



Effects of Combined Inhalation of Coal Dust and Cigarette Smoking on Haematobiochemical Parameters

Kömür Tozu Solunması ve Sigaranın Hematobiokimyasal Parametreler Üzerine Etkisi

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ABSTRACT

Purpose: The aim of this study is to determine the effect of inhaled particulate matter coal dust and smoking on the haematobiochemical profile of worker in coal analysis laboratory.

Material and Methods: Twenty nine workers, were randomly divided into three groups, one control group (n=5), one group who exposed to coal dust but non smoking (CDNS) (n=15), and one group exposed to coal dust and smoking (CDS) (n=9). The control group included subjects working in administrative division which none have been exposed to coal dust. Exposed group working on coal dust material analysis were divided as smokers and non smokers. The duration of their work period were less than 10 years.

Results: Mean of eosinophil and monocytes counts were significantly lower in both of coal dust exposure compared to control group (p<0.05). Segmental neutrophil counts were significant higher in coal dust exposure group with or without smoking compared to control group (p<0.05). The level of erythrocyte sedimentation rate (ESR) was significantly lower in CDNS group compared to control group (p<0.05).

Conclusion: Combined inhalation of coal dust and cigarette smoking changes cellular immune system such as decreasing of eosinophil and monocytes counts also increasing of segmental neutrophil. Beside that, this exposure also induces an acute phase process of inflammation..

Key Words: Worker; coal dust analysis laboratory; hematological; biochemical.

ÖZET

Amaç: Bu çalışmanın amacı, solunan kömür tozu partiküllerinin ve sigaranın, kömür analiz laboratuvarlarında çalışan işçilerin hematobiokimyasal profilleri üzerindeki etkisini saptamaktır.

Materyal ve Metod: 29 işçi rastgele biçimde; kontrol (n=5), kömür tozuna maruz kalmış ancak sigara içmeyen (CDNS) (n=15) ve kömür tozuna maruz kalmış ve sigara içen (CDS) (n=9) olmak üzere üç gruba ayrılmıştır. Kontrol grubundaki bireyler idare ile ilgili birimde çalışan ve kömür tozuna maruz kalmayan bireylerden oluşmaktadır. Maruziyet grubundaki bireyler kömür tozu materyali ile çalışan bireyler olup sigara içen ve içmeyen olarak 2 gruba ayrılmıştır. Bireylerin kurumdaki çalışma süresi 10 yıldan azdır.

Bulgular: Eozinofil ve monosit sayıları kontrol grupla karşılaştırıldığında kömür tozuna maruz kalan her 2 grupta da anlamlı şekilde azalmıştır ($p<0.05$). Segmental nötrofil sayıları kontrol grupla karşılaştırıldığında kömür tozuna maruz kalan her 2 grupta da anlamlı olarak artmıştır ($p<0.05$). Eritrosit sedimentasyon oranı (ESR), kömür tozuna maruz kalmış ancak sigara içmeyen (CDNS) grupta kontrol grubuna göre anlamlı derecede azalmıştır ($p<0.05$).

Sonuç: Kömür Tozu solunması ile birlikte sigara içilmesi, eozinofil ve monosit sayısını azaltırken, segmental nötrofillerin sayısının artması gibi hücrel immün sistem mekanizmalarını değiştirmektedir. Bunun yanında bu maruziyet inflamasyonun akut fazında indüklemektedir.

Anahtar Kelimeler: İşçi, kömür tozu analiz laboratuvarı, hematolojik, biyokimyasal.

INTRODUCTION

Unhealthy lifestyle and occupational environment were risk factors for increasing incidence of disease. Inhalation of occupational and atmospheric coal dust has not only contributed significantly to the development of several respiratory disorders, also cardiovascular disease¹. Epidemiologic study demonstrated that the population residing near coal mine facilities suffer from a higher rate of cardiovascular disease². In addition, several previous studies showed that coal dust produces proinflammatory cytokines and free radicals in vitro^{3,4} in rats^{1,5} and humans⁶⁻⁸. Smoking has been established as risk factor for cardiovascular and pulmonary disease, and also bone disease, such as osteoporosis, disc disease, delayed fracture healing, and non union⁹. There is little study have conducted to explore an effect of combined inhalation coal dust and cigarette smoke. Previous study showed that inhaled combination of coal dust and cigarette smoke decreases iron level in bone rats¹⁰.

Studies on the haematobiochemical effects will therefore serve as a useful general indicator of the potential of combined inhalation coal dust and cigarette smoke to cause adverse effects on exposed organisms. This is because the blood is a pathophysiological reflector of the whole body and, therefore, blood parameters are important in diagnosing the structural and functional status of organisms exposed to toxicants¹¹. On the basis of the above, the objectives of this study are to determine the haematobiochemical effects of combined inhalation of coal dust and cigarette

smoking on people who working in coal mine laboratory.

MATERIAL AND METHODS

Subject

The study was mainly conducted on Carsurin Coal Laboratories, Banjarmasin, South Kalimantan, Indonesia. Two groups consisted control group and exposed group. Control group are subject who working in administrative division which none exposed to coal dust (control/C). Exposed group are subject who working in coal dust material analysis (coal dust non smoker/CDNS and coal dust smoker/CDS). All subject were divided into smoker and non smoker. The duration of working are less than 10 years. This research has been approved by research ethics committee Faculty of Medicine University of Lambung Mangkurat, Banjarmasin, Indonesia.

Procedures

Five milliliters of blood was obtained from mediana cubiti vein mice using heparinized hematocrite bottle and stored in an EDTA anticoagulant bottle. A complete blood profile, including WBC count, was measured using an automated cell counter (GEN-S; Beckman Coulter, Miami, FL). Plasma glucose was measured by a hexokinase method (Hitachi 911 automated analyzer; Boehringer Mannheim, Mannheim, Germany). Total cholesterol was measured by enzymatic methods on the Hitachi 911 automated analyzer using reagent kits supplied by the manufacturer. Hematobiological analysis was done

at Clinical Pathology Laboratory, Ulin General Hospital of Banjarmasin.

Statistical analysis

Data are presented as mean \pm SD and differences between groups were analyzed using

ANOVA test with SPSS 15.0 statistical package. P value < 0.05 was considered statistically significant.

RESULTS

A total of 29 workers, were randomly divided into three groups, one control group (n=5), one group who exposed to coal dust but non smoking (CDNS) (n=15), and one group exposed to coal dust and smoking (CDS) (n=9). The mean of eosinophil counts in blood in three groups were 2.80, 1.73, and 1.33 respectively; in comparison with the control group, both of coal dust exposure shows significant decreases ($p < 0.05$). The mean monocyte counts in both of coal dust exposure, also shows significant decreases compared to control group ($p < 0.05$). The mean of erythrocyte sedimentation rate in three groups were 15.20, 8.26, and 13.88 respectively and the erythrocyte sedimentation rate in CDNS group was lower significantly compared to control group ($p < 0.05$). In addition, the counts of segmental neutrophil was increase significantly in coal dust exposure group with or without smoking compared to control group ($p < 0.05$).

DISCUSSION

Inflammation is a pathophysiological processes mediated by various signaling molecules produced mainly by circulated cells, mainly leukocytes, macrophages, and plasma cells^{12,13}. Elevated white blood cell count (WBC) that is well within the normal range was associated with an increased risk for developing cardiovascular disease^{14,15}, or for the re-occurrence of myocardial ischemia^{16,17}.

Monocytes are derived from myelomonocytic stem cells in the bone marrow where they mature to monocytes. Once in the blood, they develop further and migrate in response to chemokines to the tissue where they differentiate into functionally and phenotypically distinct macrophage types¹⁸. Monocytes were also sources of growth factors¹⁹. Their phenotype and function are known to be altered in response to multiple factors, including bacterial endotoxins, tissue injury and inflammatory signals. In this study we found significant decrease of monocytes counts in coal dust exposure with or without smoking groups. This finding resulted from balance between stem cells population and differentiation process become macrophages. Our previous study showed that coal dust exposure does not influences hematopoietic stem cells in rats²⁰. In addition, we proof that coal dust exposure increase macrophage numbers in lung rats²¹. We suggested that the reason of decreasing monocyte in this study is elevated signal to monocytes differentiation become macrophage for phagocytosis of coal dust particle and cigarette smoke compound.

We also confirmed the previous study of a substantial increase of neutrophil in smoker²². Combined inhaled coal dust particulate matter and cigarette smoking also increase neutrophil significantly. The responsible mechanisms might be influence of smoking and coal dust on neutrophil supportive phagocytosis capability to monocytes or macrophages. Beside that we also found that decreased of eosinophil count in coal dust exposures with or without smoking. Previous study showed decreased^{23,24} or not influence of smoking on eosinophil counts²⁵. This finding indicate that smoking alone or combination with coal dust exposure affect cellular immune system. The responsible mechanisms might be depressive influence of smoking and coal dust on stimulative factor for eosinophil.

The ESR is determined by the balance between pro-sedimentation factors, mainly fibrinogen and those anti-sedimentation factors.

The erythrocytes normally have net negative charges call as zeta potential and, therefore, repel each other. High molecular weight proteins, especially when positively charged, increase viscosity and favor rouleaux formation and thus would raise the ESR. When an inflammatory process is present in the blood, the high proportion of fibrinogen as acute phase reactant, causes red blood cells to stick to each other. Fibrinogen has the greatest effect on the elevation of ESR when compared with other acute phase proteins. In addition, paraproteins are positively charged molecules and will increase the ESR levels²⁶. The level of ESR is lower significantly in CDNS group compared to control group ($p < 0.05$). Smoking activity in coal dust exposure worker increase ESR level to reach similar level in control group. This

finding indicate that component of coal dust has properties as anti-sedimentation factors or increase net negative charges in blood totally. Addition of smoke component in blood people who exposed to coal dust increases ESR level but still in normal value.

In summary, combined inhalation of coal dust and cigarette smoking changes cellular immune system such as decreasing of eosinophil and monocytes counts also increasing of segmental neutrophil. Beside that, this combined exposure also induces acute phase process of inflammation.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

Table 1. Hematobiochemical profile of worker exposed to coal dust and cigarette smoking

	Control (n=5)	Coal dust exposure	
		Non smoker (n=15)	Smoker (n=9)
Hemoglobin	14.30 ± 1.67	14.54 ± 1.28	14.71 ± 1.23
RBC	5.58 ± 0.48	5.68 ± 0.43	5.92 ± 0.61
WBC	8.54 ± 1.85	8.76 ± 3.34	9.96 ± 1.70
Hematocrit	48.80 ± 4.42	49.73 ± 4.65	50.31 ± 3.51
Platelet	342.00 ± 66.50	292.200 ± 42.33	316.88 ± 64.17
MCV	87.60 ± 2.80	87.66 ± 5.16	85.77 ± 8.56
MCH	25.54 ± 1.03	25.52 ± 1.03	24.98 ± 2.75
MCHC	29.18 ± 0.92	29.20 ± 1.03	29.20 ± 1.33
Basofil	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Eosinophil	2.80 ± 1.16	1.73 ± 1.10 ^a	1.33 ± 0.55 ^a
Neutrofil-seg	45.00 ± 3.16	50.80 ± 5.25 ^a	56.11 ± 8.85 ^a
Neutrofil-tab	4.40 ± 1.02	4.40 ± 1.40	4.44 ± 1.42
Monocyte	6.40 ± 3.38	3.86 ± 2.58 ^a	3.11 ± 0.92 ^a
ESR	15.20 ± 4.49	8.26 ± 4.21 ^a	13.88 ± 6.54 ^b
ALT	23.20 ± 8.28	23.86 ± 10.25	18.88 ± 5.88
AST	21.80 ± 3.37	25.66 ± 10.88	21.11 ± 4.67
Cholesterol	173.11 ± 26.76	190.73 ± 41.26	190.00 ± 27.65
Glucose	29.18 ± 0.92	29.20 ± 1.03	29.20 ± 1.33

Note: Hb: hemoglobin; RBC: red blood cells; WBC: white blood cells; Ht: hematocrit; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; ESR: erythrocyte sedimentation rate; ALT: alanin aminotransferase; AST: aspartate aminotransferase. values are presented as mean ± SD; ^a $p < 0.05$; in comparison with control group; ^b $p < 0.05$; in comparison with coal dust exposure + non smoker; ^c $p < 0.05$; in comparison with coal dust exposure + smoker.

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