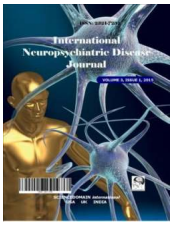

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Headache Due to Cerebral Venous Thrombosis after Carbonmonoxide Intoxication

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Carbon monoxide; CO is a colourless, odourless, tasteless and non-irritant type of gas. CO emerges as a consequence of poor combustion of carbon-based fuels, which can lead to acute and chronic poisoning. Despite the same exposure conditions, individuals may be confronted with different symptoms of CO poisoning. There are no qualitative studies on the long-term effects of CO poisoning on the headache, but there exist some reports of headache after chronic poisoning. The combination of cerebral venous thrombosis; CVT with carbonmonoxideintoxication has not been reported before. The most common symptom of patient's admission for CVT was reported as headache [6]. This rate has been reported as between 77-88% in various studies. Papillary oedema and optical atrophy after CO toxicity have often been reported, but this situation could not be related to increased intracranial pressure.Here we present a patient with headache due to carbon monoxide poisoning 3 months later.The headache of this patient was temporally related to carbon monoxide intoxication,and CVT and intracranial hypertension findings were detected. We have never encountered such a case presentation in the literature before. It is interesting because of the first instance. We would like to draw attention to the possibility of intracranial hypertension and CVT in a headache after carbon monoxide poisoning.

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Keywords: Carbonmonoxide intoxication; headache; papillary oedema; cerebral venous thrombosis.

1. INTRODUCTION

CO is a colourless, odourless, tasteless and non-irritant type of gas. CO emerges as a consequence of poor combustion of carbon-based fuels, which can lead to acute and chronic poisoning.In our country, CO poisoning is frequent in small areas where ventilation is inadequate especially in winter, as a result of coal use in stoveand water heater without achimney, and affected patients frequently refer to the emergency services of hospitals. Despite the same exposure conditions, individuals may be confronted with different symptoms of CO poisoning. Most patients with carboxyhemoglobin; COHb levels below 10% are asymptomatic.COHb concentrations below 20% are usually associated with nausea, headache and mild dyspnoea, between 20% and 40% with vomiting, evaluation difficulty and visual impairment, and values above 40% are associated with ataxia, confusion, syncope, coma and tachypnea [1]. There are no qualitative studies on the long-term effects of CO poisoning on the headache, but there exist some reports of headache after chronic poisoning [2]. Although CVT has a broad spectrum of symptoms, a majority of cases can be diagnosed only by imaging tests which are applied as a result of a clinical suspicion.Headache complaint is the most common symptom of CVT. However, it never appears alone, and this may be accompanied by findings due to an increase in the intracranial pressure [3]. In this work, we planned to submit a case of a CVT combined with CO intoxication.

2. CASE

A 44-year-old female patient was admitted to our outpatient. Algology clinic with a complaint of headache. It was observed in the anamnesis of the patient that she had the headache for 3-month. The headache was bilateral and pulsating, accompanied by nausea and vomiting, photophobia and phonophobia. The patient did not have any medication overuse. Headache was used to happen everyday. The patient had no oral contraceptive, alcohol or medication use and did not used to smoking. On neurological examination of the patient, her conscious was open, she was oriented and cooperated, cranial nerves were intact, and muscle strength was normal. The patient had bilateral papillary oedema. Cerebral MRIwas requested from the patient due to the detection of papillary oedema in the neurological examination.In the cerebral MRI, due to an appearance of thesuprasellar cistern as herniated in the sella cavity, it was reported as "bilateral perioptic CSF distances are evident; BIH". MR venography was appliedregardingaetiology due to patient's symptoms of increased intracranial pressure. In MR venography, flow and wall contour irregularities were observed at the distal part of the left transverse sinus (Fig. 1), and this was reported as "significant for chronic thrombosis". The patient stated that she underwent carbon monoxide intoxication 3 months ago, stayed in anaesthesia intensive care unit, received hyperbaric oxygen therapy, her headache started later, and had no headache before. The patient's thrombophilia panel such as level of protein C and S were normal, factor 5 leiden and prothrombin mutation were negative. Andalsoshe had no previous ny infection history. The patient has applied acetazolamide and enoxaparin treatment (1.5 mg / kg singledose daily sc). At the second month follow-up, headachewas reduced.The patient is still under clinical follow-up.

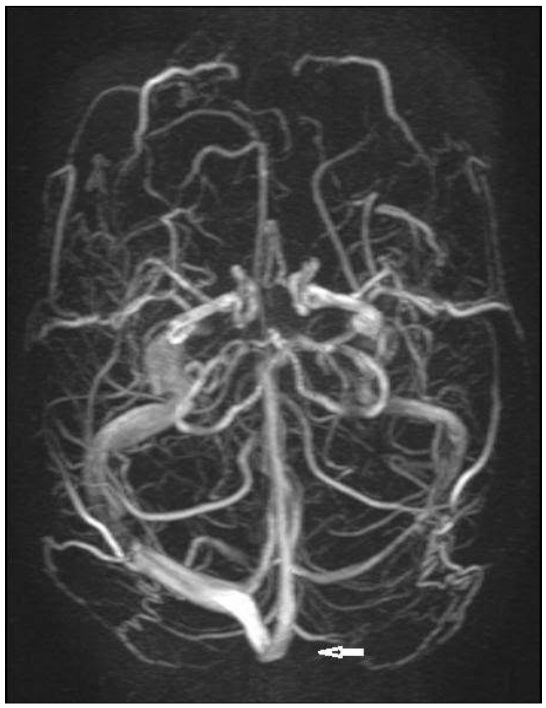

Fig. 1. In MR venography, flow and wall contour irregularities were observed at the distal part of the left transverse sinus

3. DISCUSSION

The decrease in arterial oxygen transport and shift of the oxyhemoglobin dissociation curve to the left explain the acute hypoxic symptoms observed in CO poisoning. Children, older adults and active individuals are affected more rapidly; exercise, stress and anaemiaincrease the tendency. Due to the high oxygen requirement, the brain and heart are the most sensitive organs to hypoxic effects of CO exposure. However the clinical manifestations of CO poisoning are very varying, they are often nonspecific. Headache is the most common symptom, and in the acute phase, there may be observed malaise, nausea, dizziness, mental instability, lethargy, somnolence, stroke, arrhythmia and cardiac arrest. Severe CO poisoning can cause neurological symptoms such as stroke, syncope, or coma and can lead to metabolic symptoms such as myocardial ischemia, ventricular arrhythmia, pulmonary oedema, and severe lactic acidosis.Neuropsychiatric disorders such as dementia, memory loss, personality changes, learning difficulty, behaviour, attention and concentration impairment, psychosis, parkinsonism, paralysis, chorea, apraxia, peripheral neuropathy and incontinence can be observed in 10-30% of cases after approximately 3-240 days after CO poisoning [4]. The International Classification of Headache Disorders (ICHD-III beta) defines carbon monoxide-induced headache as a bilateral headache, with quality and intensity that is related to the severity of CO intoxication. According to ICHD-IIIbeta, it develops within 12 hours of exposure and resolves within 72 hours after elimination of CO [2]. CO may induce headache and pain through the following mechanisms; hypoxia, impairment of mitochondrial function, modulation of gene expression, activation of cyclic guanosine monophosphate; cGMP signalling pathways, activation of calcium-gated potassium channels, interactions with nitric oxide [5]. In the CNS, neurones are the cells which require glucose and oxygen and are the most sensitive cells for hypoxia and ischemia. Acute and intense CO intoxication causes hypoxic-ischaemic encephalopathy mainly in grey matter. Focal involvement of the cerebral cortex in acute CO poisoning occurs less frequently. Temporal lobe and hippocampus are more sensitive to be affected. Damage causes transient vasogenicoedema or opens necrosis without occlusion in the cerebral arteries [6]. Damage often occurs immediately. For unclear reasons, the globus pallidus is affected more, but it is likely that there is much arterial blood flow to the watershed areas and the CO is excessively attached to the globus pallidus rich in iron [7]. CT often shows symmetrical hypodensity. MRI shows low signal densities in T1 sequences and high signal densities in T2 and FLAIR sequences [8]. In cerebral MRI due to CO intoxication, involvements can be seen in cerebral regions such as globus pallidus, hippocampus, and parsreticularis of thesubstantianigra. These can sometimes be accompanied by Purkinje cell loss in the cerebellum and laminar necrosis in the cortex [9]. In the medical literature, the character of the headache is not helpful to distinguish the CVT from other types of headaches [10]. Headache can be acute, subacute, chronic onset, localised or diffuse and variable. Headache may be associated with nausea and vomiting and may be continuous or vascular [11]. Because headache can be a lateralized, and vascular type, it can be misdiagnosed withmigraine [12]. Headachecan sometimes be in the form of a thunderclap mimicking subarachnoid haemorrhage [13,14]. Headache occurs more commonly with hemorrhagic than with ischemic stroke. Arboix and et al. found that headache occurred in 38% patients with ischemic stroke and 64.5% patients with hemorrhagic stroke. Also, it was shown that in ischemic stroke, headache was observed in 41% of thrombotic infarcts, in 39% of cardioembolic infarcts, in 23% of lacunar infarcts and 26% of TIA, as well [15]. Headache in cerebral venous thrombosis is 80% which is quite high [16].

Etiological factors include pregnancy and puerperium, oral contraceptive use, coagulation disorders, haematological diseases, systemic inflammatory diseases, infections, malignancies, severe dehydration, heart diseases and trauma. In spite of this diversity in aetiology, in 5-20% of cases, no etiological factor was detected [17]. None of these etiological factors were present in our case. The combination of cerebral venous thrombosis with CO intoxication has not been reported before. Papilloedema and optical atrophy after CO toxicity have often been reported, but this situation could be related to increased intracranial pressure [18]. This headache is coded as headache attributed to intracranial hypertension secondary to metabolic, toxic or hormonal causes in classification of IHS –III beta (2) and need to intracranial diseases (including venous sinus thrombosis) ruled out by appropriate investigations. Cerebral venous thrombosis was detected in addition to findings of

intracranial pressure increase in our patient. We think that CO poisoning in our patients may lead to venous sinus thrombosis.

Acute occlusion cases in coronary vessels have often been reported, although acute occlusions due to CO intoxication in cerebral vessels have not been reported before. It is known that vasoconstrictionis caused by CO gas which increases platelet aggregation as well as effects the vessels directly. This situation can be related with hypercoagulopathy [19]. Venous thrombosis should come to mind when findings of intracranial pressure are detected on resistant and persistent headaches after the CO intoxication.

4. CONCLUSION

Headache is a common finding after CO poisoning. It is known that toxic substances lead to intracranial pressure increase and cause headache. There is no evidence that CO intoxication is associated with cerebral venous sinus thrombosis. In our case, there were findings of cerebral venous sinus thrombosis and intracranial hypertension which we thought were after CO intoxication. There is some opinion in the literature about CO poisoning being able to cause clotting. Our aim in publishing our case is the first in the literature to show that CO poisoning may lead to cerebral sinus thrombosis. It is known that vasoconstrictionis caused by CO gas which increases platelet aggregation as well as effects the vessels directly. This situation can be relatedwithhypercoagulopathy and cerebral venous sinus thrombosis. We would like to draw attention to the possible association between CO poisoning and cerebral venous sinus thrombosis in this case.

CONSENT

All authors declare that 'written informed consent was obtained from the patient for publication of this paper and accompanying images".

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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