A Rare Case of *Coniochaetta Hoffmannii* Fungus Ball in an Immunocompetent Host*

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ABSTRACT

Introduction: Fungal balls are commonly associated with the *Aspergillus* species. The hyaline hyphae is not diagnostic of a fungus ball's causative organism hence the need for fungal culture. Systemic fungal infections are rarely seen in immunocompetent persons.

Case: A 45-year-old male presented with a nine month history of nonproductive cough progressing to hemoptysis. His chest tomography with contrast revealed a mass measuring 6.5cm x 5.5cm x 6.9cm located in the left upper lobe, with a smooth lining and air crescent sign consistent with aspergilloma. Serum galactomannan assay was positive. Patient was treated medically for *Aspergillus sp* infection with voriconazole and itraconazole for six months with no response. A left upper lobectomy was done. Lung tissue biopsy and histopathologic examination showed hyphal elements with branching short lateral necks. Culture studies revealed a rare microorganism namely *Coniochaetta hoffmannii*. Post-operatively, our patient improved and was eventually discharged.

Discussion: *Coniochaetta hoffmannii* is a rare human pathogen and is only implicated in those immunocompromised. Thorough clinical investigation led to the identification of this organism. Literature review reveals scant inconclusive treatment approaches. Surgical intervention proved therapeutic for our patient.

Conclusion: Not all fungal balls are caused by *Aspergillus sp*. Culture studies remains the gold standard in identifying specific organism causing fungus balls. Rare micro-organisms such as *Coniochaeta hoffmanii*. can be isolated. Invasive fungal infection can occur in an immunocompetent host. The outcome of this study will contribute to the limited pool of information on the diagnosis and management of similar cases.

Keywords: *aspergilloma, fungus ball, coniochaeta*

INTRODUCTION

Over 300 million people are afflicted with a serious fungal infection globally and 25 million are at risk of dying.[1] *Aspergillus spp* (specifically, *A fumigatus*) is by far the most common etiologic agent and aspergilloma as its best recognized form.[2] A non-aspergillus species as a primary cause is rarely documented. It is even rarer to isolate a known non-pathogenic fungus in an immunocompetent host.

Currently, there is paucity in clinical data about *C. hoffmanii* and there are no existing standardized medical guidelines on diagnosis and treatment. This paper presents a rare case of a pulmonary fungal ball formed by a rare mold, *Coniochaeta hoffmanii* in an immunocompetent 45-year old male. The aim of this case report is to highlight the importance of a complete and cost-effective diagnostic and treatment approach to fungal balls. The use of culture studies to identify other possible causative agents of fungal balls is pivotal to provide targeted therapy.

CASE PROTOCOL

A 45-year old male, married, Filipino, from Davao del Norte, was admitted due to persistent hemoptysis.

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Seven months prior, his cough increased in frequency with dyspnea on exertion. Consultation was done and chest radiograph revealed a confluent density in the left upper lobe.

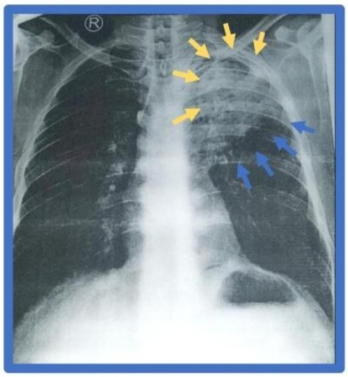

Figure 1. A chest radiograph taken at posterior-anterior view with a subtle crescentic lucency (in yellow arrows) around a confluent density in the left upper lobe (in blue arrows).

Chest computed tomography (CT) scan with contrast confirmed a huge regular oval mass measuring 6.5cm x 5.5cm x 6.9cm in the left upper lobe with a smooth lining. Dilated airways communicated with the contents of the mass. Fibrotic and bronchiectatic changes were present in the rest of the left upper lobe. Findings were consistent with aspergilloma and pulmonary bronchiectasis.

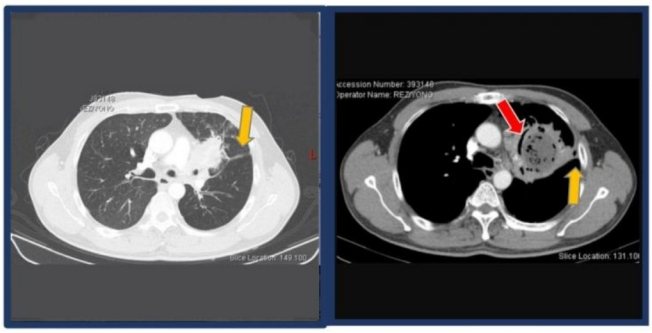

Figure 2. A sagittal chest computed tomography scan showed a 6.5cm x 5.5cm x 6.9cm mass located in the left upper lobe surrounded by air crescent sign (in red arrow). Note the presence of septation between the mass and the cavity wall (in yellow arrow).

Pulmonary malignancy was considered. However, features of the mass revealed a soft tissue attenuation and a characteristic septated separation of the mass from the cavity wall, the 'air crescent sign', hence malignancy was ruled out. Pulmonary tuberculosis was also considered however, the sputum acid fast bacilli and GeneXpert tests revealed negative results. A serum galactomannan antigen test was done to confirm chest CT scan finding of an aspergilloma and it tested positive (Appendix B).

Left upper lobectomy was advised but he opted for a trial of medical management. He was started on voriconazole 200mg tablet once a day for four months with good compliance. However, the cough persisted this time associated with weight loss.

Three months prior, voriconazole was shifted to itraconazole 130mg tablet once a day for two months. His cough increased in frequency and severity associated with episodes of blood-streaked sputum despite good compliance with medications.

He was tested for human immunodeficiency virus which turned out negative. The fasting blood glucose was normal and other inflammatory markers were also negative (See appendix).

Two weeks prior, hemoptysis increased in frequency prompting surgical referral for lung resection hence this admission.

He had pulmonary tuberculosis infection 20 years ago with six months therapy. He had no reported heredofamilial disease. He plants trees and trades lumber for 10 years. He is a 12 pack year smoker and occasional drinker.

On admission, vital signs were normal except for tachypnea of 22 breaths per minute. Pertinent physical examination findings centered on the lungs. Chest examination revealed decreased breath sounds on the left upper lung field with increased vocal and tactile fremitus.

Laboratory tests showed only a slight elevation of WBC 10.75 (normal value 5.0-10). Other laboratory tests were within normal limits. (Please see Appendix B).

He underwent intra-operative bronchoscopy and open posterolateral thoracotomy with left lung

lobectomy. The fungus ball was successfully removed encased within a capsule along with its draining lymph nodes. Histopathologic findings were consistent with aspergilloma.

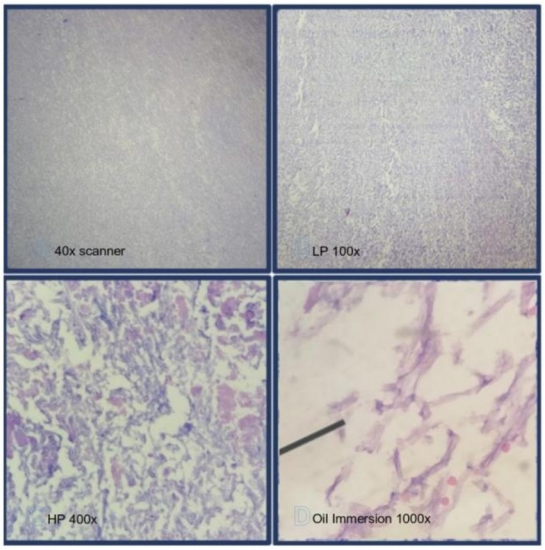

Figure 3. Histopathologic appearance of sample biopsy on hematoxylin-eosin stain showed fungus with septated hyphae (A) at scanner objective 40x, (B) at low power objective 100x, (C) at high power objective 400x, (D) and noted hypha on oil immersion 1000x (black pointer).

Post operatively, the patient developed fever and was started on cefepime 1 gram every 8 hours for secondary bacterial infection. He showed improvement and his hemoptysis disappeared.

Standard pleural fluid gram stain and culture were negative. Culture with Saboraud dextrose agar showed narrow hyaline hyphae with conidia. Small collarettes formed directly laterally on the hyphae. Identification of the organism showed *Conochaeta hoffmanii* previously known as *Lecythophora hoffmanii*.

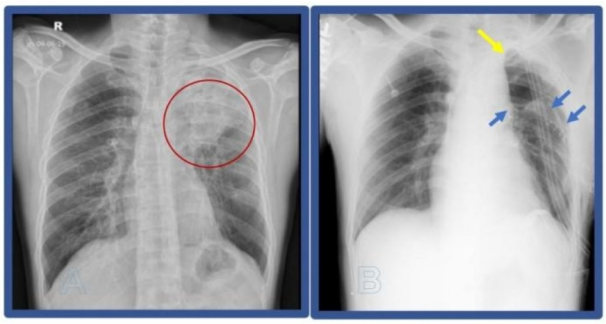

Figure 4. Frontal chest radiograph of the patient (A) Pre-surgical chest radiograph taken in antero-posterior view showing an inhomogenous opacity (in red circle) with air bronchograms see in the left upper lung field; (B): Post-surgery taken at postero-anterior view showing recent area of lucency devoid of pulmonary vascular markings with an approximate volume of 35% (in yellow line). Staple wires are seen in the left chest. Surgical clips are likewise seen in the left hilar region (in blue arrows).

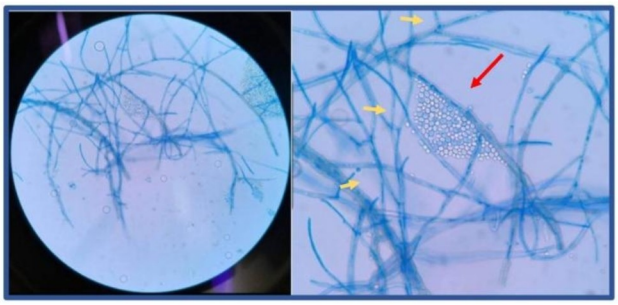

Figure 5. *Coniochaeta hoffmanii*'s conidia (in red arrow). Note presence of branching, septated, hyphae (in yellow arrow).

A normal CD4+ count, positive Tetanus Toxoid, and positive Mantoux skin test (Appendix C) indicate an intact cellular immunity and inflammatory response.

The patient gradually improved and was subsequently discharged. Cough and dyspnea resolved after one week. A repeat chest CT scan done 2 months post-operatively revealed bullae formation in the post-operative area.

DISCUSSION

The global burden of fungal infections is increasing.[3] Fungal lung disease leads to over a million deaths annually. It is the third most common fungal disease and the most serious.[4]

The diagnosis of fungal pneumonias is difficult to prove and is often made on a presumptive basis. It relies on a combination of clinical, radiologic, and microbiological factors.[5] Some fungal diseases are only recently recognized.[6] Emerging fungi previously thought to be nonpathogenic are now recognized as playing a significant role in the increased incidence of fungal diseases.[8]

A fungal ball or mycetoma consists of a rounded conglomerate of hyphae, mucus, and

debris which is contained within a thick fibrotic wall. It usually forms in approximately 15 – 25% of patients within a preexisting cavitating lung disease resulting from tuberculosis, sarcoidosis, bullous emphysema, bronchiectasis, or other conditions.[8,9] The sequelae of structural deformities impairs the clearance of the infection and allows this chronic condition to take hold.[11]

Clinical presentations of fungus ball are characteristic. Most patients are often asymptomatic. Some may present with chronic cough, malaise, and weight loss. Hemoptysis occurs in 50–80% of cases and may be massive and life-threatening.[11] One retrospective study noted a male predominance with half of the patients in their fourth decade.[12] These findings are similar with our patient who came in with cough, malaise and weight loss, he was at his 4th decade, male and with a history of pulmonary tuberculosis.

The radiological signs may be variable. Classically, mycetoma is surrounded by a crescent of air in the cavity – "ball-in-a-hole". However, this radiological appearance is non-specific and differential diagnoses includes lung abscesses, cavitary carcinomas, and ruptured hydatid cysts.[22] In chest computed tomography (CT), fungus ball is characterized by a mass with soft-tissue attenuation within a lung cavity. The mass is typically separated from the cavity wall by an airspace – the "air crescent" sign and is often associated with thickening of the wall and adjacent pleura.[24] These radiologic findings were seen in our patient.

Differential diagnoses include pulmonary malignancy, tuberculosis, lung abscess, infectious consolidations, or pulmonary hemorrhage. However, thorough clinical history, characteristic imaging findings, along with supportive serological exam (galactomannan), and a history of exposure to endemic mycotic areas, a diagnosis of mycetoma is likely. The histopathologic report supports our diagnosis but isolation and microscopic determination in cultures identify the specific causative organism.

Mycetomas are most commonly caused by *A. fumigatus hence the popular term aspergilloma*. However, it can also be caused by other molds such as *Zygomycetes* and *Fusarium*.[14] All hyphate fungi can produce fungus balls in the lungs.[18]

One diagnostic tool for detecting fungal infection is histopathological examination of affected tissues.[15] However, errors in identification of fungal pathogens often occur.[19] The histopathologic appearance of the aspergilli in tissue is similar to that observed with other hyaline molds and differentiation among genera and species usually is not possible.[16]

A positive serum galactomannan assay test may be supportive of aspergilloma. However, galactomannan is found in varying amounts in other fungi including *Penicillium, Fusarium, Altenaria* and *Histoplasma.* Hence, a positive result on galactomannan test is not specific to *Aspergillus*.

The gold standard for the diagnosis of the causative agent is culture and isolation of the fungi.[25] Histologic misidentification of fungal forms may occur. Four out 10 cases misclassified on histologic examination were interpreted as *Aspergillus*. However thru cultures, these 4 cases grew *Rhizopus*, *Fusarium*, and *Scedosporium apiospermum*.

Our fungal cultures grew *Coniochaeta hoffmanii* (formerly *Lecythophora),* a mold and a plant pathogen. It is locally known as the "soft rot fungus".[26] *C. hoffmanii* is characterized by its hyaline hyphae and intercalary phialides with very short lateral necks, periclinal wall thickening, and flaring collarettes.[28] Penetration of fine hyphae through wood cell walls initiates the process of colonization and subsequent decay.[27] Most pulmonary fungal infections occur after inhalation of fungi when their natural habitat is disturbed.[33] Once in the lung alveoli, the fungus is engulfed by macrophages and other cells involved in the primary immune response. Our patient works in the timber and lumber industry. Prolonged exposure may be a risk factor for *C. hoffmanii* infection.

An ubiquitous organism usually becomes pathogenic to humans with immunodeficient system or state. Commonly, infections are severe, complicated, in multiple sites, resistant to treatments, with unusual organism, and clustering

of cases. *C. hoffmannii* has been isolated as the cause of a prosthetic valve endocarditis in a diabetic patient.[29] Other clinical presentations of *C. hoffmanii* include a case report of septic shock in an 18 year old patient on chronic mechanical ventilator due to mitochondrial encephalopathy, chronic sinusitis in a patient diagnosed with AIDS, and a fungal peritonitis in a patient on continuous peritoneal dialysis.[30,31,32] Our patient did not have any primary or secondary immunodeficient state based on work ups done. There were no similar cases in the family precluding the diagnosis of familial immunodeficiency. Further studies regarding this microorganism's interplay with human pathogenesis remains to be determined.

Treatment options for a fungal ball include surgical or medical approach. Voriconazole is now considered the drug of choice as triazoles are preferred agents for treatment and prevention of invasive aspergillosis.[39,40] *C. hoffmanii* has been proven to be resistant to multiple antifungal agents. A study reported that it was resistant to all antifungal agents tested with no zone recorded for amphotericin B, ketoconazole, itraconazole, fluconazole, or flucytosine. This could explain the failure of medical management in our case despite use of two anti-fungal medications.

A local study reports that surgical resection of aspergilloma is effective in preventing hemoptysis and is recommended in symptomatic patients.[35] The Centers for Disease Control and Prevention strongly recommends resection for patients with symptoms, especially with significant hemoptysis and with a single aspergilloma if there are no contraindications. Peri-/postoperative antifungal therapy is not routinely required. However, if the risk of surgical spillage of the aspergilloma is moderate (related to location and morphology of the cavity), antifungal therapy with voriconazole (or another mold-active azole) or an echinocandin is suggested.[36] Despite the risks, surgery offers three potential benefits: control of symptoms, prevention of hemoptysis, and prolongation of life.[38] A study reported an in-hospital mortality of 4.1%. Postoperative complications occurred in 63.3% of patients. These were prolonged air leak (26.3%), arrhythmias (20.4%), residual pneumothorax (16.3%), respiratory failure (14.3%), atelectasis (12.3%), and bleeding (12.3%). Surgical treatment of pulmonary aspergilloma should be restricted to symptomatic patients in whom lobectomy can be performed due to the increased risk of complications.[37]

Patients who underwent surgical intervention have good long term prognosis. One case reported successful nonrecurrence and improvement sixteen months post operation.[41] Patients treated by pulmonary resection lived longer than patients treated medically in a ten year survival study.[42] Our patient subsequently had a lobectomy after failure of initial anti-fungal therapy.

SUMMARY

A 45-year-old male presented with a nine-month history of nonproductive cough progressing to hemoptysis. He was diagnosed with aspergilloma based on chest tomography with contrast which revealed a mass measuring 6.5cm x 5.5cm x 6.9cm located in the left upper lobe, with a smooth lining and an air crescent sign. Serum galactomannan assay was positive and he was treated medically for *Aspergillus spp* infection with voriconazole and itraconazole for six months. However, lack of clinical response warranted further investigation. His worsening condition prompted referral to a surgeon and lung lobectomy with removal of the aspergilloma was done. Lung tissue biopsy and histopathologic examination findings of hyphae with branching on short lateral necks was seen indicating a growth of a mold which was still consistent with initial impression of *Apergilloma*. However, culture studies which is the gold standard for fungal diagnosis, revealed a rare mold namely *Coniochaeta hoffmanii* as the specific causative organism. Post-operatively, his condition gradually improved and was eventually discharged.

No epidemiological data are available to date, although sporadic cases have been reported however treatment outcomes have been inconclusive.

On follow up, there were no recurrence of complaints although close monitoring is still advised as no treatment guidelines and prognostic data have yet been published.

CONCLUSION

This study shows that not all fungus balls are caused by Aspergillosis. This case demonstrates the importance of culture studies to identify other possible causes of fungal balls.

In this case report we were able to isolate *Coniochaeta hoffmanii* which is an extremely rare microorganism, with limited data on its pathogenesis and clinical manifestations as a human pathogen. The prognosis of this condition is yet to be known since this is the first ever documented case of *C. hoffmannii* fungal ball in an immunocompetent individual. Treatment is still surgical with possible pre-/post anti-fungal treatment. Close monitoring and surveillance is highly recommended.

The diagnosis, management, and outcomes of this study will contribute to the evolving knowledge of this disease. It will help to establish proper protocols in handling similar cases.

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APPENDIX A
Complete Blood Count

Values	1st Hospital Stay	7th Hospital Stay	10th Hospital Stay	12th Hospital Stay	16th Hospital Stay
Hgb (g/L) *135-175*	131	88	116	92	115
Hct *0.40-0.52*	0.36	0.25	0.34	0.28	0.34
WBC (x 10^3/uL) *5.0-10.0*	10.7 H	10.6 H	20 H	10.45 H	12.11 H
Neutrophil (%) *55-75*	70	79 H	96 H	82 H	83 H
Lymphocytes (%) *20-35*	23	15	2	13 L	11 L
Monocytes (%) *2-10*	5	4	2	3	4
Eosinophil (%) *1-8*	2	2	0	2	2
Basophil (%)	0	0	0	0	0
Platelet Count (x 10^3/uL) *150-400*	435 H	357	409	305	390

Blood Chemistry

Sodium (mmol/L) *136-144*	138		136		
Potassium (mmol/L) *3.6-5.1*	3.7		4.10		
Creatinine (umol/L) *57-113*	77.2		75.39		

Surgical Pathology Report

Specimen	Result
Lymph Node, Aspergilloma, left lobe with capsule	Pulmonary Aspergillosis with lung abscess, anthracosis, lymphoid hyperplasia

APPENDIX B
Complete Blood Count & Electrolytes

	Smear Microscopy		Xpert MTB/RIF
Specimen	1	2	
Visual Appearance			Mucosalivary
Reading			MTB not detected
Lab Diagnosis			

Microbiology
Serum Galactomannan

Specimen No: **********	
Specimen	Serum
Serum Galactomannan	POSITIVE
Remarks	Patent Index = 2.26 Reference Index: POSITIVE = greater than or equal to 0.50 NEGATIVE = less than 0.50

HIV Rapid

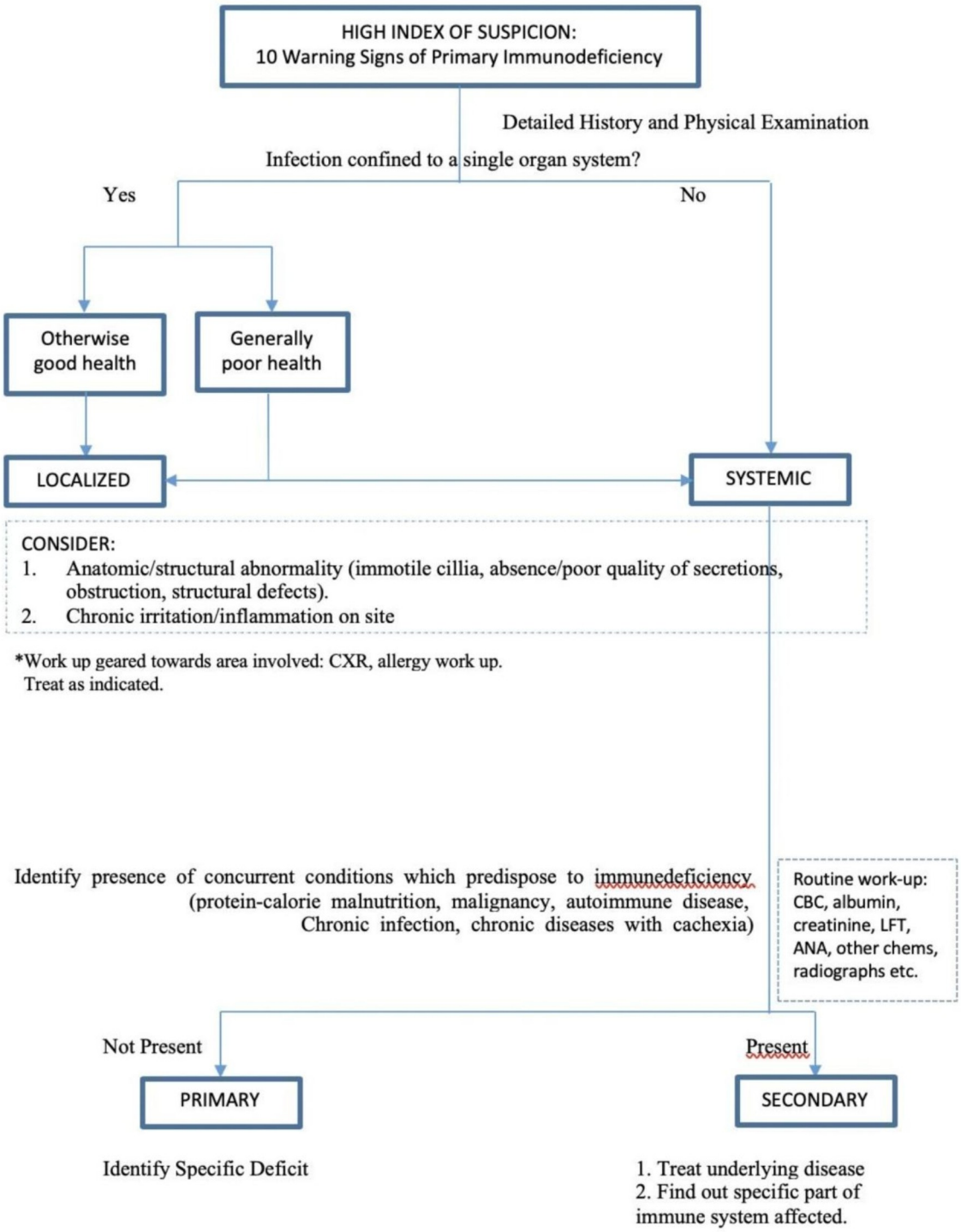
Specimen	Result
Blood	NONREACTIVE

APPENDIX C

CD4 Count

Analysis: Immune Panel CD4 Percentage and Absolute CD4 Count	
Results:	
CD4+ (cells/ul)	881
CD4%	44.92
Hemoglobin (g/dl)	14.40

APPENDIX D


HIGH INDEX OF SUSPICION:
10 Warning Signs of Primary Immunodeficiency
Detailed History and Physical Examination
Infection confined to a single organ system?
Yes
No
Otherwise good health
Generally poor health
LOCALIZED
SYSTEMIC
CONSIDER:
1. Anatomic/structural abnormality (immotile cillia, absence/poor quality of secretions, obstruction, structural defects).
2. Chronic irritation/inflammation on site
*Work up geared towards area involved: CXR, allergy work up.
Treat as indicated.
Identify presence of concurrent conditions which predispose to immunedeficiency (protein-calorie malnutrition, malignancy, autoimmune disease, Chronic infection, chronic diseases with cachexia)
Routine work-up: CBC, albumin, creatinine, LFT, ANA, other chems, radiographs etc.
Not Present
Present
PRIMARY
SECONDARY
Identify Specific Deficit
1. Treat underlying disease
2. Find out specific part of immune system affected.

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APPENDIX A
Complete Blood Count

Values	1st Hospital Stay	7th Hospital Stay	10th Hospital Stay	12th Hospital Stay	16th Hospital Stay
Hgb (g/L) *135-175*	131	88	116	92	115
Hct *0.40-0.52*	0.36	0.25	0.34	0.28	0.34
WBC (x 10^3/uL) *5.0-10.0*	10.7 H	10.6 H	20 H	10.45 H	12.11 H
Neutrophil (%) *55-75*	70	79 H	96 H	82 H	83 H
Lymphocytes (%) *20-35*	23	15	2	13 L	11 L
Monocytes (%) *2-10*	5	4	2	3	4
Eosinophil (%) *1-8*	2	2	0	2	2
Basophil (%)	0	0	0	0	0
Platelet Count (x 10^3/uL) *150-400*	435 H	357	409	305	390

Blood Chemistry

Sodium (mmol/L) *136-144*	138		136		
Potassium (mmol/L) *3.6-5.1*	3.7		4.10		
Creatinine (umol/L) *57-113*	77.2		75.39		

Surgical Pathology Report

Specimen	Result
Lymph Node, Aspergilloma, left lobe with capsule	Pulmonary Aspergillosis with lung abscess, anthracosis, lymphoid hyperplasia

APPENDIX B
Complete Blood Count & Electrolytes

	Smear Microscopy		Xpert MTB/RIF
Specimen	1	2	
Visual Appearance			Mucosalivary
Reading			MTB not detected
Lab Diagnosis			

Microbiology
Serum Galactomannan

Specimen No: **********	
Specimen	Serum
Serum Galactomannan	POSITIVE
Remarks	Patent Index = 2.26 Reference Index: POSITIVE = greater than or equal to 0.50 NEGATIVE = less than 0.50

HIV Rapid

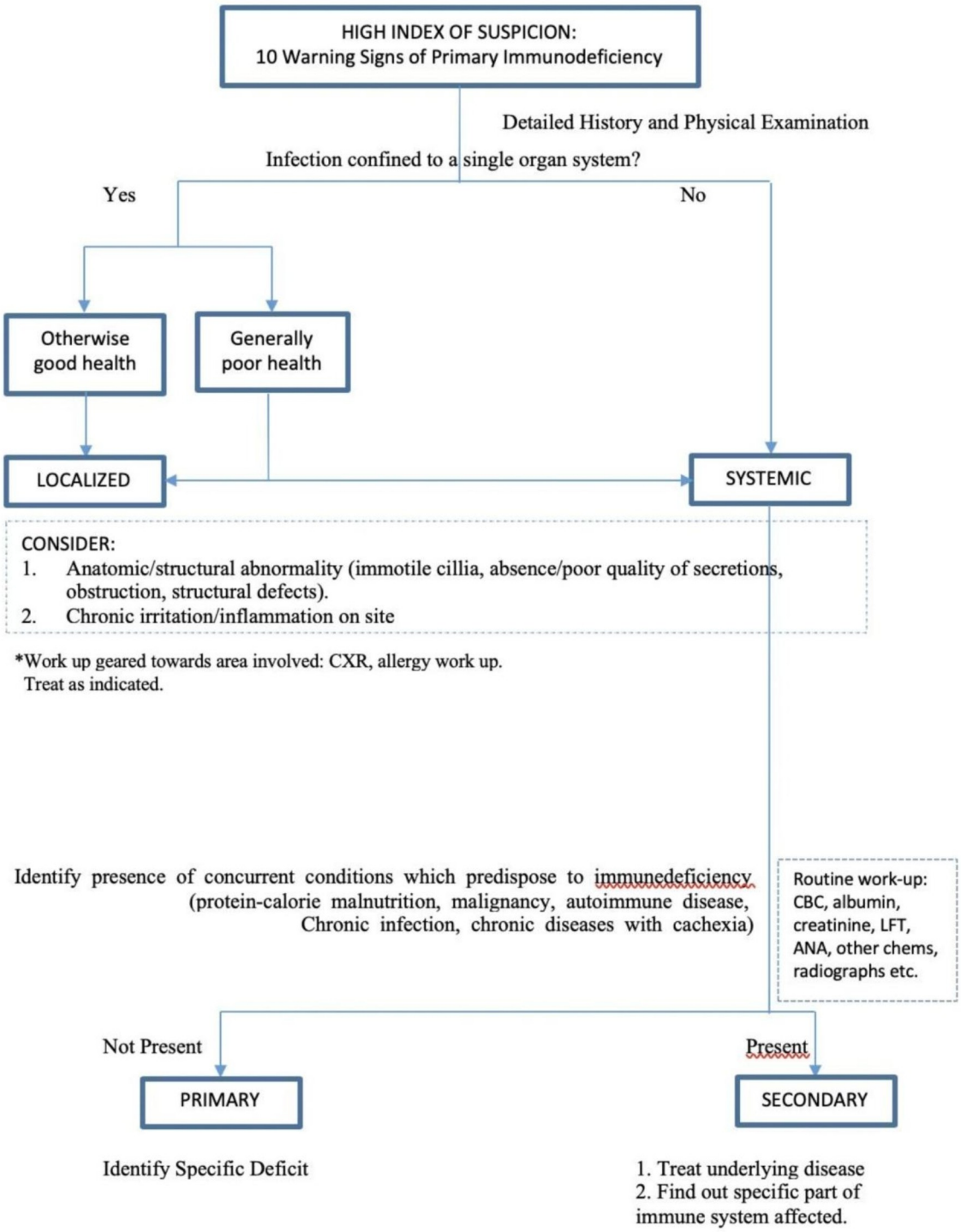
Specimen	Result
Blood	NONREACTIVE

APPENDIX C

CD4 Count

Analysis: Immune Panel CD4 Percentage and Absolute CD4 Count	
Results:	
CD4+ (cells/ul)	881
CD4%	44.92
Hemoglobin (g/dl)	14.40

APPENDIX D


HIGH INDEX OF SUSPICION:
10 Warning Signs of Primary Immunodeficiency
Detailed History and Physical Examination
Infection confined to a single organ system?
Yes
No
Otherwise good health
Generally poor health
LOCALIZED
SYSTEMIC
CONSIDER:
1. Anatomic/structural abnormality (immotile cillia, absence/poor quality of secretions, obstruction, structural defects).
2. Chronic irritation/inflammation on site
*Work up geared towards area involved: CXR, allergy work up.
Treat as indicated.
Identify presence of concurrent conditions which predispose to immunedeficiency (protein-calorie malnutrition, malignancy, autoimmune disease, Chronic infection, chronic diseases with cachexia)
Routine work-up: CBC, albumin, creatinine, LFT, ANA, other chems, radiographs etc.
Not Present
Present
PRIMARY
SECONDARY
Identify Specific Deficit
1. Treat underlying disease
2. Find out specific part of immune system affected.