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Why did she lose her sight? A case of visual damage due to methanol inhalation

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메탄올 흡입 후 발생한 시각장애 : 증례보고

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Abstract Methanol is a clear, colorless, volatile, and poisonous liquid that is commonly used as an industrial solvent. Visual impairment is a common symptom of methanol poisoning; however, visual impairment rarely occurs after exposure through inhalation. Therefore, visual loss after methanol intoxication via respiration has rarely been reported. We report a case of visual damage associated with methanol poisoning via respiratory exposure in an industrial setting. In this case in South Korea, a 28-year-old woman who worked at a cell phone factory was admitted to the emergency department with mental changes. She had blurred vision that began two days prior, but she did not come to the hospital until she experienced mental changes. She ranked 9 on the Glasgow Coma Scale and presented with severe metabolic acidosis. So, she was admitted to intensive care, and continuous renal replacement therapy was performed. Finally, she was discharged after recovery of her mental state, but had to undergo rehabilitation for six months. Also, her visual impairment was permanent. Methanol intoxication can occur through inhalation, which is difficult to detect initially. However, treatment of methanol poisoning is time-critical. Therefore, doctors should always keep in mind that methanol intoxication may occur via respiration. If in doubt, treatment should be given as soon as possible.

요 약 본 메탄올은 공업용 용매로 흔히 사용되는 무색의 휘발성, 유독성 액체이다. 메탄올 중독에 의해서 시각 장애는 흔히 나타나는 증상 및 후유증의 하나지만, 흡입으로 인한 중독에서는 잘 나타나지 않는 것으로 알려져 있다. 그렇기 때문에 메탄올을 흡인한 이후에 시력저하가 생긴 증례는 잘 발표되어 있지 않다. 우리는 산업장에서 호흡기를 통해서 메탄올이 흡입된 이후에 시력손실이 발생한 케이스를 보고하고자 한다. 한국의 한 휴대폰 공장에서 일하던 28세 여자환자가 의식저하로 응급실로 내원하였다. 그녀는 이틀 전부터 시야가 흐릿하게 보였지만 대수롭지 않게 여겨 따로 병원을 찾아가지 않았고, 의식저하가 발생하여서야 병원에 오게 되었다. 내원시 환자는 글래스고혼수척도 9점의 의식저하를 보였으며, 혈액 검사 결과 심한 대사성 산증을 보이고 있었다. 중환자실에 입원하여 응급 투석을 비롯하여 메탄올 중독에 대한 치료를 시행하였다. 치료 후 의식이 회복되어 퇴원할 수 있었으나, 6개월 후까지 재활치료를 받고 있으며, 영구적으로 시력저하가 남았다. 흡입을 통해서도 메탄올 중독이 발생 할 수 있으며, 이는 초기에 알아차리기가 쉽지 않다. 하지만 메탄올 중독의 치료는 시간과의 싸움이며, 초기에 빠르게 치료해야 예후가 좋다. 그러므로 흡입에 의한 중독 가능성을 항상 염두에 두어야 하며, 의심시에는 빠르게 치료를 해야만 한다.

Keywords : Methanol intoxication, Visual damage, Inhalation exposure, Occupational exposure, Ocular toxicology

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1. Introduction

Methanol is a clear, colorless, volatile, and poisonous liquid that is commonly used as an industrial solvent [1]. Accidental ingestion of methanol can occur because it smells and tastes like ethanol [2]. In addition, there have been reports of methanol poisoning via inhalation or absorption through skin in industrial settings [3].

Methanol poisoning can be very dangerous, and intoxication can cause convulsions, hypothermia, stupor, coma, and death depending on the dose. Visual loss occurs in most survivors [4]. Methanol toxicity is caused by its metabolites formaldehyde and formic acid. In particular, formic acid causes severe metabolic acidosis and ocular toxicity [5, 6]. However, visual loss due to methanol poisoning usually caused via oral route, is rarely caused by inhalation [7, 8]. Therefore, there is little report that visual damage via respiratory route. In Korea, Choi J-H, et al[9] reported three cases of neurological and visual complications resulting from respiratory and dermal methanol poisoning.

We report a case of visual damage associated with methanol poisoning via respiratory exposure in an industrial setting.

2. Case report

A 28-year-old woman was admitted to the emergency department with mental changes. She had no medical, surgical and family history. According to her roommate, she had blurred vision that began two days prior. On the day of admission, when her roommate arrived home after work, her consciousness was decreased. Therefore, she was transferred to the hospital by the 119 rescue service. She had no past medical or drug history. As part of her occupational history, she had started working at a cell phone factory five days earlier. Her vital signs included a blood pressure of 140/90, pulse of 92/min, respiratory rate of 40/min, body temperature of 36.5℃, and a 99% oxygen saturation on ambient air. The initial clinical examination revealed confusion, and she ranked 9 on the Glasgow Coma Scale (eye opening response: 2, best verbal response: 3, best motor response: 4).

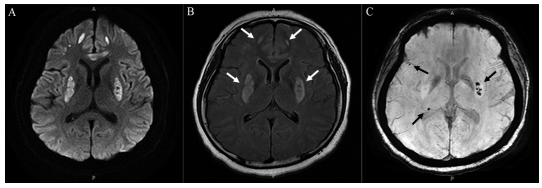

Fig. 1. Brain magnetic resonance imaging
(A) diffusion weighted image, (B) T2 weighted image, and (C) susceptibility weighted image

The patient's initial laboratory findings revealed high anion gap metabolic acidosis (pH: 7.094, pCO2: 10.0 mmHg, pO2: 148.6 mmHg, bicarbonate: 3.0 mmol/L, base excess: -24.0 mmol/L, anion gap: 30.0, sodium: 138 mEq/L, potassium: 4.96 mEq/L, chloride: 110 mEq/L, blood urea nitrogen: 8.1 mg/dL, creatinine: 1.1 mg/dL, serum osmolality: 322 mOsm/kg, urine osmolality: 532 mOsm/kg, and ethanol: < 2.20 mmol/L).

She was diagnosed with methanol intoxication based on blood test such as high anion gap metabolic acidosis and history taking. She fell into a stuporous mental state over time and had tachypnea. Therefore, she underwent tracheal intubation and was admitted into the intensive care unit. Continuous renal replacement therapy was performed owing to severe metabolic acidosis. On the second day of hospitalization, her consciousness was restored to alert, and metabolic acidosis was corrected (pH: 7.352, pCO2: 18.5 mmHg, bicarbonate: 10.0 mmol/L). Therefore, continuous renal replacement therapy was stopped. The following day, her consciousness deteriorated into a stupor. Therefore, electroencephalography was performed, resulting in non-convulsive status epilepticus. An antiepileptic drug was prescribed. On the fourth day of hospitalization, brain MRI was performed, which revealed bilateral symmetrical T2 high intensity lesions (white arrow) in the putamen and

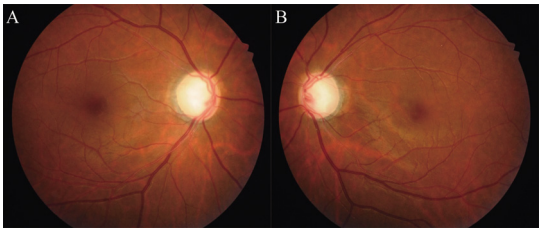

Fig. 2. Fundus examination
(A) right eye, (B) left eye

frontal subcortical area with multiple small hemorrhages (black arrow) in the left basal ganglia (Fig. 1). The next day, her consciousness recovered markedly; however, she complained of visual impairment, and she had a visual acuity of hand motion in both eyes. Fundus examination revealed bilateral pale optic disc (Fig. 2). Moreover, she had quadriparesis, with both upper and lower motor activity ranked as grade 2. She underwent rehabilitation for six months, and the quadriparesis improved, resulting in upper and lower motor activities of grade 4. However, her visual acuity did not improve within six months.

3. Discussion

Visual disturbance is a common symptom of methanol poisoning. However, visual impairment rarely occurs after methanol inhalation [7, 8]. This is because the amount of exposure is generally small via respiratory route unlike oral route. There is a report that visual damage has occurred by exposure via respiratory and dermal route in industrial field. In this report, patients are exposed to methanol while working without any protective equipment, resulting in visual impairment [9]. Ethanol is usually used in the process of cutting aluminum during cell phone manufacturing. In our case, a police investigation found that methanol had been used in the patient's workplace because methanol was one-third the price of ethanol. In addition, no protective equipment, such as masks, had been provided. Therefore, methanol vapor was inhaled into the respiratory system [10], which caused metabolic acidosis, decreased consciousness, and visual impairment. However, we could not use ethanol or fomepizole as an antidote because we did not recognize the patient was methanol poisoning at first.

Symptoms of methanol intoxication appear after a 12–24-h latency period [11]. Early symptoms of eye problems manifest as photophobia, blurred vision, and eye pain, which can worsen and cause complete vision loss [12]. Accumulation of formic acid, a metabolite of methanol, causes damage to the retina and optic nerve, and longer exposure to formic acid results in more severe symptoms and even permanent visual impairment [13]. In our case, the patient complained of blurred vision two days before she came to the hospital. Therefore, treatment was delayed and permanent eye problems were caused due to prolonged exposure to formic acid.

Bilateral putaminal and subcortical white matter edema or hemorrhagic necrosis are common findings in methanol poisoning via oral ingestion [14]. In our case, bilateral putaminal hemorrhagic necrosis occurred, even though exposure occurred via inhalation. Severe methanol poisoning may result in decreased consciousness and seizures [15], and non-convulsive status epilepticus occurred in our case.

The initial treatment of methanol intoxication is time-critical, and it is important to limit further damage by toxic metabolites. Treatment includes administration of antidotes such as fomepizole or alcohol and extracorporeal techniques [16]. Thus, it is necessary to diagnose methanol poisoning rapidly and treat it accordingly. In our case, the patient waited to go the hospital because she did not recognize that she had been exposed to methanol, resulting in a poor prognosis.

As a limitation in this case, we could not measure methanol concentrations in our hospital; therefore, the patient's serum methanol level was unknown. Regardless, the patient was believed to have experienced methanol poisoning for the following

reasons. First, methanol was used in the workplace by police investigation. And methanol was measured in the workplace and found to be in the range of 1103-2220 ppm. Other patient who worked at this factory had 7.632 mg / L of urine ethanol, which exceeded 50 percents over the exposure limit even though 72 hours had passed from the last exposure [10]. Second, the patient's symptoms included metabolic acidosis, unconsciousness, and visual impairment, parallel to those of methanol poisoning. Finally, the brain MRI was similar to that of methanol intoxication in previous reports.

4. Conclusion

Various types of intoxication can be observed in patients visiting the emergency department. Most are caused by oral exposure, for example drug intoxication, which are relatively easy to examine. However, intoxication via other routes of exposure, such as inhalation, are more difficult to detect. Therefore, a thorough patient history and physical examination should be performed while also considering the patient's occupation with effective storage plan of serum and urine samples of chemically exposed patients in preparation for delayed specific laboratory examination.

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