

中華民國比較病理學會
九十六年度第四十一次比較病理學研討會

【「困難罕見疾病之病理診斷」或其他比較病理相關病例】



主辦單位：中華民國比較病理學會
台灣大學獸醫學系

時間：中華民國九十六年十一月十七日（星期六）

地點：臺北市羅斯福路四段1號

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中華民國比較病理學會九十六年度會員大會暨第四十一次比較病理學研討會
【「困難罕見疾病之病理診斷」或其他比較病理相關病例】
議程表

時間：中華民國九十六年十一月十七日（星期六）上午 08：40~下午 17：00

地點：台灣大學獸醫學系 B01 演講廳

地址：臺北市羅斯福路四段 1 號 TEL: 02-33663854 (<http://www.vm.ntu.edu.tw/index.htm>)

主辦單位：中華民國比較病理學會 台灣大學獸醫學系

時 間	議 程
08:40~09:00	報 到 主持人
09:00~09:10	主席致詞 呂福江 理事長
09:10~10:00	【專題演講 I】 Hemangiogenesis 之研究與展望 日本德島文理大學 藥學部機能形態學教 室 井上正九 教授 (Masahisa Inoue) 阮正雄 主任
10:00~10:30	【專題演講 II】 犬癌症之免疫療法 台灣大學獸醫學系 朱瑞民 教授 劉振軒 教授
10:30~10:50	Coffee Break
10:50~11:30	【專題演講 III】 Lab Animal Health Monitoring: “Healthy” Is Not “Healthy” 台灣大學獸醫學系 萬灼華 助理教授 劉振軒 教授
11:30~12:00	病例討論 Case 281 羅東博愛醫院 施洽雯 主任
12:00~13:30	午 餐 (中華民國比較病理學會理監事會議)
13:30~14:00	病例討論 Case 282 財團法人天主教耕莘醫院 江蓉華 主任 張俊梁 主任
14:00~14:30	病例討論 Case 283 國立中興大學獸醫病理學研究 所 廖俊旺 博士
14:30~15:00	病例討論 Case 284 花蓮慈濟醫院病理科 李明勳 醫師
15:00~15:20	Rest
15:20~15:50	病例討論 Case 285 財團法人新光吳火獅紀念醫院 劉淨蘭 醫師 施洽雯 主任
15:50~16:20	病例討論 Case 286 國家實驗動物中心 梁鍾鼎 研究員
16:20~16:50	病例討論 Case 287 國立台灣大學獸醫學研究所 許家達 獸醫師

16:50~17:20

綜 合 討 論

中華民國比較病理學會章程

第一章 總則

- 第一條 本會定名為中華民國比較病理學會，英文名稱為 **Chinese Society of Comparative Pathology (CSCP)** (以下簡稱本會)
- 第二條 本會依內政部人民團體法設立，為非營利目的之社會團體，以結合人類醫學與動物醫學資源，提倡比較病理學之研究與發展，交換研究教學心得，聯絡會員友誼及促進國際間比較醫學之交流為宗旨。
- 第三條 本會以全國行政區域為組織區域，會址設於主管機關所在地區，並得報經主管機關核准設主分支機構。前項分支機構組織簡則由理事會擬訂，報請主管機關核准後行之。會址及分支機構之地址於設置及變更時應報請主管機關核備。
- 第四條 本會之任務如左：
- 一、 提倡比較病理學之研究與發展。
 - 二、 舉辦學術演講會、研討會及相關訓練課程。
 - 三、 建立國內比較醫學相關資料庫。
 - 四、 發行比較病理學相關刊物。
 - 五、 促進國內、外比較醫學之交流。
 - 六、 其他有關比較病理學術發展之事項。
- 第五條 本會之主管機關為內政部。目的事業主管機關依章程所訂之宗旨與任務，主要為行政院衛生署及農業委員會，其目的事業應受各該事業主管機關之指導與監督。

第二章 會員

- 第六條 本會會員申請資格如下：
- 一、 一般會員：贊同本會宗旨，年滿二十歲，具有國內外大專院校(或同等學歷)生命科學及其它相關科系畢業資格或高職畢業從事生命科學相關工作滿兩年者。
 - 二、 學生會員：贊同本會宗旨，在國內、外大專院校生命科學或其它相關科系肄業者 (檢附學生身份證明)。
 - 三、 贊助會員：贊助本會工作之團體或個人。
 - 四、 榮譽會員：凡對比較病理學術或會務之推展有特殊貢獻，經理事會提名並經會員大會通過者。
- 前項一、二、三項會員申請時應填具入會申請書，經一般會員二人之推

薦，經理事會通過，並繳納會費。學生會員身份改變成一般會員時，得再補繳一般會員入會費之差額後，即成為一般會員，榮譽會員免繳入會費與常年會費。

第七條 一般會員有表決權、選舉權、被選舉與罷免權，每一會員為一權。贊助會員、學生會員與榮譽會員無前項權利。

第八條 會員有遵守本會章程、決議及繳納會費之義務。

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

第十條 會員喪失會員資格或經會員大會決議除名者，即為出會。

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

第三章 組織及職員

第十二條 本會以會員大會為最高權力機構。

第十三條 會員大會之職權如下：

- 一、 訂定與變更章程。
- 二、 選舉及罷免理事、監事。
- 三、 議決入會費、常年會費、事業費及會員捐款之方式。
- 四、 議決年度工作計畫、報告、預算及決算。
- 五、 議決會員之除名處置。
- 六、 議決財產之處分。
- 七、 議決本會之解散。
- 八、 議決與會員權利義務有關之其他重大事項。

前項第八款重大事項之範圍由理事會訂定之。

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

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

本屆理事會得提出下屆理事及監事候選人參考名單。

第十五條 理事會之職權如下：

- 一、 審定會員之資格。
- 二、 選舉及罷免常務理事及理事長。
- 三、 議決理事、常務理事及理事長之辭職。
- 四、 聘免工作人員。
- 五、 擬訂年度工作計畫、報告、預算及決算。
- 六、 其他應執行事項。

第十六條 理監事置常務理事五人，由理事互選之，並由理事就常務理

- 事中選舉一人為理事長。
理事長對內綜理監督會議，對外代表本會，並擔任會員大會、理事會主席。
理事長因事不能執行職務時，應指定常務理事一人代理之，未指定或不能指定時，由常務理事互推一人代理之。
理事長或常務理事出缺時，應於一個月內補選之。
- 第十七條 監事會之職權如左：
一、監察理事會工作之執行。
二、審核年度決算。
三、選舉及罷免常務監事。
四、議決監事及常務監事之辭職。
五、其他應監察事項。
- 第十八條 監事會置常務監事一人，由監事互選之，監察日常會務，並擔任監事會主席。
常務監事因事不能執行職務時，應指定監事一人代理之，未指定或不能指定時，由監事互推一人代理之。監事會主席（常務監事）出缺時，應於一個月內補選之。
- 第十九條 理事、監事均為無給職，任期三年，連選得連任。理事長之連任以一次為限。
- 第二十條 理事、監事有下列情事之一者，應即解任：
一、喪失會員資格。
二、因故辭職經理事會或監事會決議通過者。
三、被罷免或撤免者。
四、受停權處分期間逾任期二分之一者。
- 第二十一條 本會置祕書長一人，承理事長之命處理本會事務，令置其他工作人員若干人，由理事長提名經理事會通過後聘免之，並報主管機關備查。但祕書長之解聘應先報主管機關核備。
前項工作人員不得由選任之職員（理監事）擔任。
工作人員權責及分層負責事項由理事會令另定之。
- 第二十二條 本會得設各種委員會、小組或其它內部作業組織，其組織簡則由理事會擬定，報經主機關核備後施行，變更時亦同。
- 第二十三條 本會得由理事會聘請無給顧問若干人，其聘期與理事、監事之任期同。

第四章 會議

- 第二十四條 會員大會分定期會議與臨時會議兩種，由理事長召集，召集時除緊急事故之臨時會議外應於十五日前以書面通知之。定期會

議每年召開一次，臨時會議於理事會過半數認為必要，或經會員五分之一以上之請，或監事會半數函請召集時召開之。

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

第二十六條 會員大會之決議，以出席人數過半之同意行之。但章程之訂定與變更、會員之除名、理事及監事之罷免、財產之處置、本會之解散及其他與會權利義務有關之重大事項應有出席人數三分之二以上同意。但本會如果辦理法人登記後，章程之變更應以出席人數四分之三以上之同或全體會員三分之二以上書面之同意行之。

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

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

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

第五章 經費及會計

第二十九條 本會經費來源如下：

- 一、入會費：一般會員新台幣壹仟元，學生會員壹佰元，贊助會員伍仟元，於入會時繳納。
- 二、常年會費：一般會員新台幣五百元，學生會員壹佰元。
- 三、事業費。
- 四、會員捐款。
- 五、委託收益。
- 六、基金及其孳息。
- 七、其他收入。

第三十條 本會會計年度以國曆年為準，自每年一月一日起至十二月三十一日止。

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

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

第三十三條 本章程未規定事項，悉依有關法令規定辦理。

第三十四條 本章程經大會通過，報經主管機關核備後施行，變更時亦同。

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

專 題 演 講 II

Immune responses in canine cancer patients and their applications

Rea-Min Chu

Department of Veterinary Medicine, National Taiwan University

Cancer is becoming number one cause of death in canine in many countries. To date, the most commonly used treatments are surgery, chemotherapy, and radiotherapy, which are also proven as an inefficient way to prevent a tumor from recurrence. The recurrence rates for the dogs in their second and third stage of malignant mammary gland tumors (MGT) (Gilbertson et al., 1983) and osteosarcoma after surgery are over 90% (MacEwen, 1990). This shows that it is very important and greatly urgent to develop some other strategies in dealing with the emerging diseases that found in pet animals.

The development of cancer goes through a process of evolution in an animal body (Merlo et al., 2006; Nowell, 1976). The dynamic interactions between cancer cells and its host normal cells gradually develop an environment suitable for the cancer cells to survive and to multiply (Whiteside, 2006). One of the essential elements in building the suitable environment is to develop a mechanism which weakens its immune response. A continuous research effort has been focused on breaking the established environment- the surviving zone for the cancer cells and then restoring the host immunological activities (Sadun et al., 2007). The data in the area are mostly collected from the experiments in humans and rodents; no much are available in canine species although the cancers have been already on the top of the causes of death in dogs (Proschowsky et al., 2003).

Many tumors have reduced antigen and/or MHC I antigens expression, which keeps tumor cells away from immunologic detection and elimination (Garrido et al., 1997; Goodenow et al., 1985). However, because of NK cells lyse MHC antigens deficiency or altered targets, the alteration in MHC antigen expression subjects the tumor variants to natural killer (NK)-cell cytotoxicity (Marincola et al., 2000). Tumor-derived cytokines, such as TGF- β in turn inhibits NK cell cytolytic activity, enable the tumor to escape from the host immune response (Hsiao et al., 2004; Wahl, 1994). A better understanding of the basis of molecular interactions between the tumor cells and the immune system would be helpful in developing the immunotherapeutic strategies to the spontaneous tumors.

We used a canine tumor model to study the interactions between the cancer and the host. The mechanisms discovered were applied successfully to control the tumors in the laboratory. However, the effectiveness of the treatment in a client-owned cancer dog patients still needs a lot of improvements.

References

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專 題 演 講 III

Comparative Pathology Case 281

Contributors: Chia-Wen Shih (施洽雯). MD. MS. AP.

Department of Pathology, Lo-Tung Pohai Hospital (羅東博愛醫院病理科).

Clinical history: A 70-year-old man presented right sore throat and blood-stained sputum for weeks. He has history of DM with unsatisfactory control (HbA1c: 7.1) . No history of hypertension.but CVA attacked years ago. His general physical examination findings were unremarkable. There was no cervical lymphadenopathy. Examination of oral cavity and oropharynx show an exophytic necrotic mass in right tonsil. The tumor mass measuring about 1.5 x 1.5 x 1.0 cm. Chest X-ray was normal. His hematological and biochemical examination were within normal limits. CT scan of the head and neck demonstrated an exophytic mass in right side tonsil and without significant surrounding tissue involvement. The mass showed heterogeneous post-contrast enhancement. No neck node enlargement was noted on CT scan. Tonsillectomy was performed on August 28, 2007..

Diagnosis: Angiosarcoma, tonsil.

Gross findings: The tonsil measuring 2.6 x 2.3x 1.7 cm with a protruding tumor mass measuring 1.7 x 1.5 x 1.3 cm. The tumor was soft in consistency. Focal ulceration of the tumor surface with hemorrhage was noted. Cut section of the tonsil tumor varies in color from tan to yellow to pink with marked hemorrhage.

Histopathological findings: Microscopically, the tonsil is covered by benign squamous epithelium with focal ulceration and granulation –like tissue containing blood, acute inflammatory cells and necrotic debris. The tumor shows vasoformative growth with complex anastomosing channels. The lining cells show marked cellular pleomorphism with large and hyperchromatic nuclei and distinct nucleoli. Mitosis with atypical features are identified easily .

Immunohistochemistry: Immunohistochemical staining of the tumor cells show positive staining for vimentin ,CD31 and Ki67, and negative staining for CD34 and cytokeratin.

Discussion: The angiosarcoma is a malignant vascular neoplasm with a definitively aggressive clinical course. It represents 1% of all soft tissue sarcomas in humans.

Angiosarcoma generally occurs in the skin and subcutis or skeletal muscle and has been described in the spleen, bone, liver, and breast. Half of the skin angiosarcomas occur in the head and neck region. Angiosarcomas of oral and salivary gland area are extremely rare, comprising only 2% of all angiosarcomas and 0.6 to 30% of head and neck angiosarcomas. If the angiosarcoma occurs in the head and neck region, it usually affecting the scalp of elderly men. It is very uncommon in the oral cavity but when occurring it usually involves the mandible, but other less frequent sites of involvement have included the maxilla, parotid glands, lips, tongue, floor of mouth, cheek, palate and antrum. To our best knowledge, there was no report of primary tonsil angiosarcoma in the English literature.

Angiosarcoma of the oral region is a disease of older individuals, averaging more than 65 years of age, but it has been reported in infancy. It affects predominantly males. The tumor is typically a solitary or multifocal nodule which appears as a painless, rounded, sometimes ulcerated mass or submucosal mass which may bleed spontaneously. The clinical appearance may be indistinguishable from that of oral pyogenic granuloma or hemangioma. Some tumors grow rapidly while others take many months to reach a size of 4-5 cm.

Trauma, long-standing lymphedema, the presence of foreign bodies (steel, plastic, surgical sponge, osseous wax) , the preexistent lesion such as haemangioma and lymphangioma, and irradiation of benign vascular lesions appear to be contributory factors in the onset of some cases but most cases present with no obvious etiology. Some radiation-induced angiosarcomas have been reported to occur in the oral cavity and salivary gland locations. Two cases reported in the English literature include a radiation-induced angiosarcoma of the tongue, 30 years after radiation therapy for a lymphangioma of the tongue, and a radiation-induced high-grade angiosarcoma of the parotid, 12 years after irradiation for laryngeal squamous cell carcinoma. Radiation-induced angiosarcomas of the oral cavity, like non-oral primary angiosarcomas, are reported to behave poorly, with early onset of metastases and death within 2 years after treatment.

The histopathologic appearance of angiosarcoma varies greatly, depending on the degree of cellular differentiation. Although, by definition, the lesional cells must show some degree of vascular differentiation, either at the light microscopic level or in immunohistochemical studies. The well-differentiated lesions may be quite similar to hemangioendothelioma, with distinct, endothelium-lined vascular channels with relatively flattened endothelial nuclei. There is, however, a tendency for the channels in angiosarcoma to anastomose with one another and to produced dilated sinusoids.

Overall, there are three main patterns of growth: an angiomatous pattern with epithelioid features, a spindle cell pattern, and an undifferentiated or solid pattern. These patterns can be mixed within the same tumor. Occasional proliferation of lesional cells will produce islands and sheets of tumor endothelial cells with large, hyperchromatic nuclei and mitotic activity. The less-differentiated angiosarcoma may have scattered areas of well-differentiated lesion, but it is usually comprised of pleomorphic and hyperchromatic epithelioid cells with abundant mitotic activity.

Proving the vascular phenotype, particularly in tumors with non-vasoformative areas, is essential for diagnosis. Immunohistochemically, although Factor VIII has been used to diagnose vascular phenotype, less than one in four angiosarcomas will be immunoreactive for Factor VIII antigen. CD34 is also expressed by most angiosarcomas, but it is known to have low specificity and low sensitivity. It may be seen in the occasional epithelioid sarcoma.. CD31, a membrane glycoprotein that mediates endothelial cell interactions and promotes vascular adhesion of leukocytes, is the best vascular marker for endothelial phenotype, particularly in poorly differentiated angiosarcoma, when Factor VIII and CD34 fail. CD31 antibody which labels a higher percentage of angiosarcoma lesional cells than do antibodies directed against factor VIII antigen and CD34. Previous study found that CD34 is less reactive than CD31 or Factor VIII. An immunohistochemical study of 19 cases of angiosarcoma showed that the tumor cells were positive for Factor VIII in 19/21, CD31 in 16/19, CD34 in 7/12.

The microscopic differential diagnosis should include: pyogenic granuloma, hemangioma, fibrous histiocytoma, hemangioendothelioma, papillary endothelial hyperplasia, Kaposi's sarcoma and malignant melanoma. The presence of a spindle cell component may suggest fibrosarcoma, malignant fibrous histiocytoma and other spindle cell sarcomas, while a large number of polygonal endothelial cells might suggest a carcinoma or lymphoma. Reactive papillary endothelial hyperplasia is a common process with a pattern of organized thrombus within a vessel. Spindle cell hemangioendothelioma exhibits cavernous spaces with associated thrombi and more solid spindle cell regions. Epithelioid hemangioendothelioma shows intracytoplasmic lumina in lesional cells, usually arranged in an angiocentric fashion. Lack of cytoplasmic hyaline globules, spindled cells with distinctive cytoplasmic borders, and scattered plasma cells distinguish angiosarcoma from Kaposi's sarcoma. Separating angiosarcoma from hemangioendothelioma, particularly the epithelioid subtype and the low-grade variants, can be challenging, yet the latter lesion has a very distinctive growth pattern of cords to sheets of epithelioid endothelial cells with a remarkable hyaline stroma. These features were absent in

angiosarcoma and particularly in the angiosarcomas with epithelioid features, and true cytologic atypia and collagen dissection were always present. Pyogenic granuloma can be distinguishable from angiosarcoma by its lobular growth pattern, well formed vessels and cytologically bland endothelial cells. The vascular markers separate angiosarcoma from carcinoma and melanoma, although keratins may be observed in epithelioid endothelial tumors. Pseudovascular adenoid squamous cell carcinoma in the oral cavity are described, which were characterized by acantholysis of the tumor cells, with formation of anastomosing spaces and channels mimicking an angiosarcoma.

Angiosarcoma of the oral region is treated by wide local excision, although radiotherapy is sometimes used for multifocal lesions. Some authorities favor the combination of radiotherapy and surgery even for the single lesions. Radiation therapy and chemotherapy in these tumors has been proven to be largely ineffective, unless the chemotherapy is given intra-arterially. Positive necks are treated by radical neck dissection. The prognosis is very much dependent on two features: the degree of cellular differentiation and the clinical size of the tumor.

Patients with primary angiosarcoma of the tongue, salivary gland, and lip have a better prognosis than do patients with primary cutaneous or deep soft tissue angiosarcoma. The reasons for the generally improved behavior of oral angiosarcomas over angiosarcomas at other sites may be related to the shorter time interval to diagnosis and treatment.

In conclusion, oral angiosarcomas are rare tumors that generally behave more favorably than do angiosarcomas in other sites. This is the first report of primary tonsil angiosarcoma according to the pictures of histopathology and the result of immunohistochemical stain. Because primary oral angiosarcomas generally tend to behave better than usual angiosarcoma from other locations, complete surgical excision is the preferred treatment for oral angiosarcomas.

Diagnostic criteria:

1. Histopathologic findings: vasoformative growth, cytologic atypia, mitoses.
2. Immunohistochemical stain : CD31 (+).

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Comparative Pathology Case 282

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Clinical history: A 60-years-old G4P0 married woman was admitted in our hospital because of post menopausal vaginal bleeding. She suffered from left breast cancer stage II S/P MRM at another hospital in 1995. She also received 6 course of chemotherapy. Recently she experienced post-menopausal uterine bleeding twice within one week, so she came to our OPD for help. Ultrasound examination revealed endometrial hyperplasia and uterine myoma; Diagnostic D&C reveal poorly differentiated adenocarcinoma. She was received radical hysterectomy at 96/08/27. The uterus measured 9 x 5 x 4 cm and weighed 110 gm. The endometrium measured 0.3 cm in thickness. There is an endometrial polypoid lesion about 1 x 0.5 x 0.4 cm in uterine cavity of fundus. The left fallopian tube measured 6 x 2.5 x 2.5

cm in size. There were 9 right pelvic LN and 10 left pelvic LN dissected.

Diagnosis: Malignant mixed mullerian tumor (carcinosarcoma)

Grossly finding: The resected specimen submitted consisted of a uterus with bilateral adenoxal tissues. The uterus measured 9 x 5 x 4 cm and weighed 110 gm. The endometrium measured 0.3 cm in thickness. There is an endometrial polypoid lesion about 1 x 0.5 x 0.4 cm in uterine cavity of fundus. The left fallopian tube measured 6 x 2.5 x 2.5 cm in size. There were 9 right pelvic LN and 10 left pelvic LN dissected.. The tumor masses are gray in color and soft of left fallopian tube. The tumor invaded to serosa and left ovary. All of salpingx for section.

Histopathological finding: Microscopically, the major part of tumor is adenocarcinoma mixed with minor part of sarcoma that look like endometrial stroma cells.

Immune stain:

- 1) Vimentin: (-) for carcinoma component (+) for sarcoma component
- 2) CD-10: (-) for carcinoma component (+) for sarcoma component
- 3) CK: (+) for carcinoma component (-) for sarcoma component.
- 4) mCEA: (-) for carcinoma component.
- 5) E-cadherin: (+)for carcinoma component
- 6) SMA: (-) for sarcoma component

Discussion: The carcinoma component of malignant mixed mullerian tumor is so prominent as to overgrow the epithelial element and to mimic minor sarcoma component. The malignant sarcoma-like component may be so inconspicuous in such a specimen as to be missed altogether, the lesion being misdiagnosed as an ordinary adenocarcinom. Sometime, total resection of all tumor that diagnosis as adenocarcinma is necessary. The diagnostic D&C of endometrium to make sure of malignant tumor from left fallopian tube is rare, unrespectable, and interesting.

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Comparative Pathology Case 283

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Clinical history: Male rats, 10 wk-old, were administrated a single intraperitoneally injection of streptozotocin at dose of 65 mg/kg body weight to create animal model of diabetes. After treatment, rats showed signs of body weight loss, polyuria, and increase of water and feed consumption post 4 weeks. However, rats were sacrificed due to anorexia, marked emaciation and weakness in clinic. At necropsy, bilateral enlargement and irregular surface on affected kidneys. Tissues were sent to ADDC of NCHU for pathological diagnosis.

Diagnosis: Renal cell tumor, SD rat.

Gross findings: Nine rats were administrated a single intraperitoneally injection of streptozotocin at dose of 65 mg/kg body weight to create animal model of diabetes. Grossly, bilateral enlargement and irregular surface on affected kidneys.

Staining methods: Kidneys were fixed in 10% neutral buffered formalin solution for one week. Cross and transverse sections of tissues were embedded in paraffin and cut 2 μ m thickness of sections and were stained with hematoxylin and eosin (H&E) stain routinely. Additional sections were histochemically stained with periodic acid Schiff (PAS) method. For the cell proliferation, immunohistochemistry staining was performed on deparaffinized sections and were immersed into 10 mmol L⁻¹ citrate buffer, micro-waved for 20 min, incubated with primary anti-mouse monoclonal IgG_{2a} at 1:800 dilution (PC10: sc-56, Santa Cruz Biotechnology, Inc. USA), and HA (1:100, Provided by Prof. Chen's lab) for 1 hours at room temperature. After that, slides were incubated in biotinylated secondary antibody (anti-mouse IgG antibody, 1:1000) for 45 min and followed by DAB substrate (Vectastain ABC kit, Dako, CA) for 3-10 min, and then were counterstained with Mayer's hematoxylin (Sigma, St. Louis, MO, USA) for 3 min.

Histopathological findings: Microscopically, no significant changes of glomeruli were noted in the affected kidneys. However, multiple, acute, remarked hydropic swelling of the distal renal tubules and numerous polycysts were noted in the cortex and medulla areas of kidneys. Moreover, multiple tumor masses were randomly observed. Tightly packed nests of atypical cells were observed in the nodules and tubular structures that resembled renal tubules. The cytoplasm of the atypical cells was ovoid to pleomorphic, with basophilic granules or vacuoles. The nuclei were ovoid with sparse chromatin and predominant nucleoli (Hard, 1985).

Renal neoplastic lesions were characterized as renal tubular carcinoma (RTC), renal tubular adenoma (RTA) and renal atypical tubular hyperplasia (RATH) in this case. Renal tumors were solid aggregates classifiable as carcinomas because of histological features which included cellular pleomorphism, appreciable mitotic activity, and necrosis. Typically, these were basophilic lesions consisting of granular or an admixture of granular and vacuolated cell types with a moderate amount of mitotic activity. The sheets of small densely crowded cells lacking a distinct lobular pattern, but with larger cells containing bizarre nuclei interspersed, solid lobules of more uniform epithelial cells, and trabeculae or lobules in which varying degrees of cell alignment indicated attempted tubule differentiation (Hard, 1985).

Numerous small cystic masses composed of single to multilayered cuboidal cells lining a cystic lumen and little nodular solid proliferation of epithelial cells with an eosinophilic to vacuolated cytoplasm. The smaller often microscopic lesions conformed to papillary, cystopapillary, or adenomas. Papillary adenomas consisted of tubule profiles and dilated with proliferating lining cells supported on delicate fronds. The cells were of a uniform cuboidal to columnar type, and relatively large, with a low nucleus:cytoplasm ratio. Cytoplasmic staining was typically eosinophilic with a finely granular or ground-glass appearance. The nuclei were also of a uniform size, and centrally or basally located in the cell. Although these small neoplasms portrayed a benign appearance, mitotic figures were observed in some. Cystopapillary adenomas represented identical lesions but with a more marked dilation of the tubule lumens. Another type of RATHs were focal to multifocal, characterized by enlarged to occasionally cystic tubules, lined with large eosinophilic, cuboidal to columnar cells, rarely multilayered.

Histochemistry results: A large number of PAS positive cells with intense granules were seen in the cytoplasm of swollen distal renal tubular cells and the membrane of glomeruli but not in the normal proximal tubular cells.

Immunohistochemistry Results: The tumor cells also expressed intense granular cytoplasmic staining for PCNA and vimentin.

Discussion: The compound used here, streptozotocin (STZ), is an antibiotic and diabetogenic agent produced by a strain of *Streptomyces achromogenes*. The β -cell-specific toxin streptozotocin, an analogue of GlcNAc, has been used to create animal models of diabetes. It is known that the ability of STZ to act as a NO donor has led many investigators to postulate that NO is involved, but the diabetogenic effect of STZ *in vivo* cannot be readily duplicated with *N*-methyl -*N*-nitrosourea (MNU, the portion of STZ that actually donates NO) (Konrad et al., 2001).

In our case, all of nine rats showed multiple, acute, remarked hydropic swelling of the distal renal tubules with numerous polycysts are noted in the cortex and medulla areas of kidneys within 20 weeks. It is suggested that the progression of diabetic nephropathy is associated with early adaptive changes and late structural ones. Initially, after the onset of experimental diabetes mellitus, the kidney reacts to hyperglycaemia by glomerular and tubular hypertrophy along with hyperfunction (Sanai et al., 2000).

The carcinogenicity of streptozotocin in the rat is already confirmed. The early

tubular degeneration is sited in the distal rather than the proximal convoluted tubule and subsequent glomerular lesion shows linear deposits of IgG and albumin in the basement membrane rather than in the mesangium. If the tubular dilation is exaggerated, the lesions take the form of papillary cystadenomas rather than simple papillary adenomas (Bleasel and Yong, 1982). Horton et al. (1977) reported that forty-six separate renal tumours developed in 36/80 Wistar male rats given a single i.v. dose of streptozotocin (25 mg/kg body wt) to induce diabetes mellitus. Fourteen of the tumours were epithelial in type, 8 were wholly mesenchymal and 24 were largely mesenchymal but also contained epithelial elements. Moreover, 2 of 4 rats displayed multiple tumor masses in this study.

The morphologies of renal neoplastic lesions were characterized as renal tubular carcinoma (RTC), renal tubular adenoma (RTA) and renal atypical tubular hyperplasia (RATH). The neoplasms in these animals, is composed of 2 populations of tumor cells: one was composed of large clear (PAS positive) cells with hyperchromatic nuclei and abundant clear cytoplasm variably admixed with acidophilic cells arranged in tubular or solid pattern; the other population characterised by basophilic cells with pale basophilic nuclei and a papillary pattern of growth (Okimoto et al., 2004). The occurrence of preneoplastic and neoplastic lesions in the kidneys of rats younger than 18 weeks is a highly unusual finding (Lanzoni et al., 2007). Preneoplastic lesions can occur as early as 3 weeks of age, developing into adenocarcinomas by 6 months (Okimoto et al., 2004).

Spontaneous renal tumours in the rat are found in aging animals with low incidence. The incidence of renal spontaneous neoplasms is about 1% in Fisher 344 rats (Thurman et al., 1995) and at 0.08% in Sprague–Dawley rats (Chandra et al., 1993). Although, spontaneously renal neoplasms are rare in all strains of laboratory mouse with exception of the inbred BALB/cf/Cd, which, through inbreeding and selection, has acquired an unusually high frequency of epithelial neoplasms of the kidney (Gordon and Hard, 1985; Kahan and Alden, 2002). The renal tumor frequency in male mice was lower than in the females at 73%, with only half the number of tumors classifiable as carcinomas, it is unlikely that a real difference in kidney cancer induction by streptozotocin exists between the sexes. Slightly more than one-half of the males surviving long enough to be eligible for inclusion in the effective survivor group died between Weeks 20 and 40 (Hard, 1985). However, it has been generally observed that renal tubule tumours in laboratory rodents, whenever occurring spontaneously or being induced by chemicals, are slow-growing neoplasms. Furthermore, spontaneous occurrence of rat renal neoplasia are of low incidence in F344 rats (Thurman et al., 1995; Hiasa and Ito, 1987) and uncommon or absent in

Crl:CD (SD) IGS BR (Sprague Dawley) rats from long term studies (Chandra et al., 1993, Son and Gopinath, 2004; Baldrick, 2005;).

Changes in the expression of cytoskeletal proteins within the kidneys of diabetic rats suggest a role for α -smooth muscle actin and vimentin in the pathogenesis of diabetic kidney disease. In the interstitium, it appeared in a perivascular and peritubular distribution. Vimentin was detectable within normal glomerular epithelial cells and increased rapidly (days 7 and 15) in diabetic rats. Vimentin also appeared early within the lining of the peritubular capillaries and damaged diabetic tubules. These changes were associated with a delayed increased transcription of α -smooth muscle actin and vimentin. Treatment with insulin (early or late) attenuated and reversed respectively the expression of cytoskeletal proteins and collagens within diabetic kidneys (Sanai et al., 2000).

Nuclear PCNA immunoreactivity is found in the proliferating compartment of normal and tumor cells. Immunohistochemically, there was no significant difference in percentage of PCNA-positive cells between tumor grades G 1 and G 2, while G 3 tumors contained high percentage of PCNA positive cells. The percentage of PCNA-positive tumor cells was found to be correlated with histological grading of tumor and also with the S+G2+M phase fraction and the S phase fraction measured by flow cytometry (Tanioka et al., 1993).

Renal cell carcinoma (RCC) is the third most common urologic tumour and represents about 2–3% of human malignancies. In the USA, about 30,000 patients are newly diagnosed with RCC every year. The incidence of RCC is rising and worldwide approximately 95,000 patients die each year (Horstmann et al., 2005). About 30–40% of RCC patients present with metastasized disease at the time of first diagnosis and another 30–50% with initially organ-confined tumours will develop systemic tumour dissemination regardless of an initially curative surgical approach. Whereas localized RCC (T1 or T2) are curable by resection of the tumour-bearing kidney, hereby achieving a long-term survival rate of about 80%, an effective treatment option for metastasized renal cell carcinoma patients is currently not available (Bichler and Wechsel 1999; Hartmann and Bokemeyer 1999).

In addition, the fibroblast growth factor (FGF-2) also known as basic FGF (bFGF) belongs to a family of structurally related polypeptide growth factors. FGFs mediate their signals through binding to and activation of cell surface receptors (Powers et al. 2000). RCC patients tended to express higher serum bFGF levels when compared with healthy volunteers. The value of bFGF could be as a biological marker of

prognostic relevance for RCC (Horstmann et al., 2005). Furthermore, the reduced expression of Fas and bcl-2 in RCC compared with the expression in normal kidney is a prominent alteration of apoptotic regulatory molecules. The alteration of the up-regulated Fas expression might be characterized during the tumor progression stage. It is also suggested that the effect of alteration of bcl-2 expression might be minimal during the tumor progression stage because of the reduced expression in tumors of the clear cell type, which is the most dominant cell type in RCC (Sejima and Miyagawa, 2006)

Diagnostic criteria:

1. Histopathologic findings: Renal tumors were solid aggregates classifiable as carcinomas because of histological features which included cellular pleomorphism, appreciable mitotic activity, and necrosis. The sheets of small densely crowded cells lacking a distinct lobular pattern, but with larger cells containing bizarre nuclei interspersed, solid lobules of more uniform epithelial cells, and trabeculae or lobules in which varying degrees of cell alignment indicated attempted tubule differentiation.
2. Special staining: The tumor cells exhibited vacuoles and showed reddish staining by the PAS method. The tumor cells also expressed intense granular cytoplasmic staining for PCNA and HA.

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Comparative Pathology Case 284

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Clinical History: A 52 y/o male patient, who is an interior decorator (室內裝潢師傅), came to our ENT clinic for progressive left nasal obstruction with yellowish discharge for two months. He recalled episodes of nasal bleeding two months ago, then progressive nasal obstruction with rhinorrhea occurred. In recent one month, easily nasal bleeding and progressive left periorbital swelling developed. He have smoked cigarettes about one pack per day for more than twenty years and chewed betel nuts

occasionally. He denied regular alcohol drinking.

CT scan showed soft tissue density filled in the left nasal cavity and left maxillary sinus. Sinoscopic biopsy proved malignancy.

Diagnosis: Extramedullary plasmacytoma

Gross findings: Plasmacytomas involving the upper air passages are often seen as raised smooth surface lesions which bulge into the lumen of the involved structure. Tumor tissue tends to be somewhat fleshy and demonstrate a dark red or grey color. (Fu. 1978)

Histopathological findings:

- (1) Relatively monotonous atypical large non-cohesive cells are seen with a "starry-sky" pattern.
- (2) The specimen was reported as diffuse large cell lymphoma initially at frozen section. Later, immunohistochemical stains showed faint CD-20 (+) reactivity in the periphery of tumor and no staining in the central part.
- (3) Kappa-light chain restriction and CD-138 (+) were noted.
- (4) Bone marrow biopsies during the clinical course are all negative for malignancy.

Discussion: Viewing as neoplasm of plasma cell, it was classified as (1) multiple myeloma, (2) solitary plasmacytoma of bone, and (3) extramedullary plasmacytoma. Commonly, the extramedullary plasmacytoma occurs in the upper aerodigestive pathway and it was assumed chronic stimulation caused by inhaled irritants or viral infections to be the etiology. Contrary to multiple myeloma and solitary plasmacytoma of bone, extramedullary plasmacytoma arises from plasma cells located on mucosal surface. Generally lower incidence of progression into multiple myeloma and better prognosis achieved by local treatment, some investigator even proposed that extramedullary plasmacytoma to be regarded as marginal zone lymphoma that have undergone extensive plasmacytic differentiation. As much as eight subtypes are proposed, it could be generalized into (1) low grade/well differentiated/plasmacytic; (2) intermediated grade/plasmablastic; and (3) high grade/pleiomorphic/anaplastic. Previous data considered surgery alone could achieve the best results if resectability is good. Adding radiation is recommended if complete resection is doubtful. Still, the role of adjuvant chemotherapy remains to be clarified and alkylating chemotherapy related tumorigenesis is reported in protracted clinical courses. Mis-diagnosed as diffuse large cell lymphoma, there was case treated with cyclophosphamide, doxorubicin, vincristine, and prednisolone and achieved complete remission. Local recurrence occurred 30 months later and local

irradiation achieved tumor control for more than 30 months.

Diagnostic criteria:

Criteria for Diagnosis of Solitary plasmacytoma (Galieni 1995)

- (1) Single area of bone destruction due to clonal plasma cells
- (2) Normal marrow without clonal disease
- (3) Normal results on a radiologic skeletal survey and magnetic resonance imaging of the spine, pelvis, proximal femurs, and humeri
- (4) No anemia, hypercalcemia or renal impairment attributable to myeloma
- (5) No or low serum or urinary level of monoclonal protein and preserved levels of uninvolved immunoglobulins

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Comparative Pathology Case 285

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Clinical history: A 62-year-old female patient has a painless well-circumscribed tumor on the scalp for more than 10 years. She received operation for removal of tumor. The specimen received consists of 3 pieces of grayish-whitish firm tissue fragments measuring up to 3.5 x 2.6 x 1 cm. in size.

Diagnosis: Myopericytoma

Gross findings: The specimen submitted consists of 3 pieces of soft tissue fragments measuring up to 3.5 x 2.6 x 1 cm. in size and 5.7 gm. in weight. They are grayish and soft with many dilated vessels.

Histopathological findings: Microscopic examination shows a vascular tumor with many dilated vascular spaces in lobular arrangement. Foci of multilayered concentric growth of spindle cells around lesional vessels are noted. The surrounding stroma shows edema and fibrosis. No definite malignancy is seen.

Immunohistochemistry: Immunohistochemical study shows that the spindle cells are reactive against smooth muscle actin and Vimentin antibodies and not reactive against S-100 protein and Desmin antibodies. The lining cells of vessels are reactive against CD34 antibody.

Discussion: The term “myopericytoma” was first proposed by Requena et al. in 1996 in a report of a cutaneous adult myofibroma which revealed possible myopericytic differentiation. Granter et al. adopted the term myopericytoma in 1998 to describe benign tumors with histologic features of concentric perivascular proliferation of spindle cells and showing differentiation toward perivascular pericytes. The new World Health Organization (WHO) classification of soft tissue tumors was introduced in 2002, and myopericytoma is categorized as an entity of pericytic (perivascular) tumors.

Myopericytoma may be diagnosed as myofibroma, hemangiopericytoma or sarcoma in the past. In fact, myopericytoma forms a continuous morphologic spectrum ranging from glomus tumor, myofibroma/myofibromatosis, glomangiopericytoma and angioleiomyoma. All of these tumors are characterized by perivascular actin-positive contractile cells showing variable degrees of myopericytic differentiation.

Myopericytoma occurs over a wide age range but most common in mid adulthood, with a male predominance. The lesion generally arises in the subcutaneous tissue and the dermis. The distal extremities are the most common affected sites, followed by the proximal extremities, the head and neck region, and the trunk. Rare primary sites include bone, skeletal muscle, intracranial region and visceral organ have been documented.

Clinically, myopericytoma generally presents as a slow growing, painless, and solitary nodule which is usually less than 2 cm in diameter. Multiple lesions are more frequently seen in children but usually in the same anatomical region. Most myopericytomas do not recur followed by excision. Recurrence may be related to poor circumscription of the tumor or a reflection of multifocal disease.

Macroscopically, myopericytomas are usually well demarcated and unencapsulated. They are rubbery or firm, and the cut surface is gray white or pink. Microscopically, myopericytoma is composed of cells showing a radial and perivascular concentric arrangement of monomorphic ovoid to spindle shaped neoplastic cells with eosinophilic to amphophilic cytoplasm. The stroma can be cellular, fibrotic, edematous, myxoid, or hyalinized. Scattered inflammatory cells are occasionally seen. Mitotic figure is rare, and necrosis is an infrequent finding. Lesions usually contain numerous thin-walled blood vessels. In some cases, lesional blood vessels are dilated, gaping and branching resembling features in hemangiopericytoma. Subendothelial proliferation of tumor cells is frequently seen, and entirely intravascular myopericytoma has been reported. A variant of myopericytoma called “glomangiopericytoma” shows glomoid features with uniform central round nuclei, clear to eosinophilic cytoplasm, and well-defined cell borders. Malignant myopericytoma is extremely rare. It is characterized by highly mitotic figures and necrotic areas. It is usually also associated with aggressive clinical behavior.

Immunohistochemically, the neoplastic cells in myopericytomas stained positively for smooth muscle actin (SMA). H-caldesmon is expressed in most cases. Desmin is negative for most cases, in contrast to a true smooth muscle tumor. CD34 is focal positive in some cases. S-100 protein and cytokeratin are both negative.

Diagnostic criteria:

1. Histopathologic findings: multilayered concentric growth of oval to spindle shaped myoid appearing cells with eosinophilic to amphophilic cytoplasm around blood vessels
2. Immunohistochemical stain: SMA (+), Vimentin (+), CD34 (-), S-100 (-), desmin

(-)

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Comparative Pathology Case 286

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Clinical History:05-389C, 05-0514, 8–weeks-old, Wistar rats, were submitted for health monitoring including serology, parasitology and pathology.

Diagnosis: Small intestine, mucosal crypts and intervillous spaces: Piriform, flagellated, protozoal organisms, multifocal, small to moderate numbers, Wistar rat, etiology- consistent with *Spironucleus (Hexamita) muris*.

Gross Findings:No gross lesions are observed in these cases.

Histopathological Findings:Microscopically, piriform, flagellated, protozoal organisms were often present in the duodenal and jejunal mucosal crypts and intervillous spaces multifocally.

Discussion:The two most commonly reported enteric flagellates of laboratory animals are *Spironucleus muris* and *Giardia muris*; both are members of the Hexamitidae family and are flagellated and binucleate. The trophozoites are piriform, bilaterally symmetrical. The torpedo-shaped trophozoites (2-3 x 7-9µm) have two anterior nuclei and eight flagella (two posterior and six anterior) on isolated organisms. Sucking disks, as occur in *Giardia* sp., are not observed. Both genera produce cyst forms that are not observed in this case. Small, oval cysts (5µm) have a banded “Easter egg” appearance. *Spironucleus* is often described as present in the intestinal crypts, while *Giardia* is described on villar surfaces. The incidence of infection is high in conventional weaning rat and mice colonies. Stressed weanling mice (3 to 8 weeks) of susceptible strains (C3H, NZW, DBA/2 and athymic nude) with catarrhal enteritis caused by *S. muris* often have large numbers of organisms present in the crypts, the intervillous spaces, and within the lamina propria. In clinical cases, the inflammatory infiltrate present within the mucosa and submucosa is greatly variable. Although a presumptive diagnosis of *Spironucleus* sp. can be made based on examination of histologic sections, a definitive diagnosis requires examination of stained intestinal smears. Swabbing and formalization of tissues for histology must be accomplished quickly, as the protozoa may be lysed within an hour of death. *Spironucleus muris* can be present in clinically healthy animals. Clinical spironucleosis seems to occur only in mice and possibly Chinese hamsters, usually occurring at weaning or subsequent to experimental manipulation. Acutely affected mice may have a distended abdomen and die without diarrhea. No clinical signs have been attributed directly to these organisms. Diarrhea is often exacerbated by the presence of these flagellates. However, death at the peak of an outbreak may occur 1 to 4 days following the onset of clinical signs, and mortality rates may approach 50%. More chronic cases usually exhibit stunted growth, depression, and a

hunched posture. In mice, rats, and hamsters with enteritis, increased numbers of *Spironucleus* organisms are generally believed to be a secondary change. *Spironucleus muris* has been observed in blood smears from hamsters with proliferative ileitis secondary to mucosal damage.

Spironucleus muris is a flagellated protozoan that dwells on the small intestinal mucosa. The main route of infection is ingestion of the encysted stage of the organism which is passed in feces of an infected animal. A carrier stage occurs in adults. Heavily infected mice are usually smaller in size, depressed and have abdominal distension. Dehydration and anorexia follow. A "sticky" fecal mass may be seen on the perineal area. The anterior small intestines are dilated and filled with a bubbly froth to gassy catarrhal fluid contents, giving the appearance of postmortem autolysis of the intestines. No gross lesions are observed in most juvenile and adult mice with this protozoal disease. No specific pathology accompanies these parasitic infections. Histologic exam of the pylorus and anterior small intestine reveal acute catarrhal enteritis or chronic mural hyperplasia with minimal mononuclear cell infiltration, reduction in height of villi, lymphocytic infiltration, epithelial desquamation, villous edema and small oval protozoa in the crypts and occasionally in the space between villi. Direct smears of small intestinal contents reveal fast darting protozoa.

Administration of dimetridazole (0.04-0.1 % in drinking water) has been reported to reduce mortality, but does not effectively eliminate the parasites. Mice derived by cesarian section and maintained in a barrier environment are usually free of *Spironucleus muris*. Differential diagnosis includes *Tritrichomonas* sp. (large intestinal flagellate), *Giardia* sp. (duodenum, jejunum, small intestinal flagellate), *Chilomitus* sp., *Chilomastix bettencourti*, *Entamoeba muris* (large intestinal protozoans), and other common protozoal inhabitants of the mouse and rat gut.

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Comparative Pathology Case 287

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Clinical history: An adult intact female cocker spaniel was referred for further evaluation of a mass on the right 3rd digit beside the nail bed. And the mass was remarkably enlarged in three months. Physical examination revealed a bean-shaped, firm, movable and well capsulated mass. The mass was 2x1x0.5 cm in size.

Diagnosis: Extramedullary plasmacytoma with amyloidosis, right 3rd digit mass.

Gross findings: Grossly, the mass on the right 3rd digit submitted was oval, covered skin, well-circumscribed, approximately 2x1.8x1 cm in size. It appeared pale and homogeneous on cut section.

Histopathological findings: The mass submitted is a circumscribed, but unencapsulated dermal nodule which is characterized by predominant amorphous eosinophilic deposits and contain only scattered islands of well differentiated plasma

cells arranged in tightly packed nests and cords. The majority of plasma cells have peripheral nuclei and moderate cytoplasm with a hyaline quality. Nuclei of a few cells are quite pleomorphic and vary from round to ovoid to convoluted. Variable numbers of multinucleated giant cells are also present. Congo red stains using polarizing lens for amyloid deposits are positive. Anti-canine immunoglobulins (IgG) stain demonstrates positive signal as granular appearance distributed from areas to areas within amyloid substance and a few plasma cells.

Histochemistry: Congo red stains under the polarized light show massive apple-green color in the tumor matrix.

Immunohistochemistry: Immunoglobulin (IgG) stains for some tumor cells are positive.

Discussion: Extramedullary plasmacytoma (EMP), a tumor of monoclonal immunoglobulin-producing cells, is an uncommon canine neoplasm, usually affecting the skin. Amyloid deposition associated with canine EMP is uncommon but has been described in different tissues, including the skin and intestine. In the previous study, amyloid deposits in eight canine and two feline EMPs were examined immunohistochemically with antibodies against various types of amyloid. All the EMPs exhibited tumor cells expressing λ -light chains, and the amyloid deposits invariably reacted with antibodies against A λ -type. In man, plasmacytic neoplasms associated with amyloid deposits represent about 10% of all cases of multiple myeloma and 15% of all EMPs. In canine EMPs, a figure of 6% was arrived at in two separate studies. In cats, amyloid deposits were found in two out of five EMPs.

Amyloidosis is characterized by proteinaceous extracellular deposits in various tissues and organs of the body with common morphological, structural and staining properties but variable protein composition. By light microscopy, amyloid has a homogeneous, eosinophilic appearance after staining with haematoxylin and eosin. It stains red with alkaline Congo-red dye, and under polarized light the deposits exhibit a apple-green birefringence. Classification of amyloidosis by protein type can be carried out by chemical, immunochemical or immunohistochemical methods, but the most reliable method is that of amino-acid sequence analysis. Amyloid A (AA) or reactive amyloidosis and amyloid L (AL) amyloidosis have frequently been described in man, and have also been described in domestic animals. In the case of AA amyloidosis, amino terminal portions of the acute-phase protein serum amyloid A are deposited. AL amyloid, however, consists of amino terminal fragments and completes immunoglobulin light chains. These are usually derived from

lymphoplasmacytic tumours, such as the multiple myeloma, lymphoplasmacytic lymphoma (Waldenström's macroglobulinaemia) and extramedullary plasmacytoma (EMP), which synthesize and secrete a monoclonal protein. The latter forms amyloidogenic precursors, which then aggregate to form the amyloid deposits.

In addition, in human, a disease names Amyloidomas or tumoral amyloidosis. Amyloidoma of soft tissue is a rare condition. Deposition of amyloid is usually systemic, but occasionally it is restricted to a single organ. Less commonly, nodular masses of amyloid called amyloidomas occur in isolation. They are seen most often in the nasal sinuses and the upper respiratory tract. Other sites include the lung, spleen, eyelid, skin, lower urinary tract, lymph nodes, tongue, gastrointestinal tract, breast, brain, soft tissues and bone. The deposits are usually AL fibrils formed from immunoglobulin (Ig) light chains that are typically seen in primary amyloidosis.

Diagnostic criteria:

- 1.Histopathologic findings: eosinophilic amyloid matrix and lots of plasma cells.
- 2.Histochemical stain: Congo red reveals apple-green under polar lens examination
- 3.Immunohistochemical stain: immunoglobulin (IgG) (+).

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中華民國比較病理學會
第一次至第四十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
	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 mixed tumor)	Human	佛教慈濟綜合醫院
103.	Atypical central neurocytoma	Human	新光吳火獅紀念醫院
104.	Cardiac schwannoma	SD rat	國家實驗動物繁殖及研究中心
109.	Desmoplastic infantile ganglioglioma	Human	高雄醫學院

107.	1.Primary cerebral malignant lymphoma 2.Acquired immune deficiency syndrome	Human	台北市立仁愛醫院
111.	Schwannoma	Human	三軍總醫院
114.	Osteosarcoma	Dog	美國紐約動物醫學中心
115.	Mixed germ-cell stromal tumor, mixed sertoli cell and seminoma-like cell tumor	Dog	美國紐約動物醫學中心
116.	Krukenberg's Tumor	Human	台北病理中心
117.	Primary insular carcinoid tumor arising from cystic teratoma of ovary.	Human	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科
166	Nephroblastoma	Rabbit	紐約動物醫學中心
168	Nephroblastoma	Pig	台灣動物科技研究所
169	Nephroblastoma with rhabdomyoblastic differentiation	Human	高雄醫學大學病理科
172	Spindle cell sarcoma	Human	羅東聖母醫院
174	Juxtaglomerular cell tumor	Human	新光醫院病理檢驗科
190	Angiosarcoma	Human	高雄醫學大學病理學

			科
192	Cardiac myxoma	Human	彰化基督教醫院病理科
194	Kasabach-Merrit syndrome	Human	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
239	Glioblastoma multiforme	Human	羅東聖母醫院
240	Pineoblastoma	Wistar rat	綠色四季
241	Chordoid meningioma	Human	高醫病理科

	243	Infiltrating lobular carcinoma of left breast with meningeal carcinomatosis and brain metastasis	Human	花蓮慈濟醫院病理科
	245	Microcystic Meningioma.	Human	耕莘醫院病理科
	247	Well-differentiated fetal adenocarcinoma without lymph node metastasis	Human	新光吳火獅紀念醫院
	249	Adenocarcinoma of lung.	Human	羅東聖母醫院
	252	Renal cell carcinoma	Canine	國立台灣大學獸醫學系獸醫學研究所
	253	Clear cell variant of squamous cell carcinoma, lung	Human	高雄醫學大學附設中和醫院病理科
	256	Metastatic adrenal cortical carcinoma	Human	耕莘醫院病理科
	258	Hashimoto's thyroiditis with diffuse large B cell lymphoma and papillary carcinoma	Human	高雄醫學大學附設中和醫院病理科
	262	Medullar thyroid carcinoma	Canine	臺灣大學獸醫學系
	264	Merkel cell carcinoma	Human	羅東博愛醫院
	266	Cholangiocarcinoma	Human	耕莘醫院病理科
	268	Sarcomatoid carcinoma of renal pelvis	Human	花蓮慈濟醫院病理科
	269	Mammary Carcinoma	Canine	中興大學獸醫學系
	270	Metastatic prostatic adenocarcinoma	Human	耕莘醫院病理科
	271	Malignant canine peripheral nerve sheath tumors	Canine	臺灣大學獸醫學系
	272	Sarcomatoid carcinoma, lung	Human	羅東聖母醫院
	273	Vertebra, T12, laminectomy, metastatic adenoid cystic carcinoma	Human	彰化基督教醫院
	274	rhabdomyosarcoma	Canine	臺灣大學獸醫學系
	275	Fetal rhabdomyosarcoma	SD Rat	中興大學獸醫學系
	276	Adenocarcinoma, metastatic, iris, eye	Human	高雄醫學大學
	277	Axillary lymph node metastasis from an occult breast cancer	Human	羅東博愛醫院
	278	Hepatocellular carcinoma	Human	國軍桃園總醫院
	279	Feline diffuse iris melanoma	Feline	中興大學獸醫學系
	280	Metastatic malignant melanoma in the brain and inguinal lymph node	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 <i>Streptococcus</i> 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 <i>Leptospira</i> spp	Pig	中興大學獸醫學院
173	Ureteropyelitis and cystitis	Pig	中國化學製藥公司

	254	Pulmonary actinomycosis.	Human	耕莘醫院病理科
	259	Tuberculous peritonitis	Human	彰化基督教醫院病理科
	260	Septicemic salmonellosis	Piglet	屏東科技大學獸醫系
	261	Leptospirosis	Human	慈濟醫院病理科
	267	Mycobacteriosis	Soft turtles	屏東科技大學獸醫系
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, polymavirus infection	Lory	美國紐約動物醫學中心
	138	1.Aspergillus spp. encephalitis and myocarditis 2.Demyelinating canine distemper encephalitis	Dog	台灣大學獸醫學系
	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
	238	Hydranencephaly	Cattle	國立屏東科技大學獸醫學系
	248	Porcine Cytomegalovirus (PCMV) infection	Swine	國立屏東科技大學獸醫學系
	250	Porcine respiratory disease complex (PRDC) and polyserositis, caused by co-infection with pseudorabies (PR) virus, porcine circovirus type 2 (PCV 2), porcine reproductive and respiratory syndrome (PRRS) virus and <i>Salmonella typhimurium</i> .	Swine	屏東縣家畜疾病防所
	255	Vaccine-induced canine distemper	gray foxes	國立台灣大學獸醫學系
黴菌	265	Bronchointerstitial pneumonia (PCV II infection)	Swine	台灣大學獸醫學系
	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

寄生蟲	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 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
	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
	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
	242	Neosporosis	Cow	國立屏東科技大學獸醫學系
	263	Intestinal amebiasis	Human	彰化基督教醫院病理科
立克次體	229	Necrotizing inflammation due to scrub typhus	Human	佛教慈濟醫院病理科
	251	Scrub typhus with diffuse alveolar damage in bilateral lungs.	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.	Cholelithocyst	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-rumpe	台灣大學獸醫學系

		d agoutis	
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
237	Arteriovenous malformation (AVM) of cerebrum	Human	耕莘醫院病理科
244	Organophosphate induced delayed neurotoxicity in hens	Hens	中興大學獸醫學病理學研究所
257	Severe lung fibrosis after chemotherapy in a child with Ataxia-Telangiectasia	Human	慈濟醫院病理科

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

402 台中市南區國光路 250 號

中興大學獸醫學院動物疾病診斷中心 張文發秘書長 收

Tel: (04) 22840894

Fax: (04) 22852186

e-mail address: boovet@mail.vm.nchu.edu.tw

-----中華民國比較病理學會-----

會員資料更改卡

姓 名：_____ 會員類別：☐一般會員

☐學生會員

☐贊助會員

最高學歷：_____

服務單位：_____職 稱：_____

永久地址：_____

通訊地址：_____

電 話：_____傳 真：_____

E-Mail Address：_____

中 華 民 國 比 較 病 理 學 會

誠摯邀請您加入

入 會 辦 法

一、 本會會員申請資格為：

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

二、 會員：

- (一) 入 會 費：一般會員新台幣一仟元，學生會員一百元，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：
402 台中市南區國光路 250 號中興大學獸醫學院動物疾病診斷中心張文發秘書長收
電話：04-22840894-112、傳真 04-22852186。

中華民國比較病理學會入會申請及會員卡

會籍電腦編號

姓名	中文		性別	男 <input type="checkbox"/>	出生	民國	年	月	日	出生地	省 縣/市
	英文			女 <input type="checkbox"/>	身分證字號						
			會員身份: <input type="checkbox"/> 一般 <input type="checkbox"/> 學生 <input type="checkbox"/> 贊助								
學歷	(1)				稱謂(請圈選) 先生 小姐 醫師 獸醫師						
					研究員 博士 教授 主任 其他: _____						
	(2)				研究興趣	(1)					
	(3)					(2)					
	(4)					(3)					
主要經歷	機關名稱			職務		起			止		
						年 月			年 月		
						年 月			年 月		
						年 月			年 月		
現職						年 月			年 月		
通訊地址: 現在 電話: 傳真: 永久 電話: 傳真: 電子郵件遞(E-mail)地址:											
茲 贊 同 貴會宗旨擬加入為會員嗣後並願遵守一切規章共圖發展 此 致 中華民國比較病理學會 申請人 簽章 介紹人 簽章 介紹人 簽章 中華民國 年 月 日										審核結果	