Impact of Time to Report Positive Hemoculture on Mortality in Surgical Critically Ill Patients with Septicemia

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Objective: *In patients receiving inappropriate empirical antimicrobial agents, delaying of time to report positive hemoculture (TRH) may cause delay switching from inappropriate antimicrobial agents to appropriate antimicrobial agents, and thus may increase mortality. We hypothesized that the patients with septicemia from different types of pathogens may have differ duration of TRH, and duration of TRH may had an impact on hospital mortality.*
Material and Method: *We performed observational study on the patients who were reported to have bacteremia or fungemia and admitted to the surgical intensive care unit, Phramongkutklao Hospital during a 2-year period. Type of pathogens grew in blood cultures and the sensitivities to antimicrobial agents were collected from blood culture reports. Patients were categorized into three groups based on their blood culture reports which were gram-positive (GP) bacteria, gram-negative (GN) bacteria, and fungus. Primary outcome was duration of TRH and secondary outcome was hospital mortality among the three groups of patients.*
Results: *There were 9, 32, and 7 patients for whom growth of GP bacteria (18.8%), GN bacteria (66.7%), and fungus (14.6%) were reported in their blood cultures respectively. Patients with fungemia had the longest TRH (130 hours, interquartile range (IQR) 100-137 hours), followed by patients with GN (64 hours, IQR 48-78 hours), and GP bacteremia (55 hours, IQR 42-71 hours) (p = 0.001). There was no difference in hospital mortality (GP 89%, GN 66%, fungus 71%, p = 0.4). TRH was found significantly longer in survivors (n = 14) (81 hours, IQR 56-105 hours) than non-survivors (n = 34) (64 hours, IQR 48-71 hours) (p = 0.035). In multivariate analysis, we found that every 1-hr increasing of TRH was associated with lower risk of hospital mortality with adjusted odd ratio of 0.95 (0.92-0.99, p = 0.01).*
Conclusion: *Patients with fungemia had significantly longer TRH than patients with GP and GN bacteremia. TRH was found significantly longer in patients with septicemia who survived than non-survivors.*

Keywords: *Hemoculture, Mortality, Septicemia, Bacteremia, Fungemia*

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Septicemia is a serious bloodstream infection that leads to morbidity and mortality, especially in an intensive care unit. Previous studies demonstrated that the incidence of sepsis and the number of sepsis-related deaths are increasing during 1979 through 2000[1]. In addition, one study, in emergency medical services encounters, found that while the crude rate of acute myocardial infarction hospitalization decreased by 2.7% per year, the crude rate of severe sepsis hospitalization increased by 11.8% per year during 2000 through 2009[2].

It is noticeable that effect of the "timing" is crucial and is considered as one of the primary concerns regarding to the management of patients with severe sepsis. For example, Surviving Sepsis Campaign (SSC) guideline clearly stated that, anatomical diagnosis of infection or source control should be diagnosed or excluded as rapidly as possible, intravenous antimicrobials should be administered within the first hour of recognition of sepsis[3], as its delay was found associated with increased mortality[4-6].

In addition, broad-spectrum antibiotics should be de-escalated to the most appropriate antimicrobial agent as soon as the susceptibility profile is known to improve the patient's outcomes[7-10]. Regarding the

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crucial concern of timing, the lag time in reporting positive hemoculture (TRH) associated with increased mortality is not clearly defined in patients with documented septicemia. We hypothesized that the patients with documented septicemia from different types of pathogens (i.e. fungus, gram-positive (GP), and gram-negative (GN) bacteria) may have differ duration of TRH, and duration of TRH may had an impact on hospital mortality.

Material and Method

Patients

We performed a retrospective observational study in patients with septicemia admitted to the surgical intensive care unit (SICU), Phramongkutklao Hospital, Thailand, during 2-year period (September 1, 2013 to October 1, 2015). Based on hemoculture reports, the patients were categorized into three groups which were GP bacteremia, GN bacteremia, and fungemia. The inclusion criteria were patients older than or equal to 18 years old and diagnosed with septicemia confirmed by hemoculture reports. Patients with previously known hemoculture before admission to the SICU or a hemoculture report indicating contamination were excluded from this study. Baseline characteristics of the patients were collected included age, sex, pre-existing diseases, Acute Physiology and Chronic Health Evaluation (APACHE) II severity score, and primary sources of infection. Initial and maximum serum lactate level, serum bicarbonate, and maximum dosage of norepinephrine during SICU admission were also measured. The types of pathogens grew in blood culture, their sensitivities to antimicrobial agents, and the types of initial empirical antimicrobial agents were also recorded. This study was reviewed and approved by the Royal Thai Army, Institutional Review Board.

Outcome measurements

The primary outcome was duration of TRH among three groups of patients, and secondary outcome was hospital mortality, SICU length of stay, and hospital length of stay among three groups of patients. TRH was defined as the time since the blood specimen of the patient was incubated to the time which pathogen species was identified and hemoculture was reported.

Statistical analysis

We tested for differences in baseline characteristics among the three groups of patients using the Kruskal-Wallis test for continuous variables and a Chi-square test for categorical variables, and we report the median and interquartile range (IQR). The differences in duration of TRH among patients with GP bacteremia, GN bacteremia, and fungemia were analyzed using Kruskal-Wallis test, and report the median and IQR. The differences in the duration of TRH between survivors vs. non-survivors were analyzed using Mann-Whitney U test, and report the median and IQR. In multivariate analysis for risk of hospital mortality, we tested for the influence of covariates, including gender, APACHE II score, types of pathogen, and TRH by using logistic regression analysis, and result was expressed as odds ratio (OR) with 95% confidence interval (CI). The differences were considered significant by using a two-tailed *p*-value of less than 0.05. All statistical analyses were performed using SPSS (version 23.0; IBM Corporation, NY, USA) statistical software packages.

Results

Patient characteristics and microbiological profiles

Forty-eight patients reported growth of bacteria (GN; n = 32; 66.7%, GP; n = 9; 18.8%) or fungus (n = 7; 14.6%) in their blood (Table 1). Thirty-two patients with GN bacteremia were identified GN pathogens including *Escherichia coli* (n = 2), *Klebsiella pneumoniae* (n = 4), *Pseudomonas aeruginosa* (n = 9), *Pseudomonas spp.* (n = 5), *Acinetobacter baumanii* (n = 4), *Acinetobacter spp.* (n = 1), *Proteus mirabilis* (n = 2), *Aeromonas hydrophila* (n = 2), and *Providencia spp.* (n = 3). Nine cases were reported of aerobic gram positive bacteremia including *Methicillin-resistant Staphylococcus aureus* (n = 3), *Streptococcus viridians* (n = 2), *Enterococcus faecalis* (n = 3), and *Corynebacterium spp.* (n = 1). All seven cases of fungemia were reported growth of *Candida albicans* in their blood cultures. There was no polymicrobial bloodstream infecion found. Of 48 patients, 32 patients (67%) received a combination of antimicrobial agents, which mostly were a combination of imipenem plus vancomycin (n = 10), meropenem plus vancomycin (n = 8), and imipenem plus colistin (n = 4). Sixteen patients (33%) received single antimicrobial agents as initial empirical antimicrobial agent included imipenem (n = 6), meropenem (n = 6), vancomycin (n = 3), and ceftriaxone (n = 1). Four of seven cases in patients with fungemia received amphotericin-B intravenously before hemoculture growth of *Candida albicans* was reported. According to sensitivities of pathogens to empirical antimicrobial agents, 25 patients (52%) reported pathogens had sensitivities to initial

Table 1. Baseline characteristic data among three groups of patients with documented septicemia

	Bacteremia		Fungemia (n = 7)	*p*-value
	Gram positive (n = 9)	Gram negative (n = 32)		
Age (year)	85 (66-94)	71 (56-76)	71 (59-87)	0.11
Male, n (%)	6 (66.7)	21 (65.6)	3 (42.9)	0.51
APACHE II score	16 (15-23)	16 (13-22)	17 (14-25)	0.83
Comorbid disease, n (%)				
Diabetes	2 (22.2)	15 (46.9)	4 (57.1)	0.31
Hypertension	4 (44.4)	20 (62.5)	5 (71.4)	0.50
Dyslipidemia	2 (22.2)	12 (37.5)	3 (42.9)	0.63
Chronic kidney disease	6 (66.7)	13 (40.6)	4 (57.1)	0.34
Liver disease	0 (0)	2 (6.3)	0 (0)	0.59
Source of infection; n (%)				
Lower respiratory tract	0 (0)	4 (12.5)	0 (0)	0.34
Intra-abdominal	2 (22.2)	10 (31.3)	3 (42.9)	0.68
Urinary tract	2 (22.2)	6 (18.8)	0 (0)	0.43
Skin and soft tissue	2 (22.2)	8 (25)	2 (28.6)	0.96
Intravascular catheter	0 (0)	2 (6.3)	1 (14.3)	0.50
Laboratory data				
Initial hemoglobin (g/dL)	9.9 (9.3-10.9)	10.4 (8.7-12.0)	11.8 (10.1-13.0)	0.24
Initial blood lactate (mmol/L)	2.5 (1.7-5.0)	2.8 (2.1-3.1)	2.2 (1.4-5.7)	0.89
Maximum blood lactate (mmol/L)	3.0 (2.5-6.8)	3.3 (2.6-3.9)	5.2 (2.0-11.8)	0.87
Initial serum HCO_3 (mmol/L)	22.5 (17.4-23.5)	19.8 (17.8-22.5)	15.0 (12.0-23.1)	0.36
Maximum dose of norepinephrine (mcg/kg/min)	0.89 (0.55-1.27)	0.41 (0.26-0.63)	0.90 (0.44-1.29)	0.02*

empirical antimicrobial agents, 11 patients (23%) reported that the pathogens resisted initial empirical antimicrobial agents, and 12 patients (25%) reported no sensitivities of pathogens to empirical antimicrobial agents. After receiving of positive hemoculture reports, 16 patients were de-escalated to appropriated antimicrobial agents; the remaining 32 patients were not de-escalated of initial empirical antimicrobial agents.

Most of the patients had intra-abdominal infection as a primary source of infection. There was no difference in APACHE II severity scores and other baseline characteristics among the three patient groups, except that the patients who had fungemia had received higher doses of norepinephrine (0.90 mcg/kg/min, IQR 0.44-1.20 mcg/kg/min), than patients with GP (0.89 mcg/kg/min, IQR 0.55-1.27 mcg/kg/min), and GN bacteremia (0.41 mcg/kg/min, IQR 0.26-0.63 mcg/kg/min) ($p = 0.02$); there was no difference in dose of norepinephrine between patients with fungemia vs. patients with GP bacteremia ($p = 0.95$).

Patients with fungemia had the longest TRH

Interestingly, we found that the patients with fungemia had significantly longer TRH (130 hrs, IQR 100-137 hours) than patients with GN bacteremia (64 hours, IQR 48-78 hours), and GP bacteremia (55 hours, IQR 42-71 hours) ($p = 0.001$) (Fig. 1). We also found that, compared to non-survivors, septicemia patients who survived had a significantly longer TRH (non-survivor 64 hours (IQR 48-71 hours) vs. survivor 81 hours (IQR 56-105 hours), $p = 0.035$) (Fig. 2).

Hospital outcomes among patients with GP bacteremia, GN bacteremia, and fungemia

We found no difference in hospital mortality [GP 88.9%, GN 65.6%, and fungus 71.4%, $p = 0.4$) (Fig. 3), SICU length of stay [GP 11 days (IQR 6-33 days), GN 13 days (IQR 8-20 days), and fungus 7 days (IQR 4-45 days), $p = 0.96$], and hospital length of stay [(GP 22 days (IQR 10-34 days), GN 29 days (IQR 21-44 days), and fungus 22 days (IQR 6-140 days), $p = 0.36$] among the three groups of patients ($p = 0.12$).

Impact of TRH on hospital mortality

Having found that survivors had significantly higher duration of TRH than non-survivors, we then further analyzed the effect of TRH on hospital mortality using logistic regression model for multivariate analysis.

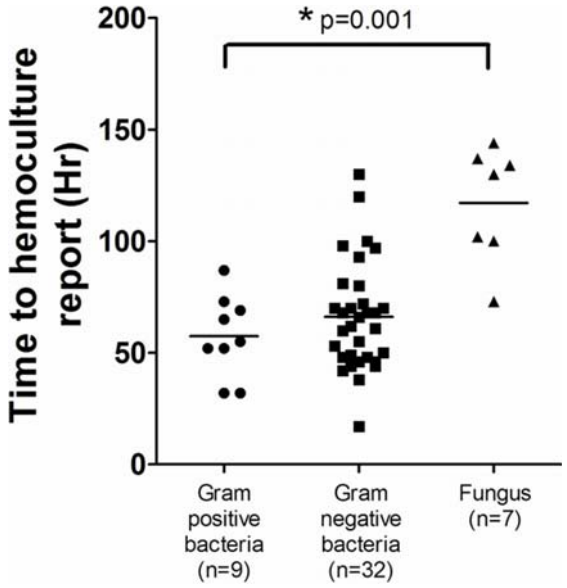

Fig. 1 Duration of time to report positive hemoculture (TRH) compared among three groups of patients (Krusskal-Wallis test, **p* = 0.001). Compared TRH between two groups of patients using Mann-Whitney U test, the *p*-values compared between fungus vs. gram negative bacteria, fungus vs. gram positive bacteria, and gram negative vs. gram positive bacteria were $p<0.001$, $p<0.001$, and $p = 0.5$, respectively.

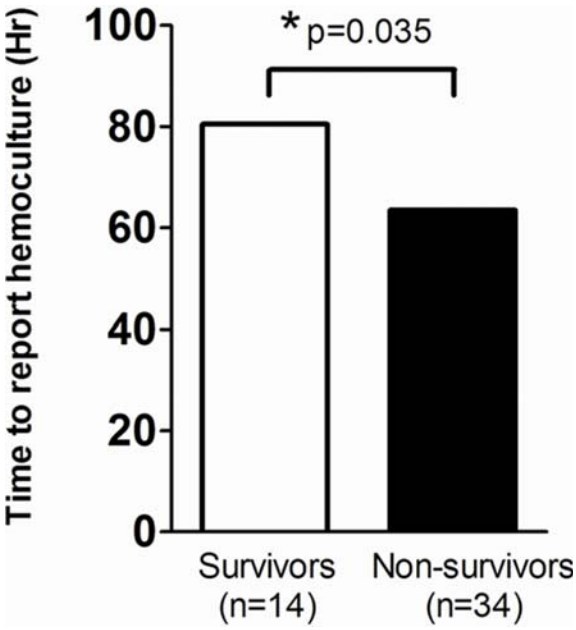

Fig. 2 Time to report hemoculture between survivors (n = 14) and non-survivors (n = 34) (80.5 vs. 63.5 hours) (Mann-Whitney U test, * $p = 0.035$).

After adjustment for the influence of covariates, including gender, APACHE II score, types of the pathogen, and TRH, we found that every 1-hr increasing of TRH was significantly associated with lower risk of hospital mortality with OR of 0.95 (95% CI 0.92-0.99, $p = 0.01$) (Table 2).

Discussion

Regarding our study, we found that the shortest TRH was demonstrated in the patients with GP bacteremia, followed by GN bacteremia, and fungemia, respectively. However, previous study[11-15] reported that patients with GN bacteremia had a shorter TRH than GP bacteremia, which contradicted to our study. One possible explanation of this may be because of our study was studied in patients admitted to SICU which the GN bacteremia are mostly infected by *Pseudomonas spp.* and they took longer TRH than other GN[16,17]. Therefore, it could affect to a longer overall TRH of GN bacteremia patient group in our study.

For hospital mortality, it can be seen that the hospital mortality of the patients in our study was quite high (66 to 89%). Possible explanation for this was we recruited only the patients with documented bloodstream infection (either bacteremia or fungemia) which this kind of patient had previously demonstrated significantly high mortality[18]. Our result was aligned to study by Smith et al which studied in 34 critically ill patients with bloodstream infection, and they found that an observed mortality of the patients with bloodstream infection was as high as 82.4%[18]. Also, in our study, at initial phase of septicemia, 11 patients (23%) were received inappropriate empirical antimicrobial agents, while 12 patients (25%) had no report of sensitivities of pathogens to empirical antimicrobial agents. This may additionally lead to a high mortality rate in our study.

In terms of TRH, previous studies demonstrated that a shorter TRH is associated with increased hospital mortality rate[12-16], this result was aligned to our finding which we found that every 1-hr increasing of TRH was associated with lower hospital mortality with an OR of 0.95. The reasons why non-survivor had shorter TRH, compared to survivor, may be because of a higher bacterial load in the blood of non-survival patients, or non-survivor may had severe bacteremia with more virulence pathogens cause bacteria grew in the blood quicker and may shorten the TRH. However, we have no data of a colony forming unit amount in our database collection. Therefore, this

assumption is hypothesis-generating only.

Compared to the patients with bacteremia, the maximum dose of norepinephrine was used more in the fungemia group. This result may be because of the patients with fungemia had a higher severity than the patients with bacteremia at initial stage of sepsis, as we found that our patients with fungemia had a trend of higher maximum blood lactate level compared to patients with bacteremia, this indicated the evidence of poorer tissue perfusion in fungemia group.

Possible clinical implications from this study were that the patients with shorter TRH may have potentials of higher mortality. On the other hand, patients with suspected septicemia and have longer TRH may have higher possibility of fungal infection than patients with shorter TRH. Nevertheless, this study has some limitations. Because of our retrospective study design and our study had a small sample size; an ability to detect clinical variables as significant predictors of hospital mortality was limited. We also had no data whether bloodstream infection in our patients were primarily from community or nosocomial infection. In addition, duration of TRH is influenced by several factors, such as incubation condition, blood volume in culture bottles, initial inoculums, virulence of each pathogen type, and bacterial species (for example, *Acinetobacter spp.* vs. *Acinetobacter baumanii*). These factors must be taken into consideration when analysis of TRH was considered as a primary outcome. Also, we studied in a single center and studied only in patients with reported positive pathogen in hemoculture; therefore, our results can not apply to a septic patient with negative hemoculture nor had polymicrobial bloodstream infection. Therefore, our results still need validation in further trials with a larger number of patients or multicenter study.

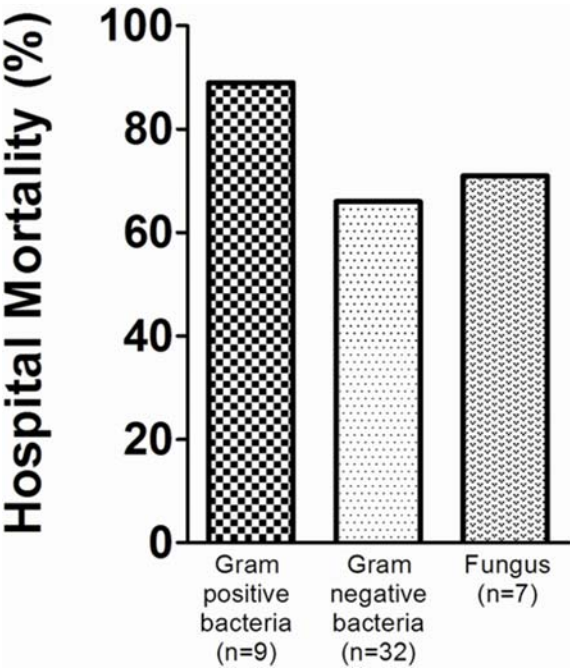

Fig. 3 Hospital mortality among three groups of patients (Krusskal-Wallis test, $p = 0.4$).

Conclusion

In critically ill surgical patients, we found that patients with fungemia had significantly longer TRH than patients with bacteremia. We also found that septicemia patients who survived had a significantly longer TRH than non-survivors. However, due to our small sample size study, further, well-controlled trials with a greater number of patients with septicemia are needed to validate our findings.

What is already known on this topic?

Patients with documented bloodstream infection had high mortality.

What this study adds?

Patients with fungemia had significantly longer time to report positive hemoculture than patients

Table 2. Multivariate analysis for hospital mortality

	Odds ratio	95% CI	*p*-value
Male (vs. female)	2.53	0.47-13.58	0.280
APACHE II (per score)	1.16	0.98-1.37	0.080
Gram positive (vs. gram negative)	4.33	0.46-19.72	0.100
Fungus (vs. gram negative)	2.56	0.08-15.93	0.600
Time to hemoculture report (per hour)	0.95	0.92-0.99	0.014*

(Analysis using logistic regression,* $p<0.05$)

with gram-positive and gram-negative bacteremia. Patients who had pathogen growth in hemoculture and survived had significantly longer TRH than patients who not survived.

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Potential conflicts of interest

None.

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ผลกระทบของระยะเวลาในการรายงานผลเพาะเชื้อจากเลือดกับอัตราตายในผู้ป่วยหนักศัลยกรรมซึ่งมีภาวะติดเชื้อในกระแสเลือด

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วัตถุประสงค์: ความล่าช้าในการรายงานผลเพาะเชื้อจากเลือดในผู้ป่วยที่มีภาวะติดเชื้อในกระแสเลือด และได้รับยาปฏิชีวนะไม่เหมาะสม อาจส่งผลให้เกิดความล่าช้าในการเปลี่ยนยาปฏิชีวนะที่เหมาะสม และอาจเพิ่มอัตราตายของผู้ป่วย การศึกษานี้มีสมมุติฐานคือ เชื้อก่อโรคที่ต่างชนิดกันอาจใช้ระยะเวลาในการรายงานผลเพาะเชื้อต่างกัน และระยะเวลาในการรายงานผลเพาะเชื้อที่ต่างกัน อาจมีผลต่ออัตราตายของผู้ป่วยที่มีภาวะติดเชื้อในกระแสเลือด

วัสดุและวิธีการ: ศึกษาโดยวิธีสังเกตการณ์เป็นระยะเวลา 2 ปี ในผู้ป่วยที่รับการรักษาตัวในหออภิบาลผู้ป่วยหนักศัลยกรรม โรงพยาบาลพระมงกุฎเกล้า และมีภาวะติดเชื้อในกระแสเลือด โดยแบ่งผู้ป่วยออกเป็น 3 กลุ่มตามชนิดของเชื้อก่อโรคที่รายงานในผลเพาะเชื้อจากเลือด ได้แก่ กลุ่มที่ผลเพาะเชื้อขึ้นแบคทีเรียกรัมบวก กลุ่มที่ผลเพาะเชื้อขึ้นแบคทีเรียกรัมลบ และกลุ่มที่ผลเพาะเชื้อขึ้นเชื้อรา โดยศึกษาเปรียบเทียบระยะเวลาในการรายงานผลเพาะเชื้อและอัตราตายระหว่างผู้ป่วยทั้ง 3 กลุ่ม

ผลการศึกษา: พบผู้ป่วยที่มีเชื้อก่อโรคขึ้นในเลือดเป็นแบคทีเรียกรัมบวก 9 ราย (ร้อยละ 18.8) แบคทีเรียกรัมลบ 32 ราย (ร้อยละ 66.7) และเชื้อรา 7 ราย (ร้อยละ 14.6) ผู้ป่วยที่มีผลเพาะเชื้อขึ้นเชื้อราพบว่ามีระยะเวลาในการรายงานผลเพาะเชื้อนานกว่าผู้ป่วยอีก 2 กลุ่มอย่างมีนัยสำคัญ (กลุ่มเชื้อรา 130 ชม. กลุ่มแบคทีเรียกรัมลบ 64 ชม. และกลุ่มแบคทีเรียกรัมบวก 55 ชม.) ($p = 0.001$) แม้ไม่พบความแตกต่างของอัตราตายระหว่างผู้ป่วยทั้ง 3 กลุ่ม ($p = 0.4$) แต่พบว่าผู้ป่วยที่รอดชีวิตมีระยะเวลาในการรายงานผลเพาะเชื้อนานกว่าผู้ป่วยที่เสียชีวิตอย่างมีนัยสำคัญ (81 ชม. กับ 64 ชม., $p = 0.035$) เมื่อวิเคราะห์โดยการควบคุมตัวแปร พบว่าทุกๆ 1 ชม. ของระยะเวลาในการรายงานผลเพาะเชื้อที่นานขึ้นจะสัมพันธ์กับอัตราตายที่ลดลงโดยมีอัตราส่วนออด (odds ratio) อยู่ที่ 0.95 (95% CI 0.92-0.99, $p = 0.01$)

สรุป: ผู้ป่วยที่ผลเพาะเชื้อจากเลือดขึ้นเชื้อราจะมีระยะเวลาในการรายงานผลเพาะเชื้อนานกว่าผู้ป่วยที่ผลเพาะเชื้อจากเลือดขึ้นแบคทีเรียกรัมบวกและกรัมลบ และพบว่าผู้ป่วยที่ติดเชื้อในกระแสเลือดซึ่งรอดชีวิตจะมีระยะเวลาในการรายงานผลเพาะเชื้อนานกว่าผู้ป่วยที่เสียชีวิตอย่างมีนัยสำคัญ