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The Pulmonary Pathology in the Acquired Immunodeficiency Syndrome (Aids)

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ABSTRACT

The pulmonary pathology in 120 autopsies of venezuelan AIDS patients was reviewed. A detailed histophatologic study of numerous sections of the lung using hematoxylin-eosin and special stains was done to detect alterations of the pulmonary parenchyma and opportunistic agents. The percentage of microorganisms found was Histoplasmosis (40,8%), cytomegalovirus infection (34,7%), bacterial pneumonia (33,9%), pneumocystis carinii pneumonia (29,9%), micobacterial infections (10,4%), candidiasis (8,6%) and cryptococcosis (6,9%). Histological changes in the lungs were related to infectious agents as well as to diffuse alveolar damage, interstitial pneumonitis and focal or diffuse necrotizing pneumonia. Kaposi's sarcoma and non Hodkin's lymphomas were also observed in the lung tissue. Differences between opportunistic agents in our series and series described in other countries seems to be related to socioeconomic and geographic factors. The importance of examining the autopsy pathology in venezuelan AIDS patients is stressed.

KEYWORDS:
AIDS opportunistic agents, pneumonitis, neoplasms.

INTRODUCTION

Patients with the acquired immunodeficiency syndrome (AIDS) initially consult by non specific pulmonary symptoms and the majority of them often died of pulmonary complications related to opportunistic infectious agents (1).

Clinical physicians and pathologists should be aware of the variety of alterations observed in the lung of AIDS patients since pulmonary lesions can diagnosed by bronchoscopic procedures such as lavage, brushing and bronchial or transbronchial biopsies.

The aim of this paper is to discuss the pathology of the lung from 120 AIDS patients autopsied at the Central University of Venezuela-Anatomopathologic Institute in Caracas. The significance of this study made with a large group of autopsies from Venezuelan AIDS patients is described [2,3].

MATERIALS AND METHODS

Autopsies performed in adults who met the the strict cirteria of the Centers for Disease Control for AIDS were studied. Fragments of the lung from 120 autopsies were fixed in 10% formalin and embedded in paraffin. Hematoxylin-eosin stained sections were reviewed. PAS, Gomori's and Masson's trichrome stains, Ziehl-Nielsen, reticulin, Alcian blue, Prussian blue and Grocott's stain were also examined in order to determine the true extent of pulmonary alterations or to detect opportunistic microorganisms. Clinical data as well as autopsy diagnoses were carefully compared. A detailed microscopic examination of all the sections from the lungs was performed and numerous histologic sections from the paraffin embedded tissue were made in order to clarify or confirm diagnostic findings.

RESULTS

The patients ranged in age from 18 to 60 years old (an average of 34.5 years for male and

30.28 years for female) The patients were 112 men and 8 women. The time elapsed between death and autopsy performance varied from 3 to 30 hours. The risk factors detected in the patients clinical history are briefly stated on table 1.

TABLE 1

Risk factors on 120 autopsies in AIDS patients.

Homosexuals	73.00
IV drug users	12.17
Sexual promiscuity	6.08
Bisexuals	3.40
Hemophiliacs	0.86

Post mortem findings regarding opportunistic agents found at the time of final autopsy study are summarized on Table 2.

TABLE 2

Opportunistic agents (%) in 120 autopsies of AIDS patients.

Cytomegalovirus	51.6
Histoplasma capsulatum	39.1
Human Papiloma carinii	28.3
Pneumocystis carinii	27.5
Candida sp	23.3
Toxoplasma gondii	15.0
Criptosporidium sp	13.3
Criptococcus neoformans	10.8
Mycobacterium sp	8.3
Herpes simplex virus	2.5
Molluscum contagiosum	1.6
Strongyloides stercoralis	0.8
Paracoccidioides brasiliensis	0.8

The most common recognized infection was widespread cytomegalovirus (CMV) infection (Figs. 1 and 2). The adrenal glands were the preferential site for detection of CMV and necrotizing adrenalitis were often found. The adrenal involvement by opportunistic agents is summarized on Table 3.

TABLE 3

Autopsy pathology of the adrenal glands: opportunistic agents found in 62 autopsies of AIDS patients.

	n	%
Cytomegalovirus	35	58.3
Histoplasma capsulatum	15	25.0
Cryptococcus neoformans	5	8.3
Mycobacterium sp	5	8.3
Toxoplasma gondii	3	4.8

On Table 4 the opportunistic organisms found in the lungs of our autopsies on AIDS patients are listed.

TABLE 4

Pulmorary pathology in 120 autopsies of AIDS in Venezuelan opportunistic agents

	n	%
Histoplasma capsulatum	47	40.86
Cytomegalovirus	40	34.78
Bacterial pneumonia	39	33.90
Pneumocystis carinii	31	29.95
Mycobacterium sp	12	10.43
Candida sp	10	8.69
Criptococcus neoformans	8	6.95
Mycobacterium tuberculosis	3	2.60
Paracoccidioides brasiliensis	1	0.86
Ficomicetae	1	0.86

The most important opportunistic agent detected in the lung was *Histoplasma capsulatum.* Macrophages loaded with fungi and necrotic foci in the pulmonary parenchyma were the main changes detected on microscopic examination (Fig. 3). Occasionally caseating or non caseatin granulomas were detected. The silver methenamine Grocott's stain was used to detect ovoid yeasts of 2 to 5 µm in diameter. Cytomegalovirus infection of the lungs was characterized by inclusion bodies in the nucleus of enlarged cells associated with thickening of the alveolar walls and hyalin membranes. PNC pneumonia revealed a foamy or granular exudate with fibrosis within the alveolar. Grocott silver methenamine stain is also useful to detect the cysts of PNC (Figure 4). In bacterial pneumonia

polymorphonuclear leukocytes were seen in the alveoli and focal areas of necorsis in the lung parechyma. Areas of necrosis with masses of ecsinophilic granular material and focal granulomatous lesions with enlarged lymphocytes were induced by mycobateriae (Fig. 5), Zielh-Nielsen stain revealed numerous rod shaped or filamentous acid fast bacilli. Other opportunistic fungi like ***cryptococcocus neoformans, Candida sp, Paraccocidioides brasiliensis*** and a case of pulmonary phycomicosis were detected using special stains (Alcian blue, PAS, and Grocott's). Alterations of the lung induced by DAD in the early and late phase of well non-specific interstitial pneumonitis and a case of interstitial pneumonitis were found. The pulmonary neoplastic diseases associate to AIDS were Kaposi's sarcoma (Fig. 6) (16 cases), and non Hodkin lymphoma (5 cases), one of them was considered to be primary of the lung.

DISCUSSION

In this s:ady the inflamatory changes induced in the lung by opportunistic organisms and alterations leading to alveolar damage, interstitial pneumonitis or malignant tumors were made by examining in 120 autopsies of Venezuelan AIDS patients. The opportunistic infectious agents were studied on the basis of their prevalence in the pulmonary parenchyma (Table 4).

Histoplasmosis: ***Histoplasma capsulatum*** was identified in 47 samples among (40,86%) 120 autopsies. In 27 cases dissemi nated histoplasmosis was observed. Dissemination of Histoplasma capsulatum seems to be the rule in HIV infected patients [4]. There are highly endemic areas of histoplasmosis in the United States and Central and South America, particularly in temperate areas of the Caribbean [1]. In Venezuela, histoplasmosis is endemic in many cities. ***Histoplasma capsulatum*** is a dimorphic yeast that survives in soil contaminated with bird or bat droppings; when inhaled the spores are deposited in the alveolar spaces and a granulomatous inflammatory reaction is observed [4, 5]. In the HIV infected patients reactivation of a latent infection or progression of a newly acquired infection is the mechanism involved in the pulmonary alterations [5]. Macrophages filled with fungi, necrotic foci with neutrophils and occasionally caseating and non caseating granulomas are observed in the lungs. In our autopsies the diagnosis of disseminated histoplasmosis was made on the basis of morphologic identification of ovoid yeasts of 2 to 5 µm in diameter [6]. Severe clinical findings are related to the multifocal involvement of the lung by the necrotizing lesions induced by this fungus. The importance of pulmonary histoplasmosis in Venezuelan AIDS patients should be stressed.

Cytomegalovirus: Cytomegalovirus (CMV) is the most usually identify pathogen with widely disseminated infection in our general autopsies (Table 2) and the second opportunistic agent observed in the lung. While in some series the lung has been described as the most frequently involved site of CMV infection [7, 8], in our autopsies from AIDS patients, we observed that the adrenal gland was involved by CMV more than the lung [2, 3] (Table 3). Discrepancies in the accuracy of the clinical diagnosis of CMV disseminated infection in AIDS patients when compared with our autopsy findings. Thus may be mainly due to the fact that CMV infected cells are focally distributed in the lungs [9]. Infection with CMV occurs either in childhood or in adulthood and invariably the infections becames latent in the immunocompetent host. Some autopsy series indicate that CMV infection is found in approximately 90% of AIDS patients [10]. When CMV causes disease in the lung of patients with AIDS a syndrome of interstitial pneumonia is observed [11]. Associations of CMV with Kaposi's sarcoma in AIDS patients have been related to the incidence of CMV infection in the homosexual men [12], on the other hand, CMV have been also considered an oncogenic virus because of its relationship with Kaposi's sarcoma. On similar basis the group of Epstein-Barr virus (EBV) is associated to Burkitt's lymphoma and to nasopharyngeal carcinoma and the hepatitis B virus is also involved in the genesis of hepatocellular carcinoma. The importance of AIDS and neoplasia have been previously discussed [3]. The histopathology of CMV pneumonia in patients with AIDS may show different patterns; a diffuese alveolar damage (DAD) [8] is frequently noted when cytomegalic cells are numerous and they are packing the alveolar spaces or lining the alveolar walls. Pneumocytes, endothelial cells and the

bronchial epithelial cells may also exhibit CMV inclusion bodies; in such instances the diagnosis of a severe CMV pneumonitis is made. The clinical significance of CMV infection of the lung in AIDS patients is difficult to detect, however, evidences suggests that CMV may be the sole cause of DAD, even with a few enlarged cells exhibiting inclusion bodies [3].

Bacterial infections: Coexisting CMV infections with other opportunistic agents such as ***Pneumocystis carinii, Histoplasma capsulatum,*** and particulary with non specific inflammatory bacterial agents is the rule. Patients in all stages of AIDS are known to be infected by pyogenic bacteria [14]. On the other hand, many of our AIDS patients were in a late stage of HIV infection with coexisting risk factors for pneumonia such as neutropenia and indwelling venous catheter. Bacterial infections are most often due *Streptoccoccus, **Haemophilus influenza, Klebsiella pneumonial, Pseudomona aeruginosa*** and ***Staphylococcus aureus*** [15]. Autopsy findings of bronchopneumonia most likely represent a terminal infection. Mycobacterial infections in our AIDS patients will be discussed later in this paper.

Pneumocystis carinii. Before the AIDS pandemic ***Pneumocystis carinii*** (PNC) pneumonia was an interstitial plasmocellular pneumonia observed in newborns or immunosupressed children [16]. ***Pneumocystis carinii*** infection is the most common pulmonary opportunistic agent described in AIDS patients elsewhere [17]. Originally classified as a protozoan, PNC show ultrastructural and biochemical similarity to fungi [18, 19]. Methenamine silver stains the cyst wall black, it measures from 5 to 7 µm in diameter, and when collapsed may show a crescentic appearance. Tropozoites of PNC are amoeboid, they measure from 2 to 8 µm and they are released from the cysts and attached themselves to type I pneumocyte causing subepithelial blebs and exudation of fibrin and plasma proteins into the alveoli [20]. The characteristic eosinophilic intralveolar foamy exudate is composed of the trophozoites immersed in serum proteins; eventual denudation of basement membrane of type I pneumocyte and hyaline membrane fibrinous material is also found. Proliferative changes of the epithelial cells, thickening of the alveolar walls, interstitial infiltrates with mononuclear cells are related to proliferative phase of DAD. Diagnostic procedures, histologic and ultrastructural details of the PNC infection of the lung have been well documented [21, 22, 23]. The presence of PNC in the lung of our autopsies was not related to the clinical diagnosis of pneumocystis carinii infection perhaps because of the treatment, however a variety of techniques have been described in order to detect PNC; fluorescence methods, Giemsa or Wright stains, the Grocott's modification of Gomori's silver methenamine stain used in our investigation and monoclonal antibodies used for immunohistochemical identification of PNC have been also described [24, 25]. While PNC pneumonia may be fulminant in some AIDS patients with rapidly progressive respiratory failure, most AIDS patients show a subacute insidious pulmonary disease.

Mycobacterial infections: The diagnosis of mycobacterial infections of the lung in our 120 autopsies performed in Venezuelan patients is based on the histopathologic changes and the presence of acid fast bacilli in the tissues. Mycobacterial infections may be due to ***Mycobacterium tuberculosis, M. avium*** intracellulare, or ***M. kansasii.*** Tuberculosis has long been associated with impaired cellular immunity. HIV infection in areas with a high prevalence of tuberculous infection is likely to induce reactivation of old tuberculous pulmonary lesions [26]. Since the inflammatory response to ***M. tuberculosis*** depends on the impairment of the cellular immunity, granulomas are not formed in advanced stages of the disease. Our cases of tuberculosis showed poorly formed granulomas and groups of acid fast bacilli. We considered 3 cases of infection with M. tuberculosis, two of them were clinically confirmed, the other seemed to be a case of reactivation. We found acid fast bacilli in 12 cases with characteristics of the so-called ***mycobacterium avium*** complex (AMC) and focal necrotizing areas in other organs such as the spleen, the liver and lymph nodes were observed in all cases. Occasionally a poorly granulomatous appearance was detected, but most cases showed a necrotizing type of inflammation with macrophages filled with a large amount of acid fast bacilli. The minimal inflammatory response and the presence of histiocytes packed with rod-shaped bacilli stained with PAS is diagnostic of this severe opportunistic infection [27, 28]

Candidiasis: It is known that mucocutaneous candidiasis of the oral cavity and pharynx is extremely common in AIDS patients, however, involvement of the lung by Candida sp is uncommon [1,29]. We found 10 cases (8,9%) with ***Candida*** sp and it was always associated to other opportunistic agents. Pulmonary candidiasis is a common late complication of AIDS. Systemic candidiasis in patients with immunodeficiency was recently reported to be higher when compared with a group of AIDS patients [30].

Cryptoccocosis: The most common cause of fungal pneumonia in AIDS is the infection caused by *Cryptococcus neoformans* [31]. It has been reported that cryptococcal pneumonia in AIDS patients usually occurs in individuals who have meningoencephalitis or disseminated cryptococosis, and on the other hand less than 10% of these patients showed pulmonary cryptococcal infection as the primary site [32]. Pulmonary infection with *cryptococcus neoformans* (CN) was detected in 8 cases (6,95%), meanwhile 7 cases pulmonary opportunistic agents coexisted with cryptococcal infection; four cases with *Histoplasma capsulatum*, three with CMV and *Histoplasma capsulatum*, CMV and PNC were also found in two cases of pulmonary cryptococcosis. *Cryptococcus* is an encapsulated yeast with a diameter of 4 to 7 µm and characterized by a thick mucopolysacharide capsule which makes a clear space of 3 to 5 µm between the surrounding tissues and the yeast.

Interstitial Lung Diseases: Since most opportunistic infections of the lung led to DAD or to interstitial alveolar changes resembling non specific neumonitis, it is important for pathologists to examine interstitial lung disease because it is the main life threatening complication of HIV infection. Several types of histologically well defined pneumonitis in absence of opportunistic agents have been described; lymphoid interstitial pneumonitis (LIP), Non specific pneumonitis (NSP), and drug induced pneumonitis have been also described in the lung of AIDS patients [33]. Interstitial pneumonitis in our autopsies were mainly related to infectious agents. Lymphoid interstitial pneumonia (LIP) was only found in one of the 120 autopsies. Recent studies of LIP associated with HIV infection have demonstrated the presence of Epstein-Barr virus (EBV) genome [34], however evidences seems to indicate that LIP is the morphologic expression of variety of different disease processes; diffuse alveolar damage (DAD) is also a non specific pattern of pulmonary injury induced by a variety of causes [35]. It is important to note that the presence of two morphologic stages in DAD should be recognized by pathologists, an acute exudative and an organizing or proliferative phase [35, 36] DAD have been reported in the lung of numerous AIDS autopsies [37, 38] and differences between DAD and interstitial pneumonitis leading to pulmonary fibrosis are based in the extensive fibroblastic proliferation with large amount of hyaluronic acid and eptithelial regeneration and little collagen in DAD while pneumonitis show dense collagen deposition and little fibroblastic and regenerative changes [39].

Malignant Tumors: Some autopsy series described Kaposis's sarcoma (KS) in 94,2% of the AIDS patients [40] however the recognition of an inflammatory variant of KS was considered to be responsible for this unusual high prevalence when compared to 30-70% of other series (8, 100. In our 120 autopsies KS was found in the lung of 13(11,3%) cases. We observed peribronchial areas with plump, spindle cells, slits and red blood cells with hemosiderin in all cases with histologic criteria for KS. When diffuse bilateral pulmonary infiltrates are clinically detected transbronchial bipsy by bronchoscopic procedures is recommended [41]. Evidence of vascular proliferation in the lungs was frequently related to CMV infection. We observed 17 cases, many of them with focal hemorrhages among 23 cases of pulmonary CMV infection. Because of the focal appearance of KS in the lung parenchyma clinical diagnosis is based in bronchial or transbronchial biopsies that not always show positive results. In spite of initial evidence of non involvement of the lung by non Hodgkin lymphomas, in our 120 cases we have 5 cases (4,3%) of non Hodgkin lymphomas, one of them primary in the lung. Some reports described from 6 to 25% the incidence of non Hodgkin lymphoma in the lung [32, 42].

The pulmonary pathology reviewed in 120 Venezuelan AIDS patients point out the importance of the autopsies. We described a wide spectrum of pulmonary diseases and on the other hand a variety of opportunistic agents that

statistically differ of the series reported in other countries. The present report emphasizes the importance of the pulmonary pathology in the demise of venezuelan AIDS patients.

RESUMEN

Se estudió la patología pulmonar en autopsias de 120 pacientes venezolanos enfermos de SIDA. Se examinaron detalladamente numerosos cortes histológicos del pulmón teñidos con hematoxilina-eosina y con coloraciones especiales con el fin de detectar gérmenes oportunistas y alteraciones del parenquima pulmonar. Se observaron casos de histoplasmosis (40,8%) infecciones por CMV (34,7%), neumonias bacterianas (33,9%), neumonias por ***pneumocistis carinii*** (29,9%), infecciones por micobacterias (10,4%), candidiasis (8,6%) y criptococosis (6,9%). Se descubrieron también casos de sarcoma de Kaposi y de linfomas de no Hodgkin. Se destaca la importancia del estudio de la patología en las autopsias de SIDA en Venezuela ya que nuestros agentes oportunistas están relacionados con condiciones geográficas o socioeconómicas y difieren en algunos aspectos de los gérmenes descritos en el SIDA en otros países.

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LEGEND FOR FIGURES

Figure 1: Cytomegalovirus infected cells in the mucous glands of thegastrointestinal tract.
100x.

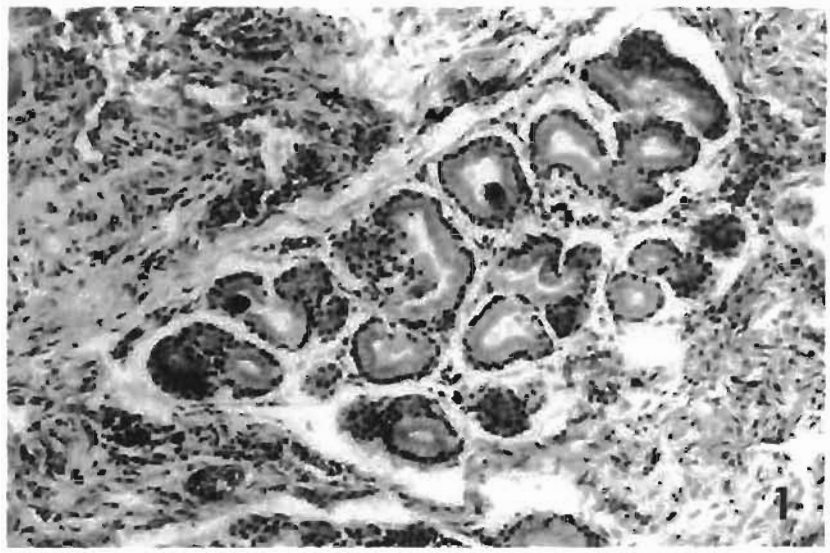


Figure 2: Macrophages loaded with Histoplasma capsulatum in the alveolar spaces. H&E stain.
400x.

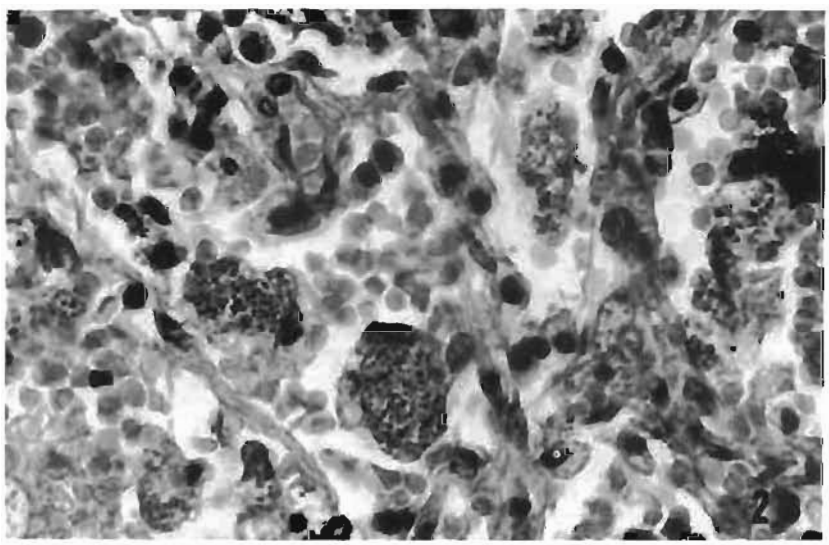
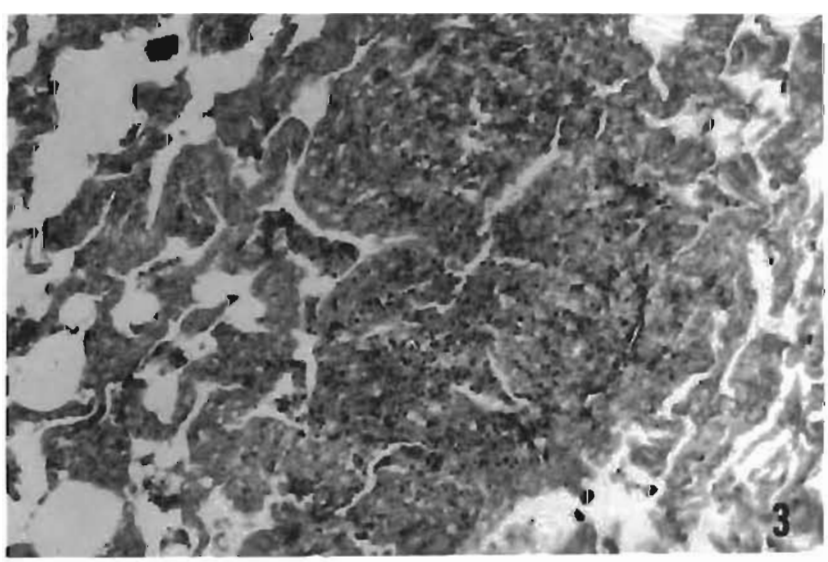


Figure 3: Grocott' s stain of a necrotic area in the lung parenchyma. Numerous Histoplasma yeasts are seen.
100x.



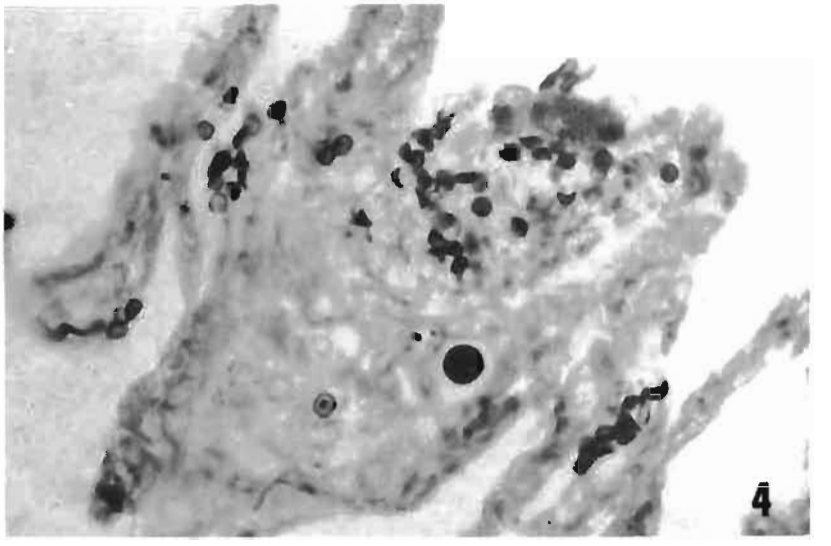


Figure 4: Pneumocystis carinii and a Criptococcus in the alveolar space stained with Grocott's silver methenamine.
400x.

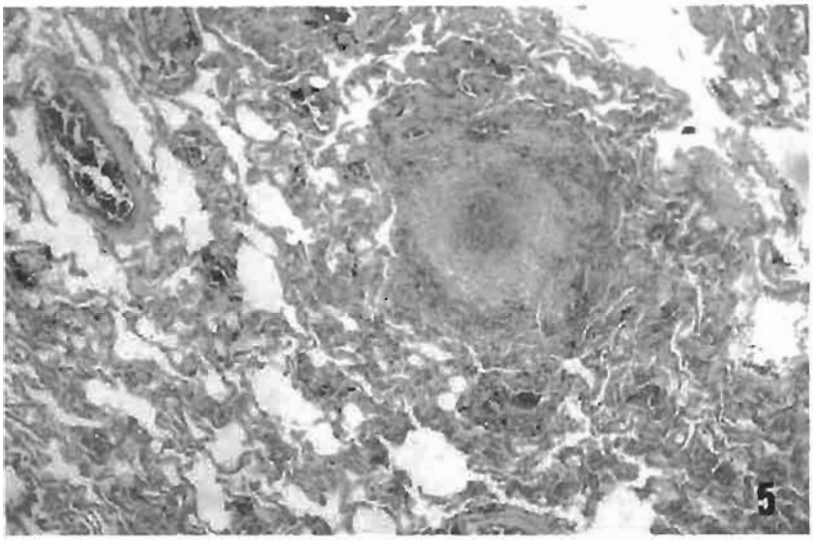


Figure 5: Nodular necrotic granulomatous lesion in the lung parenchyma induced by Mycobacteriae.
40x.

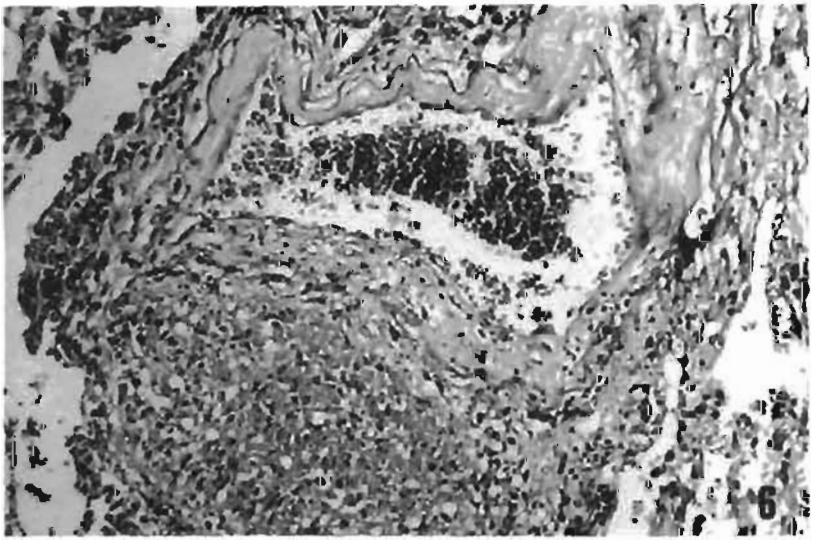


Figure 6: Necrotizing nodule with mycobacteriae in the wall of a pulmonary blood vassel. Zielh-Nielsen stain.
400x.

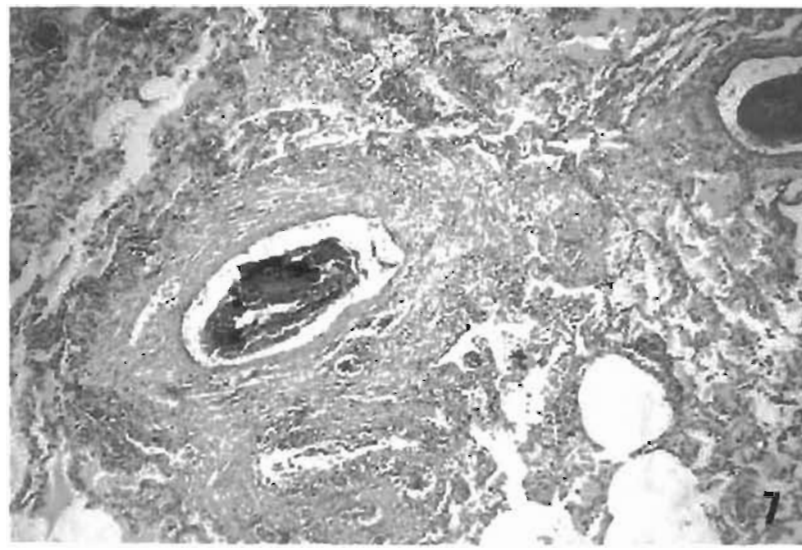


Figure 7: Perivascular and peribronchiolar proliferation of spindle cells, slits, hemosiderin and blood vassels in Kaposi's sarcoma involving the lung.
40x.

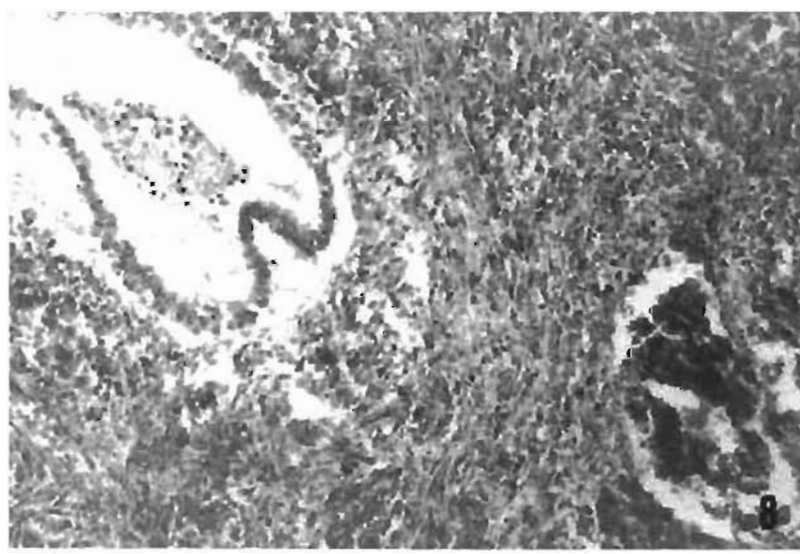


Figure 8: Red blood cells hemosiderin and Kaposi's sarcoma spindle cells in the pulmonary parenchyma.
400x.