CORRELATION BETWEEN MAGNESIUM SERUM LEVELS AND PEAK EXPIRATORY FLOW IN CHRONIC ASTHMA PATIENTS

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ABSTRACT

***Background**: Magnesium Serum in some previous studies had been related to asthma events and severity levels. Correlation between magnesium serum levels and severity of asthma was controversies. It might be influenced by race, genetic pattern, diet and demography factor.*

***Objective**: The objective of this study was to know relationship between magnesium serum levels and predictive PEF (Peak Expiratory Flow) in chronic asthma patients in Yogyakarta.*

***Method**: This was a cross-sectional study; subjects were asthma patients who visited Pulmonology Outpatient Department in Dr. Sardjito General Hospital Yogyakarta, from September 2004 - March 2005. We performed clinical evaluation, magnesium serum levels (normal 0.65 mmol/L-1.04 mmol/L), predictive PEF and medications. Correlation between magnesium serum levels and PEF were analyzed by Pearson correlation test. This study used analysis of variance (anova) to analyze mean difference among more than 2 groups and multiple regressions to know the variables which influenced serum magnesium levels.*

***Result**: There were 62 asthma patients in this study. There was no hypomagnesaemia. The mean of magnesium was 0.89 ± 0.08 mmol/L. Results showed that serum magnesium levels and predictive PEF had weak correlation (r= 0.281; p=0.027). There was no significant correlation between admission rate to Emergency Room due to asthma by magnesium levels (r=0.029 p= 0.8).*

***Conclusion**: Results showed weak relationship serum magnesium levels and PEF in chronic asthma patients.*

***Keywords**: bronchial asthma; serum magnesium levels, Peak Expiratory Flow rate*

INTRODUCTION

Asthma is a serious problem. The prevalence of asthma increased in the developing country during the last 20 years [1]. The increasing number of asthma patients registered in Indonesia is approximately 5-7% [2]. Magnesium might be a risk factor for severity of asthma. Magnesium is closely involved in numerous important biochemical reactions in the body, particularly those processes that entail the formation and utilization of ATP. As a cofactor of over 300 intracellular enzyme reactions utilizing high-energy phosphate bonds, magnesium has been implicated in smooth muscle contraction[3,4]. In fact, magnesium is a chemical element with modulator effects on the contractile state of smooth muscle cells in various tissues: hypomagnesaemia leads to contraction and hypermagnesaemia leads to relaxation. Magnesium has been shown to relax bronchial smooth muscle in vitro and to bronchodilate asthmatic airways in vivo[4,5,6]. Potential mechanisms for the direct relaxing effects of magnesium on bronchial smooth muscle was including calcium channel blocking properties, inhibition of cholinergic neuromuscular transmission with decreased sensibility to the depolarizing action of acetylcholine, stabilization of mast cells, and reduce respiratory burst activity of neutrophil [5].

In accordance with this hypothesis, a study involving 2,633 subjects in England, demonstrated that dietary magnesium intake is independently related to lung function and to the occurrence of airway hyperreactivity, and wheezing[7].Asthma patients with hypomagnesaemia had a correlation with severity of asthma compare with normomagnesaemia population[8].Study with 33 subjects in Thailand found serum magnesium in severe asthma low than serum magnesium in normal

population[9]. Some studies and meta-analysis reported that magnesium intravenous effective to treat intermediate and severe acute exacerbation of asthma[10,11,12,13].

Correlation between serum magnesium and asthma is controversy. The concentration of serum magnesium in asthma population in Yogyakarta is unknown. The prevalence of hypomagnesaemia and evidence of the correlation can support of the prevention and therapeutic of asthma. Status of magnesium was measured by serum magnesium level[14]. Peak Expiratory Flow (PEF) was used to measure lung function[15,16].

The hypothesis was the serum magnesium had the correlation with Peak Expiratory Flow (PEF) in chronic asthma patients.

SUBJECTS AND METHODS

It was a cross-sectional study. Subjects were chronic asthma patients who visited to Pulmonology Outpatient Department Dr. Sardjito General Hospital Yogyakarta, from September 2004 - March 2005.

Eligibility criteria for entry in this study were as follows: diagnosed as chronic asthma patients; more than 18 years old; signs informed consent. The exclusion criteria were as follows: used diuretic, thiazid, aminoglicoside, siclosporine, cisplatin, foscarnet, laxative, oral magnesium, enema, antacide, magnesium intravena; alcohol abuse; history of diabetes mellitus, heart and renal diseases; current diarhhoea; and current pregnancy.

The independent variable is magnesium serum. The dependent variable is PEF. The study used consecutive sampling. Chronic asthmatics, who agreed to take apart in the study were interviewed and clinically evaluated. A questionnaire that included the patients age, sex, duration of asthma, number of hospitalization was obtained from each subject. PEF was measured 3 times to get the best value. Measurements of serum magnesium level were carried out using colorimetric from Randox.

STATISTICAL ANALYSIS

The statistical analysis was done using SPSS 11. Characteristic features were showed by mean and standard deviation. Distribution was tested by Kolmogorov Smirnov. Correlation between serum magnesium levels and PEF were analyzed by Pearson correlation test. In all tests, a p value of <0.05 was considered significant. The value of power was 90%. We used anova to compare mean when analyze more than 2 group.

RESULTS

Total 62 chronic asthma patients were studied. They were 26 male subjects (41.9%) and 46 female subjects (58.1%). Mean of ages were 44.46±11.68 years. Mean of magnesium was 0.89 ± 0.08 mmol/L. There was no prevalence of hypomagnesemia. The study found 1 (2%) subject had hypermagnesaemia and 61 (98%) normomagnesaemia subjects.

Mean of predictive PEF value was 61.69±18.16%. Mean duration of asthma was 10.34±8.88 years. The study found 6 subjects (9.7%) intermittent asthma, 6 subjects (9.7%) mild persistent asthma, 15 subjects (24.2%) intermediate persistent asthma and 35 subjects (56.5%) severe persistent asthma (table 1). Results showed that magnesium serum levels and predictive PEF had weak correlation ($r= 0.281$; $p=0.027$) (table 2). The study used scatter plot to show correlation between magnesium serum and PEF (graphic 1).

Table 1. Characteristic features

Variable	Proportion or mean ± SD	95% CI	Minimum - Maximum
Sex			
Male	26 (41.9%)		
Female	36 (58.1%).		
Age (years)	44.46±11.68	41.4 – 47.4	18-66
Magnesium (mmol/L)	0.89 ± 0.08	0.87 – 0.91	0.7-1.2
hypomagnesemia	0		
normomagnesemia	61 (98%)		
hypermagnesemia	1 (2%)		
PEF predictive (%)	61.69±18.16	57.0 – 66.3	30-101
Duration of asthma	10.3	8.1 – 12.5	1-35
Severity:			
intermitten	6 (9.7%)		
mild persistent	6 (9.7%)		
intermediate persistent	15 (24.2%)		
severe persistent	35 (56.5%)		

Table 2. Correlation between Magnesium and PEF

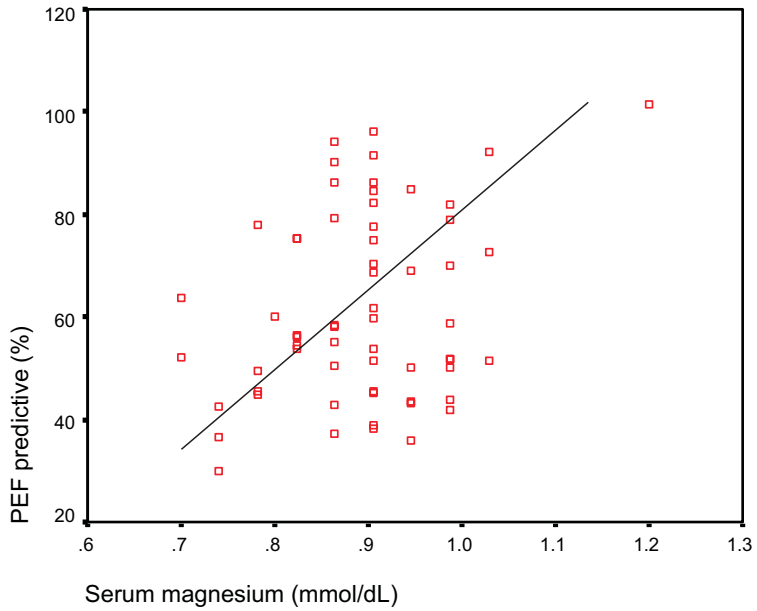
Variabel	*r*	*p*
Magnesium	0.281	0.027*

*significant (<0,05), Pearson test

Table 3. Correlation between magnesium serum and admission to emergency room or hospitalization

Variabel	*r*	*p*
Number of admission to ER**	0.029*	0.8
Number of hospitalizations***	0.13*	0.13

*used spearman correlation test
** Number of admission to ER on the last one year.
*** Number of hospitalizations on the last one year.



Graphic 1. *Scatter Plot* of correlation of predictive PEF (%) and magnesium serum (mmol/L).

DISCUSSION

This study did not find hypomagnesemia. This result was different with previous study [8,17]. This result might be interfered by some factors including diet and genetic pattern. The type of diet of Yogyakarta population and the method of cooking may influence magnesium status. Some impairment and variant of genetic pattern could also influence magnesium absorption from intestinal and magnesium excretion through renal [18,19,20]. This factor could bias of the study. The tight exclusion criteria might be excluding the population of hypomagnesemia. But the same exclusion criteria was used on Alamoudi study in Arab Saudi and found prevalence of hypomagnesemia at approximately 27%[8,17].

The serum magnesium may not reflect a true status of magnesium. Magnesium predominantly was in intracellular and only 1% in serum[21]. The correlation between concentration of serum magnesium and intracellular magnesium is still unclear. Zervas reported that the asthmatic acute attack patients had erythrocytes magnesium level lower than healthy people or chronic asthma patients, but the concentration increased when their condition improved [21]. He reported that erythrocytes magnesium level may decrease although the magnesium serum did not change[21]. However, a more extended research is needed to support this argument. We suggested a larger sample in next extended study to achieve a more conclusive result.

CONCLUSION

The study showed a weak correlation of serum magnesium and PEF(r=0.28 p<0.05). This result could be influenced by some factors. The strong correlation was higher in hypomagnesemia population than in normomagnesaemia population. This study did not have a hypomagnesaemia population so that we cannot analyze this probability.

The uncontrolled factor like allergen factor, air pollution infection, weather, emotion, food and additive might cause bias in this study.

REFERENCES

1. Global Initiative for Asthma (GINA), 2005, Global Strategy For Asthma Management and Prevention, National Instititute of Health. www.Ginasthma.com,
2. Sundaru, H. 2001, Asma Bronkial, in S. Suyono, , S. Waspadji., (eds) *Buku Ajar Ilmu Penyakit Dalam*, Balai Penerbit FKUI p. 21-32
3. Reinhart, R..A., 1988, Magnesium Metabolism: a Review with Special Reference to the Relationship Between Intracellular Content and Serum Level, *Arch Internal Medicine* 148:2415-2420

4. Spivey, W. H., Skobelpff, E. M, Levin R.M., 1984, Effect of Magnesium Chloride on Rabbit Bronchial smooth Muscle, *Annual Emergency Medicine* 19:1107-1112
5. Dominguez L.J, Barbagallo, M., Lorenzo, G. D., Drago A. *et al.,* 1998, Bronchial Reactivity and Intracellular Magnesium: a Possible Mechanism for the Bronchodilating Effects of Magnesium in Asthma*, Clinical Science* 95: 137-142.
6. Fassler, C.A., Rodriguez, M., Badesch, D.B., Stone, W.J, Marini J.J , 1985, Magnesium toxicity as a Cause of Hypotension and Hypoventilation. *Arch Internal Medicine* 145, 1604-1606
7. Britton, J., Pavord, I., Richards, K., 1994, Dietary Magnesium, Lung function, Wheezing, and Airway Hypereactivity in a Random Adult Population Sample, *Lancet* 344: 357-62
8. Alamoudi, O. S, 2000, Hipomagnesemia in Chronic Stable Asthmatics: Prevalence, Correlation with Severity and Hospitality, *Europe Respiratory Journal*, 16: 427- 431
9. Alter H. J., Thomas D. K. Hilty W. M., 2000, Intravenous Magnesium as an Adjuvant in Acute Bronkospasme: A Meta Analysis, *Annals of Emergency Medicine* vol 36:191-197.
10. Ciarallo, L., Broisseau, D. Reinert, S., 2000, Higher Dose Intravenous Magnesium Therapy for Children with Moderate to Severe Acute Asthma, *Arch Pediatric Adolescence Med* ; 154 (10):979-83
11. Silverman R. A., Osborn H. Runge J., Gallagher E. J., 2002, IV Magnesium Sulfate in the Treatment of Acute Severe Asthma, a Multicenter Randomized Controlled Trial, *CHEST*, 22 : 489-497.
12. Ian J , Johnson K , Montelpare , McAuliffe, 2002, Variability within individuals of plasma ionic magnesium concentrations, *BMC Physiology,* 2:6 ,*www.biomedcentral.com*
13. National Asthma Education an Prevention Program.Expert (NAEPP) ,1997, Panel Report 2: guidelines for Diagnosis and Management of Asthma, Bethesda, MD: National Institutes of Health, in B.D. Rose (Eds), *UpToDate* version 14.1
14. Prasson J.,Mani S.K.,2003, Peak Expiratory Flow vs spirometry in Patients with Asthma, *www.rcjournal.com*
15. Alamoudi, O.S., 2001, Electrolyte Disturbance in Patients With Chronic, Stable Asthma, *Chest* 120:431-436
16. Agus, Z. A.,2005 [1], Causes of Hypomagnesemia, dalam B.D Rose, *UpToDate ed 14.1*
17. Agus, Z. A.,2005 [2], Causes and Treatment of Hypermagnesemia, dalam B.D Rose *UpToDate ed 14.1*
18. Agus, Z. A.,2005 [3], Regulation of Magnesium Balance, dalam B.D Rose *UpToDate ed 14.1*
19. Zervas, E., Papatheodorou G., 2003, Reduced Intracelluler Mg Concentration in Patients With Acute Asthma, *Chest*:123:113-118
20. Cohen J, 1977, *Statistical Power Analysis for the Behavioral Science*, Academy Press, p. 93
21. Zervas E., Loukides S., Papatheodorou G., Psathakis K., Tsindiris K., Panagou P., Kalogeropoulos N., 2000, Magnesium levels in plasma and erythrocytes before and after histamine challenge, *Eur Respir J*; 16: 621-625

PROGNOSTIC FACTORS OF LEPTOSPIROSIS PATIENTS IN DR. SARDJITO GENERAL HOSPITAL, YOGYAKARTA, INDONESIA

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ABSTRACT

Background: *Leptospirosis, an infectious disease that affects humans and animals, is a common zoonosis with a variety of clinical manifestations. Yogyakarta is one of the cities with a high incidence of leptospirosis. It is important to recognize the clinical features and prognostic factors of this disease. Severe disease can be fatal, although majority of cases are mild and self-limited.*

Objective: *To determine the prognostic factors for leptospirosis that associated with mortality in patients with leptospirosis in Dr. Sardjito General Hospital, Yogyakarta.*

Methods: *We conducted a retrospective study of data collected in our hospital between Jan 2010 until May 2011, from whom the diagnosis of leptospirosis was confirmed based on pertinent clinical and epidemiological data and positive serology.*

Result: *Thirty two patients were included in this study, including 29 survivors (90.62%) and 3 non-survivors (9.38%). Of these 32 patients, 26 patients (81.25%) were admitted to the medical ward and 6 patients (18.75 %) were admitted to the ICU. Multivariate logistic regression demonstrated that three factors were independently associated with mortality: higher level of potassium (OR 10.8; CI 1.194-97.728; p<0.01) on admission and neurological dysfunction (altered mentation or seizure) (OR 30; CI 4.367–206.07; p<0.01)*

Conclusion: *The mortality of leptospirosis remains high despite improvements in patients care. In order to improve the early treatment of high-risk patients, these higher levels of potassium on admission and neurological dysfunction, which are associated with mortality, can be used at the time of admission as prognostic factors.*

Keywords : *Leptospirosis, prognostic factors, mortality*

Background

Leptospirosis is the most widespread zoonosis with recent outbreaks in several Asian, Central, and South American countries.[1] Leptospirosis is a *zoonotic* disease, which is caused by *Leptospira* and transmitted to human by contact with *Leptospira* contaminated animal urine or *Leptospira* contaminated environment. Thirty-two patients with Leptospirosis had admitted in Dr. Sardjito General Hospital from January 2010 until May 2011.[2] In Indonesia, the spread of leptospirosis is in the Province of West Java, Central Java, Yogyakarta, Lampung, South Sumatra, Bengkulu, Riau, West Sumatra, North Sumatra, Bali, NTB, South Sulawesi, North Sulawesi, East Kalimantan and West Kalimantan. The mortality rate of leptospirosis in Indonesia is high, reaching 2.5 to 16.45%. At the age of over 50 years mortality rate can be up to 56%. When Leptospirosis patient is accompanied by a yellow lining of the eye indicating damage to liver tissue, the risk of death will be higher. Several publications reported mortality rates between 3% -54% depending on the infected organ system.[3]

We believe that early evaluation of disease severity at the time of admission might be useful in improving the care of patients with leptopsirosis.

Objective

The purpose of this study is to determine the prognostic factors for leptospirosis that associated with mortality in patients with leptospirosis at Dr. Sardjito Hospital, Yogyakarta.

Methods

Patient population. This study was performed in Dr. Sardjito General Hospital, Yogyakarta. All patients admitted over period January 2010 until May 2011 with suspected leptospirosis were retrospectively included in the study. Leptospirosis was defined in accordance with modified Feine's score criteria. Briefly, six clinical criteria (headache, fever, conjunctiva suffusion, meningeal signs, myalgia, and jaundice), two laboratory-determined criteria (albuminuria or azotemia), and epidemiological criterion (contact with rats or contaminated water) were scored. Leptospira was suspected when the score was ⩾ 20, with a strong presumption when the score was > 26. In every case, the diagnosis was later confirmed using IgM ELISA (Panbio, Australia) for leptospirosis. From the total of 39 patients tested, it was found that 32 patients were positive for IgM ELISA and were included in the final analysis. We did not identify the serogroups involved. [4,5]

When the diagnosis had been confirmed, the patients' medical records were reviewed. The demographic data (age, gender, and occupation) and epidemiological data (exposure and time between the onset of clinical signs and hospitalization) were collected.

Clinical definitions. From the clinical data collected, there were presence of jaundice, fever (temperature ⩾ 38.5°C), and dyspnea (respiratory rate > 20). Organ dysfunction was defined as: (a) Neurological: presence of seizures or altered sensorium; (b) Respiratory: abnormal chest x-ray, clinical evidence of pneumonia or pleural effusion; (c) Cardiac: evidence of cardiac failure, S3 or EKG changes : rhythm abnormalities (sinus tachycardia [> 120 bpm], bradycardia [< 50 bpm], atrial or ventricular extra-systoles, and atrial fibrillation), repolarization abnormalities (T wave inversion, abnormal positive T wave, and depression of the ST segment), or conduction abnormalities (right or left branch block, left anterior hemiblock, and atrioventricular block); (d) Renal: oliguria (<0.5 L/24 hours) or serum creatinine >3 mg/dL; (e) Hepatic: serum bilirubin >10 mg/dL and elevated hepatic enzymes, hepatic encephalopathy; (f) Bleeding: petechiae/purpura, hemetemesis, hemoptysis, epistaxis, or melena.

Outcome. The patients were observed until they completely recovered and discharged from the hospital or until they died. The cause of death was registered in every case, but no postmortem examinations were performed. [4,5]

Statistical Methods. Results are expressed as means ± SD or as percentages. Clinical and laboratory data were statistically analyzed with the use of the χ^2 tests or Fisher's exact test for the comparison of proportions and analysis of variance for the comparison of intergroup difference. Risk factors for death were identified with the use of the χ^2 statistic for the differences in the distribution of categorical variables between survivors and non-survivors. Variables found to be relevant and associated with death ($p < 0.05$) were entered in a multiple logistic regression model (SPSS software for Windows). Adjusted odds ratios and 95% confidence intervals were calculated. A value of $p < 0.05$ was considered significant. [6,7]

Result

Thirty-two patients met the inclusion criteria, including 29 survivors (90, 62%) and 3 non-survivors (9.38%). Of these 32 patients, 26 patients (81.25%) were admitted to the medical ward and 6 patients (18.75 %) were admitted to the ICU. The 3 non-survivors all died in medical ward. All death was attributed to irreversible septic shock. The mean hospital stay ± SD for the survivors was 10.4 days ± 4.2 days. Demographic data are presented in Table 1.

Table 1. The mean hospital stay ± SD for the survivors

Variabel	Survivors	Non-survivors	p
Gender, n (%)			
Man	21	3	
Woman	8	0	0.55
Age (years), mean ± SD	42.41 ± 13.44	57.33 ± 14.43	0.2
20-46 years	17	0	
47-76 years	12	3	0.092
At risk occupation, n (%)			
Yes	12	2	
No	17	1	0.57
Rodent exposure, n (%)			
Yes	21	2	
No	8	1	1
Hemodialisis, n			
Yes	10	1	
No	19	2	1
ICU, n			
Yes	4	2	
No	25	1	0.083

The mean age ± SD for the survivors was 42.41 years ± 13.44 years, and for the non-survivors was 57.33 years ± 14.43 years. In age variable there was no differences between the survivors and non survivors (p = 0.092)

The gender ratio between male and female patients with leptospirosis was 3:1, man 75% and women 25%. There was no differences in gender between the survivors and non survivors (p = 0.55)

Table 2. Clinical sign and symptoms in survivors and non survivors among patients with Leptospirosis

Sign and symptoms	Survivors (n = 29)	Non survivors (n = 3)	p value
Fever	2	0	1.000
Dyspnea	15	2	1.000
Jaundice	19	3	0.534
Cardiovascular collapse	16	1	0.589
Conjunctival Suffusion	21	2	1.000
Oliguria	9	3	0.044*
Respiratory symptoms	7	2	0.184
Neurological dysfunction	0	2	0.006*
Hemorragic	1	1	0.181

*statistically significant

Table 3. Result of univariate analysis of laboratory values between survivors and non-survivors among patients with leptospirosis.

Value	Survivors (n = 29)	Nonsurvivors (n = 3)	p
Urea nitrogen (mg/dL)	79.12 ± 43.14	139 ± 6.56	0.000*
Creatinine (mg/dL)	6.17 ± 3.32	8.43 ± 3.75	0.275
Total protein (g/L)	5. 71 ± 0.69	5.6 ± 0.21	0.793
Albumin (g/L)	2.11 ± 0.46	1.61 ± 0.27	0.081
Total Bilirubun (mmol/L)	7.76 ± 8.59	14.48 ± 4.78	0.099
Sodium (mmol/L)	125.9 ± 9.67	144.2 ± 15.77	0.027
Potassium (mmol/L)	4.05 ± 0.81	5.29 ± 1.08	0.02*
Chloride (mmol/L)	93.98 ± 10.23	100 ± 15.62	0.359
Uric (mg/dL)	9.46 ± 3.66	11.5 ± 2.95	0.286
AST (IU/L)	71 ± 46.2	123 ± 110.59	0.365
ALT (IU/L)	53.58 ± 24.38	84.67 ± 48.44	0.064
Glucose (mg/dL)	132.17 ± 72.02	115 ± 33.41	0.628
WBC count (10^9/L)	14.39 ± 5.29	13.74 ± 2.53	0.837
Platelet count (10^9/L)	115.41 ± 87.05	76.67 ± 84.51	0.301
Hemoglobin (g/L)	11.84 ± 1.54	11.5 ± 0.26	0.305
Hematocrit (%)	33.19 ± 4.59	32.5 ± 1.9	0.651

*statistically significant

Table 4. Result of multivariate stepwise logistic regression analysis of risk factors for patients with Leptospirosis.

Risk Factor	OR	95% CI	P value
Oliguria	0.75	0.541 – 1.04	0.018
Urea nitrogen (mg/dL)	0.813	0.642 - 1.028	0.025
Potassium (mmol/L)	10.8	1.194 – 97.728	0.009
Neurological symptoms	30	4.367 – 206.07	0.000

Discussion

In this study, several poor prognostic factors in leptospirosis have been identified, including higher admission serum potassium and neurological dysfunction (altered mentation or seizures) were associated with high in-hospital fatality.[8] In another retrospective study, advanced age, oliguria, cardiac arrhythmia, dyspnea, and pulmonary rales were associated with mortality.[9]

Mortality in leptospirosis ranges from 1% to 20 %. [1, 10, 11] In South India, a mortality of 5.32% was reported from Kolenchery and 3.5% from Madras. The study showed that there was no difference in mortality when patients were grouped by age, gender, at-risk occupation, or rodent exposure.

ARF occurs in 16% to 40% of leptospirosis and approximately 30% to 50% are non-oliguric,

with a worse prognosis for oliguric renal failure.[10] Ninety-seven percent of the patients had renal failure and all patients who died were oliguric. In our study, oliguria was a very sensitive criterion of the severity of the disease. Similarly, Seguro et al noted that the mortality rate for oliguric patients with acute renal failure were higher than that for patients with persistent dieresis. Again, 90% of leptospirosis is anicteric with a lower mortality compared with icteric forms. In our group, 68% of patients were icteric and there was no difference in mortality.

Neurologic dysfunction (altered mentation or seizures) was the most significant predictor of mortality; most patients with neurologic dysfunction also had significant renal and hepatic disease contributing to encephalopathy. Altered mental status was the strongest independent predictor of death in urban leptospirosis in Brazil; other poor prognostic factors were oliguria, advanced age, renal and respiratory insufficiency. In this study, there was an association between altered mental status and mortality.[12]

Conclusions

The mortality of leptospirosis remains high despite improvements in patients care. In order to improve the early treatment of high-risk patients, these two clinically and two laboratory criteria, independently associated with mortality, could be used at the time of admission.

References

1. Vinetz JM. Leptospirosis. *Curr Opin Infect Dis*. **2001**; 14:527–538.
2. Tunissea A. Anlisis Spasial Faktor Risiko Lingkungan pada Kejadian Leptospirosis di Kota Semarang. **2008;** 1-4.
3. Murtiningsih B, Budiharta S, Supardi S. Faktor Risiko Leptospirosis di Provinsi Yogyakarta dan sekitarnya. **2005**; BKM; 21:1-8.
4. Dupont H, Perdrizet DD, Perie JL, et al. Leptospirosis: Prognostic Factors Associated with Mortality. *Clinical Infectious Diseases* **1997**; 25:720–4.
5. Unnikrishnan D, Pisharody R, Vijayalakshmy N. Prognostic Factors in Leptospirosis. A study from Kerala, India. *Infectious Diseases in Clinical Practice* **2005;** 13:104-107.
6. Riwidikno H. Uji Beda. In Statistik Kesehatan : Belajar mudah teknik analisis data dalam Penelitian Kesehatan. Jogjakarta: Mitra Cendikia Press. **2007**; 4-70.
7. Dahlan, M. Sopiyudin. Uji Hipotesis. In statistika untuk Kedokteran dan Kesehatan. Jakarta : PT. Arkans. **2006**; 1-135.
8. Lopes AA, Costa E, Costa YA, et al. The association between serum potassium at hospital admission and the case-fatality rate of leptospirosis in men. *Rev Inst Med Trop Sao Paulo*. **2001**; 43:217–220.
9. Daher E, Zanetta DM, Cavalcante MB, et al. Risk factors for death and changing patterns in leptospirosis acute renal failure. *Am J Trop Med Hyg*. **1999**; 61:630–634
10. Levett PN. Leptospirosis. *Clin Microbiol Rev*. **2001**;14:296–326
11. Muthusethupathi MA, Shivakumar S, Vijayakumar R, et al. Renal involvement in leptospirosis—our experience in Madras City. *J Postgrad Med*. **1994**; 40:127–131.
12. Ko AI, Galvao Reis M, Ribeiro Dourado CM, et al. Urban epidemic of severe leptospirosis in Brazil. Salvador Leptospirosis Study Group. *Lancet*. **1999**;354:820–825