Nontuberculous Mycobacterial Infections in King Chulalongkorn Memorial Hospital

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Background: *Nontuberculous mycobacteria (NTM) can cause infections in both human immunodeficiency virus (HIV)-infected and HIV-noninfected patients. The incidence of NTM infections has been increasing since the acquired immunodeficiency syndrome (AIDS) epidemics. However, the epidemiologic and clinical data of NTM infections in Thailand are limited.*
Objective: *Determine the epidemiology, clinical manifestations, treatment, and outcome of NTM infections in King Chulalongkorn Memorial Hospital from January 2000 to December 2003.*
Material and Method: *One hundred and fourteen patients had positive NTM cultures; however, complete medical records were available in only 103 (90.3%) patients.*
Results: *There were 71 (68.9%) HIV-infected patients, and 38 (87%) of them had the CD4 counts of < 200 cells/ L (range 4-360). Among HIV-infected patients, the most common previous opportunistic infections included tuberculosis (36.6%), Pneumocystis jirovecii pneumonia (25.3%), cryptococcal meningitis (15.5%), penicilliosis (5.6%), and cytomegalovirus infection (5.6%). Most patients presented with prolonged fever (67%), chronic cough (54.4%), lymphadenopathy (52.4%), weight loss (50.5%), or chronic diarrhea (31%). The clinical manifestations included disseminated (17.4%) and localized (82.6%) infections. The localized infection included pulmonary infection (82.3%), followed by gastrointestinal infection (34.1%), skin infection (12.9%), lymphadenitis (8.2%), genitourinary tract infection (2.4%), central nervous system infection (2.4%), and keratitis (1.2%). Mycobacterium avium complex (MAC) was the predominant species (48.5%), followed by M. kansasii (19.4%), and rapidly growing mycobacteria (16.4%). Diffuse reticular infiltration was most commonly observed on chest radiography (53.4%). Abnormal laboratory findings included anemia (48.5%), hyponatremia (42.7%), and elevated alkaline phosphatase (39.8%). The overall mortality rate was 34.8% (45.9% and 11.1% in HIV- and HIV-noninfected patients).*
Conclusion: *A diagnosis of NTM infection requires a high index of suspicion in patients especially with AIDS or immunocompromised status who present with prolonged fever, with or without organ-specific symptoms and signs. Therefore, clinical specimens must be sent for mycobacterial cultures for a definite diagnosis, a determination of the species of NTM, and an appropriate management. In addition to four standard antituberculous drugs, clarithromycin should be added for the treatment of MAC in patients with AIDS who presented with disseminated opportunistic infections before obtaining the microbiologic results.*

Keywords: *Nontuberculous mycobacteria (NTM), HIV, AIDS, Mycobacterium avium complex, Mycobacterium kansasii, Rapidly growing mycobacteria*

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Nontuberculous mycobacteria (NTM) include *Mycobacterium* species that are not *M. tuberculosis* complex (*M. tuberculosis*, *M. africanum*, *M. bovis*, *M. microti*, and *M. canettii*) and *M. leprae*. Earlier, the organisms carried the epithet "atypical mycobacteria" or "mycobacteria other than tuberculosis (MOTT)"[1,2].

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NTM are generally free-living organisms that are ubiquitous in the environment. They can be recovered from water, soil, aerosols, domestic, and wild animals, milk, and foods[3,6]. NTM can cause a wide variety of infections including pulmonary, lymphatic, skin and soft tissue, skeletal, and catheter-related infections[7]. Before the acquired immunodeficiency syndrome (AIDS) epidemics, the pulmonary infections caused by NTM were found predominantly in males in the sixth decade of life. Most patients have predisposing lung conditions or work under conditions where they were exposed to contaminated dusts. The major pulmonary pathogens include *M. kansasii, M. avium* complex (MAC), and rapidly growing mycobacteria (RGM)[8]. *M. scrofulaceum* is found to be the causative agent of cervical lymphadenitis in children[2]. *M. marinum* can cause skin and soft tissue infections, mostly associated with wound or exposed to aquarium[2,9].

Since the AIDS epidemics, the picture of NTM infections has been radically changed. 25-50% of patients with AIDS in the United States and Europe are infected with NTM, mostly by MAC[10-20].

In Thailand, the first patient with NTM infection caused by *M. kansasii* was reported in 1968[21]. Since then, there have been several reports of NTM infections caused by *M. scrofulaceum*, *M. gordonae*, MAC, *M. chelonae, M. fortuitum, M. szulgai*, *M. smegmatis*, *M. marinum*, and other RGM[22-27].

The epidemiology of NTM infections has been changed since the first case of HIV infection was reported in Thailand. The incidence of MAC infections has been increasing in parallel with the increased incidence of patients with AIDS[28-32]. Furthermore, an improved technology in mycobacterial culture with the automated commercial system in fluid media and molecular microbiology has improved the yield and has reduced the time for identification of NTM. This also contributes to the changing patterns of epidemiology of NTM infections. In King Chulalongkorn Memorial Hospital (KCMH), there was only one study of NTM infections by Phowthongkum et al who described nine patients with RGM infections from 1997 to 2003[33].

The present study, thus, aimed to determine the epidemiology, clinical manifestations, treatment, and outcome in the patients with NTM infections in KCMH, a medical school, Bangkok, Thailand from January 2000 to December 2003.

Material and Method

Patients

A retrospective review of the records of the patients with positive culture of NTM from the Department of Clinical Microbiology, Chulalongkorn University, Bangkok was carried out from January 2000 to December 2003. All available medical records were then carefully examined for epidemiology, clinical manifestations, treatment, and outcome.

A diagnosis of NTM pulmonary infections was based on the American Thoracic Society (ATS) criteria[1,34]. Other NTM infections were defined based on the compatible clinical features accompanying with positive culture for the NTM from the specimens obtained from the involved organ in the absence of any other isolated pathogens. Disseminated infection was defined with the presence of one of the following: 1) multiple sites of cutaneous abscesses, 2) involvement of two or more noncontiguous extrapulmonary sites, 3) positive blood or bone marrow culture, or 4) clinical evidence of deep infection[26].

Microbiology

Blood culture was performed using the MB/BacT mycobacteria detection system[35]. The inoculated bottles were placed in the CO_2 incubator at $37 \pm 2^{o}C$, where they were continuously monitored for the growth of mycobacteria by the MB/BacT mycobacteria detection system. Other clinical specimens were inoculated on Lowenstein-Jensen media according to the standard procedures[36]. The isolates were identified by the standard methods as previously described[36,37].

Statistic analysis

The statistical analyses were performed using the SPSS software, version 13.0, for Microsoft Windows.

Results

There were 114 patients with NTM infections during the study period, but complete medical records were available in 103 patients.

Epidemiology

The demographic characteristics and associated conditions of all patients are shown in Table 1 and 2. There were 59 males and 44 females with the median age of 33 years (range 8-75 years). There were 71 (68.9%), 20 (19.4%) and 12 (11.7%) patients with, without, and unknown HIV infection, respectively. Of 32 patients without and unknown HIV infection, 15 (47%) had preexisting illness. Of 44 HIV-infected patients with known CD4 cell counts, there were 27 (61.4%) and 38 (87%) patients with CD4 cell counts of

Table 1. The demographic data of all 103 patients with nontuberculous mycobacteria (NTM) infections, according to the HIV serostatus

	HIV serostatus (No.)			Total No. (percent)
	Yes	No	Unknown	
Age (years)				
< 30	25	6	5	36 (35.0)
30-39	34	4	4	42 (40.8)
40-49	7	4	2	13 (12.6)
50-59	4	4	1	9 (8.7)
≥ 60	1	2	0	3 (2.9)
Gender				
Male	41	11	7	59 (57.3)
Female	30	9	5	44 (42.7)
Marital status				
Single	32	6	6	44 (42.7)
Married	39	14	6	59 (57.3)
Occupation				
Wage earner	51	12	8	71 (68.9)
Merchant	6	0	1	7 (6.8)
Government employee	6	2	0	8 (7.8)
Housewife	5	4	0	9 (8.7)
Student	3	2	3	8 (7.8)
Total (%)	71 (68.9%)	20 (19.4%)	12 (11.7%)	103 (100.0)

Table 2. The preexisting illness in 103 patients with NTM infections

Underlying disease	No. (percent)
HIV	71 (68.9)
CMI defect	
NHL	1 (1)
AML	1 (1)
Hepatoma	1 (1)
RA	1 (1)
Chronic organ failure	
DM	3 (2.9)
Cirrhosis	2 (1.9)
COPD	1 (1)
Cardiomyopathy	1 (1)
Other	
Hydrocephalus	2 (1.9)
HT	1 (1)
Dyslipidemia	1 (1)
None	17 (16.5)
Total	103 (100)

HIV: human immunodeficiency virus, DM: diabetes mellitus, HT: hypertension, RA: rheumatoid arthritis, NHL: non-Hodgkin lymphoma, COPD: chronic obstructive pulmonary disease, AML: acute myeloid leukemia, CMI: cell-mediated immunity

Table 3. Previous opportunistic infections in 71 HIV-infected patients with NTM infections

Previous opportunistic infection	No. (percent)
Tuberculosis	26 (36.6)
Pneumocystis jirovecii pneumonia	18 (25.3)
Cryptococcal meningitis	11 (15.5)
Penicilliosis	4 (5.6)
Cytomegalovirus infection	4 (5.6)
Toxoplasmosis	2 (2.8)
Histoplasmosis	1 (1.4)
Total	66 (92.9)

< 50 and < 200 cells/ L, respectively. 66 (92.9%) HIV-infected patients had previous opportunistic infections including tuberculosis (26, 36.6%), *Pneumocystis jirovecii* pneumonia (18, 25.3%), cryptococcal meningitis (11, 15.5%), penicilliosis (4, 5.6%), and cytomegalovirus infection (4, 5.6%) (Table 3).

Clinical manifestations

Most patients presented with prolonged fever (69, 67%), chronic cough (56, 54.4%), lymphadenopathy (54, 52.4%), weight loss (52, 50.5%), or

Table 4. The clinical manifestations of all 103 patients with NTM infections, according to the HIV serostatus

Clinical manifestations	HIV serostatus (No.)			Total No. (percent)
	Yes	No	Unknown	
Disseminated infection	16	1	1	18 (17.4)
MAC	15	1	1	17 (94.4)
M. kansasii	1	-	-	1 (5.6)
Localized infection	55	19	11	85 (82.5)
Pulmonary	54	12	4	70 (82.3)
MAC	29	3	-	32 (45.7)
M. kansasii	10	3	4	17 (24.3)
RGM	6	2	-	8 (11.4)
Other	9	4	-	13 (61.4)
Gastrointestinal tract infection	27	1	1	29 (28.1)
MAC	15	-	1	16 (57.1)
M. kansasii	4	-	1	5 (17.2)
RGM	2	1	-	3 (10.3)
Other	5	-	-	5 (17.2)
Skin	5	5	1	11 (12.9)
MAC	3	-	-	3 (27.3)
RGM	1	2	2	5 (45.5)
M. kansasii	1	1	-	2 (18.2)
Other	-	1	-	1 (9.1)
Lymphadenitis	2	2	3	7 (8.2)
M. kansasii	1	1	1	3 (42.8)
MAC	1	-	1	2 (28.6)
RGM	-	-	1	1 (14.3)
Other	-	1	1	1 (14.3)
Genitourinary tract infection	-	2	-	2 (2.4)
RGM	-	1	-	1 (50)
MAC	-	1	-	1 (50)
Central nervous system infection	-	2	-	2 (2.4)
RGM	-	2	-	2 (100)
Keratitis	-	1	-	1 (1.2)
RGM	-	1	-	1 (100)

MAC: *Mycobacterium avium* complex, RGM: rapidly growing mycobacteria

chronic diarrhea (32, 31%). The clinical manifestations included disseminated (18, 17.4%) and localized (85, 82.6%) infections (Table 4). Of 18 patients with disseminated infection, MAC was the predominant species (17, 94.4%), followed by *M. kansasii* (1, 5.6%). The localized infection (85, 82.6%) included pulmonary infection (70, 82.3%), gastrointestinal infection (29, 34.1%), skin infection (11, 12.9%), lymphadenitis (7, 8.2%), genitourinary tract infection (2, 2.4%), central nervous system infection (2, 2.4%), and keratitis (1, 1.2%) (Table 4). Of 70 pulmonary infections, there were MAC (32, 45.7%), *M. kansasii* (17, 24.3%), RGM (8, 11.4%), *M. flavescence* (4, 5.7%), *M. scrofulaceum* (3, 4.3%), *M. szulgai* (1, 1.4%), *M. gordonae* (1, 1.4%), and unspecified species (4, 5.7%). Of 29 gastrointestinal infections, there were MAC (16, 57.1%), *M. kansasii* (5, 17.2%), RGM (3, 11%), *M. flavescence* (1, 3.4%), *M. scrofulaceum* (1, 3.4%), and unspecified species (3, 10.3%). Of 11 skin infections, there were MAC (3, 27.2%), RGM (5, 45.5%), *M. kansasii* (2, 18.1%), and *M. szulgai* (1, 9.1%). Of seven lymphadenitis, there were *M. kansasii* (3, 42.8%), MAC (2, 28.6%), RGM (1, 14.3%), and *M. flavescence* (1, 14.3%).

Microbiology

The frequency of NTM infections in each

Table 5. The clinical specimens recovered for 103 NTM

Specimen	No. (percent)
Sputum	52 (50.5)
Blood	12 (11.7)
Stool	12 (11.7)
Tissue	11 (10.7)
Pus	8 (7.8)
Bone marrow	3 (2.9)
Cerebrospinal fluid	2 (1.9)
Ascitic fluid	2 (1.9)
Urine	1 (1)
Total	103 (100)

year from 2000 to 2003 varied from 26, 31, 27, and 19 patients, respectively. The clinical specimens recovered for NTM included sputum (52, 50.5%), blood (12, 11.7%), stool (12, 11.7%), pus (8, 7.8%), and tissue (11, 10.7%) (Table 5). The distribution of the species of NTM is shown in Table 6. MAC was the predominant species (50, 48.5% isolates), followed by *M. kansasii* (20, 19.4%), RGM (17, 16.4%), and *M. flavescence* (5, 4.9%). Of 20 HIV-noninfected patients, there were *M. kansasii* (5, 25%) and RGM (5, 25%), followed by MAC (4, 20%), and *M. szulgai* (2, 10%). Of 71 HIV-infected patients, there were MAC (44, 62%), followed by *M. kansasii* (11, 15.5%), RGM (6, 8.4%), and *M. flavescence* (4, 5.6%).

Table 6. The distribution of the species of 103 NTM, according to the HIV serostatus

Species of NTM	HIV serostatus No. (percent)			No. (percent)
	Yes	No	Unknown	
MAC	44 (61.9)	4 (20)	2 (16.6)	50 (48.5)
M. kansasii	11 (15.5)	5 (25)	4 (33.3)	20 (19.4)
RGM	6 (8.4)	5 (25)	6	17 (16.4)
M. flavescence	4 (5.6)	1 (5)	0	5 (4.9)
M. scrofulaceum	2 (2.8)	1 (5)	0	3 (2.9)
M. szulgai	0	2 (10)	0	2 (1.9)
M. gordonae	0	1 (5)	0	1 (1)
Unspecified species	4 (5.6)	1 (5)	0	5 (4.9)
Total	71 (68.9)	20 (27.4)	12 (11.6)	103 (100)

MAC: *Mycobacterium avium* complex, RGM: rapidly growing mycobacteria including *M. fortuitum, M. chelonae,* and *M. abscessus*

Table 7. The pattern of infiltration on chest radiography of 103 NTM infections, according to the HIV serostatus

Pattern of infiltration	HIV serostatus (No.)			Total No. (percent)*
	Yes	No	Unknown	
Diffuse reticular infiltration	41	8	6	55 (60.4)
Alveolar infiltration	13	3	0	16 (17.6)
Normal	13	3	1	17 (18.7)
Bronchiectasis	2	0	0	2 (2.2)
Pleural effusion	0	1	0	1 (1.1)
No available chest radiography	2	5	5	12
Total	71	20	12	103

* The patients with no available chest radiography were not included in the calculation of percentage

Other laboratory data

Diffuse reticular (55, 60.4%) infiltrations were most commonly observed on chest radiography (Table 7). Of 55 diffuse reticular infiltrations, there were MAC (24, 43.6%), *M. kansasii* (12, 21.8%), RGM (10, 8.2%), *M. scrofulaceum* (3, 5.4%), *M. flavescence* (2, 3.6%), *M. szulgai* (1, 1.8%), and unspecified species (3, 5.4%). Of 76 alveolar infiltrations, there were MAC (11, 68.6%), *M. kansasii* (2, 12.5%), RGM (1, 6.3%), *M. flavescence* (1, 6.3%), and *M. gordonae* (1, 6.3%). Other abnormal laboratory findings included anemia (50, 48.5%), hyponatremia (44, 42.7%), and elevated alkaline phosphatase (41, 39.8%).

Table 8. Initial antimycobacterial regimens for treatment of 103 NTM infections

Drug	Patients No. (percent)
No treatment	23 (22.3)
HRZE	31 (30.1)
HRZEK	21 (20.4)
HRZEKO	11 (10.7)
HREOS	3 (2.9)
HRZEO	3 (2.9)
HRZESC	2 (1.9)
HR	2 (1.9)
REKA	2 (1.9)
HRE	2 (1.9)
Minocycline	2 (1.9)
KC	1 (1)
HREK	1 (1)
Total	103 (100)

H: isoniazid, R: rifampin, Z: pyrazinamide, E: ethambutol, K: clarithromycin, O: ofloxacin, C: ciprofloxacin, A: amikacin, S: streptomycin

Treatment and outcome

Twenty-three (22.3%) patients did not receive antimycobacterial agents (Table 8) because they were during the investigation period, died, or lost to follow-up before obtaining the culture results (Table 9). Most patients were empirically treated as tuberculosis (patients with, without, and unknown HIV infection) or tuberculosis and MAC (HIV-infected patients) due to positive acid-fast-bacilli (AFB) staining of clinical specimens or compatible clinical features, and eventually the regimen was modified after obtaining the species identification of NTM and the susceptibility results. The overall mortality was 34.8% (45.9% and 11.1% in HIV-infected and HIV-noninfected patients). The highest mortality rate was observed in infections caused by MAC (18, 36%), followed by *M. kansasii* (6, 30%), RGM (2, 11.8%), *M. flavescence* (1, 20%), and unspecified species (4, 80%). These rates of disseminated, pulmonary, and gastrointestinal infection were 22.2%, 32.9%, and 51.7%, respectively.

All data of each patient are summarized in Table 10.

Discussion

From January 2000 to December 2003, 103 patients with NTM infections had complete medical records. Most patients had associated HIV infection or other immunocompromised condition. In the present study, there were 17 (16.5%) patients without preexisting condition. Surprisingly, only one case with pulmonary infection by MAC had preexisting chronic obstructive pulmonary disease (COPD). This is in contrast to the literature. It describes the most important

Table 9. The outcome after discharging from the hospital in 103 patients with NTM infections

Outcome	HIV serostatus (No.)			Total No. (percent)*
	Yes	No	Unknown	
Improved	33	16	9	58 (65.2)
Dead	28	2	1	31 (34.8)
Referred	10	2	2	14
Total No. (percent)*	71 (68.9)	20 (19.4)	12 (11.6)	103

* The referred patients were not included in the calculation of percentage

Table 10. A summary of 103 patients with NTM infections

No.	Sex	Age (yrs)	Year	Associated condition	CD4 (cells / L)	Species	Infection	Specimen	Chest X-ray	Treatment regimen	Outcome	FU
1	F	24	2000	No	NA	*M. fortuitum*	Skin	Tissue	Normal	Minocycline	Improved	FU
2	M	31	2000	HIV, TB, Toxoplasmosis	30	MAC	Disseminated, GI	Blood	Normal	HRZEK	Referred	Loss
3	M	35	2000	HIV, PCP	NA	MAC	Disseminated, lung	Blood	Reticular	No	Improved	Loss
4	F	42	2000	No	NA	*M. kansasii*	Lung	Sputum	Reticular	HRE	Improved	FU
5	M	33	2000	HIV, CMV	4	MAC	Disseminated, skin	Blood	Normal	HRZEK	Improved	FU
6	M	34	2000	HIV, CMV	16	MAC	Disseminated, GI	Stool	Reticular	HRZEK	Dead	Loss
7	F	57	2000	HIV, CMV, VZV	24	MAC	Disseminated, GI	Blood	Bronchiectasis	HRZE	Improved	Loss
8	M	34	2000	HIV, CMV	NA	MAC	Disseminated, lung	Blood	Reticular	No	Referred	Loss
9	M	21	2000	HIV	10	MAC	Disseminated, lung	Blood	Reticular	No	Improved	Loss
10	F	39	2000	NHL	NA	MAC	Disseminated, lung, GI	BM	Reticular	HRZE	Referred	Loss
11	F	15	2000	HIV, VZV, HT, DYS	NA	MAC	Disseminated	Blood	Normal	No	Improved	Loss
12	F	23	2000	No	NA	MAC	Lymph node	Tissue	Reticular	HRZESC	Referred	Loss
13	M	47	2000	HIV, TB	20	MAC	Disseminated, lung	Blood	Reticular	HRZE	Improved	Loss
14	M	31	2000	HIV, VZV, TB	9	MAC	Disseminated, lung, skin	Blood	Normal	HRZEK	Dead	Loss
15	F	26	2000	HIV, VZV, PCP	360	MAC	Lung	Sputum	Alveolar	No	Dead	Loss
16	F	41	2000	TB	NA	*M. fortuitum*	Skin	Pus	Reticular	HRZEO	Dead	Loss
17	M	28	2000	Hydrocephalus	NA	*M. fortuitum*	CNS	CSF	Normal	KC	Improved	Loss
18	F	19	2000	HIV	NA	MAC	Disseminated, lung	Sputum	Reticular	HRZE	Dead	Loss
19	M	20	2000	TB	NA	MAC	Disseminated, lymph node	BM	Reticular	HRZEK	Improved	Loss
20	M	42	2000	HIV	82	MAC	Disseminated, lung	Blood	NA	HR	Improved	FU
21	M	44	2000	HIV	NA	MAC	Disseminated, lung, GI	Blood	Reticular	No	Improved	Loss
22	F	18	2000	HIV, PCP	NA	MAC	Disseminated, lung, GI	Blood	Reticular	No	Improved	Loss
23	F	20	2000	No	NA	*M. flavescence*	Lymph node	Sputum	NA	No	Improved	Loss
24	M	45	2000	HIV, VZV	NA	MAC	Lung, GI	Stool	Reticular	HRZEKO	Improved	Loss
25	M	20	2000	HIV	20	*M. kansasii*	Disseminated	BM	Alveolar	HRZEKO	Dead	Loss
26	M	37	2001	AML	NA	*M. kansasii*	Lung, lymph node	Tissue	Reticular	HRZE	Dead	Loss
27	F	38	2001	HIV	NA	MAC	Disseminated, lung, GI	Sputum	Reticular	HRZEK	Improved	Loss
28	F	34	2001	HIV, TB, CRY	NA	MAC	Lung, lymph node	Tissue	Alveolar	HRZE	Dead	Loss
29	F	54	2001	Hydrocephalus	NA	*M. fortuitum*	CNS	CSF	Reticular	HRZE	Improved	Loss
30	F	31	2001	HIV, VZV	32	*M. kansasii*	Lung, lymph node	Tissue	Reticular	HRZEK	Referred	Loss
31	F	32	2001	No	NA	Unspecified	Lung	Sputum	Alveolar	HRZE	Referred	Loss
32	M	31	2001	HIV, VZV, TB, CRY	NA	MAC	Lung	Sputum	Alveolar	HRZE	Referred	Loss
33	M	37	2001	HIV, TB, CP	60	*M. fortuitum*	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
34	F	53	2001	HT, DM	NA	*M. kansasii*	Lung	Sputum	Reticular	HREK	Dead	FU
35	M	38	2001	HIV, TB	NA	MAC	Lung, GI	Sputum	Reticular	HRZE	Improved	Loss
36	F	39	2001	HIV, TB, CRY	NA	*M. flavescence*	Lung	Sputum	Reticular	HRZE	Referred	Loss
37	F	62	2001	HIV	250	*M. fortuitum*	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
38	F	13	2001	No	NA	*M. chelonae*	Lung, skin	Sputum	Reticular	No	Improved	Loss
39	F	32	2001	HIV, CRY, PEN	10	MAC	Lung	Sputum	Alveolar	No	Dead	Loss
40	M	42	2001	HIV, Cirrhosis	NA	MAC	Lung, GI	Ascitic fluid	Reticular	HRZEKO	Dead	Loss
41	M	54	2001	No	NA	*M. kansasii*	Lung	Sputum	Effusion	HRZE	Improved	FU
42	M	31	2001	HIV, VZV, TB	70	Unspecified	GI	Stool	Reticular	HRZEK	Dead	Loss
43	M	8	2001	No	NA	*M. chelonae*	Keratitis	Tissue	NA	No	Improved	Loss
44	F	42	2002	No	NA	*M. kansasii*	Lymph node	Tissue	NA	HRZEK	Improved	FU
45	F	33	2002	HIV, PEN, CRY	89	MAC	Lung	Sputum	Normal	No	Dead	Loss
46	F	34	2002	HIV, VZV, TB	NA	MAC	Lung	Sputum	NA	HRZE	Improved	Loss
47	M	27	2002	CMI defect	NA	*M. gordonae*	Lung	Sputum	Alveolar	HRZE	Improved	FU
48	M	34	2002	HIV, VZV, Cirrhosis	32	MAC	Lung, GI	Ascites	Reticular	HRZEKO	Dead	Loss
49	M	41	2002	No	NA	*M. chelonae*	Lung	Sputum	Reticular	HREOS	Improved	Loss
50	M	37	2002	HIV, TB, CRY	90	MAC	Lung, GI	Stool	Normal	HRZEK	Referred	Loss
51	F	15	2002	HIV	140	*M. chelonae*	Lung	Sputum	Alveolar	No	Improved	Loss

Table 10. A summary of 103 patients with NTM infections (Cont.)

No.	Sex	Age (yrs)	Year	Associated condition	CD4 (cells / L)	Species	Infection	Specimen	Chest X-ray	Treatment regimen	Outcome	FU
52	F	43	2002	RA	NA	*M. kansasii*	Skin	Pus	NA	No	Improved	Loss
53	M	37	2002	HIV, TB, PCP	NA	MAC	Lung, skin	Pus	Normal	No	Referred	Loss
54	M	31	2002	HIV, VZV, TB, CRY, PCP	69	MAC	Lung, skin	Pus	Alveolar	HRZE	Improved	FU
55	F	36	2002	HIV, PCP	36	MAC	Lung, GI	Stool	Reticular	HRZEO	Improved	Loss
56	M	31	2002	HIV, PCP	49	MAC	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
57	F	28	2002	HIV, TB, PEN, HEP	320	MAC	Lung	Sputum	Reticular	HRZEK	Referred	Loss
								Sputum	Alveolar	HRZEK	Dead	Loss
58	M	19	2002	HIV, TB, CRY, PCP	21	MAC	Lung, GI	Sputum	Reticular	HRZE	Improved	Loss
59	M	32	2002	HIV, PCP	13	MAC	Lung					
60	M	22	2000	HIV	NA	*M. kansasii*	Lung	Sputum	Reticular	HRZE	Improved	Loss
61	M	44	2001	No	NA	*M. kansasii*	Lung	Sputum	NA	Minocyclinr	Improved	Loss
62	F	38	2001	No	NA	*M. fortuitum*	Lymph node	Tissue	NA	No	Improved	Loss
63	M	51	2001	HIV, Toxoplasmosis	128	*M. scrofulaceum*	Lung	Sputum	Reticular	HRZE	Improved	Loss
64	F	15	2001	HIV, VZV, PCP	31	MAC	Lung, GI	Sputum	Normal	No	Dead	Loss
65	M	37	2001	No	NA	*M. kansasii*	Lung	Sputum	Reticular	No	Improved	Loss
66	M	38	2001	HIV, VZV, TB	340	*Unspecified*	Lung, GI	Sputum	Reticular	No	Dead	Loss
67	M	34	2001	HIV, CRY, PCP	NA	*M. kansasii*	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
68	M	23	2001	HIV	207	*M. kansasii*	Lung	Sputum	Reticular	HRZE	Improved	FU
69	M	21	2001	No	NA	*M. kansasii*	Lung, GI	Stool	NA	No	Improved	Loss
70	M	22	2001	HIV, TB	NA	*M. kansasii*	Lung, GI	Stool	Reticular	HRZEK	Referred	Loss
71	M	61	2001	No	NA	*M. szulgai*	Lung	Sputum	NA	No	Improved	Loss
72	M	75	2001	COPD	NA	MAC	Lung	Sputum	NA	No	Improved	Loss
73	M	33	2001	DM	NA	*M. szulgai*	Skin	Pus	Reticular	HRZEKO	Improved	Loss
74	M	38	2002	HIV	NA	*M. kansasii*	Lung	Sputum	Reticular	HRZEK	Improved	Loss
75	M	51	2002	TB	NA	*M. scrofulaceum*	Lung	Sputum	Reticular	HRZEK	Improved	FU
76	F	21	2002	HIV, VZV, TB	NA	*M. chelonae*	Lung, GI	Sputum	Reticular	HRZESC	Improved	Loss
77	M	32	2002	HIV, TB, HIS	20	*M. flavescence*	Lung	Sputum	Normal	HRZEKO	Dead	Loss
78	F	52	2002	HIV, TB	130	*M. kansasii*	Lung, GI	Sputum	Reticular	HREOS	Dead	FU
79	F	34	2002	HIV, TB	10	*M. kansasii*	Lung	Sputum	Normal	HRZE	Improved	Loss
80	F	24	2002	HIV	NA	*M. chelonae*	Lung	Sputum	Reticular	HREOS	Improved	Loss
81	M	24	2002	HIV, PCP	20	*M. flavescence*	Lung, GI	Sputum	Alveolar	HRZEK	Improved	Loss
82	M	21	2002	HIV, CRY	NA	*M. flavescence*	Lung	Sputum	Reticular	HRZE	Improved	F/U
83	F	35	2002	HIV	10	*M. scrofulaceum*	Lung, GI	Sputum	Reticular	HRZE	Improved	Loss
84	M	42	2002	HIV, TB, PCP	34	MAC	Lung	Sputum	Bronchiec	HRZEO	Improved	Loss
85	F	23	2003	HIV, PCP	80	MAC	Lung	Sputum	Alveolar	REKA	Improved	FU
86	M	54	2003	HIV	NA	MAC	Lung, skin	Pus	Normal	HRZEKO	Dead	Loss
87	M	42	2003	HIV	4	Unspecified	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
88	M	34	2003	HIV, CAR	NA	Unspecified	Lung	Tissue	Normal	HRZEK	Dead	Loss
89	F	21	2003	HIV, VZV, CRY	23	MAC	Lung, GI	Stool	Reticular	HRZEK	Dead	Loss
90	M	37	2003	HIV, PCP	12	MAC	Lung, GI	Sputum	Reticular	HRZEK	Dead	Loss
91	M	15	2003	No	NA	MAC	Lung, GU	Urine	Alveolar	HRZEKO	Improved	FU
92	F	23	2003	HIV	NA	MAC	Lung, GI	Stool	Reticular	HRZEKO	Improved	Loss
93	M	33	2003	HIV, TB	NA	*M. kansasii*	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
94	F	29	2003	HIV	300	MAC	Lung, GI	Stool	Reticular	HRZE	Improved	FU
95	F	26	2003	HIV, VZV, TB, PCP	76	MAC	Lung, GI	Stool	Normal	HRZEKO	Dead	Loss
96	F	21	2003	HIV	32	MAC	Lung	Sputum	Alveolar	HRZEK	Dead	Loss
97	M	28	2003	HIV, PCP	5	MAC	Lung, GI	Stool	Alveolar	HRZEK	Referred	Loss
98	F	39	2003	HIV, TB	16	*M. fortuitum*	Lung	Sputum	Reticular	HRZE	Improved	FU
99	M	34	2003	No	NA	*M. fortuitum*	Skin	Tissue	NA	No	Improved	Loss
100	F	22	2003	No	NA	*M. abscessus*	GU	Pus	Normal	HR	Improved	Loss
101	M	54	2003	DM	NA	*M. abscessus*	Skin	Tissue	Normal	HRZEKO	Improved	Loss
102	M	31	2003	HIV, TB, PEN	NA	MAC	Lung, GI	Sputum	Reticular	HRZE	Dead	Loss
103	F	15	2003	No	NA	*M. kansasii*	Lung, skin	Pus	Alveolar	HRZE	Improved	Loss

HIV: human immunodeficiency virus, DM: diabetes mellitus, HT: hypertension, RA: rheumatoid arthritis, NHL: nonHodgkin lymphoma, COPD: chronic obstructive pulmonary disease, AML: acute myeloid leukemia, CIR: cirrhosis, HYD: hydrocephalus, DYS: dyslipidemia, CAR: cardiomyopathy, HEP: hepatoma, TB: tuberculosis, PCP: *Pneumocystis jirovecii* pneumonia, CRY: cryptococcal meningitis, PEN: penicilliosis, CMV: cytomegalovirus infection, TOX: toxoplasmosis, HIS: histoplasmosis, GI: gastrointestinal tract, GU: genitourinary tract, CNS: central nervous system, H: isoniazid, R: rifampin, Z: pyrazinamide, E: ethambutol, K: clarithromycin, O: ofloxacin, C: ciprofloxacin, A: amikacin, S: streptomycin, NA: not applicable, BM: bone marrow, CSF: cerebrospinal fluid, FU: follow up

risk factor for acquisition of NTM pulmonary disease is the presence of underlying chronic lung disease, especially COPD[2,38,39]. Most patients in the present study had symptomatic HIV infection with CD4 cell counts of < 200 cells/ L. This is consistent with previous studies[18,40-43]. In the present study, the number of NTM cases each year from 2000 to 2003 had not increased despite the increasing number of HIV-infected patients and the improvement of diagnostic methods for NTM infections. This is probably due to most patients being HIV-infected patients, and there has been an increasing use of antiretreoviral drugs resulting in a restoration of the immune response.

The predominant clinical presentations of the presented patients were prolonged fever, chronic cough, lymphadenopathy, weight loss, and chronic diarrhea. As described elsewhere[10-12], these features accompanying with anemia and elevated serum alkaline phosphatase suggest the disseminated infection, commonly caused by MAC in HIV-infected patients. As seen worldwide, the pulmonary infection in HIV-noninfected patients was the most common clinical feature in the present study, accounting for 60% of all patients without HIV infection. The commonest NTM pulmonary pathogens in the present study were MAC (53.7% and 25% in HIV-infected and HIV-noninfected patients), consistent with previous studies[2,7,44,45]. The presented rates of *M. kansasii* in pulmonary infection were 18.5% and 25% in HIV-infected and HIV-non-infected patients, respectively. These high rates are consistent with several reports in America[8], the United Kingdom[46], Japan[47], and Switzerland[48] as well as Thailand[23,28]. However, there was little or no pulmonary infection caused by *M. kansasii* in Australia[44], Hong Kong[49], Canada[50], and Sweden[45] where the predominant pathogen was MAC. One study of NTM pulmonary infection in Thailand during 1969 and 1978 reported only one patient with *M. kansasii* infection[52]. This discrepancy may be due to the difference in geographic distribution and study period. RGM are also recognized as common etiologic pathogens of pulmonary disease, occurring mostly in elderly patients without preexisting lung disease[44,51]. In the present study, RGM accounted for 11.4% of all patients with pulmonary infections, consistent with the authors' previous report in KCMH[33].

Most patients especially with AIDS in the present study who presented with prolonged fever and watery diarrhea had MAC infection, and AFB staining was positive in most stool specimens. The authors recommend routine AFB staining of stool specimens in all patients with AIDS who presented with chronic diarrhea.

In the present study, RGM and MAC are the species of NTM that most commonly cause skin and soft-tissue infections. MAC infections occurred only in patients with AIDS, when RGM could cause infections in both HIV-infected and HIV-noninfected patients. Both direct skin infection and reactive skin lesions like Sweet's syndrome were observed in the presented patients without HIV infection, consistent with a previous report by Chetchotisakd et al[26].

Chest radiographic changes in patients with and without HIV infection were similar to those observed with tuberculosis, both localized and disseminated forms with alveolar infiltration involving the upper lobe and with diffuse reticular infiltrations involving the lower lobes, respectively. Most patients in the present study had diffuse reticular pulmonary infiltrations because most had symptomatic HIV infection or immunocompromised condition.

In the present study, most patients especially with AIDS received an empirical treatment with four standard antituberculous drugs (isoniazid, rifampin, ethambutol, and pyrazinamide) with or without anti-MAC agents (clarithromycin in addition to ethambutol and rifampin). Nearly one-fourth of the patients did not receive specific antimycobacterial agents because they were during the investigation period, died, or lost to follow-up before a definite diagnosis of NTM infection could be made. The outcome of disseminated MAC infection especially in patients with AIDS was poor in the present study, compared to those without HIV infection. This observation is consistent with previous studies[10-12,16,44]. Restoration of immune response with highly active antiretroviral therapy after some control of mycobacterial infection may be the most important factor to improve the outcome of treatment in those patients.

In conclusion, a diagnosis of NTM infection requires a high index of suspicion in patients especially with AIDS or immunocompromised status who present with prolonged fever, with or without organ-specific symptoms and signs. Thus, clinical specimens must be sent for mycobacterial cultures for a definite diagnosis, a determination of the species of NTM, and an appropriate management. In addition to four standard antituberculous drugs, clarithromycin should be added for the treatment of MAC in patients with AIDS who present with disseminated opportunistic infections before obtaining microbiologic results.

References

1. American Thoracic Society. Diagnosis and treatment of disease caused by nontuberculous mycobacteria. Am J Respir Crit Care Med 1997; 156 (Suppl): S1-25.
2. Wolinsky E. State of the art: non-tuberculous mycobacteria and associated disease. Am Rev Respir Dis 1979; 119: 107-59.
3. Wolinsky E, Rynearson TK. Mycobacteria in soil and their relation to disease-associated strains. Am Rev Respir Dis 1968; 7: 1032-43.
4. Chapman JS. The ecology of the atypical mycobacteria. Arch Environ Health 1971; 22: 41-58.
5. Goslee S, Wolinsky E. Water as a source of potentially pathogenic mycobacteria. Am Rev Respir Dis 1976; 113: 287-92.
6. Gruft H, Falkinham JO III, Parker BC. Recent experience in the epidemiology of disease caused by atypical mycobacteria. Rev Infect Dis 1981; 3: 990-6.
7. Wagner D,Young LS. Nontuberculous mycobacterial infections: a clinical review. Infection 2004; 32: 257-70.
8. Ahn CH, Lowell JR, Onstad GD, Shuford EH, Hurst GA. A demographic study of disease due to *Mycobacterium kansasii* or *M. intracellulare-avium* in Texas. Chest 1979; 75: 120-5.
9. Zeligman I. *Mycobacterium marinum* granuloma. A disease acquired in the tributaries of Chesapeake Bay. Arch Dermatol 1972; 106: 26-31.
10. Horsburgh CR Jr. *Mycobacterium avium* complex infection in the acquired immunodeficiency syndrome. N Engl J Med 1991; 324: 1332-8.
11. Horsburgh CR Jr. Epidemiology of mycobacterial disease in AIDS. Res Microbiol 1992; 143: 372-7.
12. Horsburgh CR Jr, Selik R. The epidemiology of disseminated non-tuberculous mycobacterial infection in the acquired immunodeficiency syndrome (AIDS). Am Rev Respir Dis 1989; 139: 4-7.
13. Peters M, Schurmann D, Mayr AC, Hetzer R, Pohle HD, Ruf B, et al. Immunosuppression and mycobacteria other than *Mycobacterium* tuberculosis: results from patients with and without HIV infection. Epidemiol Infect 1989; 103: 293-300.
14. Selik, RM, Starcher ET, Curran JW. Opportunistic disease reported in AIDS patients: frequencies, associations, and trends. AIDS 1987; 1: 175-82.
15. Fauci AS, Macher AM, Longo DL. Immunopathogenic mechanisms in human immunodeficiency virus (HIV) infection. Ann Intern Med 1991; 114: 678-93.
16. Hawkins CC, Gold JW, Whimbey E. *Mycobacterium avium* complex infection in patient with the acquired immunodeficiency syndrome. Ann Intern Med 1986; 105: 184-8.
17. Macher AM, Kovacs JA, Gill V, Roberts GD, Ames J, Park CH, et al. Bacteremia due to *M. avium-intracellulare* in the acquired immunodeficiency syndrome. Ann Intern Med 1983; 99: 782-5.
18. Wallace JM, Hannah JB. *Mycobacterium avium* complex infection in the acquired immunodeficiency syndrome. Chest 1988; 93: 926-32.
19. Young, LS, Inderlied CB, Berlin OG, Gottlieb MS. Mycobacterial infections in AIDS patients, with an emphasis on the *Mycobacterium avium* complex. Rev Infect Dis 1986; 8: 1024-33.
20. Nightingale SD, Byrd LT, Southern PM, Jockusch JD, Cal SX, Wynne BA, et al. Incidence of *Mycobacterium avium-intracellulare* complex in human virus positive patients. J Infect Dis 1992; 165: 1082-5.
21. Nuchprayoon C, Thanabodi P, Chinachoti N. Pulmonary disease due to photochromogenic mycobacterial infection. J Med Assoc Thai 1968; 51: 923-32.
22. Bovornkitti S, Pushpakorn R, Nana A, Charoenratanakul S. Dissertation of reported cases of nontuberculous mycobacteriosis in Thailand. Siriraj Hosp Gaz 1991; 43: 392-6.
23. Wongwatana S, Sriyabhaya N. Non-tuberculous mycobacterial infection of lung in a chest hospital in Thailand. J Med Assoc Thai 1992; 75: 1-10.
24. Chierakul N, Mekanant P, Nana A. *Mycobacterium smegmatis* infection in a Thai woman. J Infect Dis Antimicrob agents 1993; 10: 25-8.
25. Kullavanijaya P, Prachyapruit W, Erpaiboon P. *Mycobacterium marinum* granuloma: in two agriculture students. Siriraj Hosp Gaz 1993; 45: 191-4.
26. Chetchotisakd P, Mootsikapun P, Anunnatsiri S, Jirarattanapochai K, Choonhakarn C, Chaiprasert A, et al. Disseminated infection due to rapidly growing mycobacteria in immunocompetent hosts presenting with chronic lymphadenopathy: a previously unregcognized clinical entity. Clin Infect Dis 2000; 30: 29-34.
27. Chittasobhaktra T, Vibhagool A, Pracharktam R. Clinical manifestation and drug susceptibility of rapidly growing mycobacteria. J Infect Dis Antimicrob Agents 1999;16:109-14.
28. Ratanasuwan W, Anekthananont T, Techasathit W, Jearanaisilavong J, Chuenarom V, Chaiprasert A, et al. Infection due to nontuberculous *Myco-*

bacterium other than MAC in AIDS patients at Siriraj Hospital during 1998-2000: saprophyte vs pathogen. J Med Assoc Thai 2002; 85: 886-93.
29. Chuchottaworn C, Sathapatayawongs B, Tansuphasawadikul S. Prevalence of disseminated *Mycobacterium avium* complex infection in Thai AIDS patients. J Med Assoc Thai 1999; 82: 643-7.
30. Suwanagool S, Kolladarungkri T, Leelarasamee A. Prolonged fever due to *Mycobacterium avium* complex (MAC) disease in advanced HIV infection: a public heath concern. J Med Assoc Thai 1998; 81: 893-905.
31. Kongkarat N, Mansrichum A, Roengsunthia D. Fungi and mycobacteria isolated from blood of AIDS patients in Siriraj Hospital in 1997-1998. In: Program and abstract of the 25th annual meeting of the Infectious Disease Association of Thailand. Udorn Thani, 25-28 October 1999.
32. Sungkanuparph S, Pracharktam R. The yield of mycobacterial blood culture in the diagnosis of disseminated mycobacterial infection in patients with and without HIV infection. In: Program and abstract of the 26th annual meeting of the Infectious Disease Association of Thailand, Bangkok, 1-3 November 2000.
33. Phowthongkum P, Prasanthai V, Udomsantisook N, Suankratay C. Rapidly growing mycobacteria in King Chulalongkorn Memorial Hospital and review of the literature in Thailand. J Med Assoc Thai 2005; 88: 1153-62.
34. American Thoracic Society. Diagnosis and treatment of disease caused by nontuberculous mycobacteria. Am Rev Respir Dis 1990; 142: 940-53.
35. Thorpe TC, Wilson ML, Turner JE. BacT/Alert: an automated colorimetric microbial detection system. J Clin Microbiol 1990; 28: 1608-12.
36. Pfylter GE, Brown-Elliott BA, Wallace RJ Jr. *Mycobacterium:* general characteristics, isolation, and staining procedures. In: Murray PR, Baron EJ, Jorgensen JH, Pfaller MA, Yolken RH, editors. Manual of clinical microbiology. 8th ed. Washington DC: ASM Press; 2003: 532-59.
37. Vestal AL. Procedures for the isolation and identification of mycobacteria. Centers for Disease Control and Prevention, Atlanta 1975: publication no.75-8230.
38. Yeager HJ, Raleigh JW. Pulmonary disease due to *Mycobacterium intracellulare*. Am Rev Respir Dis 1973; 108: 547-52.
39. Corpe RF. Clinical aspects, medical and surgical in the management of pulmonary disease at Battey State Hospital. Dis Chest 1964; 45: 380-2.
40. Benson CA. Disease due to the *Mycobacterium avium* complex in patients with AIDS: epidemiology and clinical syndrome. Clin Infect Dis 1994; 18: 218-22.
41. Rubin DS, Rahal JJ. *Mycobacterium avium* complex. Infect Dis Clin North Am 1994; 8: 413-26.
42. Fordham von Reyn C, Arbeit RD, Tosteson AN. Epidemiology of disseminated *Mycobacterium avium* complex infection in AIDS. International MAC study group. AIDS 1996; 10: 1025-32.
43. Brodt HR, Enzensberger R, Kamps BS, Kaul HG, Helm EB. Impact of disseminated *Mycobacterium avium* complex infection on survival of HIV-infected patients. Eur J Med Res 1997; 2: 106-10.
44. O'Brien DP, Currie BJ, Krause VL. Nontuberculous mycobacterial disease in Northern Australia: a case series and review of the literature. Clin Infect Dis 2000; 31: 958-68.
45. Wolinsky E. Mycobacterial diseases other than tuberculosis. Clin Infect Dis 1992; 15: 1-12.
46. Lamden K, Watson JM, Knerer G, Ryan MJ, Jenkins PA. Opportunist mycobacteria in England and Wales: 1982 to1994. Commun Dis Rep CDR Rev 1996; 6: R147-51.
47. Tsukamura M, Kita N, Shimoide S, Arawaka H, Kuze A. Studies on the epidemiology of nontuberculous mycobacteriosis in Japan. Am Rev Respir Dis 1988; 137: 1280-4.
48. Debrunner M, Salfinger M, Brandli O, von Graevenitz A. Epidemiology and clinical significance of nontuberculous mycobacteria in patients negative for human immunodeficiency virus in Switzerland. Clin Infect Dis 1992; 15: 330-45.
49. Hosker HS, Lan CW, Ng TK, Ma HK, Chan SL. The prevalence and clinical significance of pulmonary infection due to non-tuberculous mycobacteria in Hong Kong. Respir Med 1995; 89: 3-8.
50. Contreras MA, Cheung OT, Sanders DE, Goldstein RS. Pulmonary infection with nontuberculous mycobacteria. Am Rev Respir Dis 1988; 137: 149-52.
51. Griffith DE, Girard WM, Wallace RJ Jr. Clinical features of pulmonary disease caused by rapidly growing mycobacteria. Am Rev Respir Dis 1993; 147: 1271-8.
52. Sriyabhaya N, Wongwatana S. Pulmonary infection caused by atypical mycobacteria: a report of 24 cases in Thailand. Rev Infect Dis 1981; 3: 1085-9.

การติดเชื้อมัยโคแบคทีเรียที่ไม่ใช่เชื้อวัณโรคในโรงพยาบาลจุฬาลงกรณ์

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เชื้อมัยโคแบคทีเรียที่ไม่ใช่เชื้อวัณโรค (nontuberculous mycobacteria, NTM) สามารถติดเชื้อได้ทั้งผู้ป่วยติดเชื้อเอชไอวี และผู้ป่วยไม่ติดเชื้อเอชไอวี อุบัติการณ์ของการติดเชื้อ NTM เพิ่มขึ้นในช่วงที่มีการระบาดของโรคเอดส์ อย่างไรก็ตามระบาดวิทยา และข้อมูลทางคลินิกของการติดเชื้อ NTM ในประเทศไทยยังจำกัด การศึกษานี้มีวัตถุประสงค์เพื่อศึกษาระบาดวิทยา ลักษณะทางคลินิก การรักษา และผลของการรักษาการติดเชื้อ NTM ในโรงพยาบาลจุฬาลงกรณ์ตั้งแต่มกราคม พ.ศ. 2543 ถึง ธันวาคม พ.ศ. 2546 มีผู้ป่วย 114 ราย ที่ผลเพาะเชื้อขึ้น NTM แต่เวชระเบียนที่สมบูรณ์มีเพียง 103 ราย (90.3%) มีผู้ป่วย 71 ราย (68.9%) ที่ติดเชื้อเอชไอวี และ 38 ราย (87%) ที่มีระดับCD4 น้อยกว่า 200 เซลล์ต่อไมโครลิตร (พิสัย 4-360) ในผู้ป่วยติดเชื้อเอชไอวี การติดเชื้อฉวยโอกาสที่พบบ่อย ได้แก่ วัณโรค (36.6%), Pneumocystis jirovecii pneumonia (25.3%), cryptococcal meningitis (15.5%), penicilliosis (5.6%) และ cytomegalovirus infection (5.6%) ผู้ติดเชื้อเอชไอวีและไม่ติดเชื้อเอชไอวี ส่วนใหญ่มาพบแพทย์ด้วยอาการนำ คือ ไข้เรื้อรัง (67%) ไอเรื้อรัง (54.4%) ต่อมน้ำเหลืองโต (52.4%) น้ำหนักลด (50.5%) หรือ ท้องเสียเรื้อรัง (31%) สำหรับลักษณะทางคลินิกของการติดเชื้อ ได้แก่ การติดเชื้อแบบแพร่กระจาย (17.4%) และการติดเชื้อแบบเฉพาะที่ (82.6%) การติดเชื้อเฉพาะที่ที่พบบ่อยที่สุด ได้แก่ การติดเชื้อในปอด (82.3%) ตามด้วยการติดเชื้อของระบบทางเดินอาหาร (34.1%) การติดเชื้อที่ผิวหนัง (12.9%) การอักเสบของต่อมน้ำเหลือง (8.2%) การติดเชื้อของระบบทางเดินปัสสาวะและสืบพันธุ์ (2.4%) การติดเชื้อของระบบประสาท (2.4%) และกระจกตาอักเสบ (1.2%) พบการติดเชื้อ Mycobacterium avium complex (MAC) มากที่สุด (48.5%) ตามด้วย M. kansasii (19.4%) และมัยโคแบคทีเรียที่เจริญเร็ว (16.4%) ภาพรังสีวิทยาของปอดพบลักษณะเป็น diffuse reticular infiltration มากที่สุด (53.4%) ความผิดปกติของผลตรวจทางห้องปฏิบัติการที่พบ คือ ซีด (48.5%) ปริมาณโซเดียมในเลือดต่ำ (42.7%) และ alkaline phosphatase สูง (39.8%) อัตราการตายโดยรวมสูงถึง 34.8% (45.9% และ 11.1% ในผู้ติดเชื้อเอชไอวีและผู้ไม่ติดเชื้อเอชไอวี)

โดยสรุป การวินิจฉัยการติดเชื้อ NTM ต้องอาศัยความสงสัยเป็นอย่างสูงในผู้ป่วยที่มาพบแพทย์ด้วยไข้เรื้อรัง อาจร่วมหรือไม่ร่วมกับอาการและอาการแสดงที่จำเพาะของอวัยวะต่าง ๆ โดยเฉพาะในผู้ป่วยติดเชื้อเอชไอวี หรือ ภาวะภูมิคุ้มกันลดลง สิ่งส่งตรวจทางคลินิกต้องได้รับการส่งเพาะเชื้อสำหรับมัยโคแบคทีเรียเพื่อการวินิจฉัยที่แน่นอน การหา species ของ NTM และ การรักษาที่เหมาะสม นอกเหนือจากการให้ยาต้านวัณโรคมาตรฐาน 4 ชนิด ควรเพิ่ม clarithromycin เพื่อใช้รักษาการติดเชื้อ MAC ในผู้ป่วยเอดส์ที่มีอาการและอาการแสดงของการติดเชื้อแบบแพร่กระจายก่อนทราบผลการตรวจทางจุลชีววิทยา