The 5th WFNS Spine Committee Biennial Conference in conjunction with
The 22nd Annual Scientific Meeting of Indonesian Neurosurgical Society (INS)
The 12th Asian Epilepsy Surgery Congress (AESC) and
The 2nd International Fujita Bantane Interim Meeting of Neurosurgery

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Bali 2018

October 25th - 27th, 2018
Bali Nusa Dua Convention Center (BNDCC)
Bali, Indonesia

Abtract Book of The 5th WFNS Spine Committee Biennial Conference in conjunction with
The 22nd Annual Scientific Meeting of Indonesian Neurosurgical Society (INS),
The 12th Asian Epilepsy Surgery Congress (AESC) and
The 2nd Interantional Fujita Bantane Interim Meeting of Neurosurgery

"Meeting the Challenges, Facing the Future"

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WELCOME MESSAGE

Dear Friends,

It is our great pleasure to invite you to The 5th WFNS Spine Committee Biennial Conference of WFNS which will be held at Bali, Indonesia between October 25th - 27th, 2018.

WFNS scientific committees try to contribute to the education and progress of sub disciplines of neurosurgery. Spine surgery is getting a high interest and Spine Committee Symposia every two years are the largest activity of the committee. I am happy to invite you to Bali, Indonesia to endorse activities in this part of the world. This meeting will be in conjunction with the Annual Meeting of Indonesian Neurological Society, Asian Epilepsy Surgery Congress. On October 25, a one-day cadaver dissection course will be held in Surabaya.

The meeting aims to reach a large number of audience, thus contribute to the spine education in this area more effectively. There will be “intense”, and full of excellent lectures from prominent experts, results of implementation of new procedures, case discussions, debate sessions, video demonstrations, and workshops from industry.

The location of our congress is Bali island, one of the most beautiful and exotic place of the world. We really hope that it will endow us with many precious and long-lasting memories to cherish.

We look forward to seeing you in Bali in October 2018.

Co-chairman of the WFNS Spine Committee.

Mehmet Zileli

Michael G.Fehlings

Daniel J.Hoh

OP 072

THE EFFECT OF CURCUMIN EXTRACT TOWARD MATURE BRAIN DERIVED NEUROTRPHIC FACTOR (M-BDNF) EXPRESSION AFTER TRAUMATIC BRAIN INJURY

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Background: Traumatic Brain Injury (TBI) causes disability, death and huge economic losses in various countries of the world. TBI incident varies between 67 –317 per 100.000 population, with 4-7% mortality rate in moderate brain injury, and 50% in severe brain injury. The mature BDNF (m-BDNF) pathway system is a potential therapeutic target for neurological disorders in traumatic brain injury. Curcumin extract has a neuromodulatory effect which has a modulation effect on the expression and activation of the m-BDNF system in the hippocampus area.

Methods: Laboratory experimental study in Faculty of medicine University of Brawijaya which used thirty male Sprague-Dawley rats. Rats were divided into three treatment groups, group A (negative control), group B given traumatic brain injury, group C given traumatic brain injury and Curcumin administration. Rats's brain tissue was immunohistochemically processed, to observe the number of cells expressing m-BDNF in the subgranular zone (SGZ) of the hippocampus dentate gyrus (DG). Data were analyzed with SPSS and ANOVA analysis.

Result: In ANOVA analysis, mean expression of m-BDNF group C compared to group A and group B were increased significantly (p=0.0001). Curcumin Through Induction of m-BDNF and activation of its intracellular receptor TrkB can produce neural regeneration, reconnection, and dendritic sprouting, and can enhance synaptic efficacy.

Conclusion: Curcumin can increase the expression of m-BDNF in the subgranular zone of the hippocampus dentate gyrus.

Keywords: Curcumin, TBI, m-BDNF, Neuroplasticity

OP 073

CORRELATION BETWEEN HUMAN EPITHELIAL GROWTH FACTOR 2 (HER 2) EXPRESSION WITH HISTOPATHOLOGICAL LEVEL ON INTRACRANIAL MENINGIOMA PATIENTS AT HAJI ADAM MALIK HOSPITAL MEDAN

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Background: Meningiomas are the most common intracranial primary neoplasm in adults. Meningiomas arise from the arachnoidal cells surrounding the brain and are one of the most common tumors of the central nervous system. Meningiomas account for about 15-30% of all primary intracranial tumors. According to the 2007 WHO classification, meningiomas are divided into three grades (I, II and III). Several studies have reported higher recurrence rates for males than for females. However, other studies have found no significant difference based on gender and no association between tumor development in young patients < 40 yrs) and a high likelihood of recurrence. However, some study found a significant difference between age and recurrence. The aggressiveness of meningiomas is unpredictable. HER2 represents a well-known prognostic factor in various tumors such as breast carcinomas. There are only a few studies on the relationship between meningioma and HER2 expression, and the results are different as well. The aim of this study was to determine this relationship.

Methods: this study was a crosssectional analytic study of 32 parafin block meningioma after staining with HER 2.

Result: The result from this study has significant result from expression HER 2 in intracranial meningiomas.

Conclusion: HER 2 expression plays a role in the development of more-aggressive meningiomas or not is a question that needs to be clarified in further studies. The results of our case report did not advocate this role for HER2.

THE EFFECT OF CURCUMIN EXTRACT TOWARD MATURE BRAIN DERIVED NEUROTRPHIC FACTOR (mBDNF) EXPRESSION AFTER TRAUMATIC BRAIN INJURY

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ABSTRACT

Background: Traumatic Brain Injury (TBI) causes disability, death and huge economic losses in various countries of the world. TBI incident varies between 67 –317 per 100.000 population, with 4-7% mortality rate in moderate brain injury, and 50% in severe brain injury. The mature BDNF (mBDNF) pathway system is a potential therapeutic target for neurological disorders in traumatic brain injury. Curcumin extract has a neuromodulatory effect which has a modulation effect on the expression and activation of the mBDNF system in the hippocampus area.

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Result: In ANOVA analysis, mean expression of mBDNF group C compared to group A and group B were increased significantly (p=0.0001). Curcumin through induction of mBDNF and activation of its intracellular receptor TrkB can produce neural regeneration, reconnection, and dendritic sprouting, and can enhance synaptic efficacy.

Conclusion : Curcumin can increase the expression of mBDNF in the subgranular zone of the hippocampus dentate gyrus.

Keywords: Curcumin, TBI, mBDNF, Neuroplasticity

INTRODUCTION

Head injuries are critical public and socio-economic health problems throughout the world. Head injuries are a major cause of death, especially in young adults, and generally cause lifelong disability to those who survive. Head injuries generally cause neurocognitive and psychological disorders such as attention disorders, inability to form visuospatial associations, poor executive functions and depression[1]

The incidence of head injuries shows a difference between countries. Data from the Centers for Disease Control and Prevention (CDC) shows that every year there are 1.7 million people with head injuries. 1.4 million sufferers are treated at the emergency department, around 275 thousand sufferers must be treated and 52,000 experience fatal events. A meta-analysis of 23 European countries shows the incidence of hospital care due to head injuries by 235 per 100 thousand people and around 20 per 100 thousand people which is a neurosurgical case[2].

Head injuries can be classified into primary and secondary brain injuries. Primary brain injury occurs immediately after trauma which causes tissue mechanical damage. Secondary brain injury is an advanced process that lasts for several hours until the day after the impact. Secondary injuries include complex cascade activation, excitotoxicity, and metabolic failure resulting in cell dysfunction and death. Current therapy is focused on reducing the expansion of secondary injuries compared to repairing damage from primary injuries[3]

Brain derived neurotrophic factor (BDNF), is a neurotropin that has a neuroprotective effect on brain ischemia injury. Increased BDNF in the area around the lesion is closely related to the progress of recovery of nerve function. However, after brain ischemia injury decreases the level of BDNF which results in changes in the ability of neuroplasticity or

recovery of nerve cell function, either spontaneously or with induction of rehabilitation. Brain derived neurotrophic factor (BDNF) is an important molecule in brain plasticity[4].

Brain derived neurotrophic factor (BDNF) plays a role in cell proliferation, differentiation, adhesion and maturation, through specific receptor protein tyrosine kinase (PTK), which induces growth, resistance and regeneration of central nerve cells after injury. Research reports that after in vitro induction of BDNF, differentiation of Neural stem cells (NSCs) has doubled. BDNF has strong activity in proliferation, differentiation and activation of endogenous and exogenous NSC*s*[4].

Curcumin which has a chemical structure [1, 6-heptadiene, 3-5-dione, 1, 7-bis (4-hydroxy-3-methoxyphenyl) (C21H20O6)] is a hydrophobic material of polyphenols derived from turmeric roots (Curcuma longa) namely rhizoma family herbs from ginger. Curcumin has been consumed by humans for centuries as a curry ingredient. Curcumin has been used extensively in Ayurvedic medicine in India as an herbal plant to treat eye infections, mosquito bites, burns, fever and other skin diseases. Various studies have shown that curcumin has an effect as an antioxidant, anti-inflammatory, anti-microbial, and anti-carcinogenic. In in vivo studies, curcumin has been proven to be able to penetrate the blood brain barrier and be able to maintain high biological activity in the brain. Curcumin is a strong pleiotropic molecule and can interact with a number of molecular targets[5,6].

MATERIAL AND METHOD

Sample and population

This study used 33 mice that met the predetermined criteria. Experimental rats were divided into 3 groups. Each group consisted of 11 rats. The first group is the group that is not given any treatment (Group A). The second group was a group of rats treated with traumatic brain injury (Group B). The third group was a group of rats treated with traumatic brain

injury and turmeric extract/curcumin (Group C). All rats were observed after 24 hours after the treatment of traumatic brain injury.

The experimental animals used in this study were Sprague-Dawley male rats, 2.5-3 months old, weight 280-320 grams, healthy and obtained from the Faculty of Veterinary Medicine, Bogor Agricultural University. The choice of mice as experimental animals is based on the consideration that Sprague-Dawley mice are genetically similar to humans and have adaptability to the laboratory environment[7]

Preparatiom

Maintenance of experimental animals during the research was conducted in experimental animal cages at the Pharmacology Laboratory, Faculty of Medicine, Universitas Brawijaya (FK UB). To examine brain tissue preparations carried out in the Department of Anatomical Pathology (FK UB).

The study was conducted for 2 (two) months, including the stages of preparation of materials and tools, treatment, examination, and preparation of reports. Treatment for experimental animals was carried out for 1 day, then brain tissue preparations were carried out on the 7th day in the form of cells expressing mBDNF in the cerebral cortex that experienced contusion from Sprague-Dawley male mice, with a 1000x microscope, positive cells were counted in 20 fields view of each sample. Observation of the expression of mBDNF reached its peak after 24 hours in the cerebral cortex which experienced contusion, decreasing to the point of balance after the first 24 hours in the area of the injury penumbra.

The turmeric extract used is processed at UPT Materia Medica Batu. Turmeric rhizomes are collected from the Malang area and then cleaned from dirt and then dried. A total of 400 gr was processed by maceration method, 70% ethanol solvent as much as 2 L and evaporated for 1 hour 30 minutes. Produced 115 ml of turmeric extract with 80 gr levels.

Procedures

Focal brain injury was induced on the right frontal area using rat model of closed head injury (CHI), performed with the modified Feeney's weight-drop protocol[7]. This study was approved by local ethical committe. Thirty-three Sprague dawley rats weighing 280-320 gr were randomized into three treatments group, i.e sham-operated controls (Group A), CHI only (group B), and CHI with curcumin extract (group C). All animals were given ketamine HCl (Intramuscular dosage 100 mg/kg) and xylazine base' concentration 20mg/ml (Intramuscular dosage 0.15 ml/kg).

The scalp was cleaned with povidone iodine and aseptic techniques were used throughout surgery. The scalp was opened on the right frontal. Then, the rats were placed securely in stereotactic apparatus. We gave 40 mg metal mass from 1.5 m height. Tumeric extract/ curcumin (200mg/Kg per oral) was given once daily until seventh day via nasogastric tube. Afterward, animals were sacrificed through cervical dislocation after giving ketamine HCl (100 mg/kg, intramuscular). The brains were removed and fixed in 10% buffered formalin. The specimens were then processed for paraffin-embedded for immunohistochemistry staining preparation. Sham operated rat underwent anesthesia and surgery, without trauma and treatment.

Immunohistochemistry staining

The slides were washed with PBS pH 7.4 one time for 5 minutes. Endogenous peroxide bloking uses 3% H2O2 for 15 minutes. Wash using PBS pH 7.4 three times, each for 3 minutes. Specific protein blocking uses 5% FBS containing 0.25% Triton X-100. Wash using

PBS pH 7.4 three times, for 3 minutes each. Incubation using mBDNF antibodies for 60 minutes. Wash using PBS pH 7.4 three times, each for 3 minutes.

Incubation using anti mouse biotin conjugated for one hour at room temperature. Wash using PBS pH 7.4 three times, each for 3 minutes. Incubation using SA-HRP (Strep-Avidin Horse Radis Peroxidase) for 10 minutes. Wash using PBS pH 7.4 three times, each for 3 minutes. Tetesi with DAB (Diamino Benzidine) and incubation for 5-15 minutes. Wash using PBS pH 7.4 three times, each for 5 minutes. Counterstaining using Mayer Hematoxilen which was incubated for 1 minute and washed using tap water. Rinse using dH2O and dry air. Mounting uses an entanglement and covers with a glass cover. Observe on a light microscope

Positive molecular expression with primary antibodies will look brown with a 1000X light microscope. The calculated cerebral cortical contour cells were located in the cerebral cortex, positive cells were counted in 20 fields of view (HPF) in each sample.

Statistical Analysis

The results of the research obtained were analyzed using the Shapiro Wills normality test. If the data is normally distributed, then statistical analysis is done by ANOVA hypothesis test (analysis of variants). However, if ata is not normally distributed, then Kruskal Wallis hypothesis testing is carried out. If there are differences, then the analysis is continued with the Tukey Post Hoc test to find out the different pairs of data (to see differences from each group). This study was significant if the value of $p < 0.05$.

RESULT

Weights

Based on the results of the study, giving trauma to the study sample significantly reduced weight, from an average of 288.89 ± 28.59 grams before treatment to 275.30 ± 34.96 grams after treatment of head trauma (p = 0.0001, with t test in pairs, table 1.)

The homogeneity test (table 2) carried out using the one-way ANOVA test showed that there was no significant difference between body weight before treatment and body weight after treatment (sig> 0.05). This shows that experimental animal body weight data has homogeneous variations. Thus weight is not a confounding variable that can affect the dependent variable, in this study, namely mBDNF.

Expression of mBNDF in the Brain Tissue of the Treatment Group

The results of immunohistochemical examination of brain tissue showed that the brain tissue of the control group showed an average number of mBDNF expressions of 5.82 ± 1.32 (table 3). Treatment of traumatic and curcumin head injuries (group C) in experimental animals significantly increased the mean expression of mBDNF to group A (p = 0.0001) and group B (p = 0.0001) to 14.82 ± 2,828 seen in table 4.

It can be concluded that in the treatment of head injury and curcumin (Group C), the expression of mBDNF increased when compared to the negative control group (Group A) and the group given head injury only (Group B).

Histology of BDNF expression with immunohistochemical staining, using Olympus BX 50 microscope 1000x magnification can be seen in Figure 1.

DISCUSSION

In this study investigated the effects of oral administration of curcumin-containing turmeric extract on mBDNF expression after traumatic brain injury. This research is an experimental study of experimental laboratory.

The increase in mBDNF in the area around the lesion is closely related to the progress of recovery of nerve function. However, after brain ischemia injury there is a decrease in the level of mBDNF which results in changes in the ability of neuroplasticity or recovery of nerve cell function, either spontaneously or with induction of rehabilitation. mBDNF is an important molecule in brain plasticity[4].

The Mature Brain Derived Neurothrophic Factor (mBDNF) plays a role in cell proliferation, differentiation, adhesion and maturation, through specific receptor protein tyrosine kinase (PTK), which induces growth, resistance and regeneration of central nerve cells after injury. Research reports that after induction of mBDNF in vitro, differentiation of NSCs has doubled. mBDNF has strong activity in proliferation, differentiation and activation of endogenous and exogenous NSCs [4,9].

Curcumin has the potential to increase mBDNF, inhibit lipid peroxidation, and neutralize oxygen reactivation and free radicals derived from nitric oxide. This curcumin characteristic is supported by the ability to cross the blood brain barrier so that it can provide protection against neurons directly. From the results of the study, the administration of intranasal curcumin increased the mBDNF expression significantly ($p = 0.0001$) compared to the negative control group and the head injury treatment group. Significantly decreased mBDNF expression ($p = 0.0001$) in the head injury treatment group compared to negative controls.

Activation of BDNF (ligand) from tyrosine residues results in activation of different intracellular pathways, which leads to nerve plasticity, neurogenesis, stress resistance and cell survival. This shows the comparative flexibility of the Trk receptor in terms of pro-survival functions. Thus, the mBDNF signaling pathway activates one or both transcription factors CREB and CREB-binding protein (CBP) which regulate the expression of genes encoding proteins involved in nerve plasticity, stress resistance and cell survival[10].

The role of curcumin in increasing BDNF expression is related to the transcription of the BDNF coding gene. Curcumin has been shown to increase the levels of cAMP and ERK (extracellular signal regulated kinases) and p38 kinase. The cAMP compound activates Protein Kinase A (PKA), an enzyme needed to increase the activity of cAMP-response element binding (CREB) protein (CREB). This activated CREB will then occupy the promoter on the gene that encodes BDNF and then initiate the process of transcription of the gene which causes more quantities of BDNF protein to be produced[11]

New findings from this study are that administration of curcumin intraoral increases the expression of mBDNF in brain cells that have traumatic head injury, presumably by modulating the TrkB system, which increases the expression of PI3K and ERK. PI3K plays a role in the anti-apoptotic pathway, while ERK plays a role in neuroplasticity.

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TABLE

Table 1. Changes in Body Weight after the Trauma Treatment

	x±SD (gram)	**p**
Sebelum perlakuan	288,89 ± 28,59	0,0001*
Setelah perlakuan	275,30 ± 34,96	

Table 2. Weight of Experimental Animals based on the Treatment Group

	Sebelum perlakuan (gr)	**P**	**Setelah perlakuan (gr)**	**p**
Kelompok A	284,89 ± 38,17	0,932	276,44 ± 34,03	0,492
Kelompok B	289,78 ± 20,92		283,11 ± 20,86	

Kelompok C	276,44 ± 34,03	263,33 ± 46,39

Table 3. mBDNF Expressions Based on Treatment Groups

	x±SD	**p**
Kelompok A	5.82±1,32	
Kelompok B	10.64±1,69	0,0001*
Kelompok C	14.82±2,83	

Table 4. Analysis of mBDNF expressions based on the treatment group

		Mean Difference	Std. Error	Sig.
Kelompok A	Kelompok B	-4.818*	1.038	.000
	Kelompok C	-9.000*	1.038	.000
Kelompok B	Kelompok A	4.818*	1.038	.000
	Kelompok C	-4.182*	1.038	.001
Kelompok C	Kelompok A	9.000*	1.038	.000
	Kelompok B	4.182*	1.038	.001

FIGURE

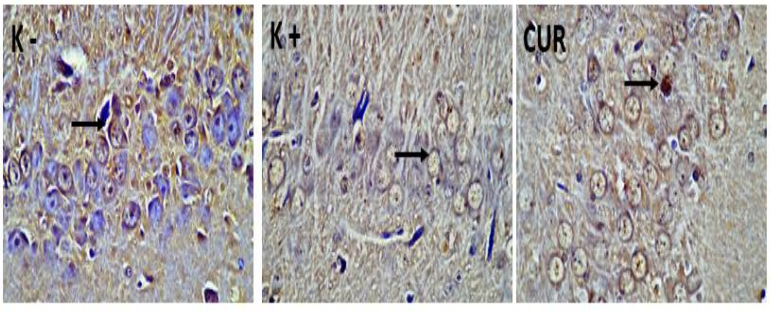

Figure 1. Comparison of mBDNF expressions