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Correlation between Acute Stroke and Concentration of Creatine Kinase Myocardial Band, Troponin T, Hemoglobin, and Electroyltes

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Abstract

Currently, there were only a few studies regarding the correlation between concentration of creatine kinase-myocardial band (CKMB), troponin T, hemoglobin, and electrolytes and acute stroke. This study aimed to understand the difference of mean concentration of CKMB, troponin T, hemoglobin, and electrolytes and their relationship with acute stroke. This was a cross sectional study using samples of 30 subjects who were selected with consecutive non random sampling. The subjects were acquired from acute stroke patients who were proven clinically and using computed tomography (CT) scan at Haji Adam Malik General Hospital. Demographic data were analyzed using descriptive statistic. The result showed that there were no difference in demographical characteristics between the subjects. In conclusion, there were no differences in mean of CKMB, troponin T, hemoglobin, and electrolytes in acute stroke. No correlation was found between acute stroke and concentration of CKMB, troponin T, hemoglobin, and electrolytes.

Introduction

Stroke is the leading cause of disability and is the third most common cause of mortality after heart disease and cancer. Stroke inflicts a huge economic burden because of its effect on treatment cost and disability (Lahano et al., 2014). Patient of post-ischemic stroke need to undergo early movement training in order to obtain optimal recovery. Administration of movement training in patient should involve the family to allow increased frequency. Movement training in stroke patient is better to be carried out at 6 months after the attack (Mahmudah, 2012).

It is known that both ischemic and hemorrhagic stroke could inflict negative influence on cardiac function (Bhavani & Tagore, 2015). Increase in cardiac enzymes, change of electrocardiography (ECG), or left ventricular dysfunction seen in echocardiography suggested cardiac abnormality. The underlying mechanism of this dysfunction is unclear. Increase in cathecolamin is presumed to be responsible for myocardial dysfunction in numerous cases (Hasirci et al., 2013).

In cardiac damage, concentration of numerous cardiac enzyme increase dramatically. Although the concentration of Creatine Kinase-Myocardial Band (CK-MB) is usually used as marker of myocardial infarction, it could also be found in non-cardiac disorder, therefore decreasing its specificity (Hasirci et al., 2013).

A case control study conducted

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from January to April 2015 found that the concentration of CKMB increased significantly in ischemic stroke patient compared to control (CK-MB concentration of 24.0±16.6 versus 10.6±4.7, p-value<0.0001). The probability of cardiac damage need to be monitored in stroke patient (Bhavani & Tagore, 2015).

Troponin is a complex protein which is situated in filament of striated muscle and consists of three subunit, namely Troponin T (TnT), Troponin I (TnI), and Troponin C (TnC) (Nigam, 2007). Troponin T is a specific and sensitive marker of myocardial necrosis and could be used as prognostic and diagnostic tools in patient with acute coronary syndrome. Troponin could be detected in blood in 3-6 hours after the onset of myocardial infarct symptoms and its concentration remains elevated for 7-14 days. Troponin T has a half time of 90 minutes. Persistent increase in the third or fourth day shows degradation of contractile element as marker of irreversible cell damage (Wells et al., 2008; Jagannadharao et al., 2010; Al-otaiby et al, 2011).

Increase in Troponin T concentration occurred in 5-34% of acute ischemic stroke patients. The cause of the increase was still unclear but was presumed to occur concurrently with acute coronary syndrome which cause myocardial necrosis (Cojocaru et al., 2014).

Cerebral infarct is a result of imbalance between energy supply and demand. Energy availability played important role in the outcome of stroke. It could be hypothesized that blood oxygen binding capacity might influence the outcome of ischemic brain tissue (Kimberly et al., 2011).

Low and high hemoglobin level at hospital admission in acute ischemic stroke patient were correlated with poor outcome and prognosis after acute ischemic stroke. Patients with high hemoglobin level during admission showed significantly higher disability when they were discharged (Furlan et al., 2016).

One of four acute ischemic stroke patients experienced anemia. Anemia was a predictor of mortality in acute ischemic stroke patient (Hao et al., 2013).

The correlation between high and low hemoglobin level and increase in stroke risk was found in women but not in men. The mechanism underlying this gender difference in stroke risk is still poorly understood. Middle aged women had low risk of cardiac disease and stroke compared to similar aged men. However, the risk of stroke was increased twice 10 years after menopause due to decrease of estrogen concentration (Panwar et al., 2016).

Elevated level of hemoglobin could also predict increase in 7^{th}, 30^{th}, and 90^{th} day mortality compared to normal hemoglobin level at admission. Meanwhile, low hemoglobin level at admission was correlated with increase in length of stay and 90 days mortality in acute ischemic stroke patient (Furlan et al., 2016).

Anemia is often found in acute stroke patient with a prevalence of 30%. A significant proportion of stroke patient suffered anemia during stroke onset and it was correlated with increase in 1 year mortality rate (Barlas et al., 2016).

Stroke patient died due to their primary disease or its complication. Medical treatment need to emphasize prevention of subacute complication, such as malnutrition, aspiration, pneumonia, electrolyte disturbance, urinary tract infection, alimentary tract dysfunction, pulmonary embolism, deep vein thrombosis (DVT), joint abnormality, and contracture (Siddiqui et al., 2012).

Electrolyte disturbance such as hypernatremia or hyponatremia were caused by syndrome of inappropriate anti diuretic hormone (SIADH), increase in brain natriuretic peptide (BNP), and imbalance in intake and expenditure which could result in seizure and even death (Siddiqui et al., 2012; Chakraborty et al., 2013).

High sodium intake might cause mortality in stroke patient. High potassium excretion was correlated with a decrease in cerebrovascular disease risk and increase in potassium intake was inversely related with mortality in coronary artery disease (Farahmand et al., 2013; Roy et al., 2014).

Method

This was an analytical descriptive study with cross-sectional design conducted from February to September 2016 in stroke patient who were treated at Rindu A4 Neurological Ward of Haji Adam Malik Hospital Medan. The subjects were recruited through consecutive

sampling. The inclusion criteria were: a) all stroke patient in acute phase who were clinically proven and had been confirmed by CT scan examination, b) agreed to participate in the study. The exclusion criteria were patient with the following condition: a) recurrent stroke, b) cardioembolic stroke, c) gastroenteritis, d) chronic kidney disease, e) history of diuretic use, f) anemia, g) hematologic malignancy, h) heart failure, i) history of cardiac surgery, j) history of myocardial infarction, and k) history of chronic atrial fibrillation.

The acquired data were then analyzed for their correlation with stroke. CKMB concentration was classified as normal if it was <25 U/L and as high if it was >25 U/L (D, Bhavani and Tagore, 2015). Troponin T concentration was classified as normal if it was <0.1 g/L and as high if it was >0.1 g/L (Durando et al., 2013). Hemoglobin level was classified as low if it was <13.5 g/dL (men) and <11.5 g/dL (women), as normal if it was between 13.5-18.0 g/dL (men) and 11.5-16.0 g/dL (women), and as high if it was >18.0 g/dL (men) and >16.0 g/dL (women) (Thomas & Lumb, 2012). In this study, the electrolytes evaluated were sodium, potassium, and chloride because according to Siddiqui et al. (2012), the most common electrolyte disturbance in stroke patient were hyponatremia and hypokalemia followed by hypochloremia. Sodium concentration was classified as low (<136 mEq/L), normal (136-145 mEq/L), and high (>145 mEq/L). Potassium concentration was classified into low (<3.5 mEq/L), normal (3.5-5.1 mEq/L), and high (>5.1 mEq/L). Chloride concentration was classified into low (<98 mEq/L), normal (98-107 mEq/L), and high (>107 mEq/L) (Harwell, 2013).

Results and Discussion

From 30 acute stroke patient, 18 people (60.0%) suffered ischemic stroke and 12 people (40.0%) suffered hemorrhagic stroke (Table 1). In ischemic stroke, the proportion of women (66.7%) were higher than men (33.3%), while in acute hemorrhagic stroke, the proportion of men and women were similar. Average age of ischemic stroke patient was 58.7 ± 8.1 years old, while in hemorrhagic stroke patient was 55.3 ± 9.7 years old (Table 2). Hypertension was the most common risk factor in acute stroke patient (Table 3).

From all of acute stroke patients, 24 subjects (80.0%) experienced increase in CKMB concentration. There was no increase in Troponin T concentration in this study. Three male acute stroke patients (25%) experienced decrease in hemoglobin level while in female acute stroke patients, three subjects (16.7%) experienced decrease in hemoglobin level and 1 subject (5.6%) experienced increase in hemoglobin level. Eleven acute stroke patients (36.7%) experienced hyponatremia, 13 subjects (43.3%) experienced hypokalemia, and 2 subjects (6.7%) experienced hypochloremia.

Chi Square test did not show significant relationship between concentration of creatine kinase-myocardial band, troponin T, hemoglobin, and electrolyte and acute stroke ($p>0.05$) (Table 4).

Ischemic stroke was more common compared to hemorrhagic stroke (Siddiqui et al., 2012). Average age of ischemic stroke patients (69.8 ± 13.6 years old) was higher than that acute hemorrhagic stroke patients (68.8 ± 12.1 years old), however, there was no significant difference between them (p=0.14).

Twenty years mortality after acute ischemic stroke was correlated with increase in expected mortality between age of 18 to 50 years old (Panwar et al., 2016).

The prevalence of cerebral infarct and intracerebral hemorrhage were more common in men than in women. Meanwhile, the prevalence of subarachnoid hemorrhage was more common in women, although not statistically significant (Roy et al., 2014).

Women had lower prevalence of stroke compared to men. This might be caused by genetic factor and the positive effect of estrogen towards cerebral circulation. However, exception exist in age 35-44 years old and >85 years old. Gender influence was reversed in older age, where incidence of stroke was higher in women compared to men at age 85-94 years old (Appelros et al., 2009; Panwar et al., 2016).

In an experimental study using animals, gender difference influenced the prevalence of stroke. In young female animal, there was a smaller brain ischemic size compared to male animal. In second experiment, ovariectomy was found to increase the occurrence of medial

Table 1. Demographic Characteristic of Research Subjects

Characteristics	N(30)	%
Gender		
- Male	12	40.0
- Female	18	60.0
Age		
- 57 years old	20	66.7
- >57 years old	10	33.3
Ethnic group		
- Acehnese	2	6.7
- Batak	14	46.7
- Javanese	3	10.0
- Karo	4	13.3
- Malay	1	3.3
- Minangkabau	5	16.7
- Sundanese	1	3.3
Occupation		
- Housewives	12	40.0
- Farmer	2	6.70
- Civil servant	7	23.3
- Private employee	8	26.7
- Temporary worker	1	3.30
Educational background		
- Undergraduate degree	3	10.0
- Elementary school	4	13.3
- Junior high school	4	13.3
- Senior high school	19	63.4
Head CT Scan		
- Ischemic stroke	18	60.0
- Hemorrhagic stroke	12	40.0
Risk factors		
- Hypertension	27	90.0
- Diabetes Mellitus	12	40.0
- Smoking	3	10.0
- Dyslipidemia	7	23.3

Table 2. Comparison of Subject's Demographic Characteristics between Acute Ischemic Stroke and Acute Hemorrhagic Stroke

Characteristics	**Acute Ischemic Stroke (n=18)**	**Acute Hemorrhagic Stroke (n=12)**	***p***
Age, mean ± SD	58.7 ± 8.1	55.3 ± 9.7	0.305*
Gender, n (%)			
Male	6 (33.3)	6 (50.0%)	0.458**
Female	12 (66.7)	6 (50.0%)	
Ethnic Group, n (%)			
Sumatera	17 (94.5)	9 (75.0)	0.560***
Others	1 (5.5)	3 (25.0)	
Occupation, n (%)			
Civil servant	6 (33.3)	1 (8.3)	0.656***
Other	12 (66.7)	11 (91.7)	
Education			
≤ 9 years	4 (22.2)	4 (33.3)	0.431***
> 9 years	14 (77.8)	8 (66.7)	

*Independent T Test, ** Fisher's Exact Test, ***Mann Whitney Test

Table 3. Demographic Characteristics of Acute Ischemic Stroke and Acute Hemorrhagic Stroke Patients According to Stroke Risk Factors and Average Concentration of CKMB, Troponin T, Hemoglobin, and Electrolytes

Characteristics	Acute Ischemic Stroke (n=18)	Acute Hemorrhagic Stroke (n=12)	p
Risk Factors			
- Hypertension	15 (83.3)	12 (100.0)	0.255**
- Diabetes Mellitus	10 (55.6)	2 (16.7)	0.058**
- Smoking	1 (5.6)	2 (16.7)	0.548**
- Dyslipidemia	6 (33.3)	1 (8.3)	0.193**
CK-MB, average ± SD (U/L)	31.2 ± 9.2	34.9 ± 7.1	0.241*
Troponin T, average ± SD (g/L)	0.03 ± 0.007	0.03 ± 0.02	0.067***
Hemoglobin,			
- Men, average ± SD (g/dl)	13.6 ± 0.9	13.9 ± 1.8	0.676*
- Women, average ± SD(g/dl)	13.0 ± 1.8	12.46 ± 2.1	0.582*
Sodium, average ± SD (mEq/L)	135.7 ± 1.5	139.7 ± 4.8	0.317***
Potassium, average ± SD (mEq/L)	3.7 ± 0.5	3.6 ± 0.5	0.625*
Chloride, average ± SD (mEq/L)	101.4 ± 7.1	104.8 ± 3.4	0.359***

*Independent T Test, ** Fisher's Exact Test, ***Mann Whitney Test

Table 4. Correlation between Concentration of Creatine Kinase-Myocardial Band, Troponin T, Hemoglobin, and Electrolytes with Acute Stroke

		Acute Ischemic Stroke n (%)		Acute Hemorrhagic Stroke n (%)		p value
CK-MB	Normal	5	(27.8)	1	(8.3)	0.204*
	High	13	(72.2)	11	(91.7)	
	Total	18	(100.0)	12	(100)	
Troponin T	Normal	18	(60.0)	12	(40.0)	0.273**
	Total	18	(60.0)	12	(40.0)	
Hemoglobin	Low	1	(66.7)	2	(33.3)	0.500*
Men	Normal	5	(83.3)	4	(66.7)	
	Total	6	(100.0)	6	(100.0)	
Hemoglobin	Low	2	(16.7)	1	(16.7)	0.747***
Women	Normal	9	(75.0)	5	(83.3)	
	High	1	(8.3)	0	(0.0)	
	Total	12	(100.0)	6	(100.0)	
Sodium	Low	8	(44.8)	3	(25.0)	0.209***
	Normal	9	(50.0)	7	(58.3)	
	High	1	(5.6)	2	(16.7)	
	Total	18	(100.0)	12	(100.0)	
Potassium	Low	6	(33.3)	7	(58.3)	0.328**
	Normal	12	(66.7)	5	(41.7)	
	Total	18	(100.0)	12	(100.0)	
Chloride	Low	2	(11.1)	0	(0.0)	0.068***
	Normal	15	(83.3)	9	(75.0)	
	High	1	(5.6)	3	(25.0)	
	Total	18	(100.0)	12	(100.0)	

*Fisher's Exact Test, **Chi-Square Test, ***Mann Whitney Test.

cerebral artery occlusion. Low dose of estradiol was enough to provide dramatic protection to brain (Wilson, 2013).

Although the absolute risk of thrombosis stroke and myocardial infarct with utilization of hormonal contraceptive method were low, the risk were increased by 0.9 to 1.7 with administration of oral contraceptive method

such as ethinyl estradiol with dose of 20 μg and by 1.3 to 2.3 with administration of ethinyl estradiol with dose of 30-40 μg (Lidegaard et al., 2012).

Hypertenion was more common in acute hemorrhagic stroke (59.6%) than in acute ischemic stroke (53.9%), however, the difference was not statistically significant (Firoozabadi et al., 2013). Hypertension was the most common cause of stroke, particularly intracerebral hemorrhage (Lahano et al., 2014).

Atherosclerosis is the cause of half of mortality in western developed countries. This arterial disease which developed progressively is characterized by intimal thickening caused by fibrous deposition which narrow the vascular lumen and progressively become place of hemorrhage and thrombus formation (Durando et al., 2013)

There were significant correlation between obesity and stress with the prevalence of hypertension in women aged 40-55 years old. Obese person had more chance to acquire hypertension (OR = 4.2) compared to non-obese person. Stress at work tend to cause severe hypertension. The sources of stress at work are work load, unsatisfactory working facility, unclear working role, interpersonal problem, and family burden. Healthcare workers should provide routine and scheduled health promotion towards community, particularly regarding hypertension. Meanwhile, female workers (teacher) was advised to undertake lifestyle changes such as routine exercise and consumption of healthy food in order to prevent obesity. For people who were already obese, they were advised to reduce their bodyweight towards normal range. For people who experience stress, they were advised to manage their stress, avoid problems which could cause stress, and do not think about things which could cause stress by managing risk factors which were correlated with hypertension, hence hypertension could be prevented (Korneliani, 2013).

People with hypertension had 4.375 times more risk to experienced stroke compared to people which did not have hypertension. High lipid level could not yet be regarded as risk factor of stroke (OR 1.375, CI 0.453-4.170). Poor smoking habit also could not yet be regarded as risk factor of stroke (OR 2.333, CI 0.809–6.730). Therefore, risk factor of stroke such as hypertension need to be managed (Sarini, 2007).

Stroke patient with diabetes mellitus had significant higher incidence of mortality and recurrency in 3 to 6 months after stroke onset compare to stroke patients without diabetes mellitus. Diabetes mellitus is a risk factor of mortality in ischemic stroke patient in 6 months after stroke onset (Jia et al., 2011).

Increase in risk of subarachnoid hemorrhage was correlated with increase in risk of aneurism in smoker. Massive smoker (>20 cigarettes/day) had relative risk of aneurism bleeding of 7.3 (95% CI 3.8-14.3) compared to men who did not smoke (RR 2.1, 95% CI 1.2-3.6). Smoking could cause intracerebral hemorrhage through damage in small intraparenchymal blood vessel wall which could trigger rupture (Shah & Cole, 2010).

Stroke in smoker was correlated with morbidity. This is because the fact that cigarette smoke contains >4000 chemicals, including heavy metals and toxin which could produce free radicals, endothelial dysfunction and inflammation which in the end caused continuous atherosclerosis process. Smoking could influenced coagulation status. Smoking also could decrease cerebral blood flow and increased the risk of clot formation and risk of subsequent stroke through flow retardation or static flow phenomenon (Shah & Cole, 2010).

Higher concentration of nicotine which accumulated in the body of smoker caused addiction and pleasure feeling which stimulated desire to smoke more. Beside that, strong intention to stop smoke habit which was shown by intention to totally stop smoking would determine the success of smoking cessation effort. The effort to stop smoking would be useless if it was not founded by strong intention. Meanwhile, the intention to stop smoking was influenced by social support factor. If the social environment rejects and did not like the habit, the person would likely able to realize their desire to stop smoking. If the social environment was also smoker, the person must tell the social environment, especially close relative such as parents or friends, therefore

they could support and appreciate the effort. However, if the environment did not know about the effort, they would continue to smoke in front of the person. This would influence the smoker to continue to smoke and the intention to stop smoking would be postponed or ceased totally. Because of that, the best step for smoker who want to stop their habit is by acquiring intention to stop smoking totally. In that way, determination of smoke cessation intention could be used to predict chance of success of the effort (Riska et al., 2012).

In a study by Lahano et al. (2014), dyslipidemia was found to be more common in acute ischemic stroke (42 people/71.2%) compared to acute hemorrhagic stroke (22 people/68.9%) (p=0.818). Dyslipidemia is the main risk factor of coronary heart disease. However, its mechanism in causing ischemic stroke is still unclear. Oxidized cholesterol could start inflammation and plaque formation in blood vessel, therefore slowing the arterial blood flow (Xu et al., 2014).

We found that average concentration of CKMB was higher in acute hemorrhagic stroke compared to acute ischemic stroke. Average concentration of CKMB in hemorrhagic stroke patients with elevated troponin level (4.70 ± 4.08 ng/ml) was higher than patients with no increase in troponin level (2.98 ± 2.01 ng/ml) (Siddiqui, 2012).

The mechanism underlying increase in CKMB concentration was increase of intracranial pressure which stimulate cathecolamine secretion through activation of central autonomous system after ischemic stroke. This activation which stimulate tachycardia, coronary vasospasm, and coronary and peripheral vasoconstriction (Bhavani & Tagore, 2015).

Average concentration of troponin T in hemorrhagic stroke was higher than in ischemic stroke. According to Durando et al. (2013), normal serum troponin T was 0-0.1 μg/L. Meanwhile, according to Abdi et al. (2015), concentration of troponin T was considered abnormal if it was >24 ng/L. A concentration of 0.07-0.5 mg/mL was presumed to be consistent with probability of cardiac damage and probability of increase in clinical risk, while concentration >0.5 mg/mL was consistent with cardiac damage and increase in clinical risk (Singh et al., 2011; Abdi et al., 2015). Elevated troponin concentration was found in other clinical condition such as sepsis, hypovolemia, atrial fibrillation, congestive heart failure, myocarditis, and stroke (Alcalai et al., 2007; Wells et al., 2008; Al-otaiby et al., 2011). Mechanism of troponin T increase could be attributed to imbalance of autonomous nervous system which cause excessive sympathetic activity and increase in cathecolamine influence towards cardiac cell (Hasirci et al., 2013).

Hemoglobin had several functions, such as: (1) delivery of oxygen from lung to tissue, (2) transfer of carbon dioxide from tissue to lung, (3) hydrogen ion buffer in erythrocyte from conversion of carbon dioxide to bicarbonate, and (4) nitrite oxide metabolism (Storz, 2007; Thomas & Lumb, 2012).

Mechanism underlying of decreased hemoglobin level in acute stroke is yet to be understood. This might be due to decrease of blood viscosity which caused a reduction in cerebral circulation. Anemia could cause myocardial ischemia and left ventricular enlargement which could stimulate stroke (Panwar et al., 2016). Gender and type of stroke had significant influence towards concentration of blood cells, serum urea, creatinine, and electrolytes (Musa et al., 2015).

Chakraborty et al., (2013) found higher average sodium concentration in acute hemorrhagic stroke (146.0 ± 2.61mEq/L) compared to ischemic stroke (142.3 ± 3.01 mEq/L). We found significant difference in average sodium concentration in acute ischemic stroke compared to acute hemorrhagic stroke ($p<0.001$). Hasan et al., (2013) found electrolyte disturbance (chloride) in 10 acute stroke patients (14.28%), in which chloride disturbance was found to be more common in ischemic stroke (8.57%) compared to hemorrhagic stroke (4.28%). Six people (8.57%) had hypochloremia and 4 people (5.71%) had hyperchloremia. Meanwhile, average serum potassium concentration was higher in acute ischemic stroke (4.0 ± 0.2 mEq/L) compared to acute hemorrhagic stroke (3.6 ± 0.2 mEq/L). There was a significant difference of average serum potassium concentration between acute ischemic stroke and acute hemorrhagic stroke

(p<0.001) (Chakraborty et al., 2013).

Electrolyte disturbance is a mechanism that caused cellular damage during brain injury. Electrolyte disturbance such as hyponatremia or hypernatremia were caused by SIADH, increase in brain natriuretic peptide, imbalance in fluid intake and expenditure. The cause of electrolyte disturbance in cerebrovascular disease is SIADH (Siddiqui et al., 2012; Roy et al., 2014).

Reduction of cerebral blood flow could stimulate increase of cellular membrane permeability to sodium. Chloride would follow sodium to enter the cell through chloride channel (Kahle et al., 2009).

Concentration of CKMB increased significantly in 28% patients compared to control. Increase of CKMB in acute ischemic stroke was correlated with non-cardiac cause (Bhavani & Tagore, 2015). Stroke severity, not its location, was correlated with higher troponin concentration, although not particularly caused by heart and kidney compared to brain (Abdi et al., 2015).

Acute ischemic stroke patients with elevated troponin T concentration had poorer outcome compared to patients with normal troponin T concentration. Increase in troponin T was correlated with new onset atrial fibrillation (Cojocaru et al., 2014).

This study was in accordance with a study by Musa et al., (2015) which found no correlation between hemoglobin level and stroke. Low hemoglobin level was correlated with increase in mortality risk in stroke patients. This might be caused by decrease in blood oxygen delivery capacity, cerebrovascular autoregulation disturbance, and turbulence which was caused by decrease in blood flow. Anemia could cause worsening of clinical outcome which was correlated with inflammatory mediator, increase in nitric oxide synthase and CXC chemokine receptor 4 production (Musa et al., 2015; Barlas et al., 2016).

Low hemoglobin level was correlated with higher acute infarct and increase of the degree of infarct growth. Mechanism regarding the correlation between hemoglobin level and infarct severity was still unclear. We hypothesized that blood oxygen delivery capacity might influence the outcome of ischemic brain tissue. Low hemoglobin level at admission was correlated with mortality and increase of mortality risk in extreme hemoglobin level (Kimberly et al., 2011).

There was no significant difference between concentration of sodium, potassium, and chloride and acute stroke. Chakraborty et al., (2013) found that high sodium concentration might be correlated with increase in stroke prevalence (p<0.001). High sodium intake might increase mortality in stroke patient. Hyponatremia is more commonly found in hemorrhagic stroke (19%) compared to ischemic stroke (13%). Hypernatremia could also be found in ischemic stroke (3%) and hemorrhagic stroke (1%). There was no significant difference between sodium concentration and stroke (p>0.05) (Siddiqui et al., 2012).

Hypokalemia is an electrolyte disturbance which is more commonly found in hemorrhagic stroke (19%) than in ischemic stroke (11%). However, there was no significant correlation between potassium concentration and stroke (p>0.05) (Siddiqui et al., 2012). High potassium excretion was correlated with decrease in cerebrovascular disease risk and increase in potassium intake was inversely related with mortality in coronary artery disease (Chakraborty et al., 2013; Farahmand et al., 2013; Roy et al., 2014).

A study by Siddiqui et al., (2012) found no significant correlation between chloride concentration and stroke (p>0.05). All patients with hyperchloremia also had hypernatremia which might point to the same etiology for both disturbance (Hasan et al, 2013).

This study had several limitation. First of all, things which might influence concentration of CKMB, troponin T, hemoglobin, and electrolyte were not controlled. Secondly, this study only took blood samples for CKMB, troponin T, hemoglobin, and electrolyte concentration examination during the occurrence of disease and the concentration before the disease were not examined, therefore there could be biases happened. Finally, the number of samples was relatively small to provide representative result.

Conclusion

There were no difference in average

and no significant correlation between concentration of creatine kinase myocardial band, troponin T, hemoglobin, and electrolytes with acute stroke.

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