



Blood Peroxydase and Catalase Enzyme Activity in Down Syndrome in South Kalimantan

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Down syndrome is a genetic abnormality caused by abnormal autosomal chromosome 21. Oxidative stress is increased in Down syndrome patients. The stress can be measured through peroxydase and catalase activity. Peroxydase and catalase was the antioxidant enzymes that catalyzed hydrogen peroxide to water and oxygen.

This research was analytical observational research with cross sectional approach to measure blood peroxydase and catalase activities in 30 Down syndrome patients and 30 non Down syndrome patients. Samples were obtained by purposive sampling. Peroxydase activities were measured by Kanehira and Shibata method. Catalase activities were measured by the first order of reaction velocity constant parameter.

Result showed that mean peroxidase activity in Down syndrome patients was 0,335/minute and peroxidase activity in non Down syndrome patients was 0,162/minute. The mean catalase activity in Down syndrome patients was 47,478/minute and catalase activity in non Down syndrome patients was 29,480/minute. Data was analyzed by unpaired t test with $\alpha=0,05$; showed that peroxydase and catalase activities were significantly different between Down syndrome patients group and non Down syndrome patients group.

Conclusion: peroxydase and catalase activities in Down syndrome was

higher than in non Down syndrome patients.

Keywords: Down syndrome, peroxydase, catalase, oxidative stress
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Central Pontine Myelinolysis : Diagnosis and Management

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Central pontine myelinolysis (CPM) was described by Adams and colleagues in 1959 as a disease affecting alcoholics and the malnourished. The concept was extended in 1962 with the recognition that lesions can occur outside the pons, so-called extrapontine myelinolysis (EPM). When the pathologic process involves pontine and extrapontine sites, the term osmotic demyelination syndrome (ODS) is used. In 1982 a link between these disorders and the rapid correction of sodium in hyponatraemic patients was substantially established. The clinical presentation of CPM is highly variable, usually depressed level of consciousness, quadriparesis and pseudobulbar symptoms such as dysarthria and dysphagia. Less often, CPM manifests with ataxia, movement disorders, or behavioral symptoms. Diagnosis of CPM is based on clinical suspicion and confirmed by magnetic resonance imaging (MRI). Recommended treatment is supportive only. Reports of treat-

ments including Thyrotropin-releasing hormone (TRH), methylprednisolone, intravenous immunoglobulins (IVIg), and plasmapheresis. Recognizing patient at risk, preventing rapid correction of hyponatremia, prompt diagnosis, and managing associated complications will decrease morbidity and mortality.

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Muscular Dystrophy with Cardiomyopathy – case report

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Introduction: Duchenne Muscular Dystrophy is the most common type of muscular dystrophy. The incidence is 30 per 100.000 newborn male. CK level increased in 50 - 75 % cases and is very useful for disease evaluation ; CKMB level increased in 10 % cases, LDH level increased in 10 % cases, and transaminase level increased in 15 % cases.

Case: A 22 year-old male with Duchenne-type muscular dystrophy complicated by cardiomyopathy. CPK level was 2508 U/L, CKMB level was 50 U/L, LDH level was 568 U/L, SGOT level was 109 mU/L, dan SGPT level was 72 mU/L. Muscular biopsy revealed progressive muscular dystrophy pattern. Thorax photo revealed cardiomegaly, EKG pattern revealed sinus tachycardia with left and right ventricle hypertrophy.

Conclusion: A case of chronic progressive muscular dystrophy with cardiomyopathy.

Suggestion: Genetic examination with Western Blot muscle biopsy, muscular immunocytochemistry stained with dystrophin antibody, and DNA mutation peripheral blood leucocyte analysis.

Keywords : Muscular dystrophy, CK, cardiomyopathy

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