Impact of Bronchoalveolar lavage Galactomannan on the Outcome of patients at risk for Invasive Pulmonary Aspergillosis

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Background: *Invasive pulmonary aspergillosis (IPA) is an important cause of morbidity and mortality among immunocompromised patients especially in neutropenic and patients treated with immunosuppressive drugs. New diagnostic tools have been developed to improve treatment and outcome. Compared with serum galactomannan, bronchoalveolar lavage galactomannan (BAL GM) detection has higher sensitivity (81% vs. 71%) and comparable specificity (87.6% vs. 89%). No study has correlated this test result to clinical outcome.*

Material and Method: *A prospective non-randomised study was conducted from March to December 2008 in adult patients who were suspected to have invasive pulmonary aspergillosis (IPA). Serum galactomannan levels were measured and bronchoscopy was performed to obtained BAL fluid for direct examination, culture, and measurement of galactomannan level. Response to treatment and mortality within 6-weeks of follow-up were compared between positive and negative BAL GM groups. Factors influencing outcome were also analysed.*

Results: *There were 30 patients with 3 probable, 11 possible and 17 no IPA. Other causative organisms can be identified in 8 of 17 patients in the no IPA group. Overall, BAL GM at the 0.5 cut-off yielded a 46% positive result compared with 13% of serum GM (p = 0.005). There was no significant difference in positive result between BAL GM at 1.0 cut-off and serum GM. By using BAL GM as a mycological criteria, 54% of possible IPA was upgraded to probable IPA. Neither BAL GM nor serum GM results were associated with clinical response and mortality. Recovery of neutropenia was the only factor associated with response to treatment and outcome (p = 0.003).*

Conclusion: *BAL GM detection has a higher positive rate than serum GM in patients at risk for IPA. It is helpful in diagnosis and categorization of IPA, but its impact on clinical outcome cannot be demonstrated in this study.*

Keywords: *Galactomannan antigen, Bronchoalveolar lavage, Invasive pulmonary aspergillosis*

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Invasive pulmonary aspergillosis (IPA) is an important cause of morbidity and mortality among immunocompromised patients especially in neutropenic and patients treated with immunosuppressive drugs. The mortality rate ranges from 30-80%[1-3]. Early diagnosis and treatment can improve the outcome. However, diagnosis of IPA is a major challenge. The gold standard for diagnosis requires a demonstration of tissue invasion by fungus and a positive culture from the same specimen, but tissue biopsy is often precluded by thrombocytopenia and the debilitated condition of the patients and culture yields only 50% sensitivity[4]. Because of the difficulty in diagnosis, there has been an intense search for a better diagnostic method of IPA. Detection of galactomannan is the most studied among new diagnostic methods.

Galactomannan (GM), a cell wall poly-saccharide of aspergillus, is produced during the fungal growth and is released into circulation. It is produced in a much lesser extent by conidia. Circulating GM can be detected at a median of 5-8 days before clinical signs and

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symptoms of IPA appear. Its concentration correlates with fungal burden and disease severity.

A commercially available sandwich ELISA has been widely used in Europe and has been recently approved by US Food and Drug Administration for detection of GM in serum. Previous studies found serum galactomannan assay to have a sensitivity of 71% and a specificity of 89%[5]. To improve the sensitivity of diagnostic methods, bron-choalveolar lavage galactomannan (BAL GM) has been introduced and found to have a higher sensitivity than serum GM. BAL GM at the cut-off values of 0.5 and 1.0 and has a comparable sensitivity of 81.8% and a specificity of 87.6 and 93.5% respectively[6]. In addition, exposure to antifungal agents reduced sensitivity of the test in BAL fluid by 8% compared with 20% in serum.

Nevertheless, there is no data about the correlation of these findings and clinical response or outcome of the patients or about whether this test aids clinicians in decision making or alters current treatment strategies. The aim of this study was to determine whether early detection of galactomannan in bronchoalveolar lavage has an impact on clinical response and survival of immunocom-promised patients at risk for invasive pulmonary aspergillosis.

Material and Method

Study population and data collection

Patients admitted to the Department of Medicine, Siriraj Hospital during March to December 2008 were included in this study. We included patients who were at risk of invasive pulmonary aspergillosis by screening patients with at least one of following host factors: hematologic malignancies, cancer and receiving chemotherapy within 3 months, hematopoietic stem cell transplant, solid organ transplant recipient, corticosteroid use > 20 mg/day for > 3 weeks or patients receiving other immunosuppressive agents. The eligible patients must also have the following features.

For neutropenic hosts

Fever with abnormal chest x-ray or CT scan of the chest and refractory for at least 3 days of appropriate antibiotics with or without pulmonary symptoms and signs (cough, hemoptysis, dyspnea, pleuritic chest pain or physical finding of crackles).

For non-neutropenic hosts

Development of pulmonary infiltrate suspected to be infectious with or without fever or pulmonary symptoms and signs.

The patients may or may not have received an antifungal agent prior to bronchoscopy.

Patients were excluded if they had known causative organisms or contraindi-cation for bronchoscopy.

Fiberoptic bronchoscopy was performed as soon as possible. Broncho-alveolar lavage using 150 ml. of normal saline was done at the site according to the imaging. The bronchoalveolar lavage fluid returned was submitted for galactomannan antigen detection, cell count, differential count, gram stain, AFB stain, modified AFB stain, direct examination for fungus, culture for bacteria, mycobacteria and fungus. Other laboratory investigations such as special stains for Pneumocystis jirovecii and CMV Ag detection were performed if requested by the attending physician. Transbronchial lung biopsy was done if there was no contra-indication. Serum galactomannan measure-ment was repeated at the day of performing bronchoscopy if the last specimen had been done more than 3 days previously.

Septic work-up and other imaging such as HRCT of the chest and empirical antibiotics treatment were done according to the attending physician and clinical practice guidelines for management of febrile neutropenia.

The study was approved by the ethics committee and written, informed consent was obtained from the patients.

Galactomannan antigen detection

Serum and BAL galactomannan were tested with an immunoenzymatic sandwich microplate assay commercial kit (Platelia^{R}-Aspergillus EIA: BIO-RAD, Marnes-la-Coquette, France) according to the manufacturer's instruction. An optical density (OD) index of ≥ 0.5 was considered positive for serum galactomannan. For BAL GM, 2 cut-off levels of 0.5 and 1.0 were used and analysed separately.

Case definition and classifications

IPA was diagnosed on the basis of the Revised Definition of Invasive Fungal Disease proposed by the European Organization for Research and Treatment of Cancer/Invasive fungal infections Cooperative Group and the National Institute of Allergy and Infectious Diseases Mycoses Study Group (EORTC/MSG)[7] (Table 1). In this study, we used chest X-ray findings in cases without CT scan. Patients with compatible host factors but no clinical and mycological criteria are classified as no IPA. Patients in the no IPA group in whom the investigation cannot yield any etio-

logic agents were subgrouped as suspected IPA.

Response and outcome

Response, including partial and complete, is defined as improvement or resolution of symptoms and signs of IPA plus at least a 25% reduction or resolution of radiologic abnormality[8].

Outcome is defined as all-cause mortality occurring within 6 weeks of follow-up.

Statistical analysis

Categorial data were expressed as counts and percentages and compared by using the Chi-square or Fisher's exact test. Continuous data were expressed as mean or median. Comparison between groups was performed by use of T test as parametric method and Mann-Whitney U test as non-parametric method. Statistical analyses were performed with SPSS version 13.0. A p-value less than 0.05 was considered significant.

Results

Patients characteristics

Patients admitted to the Department of Medicine during March to December 2008 were screened. 30 patients (17 males and 13 females) were enrolled. The mean age of the patients was 41 years (range 16-75 years). Twenty six out of 30 patients (84%) had acute leukemia. Other host factors were lymphoma (2; 6.5%), aplastic anemia (2; 6.5%), systemic lupus erythematosus treated with cortico-steroid and immunosuppressive drug (1; 3.2%). Twenty four patients were neutropenic or had recently recovered from neutropenia after chemotherapy, 5 patients had neutropenia from disease condition and 2 patients without neutropenia received corticosteroid therapy. The absolute neutrophil count (ANC) ranged from 0 to 7,920 cell/mm^3 (median 170 cell/mm^3) and platelet count ranged from 3,000-360,000/mm^3 (median 37,000/mm^3).

CT scans of the chest were done in 17 patients (56.6%). The CT pattern showed single or multiple nodules with or without halo sign in 11 patients and an air-crescent sign in 2 patients. Three out of 15 patients whose CT chest scan was not performed had nodular infiltration on plain chest X-rays.

The median time from onset of fever to bronchoscopy was 10 days (range 4-32 days) and the median time from first abnormal chest X-ray to bronchoscopy was 5 days (range 1-25 days). Three patients had respiratory failure and were mechanically ventilated while bronchoscopy was performed. Mild hypoxemia,

Table 1. Criteria for probable invasive fungal disease except for endemic mycoses.

Host factors
Recent history of neutropenia (< 0.5 x 10^9 neutrophils/L [< 500 neutrophils/mm^3] for > 10 days) temporally related to the onset of fungal disease
Receipt of an allogeneic stem cell transplant
Prolonged use of corticosteroids (excluding among patients with allergic bronchopulmonary aspergillosis) at a mean minimum dose of 0.3 mg/kg/day of prednisone equivalent for > 3 weeks
Treatment with other recognized T cell immunosuppressants, such as cyclosporine, TNF-a blockers, specific monoclonal antibodies (such as alemtuzumab), or nucleoside analogues during the past 90 days
Inherited severe immunodeficiency (such as chronic granulomatous disease or severe combined immunodeficiency)
Clinical criteria[a] for lower respiratory tract fungal disease[b]
The presence of 1 of the following 3 signs on CT:
Dense, well-circumscribed lesions(s) with or without a halo sign
Air-crescent sign
Cavity
Mycological criteria
Direct test (cytology, direct microscopy, or culture)
Mold in sputum, bronchoalveolar lavage fluid, bronchial brush, or sinus aspirate samples, indicated by 1 of the following:
Presence of fungal elements indicating a mold
Recovery by culture of a mold (*e.g.*, Aspergillus, Fusarium, Zygomycetes, or Scedosporium species)
Indirect tests (detection of antigen or cell-wall constituents)
Aspergillosis : Galactomannan antigen detected in plasma, serum, bronchoalveolar lavage fluid, or CSF

[a] Must be consistent with the mycological findings, if any, and must be temporally related to current episode.
[b] Every reasonable attempt should be made to exclude an alternative etiology

a complication of bronchoscopy, occurred in 4 patients (13.3%) .

None of the patients had proven IPA, 3 probable IPA, 11 possible IPA, 9 suspected IPA and 7 no IPA (2 pulmonary alveolar hemorrhage). TB, 2 bacterial pneumonia, 2 PCP and 1.

Galactomannan antigen detection

Overall, BAL GM had a higher positive rate than serum GM (46.7% vs. 13.3%, p = 0.01) (Table 2). BAL GM at the 1.0 cut-off yielded positive results more than serum GM but with no statistical significance (p = 0.15). There is no significant difference in the positive result of BAL and serum GM between each category of diagnosis. Among the 7 patients in the no IPA group and proven other etiologies, positive serum GM was found in 2 and positive BAL GM was found in 2 patients (Table 3).

Antifungal treatment

Twenty three patients (76.6%) were treated with antifungal agents before bronchoscopy. The median duration of treatment before bronchoscopy was 5 days. The antifungal agent was discontinued before the complete course in 3 patients (treatment duration 8-12 days). BAL fluid was positive for acid fast bacilli in one patient and no organism was identified in the other, but this patient was diagnosed as no IPA by the attending physician. Eighteen out of 22 patients (81%) received ampho-tericin B and in three of them this was changed or combined with voriconazole. The other six patients received voriconazole. One patient with positive BAL GM but negative serum GM was not treated with antifungal agent and died in that episode. All patients were concomitantly treated with antibiotics.

Response to treatment and outcome

To assess the response to treatment with antifungal agents, we focused on 22 patients in the probable, possible and suspected IPA groups. The overall response rate was 63.6% (14 in 22 patients) and 33.3%, 63.6% and 75% in the probable IPA , possible IPA and suspected IPA groups, respectively.

Patients who survived were followed up for a mean duration of 39 days (range 14-94 days). The overall mortality was 43.3% (13 in 30 patients) and 66.7% , 41.7% and 40% in the probable IPA , possible IPA and no IPA groups, respectively. Causes of death were septic shock in 8 patients, diffuse alveolar hemorrhage in 4 patients and IPA in 1 patient.

Table 2. Positive results in each category.

		IPA : Number				
		No (n = 15)	Possible (n = 12)	Probable (n = 3)	Total (n = 30)	Exact p-value
BAL GM	< 0.5	8	6	2	16	1.000
	≥ 0.5	7	6	1	14	
	< 1.0	10	10	2	22	0.502
	≥ 1.0	5	2	1	8	
Serum GM	< 0.5	13	12	1	26	0.020
	≥ 0.5	2	0	2	4	
BAL c/s or	No	15	11	2	28	0.090
histology	Yes	0	1	1	2	

Table 3. False positive serum and BAL GM detection.

Patient	serum GM	BAL GM	Final diagnosis	remark	outcome
Case 1	+ve	-ve	alveolar hemorrhage	pip/tazo discontinue 1 day before FOB	survive
Case 2	+ve	+ve	pulmonary TB		survive
Case 3	-ve	+ve	bacterial pneumonia	pip/tazo discontinue 6 days before FOB	survive

Definition of abbreviations : pip/tazo = piperacillin/tazobactam

Factors influencing response to treatment and outcome

The response to treatment and mortality were neither related to both the 0.5 and 1.0 cut-off BAL GM results (Table 4 and 5) nor the serum GM result (p = 0.33) in all categories. We analysed other factors that may have an impact on mortality and found that recovery of neutropenia was associated with a response to treatment and outcome. Lower ANC and platelet counts have a tendency to be associated with increased mortality but there is no statistical significance (Table 6).

BAL GM as one of the mycological criteria

Considering the positive BAL GM result as positive mycological criteria, 6 out of 11 patients (54%) of the possible IPA group would be categorized in the probable IPA group. However, othere is no association

Table 4. Rate of response to treatment and BALGM results*

Diagnostic categories**	Response rate, no of response/total		p-value
	+ve BAL GM*	-ve BAL GM	
Probable IPA (n = 3)	1/2	1/1	0.386
Possible IPA (n = 10)	4/5	4/5	1.000
No IPA* (n = 9)	3/4	4/5	0.858
Total population (n = 22)	8/11	8/11	1.000

*cut-off value 0.5
**exclude 7 patients with proved other etiologies

Table 5. Mortality and BAL GM results*

Diagnostic categories	Mortality rate, no. of dead/total (%)		p-value
	+ve BAL GM	-ve BAL GM	
Probable IPA (n = 3)	1/2	1/1	1.000
Possible IPA (n = 12)	2/6	2/6	1.000
No IPA (n = 15)	2/7	4/8	0.608
Total population (n = 30)	6/14	7/16	1.000

*cut-off value 0.5

Table 6. Analysis of factors associated with outcome.

Factors	Survive (n = 17)	Dead (n = 13)	p-value
Age (mean, SD)	38, 11.7	45, 20.2	0.432
Positive BAL GM (0.5 cut-off), n	8	6	1.000
Positive BAL GM (1.0 cut-off), n	6	2	0.407
Positive serum GM, n	2	2	1.000
ANC (median, cell/mm^3)	890	110	0.094
Platelet count (mean,/mm^3)	44,000	32,000	0.079
Time to start antifungal (from onset of fever), days	5	7	0.683
Hemoptysis, n	1	1	1.000
Patients treated with voriconazole, n	1	3	0.457
Recovery of neutropenia, n	17	2	0.003

between the BAL GM result and the outcome or response to treatment in every category.

Discussion

BAL GM detection has been increasingly investigated as a tool for diagnosis of invasive pulmonary asper-gillosis. Previous studies found that BAL GM has a higher sensitivity than serum (81.8-88% vs. 42-64%)[6,9] with the specificities that are comparable[6]. Thus, detection of BAL GM is expected to help in the earlier diagnosis of IPA and the decision to start an antifungal agent and may improve the outcome.

In this study, BAL GM at the cut-off value of 0.5 also yielded a higher positive rate than the serum GM (46.7% vs. 13.3%, p = 0.005). To determine the impact of BAL GM on the clinical response and outcome of the patients, we compared the response and mortality rates between positive and negative BAL GM groups, but found no impact. The majority of our patients were neutropenic and received empirical antifungal treatment according to the current guideline. Also, the treatment was continued in 7 out of 9 patients (77%) in the no IPA group even with negative BAL and serum GM and no organism identified. However, bronchoalveolar lavage helped in identification of other etiologies in 7 out of 30 patients (23.3%).

The EORTC/MSG criteria for diagnosis of invasive aspergillosis seems to underestimate the diagnosis of IPA[10] . When the BAL GM result is considered as a mycological criteria, 54% of patients in the possible IPA will be upgraded to the probable IPA group. This improves sensitivity of the diagnostic criteria.

The response rate to antifungal treatment of the patients was 63.6% in all categories and 33.3% in probable IPA group which were comparable to previous studies using amphotericin B (33-54%)[1,11,12]. Mortality rate among our patients was also similar to other studies (30-80%)[1-3]. From this study, bone marrow recovery deter-mined the response to treatment and outcome. Patients who died did not recover from neutropenia. Other factors including age, ANC, platelet counts, presence of hemoptysis, and type of antifungal agent were not associated with the outcome.

There are some limitations of this study. First, the number of patients is less than the calculated sample size so that it cannot detect any significant difference between groups. Second, the study population may not represent the whole spectrums of IPA because patients with unstable condition and profound thrombocytopenia are precluded from bronchoscopy and the majority of our patients were neutropenic host. Finally, the proportion of probable and possible cases may be underestimated by the CT scan of the chest which is the only clinical criteria proposed in the new definition and which was not performed in all cases.

In conclusion, the impact of BAL GM on the clinical outcome of the patients suspected to have IPA cannot be determined in the present study. A positive galactomannan result in an appropriate clinical setting can help to confirm diagnosis of IPA and better categorization of patients. Bronchoscopy has a limited role in the management of neutropenic hosts for whom empirical antifungal treatment strategy is implicated. However, it is useful in the identification of other etiologies and may be more helpful in the management of pneumonia in non-neutropenic hosts. Nevertheless, the impact of BAL GM in nonneutropenic patients has to be further investigated.

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ผลการตรวจแอนติเจนกาแลคโตแมนแนนในน้ำล้างปอดต่อผลลัพธ์ของผู้ป่วยที่เสี่ยงต่อการติดเชื้อราแอสเปอร์จิลลัสแบบลุกลามในปอด

ประณิธิ ด่านพรประเสริฐ, ศุภร ฟุ้งลัดดา, แจ่มศักดิ์ ไชยคุนา

วัตถุประสงค์: *การติดเชื้อราแอสเปอร์จิลลัสแบบลุกลามในปอดเป็นสาเหตุสำคัญ ของการป่วยและเสียชีวิตที่ในผู้ป่วยที่มีภูมิต้านทานต่ำ โดยเฉพาะผู้ป่วยโรคเลือดที่มีเม็ดเลือดขาวต่ำและผู้ป่วยที่ได้รับยากดภูมิ ได้มีการพัฒนาเครื่องมือการตรวจเพื่อทำให้การรักษาและผลลัพธ์ของผู้ป่วยดีขึ้น เมื่อเปรียบเทียบการตรวจแอนติเจนกาแลคโตแมนแนนในเลือดและในน้ำล้างปอด พบว่าการตรวจในน้ำล้างปอดมีความไวสูงกว่า (ร้อยละ 81 กับ 71) โดยมีความจำเพาะใกล้เคียงกัน (ร้อยละ 87.6 กับ 89) อย่างไรก็ตามไม่มีการศึกษาถึงความสัมพันธ์ของผลการตรวจกับผลลัพธ์ของผู้ป่วย*

วัสดุและวิธีการ: *เป็นการศึกษาแบบไปข้างหน้าระหว่างเดือนมีนาคม ถึง เดือนธันวาคม พ.ศ. 2551 ในผู้ป่วยผู้ใหญ่ที่สงสัยว่าติดเชื้อราแอสเปอร์จิลลัสแบบลุกลามในปอด ทำการวัดระดับแอนติเจนกาแลกโตแมนแนนในเลือดและส่องกล้องตรวจหลอดลม เพื่อนำน้ำล้างปอดมาตรวจเพาะเชื้อและวัดระดับแอนติเจนกาแลคโตแมนแนน เปรียบเทียบการตอบสนองต่อการรักษาและการเสียชีวิตใน 6 สัปดาห์ระหว่างผู้ป่วยที่มีผลการตรวจแอนติเจนกาแลคโตแมนแนนเป็นบวกและลบวิเคราะห์ปัจจัยที่มีผลต่อผลลัพธ์ของผู้ป่วย*

ผลการศึกษา: *มีผู้ป่วยจำนวน 30 ราย โดยแยกเป็นการติดเชื้อราแอสเปอร์จิลลัสแบบลุกลาม ประเภทน่าจะเป็น 3 ราย ประเภทอาจเป็นไปได้ 11 รายและไม่มีการติดเชื้อ 17 ราย ซึ่งในจำนวนนี้ ตรวจพบเชื้อก่อโรคอื่น 8 ราย โดยรวมการตรวจแอนติเจนกาแลคโตแมนแนนในน้ำล้างปอดที่ใช้ค่าจุดตัด 0.5 ให้ผลบวกร้อยละ 46 สูงกว่าการตรวจในเลือดซึ่งให้ผลบวกร้อยละ 13 ($p = 0.005$) ส่วนการตรวจน้ำล้างปอดโดยใช้จุดตัด 1.0 ให้ผลบวกไม่แตกต่างจากการตรวจในเลือด เมื่อใช้ผลแอนติเจนกาแลคโตแมนแนน ในน้ำล้างปอดเป็นเกณฑ์การวินิจฉัยพบว่าผู้ป่วยในกลุ่มที่อาจเป็นไปได้ร้อยละ 56 จะจัดอยู่ในกลุ่มน่าจะเป็นผลการตรวจแอนติเจนกาแลคโตแมนแนนทั้งในน้ำล้างปอดและเลือดไม่มีความสัมพันธ์กับการตอบสนองต่อการรักษา และการเสียชีวิตของผู้ป่วย การหายจากภาวะเม็ดเลือดขาวนิวโตรฟิวต่ำเป็นปัจจัยเดียวที่มีผลต่อการตอบสนองต่อการรักษาและการเสียชีวิต ($p = 0.03$)*

สรุป: *ในผู้ป่วยที่สงสัยการติดเชื้อราแอสเปอร์จิลลัสแบบลุกลามในปอด การตรวจแอนติเจนกาแลคโตแมนแนนในน้ำล้างปอดให้ผลการตรวจเป็นบวกมากกว่าการตรวจในเลือด การตรวจนี้ช่วยในการวินิจฉัยและจัดกลุ่มผู้ป่วย จากการศึกษานี้ไม่พบว่าการตรวจมีผลต่อผลลัพธ์ของผู้ป่วย*