

Relationship Between Biomass Exposure, Chronic Headache and Brain Damage in Young Women

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ABSTRACT

Biomass, an energy source, is the general name of all non-fossilized biological material obtained from living or recently living organism. Household use of solid biomass fuels is the most common cause of indoor air pollution (IAP) worldwide. The aim of this study was to investigate the relationship between biomass exposure and the presence of headache and brain damage in young women.

This cross-sectional study included patient group consisted of 69 women who applied to the neurology outpatient clinic with complaints of chronic headache and fatigue and the control group consisted of 26 healthy volunteer women who were not exposed to *tandır* smoke. In all subjects, blood samples were obtained for biochemical analysis and brain Magnetic Resonance Imaging (MRI) scan was performed to evaluate brain damage.

In the patient group, ischemic gliotic foci were detected in 56 patient (81.2%) while in the control group, gliotic foci was detected in 3 subjects (11.5%). The prevalence of gliotic foci was almost 8 times higher in subjects with a history of biomass smoke exposure than in subjects without a history of biomass smoke exposure (81.2% *vs.* 11.5%).

These findings suggest that indoor air pollution may have an effect on the risk of headache and brain damage. However additional studies with larger sample sizes and including other ethnic or environmental communities are needed to determine in detail the role of indoor air pollution in the occurrence of headaches and brain damage.

Key Words: Biomass, headache, indoor air pollution, gliotic foci

Introduction

Biomass is a source of primary energy consisting of all substances of vegetal and/or animal origin such as wood, manure, and crop residue. Household use of solid biomass fuels is the most common cause of indoor air pollution (IAP) worldwide (1). The IAP caused by the combustion of solid fuels is a significant cause of morbidity and mortality particularly in developing countries. According to World Health Organization (WHO), about 1.6 million people around the world die prematurely from diseases associated with IAP caused by the inefficient use of solid fuels every year, and the morbidity rate among adult women is 50% higher than that of adult men (2). Most of these women are exposed to biomass smoke when cooking bread or meals over a period of more than 5 hours each day. In developing countries, biomass fuels are commonly used for cooking and heating homes and more than 2 billion people worldwide use these fuels as a source of primary energy (3,4).

Inefficient combustion of biomass fuels on an open fire or traditional stove substantially increases indoor levels of health-threatening pollutants such as hydrocarbons, nitrogen dioxide (NO₂), and carbon monoxide (CO). The resultant IAP has been associated with numerous health conditions including chronic obstructive pulmonary disease (COPD), lung cancer, lower respiratory tract infections, cataract, cardiovascular diseases, and adverse pregnancy outcomes (5,6). Biomass smoke (particulates) and sulfur dioxide (SO₂) are known to cause eye discomfort and CO is known to be a cause headache. However, there is little evidence suggesting an association between these results and IAP (7).

In Turkey, biomass fuels are mostly used in regions with lower socioeconomic status (such as Eastern Anatolia Region). In these regions, the most widely used biomass fuel is called *turd*, which is a fuel type produced by dehumidifying animal waste. *Turd* is utilized for cooking bread and meals in a covered oven called *tandır*, which has no ventilation source except for the entry door

and a small chimney. Biomass smoke is a mixture of complex particles, gases such as carbon monoxide, nitrous oxides, aldehydes, sulfur oxides, and polycyclic aromatic hydrocarbons (6,8).

Headache is a major cause of morbidity in modern societies. An association between air pollutants or other environmental factors and migraine or other headache types has not been widely accepted by clinicians. Nevertheless, it has been suggested that exposure to CO triggers neurogenic inflammation, thereby leading to migraine attacks and headache (9). Some previous studies also suggested that IAP could be associated with migraine and other headache types (10,11).

In this cross-sectional study, we aimed to investigate the relationship between IAP exposure, headache and brain damage, which, to our knowledge, has never been investigated in the literature.

Materials and Methods

A total of 95 women were included in this study. Patient group included 69 women aged 22-40 years who applied to out-patient headache clinic in the Department of Neurology at Van Training and Research Hospital. All the patients declared that they spent at least 6 h/week cooking bread and/or meals in tandir for the last 5 years. A control group was formed by 26 volunteering healthy women with no history of exposure to tandir smoke. In both groups, the subjects were young women that had no history of passive or active smoking exposure and were living in rural areas of low socioeconomic status with little air pollution. Total duration of exposure to biomass smoke was expressed as hour/week. Patients with systemic diseases and those detected with migraine, tension-type headache, cranial neuralgia, autonomous headache based on patient history and physical examination findings were excluded from the study. In addition, for the exclusion of vasculitis, patients positive for antinuclear antibody (ANA), anti-smooth muscle antibodies (ASMA), perinuclear antineutrophil cytoplasmic antibodies (p-ANCA), cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA), antistreptolysin O (ASO), C-reactive protein (CRP), and rheumatoid factor (RF) and patients with genetic factors associated with hypercoagulability including protein C deficiency, protein S deficiency, Factor V Leiden, and prothrombin gene mutation were excluded from the study. The subjects in both groups had normal lung function

and symptoms. Blood samples were collected in all subjects and were sent for biochemical analysis. Following centrifugation, plasma and serum samples were stored at -20 °C until analysis. Brain injury was assessed in each subject using cranial magnetic resonance imaging (MRI) (General Electric 1.5T SIGNA Explorer MRI System) with 5-mm-thick slides. All patients were informed about the details of the study, and written consent was obtained. The study was approved by Van Regional Training and Research Hospital, Non-Invasive Clinical Research Ethics Committee and was conducted in accordance with the Declaration of Helsinki.

Statistical Analysis: Descriptive statistics for the continuous variables were presented as Mean and Standard deviation, while count and percentages for categorical variables. Student t test was used to compare control and patient groups. Chi-square test was performed to determine the relationship between categorical variables. Statistical significance level was considered as 5% and SPSS (ver: 13) statistical program was used for all statistical computations.

Results

Table 1 presents the clinical and demographic characteristics of both groups. Mean age was similar in the patient and control groups (31.49 ± 5.84 and 31.58 ± 5.57 years, respectively). The two groups were divided into three subgroups based on their educational level: (I) illiterate (patients, $n=24$ [34.8%]; controls, $n=10$ [38.5%]), (II) primary school graduate (patients, $n=40$ [58.0%]; controls, $n=15$ [57.7%]), and (III) high school graduate (patients, $n=5$ [7.2%]; controls, $n=1$ [3.8%]). All the subjects had normal systolic and diastolic blood pressures. In the patient group, 40 (58.0%) patients had itching and burning in the eyes associated with biomass smoke exposure and 36 (52.2%) patients had backache. In both groups, there were no active smokers and the subjects were receiving no drug therapy. Additionally, no subject had a systemic disease (e.g. hypertension, diabetes mellitus, vasculitis) and all the subjects had normal respiratory function test results.

As shown in Table 2, no pathology was detected in the brain of 19 subjects (73.1%) in the control group, while gliotic foci was detected in 3 subjects (11.5%) and other pathologies (sinusitis, arachnoid cyst, etc.) were detected in 4 subjects (15.4%). In the patient group, no pathology was detected in the brain in 3 subjects (4.3%) while ischemic gliotic foci were detected in 56 patient (81.2%) and other pathologies (sinusitis, arachnoid cyst) were detected in 10 (14.5%)

Table 1. Descriptive characteristics of the study group

	Patient group (n=69)	Healthy control group (n=26)
Age (year) (Mean ± SD)	31,49 ± 5,84	31,58 ± 5,57
Biomass exposure (year)	5-6	-
Education status		
- Illiterate	24	10
- Primary school	40	15
- High school	5	1
Systolic blood pressure (Mean±SD)	117±6	110±5
Diastolic blood pressure (Mean±SD)	75±7	70±8
Itching of the eyes (yes / no)	40/29	-
Back pain (yes / no)	36/33	-
Smoking	none	none
Medication use	none	none

Table 2. Distribution of gliotic foci status in patient and control groups

		Group		Total
		Control	Patient	
Normal	Count	19	3	22
	% within foci	86,4%	13,6%	100,0%
	% within Group	73,1%	4,3%	23,2%
	% of Total	20,0%	3,2%	23,2%
Gliotic foci	Count	3	56	59
	% within foci	5,1%	94,9%	100,0%
	% within Group	11,5%	81,2%	62,1%
	% of Total	3,2%	58,9%	62,1%
Other pathology	Count	4	10	14
	% within foci	28,6%	71,4%	100,0%
	% within Group	15,4%	14,5%	14,7%
	% of Total	4,2%	10,5%	14,7%
Total	Count	26	69	95
	% within foci	27,4%	72,6%	100,0%
	% within Group	100,0%	100,0%	100,0%
	% of Total	27,4%	72,6%	100,0%

Chi-square = 53.268; p = 0,001

patients. These findings suggest that the prevalence of gliotic foci was almost 8 times higher in subjects with a history of biomass smoke exposure than in subjects without a history of biomass smoke exposure (81.2% *vs.* 11.5%). Figure 1 presents Hyperintense gliotic foci in white matter on Axial Flair MR image in a patient.

Discussion

In developing countries, household use of solid biomass fuels for cooking and heating exposes many

women to high levels of toxic air pollutants. In such countries, these fuels are mostly burned on open fires or in simple, inefficient stoves with no ventilation, resulting in the production of numerous harmful pollutants, ultimately leading to IAP levels that are typically many times higher than the standards for ambient air quality. The particulates in the air are known to cause acute and chronic respiratory and cardiovascular diseases via inflammation and oxidative stress. In addition, a limited number of studies have shown a relationship between particulate matter and headache and migraine. (10-13).

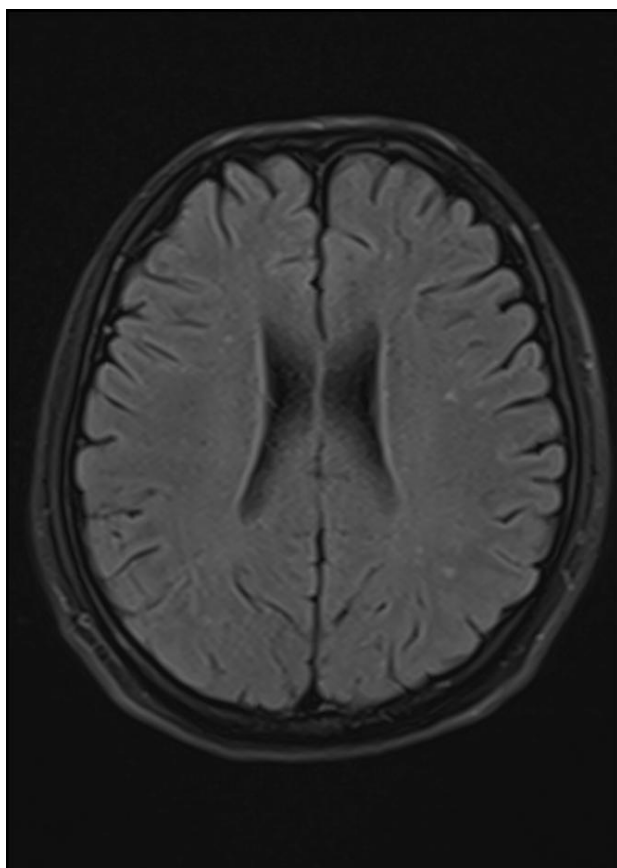


Fig. 1. Hyperintense gliotic foci in white matter on Axial Flair MR image

Nevertheless, to our knowledge, there has been no study in the literature evaluating the relationship between IAP and brain damage.

In rural areas of Van Province, women commonly use biomass fuels (turd, firewood) for cooking and heating. The present study found that the prevalence of headache and cerebral gliotic foci was relatively higher in women with a history of biomass smoke exposure. Similarly, neurogenic switching, in which exposure to irritants generates an afferent signal that induces a distant response, potentially in a different organ system, has been suggested as a mechanism through which neurogenic inflammation induced by exposure to air pollution may lead to migraine headache (14).

A common MRI finding in old-age individuals is single or multiple white matter hyperintensities (WMH) which can be focal or confluent. Although these foci can be seen at any age, they are detected in almost any individual aged over 75 years. Moreover, although they cause no symptoms in healthy individuals, they can be an indicator of a significant clinical condition such as multiple sclerosis (MS) and other demyelinating diseases, metabolic and toxic diseases, cardiovascular abnormalities, chemo- and radiation-induced

alterations, and infectious, tumoral, vascular, and traumatic lesions. However, the most common two mechanisms resulting from WMH include (i) insufficient remyelination and the resultant gliosis and (ii) vascular insufficiency that cause gliotic changes. While focal WMH have no ischemic findings, confluent WMH typically present with ischemic findings (15,16). In our study, the WMH detected in the patients showed no tendency for confluence. On the other hand, literature indicates that the prevalence of white matter lesions increases with age, particularly as a result of systemic diseases including hypertension, diabetes mellitus, and vasculitis (16). In our study, all the patients were young and no patient had a systemic disease.

Diaz et al. found a high prevalence of headache, backache, and eye problems in women exposed to high levels of indoor smoke pollution (≥ 5 h/day) when cooking meals on an open wood fire (17). However, the authors performed no evaluation of brain injury in the patients. On the other hand, some other studies reported a significant relationship between air pollutants and the frequency of emergency department visits for headache and migraine (9,18). In addition, no research has been conducted on brain damage in these studies.

Systemic oxidative stress caused by air pollution leads to endothelial dysfunction which is considered as a primary event in the formation of white matter damage. Endothelial dysfunction may have two major pathogenic outcomes including (i) reduced perfusion or cerebral blood flow (CBF) in the white matter and (ii) alterations of blood-brain barrier (BBB) permeability. The resultant hypoperfusion and BBB impairment causes additional oxidative stress by inducing tissue hypoxia and by extravasation of plasma proteins such as fibrinogen. Moreover, tissue edema caused by increased BBB permeability may worsen these changes by compressing blood vessels and reducing CBF further. Tissue hypoxia and oxidative stress activate inflammatory pathways via nuclear factor κ B (NF- κ B)-dependent transcription, thereby causing production of cytokines and adhesion molecules in vascular cells, activated microglia, and reactive astrocytes. Hypoxia, inflammation, and oxidative stress damage oligodendrocytes and cause trophic uncoupling in the neurovascular unit, thereby contributing to the damage to vascular cells and oligodendrocytes. Moreover, oligodendrocyte damage, inflammation, oxidative stress cause demyelination. When demyelination is formed, the

increased energy requirement of the denuded axons intensifies the hypoxic stress of the tissue, thus resulting in a vicious circle that administers these pathogenic processes and aggravates the tissue damage (19,20).

The present study had several limitations. First, all the subjects included in the study were young women who were residing in rural areas in Van province and thus the study included no subjects from urban areas. Accordingly, the results of the study may not be generalized to individuals of other races/ethnicities or to individuals residing in other regions/areas. Second, factors such as the use of ventilated tandirs and the total duration of exposure to tandir smoke are likely to affect the intensity of subjects' exposure to tandir smoke and, in turn, may affect the cerebral findings of the subjects. Third, no central-site ambient monitor was used to quantify the subjects' level of exposure to tandir smoke. Finally, although the study indicated a potential relationship between long-term exposure to biomass smoke and the presence of headache and cerebral gliotic foci in young women, the study did not evaluate other qualitative variables that could have a potential effect on brain health. Further longitudinal studies are needed to substantiate our findings and to provide a better understanding of the relationship between brain damage and exposure to biomass smoke in young women. Additionally, further studies are recommended to evaluate the effects of potential factors that were not investigated in the present study.

In conclusion, to our knowledge, the present study is the first to investigate the association between biomass smoke exposure and the presence of headache and cerebral ischemic gliotic foci in young women. The results indicated that the prevalence of cerebral ischemic gliotic foci was relatively higher in women exposed to biomass smoke. Physical interventions such as improvement of ventilation and regular cleaning and maintenance of stoves can contribute to reducing adverse effects of biomass fuels.

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