

# 中華民國比較病理學會第七次比較病理研討會 (呼吸道感染專題) 議程表

時間：中華民國八十五年十一月十日（星期日）08:00-17:00

地點：國立屏東技術學院獸醫系館（屏東縣內埔鄉學府路一號）  
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主辦單位：中華民國比較病理學會  
國立屏東技術學院獸醫學系  
屏東縣家畜疾病防治所

08:00- 08:40 Registration

08:40- 09:00 Welcome & Opening Remark（黃文哲/郭謨/洪信雄）

Section【1】 Moderator: 蔡信雄教授

09:00- 09:30 Case 46 SK Liu, DVM, PhD (劉錫光教授)

09:30- 10:00 Case 47 Ann Chen, MD, DMS (陳安醫師)

10:00- 10:30 Coffee Break

Section【2】 Moderator: 劉錫光教授

10:30- 11:00 Case 48 Victor Fei Pang, DVM, PhD (龐飛教授)

11:00- 11:30 Case 49 TY Chou, MD, PhD (周德盈博士)

11:30- 12:00 Case 50 SH Lee, DVM, MS (李淑慧研究員)

12:00- 13:30 Luncheon 中華民國比較病理學會第三次理監事聯席會議

Section【3】 Moderator: 方中民教授

13:30- 14:00 Case 51 Hsiaoling Chen, MD (陳小玲醫師)

14:00- 14:30 Case 52 MT Tsai, DVM, MS (蔡睦宗研究員)

14:30- 15:00 Case 53 JP Juch, MD, MS (祝志平醫師)

15:00- 15:30 Coffee Break

Section【4】 Moderator: 何逸僊教授

15:30- 16:00 Case 54 YS Ho, MD, MS (何逸僊醫師)

16:00- 16:30 Case 55 TC Chang, DVM, MS (張聰洲老師)

16:30- 17:00 Discussion

# Comparative Pathology Case 46

**Contributor:** Si-Kwang Liu (劉錫光), DVM, PhD

Consultant, Pig Research Institute Taiwan

Senior Pathologist, The Animal Medical Center

**Clinical History:** This is a 7-year-old, castrated male, domestic shorthair cat with sudden onset of vomiting, choking, and then acute dyspnea. When the cat was examined at The Animal Medical Center, it had cyanosis, open-mouth breathing, salivation, and very harsh lung sounds. Fluid was removed (100 ml) from the right thoracic cavity. The cat was put in an oxygen cage; the cat had respiratory arrest and then cardiac arrest after echocardiographic examination.

**Diagnosis:**

1. Endoarteritis, eosinophilic, granulomatous, pulmonary arteries
2. Granulomatous pneumonitis
3. Pulmonary fibrosis, congestion, and edema

**Gross Findings:** The right side of the heart was dilated and a male heartworm (*Dirofilaria immitis*) was found in the right ventricle. The lung lobes had marble patterns, were consolidated, and consisted of dark purple and gray areas mixed with a few whitish, emphysematous areas throughout all the lung lobes.

**Histopathologic Findings:** Examination of pulmonary arteries revealed endothelial roughenings; some arteries had fibrous endothelial proliferation and formation of villous projections on the endothelial surface. Other pulmonary arteries had fibrous endothelial proliferation and thrombi. A heavy infiltration of lymphocytes, plasma cells, and eosinophils was seen in the intima and proliferation of endothelial cells. Examination of myocytes revealed coagulation, granulation, and vacuolization of the myoplasm in the tunica media. A heavy infiltration of lymphocytes, plasma cells, and a few eosinophils was observed in the arterioles. Lumen of small arteries and arterioles were

occluded by endothelial proliferation and hyperplasia of subintimal myofibrocytes, hyperplasia of myocytes with myocytolysis in the tunica media, and sprinkling of lymphocytes in the adventitia. The pulmonary parenchyma was fibrotic, with hyperplasia of smooth muscle cells and peribronchial accinar and lymphoid hyperplasia. Infiltration of eosinophils was observed in the parenchyma.

**Discussion:** Eosinophilic granulomatous pneumonitis and active endoarteritis of the pulmonary arteries are typical pulmonary lesions found in cats with dirofilariasis or with *Aelurostrongylus abstrusus* infection. These lesions are identical to those found in pulmonary arteries of dogs with the same disease. Since the cat is not the final host for heart-worm infection, the parasite might be able to reach the hearts; but it is usually infertile, and pulmonary lesions in cats are usually more severe than those found in dogs with the disease. Arterial lesions are commonly found in the lungs of dogs with dirofilariasis, and the worms are found in the right ventricle; but the lesions have also been observed in the aorta and iliac, femoral, popliteal, renal, and testicular arteries of a dog with massive heart-worm infection, and the worms are found in the left and right sides of the heart and arterial system.

Characteristic, progressive, intimal proliferative lesions which lead to obstruction of the pulmonary arteries have been commonly observed in dogs with dirofilariasis on histologic examination of the lungs. Obstruction, pruning, and abnormal tapering of the pulmonary arteries have been demonstrated by angiocardigraphy and postmortem arteriography of the lungs of affected dogs. Anastomoses between bronchial and pulmonary arteries normally does not occur in the lungs of cats and dogs, but pulmonary and bronchial arterial anastomoses have been commonly demonstrated in the lungs of dogs with chronic dirofilariasis; the anastomoses usually occur at the sites of obstruction of the affected pulmonary arteries. The bronchial arterial circulation through the broken vasa vasorum and communication with the pulmonary arteries distal to the obstruction have been demonstrated by histologic technique and by postmortem bronchial arteriography. Pulmonary arterial circulation distal to the obstruction is supplied by bronchial arterial blood in dogs with chronic dirofilariasis.

The cause of the pulmonary arterial lesions is unclear; however, it is possible that antigenicity of the metabolic byproducts from the parasite can induce an allergic reaction in the pulmonary arteries.

**References:**

1. Adcock JO: Pulmonary arterial lesion in canine dirofilariasis. Am J Vet Res 22:655-662, 1957.
2. Liu SK, Das KM, Tashjian RJ: Adult *Dirofilaria immitis* in the arterial system of a dog. J Am Vet Med Assoc 148:1501-1507, 1966.
3. Liu SK, Yarns DA, Tashjian RJ: Postmortem pulmonary arteriography in canine dirofilariasis. Am J Vet Res 30:319-329, 1969.
4. Liu SK, Yarns DA, et al.: Pulmonary collateral circulation in canine dirofilariasis. Am J Vet Res 30:1723-1735, 1969.
5. Tashjian RJ, Liu SK, Yarns DA, et al. : Angiocardiography in canine heartworm disease. Am J Vet Res 31:415-436, 1970.
6. Liu SK, Hsu F, Lee RCT: An Atlas of Cardiovascular Pathology. Pig Research Institute Taiwan, ROC 1989, p. 209-215.
7. Hieronymi E: Zur Entwicklung von *Aelurostrongylus abstrusus* in der Katzenlunge. Tierarztl Umsch 8:230-233, 1953.

## Comparative Pathology Case 47

**Contributors:** Ann Chen, MD, DMS (陳安), Bao-Chiien Chang, MD (張保欽), Hueng-Chin Han, MD, PhD (韓鴻志), Wei-Hwa Lee, MD, PhD (李偉華)  
Department of Pathology, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan, R.O.C.

**Clinical History:** This 36-year-old man was admitted due to hypertension for several years. CT scan showed multiple tumor masses in the right suprarenal region, liver, and lungs. The patient received thoractomy with lobectomy of the right lower lobe, removal of suprarenal mass and wedge resection of hepatic mass.

**Gross Findings:** On gross examination, the lung tissue measured 12 x 9 x 5 cm with two tumor lesions, 3 x 2 x 2 and 3 x 1.8 x 2 cm, on the lower pole and apical region, respectively. The adrenal mass measured 3 cm in diameter and the liver mass 4.9 cm in diameter.

**Histopathological Findings:** Light microscopically, the sections appeared as follows:

- (1) Lung: metastatic carcinoma associated with cryptococcal infection.
- (2) Liver: metastatic carcinoma.
- (3) Adrenal gland, right: carcinoma (primary).

**Histochemical Results:** The fungi localized in the lung tissue examined were positive for GMS, mucin, and PAS staining.

**Immunohistochemical Results:** The tumor cells were positive for neuron-specific enolase focally, but negative for alpha-fetal protein, cytokeratin, S-100, or chromogranin-A.

## Comparative Pathology Case 48

**Contributors:** Victor Fei Pang<sup>1\*</sup> (龐飛), DVM, PhD; Shih-Hsuan Hsiao<sup>1</sup> (蕭世烜), DVM, MS; Ming-Tang Chiu<sup>1</sup> (邱明堂), DVM; Chun-Teng Tsai<sup>1</sup> (蔡群騰), DVM; Chian-Ren Jeng<sup>1</sup> (鄭謙仁), DVM, PhD; Hsuan-Wien Chen<sup>2</sup> (陳宣汶), MS; Pei-Fen Lee<sup>2</sup> (李培芬), PhD; Chung-Tiang Liang<sup>3</sup> (梁鍾鼎), DVM, MS; Chou-Chu Hong<sup>3</sup> (洪昭竹), DVM, PhD.

<sup>1</sup>Department of Veterinary Medicine, National Taiwan University

<sup>2</sup>Department of Zoology, National Taiwan University

<sup>3</sup>National Laboratory Animal Breeding and Research Center

**Clinical History:** This was an incidental finding in a disease survey of wild rodents captured in mountainous regions of northern Taiwan. Sample collection areas included Kuan-Wu, An-Ma, Tai-Ping mountain, Yuan-Yang lake, and La-La mountain at heights ranging from 1650 to 2550 meters above sea level. A total of 146 animals, including 110 *Apodemus semotus*, 21 *Niviventer culturatus*, 9 *Eothenomys melanogaster*, 4 *Tamias swinhoei formosanus*, 1 *Microtus kikuchii*, and 1 *Niviventer coninga*, were necropsied and examined histopathologically.

**Diagnosis:** Adiaspiromycosis

**Gross Findings:** Seventeen out of 110 (17/110) *Apodemus semotus*, 1/21 *Niviventer culturatus*, and 1/1 *Niviventer coninga* contained scattered to multiple, pinpoint, clear to translucent, spherical structures protruding from the surface of the lungs. The distribution of these spherules was random but was predominant in the front lung lobes.

**Histopathological Findings:** Microscopically, 35/110 *Apodemus semotus*, 4/21 *Niviventer culturatus*, and 1/1 *Niviventer coninga* contained various numbers of 50-300µm, round, thick-walled, spherical bodies (adiaspores) randomly distributed in the lung parenchyma. The wall was composed of 3 indistinct layers that had different affinity to eosin and hematoxylin. The outmost layer was hyalinized and dark red-pink; the middle layer was amorphous and dark pink; the inner layer was

homogenous but light pink with slight basophilia. The interiors of the adiaspores were basophilic and granular in appearance with evident marginal alignment. The tissue response was generally limited. There was only a minimal infiltration of lymphocytes, macrophages, and plasma cells along with some fibroblasts around the adiaspores. Typical granuloma or granulomatous inflammation was only occasionally noted.

**Histochemistry Results:** Periodic acid-Schiff staining stained the outmost layer of the adiaspore wall bright red-pink and the middle and inner layers equally pink but indistinctly; the interior was stained light pink and slightly granular. Gomori methenamine-silver nitrate staining stained the entire wall equally dark black and the interior light black and granular, but there was a thin unstained zone present between the wall and interior. Gram's staining revealed a limiting membrane present in the wall that demarcated the adiaspores from surrounding tissue; it stained the middle and inner layer of the wall leaving the outmost layer and the interior unstained.

**Discussion:** Adiaspiromycosis is a worldwide, noninfectious, nonarthropod transmitted pulmonary fungal infection of man and animals, most commonly rodents, caused by the species *Emmonsia parva*. There are two varieties within the species, *E. parva* var. *parva* and *E. parva* var. *crecens*. Although both varieties can cause infection in animals, humans only become an accidental host by inhaling dust-borne spores (conidia) of the saprophytic soil fungus, *E. parva* var. *crecens*.

*Emmonsia parva* var. *parva* and *E. parva* var. *crecens*. were renamed by Carmichael as *Chrysosporium parva* var. *parva* and *C. parva* var. *crecens* in 1962; however, these were refuted by von Arx in 1973 on the basis that, unlike the species of *Chrysosporium*, *Emmonsia* produces blastic conidia and, at an elevated temperature, the conidia become adiaspores. Cultures of both varieties closely resemble each other at 25°C. However, the size of the adiaspores produced either in vivo or in vitro by *E. parva* var. *crecens* at 37°C is 5 to 10 times larger than those of *E. parva* var. *parva*.

The mycosis is unique in that the inhaled fungal spores only enlarge and never germinate or reproduce in the host tissue. The name of the mycosis stems from the word adiaspore, which is derived from Greek roots signifying enlargement without multiplication. Each inhaled conidium grows at the site of implantation in the lung of the host and becomes an enormous, thick-walled adiaspore. The

adidaspores eventually die and may become calcified. The severity of the disease depends on the number of spores inhaled. In limited inoculum, the disease remains localized, whereas in heavy inocula the fungus may involve the entire lungs and presents as a multifocal discrete nodular to confluent granulomatous infiltrate. In this disseminated form, patients usually develop cough, dyspnea on exertion, and low-grade fever mimicking other systemic fungal infections and tuberculosis.

It is difficult to unmask the fungus because it is not easily cultured nor is there a reliable serologic test; therefore, a biopsy is required. The pathologist must recognize the large (ranging in size from 50 to 500 m), round, Gomori methenamine-silver nitrate, periodic acid-Schiff, and Gram s stains positive adiaspores with a trilaminar wall. The adiaspores can be surrounded by either limited suppuration, epithelioid granulomas with or without necrosis, or thin layers of concentric, hyalinized fibrosis. In the latter chronic stage, the organism may collapse, forming a variety of sizes and shapes thereby resembling other fungi, helminths, mineral particles, or inhaled pollen grains.

Clinically, the infection most commonly regresses spontaneously, but may persist, or rarely progress, requiring surgical intervention with limited resection to attain cure.

#### **Diagnostic Criteria:**

1. Large (50-500 m), spherical adiaspores with a thick trilaminar wall
2. GMS, PAS, and Gram s staining positive
3. Lack of significant host responses
4. An incidental finding

#### **References:**

1. Albassam, M. A., Bhatnagar, R., Lillie, L. E., and Roy, L. 1986. Adiaspiromycosis in striped skunks in Alberta, Canada. J. Wildlife Dis. 22:13-18.
2. Arx, J. A. von. 1973. Further observations on *Sporotrichum* and some similar fungi. Persoonia 7:127-130.
3. Carmichael, J. W. 1962. *Chrysosporium* and some other aleurosporic *Hyphomycetes*. Can. J. Bot. 40:1137-1173.
4. England, D. M. and Hochholzer, L. 1993. Adiaspiromycosis: an unusual fungal infection of the lung. Report of 11 cases. Am. J. Surg. Pathol. 17:876-886.



5. Kodousek, R., Vortel, V., Fingerland, A., Vojtek, V., Sery, Z., Hajek, V., and Kucera K. 1972. Pulmonary adiasporiomycosis in man caused by *Emmonsia crescens*: report of a unique case. Am. J. Clin. Pathol. 56:394-399.
6. Koller, L. D. and Helfer, D. H. 1978. Adiaspiromycosis in the lungs of a goat. J. Am. Vet. Med. Assoc. 173:80-81.
7. Koller, L. D., Patton, N. M., and Whitsett, D. K. 1976. Adiaspiromycosis in the lungs of a dog. J. Am. Vet. Med. Assoc. 169:1316-1317.
8. Kwon-Chung, K. J. and Bennet, J. E. 1992. Infections due to miscellaneous molds. In: Medical Mycology. pp. 733-767. Lea & Febiger, Malvern, PA, USA.
9. Peres, L. C., Figueiredo, F., Peinado, M., and Soares, F. A. 1992. Fulminant disseminated pulmonary adiaspiromycosis in humans. Am. J. Trop. Med. Hyg. 46:146-150.
10. Taniyama, H., Furuoka, H., Matsui, T., and Ono, T. 1985. Two cases of adiaspiromycosis in the Japanese pika. Japn. J. Vet. Sci. 47:139-142.

## Comparative Pathology Case 49

**Contributors:** T.Y. Chou (周德盈), MD, Ph D; SM Liu, MD, MS, H Chiang (江宏), MD

Department of pathology

National Yany-Ming U.I. Veterans General Hospital-Taipei

**Clinical History:** One week before admission, this 68-year-old male went to a local hospital because of a "common cold." The chest X-ray revealed a nodular shadow in right upper lobe of lung. He had smoked half pack per day for more than 20 years but quit 25 years ago. He complained of cough with sticky, greenish sputum. At admission, physical examination showed no remarkable change. Under the impression of lung cancer, he received exploratory thoracotomy and lobectomy of right upper lobe. The surgical specimen consisted of a lobe of lung tissue, 13 x 8 x 2 cm, gray and spongy, containing a cystic space 2.5 cm in diameter. Representative parts of the cyst wall were submitted for microscopic pathology.

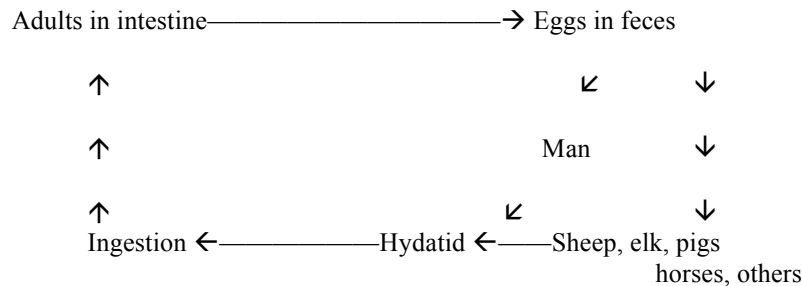
**Diagnosis:** Lung, RUL, Lobectomy--Echinococcosis

**Gross Findings:** The cyst, already opened by surgeon, had a fibrous capsule with focal consolidation of the adjacent lung tissue.

**Histopathological Findings:** Three layers of the cyst wall were identified: the external fibrous layer, the middle laminated layer, and the internal germinal layer. Protoscolices and residual blood capsules were found along with the cyst wall. Marked granulomatous inflammation with histiocytes, giant cells, and infiltration of eosinophils and neutrophils was noted surrounding the cyst and around fragments of the laminated layer of the cyst wall, where protoscolices were also seen.

**Discussion:** The tapeworms Echinococcus have larval stages known as hydatid cysts, and the disease they produce is called echinococcosis or hydatidosis. At least three species of Echinococcus have been recognized to have man as their intermediate host: *E. granulosus* (unilocular hydatid disease), *E.*

*multilocularis* (alveolar h.d.), and *E. Vogeli* (polycystic h.d.). Echinococcus has worldwide distribution, but is more prevalent in the temperate and arctic regions. The life cycle of Echinococcus is illustrated here:



The onchospheres of *E. granulosus* usually enter the venules of the intestinal mucosa and lodge in the liver (75% of cases) or travel through the lymphatics (or venous system) to lungs (9%), muscles (5%), spleen (2%), brain (1.5%), bone (1%), and heart (< 1%). Approximately 90% of pulmonary hydatid cysts are solitary and the remaining occur concomitantly with a liver cyst.

The diagnosis of hydatid cyst is made primarily on clinical grounds, based on the presentation and the history of exposure. X-rays and other imaging techniques will help demonstrate the lesion. Cassoni intradermal test is positive in 65-85% of cases. Serological tests are not reliable. The definitive diagnosis will be the gross and histological examination.

#### **Diagnostic Criteria:**

1. Unilocular, spherical, or subspherical cyst filled with clear fluid.
2. Three layers of the cyst wall: fibrous layer (from host), laminated membrane, and germinal membrane.
3. PAS-positive laminated layer, approximately 10 times the thickness of the germinal layer.
4. Protoscolices with rostellum and hooklets.

#### **References:**

1. Y Gutierrez. 1990. Echinococcus-Hydatid Disease or Echinococcosis. in Diagnostic Pathology of Parasitic Infections with Clinical Correlations. pp. 460-80 Lea & Febiger, Philadelphia.

2. PJ Cant. 1995. Migration of *Echinococcus granulosus* via lymphatics. *Lancet* 345:393-4.

## Comparative Pathology Case 50

**Contributor:** Shu-Hwae Lee (李淑慧), DVM, MS

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**Clinical History:** In a breeder to grown-up farm, one herd of 350 weaned piglets (about 30 days of age) gradually developed depression, anorexia dyspnea, swelling of legs, and fever. In a few piglets the fever was as high as 41°C. After these piglets had been treated with antibiotics but without improvement, eight pigs were submitted to Taiwan Animal Health Research Institute for diagnosis. The epizootic lasted for 3 weeks, but no clinical signs were found in breeder or adult pigs. The mortality was 13% (47/500) in the herd.

**Diagnosis:** Porcine Cytomegalovirus Infection

**Gross Findings:** In two of the piglets, the nasal cavity was filled with gray viscid mucus, and the nasal mucosa was moderately congested. Except for this, all the lesions described in the following were present in all 8 piglets examined. There was transparent fluid, about 400 to 500 ml in volume, deposited in the pleural cavity. The lungs were swollen, and their surface covered by a thick, whitish membrane. Lobular septas were clearly separated all over the lung due to transparent fluid depositions. In both apical and cardiac lobes there were areas of focal hepatization. In the heart, pericardial fluid was increased to 30 to 50 ml in volume. The systemic lymph nodes were mildly swollen. The liver had a few miliary, whitish gray spots on the surface and the parenchyma.

**Histopathological Findings:** The turbinate mucosa of the nasal cavity had congestion and focal necrosis with mild lymphocyte infiltrations. Underneath, the tubuloalveolar gland epithelia were enlarged; some of the epithelia became 10 times their normal size. The epithelia had vacuolated pale staining cytoplasm and large basophilic intranuclear inclusions. The inclusions were variable in shape and size. Slight fibroblast proliferations were present in the surrounding tissue. In the lung, dilated lobular septas with protein exudates in the septas and the surrounding alveoli were noted. Focal

depositions of epithelial cells and macrophages in the alveoli were noted. The liver had mild fatty changes, focal necrosis with lymphocyte infiltrations, and fibrocyte infiltrations in the cortex. The lymph nodes were edematous. There was hemosiderin deposition, and lymphocytic depletions and neutrophil infiltrations were found in the follicles. The brain had multiple focal microglial cell proliferations and mild lymphocytic perivascular accumulations.

**Electron Microscopic Findings:** Mixture of turbinate mucosa and the grayish mucoid mucus in the surface were emulsified to prepare 10 X suspension. The suspension was fixed in 2.5% glutaraldehyde solution and centrifuged. The pellet was stained with 2% phosphotungstic acid. Herpesviral nucleocapsids were found under TEM examination.

**Discussion:** In this case, the demonstration of intranuclear inclusions in the tubuloalveolar gland of turbinate mucosa was the characteristic lesion induced by cytomegalovirus infection in pig. Mild inflammatory reactions in the brain and other organs were probably associated with the infection, although no characteristic inclusions were found. Except for histopathologic examination, the emulsified tissues were inoculated into PK-15, RK-13, and ESK cell lines, but no virus was recovered. Similar results have been reported in other countries.

**Diagnostic Criteria:**

1. Clinical history, demonstration of characteristic lesions, and intranuclear viral inclusion bodies.
2. Virus isolation-difficult.
3. Characteristic features of Herpesvirus by TEM examination.

**References:**

1. Duncan, J.R. et al., 1965. Electron microscopy of cytomegalic inclusion disease of swine (inclusion body rhinitis). Am. J. Vet. Res. 26:939-947.
2. Edington, N. 1992. Cytomegalovirus. Disease of swine. 7th ed. Ames: Iowa State University Press. pp.250-256.
3. Kawamura, H. et al., 1992. Replication of porcine cytomegalovirus in the 19-PFT cell line. J. Vet.

- Med. Sci. 54(6):1209-11.
4. Tsjima, T. et al., 1994. Detection of the antibodies against porcine cytomegalovirus from whole blood collected on the blood sampling paper. J. Vet. Med. Sci. 56(1):189-90.
  5. Yang, S. Y. et al., 1988. The first case report of porcine cytomegalovirus infection in Taiwan. J. Chinese Soc. Vet. Sci. 4:57-63.
  6. Yoshikawa T. et al., 1977. Pathology of cytomegalic inclusion body disease in swine. Jap. J. Vet. Sci. 39:47-58.

# Comparative Pathology Case 51

**Contributor:** Hsiaoling Chen (陳小玲), MD

Taipei Institute of Pathology

**Clinical History:** A 24-year-old male, suffering from exertional dyspnea for 10 days, was admitted to the hospital. The CT scan of chest disclosed diffuse ground-glass infiltrate in both lung fields. Open lung biopsy was performed. The laboratory data revealed white count: 18,300/cumm, CD4 cell: 21/cumm, CD4/CD8: 0.1, anti-HIV: (+), and anti-HIV1: (+)(western-blot). The patient did not response well to the therapy for AIDS. He died two and a half months later.

**Diagnosis:** *Pneumocystis carinii* pneumonia (PCP).

**Histopathological Findings:** The lung parenchyma shows that alveoli are filled with foamy proteinaceous edema fluid. The alveolar wall appears normal.

**Histochemical Studies:** The Gomori methenamine silver stain reveals discrete clusters of ovoid to round, collapsed cup-like organisms in the alveolar spaces. The clusters of cysts, which do not stain for H & E, impart foamy appearance to alveolar edema.

**Electron Microscopic Findings:** There are crescent and ovoid cysts in the alveolar exudate.

**Discussion:** The *Pneumocystis carinii* was first described in 1909-1910 in rats by Chagas and Carinii and was believed to be the part of the life cycle of trypanosoma (schizotrypanum). In 1912, Delanoes found the organisms in rats and guinea pigs and named it *Pneumocystis carinii*. Vanek and Jirovec in 1952 identified *P. carinii* as the agent in the outbreaks of "plasma cell" pneumonia in malnourished neonates and children after World War II. In 1960s, the use of immunosuppressive therapy became common for many diseases, rendering many patients at risk for PCP. In 1980s, PCP emerged as the most common opportunistic infection in patients with AIDS.



*P. carinii* infect the human beings as well as other animal including rats, mice, shrews, ferrets, thoroughbred foals, rabbits and dogs. Under the molecular techniques, it has been proven that different species of *P. carinii* infect different animal hosts. Genetic variation within rat-derived *P. carinii* has been reported, but strain stability in the human hosts is also seen.

*P. carinii* produces no disease in normal individuals but causes severe pneumonia in immunocompromised hosts and in children with protein-calorie malnutrition. In general, most patients at risk for PCP have a defect in the T cell arm of immunity, whether from a disease such as leukemia or HIV infection ( $CD4 < 200/\text{cumm}$ ) or from immunosuppressive therapy, especially with corticosteroids.

The inhaled trophozoites tend to parasitize type I pneumocytes, leading to pulmonary edema. Multiplication of *P. carinii* appears to be extracellular and is within the alveolar spaces.

The onset is usually insidious, but there may be sudden onset or exacerbation in patients treated with corticosteroids. The standard presentation of PCP are fever, dry cough and progressive dyspnea. The chest radiograph varies considerably from normal to mild involvement with interstitial or reticulonodular infiltrates through both lungs, to more severe disease with patches of alveolar consolidation.

The pathological manifestation of early (incipient) PCP is characterized by individual cysts and trophozoites can be seen attached to the alveolar septae. As the infection proceeds the alveoli become progressively filled with foamy proteinaceous exudates composed of large numbers of trophozoites with their microtubular extensions, cysts, fibrin and cellular debris. Frequently, there is concurrent infection by opportunistic bacteria, fungi or virus, especially cytomegalovirus, which may overshadows the pathology caused by *P. carinii*. Several atypical manifestations of this infection include: (1) interstitial lung disease; (2) lymphoplasmcytic interstitial infiltrates; (3) nodular and granulomatous PCP; (4) cavitory PCP and (5) extrapulmonary PCP.

*P. carinii* is diagnosed by the demonstration of round, ovoid or collapsed cup-like structures, measuring 4 to 6 microns in diameter with a membrane of variable thickness. Special stains used include Gram-Weigert, toluidine stains, or methenamine silver. Methenamine silver stains demonstrate the cyst wall clearly. The morphology of the trophozoites is best studied with the Giemsa stain.

The various means of diagnosis of PCP are demonstrating *P. carinii* in a sample of induced sputum, a bronchoalveolar lavage sample, a biopsy specimen and polymerase chain reaction (PCR). It

has recently been shown that a PCP severity score based on serum LDH enzyme levels, the alveolar arterial oxygen gradient, and the percentage of neutrophils in bronchoalveolar lavage fluid has very high prognostic significance for survival.

A wide range of antimicrobial agents are effective against *P. carinii* as followings: first choice, Co-trimexazole; second choice, pentamidine, dapsone and trimethoprim, clindamycin and primaquine, atovaquone; and adjuvant therapy: corticosteroids. Anti-PCP prophylaxis is effective, the drug of first choice being oral co-trimoxazole and pentamidine aerosol.

*P. carinii* genomes are not found in autopsy lung where the cause of death is not clinical PCP, by PCR, or normal sputum in general population. Serologic studies indicate 85% of adults have been subclinically infected. Therefore *P. carinii* is a ubiquitous organism in our environment. Each episode of pneumonia represents de novo infection and not reactivation of previous infection.

#### **Diagnostic Criteria:**

1. Histopathology: foamy protein-rich alveolar edema.
2. Special stain: Methenamine silver-positive collapsed cup-like cysts in the alveolar exudate.

#### **References:**

1. Frenkel JK: pneumocystosis In: Binford CH, Connor DH: Pathology of tropical and extraordinary diseases. Washington D.C. Armed Forces Institute of Pathology, 303-307, 1976.
2. Diseases of immunity In: Cortan RS, Kumar V, Robbins SL: Pathologic basis of disease. Philadelphia, W. B. Saunders Co., 228-229, 1994.
3. Infectious diseases In: Cortan RS, Kumar V, Robbins SL: Pathologic basis of disease. Philadelphia, W. B. Saunders Co., 357, 1994.
4. The lung In: Cortan RS, Kumar V, Robbins SL: Pathologic basis of disease. Philadelphia, W. B. Saunders Co., 703, 1994.
5. Saldana MJ, Mones JM: Pulmonary pathology in AIDS: atypical Pneumocystis carinii infection and lymphoid interstitial pneumonia. Thorax 49 (suppl): s46-s55, 1994.
6. Sepkowitz KA: *Pneumocystis carinii* pneumonia in patients without AIDS. Clin Infect Dis 17(suppl 2): s416-s422, 1993.
7. Miller RF, Mitchell DM: *Pneumocystis carinii* pneumonia. Thorax 47: 305-314, 1992.

8. Miller RF, Mitchell DM: *Pneumocystis carinii* pneumonia. Thorax 50: 191-199, 1995.

## Comparative Pathology Case 52

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**Clinical History:** A poultry farm reared about 2300 goslings in Pingtung prefecture. The accumulative morbidity and mortality within one week period were about 50% and 80%, respectively. Clinically, goslings flock at 12 to 14 days of age showed signs of somnolence, inappetence, dyspnea, gasping, respiratory difficulties for about one week. In more severe cases, nervous signs appeared (incoordinated movements, twisting of neck and head, ataxia), along with listlessness, ocular discharge, and corneal opacity. The owner sent dead goslings to our laboratories for pathological diagnosis.

**Diagnosis:** Aspergillosis in goslings

**Gross Findings:** There were yellow or gray, firm nodules (about 1-6 mm in diameter) in the lungs, air sacs, cerebrum, surfaces of kidney and mesentery. Numerous nodules were present in the lungs but few discrete ones in others. A gray-white opacity was noticed in the eye.

**Histopathological Findings:** The lungs showed multiple granulomas consisting of a central necrotic core associated with filamentous mycelia, surrounded by epithelioid cells, giant cells, heterophils and lymphocytes. The lesions were contained within a thin walled fibrous capsules. The air sacs showed marked thickening with caseating necrosis. Cerebral lesions consisted of solitary abscesses with necrotic centers infiltrated with heterophils and surrounded by giant cells. Septate and dichotomously branched hyphae were seen in the central area of lesions. Thrombi were observed in blood vessels. Fungal hyphae with granulomas were found in the posterior chamber of the eye.

**Histochemistry Results:** The typical, dichotomous branching, regular hyaline, septate hyphal forms were identified in the necrotic lesions of the granulomas when stained by PAS stain with pink-red color.

**Microbiological Examinations:** *Aspergillus* spp. grew readily On Sabouraud dextrose agar (25-37 °C). The colonies of *Aspergillus fumigatus* were at first white and slightly fuzzy, then bluish-green as conidia begin to mature. As the colony matures, The conidial masses became gray-green while the colony edge remains white. On microscopic examination, Conidiophores with vesicles, phalides and conidia stained by lactophenol cotton blue were characteristic for *Aspergillus* spp.

**Discussion:** Aspergillosis, caused by *Aspergillus fumigatus*, *Aspergillus flavus* and other *Aspergillus* species, is a mycotic disease, usually of the respiratory tissues (including the air sacs) characterized by inflammatory granulomatous lesions. Hematogenous dissemination to other organs may occur.

Occasionally the eye, skin, brain and reproductive tract are invaded. The disease is relatively rare in domestic and pet animals but occurs frequently in avian species. Avian aspergillosis may be classified as follows: (1) an acute form, caused by a single exposure to an overwhelming number of spore from a source of environmental contamination; (2) a tracheal form, which is generally a localized lesion occurring in and near the syrinx; (3) a localized abscess or series of granulomas in the air sacs and lungs; and (4) an invasive form, which initially affects the respiratory system and then spreads hematogenously or through air sacs passages to other parts of the body. Accordingly, lesions in the eye, brain, liver, and cardiac muscle may occur. Avian hosts include turkeys, chickens, ducks, geese, many other kinds of poultry, wild birds and cage birds. Penguins raptors, migratory waterfowl and psittacines frequently have aspergillosis. Poult, geese and chicks less than three weeks of age are affected more frequently than adults but adults are susceptible if they are heavily exposed. The disease has been seen in newly hatched chicks infected as embryos following egg shell penetration by fungus. Aspergillosis appears to be more significant in confinement situations where stress factors may be involved or where moldy litter or grain are present. A differential diagnosis for the granulomatous lesions in avian respiratory system should include *Mycoplasma gallisepticum* and another fungus, *Dactylaria gallopavum* infection. In human, the respiratory tract is the principal portal of entry, and inhaled spores can cause disease in healthy as well as compromised patients. The species most frequently associated with disease is *A. fumigatus*, but other species such as *A. flavus*, *A. niger*, and *A. terreus* are occasionally involved. The clinical manifestations of aspergillosis can be divided into three types: allergic, colonizing (aspergilloma), and invasive. Allergic aspergillosis is an asthma-like response to

the fungal spore. Colonizing aspergillosis is characterized by the formation of fungus ball (aspergilloma), a dense collection of hyphae. Invasive aspergillosis can develop from the allergic or colonizing forms of the disease, or it can arise independently. Several factors may predispose the patient to invasive disease: (1) cytotoxic chemotherapy, (2) therapy with broad-spectrum antimicrobial agents, (3) leukopenia, and (4) acute leukemia. Mortality is very high, especially among renal transplant patients and leukemia or lymphoma patients.

#### **Diagnostic Criteria:**

1. The physical examination, tracheal culture, endoscopic examination of the trachea and air sacs, blood work, and serological tests represent a diagnostic battery for detecting aspergillosis.
2. Systemic, yellow or gray nodules in the lungs, air sacs, brains, skin and corneal opacity.
3. Typical necrotizing granulomatous inflammation.
4. Typical pink-red septate, branching hyphae within necrotic zone of the granuloma stained by PAS method.
5. Conidiophores with vesicles, phalides and conidia stained by lactophenol cotton blue in colonial smear.

**Treatments:** In goslings, medication with mycostatin at 100 ppm in feed for 1 week and decontamination of all feed and litter had improved the outcome of treatments.

#### **References:**

1. Barnes HJ, Eckroade RJ, Fletcher OJ, Hitchner SB, Straluss AC, Fite RW, and Hoerr FJ. 1989. Avian Disease Manual. Kendall/ Hunt publishing Co. Iowa. pp:135-138.
2. Boado E, Fonseca C, and Toledo R. 1987. Aspergillosis in geese (*A. anser*) caused by *Aspergillus flavus*. Revista Cubana de Ciencia Avicola. 14(1):85-89.
3. Boyd RF, and Hoerl BG. 1986. Basic Medical Microbiology. Little, Brown and Co. Boston/Toronto. pp:805-807.
4. Calnek BW, Barnes HJ, Beard CW, Reid WM, and Yoder HW. 1991. Diseases of Poultry. Iowa State University Press. Iowa. pp:326-334.

5. Chaudhry SI, Hameed A, and Javed T. 1992. Clinicol-pathological studies on aspergillosis in broilers. *Pakistan Vet. J.* 12(1):32-35.
6. Chihaya Y, Furusawa Y, Okada H, Matsukawa K, and Matsui Y. 1991. Pathological studies on systemic mycoses in calves. *J. Vet. Med. Sci.* 53(6):1051-1058.
7. Finegold SM, and Baron EJ. 1986. *Diagnostic Microbiology*. V. Mosby Co. Toronto/Princeton. pp:740-742.
8. Flach EJ, Stevenson MF, and Henderson GM. 1990. Aspergillosis in gentoo penguins (*Pygoscelis papua*) at Edinburgh Zoo, 1964 to 1988. *Vet. Rec.* 126: 81-85.
9. Fowler ME. 1993. *Zoo and Wild Animal Medicine*. W.B. Saunders Co. Philadelphia. pp:178-181.
10. Jubb KVF, Kennedy PC, and Palmer N. 1993. *Pathology of Domestic Animals*. Academic Press, Inc. San Diego/New York/Boston/Sydney. Vol 2. pp:665-666.
11. Jungerman PF, and Schwartzman RM. 1972. *Veterinary Medical Mycology*. Lea & Febiger. Philadelphia. pp:75-86.
12. Kardevan A, and Palyusik M. 1967. Pathogenic effect of *Aspergillus flavus* on geese. *Acta. Vet. Hung.* 17:301-310.
13. Mahmoud AZ. 1988. Contact infection with aspergillosis of pigeons. II. Pathology of the respiratory system in this disease and role in natural infection with *Aspergillus fumigatus*. *Assiut Vet. Med. J.* 20(39):91-94.
14. Pal M, and Mehrotra BS. 1986. Studies on the association of *Aspergillus fumigatus* with ocular infections in animals. *Vet. Rec.* 118:42-44.
15. Palya V, and Balogh T. 1971. Cerebral aspergillosis in geese and turkeys. *Magyar Allatorvosok Lapja.* 26(6):307-310.
16. Perelman B, and Kuttin ES. 1992. Aspergillosis in ostriches. *Avian Pathol.* 21:159-163.
17. Richard JL, and Thruston RT. 1983. Rapid hematogenous dissemination of *Aspergillus fumigatus* and *A. flavus* spores in turkey poults following aerosol exposure. *Avian Dis.* 27(4):1025-1033.
18. Ross FC. 1986. *Introductory Microbiology*. Scoot, Foresman and Co. Glenview/London. pp:399-400.
19. Slocombe RF, and Slauson DO. 1988. Invasive pulmonary aspergillosis of horses: an association with acute enteritis. *Vet. Pathol.* 25(4):277-281.

## Comparative Pathology Case 53

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**Clinical History:** A 44-year-old female came to ER with chief complaint of hemoptysis and chest pain for one day. Past history revealed chest tapping for pleural effusion and anti-TB treatment were administered during the last hospitalization. She was discharged in stable condition. Chest X-ray examination was performed and revealed a cavitory lesion in the left upper lung field with "ball in hole" finding. Then, she was admitted and received lobectomy of left upper lobe of lung.

**Diagnosis:** Intracavitary aspergilloma and cavitory tuberculosis, Lung.

**Gross Findings:** The lung lobe measured 10.0 x 8.5 x 2.9cm and weighed 110gm. The surface showed diffuse fibrous adhesion with partial collapse. Cut surface showed partially collapsed cavity, measuring 5 x 3.5 x 1.5cm in size and walled by elastic firm fibrous tissue. The cavity was filled with multiple soft tissue, weighing 76gm in total. It was friable and clay-colored. The slide (A3) presented was taken from the soft tissue.

**Histopathological Findings:** Sections from the "soft tissue" nodules showed intracavitary aspergilloma. PAS and GMS stains demonstrated acute angle branching septate hyphae with conidiophore. The wall of cyst and adjacent lung tissue showed caseating granulomatous inflammation and granulation tissue with heavy plasmolymphocytic infiltration. Acid fast stain showed negative result. The lung elsewhere was unremarkable. The section from the lymph node showed anthracosis.

**Histochemistry Results:**



1. PAS stain, Grocott's methenamine Silver Nitrate method and Gridley fungal stain showed hyphae- and spore-bearing conidrophores of aspergillus.
2. Acid fast stain showed negative result.

**Discussion:** Pulmonary aspergillosis represents a common, potentially lethal opportunistic infection that has four unique forms:

1. allergic bronchopulmonary aspergillosis ( ABPA ).
2. aspergilloma.
3. invasive aspergillosis.
4. semi-invasive aspergillosis.

In individuals who are at risk, pulmonary aspergillosis is characterized by a spectrum of clinical and radiographic findings that are intrinsically related to the status of the immune system or the presence of structural lung disease.

Aspergilloma is the result of colonization by fungus of bronchial or pulmonary cavities.

An aspergilloma, or ball of aspergillus fungus, can form in any area of damaged pulmonary tissue in which there is a persistent abnormal space. The most common cause of such pulmonary damage is tuberculosis, but aspergillomas can develop in abscess cavities, bronchiectatic spaces, and cavitated tumors.

Aspergillomas do not occur in healthy lungs. Most, but not all, aspergillomas are caused by *A. fumigatus*. *A. fumigatus* is the most common cause of bronchopulmonary fungal disease in Britain.

Many aspergillomas are asymptomatic and are diagnosed by routine chest X-ray. In symptomatic cases of aspergillomas, the most common symptom is hemoptysis, which is usually recurrent and may be massive and fatal. Systemic symptoms are not common, but general debility and weight loss occurs in a few cases. There are no special clinical findings. Most cases have the clinical finding of the underlying disease, e.g. inactive pulmonary tuberculosis, and bronchiectasis.

The chest X-ray is the single most important test in the diagnosis of aspergilloma. Aspergilloma presents radiographically opportunistic as a focal intracavitary mass and is characterized initially by an increase in the wall thickness of pre-existing cavities. The aspergilloma appears as a solid opacity within the cavity and is usually spherical and may appear to totally fill the cavity. Tomography shows the

aspergilloma to be separated from the wall of the cavity by an air space or "halo."

**Sputum:** Sputum of patient with an aspergilloma consistently shows the presence of mycelial fragments on microscopy and produces a positive culture. Failure to demonstrate *A. fumigatus* in the sputum excludes the diagnosis. The microscopical visualisation of hyphae in sputum indicates fungal colonization of some part of the respiratory tract and is always of diagnostic significance.

**Blood:** The vast majority of patients with an aspergilloma have precipitating antibodies to the offending fungus (usually *A. fumigatus*) in the serum. If the aspergilloma has developed while the patient has been treated with systemic corticosteroids, serum precipitins may be absent.

**Skin tests:** Approximately one-third of patients exhibit skin hypersensitivity to *A. fumigatus*.

**Treatment:** Most aspergillomas do not require specific therapy when recurrent hemoptysis and general ill health are features of the disease. Surgical removal of the fungus ball and cavities is the only treatment that offers permanent cure. Unfortunately, most patients, because of the underlying pathology, are not suitable candidates for formal thoracotomy. Local incision of the chest wall and removal of the aspergilloma without attempt to remove the lung cavity have occasionally been performed with success. However, recurrence is common after this procedure. Drug therapy is generally unsatisfactory. In most patients the development of an aspergilloma does not significantly alter prognosis, which is often poor because of the underlying pulmonary damage. General ill health and weight loss associated with an aspergilloma usually mean a poor prognosis unless surgical removal of the aspergilloma is possible.

**Diagnostic Criteria:**

1. Radiological demonstration of a fungus ball (chest X-ray and tomography).
2. Sputum consistently shows the presence of mycelial fragments on microscopy and produces a positive culture.
3. Serum precipitins to *A. fumigatus*, (90% of cases of aspergilloma, 70% of cases of ABPA).
4. Skin test: hypersensitivity to *A. fumigatus*.
5. Histologically demonstrated fungal hyphae with regular septa and dichotomous branching at 45° angles. Conidiospores are formed if the cavity communicated with a bronchus.

## **References:**

1. Bronchopulmonary and disseminated granulomatous disease associated with *aspergillus fumigatus* and *candida* species infection in a golden retriever. *J. Am. Anim. Hosp. Assoc.* 1996 Mar-Apr; 32(2): 139-45.
2. Aerosol amphotericin B inhalations for prevention of invasive pulmonary aspergillosis in neutropenic cancer patients. *Ann. Hematol.* 1995 Dec; 71(6): 287-91.
3. Retained sponge after thoracotomy that mimicked aspergilloma. *Ann. Thorac. Surg.* 1996 May; 61 (5): 1535 - 6.
4. The use of respiratory-tract cultures in the diagnosis of invasive pulmonary aspergillosis. *Am. J. Med.* 1996 Feb; 100 (2): 171-8.
5. Serodiagnosis and monitoring of *Aspergillus* infections after lung transplantation. *Ann. Intern. Med.* 1996 Aug 1; 125 (3):197 - 201.
6. [Invasive pulmonary aspergillosis associated with influenza virus]. *Ann. Med. Interna.* 1996 Jan; 13 (1):34 - 6.
7. [palliative percutaneous treatment under x-ray computed tomographic control of inoperable pulmonary aspergilloma. Apropos of 30 cases] Senac = JP; Railhac - JJ; *Rev. Mal. Respir.* 1995; 12 (6): 593-9.
8. Comparison of antigen detection and PCR assay using bronchoalveolar lavage fluid for diagnosing invasive pulmonary aspergillosis in patients receiving treatment for hematological malignancies. *J. Clin. Microbiol.* 1995 Dec; 33 (12): 3150-3.
9. Pulmonary aspergillosis: early diagnosis improves survival. *Respiration.* 1995; 62 (6) : 341-7.
10. Opportunistic bronchopulmonary infections after lung transplantation: clinical and radiographic findings. *of columbia*

## Comparative Pathology Case 54

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**Clinical History:** This 40-year-old male has suffered from DM and hasn't received regular treatment for 7 years. Two years ago, he was treated with anti-TB drugs at LMC because of hemoptysis. Unfortunately, 3 weeks before this admission to Chang-Gung Medical Center, he experienced 3 episodes of hemoptysis again. Chest computed tomography with contrast enhancement showed irregular infiltration and partial atelectasis with fungal ball over left upper lung. Bronchoscope examination revealed a whitish fragile mass at the left apical segment. Blood sugar test was AC=204-286 mg/100ml and 2 hr PC=224-237 mg/100 ml. Lung biopsy at the lesion was taken and showed necrotic debris with sulfa granules and necrotic fungi hyphae. He received a lobectomy of left upper lung and decortication of left lung. Fungi and mycobacterial culture were negative. He was treated with Fludiazepam 0.25 gm/qid.

**Diagnosis:** Fibrocalcified pulmonary TB, left Apex

Mixed actinomycosis and aspergillosis lung infection with abscess

DM, NIDDM

**Gross Findings:** The left upper lung measured 15 x 9 x 4 cm. The cut sections revealed a thick-walled cavity measuring 5 x 4 cm with sand-like materials inside. The lung parenchyma elsewhere showed focal hemorrhage and consolidation change.

**Histopathological Findings:** The bronchi of the lung were partially denuded and lined by marked granulation tissue with acute and chronic infection. Focal hemorrhagic necrosis and abscesses formation were seen in pulmonary parenchyma. Foci of necrotic vasculitis, necrotic fungi hyphae, and sulfur granules were seen. Gram's stain demonstrated Gram(+) bacteria. GMS stain showed dichotomous branching fungi hyphae with  $35^{\circ}$  to  $45^{\circ}$  degree.

**Discussion:** The underlying cause of the patient to get mixed actinomycosis and aspergillosis lung infection was his uncontrolled DM. DM, predisposition to tuberculosis, is well known. Initially, the patient suffered from pulmonary TB, followed by actinomycosis lung abscesses and aspergillosis lung 2 years later. This may have been due to the incomplete healing process of the pulmonary TB. Thereafter, the cavity was not able to drain freely into the bronchial tree and left a deadspace. Finally, secretions may have accumulated in it and become predisposed to secondary bacterial and fungal infection.

## Comparative Pathology Case 55

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**Clinical History:** Broilers, 5 to 6wks old, were presented with signs of coughing and gasping, marked dyspnea and expectoration of blood-stained sputum. Morbidity was high and mortality was about 15%.

**Gross Findings:** Lesion was limited to the trachea and larynx in all carcasses. It was characterized by the presence of bloody exudate in the lumen or yellowish-white pseudomembrane on the mucous membrane.

**Histopathological Findings:**

**Trachea:** The epithelium was desquamated, and the underlying lamina propria showed edematous, hyperemic, and hemorrhagic changes with infiltration of some heterophils and lymphocytes. The lumen was filled with mucofibrinous exudates, erythrocytes, and scattered heterophils. Syncytial formation of epithelial cells containing intranuclear inclusion bodies was found in the lumen.

**Lung:** The pneumonia occurred mainly around the primary bronchus of the lung. Tertiary bronchi were plugged with fibrin and heterophils, and the epithelium was hyperplastic with syncytial cells containing intranuclear inclusion bodies in the lumen.

**Discussion:** ILT is a viral infection. The virus belongs to Herpes viridae. It is of varying virulence; the very mild type causes little or no evidence of disease while the very severe form may cause as much as 50 to 70% mortality in infected stock. The organism mainly attacks the respiratory tract and eyes, and death often results from asphyxia. Gross lesions are characterized by caseous exudate and blood in the trachea, bronchi, and air sacs. Some gross lesions in chickens occur in the form of casts or plugs. Caseous exudate in the larynx and trachea is sometimes seen in young chickens infected with trichomoniasis, fowl pox, or aspergillosis.

Herpes viruses cause respiratory diseases in domestic animals in southern Taiwan as shown in the following :

Virus	Disease
Bovine herpes virus 1	Infectious bovine rhinotracheitis (IBR)
Porcine herpes virus 1	Pseudorabies (Pr)
Canine herpes virus 1	Hemorrhagic disease of pups
Avian herpes virus 1	Infectious laryngo-tracheitis (ILT)
Avian herpes virus 2	Marek's disease (MD)
Bovine herpes virus 3	Bovine malignant catarrhal fever (MCF)
Columbid herpes virus 1	Pigeon herpesvirus (PHV)