagnostic criteria:

1. Innumerable intercellular, pale, mucin containing microcysts encircled by thin tumor cell processes.
2. Scattered large dark nuclei.
3. Copious hyalinized vessels.
4. Absence of well-formed lobules, whorls, and psammoma bodies commonly seen in classical meningioma (meningotheliomatous, transitional, fibroblastic, and psammomatous)
5. Immunoreactive for EMA

References:

1. Peter C. Burger, et al. Surgical Pathology of the Nervous System and its Coverings. 4th ed. Churchill Livingstone 2002.
2. Perry A, Stafford SL, et al. Meningioma grading: An Analysis of Histologic Parameter. The American Journal of Surgical Pathology 1997; 21(12):1455-1465
Perry A, Stafford SL, Scheithauer BW, et al. "Malignancy" in meningiomas: a Clinocopathologic study of 116 patients with grading implications. Cancer 1999;85:2046-2056
4. De Jesus O, Rifkinson N, Negron B. Cystic meningiomas: a review. Neurosurgery 1995;36:599-605
5. Ng HK, Tse CCH, Lo STH. Microcystic meningioma- an unusual morphological variant of meningiomas. Histopathology 1989;14:1-9

Comparative Pathology Case 246

Contributors: Che-Ming Shu¹ (許哲銘), DVM; Chen I Liu² (劉貞怡), DVM, MS; Chung-Tiang Liang(梁鍾鼎) DVM, MS¹, Chen-Hsuan Liu¹ (劉振軒), DVM, PhD.

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Clinical History: The 1-year-old female pug dog was referred to the emergency severe with sudden onset of seizure-like episode, hemiparesis of right limbs, anisocoria and ptialism on Jan 22. Results of a complete blood count and serum biochemical analyses were within reference ranges. Sampling whole blood for canine distemper PCR was negative. During hospitalization, fever (40-41°C) sometimes happened. The dog was treated with prednisolone, doxycycline and diazepam and gradually improved. The dog was discharged on the 7th day. The dog returned on June 14 because of seizures and ataxia. After treatment with prednisolone and phenobarbital, the dog improved for a few days then the symptoms gradually worsens. On July 10, the most conspicuous findings were a head tilt and circle to the right, tetraparesis, and nystagmus. Pupil light reflexes were normal bilaterally. Menace responses were absent. The dog had normal appetite but crying when she was awake. Cerebrospinal fluid evaluation revealed normal protein content and the predominant cell type was the small lymphocyte. Despite therapy, the dog's neurological status worsened. Based on poor prognosis, the owner requested euthanasia.

Diagnosis: Pug dog encephalitis

Gross Findings: The vessels over the leptomeninges were hyperemia. The gyri of the cerebral hemispheres were flattened and thin cortex of bilateral pyriform lobe. Transverse sections of brain showed severe asymmetrical dilation of the lateral ventricles containing clear fluids, and discoloration, malacia or cavitation.

Histopathological Findings: In the cerebrum section, the lateral ventricles are extremely dilated with flattened ependymal cells. There is a severe necrotizing meningoencephalitis involving the leptomeninges, gray and white matter. The parenchyma is rarefied and parenchymal infiltrates of lymphocytes, plasma cells, and histocytes; many mononuclear cells have small, round,

dense nuclei and are difficult to identify. Reactive astrogliosis is extensive. Multifocal inflammatory change in the cerebral cortex, vacuolation and laminar cortical necrosis are also found. Severe lymphoplasmacytic perivascular cuffing is found in the meninges of cerebellum, but less in cerebellum parenchyma. Necrotic foci are also present between molecular layer and granular cell layer.

Laboratory Results:

- Immunohistochemistry:
CDV: Negative for cerebellum.
- Polymerase chain reaction:
CDV: Negative for peripheral whole blood.

Discussion: Canine necrotizing meningoencephalitis (NME) is a unique inflammatory disorder in small-sized breed dogs, especially in Pug dogs, and similar condition have been described in Maltese, Shin Tzu, and Chihuahua.

The cause of necrotizing meningoencephalitis in Pug dogs is still unknown. Although several authors have suggested that some viruses such as canine distemper virus or the canine herpes virus may be responsible for the initial pathogenesis of Pug dog encephalitis, there has been no evidence to support an infectious etiology in this unique disease. Several differential etiologies other than viral infections have been studied, including canine granulomatous meningoencephalitis, toxoplasmosis, seizure-related cortical necrosis and metabolic causes, but these would be unlikely primary events in Pug dog encephalitis.

Lesion of NME should be differentiated with granulomatous meningoencephalomyelitis (GME) in pug dog. Gross observation exhibited lateral ventricular dilation and discoloration, malacia and/or cavitation of the cerebrum in NME. On the contrary, gross changes were milder in GME, except for occasional visible granulomatous mass formation. Histopathologically, the lesions of NME were distributed predominantly in the cerebral cortex and various degrees of inflammatory and necrotic changes were observed according to clinical stages. Besides, microscopic lesions of GME were mainly distributed in the white matter of the cerebrum, cerebellum and brainstem, which are characterized by perivascular cuffing, multiple granulomas and leptomeningeal infiltrates.

In the previous study, prevalence of the anti-astrocyte autoantibody was highly specific to NME and GME, and indicates that the anti-astrocyte autoantibody could be used as a novel hallmark of NME and/or GME.

Diagnostic Criteria:

In H&E section, lymphocytic perivascular cuffing are obvious in the cortex and meninges of brain in pug dog, especially in the cerebrum. Malacia, microglial nodule hyperplasia, lamina cortical necrosis, various degree of inflammation and necrotic changes are observed. Occasionally, various degree of hydrocephalus may also be found in the lateral ventricles.

Laboratory examination revealed negative for virus, bacteria, parasites, fungi and saprophytes in specimens.

References:

1. Matsuki N, Fujiwara K, Tamahara S, Uchida K, Matsunaga S, Nakayama H, Doi K, Ogawa H, Ono K. Prevalence of autoantibody in cerebrospinal fluids from dogs with various CNS diseases. J Vet Med Sci 66: 295-7, 2004.
2. Polymerase chain reaction screening for DNA viruses in paraffin-embedded brains from dogs with necrotizing meningoencephalitis, necrotizing leukoencephalitis, and granulomatous meningoencephalitis. J Vet Intern Med 19: 553-559, 2005.
3. Suzuki M, Uchida K, Morozumi M, Hasegawa T, Yanai T, Nakayama H, Tateyama S. A comparative pathological study on canine necrotizing meningoencephalitis and granulomatous meningoencephalomyelitis. J Vet Med Sci 65: 1233-9, 2003.
4. Uchida K, Hasegawa T, Ikeda M, Yamaguchi R, Tateyama S. Detection of an autoantibody from Pug dogs with necrotizing encephalitis (Pug dog encephalitis). Vet Pathol 36: 301-7, 1999.

中華民國比較病理學會
第一次至第三十四次比較病理學研討會病例分類一覽表

分 類	病 例 編 號	診 斷	動 物 別	提 供 單 位
腫 瘤	1.	Myxoma	Dog	美國紐約動物醫學中心
	2.	Chordoma	Ferret	美國紐約動物醫學中心
	3.	Ependymoblastoma	Human	長庚紀念醫院
	8.	Synovial sarcoma	Pigeon	美國紐約動物醫學中心
	18.	Malignant lymphoma	Human	長庚紀念醫院
	19.	Malignant lymphoma	Wistar rat	國家實驗動物繁殖及研究中心
	24.	Metastatic thyroid carcinoma	Human	省立新竹醫院
	25.	Chordoma	Human	新光吳火獅紀念醫院
	34.	Interstitial cell tumor	Dog	中興大學獸醫學系
	35.	Carcinoid tumor	Human	長庚紀念醫院
	36.	Hepatic carcinoid	Siamese cat	美國紐約動物醫學中心
	38.	Pheochromocytoma	Ferret	美國紐約動物醫學中心
	39.	Extra adrenal pheochromocytoma	Human	新光吳火獅紀念醫院
	40.	Mammary gland fibroadenoma	Rat	國家實驗動物繁殖及研究中心
	41.	Fibroadenoma	Human	省立豐原醫院
	42.	Canine benign mixed type mammary gland tumor	Pointer bitch	中興大學獸醫學系
	43.	Phyllodes tumor	Human	台中榮民總醫院
	44.	Canine oral papilloma	Dog	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

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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	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	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	Human	佛教慈濟綜合醫院

	mixed tumor)		
103.	Atypical central neurocytoma	Human	新光吳火獅紀念醫院
104.	Cardiac schwannoma	SD rat	國家實驗動物繁殖及研究中心
109.	Desmoplastic infantile ganglioglioma	Human	高雄醫學院
107.	1.Primary cerebral malignant lymphoma 2.Acquired immune deficiency syndrome	Human	台北市立仁愛醫院
111.	Schwannoma	Human	三軍總醫院
114.	Osteosarcoma	Dog	美國紐約動物醫學中心
115.	Mixed germ-cell stromal tumor, mixed sertoli cell and seminoma-like cell tumor	Dog	美國紐約動物醫學中心
116.	Krukenberg's Tumor	Human	台北病理中心
117.	Primary insular carcinoid tumor arising from cystic teratoma of ovary.	Human	花蓮慈濟綜合醫院
119.	Polypoid adenomyoma	Human	大甲李綜合醫院
120.	Gonadal stromal tumor	Human	耕莘醫院
122.	Gestational choriocarcinoma	Human	彰化基督教醫院
123.	Ovarian granulosa cell tumor	Horse	中興大學獸醫學系
129.	Kaposi's sarcoma	Human	華濟醫院
131.	Basal cell carcinoma (BCC)	Human	羅東聖母醫院
132.	Transmissible venereal tumor	Dog	臺灣大學獸醫學系
137	Canine Glioblastoma Multiforme in Cerebellopontine Angle	Dog	中興大學獸醫病理研究所
143	Osteosarcoma associated with metallic implants	Dog	紐約動物醫學中心
144	Radiation-induced osteogenic sarcoma	Human	花蓮慈濟綜合醫院
145	Osteosarcoma, osteogenic	Dog	臺灣大學獸醫學系
146	Pleomorphic rhabdomyosarcoma	Human	行政院衛生署新竹醫院
147	Papillary Mesothelioma of pericardium	Leopard	屏東科大學獸醫學系
148	Cystic ameloblastoma	Human	台北醫學院
149	Giant cell tumor of bone	Canine	中興大學獸醫學院
150	Desmoplastic small round cell tumor (DSRCT)	Human	華濟醫院
152	Hepatocellular carcinoma	Human	羅東聖母醫院
158	Hemangiopericytoma	Human	羅東聖母醫院
160	Cardiac fibroma	Human	高雄醫學大學病理學

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166	Nephroblastoma	Rabbit	紐約動物醫學中心
168	Nephroblastoma	Pig	台灣動物科技研究所
169	Nephroblastoma with rhabdomyoblastic differentiation	Human	高雄醫學大學病理科
172	Spindle cell sarcoma	Human	羅東聖母醫院
174	Juxtaglomerular cell tumor	Human	新光醫院病理檢驗科
190	Angiosarcoma	Human	高雄醫學大學病理學科
192	Cardiac myxoma	Human	彰化基督教醫院病理科
194	Kasabach-Merrit syndrome	Human	慈濟醫院病理科
195	Metastatic hepatocellular carcinoma, right atrium	Human	新光醫院病理科
197	Papillary fibroelastoma of aortic valve	Human	新光醫院病理科
198	Extraplacental chorioangioma	Human	耕莘醫院病理科
208	Granulocytic sarcoma (Chloroma) of uterine cervix	Human	高雄醫學大學病理學科
210	Primary non-Hodgkin's lymphoma of bone, diffuse large B cell, right humerus	Lymphoma	彰化基督教醫院病理科
213	Lymphoma, multi-centric type	Dog	中興大學獸醫系
214	CD30 (Ki-1)-positive anaplastic large cell lymphoma (ALCL)	Human	新光醫院病理科
215	Lymphoma, mixed type	Koala	台灣大學獸醫學系
217	Mucosal associated lymphoid tissue (MALT) lymphoma, small intestine	Cat	臺灣大學獸醫學研究所
218	Nasal type NK/T cell lymphoma	Human	高雄醫學大學病理科
222	Acquired immunodeficiency syndrome (AIDS) with disseminated Kaposi's sarcoma	Human	慈濟醫院病理科
224	Epithelioid sarcoma	Human	彰化基督教醫院病理科
226	Cutaneous B cell lymphoma , eyelid , bilateral	Human	羅東聖母醫院病理科
227	Extramammary Paget's disease (EMPD) of the scrotum	Human	萬芳北醫皮膚科, 病理科
228	Skin, back, excision, CD30+diffuse large B cell lymphoma, Soft tissue, leg , side not stated, excision, vascular leiomyoma	Human	高雄醫學大學附設醫院病理科

	231	Malignant melanoma, metastasis to intra-abdominal cavity	Human	財團法人天主教耕莘醫院病理科
	232	Vaccine-associated rhabdomyosarcoma	Cat	台灣大學獸醫學系
	233	1. Pleura: fibrous plaque, 2. Lung: adenocarcinoma, 3. Brain: metastatic adenocarcinoma	Human	高雄醫學大學附設中和醫院病理科
	235	1. Neurofibromatosis, type I 2. Malignant peripheral nerve sheath tumor (MPNST)	Human	花蓮慈濟醫院病理科
細菌	6.	Tuberculosis	Monkey	臺灣大學獸醫學系
	7.	Tuberculosis	Human	省立新竹醫院
	12.	H. pylori-induced gastritis	Human	台北病理中心
	13.	Pseudomembranous colitis	Human	省立新竹醫院
	26.	Swine salmonellosis	Pig	中興大學獸醫學系
	27.	Vegetative valvular endocarditis	Pig	台灣養豬科學研究所
	28.	Nocardiosis	Human	台灣省立新竹醫院
	29.	Nocardiosis	Largemouth bass	屏東縣家畜疾病防治所
	32.	Actinomycosis	Human	台灣省立豐原醫院
	33.	Tuberculosis	Human	苗栗頭份為恭紀念醫院
	53.	Intracavitary aspergilloma and cavitory 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	中興大學獸醫學系
	108.	Listeric encephalitis	Lamb	屏東縣家畜疾病防治所

	113.	Tuberculous meningitis	Human	羅東聖母醫院
	134.	Swine salmonellosis with meningitis	Swine	中興大學獸醫學系
	135.	Meningoencephalitis, fibrinopurulent and lymphocytic, diffuse, subacute, moderate, cerebrum, cerebellum and brain stem, caused by Streptococcus spp. infection	Swine	國家實驗動物繁殖及研究中心
	140	Coliform septicemia of newborn calf	Calf	屏東縣家畜疾病防治所
	161	Porcine polyserositis and arthritis (Glasser's disease)	Pig	中興大學獸醫學院
	162	Mycotic aneurysm of jejunal artery secondary to infective endocarditis	Human	慈濟醫院病理科
	170	Chronic nephritis caused by Leptospira spp	Pig	中興大學獸醫學院
	173	Ureteropyelitis and cystitis	Pig	中國化學製藥公司
病毒	21.	Newcastle disease	Chickens	台灣大學獸醫學系
	22.	Herpesvirus infection	Goldfish	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	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
	128.	Contagious pustular dermatitis	Goat	屏東縣&台東縣家畜疾病防治所
	130.	Fowl pox and Marek's disease	Chicken	中興大學獸醫學系
	133.	Japanese encephalitis	Human	花蓮佛教慈濟綜合醫院
	136	Viral encephalitis, polyomavirus infection	Lory	美國紐約動物醫學中心
	138	1.Aspergillus spp. encephalitis and myocarditis	Dog	台灣大學獸醫學系

	2.Demyelinating canine distemper encephalitis		
153	Enterovirus 71 infection	Human	彰化基督教醫院
154	Ebola virus infection	African Green monkey	行政院國家科學委員會實驗動物中心
155	Rabies	Longhorn Steer	台灣大學獸醫學系
163	Parvoviral myocarditis	Goose	屏東科技大學獸醫學系
199	SARS	Human	台大醫院病理科
200	TGE virus	swine	臺灣動物科技研究所
201	Feline infectious peritonitis(FIP)	Feline	台灣大學獸醫學系
209	Chicken Infectious Anemia (CIA)	Layer	屏東防治所
219	1.Lymph node:Lymphdenitis, with lymphocytic depletion and intrahistiocytic basophilic cytoplasmic inclusion bodies. Etiology consistent with Porcine Circovirus(PCV)infection. 2.Lung: Bronchointerstitial pneumonia,moderate, lymphoplasmacytic, subacute.	Pig	臺灣動物科技研究所
220	Cytomegalovirus colitis	Human	彰化基督教醫院病理科
221	Canine distemper virus Canine adenovirus type II co-infection	Canine	國家實驗動物繁殖及研究中心
223	1. Skin, mucocutaneous junction (lip): Cheilitis, subacute, diffuse, sever, with epidermal pustules, ballooning degeneration, proliferation, and eosinophilic intracytoplasmic inclusion bodies, Saanen goat. 2. Haired skin: Dermatitis, proliferative, lymphoplasmacytic, subacute, diffuse, sever, with marked epidermal pustules, ballooning degeneration, acanthosis, hyperkeratosis, and eosinophilic intracytoplasmic inclusion bodies.	Goat	台灣動物科技研究所
黴菌	23. Chromomycosis	Human	台北病理中心
	47. Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma.	Human	三軍總醫院

		Adrenal gland, right: carcinoma (primary)		
	48.	Adiaspiromycosis	Wild rodents	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
	127.	Eumycotic mycetoma	Human	花蓮佛教慈濟綜合醫院
	138	1.Aspergillus spp. encephalitis and myocarditis 2.Demyelinating canine distemper encephalitis	Dog	台灣大學獸醫學系
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	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 by Toxocara canis larvae migration	Dog	臺灣養豬科學研究所
	139	Disseminated strongyloidiasis	Human	花蓮佛教慈濟綜合醫院
	141	Eosinophilic meningitis caused by Angiostrongylus cantonensis	Human	台北榮民總醫院病理檢驗部
	156	Parastrongylus cantonensis infection	Formosan gem-faced civet	中興大學獸醫學院
	157	Capillaria hepatica, Angiostrongylus cantonensis	Norway Rat	行政院農業委員會農業藥物毒物試驗所
	202	Colnorchiasis	Human	高雄醫學院附設醫院

	203	Trichuriasis	Human	彰化基督教醫院
	204	Psoroptes cuniculi infection (Ear mite)	Rabbit	農業藥物毒物試驗所
	205	Pulmonary dirofilariasis	Human	和信治癌中心醫院
	206	Capillaries philippinesis	Human	和信治癌中心醫院
	207	Adenocarcinoma with schistosomiasis	Human	花蓮佛教慈濟綜合醫院
原蟲	4.	Cryptosporidiosis	Goat	台灣養豬科學研究所
	15.	Amoebiasis	Lemur fulvus	台灣養豬科學研究所
	16.	Toxoplasmosis	Squirrel	台灣養豬科學研究所
	17.	Toxoplasmosis	Pig	屏東技術學院獸醫學系
	51.	Pneumocystis carinii pneumonia	Human	台北病理中心
	57.	Cecal coccidiosis	Chicken	中興大學獸醫學系
	65.	Cryptosporidiosis	Carprine	台灣養豬科學研究所
	211	Avian malaria, African black-footed penguin	Avian	臺灣動物科技研究所
立克次體	70.	Acute Q fever hepatitis	Human	佛教慈濟綜合醫院
	229	Necrotizing inflammation due to scrub typhus	Human	佛教慈濟醫院病理科
皮膚	216	Cytophagic histiocytic panniculitis with terminal hemophagocytic syndrome	Human	佛教慈濟綜合醫院病理科
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	中興大學獸醫學系
	76.	Polycystic kidney bilateral and renal failure	Cat	美國紐約動物醫學中心
	151	Osteodystrophia fibrosa	Goat	台灣養豬科學研究所 & 台東縣家畜疾病防治所
	80.	1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate 2.Benign hypertension	SHR rat	國防醫學院 & 國家實驗動物繁殖及研究中心
	83.	Phagolysosome-overload nephropathy	SD rats	實驗動物繁殖中心
	85.	Renal amyloidosis	Dog	台灣養豬科學研究所
	89.	1.Severe visceral gout due to kidney damaged 2.Infectious serositis	Goose	中興大學獸醫學系

91.	Hypervitaminosis D	Orange-rumped agoutis	台灣大學獸醫學系
118.	Cystic endometrical hyperplasia	Dog	臺灣養豬科學研究所
121.	Cystic subsurface epithelial structure (SES)	Dog	國科會實驗動物中心
124.	Superficial necrolytic dermatitis	Dog	美國紐約動物醫學中心
125.	Solitary congenital self-healing histiocytosis	Human	羅東博愛醫院
126.	Alopecia areata	Mouse	實驗動物繁殖及研究中心
142	Avian encephalomalacia (Vitamin E deficiency)	Chicken	國立屏東科技大學獸醫學系
159	Hypertrophic cardiomyopathy	Pig	台灣大學獸醫學系
165	Chinese herb nephropathy	Human	三軍總醫院病理部及腎臟科
167	Acute pancreatitis with rhabdomyolysis	Human	慈濟醫院病理科
171	Malakoplakia	Human	彰化基督教醫院
183	Darier's disease	Human	高雄醫學大學病理科
191	1. Polyarteritis nodosa 2. Hypertrophic Cardiomyopathy	Feline	台灣大學獸醫學系
193	Norepinephrin cardiotoxicity	Cat	台中榮總
196	Cardiomyopathy (Experimental)	Mice	綠色四季
212	Kikuchi disease (histiocytic necrotizing lymphadenitis)	Lymphadenitis	耕莘醫院病理科
225	Calcinosis circumscripta, soft tissue of the right thigh, dog	Dog	台灣大學獸醫所
230	Hemochromatosis, liver, bird	Bird	台灣大學獸醫學系
234	Congenital hyperplastic goiter	Holstein calves	屏東縣家畜疾病防治所
236	Hepatic lipidosis (fatty liver)	Rats	中興大學獸醫學病理學研究所

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

350 苗栗縣竹南鎮頂埔里科東二路 52 號

台灣動物科技研究所動物醫學組 病理室收

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-----中華民國比較病理學會-----

會員資料更改卡

姓 名：_____ 會員類別：☐ 一般會員

☐ 學生會員

☐ 贊助會員

最高學歷：_____

服務單位：_____ 職 稱：_____

永久地址：_____

通訊地址：_____

電 話：_____ 傳 真：_____

E-Mail Address：_____

中 華 民 國 比 較 病 理 學 會

誠摯邀請您加入

入 會 辦 法

一、 本會會員申請資格為：

- (一) 一般會員：贊同本會宗旨，年滿二十歲，具有國內外大專院校（或同等學歷）生命科學及其它相關科系畢業資格或高職畢業從事生命科學相關工作滿兩年者。
- (二) 學生會員：贊同本會宗旨，在國內、外大專院校生命科學或其他相關科系肄業者（請檢附學生身份證明）。
- (三) 贊助會員：贊助本會工作之團體或個人。
- (四) 榮譽會員：凡對比較病理學術或會務之推廣有特殊貢獻，經理事會提名並經會員大會通過者。

二、 會員：

- (一) 入 會 費：一般會員新台幣一仟元，學生會員一百元，贊助會員伍仟元，於入會時繳納。
- (二) 常年會費：一般會員新台幣伍佰元，學生會員一百元。

【註：學生會員身份變更為一般會員時，只需繳交一般會員之常年會費】

- ### 三、 請填妥入會申請表郵寄或傳真方式寄回中華民國比較病理學會秘書處收。地址：350 苗栗縣竹南鎮頂埔里科東二路 52 號 台灣動物科技研究所動物醫學組 電話：037-585872、傳真 037-585850。