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Pathology of spontaneous air sacculitis in 37 baboons and 7 chimpanzees and a brief review of the literature

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Abstract

Background—Air sacculitis is an important clinical condition in non-human primates.

Methods—We evaluated 37 baboons and 7 chimpanzees with spontaneous air sacculitis submitted to pathology over a 20 year period.

Results—Air sacculitis was observed almost exclusively in males. Common reported signs were halitosis, coughing, nasal discharges, depression, anorexia, and weight loss. Gross lesions included thickened air sacs and suppurative exudate lining the walls. Microscopic lesions included marked epithelial hyperplasia or hypertrophy, necrosis, fibrosis, cellular infiltrates, and bacterial colonies. Mixed bacterial infections were more common than infections by single species of bacteria. *Streptococcus* sp. was the most frequent bacteria isolated in both baboons and chimpanzees.

Conclusions—This is the first report describing the gross and microscopic lesions of air sacculitis in chimpanzees. The preponderance of males suggests a male sex predilection in baboons.

Keywords

Air sac; Airsacculitis; Pan; Papio; Non-human primate

Introduction

Air sacs are an anatomic feature in many Old World and New World primates [8]. Among Old World primates, they are very distinct in siamangs (*Symphalangus syndactylus*) and orangutans (*Pongo pygmaeus*), [5,28] and are less prominent in macaques (*Macaca spp.*), baboons (*Papio spp.*), chimpanzees (*Pan troglodytes*) and gorillas (*Gorilla gorilla*) [8]. Among New World primates they have been described in owl monkeys (*Aotus trivirgatus*), howler monkeys (*Alouatta spp.*), titi monkeys (*Callicebus spp.*), saki monkeys (*Pithecia spp.*), capuchin monkeys (*Cebus spp.*) and marmosets (*Callithrix spp.*) [8].

Anatomically, air sacs arise from anterior laryngeal diverticula and extend from the larynx through the neck, often into the axillae and cheeks [5]. In baboons the air sac is ventrally

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located and communicates with the larynx via an opening in the lateral ventricles of the vocal cord[9]. In contrast, in chimpanzees the air sac has a central and two lateral parts[14]. The central part extends from hyoid bone cranially to the manubrium sternii caudally[14]. The right and left diverticula are very often of unequal sizes and may extend into the axilla[26]. Various functions of air sacs have been proposed and vary with species. Some of the functions are deepening and increasing the volume of voice, providing cushioning to head, stimulating a mate sexually, communicating over long distances in solitary apes, displaying aggressive behavior, and aiding in swimming[16,17].

Bacterial infections of air sacs have been reported in many non-human primate (NHP) species such as baboons (*Papio spp.*), owl monkeys (*Aotus trivirgatus*), orangutans (*Pongo abelii*, *Pongo pygmaeus*), chimpanzees (*Pan troglodytes*), bonobos (*Pan paniscus*) and gorillas (*Gorilla gorilla beringei*)[3–6,8,9,11,14,16,17,19,20,28] in captive and wild populations[5,8,9,16]. In most reported cases, the etiological agents have been either a gram negative organism or mixed infections of gram negative and gram positive organisms[8,9,14,16,25]. Bacterial air sacculitis varies from a mild subclinical infection to a potentially life threatening syndrome when complicated by aspiration pneumonia, thromboembolism, and septicemia[14]. Often, air sacculitis remains subclinical and causes chronic infections. Common symptoms include mild upper respiratory tract infection, with a nasal discharge, halitosis, and distension of the cervical portion of the air sac[9,12,14].

Limited information is available about the incidence of air sacculitis in captive non-human primate populations, especially in baboons and chimpanzees[3,9,14,17,25]. Most of the literature is in the form of case reports, and the majority of them are more than 20 years old[5,8,9,12,14,16,17,25]. Two reports have discussed the recovery and management of air sacculitis in chimpanzees (*Pan troglodytes*)[14,25] and two in bonobos (*Pan paniscus*) [13,21]. There is no published report of death directly due to air sacculitis in a chimpanzee. Two reports have described air sacculitis induced deaths in baboons in experimental conditions [9,17] but a comprehensive study of air sacculitis pathology in natural conditions is lacking. A recent survey identified air sacculitis in 0.7 % of all baboon necropsies over a 20 year period at our facility[2]. All of these factors emphasize need for more information regarding air sacculitis in captive non-human primate populations. Here, we present a retrospective study of all diagnoses of air sacculitis made at necropsy over a 20 year period in the captive non-human primate population housed at Southwest National Primate Research Center (SNPRC) at the Texas Biomedical Research Institute, San Antonio, TX. We report the presenting complaint, gross pathology, histopathology, and bacteriology associated with air sacculitis in baboons and chimpanzees at SNPRC and a summary of published literature related to NHP.

Materials and methods

Animals

The SNPRC baboon colony population averaged approximately 3250 animals during the 20-year period. Baboons were housed in two six-acre outdoor corrals, outdoor metal and concrete gang cages, and indoor-outdoor cages. For variable periods of time, baboons were housed inside buildings individually or in groups for research or other special purposes. The baboons were fed a diet of commercial monkey chow supplemented with grains, fruits and vegetables. Water was supplied *ad libitum*. The chimpanzees were maintained under similar conditions in indoor/outdoor metal and concrete cages. Standard commercial monkey chow (Teklad®, PMI Nutrition International, LLC, Brentwood, MO 63144) was fed and water was available *ad libitum*. Fresh fruits and vegetables were fed daily. All procedures and care were approved by the Institutional Animal Care and Use Committee.

Animals that died or were euthanized were necropsied and appropriate tissue samples were taken for histological evaluation. The tissues were fixed in 10% neutral buffered formalin, processed conventionally, embedded in paraffin, cut at 5 microns and stained with hematoxylin and eosin or other stains as indicated, and evaluated by light microscopy. When indicated, individual tissues were frozen in liquid nitrogen, and stored at -80°C , fixed with 2% glutaraldehyde for electron microscopy, placed in normal saline or transport medium for cytogenetic evaluation, cultured for bacteria and viruses, or frozen in optimal cutting temperature (OCT) compound for frozen sectioning and further evaluation using immunochemistry. The necropsies and histopathological diagnosis were made by board-certified veterinary pathologists.

Case Selection

A computer search was performed for spontaneous cases which included “air sacculitis” as a pathological finding. The gross necropsy reports, histopathology reports and archived specimens were retrieved and reviewed as needed for confirmation or clarification. All the cases with air sacculitis were searched against another internal database to obtain bacterial culture results.

Results

The results are summarized in tables 1 – 4. We identified 44 cases of air sacculitis, which included 37 baboons and 7 chimpanzees over a period of 20 years (1989–2009). Culture results were available for 14 baboons and 6 chimpanzees. Table 5 summarizes cases of air sacculitis in non-human primates from the literature.

Baboons

Of the 37 cases identified in baboons, 36 were males and 1 was female (Table 1). The average age at death in baboons was 14 ± 5.2 years and ranged from 5 to 26 years. Twelve baboons had clinical diagnosis of air sacculitis. Ten of the twelve clinically diagnosed baboons were euthanized due to poor prognosis; one was admitted to the hospital in a severely chronic condition, remained comatose and died. One baboon (#22) had a clinical diagnosis of air sacculitis confirmed by biopsy; the animal was euthanized three years later without evidence of air sacculitis at necropsy. Among the 25 baboons which were not clinically diagnosed with air sacculitis, the most common complaints were related to the central nervous system (CNS; $n=10$; seizures (3), paralysis (3), other nonspecified CNS (3), and blindness (1)), or respiratory system ($n=6$; pneumonia (3), nasal discharge (2), and rhinitis (1)).

Gross pathology

Air sacculitis was bilateral in 31 baboons, left sided in 4, and right sided in 1; a location was not specified in the biopsied baboon. Air sac walls were moderately to severely thickened, dark red to tan in color, diffusely necrotic, and contained white to yellow exudate which varied in amount from minimal to filling the air sac cavity (Fig. 1). In some cases bronchial lymph nodes were swollen. Fifteen baboons had concurrent pneumonia ranging from a few areas of firmness to consolidation of entire lobes often accompanied with presence of suppurative exudate; three baboons had lung abscess. Three baboons had suppurative meningitis in addition to air sacculitis, including two with clinical histories of ear infections, and one with a clinical history of seizures. Other frequent lesions were amyloidosis, hepatitis, hepatic lipidosis, nephritis, pyelonephritis, and cardiomyopathy, many previous described in baboons[2,22,23,27].

Histopathology

The most common histopathologic lesions of the air sacs are summarized here. There was marked hypertrophy and hyperplasia of air sac epithelium with variable erosion and necrosis, the mucosa and sub mucosa were thickened by fibrous tissue, and the lamina propria and sub mucosa were infiltrated by mostly lymphocytes with fewer plasma cells, histiocytes, neutrophils and colonies of bacteria (Fig. 2). There was marked perivascular infiltration of lymphocytes. Histopathological features of affected lungs were consistent with inhalation of suppurative air sac material with lesions primarily localized in and around airways and consisting of inflammatory cell infiltrates, necro-suppurative exudates and organizing fibrin with bacterial colonies. Alveoli contained proteinacious eosinophilic edema fluid with variable fibrin, macrophages and neutrophils. In cases of concurrent meningitis, the meninges contained a mixed cellular infiltrate primarily composed of neutrophils.

Bacterial culture

Seventeen cultures were collected from 14 of the 37 baboons (Table 2). Ten cultures were from the air sacs, four from lungs, two from the brain/meninges, and one from the blood. Three animals had multiple cultures: two from the air sacs and lungs, and one from the air sac and meninges. In most cases there was mixed infection (n = 8). *Streptococcus spp.* were the most common (n = 8), followed by *Escherichia coli* (4), *Pasteurella multocida* (3), and *Staphylococcus aureus* (2). One case each of *Citrobacter amalonaticus*, *Enterococcus faecium*, *Hemophilus spp.* (not *influenzae*), *Klebsiella pneumoniae*, *Pasteurella haemolytica*, *Fusobacterium spp.*, and *Lactobacillus spp.* were identified. Among the Streptococci, those belonging to the beta-hemolytic subgroup were more common (n = 4), three belonged to the alpha-hemolytic subgroup, and one species was not specified. One case, where only lung cultures were taken, had no bacterial growth.

Chimpanzees

A total of seven cases of air sacculitis in chimpanzees were identified (6 males, 1 female) (Table 3). Three chimpanzees had a clinical history of air sacculitis while air sacculitis was a post mortem finding in four animals. The average age at necropsy was 22 years. Four animals were euthanized and three died naturally. The causes of deaths and reasons for euthanasia as determined by the pathologist at the time of necropsy were: air sacculitis (n = 2), heart failure (n = 2), renal failure (n = 1), chronic liver disease (n = 1), and brain infarction leading to pneumonia and upper respiratory tract infection (n = 1). One chimpanzee had clinical symptoms of air sacculitis; however, the primary cause of death was found to be congestive heart failure.

Gross pathology

Six chimpanzees had bilateral air sacculitis with exudate in both air sacs (Fig. 3); one chimpanzee had left side air sacculitis with dried suppurative exudate in the nasal turbinates. The air sacs were thick walled (n = 4), and lined by viscous, suppurative exudate (n = 7) that varied in color from white to dark tan. The air sac walls were multifocally reddened. Other frequent lesions included pulmonary edema and congestion (n = 4), cardiomegaly (n = 3), hepatomegaly (n = 3), cardiomyopathy (n = 2), hydropericardium (n = 2), hydronephrosis (n = 1) and nephritis (n = 1), many of which have been previously described in chimpanzees[15,24,25].

Histopathology

There was marked hyperplasia of air sac epithelium in all cases. The air sac mucosa and submucosa layers had marked fibrosis, edema, and mixed inflammatory cell infiltration

which ranged from mild to severe and sometimes extended into subjacent tissues including skeletal muscles (Fig. 4). Abundant bacteria were sometimes visible in the mucosal layer of air sacs. There was variable loss of epithelium and the lumen was filled with neutrophils and necrotic debris. Lung lesions included neutrophilic accumulation in airways and interstitium, necrosis, bacterial colonies in necro-suppurative exudate, and proteinaceous fluid in alveolar spaces.

Bacterial culture

Ten cultures were collected from six of the seven chimpanzees (Table 4). Air sac was cultured in five animals. Most cases had mixed infection ($n = 3$); two had solitary infection (*Escherichia coli*, *Staphylococcus aureus*). Other bacteria isolated from the air sacs included *Micrococcus sp.* ($n = 2$), *Pseudomonas sp.* ($n = 2$), *Streptococcus sp.* ($n = 2$), *Acinetobacter* ($n = 1$), and *Escherichia sp.* ($n = 1$). In one chimpanzee with a diagnosis of air sacculitis, nasal cultures were positive for *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Four additional tissues were cultured in three animals: thoracic cavity (positive for *Acinetobacter*, *Enterococcus*, *Alcaligenes*); lung (positive for beta-hemolytic *Streptococcus* Group C, and *Micrococcus sp.*); pericardial sac (positive for *Micrococcus sp.*); and nasopharynx (positive for *Staphylococcus aureus* and *Streptococcus milleri*).

Discussion

We report 37 baboon and 7 chimpanzee cases of air sacculitis during the years 1989–2010 observed among the NHP populations at Southwest National Primate Research Center, San Antonio, TX.

The average age of baboons at the time of necropsy was 14 ± 5.2 years, ranging from 5–26. The sex distribution was very biased towards males in baboons (36 male: 1 female), even though more females are in the colony, as previously reported in a large survey of baboons from the SNPRC[2].

Approximately one third (12 of 37) of the baboons had clinical diagnosis of air sacculitis. The remaining animals either lacked clinical signs of air sacculitis or had signs that were not directly indicative of air sacculitis, such as respiratory or CNS signs that may be related to or extensions of air sac infections. This is in agreement with a previous report where only one out of six affected baboons were clinically diagnosed with air sacculitis[17]. Gross and histopathological lesions were similar to those published previously[9,17].

Most of our cases were due to mixed infection ($n = 8$). *Streptococcus sp.* were the most frequent bacteria isolated, followed by *Escherichia coli*, *Pasteurella multocida*, and *Staphylococcus aureus*. Gross *et al* identified *Pasteurella multocida* as the causative organism for air sacculitis in one baboon under experimental conditions[9]. In the two cases with only one type of bacteria cultured, the bacteria were gram positive, as opposed to most other reports that cultured gram negative bacteria in single infections[5,6,9,14,16].

Previous reports[9,17] of air sacculitis in baboons refer to air sacculitis arising in chronically instrumented chaired baboons. Lewis [17] described air sacculitis in six baboons (one diagnosed clinically and five diagnosed post mortem) all of which were given an opiate drug using an indwelling catheter. Lewis[17] described that one out of six baboons, a swelling in upper portion of thorax and lower portion of neck was observed clinically, and upon repeated paracentesis yielded from 300 ml to 1L of muco-purulent odoriferous exudate containing anaerobic organisms. Air sacs in this animal were surgically removed and the animal survived. Subsequently, five other cases of air sacculitis were identified during necropsy at the same institute and apparently all were subjected to venous catheterization for

periods of up to six months. Histologically, all affected air sacs had mild erosion of ciliated epithelium and a slight increase in mononuclear cells in the lamina propria. The authors[17] proposed three possible reasons for development of air sacculitis in these animals; the first possibility was that opiate compounds increase the respiratory secretions, the second one was possible infection through the catheter, and the third one was the continuous upright posture of chaired baboons may lead respiratory secretions to drain into the duct leading to the air sac based on the anatomic design of larynx. They suggested that if baboons are restrained they tend to accumulate fluids in unusual places including air sacs. In another report[9] air sacculitis developed in a mature male baboon that was catheterized and confined to a special chair for an extended period of time; because the catheter was repeatedly negative for bacteria, the upright position was considered to be more important than catheterization as the cause of infection. Air sacculitis was diagnosed by the presence of fluid in air sacs; the fluid was initially clear but later infection progressed, and the fluid turned purulent and tested positive for *Pasteurella multocida*. Histopathological lesions included goblet cell hyperplasia and marked lymphoplasmacytic infiltrates in the sub-epithelial tissues. In comparison to the previous reports[9,17], these baboons were not part of experimental study, and the air sacculitis in these 37 baboons can be considered as spontaneous infections.

The age distribution in chimpanzees was from 4 to 37 which is in similar range as reported previously (3–37 years)[25]. Three chimpanzees had a clinical diagnosis of air sacculitis and four were diagnosed at necropsy. The sex distribution in our results was very biased towards male chimpanzees (six males to one female), which is different from the previous reports in chimpanzees with an equal distribution[14,25]. However, approximately three times as many male chimpanzees came to necropsies as females, so the significance of this distribution is questionable. The chimpanzees described in the previous two reports[14,25] were treated and survived, so details about the gross and histopathological lesions are not available for comparison. Bacterial culture suggested that mixed infections were predominant in air sacs, and our results are in agreement with previous publications.

Previously, Strobert and Swenson[25] reported the occurrence of air sacculitis in six chimpanzees at Yerkes Regional National Primate Center, Yerkes, GA. There was an even sex distribution and ages ranged from 3–37 years old. Only one chimpanzee had clinical signs of air sacculitis consisting of swollen air sacs, halitosis, and anorexia, four other chimpanzees had persistent distension of air sac but did not appear ill, and in one case air sacculitis was identified during routine physical examination. Air sacculitis was confirmed by aspirating exudate from air sacs. Four infections were mixed infections, bacterial cultures were positive for *Pseudomonas aeruginosa*, *Pseudomonas putrefaciens*, *Mima polymorpha*, *Proteus sp.*, *Escherichia coli*, *Enterococcus sp.*, *Streptococcus sp.*, *Staphylococcus aureus*, *Staphylococcus epidermis*, *Pasteurella multocida*, *Bacteroides spp.*, *Haemophilus influenzae* and *Diphtheroids spp.* Out of the six chimpanzees one recovered after surgical intervention while the remaining ones recovered following irrigation treatment. Hill *et al*[14], reported air sacculitis in a 10 year old chimpanzee. The animal was apparently healthy and the only clinical signs noticed were swollen air sac and halitosis. Upon surgical intervention, approximately 1 L of viscous, foul-smelling, pale-green, purulent exudate was removed from the air sac; culture was positive for beta-hemolytic *Escherichia coli*. The chimpanzee recovered following surgical intervention. A previous case report by Rietschel and Kleeschulte describes the diagnosis and therapy of air sacculitis in Pygmy chimpanzee (*Pan paniscus*)[20]. Two previous case reports describe the diagnosis and successful therapy of air sacculitis in Pygmy chimpanzees (*Pan paniscus*)[13,21].

Among other species, Giles[8] reported air sacculitis in 12 owl monkeys. In two cases there was a clinical history of purulent nasal discharge and swelling in cervical and

intermandibular areas while remaining animals showed symptoms of anorexia, depression and died acutely[8]. Pathologic changes consisted of acute thromboembolic air sacculitis in five animals and subacute suppurative air sacculitis in six animals[8]. Pure culture of *Klebsiella pneumoniae* was isolated from eight owl monkeys, and mixed infections were isolated from three animals (*Proteus mirabilis*, *Escherichia coli*, *Streptococcus sp.*). *Pasteurella multocida* was isolated from one infected air sac.

In one report of air sacculitis in a free ranging mountain Gorilla[12], air sacculitis was considered a complication of upper respiratory infection. Approximately 1L of purulent exudate was removed from the air sacs and showed numerous gram-negative rods and inflammatory cells (75% PMNs, 19% lymphocytes, 5% monocytes, 1% eosinophils). Upon culture, *Staphylococcus aureus* and a species of non-lactose fermenting *Enterobacteriaceae spp.* were isolated[12].

Orangutans are suspected to be more susceptible to air sacculitis, probably due to the well-developed structure in adult males[8,16]. In seven cases of suppurative air sacculitis in orangutans, the causes of death were suppurative pneumonia, chronic airway infection, and toxemia, which suggests that upper respiratory tract infection can induce air sacculitis[1,7,10,11]. Bacterial cultures from these cases tested positive for mixed infection of *Pseudomonas aeruginosa*, *Escherichia coli* and *Streptococcus sp.* in three cases, and one case each of *Proteus vulgaris* and *E.coli*, *Proteus rettgeri*, *Proteus morganii*, *Aerobacter cloacae* and *Alcaligenes spp.* [1,7,8,10,11]. Cambre *et al* [5] described air sacculitis in a female orangutan that experienced severe acute respiratory distress and collapsed and died. Upon necropsy, a turbid brown fluid was observed throughout the tracheobronchial tree and the submandibular air sac was filled with turbid dark brown fluid. Gross lesions included thickening of the air sac wall and medial septum wall. The inner surface of the air sacs was nodular and hemorrhagic. Histopathological lesions included fibrinous inflammatory exudate, ulceration of mucosa, submucosal inflammation, and fibrosis. *Escherichia coli* was isolated from the air sac tissue and fluid as well as from heart blood, tracheal fluid, lung and bile[5]. In a recent report Lawson *et al* [16] reported air sacculitis in 14 juvenile orangutans. Common symptoms were cough, halitosis and nasal discharge, and bacteria isolated included *Pseudomonas spp.*, *Enterobacter spp.*, and *Klebsiella pneumonia*. Four animals recovered following antibiotic therapy, while 10 recovered after surgical intervention.

In 2 cases of recurrent air sacculitis in Sumatran male orangutans (*Pongo pygmaeus abelii*) surgical intervention was successful; *Pasteurella multocida* was identified in one case [13]. In 2 cases of air sacculitis in Southern Bornean orangutans (*Pongo pygmaeus wurmbii*), the female recovered following antibiotic therapy and *Pseudomonas spp.* were identified, while in the male the air sacculitis was a post mortem finding[20]. In 1 case of air sacculitis described in silver leaf langur (*Trachypithecus cristatus ssp ultima*), *Klebsiella* induced air sacculitis was successfully treated with antibiotics and surgical intervention[18].

One caveat in the present study is that it includes only those cases which were confirmed at necropsy or biopsy. In many cases air sacculitis may not present specific symptoms and may resolve on its own or with treatment[14,21,25]. Thus, the frequency of cases described here must be considered a conservative estimate only and the actual number of cases is likely greater in number. It is also possible that the bacteria identified in these cases represent only the more severe cases, those that do not clear spontaneously or are refractory to treatment.

We report 37 baboon and 7 chimpanzee cases of air sacculitis and the first description of the gross and histopathological lesions of air sacculitis in chimpanzees. The preponderance of affected male baboons suggests a sex predilection, though the reason for this is undetermined. Although *Streptococcus* was the most frequent isolate in baboons,

bacteriological results do not strongly suggest that any one organism is more pathogenic in the air sacs. These results provide valuable information concerning an important problem in the captive non-human primate population.

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Fig. 1. Gross pathology of baboon air sacs. (A) Both air sacs thickened and covered by thick tan material (fibrinosuppurative exudate). (B) Air sacs filled with cloudy, viscous fluid (suppurative exudate) (AS= Air sac, T= thorax, H=head).

Fig. 2.

Histology of baboon air sacs. (A) Normal – Ciliated columnar epithelium with mucous cells. Note few lymphocytes in connective tissue. (B) Mild lymphoplasmacytic inflammation and hyperplasia of the epithelium with increased mucous cells and fewer ciliated cells. (C) Lymphocytic inflammation and hyperplasia of the epithelium with squamous metaplasia. (D) Erosive, necrotizing air sacculitis with neutrophils and bacteria. (E) Marked epithelial hyperplasia with squamous metaplasia and lymphoplasmacytic, neutrophilic, and histiocytic inflammation. (F) Epithelial hyperplasia with squamous metaplasia, and lymphoplasmacytic inflammation with lymphoid nodule

Fig. 3.
Gross pathology of chimpanzee air sacs. Air sacs filled with tick, tan-yellow (purulent) fluid. The connective tissues are icteric. (AS= Air sac, T= thorax , H=head)

Fig. 4. Histology of chimpanzee air sacs. (A) Normal – Ciliated columnar epithelium with mucous cells. (B) Hyperplasia of epithelium, lymphocytic inflammation, and necrotic cells and mucus in lumen. (C) Epithelial hyperplasia, mixed inflammation in the mucosa and connective tissue, and suppurative exudate in lumen. (D) Epithelial hyperplasia, necrosis and ulceration of mucosa, neutrophilic and lymphocytic inflammation in the connective tissue.

Table 1

History, gross findings, and signalment of baboons

Animal #	Submitted history	Relevant gross findings	Sex	Age at death
1	CNS signs, Blind, muscle wasting	BAC	M	14
2	Air sacculitis	BAC, pneumonia (right side)	M	20
3	Hypothermia, ear infection	BAC, suppurative meningitis	M	12
4	No significant clinical history	BAC	M	14
5	CNS signs, Seizures	BAC	M	5
6	Air sacculitis, pneumonia	BAC, pulmonary abscesses	M	21
7	Air sacculitis	BAC, pleuritis, pneumonia, tracheitis	M	13
8	Air sacculitis	BAC	M	15
9	Paralysis	LSAC	M	16
10	Spondylosis	RSAC	M	22
11	Air sacculitis, pneumonia	BAC, suppurative pneumonia	M	21
12	Air sacculitis, pneumonia	BAC, pneumonia, pleural effusion	M	22
13	Moribund	BAC, pneumonia, cellulitis	F	6
14	Air sacculitis, aspiration, cellulitis	BAC, cellulitis, necrotizing pneumonia	M	7
15	Found dead - No significant clinical history	BAC	M	7
16	Severe spondylosis, periodontal disease	BAC, periodontal disease, spondylosis, arthritis	M	26
17	No significant clinical history	BAC	M	23
18	Renal failure, weight loss	BAC, end stage kidneys	M	8
19	Seizure, cellulitis, paralysis	BAC, suppurative meningitis, myositis	M	17
20	Depressed, hepatopathy	BAC, minimal pneumonia	M	10
21	Nasal discharge, diarrhea, CNS signs	BAC, neoplasia (disseminated lymphoma), rhinitis	M	11
22	Air sacculitis	Biopsy	M	N/A
23	Air sacculitis	BAC	M	10
24	Found dead - No significant clinical history	BAC, hepatitis	M	13
25	Pneumonia	BAC, pneumonia	M	17
26	Air sacculitis, renal failure	LSAC, focal abscess on lungs	M	13
27	Dehydration, oral discharge	BAC, pneumonia, pleural effusion	M	7
28	Ear infection	BAC, otitis, meningitis	M	17
29	Seizures	BAC	M	10
30	Pneumonia, weight loss	LSAC, pneumonia	M	14
31	Depressed, anorexia, rhinitis	BAC, rhinitis, hepatic lipidosis	M	16
32	Pneumonia	BAC, pneumonia	M	16
33	Air sacculitis	BAC, pleuropneumonia	M	8
34	No significant clinical history	BAC, mild pneumonia, fibrinous peritonitis	M	10
35	Air sacculitis, gingivitis, dermatitis/cellulitis	BAC, cellulitis, pulmonary abscess	M	11
36	Found dead- No significant clinical history	BAC, pneumonia	M	14
37	Paralysis	LSAC, pyelonephritis, cystitis	M	15

Key: BAC = Bilateral air sacculitis; LSAC = Left sided air sacculitis; RSAC = Right sided air sacculitis

Table 2

Bacterial cultures for baboons

Animal #	Specimen	ETIOLOGY
1	Air sac	<i>Klebsiella pneumoniae</i> , <i>Escherichia coli</i>
2	Air sac	<i>Staphylococcus aureus</i> , <i>Citrobacter amalonaticus</i>
7	Air sac	<i>Streptococcus equisimilis</i> , <i>Haemophilus sp</i> beta lactamase negative
12	Lung	<i>Streptococcus spp.</i> Beta group
13	Air sac	<i>Streptococcus spp.</i> Beta group, <i>Escherichia coli</i> , <i>Pasteurella multocida</i>
	Lung	<i>Pasteurella multocida</i>
14	Air sac	<i>Streptococcus spp.</i> Alpha group, <i>Escherichia coli</i>
19	Brain	<i>Enterococcus faecium</i>
21	Air sac	<i>Streptococcus spp.</i> Beta group, <i>Pasteurella multocida</i>
24	Air sac	<i>Streptococcus spp.</i>
25	Blood	<i>Pasteurella haemolytica</i>
30	Lung	Negative
28	Air sac	<i>Staphylococcus aureus</i>
	Meninges	<i>Staphylococcus aureus</i>
32	Air sac	<i>Streptococcus viridans</i> , <i>Streptococcus spp.</i> Beta group, <i>Lactobacillus spp.</i>
33	Air sac	<i>Streptococcus viridans</i> , <i>Escherichia coli</i> , <i>Pasteurella multocida</i>
	Lung	<i>Fusobacterium spp.</i> , anerobic gram – rod, aerobic gram + cocci

Table 3

History, causes of death, gross lesions, and signalment of chimpanzees

Animal #	Submitted history	Cause of Death	Gross lesions	Sex	Age
1	Air sacculitis	Chronic air sacculitis	BAC, airs ac contained pus, pulmonary edema and congestion, hydrothorax	M	20
2	Died acutely, blood in trachea and urine	Chronic air sacculitis, septicemia, pneumonia, hemolytic crisis and hemoglobinuric nephrosis	BAC, disseminated hemolysis	M	4
3	Air sacculitis, congestive heart failure	Congestive heart failure	BAC, ascites, pulmonary congestion and edema, cardiomegaly, cardiomyopathy, hepatopathy	M	25
4	Icteric, enlarged liver, biliary obstruction	Hepatic amyloidosis with hemorrhage	BAC, icterus, hepatomegaly, hydropericardium, abdominal hemorrhage, rectal mass	F	26
5	Air sacculitis, renal failure	End stage renal disease	BAC, hydronephrosis, uroliths, end stage kidneys, hepatomegaly	M	31
6	Lethargic and vomiting	Cardiomyopathy	BAC, cardiomegaly, hydrothorax, hydropericardium, pulmonary edema and congestion	M	13
7	Chronic scoliosis, dwarfism, neurological symptoms	Brain infarct with pneumonia	LSAC, pneumonia, cardiomegaly, cardiomyopathy, hepatopathy, ascites	M	38

Key: BAC = Bilateral air sacculitis; LSAC = Left sided air sacculitis; RSAC = Right sided air sacculitis

Table 4

Bacterial cultures for chimpanzees

Animal #	Specimen	ETIOLOGY
1	Nasal cavity	<i>Staphylococcus aureus</i> , <i>Pseudomonas aeruginosa</i>
2	Air sac	<i>Micrococcus spp.</i> , <i>Streptococcus pneumoniae</i>
	Lung	<i>Micrococcus spp.</i> , <i>Streptococcus spp. Beta group</i>
	Pericardial sac	<i>Micrococcus spp.</i>
3	Air sac	<i>Streptococcus spp.</i> , <i>Escherichia spp.</i> , <i>Pseudomonas spp.</i> , <i>Acinetobacter spp.</i>
	Thoracic cavity	<i>Acinetobacter</i> , <i>Enterococcus</i> , <i>Alcaligenes spp.</i>
4	Air sac	<i>Pseudomonas aeruginosa</i> , <i>Micrococcus spp.</i>
6	Air sac	<i>Staphylococcus aureus</i>
7	Air sac	<i>Escherichia coli</i>
	Nasal cavity	<i>Staphylococcus aureus</i> , <i>Streptococcus milleri</i>

Table 5

Summary of reports of air sacculitis in non-human primates

Species	# animals (sex)	Other lesions	Experimental?	Reference	Bacteria isolated
1 Baboon (<i>Papio anubis</i>)	1 (M)	-	Yes	Lewis, 1975	<i>Aerobic organism</i>
2 Baboon (<i>Papio anubis</i>)	1 (M)	-	Yes	Gross, 1978	<i>Pasteurella multocida</i>
3 Baboon (<i>Papio cynocephalus</i>)	2 (M)	Pleuritis, bronchopneumonia, cardiac arrest	Yes	Brondson, 1993	<i>P. multocida</i> , <i>Neisseria</i> , <i>Corynebacteria</i>
4 Chimpanzee (<i>Pan troglodytes</i>)	6 (3M, 3F)	-	No	Strobert, 1979	<i>Pseudomonas putrefaciens</i> <i>Mima polymorpha</i> <i>Proteus vulgaris</i> <i>Escherichia coli</i> <i>Enterococcus</i> <i>Beta hemolytic streptococci</i> <i>Staphylococcus aureus</i> <i>Staphylococcus epidermidis</i> <i>Pasteurella multocida</i> <i>Proteus morganii group D</i> <i>Bacteroides</i> <i>Haemophilus influenzae</i> <i>Pseudomonas aeruginosa</i>
5 Chimpanzee (<i>Pan troglodytes</i>)	1 (F)	-	No	Hill, 2001	<i>Beta hemolytic Escherichia coli</i>
6 Chimpanzee, Pygmy (<i>Pan paniscus</i>)	1 (F)	-	No	Rietschel, 1989	<i>Streptococcus spp</i> <i>Klebsiella spp</i> <i>Coliform</i> <i>Pseudomonas aeruginosa</i> <i>Providentia stuartii</i>
7 Chimpanzee, Pygmy (<i>Pan paniscus</i>)	NR	NR	NR	Brown, 1995	NR
8 Chimpanzee, Pygmy (<i>Pan paniscus</i>)	1 (M)	-	No	Herrin, 2002	No
9 Mountain gorilla (<i>Gorilla gorilla beringei</i>)	1 (M)	-	No	Hastings, 1991	<i>Staphylococcus aureus</i>
10 Orangutan (<i>Pongo pygmaeus</i>)	1 (M)	Pneumonia, toxemia	No	Hassko, 1929	No bacteriology report
11 Orangutan (<i>Pongo pygmaeus</i>)	1 (M)	terminal septicemia	No	Anonymous, 1963	No bacteriology report
12 Orangutan (<i>Pongo pygmaeus</i>)	1 (M)	-	No	Giene, 1965	<i>Proteus vulgaris</i>

Species	# animals (sex)	Other lesions	Experimental?	Reference	Bacteria isolated
13 Orangutan (<i>Pongo pygmaeus</i>)	4 (2M, 2F)	Consolidated lung Lung abscesses Lympho reticular cell hyperplasia Lymphadenitis	No	Guilloud, 1969	<i>E. coli</i> <i>Pseudomonas aeruginosa</i> - 3 cases <i>Escherichia coli</i> - 3 cases <i>Alpha hemolytic streptococci</i> - 3 cases <i>Aerobacter cloacae</i> - 1 case <i>Proteus rettgeri</i> - 1 case <i>Proteus morganii</i> - 1 case <i>Alkaligenes</i> - 1 case <i>Escherichia coli</i>
14 Orangutan (<i>Pongo pygmaeus</i>)	1 (F)	Tracheitis, Pneumonia, Splenic necrosis Cervical and thoracic lymphoid hyperplasia Fatty liver	No	Cambre, 1980	
15 Orangutan, Southern Bornean (<i>Pongo pygmaeus wummbi</i>)	14 (5M, 9F)	-	No	Lawson, 2006	<i>Pseudomonas aeruginosa</i> <i>P. alcaligenes</i> <i>Klebsiella pneumoniae</i> <i>Enterobacter species</i> <i>Staphylococcus aureus</i> <i>Pseudomonas aeruginosa</i> in female
16 Orangutan, Southern Bornean (<i>Pongo pygmaeus wummbi</i>)	2 (1 M, 1 F)	Female - sarcoma in neck, myocarditis, lymphadenitis Male - Dilated cardiomyopathy, vascular thrombosis, renal cortical necrosis	No	Plesker, 2006	
17 Orangutan, Sumatran (<i>Pongo pygmaeus abelii</i>)	2 (1M, 1F)	-	No	Herrin, 2002	<i>Pasteurella multocida</i> - 1 case
18 Owl monkey (<i>Aotus trivirgatus</i>)	12 (NR)	Suppurative bronchopneumonia Suppurative lymphadenitis Necrotic hepatitis Thromboembolism		Giles, 1974	<i>Klebsiella pneumoniae</i> <i>Proteus mirabilis</i> <i>Escherichia coli</i> <i>Beta hemolytic streptococcus</i> <i>Pasteurella multocida</i> <i>Non-lactose fermenting Enterobacteriaceae spp.</i> <i>Mycoplasma pneumoniae</i> by serology
19 Pig-tail macaque (<i>Macaca nemestrina</i>)	NR	NR	NR	Brown, 1995	NR

	Species	# animals (sex)	Other lesions	Experimental?	Reference	Bacteria isolated
20	Silver leaf langur (<i>Trachypithecus cristatus ssp ultima</i>)	1 (M)	pneumonia	No	Luz, 2005	<i>Klebsiella ssp.</i>