中華民國比較病理學會

Chinese Society of Comparative Pathology

第 51 次比較病理學研討會

（免疫血液相關疾病）

暨第六屆第一次會員大會議程表

School of Veterinary Medicine, National Taiwan University

國立臺灣大學獸醫專業學院

March 12, 2011 (中華民國 100 年 3 月 12 日)

Chinese Society of Comparative Pathology

中華民國比較病理學會

SCHEDULE

51ST MEETING OF COMPARATIVE PATHOLOGY

中華民國比較病理學會第 51 次比較病理學研討會

免疫血液相關疾病

暨第六屆第 1 次會員大會議程表

Date: March 12, 2011 (Sat) 08:30~17:00

Location: B01,School of Vet Med, NTU

Address: No.1, Sec. 4, Roosevelt Road, Taipei

Telephone: 02-33663858

時間：100 年 3 月 12 日(星期六) 08:30~17:00

地點：國立台灣大學獸醫學系 B01 演講廳

地址：台北市羅斯福路四段 1 號

電話：02-33663858

Time(時間)	Schedule(議程)		Moderator(主持)
08:30~09:00	Registration (報到)		
09:00~09:10	Opening Ceremony (致詞)		
09:10~09:40	專題演講	Dr. C. H. Wan (萬灼華 助理教授) 美國 Emory 大學 Yerkes Primate Center 參訪介紹：相關生醫研究應用之簡介	Dr. C. H. Liu 劉振軒 院長
09:40~10:00	Coffee Break		
10:00~10:30	Case 357	Dr. Y.L. Chen (陳燕麟 醫師) Cardinal Tien Hospital, Taiwan (天主教耕莘醫院)	Dr. F. J. Leu 呂福江 主任
10:30~11:00	Case 358	Dr. C. W. Shih (施洽雯 醫師) Lotung Poh-Ai Hospital, Taiwan (羅東博愛醫院)	
11:00~11:30	Case 359	Dr. Y. Lee (李遙 獸醫師) National Taiwan University, Taiwan (國立臺灣大學)	
11:30~13:30	General Assembly, Lunch, and Board Meeting (中華民國比較病理學會會員大會暨理監事改選暨理監事會議)		
13:30~14:00	Case 360	Dr. Y. H. Chen (陳昱宏 醫師) Buddhist Tzu Chi General Hospital and University, Taiwan (慈濟綜合醫院暨慈濟大學)	Dr. C. W. Shih 施洽雯 主任
14:00~14:30	Case 361	Dr. K. J. Yu (余國睿 獸醫師) National Chung-Hsing University, Taiwan (國立中興大學)	
14:30~14:50	Coffee Break		
14:50~15:20	Case 362	Dr. C. T. Liang (梁鐘鼎 獸醫師) National Applied Research Laboratories, Taiwan(國家實驗研究院)	Dr. Y. H. Hsu 許永祥 主任
15:20~15:50	Case 363	Dr. Y. W. Lo (羅雅文 獸醫師) National Taiwan University, Taiwan (國立臺灣大學)	
15:50~16:20	Case 364	Dr. Y. T. Chen (陳怡庭 醫師) Kaohsiung Medical University Chung-Ho Memorial Hospital, Taiwan (高醫大附設中和紀念醫院)	
16:20~17:00	General Discussion (綜合討論)		

目　　錄

中華民國比較病理學會章程

第一章　總則

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

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

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

第四條　本會之任務如左：

一、　提倡比較病理學之研究與發展。

二、　舉辦學術演講會、研討會及相關訓練課程。

三、　建立國內比較醫學相關資料庫。

四、　發行比較病理學相關刊物。

五、　促進國內、外比較醫學之交流。

六、　其他有關比較病埋學術發展之事項。

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

第二章　會員

第六條　本會會員申請資格如下：

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

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

三、　贊助會員：贊助本會工作之團體或個人。

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

前項一、二、三項會員申請時應填具入會申請書，經一般會員二人

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

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

第八條　會員有遵守本會章程、決議及繳納會費之義務。

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

第十條　會員喪失會員資格或經會員大會決議除名者，即為出會。

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

第三章　組織及職員

第十二條　本會以會員大會為最高權力機構。

第十三條　會員大會之職權如下：

一、　訂定與變更章程。

二、　選舉及罷免理事、監事。

三、　議決入會費、常年會費、事業費及會員捐款之方式。

四、　議決年度工作計畫、報告、預算及決算。

五、　議決會員之除名處置。

六、　議決財產之處分。

七、　議決本會之解散。

八、　議決與會員權利義務有關之其他重大事項。

前項第八款重大事項之範圍由理事會訂定之。

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

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

本屆理事會得提出下屆理事及監事候選人參考名單。

第十五條　理事會之職權如下：

一、　審定會員之資格。

二、　選舉及罷免常務理事及理事長。

三、　議決理事、常務理事及理事長之辭職。

四、　聘免工作人員。

五、　擬訂年度工作計畫、報告、預算及決算。

六、　其他應執行事項。

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

理事長對內綜理監督會議，對外代表本會，並擔任會員大會、理事會主席。

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

理事長或常務理事出缺時，應於一個月內補選之。

第十七條　監事會之職權如左：

一、監察理事會工作之執行。

二、審核年度決算。

三、選舉及罷免常務監事。

四、議決監事及常務監事之辭職。

五、其他應監察事項。

第十八條　監事會置常務監事一人，由監事互選之，監察日常會務，並擔任監事會主席。

常務監事因事不能執行職務時，應指定監事一人代理之，未指定或不能指定時，由監事互推一人代理之。監事會主席（常務監事）出缺時，應於一個月內補選之。

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

第二十條　理事、監事有下列情事之一者，應即解任：

一、喪失會員資格。

二、因故辭職經理事會或監事會決議通過者。

三、被罷免或撤免者。

四、受停權處分期間逾任期二分之一者。

第二十一條　本會置祕書長一人，承理事長之命處理本會事務，令置其他工作人員若干人，由理事長提名經理事會通過後聘免之，並報主管機關備查。但祕書長之解聘應先報主管機關核備。

前項工作人員不得由選任之職員（理監事）擔任。

工作人員權責及分層負責事項由理事會令另定之。

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

第二十三條　本會得由理事會聘請無給顧問若干人，其聘期與理事、監事之任期同。

第四章　會議

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

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

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

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

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

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

第五章　　經費及會計

第二十九條　本會經費來源如下：

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

二、常年會費：一般會員新台幣五佰元，學生會員壹佰元。

三、事業費。

四、會員捐款。

五、委託收益。

六、基金及其孳息。

七、其他收入。

第三十條　　本會會計年度以國曆年為準，自每年一月一日起至十二月三十一日止。

第三十一條　本會每年於會計年度開始前二個月由理事會編造年度工作計劃、收支預算表、員工待遇表，提會員大會通過（會員大會因故未能如期召開者，先提理監事聯席會議通過），於會計年度開始前報主管機關核備．並於會計年度終了後二個月內由理事會編造年度工作報

告、收支決算表、現金出納表、資產負債表、財產目錄及基金收支表，送監事會審核後，造具審核意見書送還理事會，提會員大會通過，於三月底前報主管機關核備（會員大會未能如期召開者，需先報主管機關備查）。

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

第三十三條　本章程未規定事項，悉依有關法令規定辦理。

第三十四條　本章程經大會通過，報經主管機關核備後施行，變更時亦同。

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

中華民國比較病理學會第五屆理監事名單簡歷冊								
職別	姓名	性別	出生 年月日	學歷	經歷	現任本職	電話	傳真
理事長	劉振軒	男	42/10/9	美國加州大學戴維斯校區比較病理學博士	台灣養豬科學研究所主任	國立台灣大學獸醫專業學院院長	02-33663760	02-23633289
常務理事	呂福江	男	37/11/21	美國漢尼門大學病理學博士	國防醫學院病理學研究所所長	耕莘醫院病理部主任	02-22193391-65236 0968-666741	02-2193506
常務理事	祝志平	男	46/02/25	台大病理研究所碩士	台北醫學院講師	羅東聖母醫院病理科主任	039-544106-6113 0913-379889	039-572916
常務理事	李進成	男	49/06/06	英國倫敦大學神經病理博士	長庚醫院內科醫師	新光吳火獅紀念醫院病理檢驗科醫師	02-28389306	02-28389306
常務理事	葉坤土	男	43/01/05	中國醫藥學院醫藥研究所	彰化基督教醫院病理科主任	彰化基督教醫院病理科主任	(04)723-8598轉3370	
常務理事	張文發	男				國立中興大學獸醫學院動物疾病診斷中心副主任		
理事	許永祥	男	48/10/30	國立台大醫學院病理研究所碩士	台大醫院病理科住院醫師	慈濟醫院病理科主任	03-8565301-2197	03-8574265
理事	施洽雯	男	46/08/30	國防醫學院病理研究所	中山醫學院病理科副教授	羅東博愛醫院病理科主任	039-543131-2716	039-551543
理事	徐榮彬	男	49/01/10	中興大學獸醫博士	屏東縣家畜疾病防治所課長	屏東縣家畜疾病防治所所長	087-7224109	087-7224432
理事	陳憲全	男						
理事	張俊梁	男	45/5/6	國防醫學院醫學科學研究所博士	國防醫學院兼任助理教授	國軍桃園總醫院病理檢驗部主任	02-2303-2209 03-4799595 0966008531	02-2303-5192
理事	張聰洲	男	41/11/29	國立中興大學獸研所碩士班	國立屏東技術學院助教	國立屏東科技大學副教授	06-2333529	08-7740295
理事	賴銘淙	男	47/10/14	清華大學生命科學院博士	華濟醫院病理科主任	彰濱秀傳紀念醫院病理科主任	04-3250487	
理事	蔡睦宗	男				屏東縣防治所		
理事	廖俊旺	男		國立台灣大學獸醫學研究所博士	農業藥物毒物試驗所應用毒理組副研究員	中興大學獸醫病理學研究所副教授	04-22840894 ext 406	
常務監事	邱慧英	女	57/6/12	台大獸醫學研究所博士班	台灣動物科技研究所動物醫學組助理研究員	台灣動物科技研究所動物醫學組助理研究員	(037)585872	(037)585850
監事	林永和	男	46/02/24	台大病理研究所	台北醫學院病理科講師	台北醫學院病理科講師	02-27361661-641	02-23770054
監事	梁鍾鼎	男	51/01/25	台灣大學獸醫學研究所博士班	國家實驗動物中心副研究員	國家實驗動物中心首席獸醫師	02-2789-5569	02-2789-5588
監事	阮正雄	男		國立岡山大學大學院醫齒綜合研究科博士		童綜合醫院		
監事	江蓉華	男		國防醫學院醫學士	國軍花蓮總醫院病理部主任	耕莘醫院組織病理科主任		

中華民國比較病理學會

資產負債表

中華民國 99 年 12 月 31 日　　單位：新臺幣(元)

資　　產		負債 基金 暨　餘絀	
歷年歲末累計結餘	96,349		
提撥準備基金	0		
99 年度餘絀	-35,364		
		合作金庫活存	60,985
合　計	60,985	合 計	60,985

理事長:　　常務監事:　　秘書長:　　會計:

中華民國比較病理學會

收支決算表

中華民國 99 年 1 月 1 日至 99 年 12 月 31 日　　單位：新臺幣(元)

科目				決算數	預算數	決算與預算比較數		說明
款	項	目	名稱			增加	減少	
1			本會經費收入	**32,736**	**55,542**		22,806	
	1		入會費	200	6,000		5,800	
	2		常年會費	6,900	22,000		15,100	
	3		贊助會費	0	20,000		20,000	
	4		利息收入	84	42	42		
	5		其他收入	25,552	7,500	18,052		
2			本會經費支出	**68,100**	**55,200**	9900		
	1		人事費	22,600	18,000	1,600		
		1	兼職人員車馬費	15,600	12,000	3,600		第 48 次為聯合學術研討會，多支付講者費用
		2	其它人事費	7,000	6,000	1,000		
	2		辦公費	28,351	12,000	16,351		
		1	印刷費	1,925	9,000		7,075	
		2	旅運費	12,291	0	12,291		第 50 次邀請外國學者 Dr. E. Arifin 差旅費
		3	郵電費	5,955	3,000	2,955		
		4	公共關係費	8,180	0	8,180		第 50 次招待外賓餐費
	3		業務費	17,149	18,000		851	
		1	會議費	17,149	18,000	851		
	4		雜費支出	0	6,500		6,500	
	5		提撥基金	0	700		700	
3			本期餘絀	-35,364				

理事長：　　常務監事：　　秘書長：　　會計：

中華民國比較病理學會

基金收支表

中華民國 99 年 1 月 1 日至 99 年 12 月 31 日止

收　　入		支　　出	
準備基金		準備基金	0
歷年累存	10,400		
本年度提撥	0		
		結　餘	10,400

理事長：　　常務監事：　　祕書長：　　會計：

中華民國比較病理學會現金出納表

中華民國 99 年 1 月 1 日至 99 年 12 月 31 日

收　　入		支　　出	
科目名稱	金　　額	科目名稱	金　　額
上期結存	96,349	本期支出	68,100
本期收入	32,736	本期結存	60,985
合　　計	129,085	合　　計	129,085

理事長:  　　常務監事:  　　祕書長:  　　會計: 

中華民國比較病理學會

收支預算表

中華民國 100 年 1 月 1 日至 100 年 12 月 31 日

科目				預算數	上年度預算數	本年度與上年度預算比較數		說明
款	項	目	名稱			增加	減少	
1			本會經費收入	**55,542**	**55,542**			
	1		入會費	6,000	6,000			
	2		常年會費	22,000	22,000			
	3		贊助會費	20,000	20,000			
	4		利息收入	42	42			
	5		其他收入	7,500	7,500			
2			本會經費支出	**55,200**	**55,200**			
	1		人事費	18,000	18,000			
		1	兼職人員車馬費	12,000	12,000			1 人x1,000x12=12,000
		2	其他人事費	6,000	6,000			臨時人員工資(協助研討會辦理、資料寄發、會務連絡等)
	2		辦公費	12,000	12,000			
		1	印刷費	9,000	9,000			
		2	旅運費	0	0			
		3	郵電費	3,000	3,000			
		4	公共關係費	0	0			
	3		業務費	18,000	18,000			
		1	會議費	18,000	18,000			
	4		雜費支出	6,500	6,500			
	5		提撥基金	700	700			
3			本期餘絀					如有盈餘，得依規定提列 5%以上

理事長：　常務監事　秘書長：　會計：

CASE SIGNALMENT

51ST MEETING OF COMPARATIVE PATHOLOGY

March 12nd, 2011

(中華民國比較病理學會第 51 次比較病理學研討會)

Case No.	Presenter	Institution	Slide No.	Signalment
Case 357	Dr. Y.L. Chen (陳燕麟 醫師)	Department of Pathology, Cardinal Tien Hospital, Taiwan (天主教耕莘醫院病理科)	CTH	93-year-old, women
Case 358	Dr. C. W. Shih (施洽雯 醫師)	Department of Pathology, Lotung Poh-Ai Hospital, Taiwan (羅東博愛醫院病理科)	Cp10-11579	38-year-old, women
Case 359	Dr. Y. Lee (李遙 獸醫師)	School of Veterinary Medicine, National Taiwan University, Taiwan (國立臺灣大學獸醫專業學院)	NTU98-299	7-month-old, female, mixed breed cat
Case 360	Dr. Y. H. Chen (陳昱宏 醫師)	Buddhist Tzu Chi General Hospital and University, Taiwan (慈濟綜合醫院暨慈濟大學)	S2007-15618	27-year-old, women
Case 361	Dr. K. J. Yu (余國睿 獸醫師)	Graduate Institute of Veterinary Pathology, National Chung Hsing University, Taiwan (國立中興大學獸醫病理生物學研究所)	1010-669114	8-week-old, male, ICR strain mouse
Case 362	Dr. C. T. Liang (梁鐘鼎 獸醫師)	National Laboratory Animal Center, National Applied Research Laboratories, Taiwan(國家實驗研究院)	NYU00-233	3-year-old, male, mixed dog
Case 363	Dr. Y. W. Lo (羅雅文 獸醫師)	School of Veterinary Medicine, National Taiwan University, Taiwan (國立臺灣大學獸醫專業學院)	NTU2004-141R	11-year-old, male, domestic short hair cat
Case 364	Dr. Y. T. Chen (陳怡庭 醫師)	Dept. of Path., Kaohsiung Medical University Chung-Ho Memorial Hospital, Taiwan (高醫大附設中和紀念醫院病理科)	Kmu-11-00761	33-year-old, women

CASE DIAGNOSIS

51ST MEETING OF COMPARATIVE PATHOLOGY

March 12nd, 2011

（中華民國比較病理學會第 51 次比較病理學研討會）

Case No.	Presenter	Institution	Slide No.	Signalment
Case 357	Dr. Y.L. Chen (陳燕麟 醫師)	Department of Pathology, Cardinal Tien Hospital, Taiwan （天主教耕莘醫院病理科）	CTH	Small cell Carcinoma, Urinary bladder
Case 358	Dr. C. W. Shih (施洽雯 醫師)	Department of Pathology, Lotung Poh-Ai Hospital, Taiwan (羅東博愛醫院病理科)	Cp10-11579	Eosionphilic granuloma, Right suboccipital epidural mass
Case 359	Dr. Y. Lee (李遙 獸醫師)	School of Veterinary Medicine, National Taiwan University, Taiwan （國立臺灣大學獸醫專業學院）	NTU98-299	Eosinophilic granuloma with fungal infection, Skin
Case 360	Dr. Y. H. Chen (陳昱宏 醫師)	Buddhist Tzu Chi General Hospital and University, Taiwan (慈濟綜合醫院暨慈濟大學)	S2007-15618	Septa panniculitis with lymphocytic vasculitis
Case 361	Dr. K. J. Yu (余國睿 獸醫師)	Graduate Institute of Veterinary Pathology, National Chung Hsing University, Taiwan (國立中興大學獸醫病理生物學研究所)	1010-669114	Hepatotoxicity of SMA-AgNPs
Case 362	Dr. C. T. Liang (梁鐘鼎 獸醫師)	National Laboratory Animal Center, National Applied Research Laboratories, Taiwan(國家實驗研究院)	NYU00-233	Canine distemper virus infection combined pulmonary dirofilariasis
Case 363	Dr. Y. W. Lo (羅雅文 獸醫師)	School of Veterinary Medicine, National Taiwan University, Taiwan （國立臺灣大學獸醫專業學院）	NTU2004-141R	Hypertrophy osteopathy
Case 364	Dr. Y. T. Chen (陳怡庭 醫師)	Dept. of Path., Kaohsiung Medical University Chung-Ho Memorial Hospital, Taiwan (高醫大附設中和紀念醫院病理科)	Kmu-11-00761	Perivascular epithelioid cell tumor, in favor of lymphangiomyomatosi

CASE DIAGNOSIS

51ST MEETING OF COMPARATIVE PATHOLOGY

March 12nd, 2011

(中華民國比較病理學會第 51 次比較病理學研討會)

Case 357	Human	Small cell Carcinoma, Urinary bladder
Case 358	Human	Eosionphilic granuloma, Right suboccipital epidural mass
Case 359	Cat	Eosinophilic granuloma with fungal infection, Skin
Case 360	Human	Septa panniculitis with lymphocytic vasculitis
Case 361	Mouse	Hepatotoxicity of SMA-AgNPs
Case 362	Dog	Canine distemper virus infection combined pulmonary dirofilariasis
Case 363	Cat	Hypertrophy osteopathy
Case 364	Human	Perivascular epithelioid cell tumor, in favor of lymphangiomyomatosi

Contributors：
Y.L.Chen(陳燕麟)MD,孫政宏 MD,F.J. Leu 呂福江 MD PhD,江容華 MD
Department of Pathology, Cardinal Tien Hospital(天主教耕莘醫院病理科)

Clinical history：

This 93-year-old female complained of painless gross hematuria and difficult voiding 5 months ago. She came to ER for management and lab data showed pyuria and hematuria. KUB showed no radio-opaque urinary tract stone. Renal echo revealed only renal parenchymal disease. Cystoscopy was done and a papillary-growth bladder tumor was found. Then TUR-BT was performed and her symptoms improved. 2 weeks ago she had painless gross hematuria again. Under the impression of recurrent tumor, she was admitted. TUR-BT was performed again and symptoms resolved. 3 months later she had low back pain and MRI showed bone metastasis. Radiotherapy was suggested but the disease progressed. The patient was transferred to hospice care and expired 5 month later.

Diagnosis：Urinary bladder --- Small cell carcinoma

Gross Finding：

The specimen submitted consisted of multiple small pieces of bladder tissue measuring 2 gm in weight, fixed in formalin.

Grossly, they were white in color and soft in consistency. They were totally embedded.

Histopathologic Finding：

Microscopically, the sections show picture of small cell carcinoma. The neoplastic cells show scanty cytoplasm and small to medium-sized nucleus with nuclear molding and salt-and-pepper chromatin pattern.

Immunostain:

1) CK: (+) 2) CD56: (+)
3) synaptophysin: (+) 4) chromagranin: (-) 5) TTF-1: (-)

Differential diagnosis：

1. poorly differentiated urothelial carcinoma
2. metastasis small cell carcinoma
3. malignant lymphoma

Diagnostic criteriae：

1. Small cell carcinoma of the bladder is very similar to that of lung.
2. A malignant epithelial tumour consisting of small cells with scant cytoplasm, illdefined cell borders, finely granular nuclear chromatin, and absent or inconspicuous nucleoli.
3. Nuclear molding is prominent.
4. Positive for either neuroendocrine markers (CD56, chromogranin and synaptophysin)

Discussion：

Small cell carcinoma of the bladder defined as malignant neuroendocrine neoplasm derived from the urothelium which histologically mimics its pulmonary counterpart.

It's mean age is 66 y/o with male predominant and accounts for only 0.35-0.70% of all bladder tumors. Cigarette smoking may be associated (65%). Gross hematuria is the most common symptoms and other symptoms include dysuria or localized abdominal/pelvic pain.

Almost all the small cell carcinoma of urinary tract arises in the bladder. They presented as large solid, isolated, polypoid, nodular mass with or without ulceration, and may extensively infiltrate the bladder wall. The vesical lateral walls and the dome are most frequent topographies. Molecular evidence for the development of invasive small cell carcinoma out of urothelial carcinoma in situ is proved by identical point mutation of TP53 on 2008.

Most cases consists of small cell carcinoma with other histological type (urothelial carcinoma 70%, adenocarcinoma 8%, squamous cell carcinoma10%). The small cell carcinoma part is similar histopathological features as pulmonary counterpart but lower immunoreactivity for TTF-1. There was no positive staining with either CK20 or uroplakin.

Differential diagnosis should be considered poorly differentiated urothelial carcinoma, metastasis small cell carcinoma and malignant lymphoma. IHC can be quite helpful in distinguish these entities. Poorly differentiated urothelial carcinoma is negative for neuroendocrine markers. Metastasis small cell carcinoma can be noted by co-existing carcinoma and clinical settings. Malignant lymphoma is negative for both CK and neuroendocrine markers.

Aggressive behavior with dismal prognosis was noted. 5-year survival rate less than 10%. Most common locations of disease spread include: regional L.N, 56%; bone, 44%; liver 33%; and lung, 20%.

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Contributors：
C.W. Shih (施洽雯), M.D., M.S [1], H.H.Chen (陳幸鴻), M.D. [2], H.W. Lin (林湘文), M.D [2]
1. Department of Pathology, Lotung Poh-Ai Hospital (羅東博愛醫院病理科)
2. Department of neurosurgery, Lotung Poh-Ai Hospital (羅東博愛醫院神經外科)

CASE HISTORY:

Signalment: 38-year-old woman.

Clinical History:
The 38-year-old woman was admitted to our hospital with the chief complaint of diplopia and dizziness for one month. She went to the OPD of ophthalmology on 99-11. Diabetes mellitus with retinopathy was suspected by the ophthalmologist .
For the symptoms were persistent, she was referred to the neurologist on 99-11-22.
CT and MRI were arranged and a tumor mass was noted on the occipital area and measuring 2.5 x 2.1 x 1.4 cm. Bone destruction and suboccipital epidural involvement were also noted. Bone scan was performed on 99-12-23 and revealed increased MDP uptake in the right occipital bone, no other definite bony abnormality was demonstrable elsewhere. Under the impression of R/O malignancy, she was referred to the department of neurosurgery and admitted for further evaluation and management. Craniectomy for removal of right suboccipital epidural tumor was performed on 99-12-16. The tumor tissues were sent for frozen and paraffin sections.

Clinical Pathology:
RBC: 4.18x10^6/uL (0-5 x10^6/uL), Hb: 12.1 gm/dL (12.0-16.0 gm/dL), Hct: 35.7 % (37-47%), WBC: 10200/uL (4500-11000/uL), Plt: 41.1 x10^4/dL (15-40 x10^4/dL), Lymphocyte: 40.0% (20.0-45.0%), Neutrophil: 53.8% (45.0-75.0%), Monocyte:4.7% (0.0-9.0%), Eosinophil:0.9% (1.0-3.0%), Basophil:0.6% (0.0-1.0%). BUN:9 mg/dL (7-22 mg/dL), Creatinine:0.4 mg/dL (0.6-1.3 mg/dL), Glucose:129 mg/dL (70-110 mg/dL), AST: 26 U/L (5-40 U/L), ALT: 21 U/L (5-40 U/L), Na:143.4 mmol/L (133-145 mmol/L), K:3.7 mmol/L (3.3-5.1 mmol/L).

Gross Findings:
On gross examination, the tissue fragments were soft or hard in consistency, grayish- red to brown with flecks of yellow in color.

CASE RESULT:

Histopathologic Findings:
The tumor shows an abundance of histiocytes with grooved nuclei intermingled with large numbers of eosinophils. Also present in the lesion are lymphocytes, polymorphonuclear cells, and multinucleated giant cells. Hemorrhage is noted in areas. Nuclear atypia and mitosis are occasionally seen. No tumor necrosis is seen.

Immunohistochemistry:
The tumor cells were positive for LCA, CD68 and S100, and negative for cytokeratin.

Differential Diagnosis:
1. Osteomyelitis.
2. Giant cell tumor.
3. Giant cell reparative granuloma.
4. Aneurysmal bone cyst.
5. Eosinophilic granuloma.

Diagnosis: Eosinophilic granuloma.

Comments:
Langerhans' cell histiocytosis (formerly known as histiocytosis X) is characterized by a monoclonal proliferation of histiocytes. The disorder was first described on the basis of purely clinical observations more than 145 years age, after the Langerhans' cell was detected by the Paul Langerhans in 1865. The disease is manifested in three different clinical conditions: eosinophilic granuloma, Hand-Schuller-Christian disease, and Letterer-Siwe disease. Eosinophilic granuloma is the most benign of the three conditions. Hand-Schuller-Christian disease is the chronic systemic variant, and Letterer-Siwe disease is an acute, fulminant, systemic condition

Eosinopilic granuloma (EG) is the most common form of Langerhans' cell histiocytosis, accounting for 70% of all cases. EG manifests clinically as osteolytic lesions of the skull, long bones, ribs, and/or vertebrae; there is a notable lack of systemic involvement. Its incidence is slightly less than 1 per 200,000 population. Approximately 75% of affected patients are younger than 20 years, and men are affected twice as much as woman. The disease has no predilection to any particular race. Skull lesions are present in approximately

40% of all cases; some 20 to 30% of these lesions involve the temporal bone.
Little is yet known about the etiology and pathogenesis of Langerhans' cell histiocytosis, inflammation, autoimmunity and loss of controlled proliferation of Langerhans'cells are the assumed etiologies. There is no evidence that the disease originates in a malignant neoplastic process, and no underlying viral or genetic cause has been identified.
Langerhans' cells are highly differentiated histiocytes of the stellate dendritic antigen-presenting cell lineage. First recognized as a component of the normal human skin by Paul Langerhans in 1865, they originally derive from bone marrow. The importance of their immunologic role is now recognized: They are involved in immunosurveillance as they engage and present antigens to T-cells. They are able to detect foreign antigens entering the body through the skin and then to migrate from the skin to the lymph nodes. It is now known that the pathognomonic cell, the Langerhans' cell, excretes IL-1 and PG-E2 as to damage surrounding tissues. Interleukin-1 is a major osteoclast-activating factor and inhibitor of bone formation, thereby producing the classic osteolytic lesion of LCH.

EG is benign tumor is usually asymptomatic or may appear as a palpable, tender mass over the affected bone. Other symptoms are related to the affected bone and can include an infected ear when the mastoid is involved or severe back pain and scoliosis when the spine is affected. Rarely does it cause epidural hematoma, suppression of bone marrow and pathological fractures. When at skull, headache, neurological symptoms, chronic mastoiditis and exopthalmos may be the findings. The most common manifestations of the disease are bone lesions, whereas extraosseous involvement is less frequently seen. In order of decreasing frequency: skin
(55%), central nervous system (35%), hepatobiliary system and spleen (32%), lungs (26%), lymph nodes (26%), soft tissues (26%), bone marrow (19%), salivary glands (6%), and digestive tract (6%).

Radiological depiction of EG is necessary as to determine the activity and nature of the tumor. On plain radiographs, EG typically presents as a punched out lesion with reactive sclerosis. The cortex of the affected bone may present as thin, eroded or thickened due to new bone formation. The calvaria and especially the parietal bones are most often affected, followed by the mandible, the ribs and the pelvis. Lesions in the long bones are most often in the diaphysis (58%). Epiphysis is rarely involved (2%).

CT scan and MRI delineate the extent of the intramedullary and cortical penetration.
CT imaging of these patients best demonstrates the bony erosion caused by these lesions.

In early stages, the solitary EG appears as a radiolucent area adjacent to bone. As the soft tissue component breaks through the bony cortex, moth-eaten osteolytic defects can be appreciated. On MRI most lesions are hypointense on T1- weighted images and have a heterogeneous, intermediate to high signal intensity on T2-weighted images.

On gross examination, EG is a soft, granular or gelatinous mass. It appears gray red to brown with flecks of yellow. Microscopically, EG shows an abundance of histiocytes with grooved nuclei intermingled with large numbers of eosinophils and variable amount of neutrophils, lymphocytes and multinucleated giant cells. , Hemorrhage, necrosis and mitotic figures can also be seen. The histiocytes are positive with immunohistochemical stains CD1a and S-100. However, CD1a positivity also may occasionally be seen in other histiocytes, such as juvenile xanthogranuloma and Rosai-Dorfman disease. Finally, the presence of S100 protein remains a useful indicator of histiocytic disorders, although it is not diagnostically specific. A positive result of staining for S100 indicates the likelihood that lesional histiocytes are Langerhans' cells, indeterminate cells, or interdigitating dendritic cells. Under the electron microscope, the Langerhan's cell has tennis-racket--shaped cytoplasmic organelles called Birbeck's granules. Their function is unknown.

EG can heal without treatment, if not, the suggested treatment is the surgical curettage of the tumor or local infusion of cortisone. Treatment is almost always curative for EG.. Chemotherapy, radiotherapy and systemic use of cortisone are effective for cases of Hand-Schuller-Christian or Letterer-Siwe disease, in which multiple organ systems are involved.

In conclusion, EG is an uncommon condition that primarily affects children and adolescents. It is characterized by unifocal osteolytic lesion that often affect the skull. In general, no treatment is needed for localized osseous EG and often the biopsy is enough to initiate healing. Steroid injection, curettage excision or radiation may be necessary depending on the extent of the disease and the symptoms.

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Contributors：
Y. Lee (李遙), DVM , C.R. Jeng, (鄭謙仁), DVM, Ph. D
Graduate Institute of Molecular and Comparative Pathobiology, School of Veterinary Medicine National Taiwan University.

CASE HISTORY

Signalment: 7-month-old cat, mixed breed, female. Body weight was 2.4 Kg.

Clinical history:
There was a mass with ulceration on the left lateral hock joint and it had been operated for 3 ties by local private animal hospital. The mass was taken for biopsy on 4/16 in 1998.

Clinical pathology:
The results of blood examinations including CBC and leukocyte differential count on 4/16 were unremarkable.
RBC: 9.44 $x10^6$/μL (5-10 $x10^6$/μL), Hb: 14.8 g/dL (8-15 g/dL), Hct: 47.2% (30-45%), MCV: 50.0 fl (39-55 fl), MCH: 15.7 pg (13-17 pg), MCHC: 31.4 g/dL (30-36 g/dL). WBC: 3400 /μL (5500-19500 /μL), Neutrophil: 1972 /μL (2500-12500 /μL), Eosinophil: 272 /μL (0-750 /μL), Lymphocyte: 1088 /μL (1500-7000 /μL), Monocyte: 68 /μL (0-850 /μL), Plt: 88 $x10^3$/μL (300-700 $x10^3$/μL).
Albumin: 3.0 g/dL (2.3-3.9 g/dL), ALP: 76 U/L (14-111 U/L), ALT: 31 U/L (12-130 U/L), AST: 27 U/L (0-48 U/L), BUN:20 mg/dL (16-36 mg/dL), Creatinine: 1.3 mg/dL (0.8-2.4 mg/dL), Glucose:118 mg/dL (71-159 mg/dL), TP: 6.6 g/dL (5.7-8.9 g/dL).

Gross findings:
This tumor-like mass was on the left lateral hock joint, with ulceration.

CASE RESULT

Histopathologic findings:
The dermis is diffusely inflamed and contains generally large as well as irregular foci. These foci are composed of abundant eosinophilic materials and cell debris, girdled by numerous of macrophages, multinucleated giant cells, eosinophils, and other inflammatory cells including neutrophils and lymphocytes. Some fungal hyphae are suspected in the central region of foci. Many plasma cells and lymphocytes aggregate around the blood vessels.

The epidermal lesions consist of severe ulceration and exudation. Superficial crusts contain degenerating neutrophils and serum, in addition to necrotic epidermal debris.

Histochemistry:
Fungal hyphae can be recognized either in the middle of inflammatory and necrotic foci but also in necrotic dermoepidermal junction in GMS staining. In Masson trichrome staining, collagen fibers seldom present in the middle of inflammatory and necrotic foci in dermis, instead, the architecture of fibrostroma is compressed and destroyed multifocally by this inflammatory and necrotic foci.

Morphological diagnosis:
Necrotizing ulcerative dermatitis, severe, locally extensive, chronic, eosinophilic pyogranulomatous, with intralesional fungal elements, skin, feline.

Differential diagnoses:
1. Mycotic dermatitis
2. Eosinophilic granuloma complex

Final diagnosis: Eosinophilic granuloma with secondary fungal infection.

Discussions:
The eosinophilic granuloma complex (EGC) in the cat actually consists of three similar diseases: Eosinophilic plaque (EP), indolent ulcer (IU), and eosinophilic granuloma (EG). These diseases are best thought of as inflammatory reactions of the skin. The term eosinophilic dermatoses has been proposed to describe those feline diseases included in EGC, but the term EGC is still commonly used.

EP in cats is usually seen on the ventral abdomen or inner thigh, and typical lesions show as pruritic, erythematous and erosive coalescing papules and plaques. Epidermal hyperplasia with spongiosis and prominent eosinophilic exocytosis with possible formation of intra-epidermal eosinophilic vesiculo-pustules and diffuse eosinophilic infiltration of the dermis are reported as common features.

Feline IU (synonyms: rodent ulcer, eosinophilic ulcer, lip ulcer) refers to a painless, non-pruritic and non-bleeding ulcerated lesion most commonly located on the upper lip.

Reported histopathological findings vary from an ulcerative dermatitis with a diffuse eosinophilic infiltration and foci of collagen degeneration (less prominent than in EG) to an ulcerative neutrophilic and fibrosing dermatitis.

EG (synonyms: collagenolytic granuloma, linear granuloma) classically occurs as multiple nodules, variably pruritic, orientated linearly on the caudal thigh or, less commonly, as single nodular lesions located anywhere on the body, including the footpads, the lower lip and the oral cavity in cats. Dermal foci of an amorphous to granular, eosinophilic to partly basophilic debris, which is considered as a mix of degenerated collagen and degranulated eosinophils, are reported as being distinctive histopathological features. Moreover, small foci of collagen degeneration, in which degenerated collagen fibers are surrounded by degranulated eosinophils, are commonly described and named flame figures. However, collagenolysis is not a consistent or predictable finding in feline EG stated by some researches. Dermal infiltrates in EG vary from predominantly eosinophilic to lymphocytic and histiocytic. In some cases epithelioid and multinucleated cells form a palisading granulomatous reaction around the eosinophilic debris. The epidermis is moderately hyperplastic and occasionally ulcerated.

In spite of the report of a distinctive histopathological appearance, pathological features consistent with those recognized entities of the EGC are sometimes observed simultaneously in feline cutaneous biopsies.

EGC has been attributed, although not definitely proven, to hypersensitivity reactions to arthropod (mosquito-bite or flea), parasite, food and environmental allergens. Over the last two decades, several other causes have been proposed for EGC, including viral and bacterial infections, chronic trauma and autoimmune reactions. Nevertheless, in most cases EGC remains idiopathic. The etiopathogenesis of EGC is still unknown.

Definitive diagnosis of the EGC must be made on histopathology. There are simply too many differential diagnoses when just based on visual examination, such as neoplasias (lymphoma, mast cell tumor, etc.), proliferative, non-neoplastic conditions (plasmacytic dermatitis) or herpesvirus dermatitis. In the case presented here, those diseases can be ruled out since multifocal foci in dermis composed of large amorphous eosinophilic debris with eosinophils, macrophages, as well as multinucleated giant cells surrounding, which highly corresponds with eosinophilic granuloma in histopathological findings.

There is a contradiction in the presented case. Moderate amount of fungal hyphae in the middle of foci is observed and evidenced by GMS staining, which is likely associated with granulomatous lesions. However, fungal elements should not trigger that much eosinophils as seen in this case, unless these fungi are not the primary pathogens. Therefore, it suggests that eosinophilic granulomatous lesions may occur followed by secondary fungal infection, which is possibly by patient's licking or self-traumatism. Phaeohyphomycosis, a wide variety of opportunistic dematiaceous fungi, is the most suspected category based on morphology of hyphae.

Therapeutic options for EGC include the use of glucocorticoids, essential fatty acids and cyclosporine. Some veterinarians recommend interferon alpha as alternative medicine. Other therapies, such as chlorambucil, aurothioglucose and progestagens, have been recommended in the literature; these are expensive, usually unlicensed, require careful monitoring and are not always effective but are, nevertheless, worth considering. If the patient has been confirmed as allergy, diet control, parasite prevention, or environmental cleaning should be considered. Although our patient was negative for FIV/FeLV and parasite examinations, it is hard to rule out these factors. Finally, the skin ulcer of our patient got worse regardless oral medicine was administered, thus skin transplantation was operated.

In conclusion, cats frequently present with various manifestations of the so-called EGC including three major forms: EP, IU, and EG. These forms have distinct histological patterns and may be regarded as different cutaneous reaction patterns to the same underlying cause. The etiopathogenesis is not sure, but several risk factors have been emphasized. In the present case, the progression of disease should be chronic since EG accompanied with fungal infection are diagnosed, and the latter just complicates not only the pathological diagnosis but also the treatment.

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Contributors：
Chen Yu-Hung (Medical student)(陳昱宏),Chang Yin-Sho (張茵琇), Hsu Yung-Hsiang (許永祥), MD
Buddhist Tzu-Chi General Hospital and University (佛教慈濟綜合醫院暨慈濟大學)

Case History:

Signalment: 27-year-old woman

Clinical History:
A 27-year-old female complained of acute painful red eyes with floaters, which developed 4 days after the third vaccination of quadrivalent Human pappiloma virus (HPV) vaccine. She denied any medication use, recent life change or family history of autoimmune disease. There was no discomfort following the first and second doses of vaccination. There were also bilateral painful knees with morning stiffness, painful erythematous nodules at bilateral anterior legs, vertigo and hearing impairment. On examination, a visual acuity of 16/20 was noticed in each eye, and an intraocular pressure of 6 and 7 mmHg in her right and left eye respectively. Biomicroscopy examination revealed ciliary injection, an anterior chamber reaction of three plus cells with keratic precipitates and snowball aggregates of inflammatory cells in vitreous. Fundus examination showed some creamy choroidretital infiltrates around left macula. There was also peripheral leakage on fluorescein angiography. Laboratory examination revealed an elevated erythrocyte rate (34 mm/hr). Complete blood cell count was unremarkable. Rapid plasma antigen, Treponema Pallidum Hemagglutination, anti-nuclear antibody and rheumatic factor were non-reactive. The human leukocyte antigen B27 was negative. Chest X-ray showed no hilar lymphadenopathy. Skin biopsy of those local erythematous papules disclosed panniculitis with lymphocytic vasculitis. Oral prednisolone 1 mg/kg/day, methotrexate 5mg weeksly and topical betamethasone 1% hourly were prescribed. Prednisolone was tapered in 4 months. The skin lesions and arthralgia resolved after the treatments and the final visual acuity was 20/15 in both eyes.

Clinical Pathology:

2007 Nov. 26th WBC = 8320/uL, ESR = 34, CRP = 0.46 mg/dL
2007 Nov. 27th ANA, RF, HLA B27, STS-RPR, TPHA were negative
WBC 9750/uL, ESR = 32, CRP = 0.545 mg/dL
IgA = 448.9
M. pneumonia Ab = 1:320, Cold HA = 1:16
ASLO = 1:80
2007 Nov. 30th WBC = 11540/uL, ESR = 29, CRP = 0.149 mg/dL

Gross Findings: Some erythematous papules formation in the bilateral legs

Case result:

Histopathological Findings:

Septal panniculitis with lymphocytic vasculitis

Immunohistochemistry: diffuse CD3(pan-T) and CD68(macrophage) positive along the capillary and subcutaneous adipose tissue.

Differential Diagnosis:

1. Panuveitis and septal panniculitis (erythema nodosum) due to human papilloma virus vaccine
2. Streptococcal infection related erythema nodosum
3. Lupus erythematous panniculitis (Lupus profundus)
4. Behcet's disease
5. Sarcoidosis
6. Weber-Christian Disease

Diagnosis: Panuveitis and septal panniculitis (erythema nodosum) and lymphocytic vasculitis due to human papilloma virus vaccine

Discussion: HPV vaccine-related adverse events have been reported, including HPV-related autoimmune phenomena such as scleroderma, rheumatoid arthritis and once been reported, uveitis [1, 2]. To our patient, there was pan-uveitis with also arthritis, panniculitis and some acoustic symptoms. The pathology of skin biopsy revealed panniculitis with lymphocytic vasculitis. There are many diseases that are associated with panniculitis, including Weber-Christian Disease, Erythema nodosum and erythema induratum or certain autoimmune diseases. Weber-Christian Disease can be excluded for that there are no febrile symptoms and the paniculitis was localized to leg in our patient. Autoimmune disease related panniculitis was unlikely due to absence of autoimmune serology and history. Erythema nodosum, according to the physical finding, was the most possible diseases considered at that moment. However, the etiologic survey for erythema nodosum revealed no satisfactory result. Another problem is that though small vessel inflammation occasionally occurs in erythema nodosum [3], lymphocytic vasculitis was not a typically finding. The mystery left now is “what” caused the panniculitis. There was currently no report of HPV vaccine related Erythema nodosum. With the strong temporal relationship and clustering of many auto-immune-like symptoms, the most possible explanation now is that the panniculitis may be related to HPV vaccine. Recently, Muñiz AE had reported a case of anthrax vaccine related lymphocytic vasculitis [4]. This may indicates that vaccine can produce lymphcytic vasculitis, just as in our patient. Furthermore, Costanzo et al. had reported that there was molecular mimicry between HPV type 16 E7 oncoprotein and human self-proteins, and there were also wide-spread similarities in many regulatory processes [5]. This explains that the post-vaccination auto-immune response can be extensive through-out multiple organ systems.

The licensed age for this vaccine was between 9 and 26 years old. Ages older than 26 is not recommended for vaccination due to the possibility of existing HPV infection. The vaccination age was 27 in our patient; this may indicates that there might be existing HPV infection before vaccination. This may explains that why the immune response is more severe in our patient.

We believe that this is a novel manifestation of post-HPV vaccination response. Recently, HPV vaccine licensed age has been considered prolonged in certain countries. Nevertheless, older age means higher risk of existing HPV infection, and, higher risk of developing severe adverse event after vaccination. For all clinical physicians, caution should be paid while prescribing quadrivalent HPV vaccine for woman who aged over 26 years old or risk of existing HPV infection.

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Contributors:

Yu, K.J.(余國睿), DVM,[1]; Hsu, S.H.(徐善慧), PhD,[2], Lin, C.C.(林江珍), PhD,[2], Chou, F.Y. (周鳳英), PhD,[3], Chang, W.F.(張文發) DVM., MS,[4] and Liao, J.W.(廖俊旺), DVM., PhD. [1, 4, *]

[1] Graduate Institute of Veterinary Pathology, National Chung Hsing University (國立中興大學獸醫病理生物學研究所)

[2] Institute of Polymer Science and Engineering, National Taiwan University (國立台灣大學高分子科學與工程學研究所)

[3] Institute of Nuclear Engineering and Science, National Tsing Hua University (國立清華大學核子工程與科學研究所)

[4] Animal Disease Diagnostic Center, National Chung Hsing University (中興大學動物疾病診斷中心)

CASE HISTORY:

Signalment: Male mouse, ICR strain, 8 week-old, experimental toxicity test

Case History:

Eighty 8-week-old, male, ICR mice were divided 16 groups. Dosages of 0, 0.25, 0.5 and 1 mg/kg body weight of poly(styrene-*co*-maleic anhydrides, SMA)-nanosilver (SMA-AgNPs, 5-15 nm) were intravenously injected via tail vein to mice. Mice were sacrificed at intervals of Days 1, 2, 3, and 7 for the time course study. This case was intravenously injected with 1 mg/kg body weight of SMA-AgNPs. After injection, the mouse showed signs of dullness, paleness, rough coat, and abdominal breathing and was sacrificed at Day 1.

Gross finings:

At necropsy, liver became swelling, marked congested with multiple hemorrhages on the surface. No significant gross lesion was observed in the other organs. The organs were fixed by 10% formalin for histopathological examination.

CASE RESULTS:

Microscopic findings:

Results from histological examination displayed that multiple, severe congested and hemorrhage was noted on livers. Injured hepatic cells presented marked acute swelling, vacuolization and apoptosis that accompanied with increasing inflammatory cells in the blood vessels and hemorrhages were also noted in the gall bladder.

Hematology and biochemistry examination:

The mouse showed elevation of white blood cell count, normocytic and normochromatic anemia with lower platelet counts in blood examination. The ratio of monocyte was increased in the white blood cell differentiation. Unfortunately, biochemistry parameters of these toxic mice could not be evaluated due to interval death or hemolysis was found.

Transmission electron microscopy:

TEM's results presented that the rough endoplasmic reticulum in hepatocytes was dilated and compressed to nucleus. The hepatic mitochondria appeared enlarged and abnormal in shape with vacuolization, partial loss of their double membrane and reduced mitochondrial cristae. The mitochondria had assumed a rounder, almost circular profile, many with central holes, so-called ring mitochondria.

Apoptosis assay:

Cellular DNA were extracted from this mouse liver, and DNA laddering visualized in an 2% agarose gel by ethidium bromide staining. Moreover, the damaged hepatocytes were positive staining by using the TUNEL immunohistochemically assay.

Silver concentration analysis:

Silver residues in organs were analyzed by using inductively coupled plasma mass spectrometry (ICP-MS), results indicated that SMA-AgNPs distributed mainly in spleen, followed by liver and lung; otherwise only a small amount of silver were detectable in the brain after 1 day of i.v. injection.

Diagnosis: Hepatotoxicity of SMA-AgNPs in mice

Discussion:

Nanotechnology is an enabling technology that deals with structures ranging from approximately 1-100 nm in at least one dimension. Because of their small particles size,

novel physicochemical properties, and easy surface modification, nanotechnology is rapidly expanding and used in various areas, such as health care, consumer products, ICT, food and feed, environmental health, and agriculture (Wijnhoven et al., 2009). One of the substances used in nanolized formulation is silver (nano-silver), due to their superiority in antibacterial activity. Despite the benefit of nanosilver in the fields of biomedical and industrial applications, there is only limited information about the possible risks of different exposures to nanosilver particles *in vivo*.

SMA-AgNPs were conducted *in vivo* tests to evaluate its biocompatibility and biosafety via intravenous. In this case presentation, mouse was intravenously injected 1 mg/kg body weight of SMA-AgNPs via tail vein, and then sacrificed after 24 hours of treatment. Results revealed that toxic signs such as depression, abdominal breathing, and paleness after treatment. This mouse also showed elevation of white blood cell count, and normocytic and normochromatic anemia with lower platelet in blood. Nanosilver particles (AgNPs) may be as foreign bodies, when entering the blood vessels to induce severe tissue damage. Such as high dose of SMA-AgNPs (1 mg/kg) could be immediately stimulated chemokine increasing, and lead to the proliferation of white blood cells, especially monocytes are response to clean foreign objects and injury tissue.

In addition, SMA-AgNPs also decreased the number of red blood cell, hemoglobin and hematocrit compared with normal. Studies have shown that nanosilver particles may increase interleukin-1 and TNF-α release (Park et al., 2010), resulting in the activation of coagulation system and inhibit blood clotting mechanisms. Nanosilver particles also makes the endothelial cells injured, and prompting release of tissue factor, platelet aggregation and activation of the intrinsic path caused disseminated intravascular coagulation. Silver nanoparticles showed low hemolytic performance on human red blood cells (Kim et al., 2008). Similar lesions were found in this case, for this, we suggested that hepatocytic death and hemorrhage are related to the treatment of SMA-AgNPs. However, the toxicity in the hematology is still unknown.

Liver is the main target organ after i.v. treated with SMA-AgNPs in mice. Grossly, liver became swelling, marked congested with multiple hemorrhages on the surface. Histological examination displayed multiple, severe congested and hemorrhage on livers. Injured hepatic cells presented marked acute swelling, and apoptosis toxicity. DNA laddering and TUNEL assay also confirmed that SMA-AgNPs induced cell death via apoptosis. Nanosilver is also known cytotoxic agents, inducing apoptosis in NIH3T3 fibroblast cells. Treatment with

nanosilver induced the release of cytochrome c into the cytosol and translocation of Bax to mitochondria, indicating that nanosilver-mediated apoptosis is mitochondria-dependent (Hsin et al., 2008). In this case, transmission electron microscopy showed that chromatin compaction/margination and many abnormal mitochondria obverved within hepatocytes. This phenomenon may also indicate that programmed cell death is related to mitochondrial injury in hepatocytes.

Nanoparticles less than 100 nm in diameter can enter cells, those with diameters below 40 nm can enter the cell nucleus, and those that are smaller than 35 nm can pass through the blood–brain barrier and enter the brain (Dawson et al., 2009). The 20 nm silver particles distributed mainly to liver, followed by kidneys and spleen, whereas the larger particles (80 and 100 nm) distributed mainly to spleen followed by liver and lung (Lankveld et al., 2010). In this case, SMA-AgNPs distributed mainly to spleen, followed by liver and lung after 24 hours of treatment. The size of nanoparticles used in this experiment are approximately from 5 to 15 nm, but the distribution pattern of silver is similar with 80 and 110 nm (Lankveld et al, 2010), and the silver concentration is lower in the brain. It is suggesting that SMA-AgNPs can integrate into larger particles *in vivo*, thus changing the particle distribution kinetics in body. However, the mechanism of hepatotoxicity is needed to elucidate in the future study.

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Contributors:
Chung-Tiang Liang [1,2](梁鍾鼎) DVM,MS；Ling-Ling Chueh [1](闕玲玲) DVM, PhD；Victor Fei Pang [1](龐飛) DVM, PhD；Chin-Cheng Lee[3](李進成) MD, PhD；Chen-Hsuan Liu[2](劉振軒) DVM, PhD
[1]National Laboratory Animal Center, National Applied Research Laboratories, Nan-Kang, Taipei 115
[2]Department and Graduate Institute of Veterinary Medicine, College of Bioresources and Agriculture, National Taiwan University, Taipei 106.
[3]Shin Kong Wu-Ho-Su Memorial Hospital, Taipei 111

Clinical History: Canine, NTU00-233, male, mixed, 3-year-old, came from shelter with neurological signs

Diagnosis:
Canine distemper virus
Pulmonary dirofilariasis co-infection

Gross Findings:
Consolidated, congested pulmonary lesions were noted. Adult heartworms were noted in the pulmonary artery.

Histopathological findings:

Significant vacuolating, demyelinating lesions in cerebral periventricular, and cerebellar white matter were noted. Extensive alveolar and bronchiolar plugging of neutrophils, necrotic debris, as well as macrophages were present. Eosinophilic intranuclear and cytoplasmic inclusion bodies were present in the bronchiolar and alveolar epitheliar cells, pulmonary macrophages, as well as astroglia. Lymphoid tissues depletion and necrosis was noted in the spleen .Eosinophilic intranuclear inclusion bodies were present in the white pulps. Few eosinophilic small cytoplasmic inclusion bodies were present in the pelvis and bladder mucosa epithelium.

Within the pulmonary artery lumen there are multiple cross sections of adult male and female, 1 mm in diameter nematodes, with a thin eosinophilic cuticle; prominent lateral cords that possess an internal lateral cuticular ridge; tall coelomyarian/polymyarian musculature; a small intestine lined by few multinucleate epithelial cells; and paired uteri or a single gonad.

Quite a few of microfilariae were present in pulmonary capillaries but were also noted in the alveolar interstitium with mixed leukocytic aggregates and erythrocytes.

Laboratory results:

RT-PCR for CDV (+)

Discussion:

Canine distemper (CD) is a naturally-occurring, Morbilliviral (family Paramyxoviridae)-induced disease of the systemic infections or CNS infection of dogs and their relatives and exotic species. CD has been the most important viral infection in dogs with high mortality in Taiwan. The disease often affected 3 to 6-momth-old unvaccinated dogs, causing respiratory, digestive and central nervous system（CNS）signs. However, since 1995, the cases of canine demyelinating encephalitis resembling different CDV strain infection have been emerging. The disease mainly affected mature dogs with CNS signs and high mortality without any other signs. The characteristic features of the histologic lesions were white matter demyelination of CNS, astrogliosis, eosinophilic intranuclear inclusion bodies, and occasional syncytial giant cells.

A study of CD virus infection indicated that demyelination occurred about 21 to 24 days after infection, whereas the pathological mechanism of this selective destruction of myelin sheaths was not clearly identified. Based on the previous studies, it is proposed that demyelination may be caused by the following mechanisms; (1) deleterious effects of CD virus on astrocytes associated with a variety of cytokines release, (2) lytic infection of the oligodendrocytes, (3) an immune attack oligodendrocytes and myelin sheath, or (4) by a "bystander effect" of inflammatory mediators. Multinucleated syncytial cells in measles and CD virus infection may be induced by the viral fusion protein mediated by host tissue proteases. Previous studies proposed that syncytia may be sites of defective viral replication, thus providing the mechanism of viral persistence in the CNS. Additionally, Toxoplasmosis (cases 16 & 17 in 2nd Comparative Pathology Conference) often occurs with CD in dogs. Lesions of both diseases should be carefully sought in pathological examination.

In CDV infection, the inflammatory response is associated with viral clearance from the lesions. Only 53% (42/79) inflammatory demyelinating lesions, CDV antigen could be demonstrated. Sometimes, the negative result in IHC staining of CDV antigen should consider destroyed by ethanol fixation, different mAb directed against the H and F protein showed no immunoreaction in the diseased brains and immunostaining was prominent in early and subacute and reduced in chronic demyelinating lesions. The NP-2 epitope showed strong positive results in all stages of demyelination.

In CDV infection, it was shown that 95% of all infected cells were astrocytes, only 2% to 8% restricted infection of oligodendrocytes and expressing CDV viral RNA. The infection of glial cells in the white matter precedes demyelintion. It is generally believed that the acute demyelinating lesions are due to the direct lytic effect of CDV, whereas the chronic demyelinating lesions are probably immune mediated. CDV antigen was observed within the inclusion bodies, neuron, astrocytes, and cerebellar cortex granule and cerebellar Purkinje cells.similar to previously reports. However CDV viral antigen-postive cells were frequently found at the edge and outside of the lesions, where the white matter appeared to be still intact. Additionally, CDV infection in neurons often involved the perikaryon and neuronal process.

Immunohistochemical detection of CDV antigen in tissue sections is superior to the demonstration of inclusion bodies or syncytial cells for the diagnosis of canine distemper encephalitis. However, inclusion bodies were only found in 31(6/19) to 48% (16/33) cases of CDV infection. Inclusion bodies are often scarce, and sections had to be scanned for a long time to detect them. This was not the case with immunohistochemical demonstration of CDV antigen.

Dirofilariasis (Heartworm Disease) is caused by the filarial organism, *Dirofilaria immitis.* While *D. immitis* can infect over 30 species of animals, the dog is considered its definitive host. *D. immitis* is a vector borne disease that is transmitted via the mosquito. *Aedes, Anopheles*, and *Culex* spp. are the most prominent mosquitos responsible for transmitting this disease. Adult heartworms are present in the right ventricle and extend into the pulmonary artery. Adults have a thin cuticle with internal ridges, coelomyarian/polymyarian muscles, prominent lateral cords, small diameter intestine lined by few multinucleate cells, and paired uteri. Adult females are 230-310 mm long and 1-2 mm in diameter; adult males are 120-190 mm long and 0.7-0.9 mm in diameter. The high incidence of microfilariae of Dirofilarisis with CDV was also noted. However, the discrimination between different species of canine microfilariae must depend on PCR.

The mosquito obtains microfilariae from feeding on an infected host Depending on environmental conditions, the first stage larvae (L1 microfilariae) will progressively mature to second stage larvae (L2) and then to third stage larvae (L3). Studies suggest that development of the infective L3 stage is directly related to temperature. Maturation requires 24 hours of temperatures above 64˚F for at least one month. If temperatures are consistently greater than 80˚F, larvae will mature to L3 within 10-14 days. If at any point the temperature

drops to 57˚F, larval development will be retarded. When mature, the L3 larvae migrate to the labium of the mosquito. During the mosquito's next meal, the larvae are deposited onto the host and molt to L4 larvae within 3 days. After two months the L4 larvae will molt into L5 larvae and relocate from the subcutaneous tissue to the pulmonary arteries. These larvae will become sexually mature adults over the next 2-3 months. If both sexes of *D. immitis* are present, they will mate and microfilariae will be released into the bloodstream. Microfilariae can be seen in the blood approximately 6-7 months after exposure to the infective L3 larvae.

Antigen testing is the preferred diagnostic method to detect *D. immitis* infections. These tests detect parasitic antigens that are found on mature female heartworms. Therefore, it is imperative that the test be used at least 7 months post-infection. Antigen tests have both high specificity and high sensitivity. As with all tests, false positives and false negatives may occur. It is very important to follow the manufacture's guidelines when using their antigen test. A negative result can have three interpretations: 1) heartworm infection is not present, 2) infection with immature filarids (subadults) may be present, or 3) the dog has a unisex heartworm infection consisting of male adult worms (remember that the test detects female heartworm antigens), and 4) the test kit has been refrigerated and not warmed to room temperature before use. A positive result can mean the dog is infected with heartworms or an infection has recently been cleared but antigens are still present. False positive test results may occur due to technical error, such as delayed reading of the test.

Parameter	***D. immitis***	***A. reconditum***
Length (in microns)	286-340	258-292
Width (in microns)	5-7.5	4.5-5.5
Shape (anterior end)	Tapered	Blunt
Shape (body)	Straight	Curved
Shape (tail)	Straight	Button hook
Number present	Few to many	Few
Movement	Stationary	Move across slide

Table 1. Morphologic differentiation of *D. immitis* and *A. reconditum* filariae.

Diagnostic criteria:

1. Lung: Bronchointerstitial pneumonia, necrotizing to granulomatous, histiocytic and lymphoplasmacytic, subacute, multifocal, severe, with ensinophilic intranuclear inclusion bodies and microfilariae of Dirofilarisis , etiology consistent with canine distemper virus and *Dirofilaria immitis* infection, canine.
2. Pulmonary artery: Endarteritis, proliferative, chronic, focally extensive, moderate, with intraarterial male and female nematodes, consistent with *Dirofilaria immitis* infection.
3. Spleen and lymph node: Spenitis and lymphadenitis, necrotizing, with lymphoid depletion, subacute, multifocal, severe.
4. Cerebellum and cerebrum: Demyelination, multifocal, subacute, severe, cerebellum and cerebral periventricular white matter.
5. Inclusion bodies: Cowdry type A, eosinophilic intranuclear and few cytoplasmic inclusion bodies, cerebellum, lung, spleen, lymph node, peyer's patch, bladder mucosa and renal pelvis.

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Contributors:
Lo, Ya-Wen (羅雅文),; Yeh, Lih-Seng (葉力森), D.V.M., Ph.D.; Pang, Victor-Fei (龐飛), DVM, Ph. D.; Jeng, Chian-Ren (鄭謙仁), DVM, Ph. D
Graduate Institute of Molecular and Comparative Pathobiology, School of Veterinary Medicine National Taiwan University (台大獸醫專業學院 分子暨比較病理生物學研究所)

CASE HISTORY:

Signalment: 11-year-old, neutered male, domestic short hair cat

Clinical History:
The patient presented hind leg paresis at 2004/01/10. Rt. carpal swelling was noted at 2004/01/24. The clinics found that hind legs muscle atrophy/ both hocks swelling (restricted motion range) and elbow joints of both sides swelling (Lt>Rt). BUN slightly increases in blood test. Atrophy of kidney was revealed under ultrasound examination. Antibiotics and steroids showed no improvement and died at home at 2004/02/13.

Gross Findings:
The body revealed obesity. The swelling of the extremities was caused by proliferation of osteoid tissue in the joints of four limbs. The increased translucent fluid was noted in the abdomen and thorax. Lung revealed edema with white spots and fibrins on the surface. Shrink of both kidneys with rough surface were noted.

CASE RESULT:

Histopathologic Findings:
Kidney: Degeneration and necrosis of proximal tubular epithelium, and massive fibrosis in the renal medulla, confirmed by trichrome Masson stain, are noted.

Thyroid and parathyroid glands: hyperplasia of parathyroid gland, which compress the thyroid gland and causes follicular atrophy of thyroid gland, shows glandular growth.

Hypertrophic bone: Osteoblasts deposit osteoid on the cortical bone and lay down loose, woven trabeculae perpendicular to the original cortex, and form a clear dark resting line. Osteoclasts and fibroblasts are also noted in the hypertrophic region. All the lesions locate outside the joints and no articular space is involved.

Diagnosis:
1. Chronic renal medullary fibrosis and nephrosis
2. Parathyroid gland hyperplasia
3. Hypertrophic osteopathy

Discussion:
Hypertrophic osteopathy (HO) is a syndrome of periosteal hyperostosis of the appendicular skeleton, typically starting from the distal portions of limbs. The new bone formation is usually symmetrical with nodular, spiculated, or palisade patterns, and leading to irregular outlines of the involved bones. Hypertrophic osteopathy may cause limb soreness and may result in ambulant disability.

Hypertrophic osteopathy is most commonly associated with intrathoracic neoplasia or inflammation and is more commonly documented in humans and dogs; it has also been noted to occur in the horse, cow, sheep, cat, fowl, and various other more exotic species. In humans, a number of thoracic or non-thoracic disorders have been linked with HO development, including cyanotic congential heart defects, neoplasia, pulmonary diseases, and gastrointestinal diseases. In cats, reports are limited and highly sporadic. Almost all reported feline HO cases have been associated with neoplasia.

The majority of affected animals are presented to the clinician with complaints of lameness and reluctance to move about. They invariably exhibit symmetric, nonedematous and firm swellings of the distal portions of all four legs. On physical examination, the limbs are generally warm to the touch, often pulsatile and sometimes painf'ul upon deep digital palpation. The normally loose skin over the metacarpal and metatarsal regions feels unusually taut.

Radiographically, hypertrophic osteopathy usually is seen as a bilaterally symmetric and generalized periosteal proliferative reaction that affects primarily the long bones of the appendicular skeleton. Initially only soft tissue swelling of the extremities may be seen with little to no bony abnormality. The periosteal proliferative changes have a variable spectrum from a smooth and regular (nonaggressive) to a scalloped or lacy (more aggressive) appearance. The distal portions of the limbs frequently have the earliest bony involvement, characteristically present on the abaxial aspects of the second and fifth metacarpal and/or metatarsal bones. The cortex and medullary portions of the bone are normal but may appear partially obliterated by the superimposed periosteal disease. The penosteal proliferative reaction may spread proximally to invest eventually all of the bones of the limbs (including scapula, carpal and tarsal bones) and less commonly the ribs, pelvis, and vertebrae; in one report even the penile bone was involved.

The precise pathogensis of HO is yet to be determined. Increased circulation to the extremities secondary to neurogenic or humorally mediated mechanisms is thought to play a major role in new bone formation. Current studies in humans suggest that vascular endothelial growth factor and platelet-derived growth factor may be key elements in the development of HO.

Grossly, specimens from advanced cases have coarse osseous exostoses that may be quite nodular, covering the entire cortical surface from end to end. Vessels and tendons are located in deep grooves within the osseous proliferation. The microscopic progression of the disease is characterized primarily as a proliferation of vascular connective tissue that invests the bones and tendons of the distal limb. Spicules of new bone form perpendicularly to the underlying cortex. Chondroid or fibrochondroid metaplasia may occur in this area of new-bone formation. As the periosteum is pushed away from the original cortex by the new

bone, the deeper layers of bone adjacent to the cortex may undergo lamellar reconstruction, but they never become as compact as the original cortex.

Treatment of hypertrophic osteopathy remains predicated on removal of the underlying primary lesions. Most reports of cases showing relief of symptoms and regression of bone changes were in patients who had received complete surgical resection of causative tumors.

In this case, according to the radiography and pathological findings, hypertrophic osteopathy is diagnosed that not associated with neoplastic or pulmonary disorders. And the cat died due to renal failure.

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Contributors:
Yi-Ting Chen (陳怡庭) MD, Shan-Yin Tsai (蔡善茵), Chee- Yin Chai (蔡志仁) MD, PhD
Department of Pathology, Kaohsiung Medical University Chung-Ho Memorial Hospital, Taiwan (高雄醫學大學附設中和紀念醫院病理科).

Case History

This 33 year-old female has the history of 1) spontaneous pneumothorax of left lung and s/p VATS wedge resection six years ago 2) Hepatitis B carrier and 3) Asthma. However, right adenxal tumor was noted accidentally during GYN OPD. In addition, she denied any symptoms/signs recently. Operation was arranged and one retroperitoneal tumor was noted during operation. Partial oophorectomy and retroperitoneal tumor resection were done.

Case Result

Histopathologic Findings:

The pelvic tumor is composed of oval or plump spindled cells in short fascicles. The cells surround spaces lined by endothelium cells in radial arrangement. The cells show clear to eosinophilic cytoplasm without nuclei pleomorphism. The lymphocytic stroma component is composed of small sized lymphocyte with germinal center formation focally. No obvious mitotic figure or necrosis

Immunohistochemical Findings:

- ✓ Positive: SMA, HMB-45 and Desmin
- ✓ Negative: CD117, CD34, S-100 and Melan-A

Differential Diagnosis:

- ✓ Extragastrointestinal stromal tumor (EGIST)
- ✓ Leiomyoma of deep soft tissue
- ✓ Clear cell sarcoma
- ✓ Paraganglioma
- ✓ Myopericytoma
- ✓ Neoplasm of perivascular epithelioid differentiation (PEComa)

Diagnosis:

Perivascular epithelioid cell tumor, in favor of lymphangiomyomatosis

Discussion:

Perivascular epithelioid cell tumor (PEComa) is a mesenchymal tumors composed of histologically and immunohistochemically distinctive perivascular epithelioid cells (PECs). In 1991, Pea et. al. first reported both angiomyolipoma (AML) and clear cell sugar tumor (CCST) of the lung. Later, in 1992, Bonetti et. al displayed cellular link between AML, CCST and lymphangioleiomyomatosis (LAM), their association with tuberous sclerosis complex (TSC) and “immunoreactive” with melanocytic markers.

Lymphangioleiomyomatosis usually occurs in reproductive aged women with mean ago for 40 years. Most of all, the patients suffer from progressive dyspnea with chylous pleural effusion. Pneumothorax and hemoptysis are also noted sometimes. When the lesion is involved in the lung, haphazard proliferation of smooth muscle cells that surround arterioles, venules and lymphatics and which diffusely thicken alveolar septa may be seen as the primary change. Secondary change, bullae formation, as a result of air trapping by obstructed bronchioles, and hemorrhage and hemosiderin deposition are present.

Microscopically, PEComa is characterized by perivascular location with radical arrangement of epithelioid and spindled cells surrounding the vascular lumen. PECs have clear to lightly eosinophilic cytoplasm and round to oval nuclei with small nucleioli. Striking hyperchromasia, nuclear irregularity or atypia and even elevated mitotic activity and necrosis may be seen in some cases. Immunohistochemically, PEComa is positive for malanocytic markers, including HMB-45, Melan-A, tyrosinase and microphthalmia transcription factor and muscle markers, such as smooth muscle actin, myosin and calponin. Cytokeratin and S-100 protein are usually absent.

Ten years survival rate after diagnosis is approximately 85% to 90%. Criteria of malignant category of PEComa has been demonstrated by Folpe and colleagues et. al. in 2005. When the tumor has two or more features as the followings, malignant PEComa is considered, including size more than 5 cm, infiltrating border, high nuclear grade, high cellularity, mitotic rate$\geqq$1/50 HPF, necrosis and vascular invasion. 71% of malignant PEComa has the aggressive behavior.

Surgery is the mainstay of treatment for primary PEComa with the aim of clear resection margins, even local recurrences and metastases. Recently, hormonal therapy including bilateral oophrectomy, progesterone, Tamoxifen or gonadotropin-releasing hormone agonist has been used. However, lung and heart transplantation are still the final treatment for patients of severe lung function induced by pulmonary PEComa.

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4. Choose a Comparative Pathology meeting (e.g. 51st CSCP)

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中華民國比較病理學會
第一次至第五十一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, palillary carcinoma, metaststic	Human	羅東聖母醫院
95.	Transmissible venereal tumor	Dog	中興大學獸醫學系
96.	Malignant lymphoma, large cell type, diffuse, B-cell phenotype	Human	彰化基督教醫院
97.	Carcinosarcomas	Tiger	台灣養豬科學研究所
98.	Mucinous carcinoma with intraductal carcinoma	Human	省立豐原醫院
99.	Mammary gland adenocarcinoma, type B, with pulmonary metastasis, BALB/cBYJ mouse	Mouse	國家實驗動物繁殖及研中心
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)-postitive 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	Faline	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
281	Tonsil Angiosarcoma	Human	羅東博愛醫院
282	Malignant mixed mullerian tumor	Human	耕莘醫院病理科
283	Renal cell tumor	Rat	中興大學獸醫學系
284	Multiple Myeloma	Human	花蓮慈濟醫院病理科
285	Myopericytoma	Human	新光吳火獅紀念醫院
287	Extramedullary plasmacytoma with amyloidosis	Canine	臺灣大學獸醫學系
288	Metastatic follicular carcinoma	Human	羅東聖母醫院病理科
289	Primitive neuroectodermal tumor (PNET), T-spine.	Human	羅東博愛醫院病理科
292	Hemangioendothelioma of bone	Human	花蓮慈濟醫院病理科
293	Malignant tumor with perivascular epithelioid differentiation, favored malignant PEComa	Human	彰化基督教醫院
297	Mucin-producing cholangiocarcinoma	Human	基隆長庚醫院
300	Cutaneous epitheliotropic lymphoma	Canine	臺灣大學獸醫專業學院
301	Cholangiocarcinoma	Felis Lynx	臺灣大學獸醫專業學院

302	Lymphoma	Canine	臺灣大學獸醫專業學院
303	Solitary fibrous tumor	Human	彰化基督教醫院
304	Multiple sarcoma	Canine	臺灣大學獸醫專業學院
306	Malignant solitary fibrous tumor of pleura	Human	佛教慈濟綜合醫院暨慈濟大學
307	Ectopic thymic carcinoma	Human	彰濱秀傳紀念醫院病理科
308	Medullary carcinoma of the right lobe of thyroid	Human	彰化基督教醫院病理科
309	Thyroid carcinosarcoma with cartilage and osteoid formation	Canine	臺灣大學獸醫專業學院
312	Lymphocytic leukemia/lymphoma	Koala	臺灣大學獸醫專業學院
313	Neuroendocrine carcinoma of liver	Human	佛教慈濟綜合醫院暨慈濟大學
314	Parachordoma	Human	羅東博愛醫院病理科
315	Carcinoma expleomorphic adenoma, submandibular gland	Human	天主教耕莘醫院病理科
316	Melanoma, tongue	Canine	國立臺灣大學獸醫專業學院
317	Renal cell carcinoma, papillary type	Canine	國立臺灣大學獸醫專業學院
323	Metastatic papillary serous cystadenocarcinoma, abdomen	Human	國軍桃園總醫院
324	Malignant gastrointestinal stromal tumor	Human	天主教耕莘醫院
329	Sclerosing stromal tumor	Human	彰化基督教醫院
330	Pheochromocytoma	Human	天主教耕莘醫院
334	Metastatic infiltrating ductal carcinoma, liver	Human	佛教慈濟綜合醫院
335	Adenoid cystic carcinoma, grade II, Rt breast	Human	天主教耕莘醫院
336	Malignant lymphoma, diffuse, large B-cell, right neck	Human	林新醫院
337	Pulmonary carcinoma, multicentric	Dog	國立臺灣大學獸醫專業學院
338	Malignant melanoma, multiple organs metastasis	Rabbit	國立中興大學獸醫學院
340	Mucinous-producing urothelial-type adenocarcinoma of prostate	Human	天主教耕莘醫院

	342	Plexiform fibromyxoma	Human	彰化基督教醫院
	343	Malignant epithelioid trophoblastic tumor	Human	佛教慈濟綜合醫院
	344	Epithelioid sarcoma	Human	林新醫院
	346	Transmissible venereal tumor	Dog	國立臺灣大學獸醫專業學院
	347	Ewing's sarcoma (PNET/ES tumor)	Human	天主教耕莘醫院病理科
	348	Malignant peripheral nerve sheath tumor, epithelioid type	Human	林新醫院病理科
	349	Low grade fibromyxoid sarcoma	Human	高醫大附設中和紀念醫院病理科
	351	Orbital embryonal rhabdomyosarcoma	Dog	Gifu University, Japan (岐阜大学)
	354	Granular cell tumor	Dog	國立臺灣大學獸醫專業學院
	356	Malignant neoplasm of unknown origin, cerebrum	Dog	國立臺灣大學獸醫專業學院
	357	Small cell Carcinoma, Urinary bladder	Human	天主教耕莘醫院病理科
	364	Perivascular epithelioid cell tumor, in favor of lymphangiomyomatosi	Human	高醫大附設中和紀念醫院病理科
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 cavitary tuberculosis, lung.	Human	羅東聖母醫院
	54.	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	Human	林口長庚紀念醫院
	58.	Tuberculous enteritis with perforation	Human	佛教慈濟綜合醫院

61.	Spirochetosis	Goose	國立嘉義農專獸醫科
63.	Proliferative enteritis (*Lawsonia intracellularis* infection)	Porcine	屏東縣家畜疾病防治所
68.	Liver abscess (Klebsillae pneumoniae)	Human	台北醫學院
77.	1. Xanthogranulomatous inflammation with nephrolithiasis, kidney, right. 2. Ureteral stone, right.	Human	羅東聖母醫院
79.	Emphysematous pyelonephritis	Human	彰化基督教醫院
89.	1. Severe visceral gout due to kidney damaged 2. Infectious serositis	Goose	中興大學獸醫學系
108.	Listeric encephalitis	Lamb	屏東縣家畜疾病防治所
113.	Tuberculous meningitis	Human	羅東聖母醫院
134.	Swine salmonellosis with meningitis	Swine	中興大學獸醫學系
135.	Meningoencephalitis, fibrinopurulent and lymphocytic, diffuse, subacute, moderate, cerebrum, cerebellum and brain stem, caused by Streptococcus spp. infection	Swine	國家實驗動物繁殖及研究中心
140	Coliform septicemia of newborn calf	Calf	屏東縣家畜疾病防治所
161	Porcine polyserositis and arthritis（Glasser's disease）	Pig	中興大學獸醫學院
162	Mycotic aneurysm of jejunal artery secondary to infective endocarditis	Human	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
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
290	*Staphylococcus* spp. infection	Formosa Macaque	中興大學獸醫病理學研究所
291	Leptospirosis	Dog	台灣大學獸醫學系
296	Leptospirosis	Human	花蓮慈濟醫院
305	Cryptococcus and Tuberculosis	Human	彰濱秀傳紀念醫院
319	Placentitis, Coxiella burnetii	Goat	台灣動物科技研究所
321	Pneumonia, Buirkholderia pseudomallei	Goat	屏東縣家畜疾病防治所
339	Mycoplasmosis	Rat	國家實驗動物中心

	352	Chromobacterium violaceum Septicemia	Gibbon	Bogor Agricultural University, Indonesia
	353	Salmonellosis	Pig	國立中興大學獸醫學院
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.	Pseduorabies	Piglet	國立屏東科技大學
	112.	Avian encephalomyelitis	Chicken	國立中興大學
	128.	Contagious pustular dermatitis	Goat	屏東縣&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 ll co-infection	Canine	國家實驗動物繁殖及研究中心
	223	1. Skin, mucocutaneous junction (lip): Cheilitis, subacute, diffuse, sever, with epidermal pustules, ballooning degeneration, proliferation, and eosinophilic intracytoplasmic inclusion bodies, Saanen goat. 2. Haired skin: Dermatitis, proliferative, lymphoplasmacytic, subacute, diffuse, sever, with marked epidermal pustules, ballooning degeneration, acanthosis, hyperkeratosis, and eosinophilic intracytoplasmic inclusion bodies.	Goat	台灣動物科技研究所
	238	Hydranencephaly	Cattle	國立屏東科技大學獸醫學系
	248	Porcine Cytomegalovirus (PCMV) 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 *Salmonella typhimurium*.	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
	265	Bronchointerstitial pneumonia (PCV II infection)	Swine	台灣大學獸醫學系
	295	Feline infectious peritonitis (FIP)	Cat	中興大學獸醫病理所
	362	Canine distemper virus infection combined pulmonary dirofilariasis	Dog	國家實驗研究院
黴菌	23.	Chromomycosis	Human	台北病理中心
	47.	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	Human	三軍總醫院

	48.	Adiaspiromycosis	Wild rodents	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
	298	Systemic Candidiasis	Tortoise	中興大學獸醫學院
	318	Alfatoxicosis in dogs	Canine	國立臺灣大學獸醫專業學院
	322	Allergic fungal sinusitis	Human	羅東博愛醫院
	326	Meningoencephalitis, Aspergillus flavus	Cat	國立臺灣大學獸醫專業學院
	331	Histoplasmosis	Human	花蓮慈濟醫院病理科
	332	Pulmonary Blastomycosis	Rat	中興大學獸醫學院
	355	Encephalitozoonosis	Rabbit	國立中興大學獸醫學院
	356	Eosinophilic granuloma with fungal infection, Skin	Cat	國立臺灣大學獸醫專業學院
寄生蟲	14.	Dirofilariasis	Dog	台灣省家畜衛生試驗所
	15.	Pulmonary dirofilariasis	Human	台北榮民總醫院
	20.	Sparganosis	Human	台北榮民總醫院
	46.	Feline dirofilariasis	Cat	美國紐約動物醫學中心
	49.	Echinococcosis	Human	台北榮民總醫院
	60.	Intestinal capillariasis	Human	台北馬偕醫院
	64.	1.Adenocarcinoma of sigmoid colon 2.Old schistosomiasis of rectum	Human	省立新竹醫院
	66.	Echinococcosis	Chapman's zebra	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	Dog	臺灣養豬科學研究所

		migration		
	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, Angiostongylus cantonensis	Norway Rat	行政院農業委員會農業藥物毒物試驗所
	202	Colnorchiasis	Human	高雄醫學院附設醫院
	203	Trichuriasis	Human	彰化基督教醫院
	204	Psoroptes cuniculi infection (Ear mite)	Rabbit	農業藥物毒物試驗所
	205	Pulmonary dirofilariasis	Human	和信治癌中心醫院
	206	Capillaries philippinesis	Human	和信治癌中心醫院
	207	Adenocarcinoma with schistosomiasis	Human	花蓮佛教慈濟綜合醫院
	286	Etiology- consistent with *Spironucleus* (*Hexamita*) *muris*	Rat	國家實驗動物繁殖及研究中心
	327	Dermatitis, mange infestation	Serow	中興大學獸醫學院
	328	Trichosomoides crassicauda, urinary bladder	Rat	國家實驗動物中心
	362	Canine distemper virus infection combined pulmonary dirofilariasis	Dog	國家實驗研究院
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
	320	Cutaneous leishmaniasis	Human	佛教慈濟綜合醫院
	325	Myocarditis/encephalitis, Toxoplasma gondii	Wallaby	國立臺灣大學獸醫專業學院
立克次體	229	Necrotizing inflammation due to scrub typhus	Human	佛教慈濟醫院病理科
	251	Scrub typhus with diffuse alveolar	Human	佛教慈濟醫院病理科

		damage in bilateral lungs.		
皮膚	216	Cytophagic histiocytic panniculitis with terminal hemophagocytic syndrome	Human	佛教慈濟綜合醫院病理科
	359	Eosinophilic granuloma with fungal infection, Skin	Cat	國立臺灣大學獸醫專業學院
	360	Septa panniculitis with lymphocytic vasculitis	Human	慈濟綜合醫院暨慈濟大學
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 lymphandenitis)	Lymphandenitis	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
294	Arteriovenous malformation of the left hindlimb	Dog	台灣大學獸醫學系
299	Polioencephalomalacia	Goat kid	屏東家畜疾病防治所
310	Hyperplastic goiter	Piglet	屏東家畜疾病防治所
311	Melamine and cyanuric acid contaminated pet food induced nephrotoxicity	Rat	中興大學獸醫學病理學研究所
318	Alfatoxicosis	Canine	國立臺灣大學獸醫專業學院
333	Lordosis, C6 to C11	Penguin	國立臺灣大學獸醫專業學院
341	Pulmonary placental transmogrification	Human	羅東博愛醫院
345	Acute carbofuran intoxication	Jacana	國立中興大學獸醫學院
350	Malakoplakia, liver	Human	慈濟綜合醫院暨慈濟大學
351	Eosionphilic granuloma, Right suboccipital epidural mass	Human	羅東博愛醫院病理科
359	Eosinophilic granuloma with fungal infection, Skin	Cat	國立臺灣大學獸醫專業學院
360	Septa panniculitis with lymphocytic vasculitis	Human	慈濟綜合醫院暨慈濟大學
361	Hepatotoxicity of SMA-AgNPs	Mouse	國立中興大學獸醫病理生物學研究所

	363	Hypertrophy osteopathy	Cat	國立臺灣大學獸醫專業學院

會 員 資 料 更 新 服 務

各位會員：

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

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

----------中華民國比較病理學會----------

會員資料更改卡

姓　　名：_____　　會員類別：☐一般會員
☐學生會員
☐贊助會員

最高學歷：_____

服務單位：_____職　　稱：_____

永久地址：_____

通訊地址：_____

電　　話：_____傳　　真：_____

E-Mail Address：_____

中華民國比較病理學會
誠摯邀請您加入

入　會　辦　法

一、本會會員申請資格為：

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

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

（三）贊助會員：贊助本會工作之團體或個人。

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

二、會員：

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

（二）常年會費：一般會員新台幣伍佰元，學生會員一佰元。

【註：學生會員身份變更為一般會員時，只需繳交一般會員之常年會費】

三、請填妥入會申請表郵寄或傳真方式寄回中華民國比較病理學會秘書處收。

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

電話：02-33663858、傳真 02-23682423。

中華民國比較病理學會入會申請及會員卡

會籍電腦編號 _____

姓名	中文		性別	男 ☐ 女 ☐	出生	民國 年 月 日 身分證字號	出生地	省 縣/市
	英文		會員身份: ☐一般 ☐學生 ☐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)

	機關名稱	職務	起	止
主要經歷			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：

電子郵遞(E-mail)地址：

茲 贊 同

貴會宗旨擬加入爲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