

Case Report

An Unusual Case of Drunk Driving in Japan: Alcohol-Related Sleepwalking

Ayako Himemiya-Hakucho^{a*}, and Tatsuya Fujimiya^b

Department of^aLegal Medicine, ^bMedical Education, Yamaguchi University Graduate School of Medicine, Ube, Yamaguchi 755-8505, Japan

Alcohol has been identified as a potential precipitating factor for parasomnia, particularly sleepwalking (SW). We report an unusual case of a Japanese drunk driver who may have experienced alcohol-related SW, based on the statements of the suspect, pharmacokinetic analyses of the suspect's breath alcohol concentration, testimonies of witnesses, driving recorder data, and medical records. The existence of sleep-related criminal acts performed while a suspect experiences memory loss under the influence of alcohol has not been sufficiently recognized, and awareness of such acts should be raised among the police, public prosecutors, and the general public in Japan.

Key words: drunk driving, sleepwalking, parasomnia, amnesia, blood alcohol concentration

Alcohol is the most commonly used drug in the world, and its abuse is a primary factor in many transport, domestic, and industrial accidents [1]. Driving a car while under the influence of alcohol has been a major social problem in Japan. A new, stricter road traffic law was enacted to prevent alcohol-related traffic accidents, injuries, and fatalities in 2002, and it has resulted in a decrease in alcohol-related traffic fatalities; however, drunk driving is still being reported [2, 3]. The police and public prosecutors often consult our department for pharmacokinetic analyses of blood alcohol concentration (BAC) and for interpretation of the effects of alcohol consumption on driving ability and/or the occurrence of road traffic accidents [4].

Alcohol has been identified as a potential precipitating factor for parasomnia, particularly sleepwalking (SW). SW, categorized as a classic form of non-rapid eye movement sleep arousal disorder, is a condition wherein an individual arises and ambulates without

fully awakening. Although it is very common in children, it is rare in adults, but it can be triggered by drugs and stimulants, including alcohol. In the SW state, an individual lacks conscious awareness and suffers from severe impairment of cognitive functions, such as attention, memory, social interaction, and planning. Nevertheless, the individual is capable of complex motor behavior, which means that SW could have devastating outcomes because of the risk associated with going outside in this state [5-7]. Previous case studies in Western countries have reported defendants who claimed to have been under a SW state during criminal acts, such as murders, attempted murders, assaults, and rapes, and who have claimed that alcohol intoxication was responsible for the occurrence of such criminal acts while SW [8, 9]; however, criminal cases of alcohol-related SW have rarely been reported in Japan. In this report, we present an unusual case of a Japanese drunk driver with complete memory loss, whose behavior was likely to have resulted from alco-

hol-related SW.

Case Report

A male suspect in his fifties (body weight, 71 kg) followed his usual routine: he took his mobile recharger, drove to work, and then changed into his work clothes and shaved his face in the dressing room at his office. The suspect entered the workroom at 5 : 00 p.m., although it was his day off and his usual work shift was in the daytime. He collided with a door when entering the workroom and staggered in. The first thing he said was "Good morning," somewhat inarticulately, but he did not react to the responses from his colleagues. After that, looking down, he sat in his seat and absently responded to a colleague who tried to explain to him that it was his day off. He did not appear to have any symptoms such as paralysis, seizure, vomiting, or injury. His heart rate was later determined to be 91 beats per min, and his blood pressure was 134/87; these data were collected by paramedics who were present because his office was at a fire station. However, his face was flushed, and his breath smelled of alcohol. His colleagues felt that he looked different than usual and were puzzled by his appearance. His superiors came to the scene about 45 min later and tried to conduct an interview with him; they ultimately called the police after surmising from the smell of alcohol that he had driven to the office while under the influence of alcohol. After the police investigation, the man was found to have a breath alcohol concentration (BrAC) of 0.55 mg/L at 7 : 07 p.m., which exceeded the legal limit in Japan.

The suspect confessed that he had woken up at around 4 : 00 a.m. and visited a hot spring, which he enjoyed until around 6 : 30 a.m., because it was a day off for him. He had played a Japanese pinball game at a pinball saloon for about 30 min beginning around 9 : 00 a.m. and then bought some food at a supermarket. Finally, he reached home at around 12 : 00 p.m., drank 2 glasses of an alcoholic beverage with a 25% ethanol content (the police determined this amount to be 338 mL by reproducing the alcohol consumed by the suspect) with lunch until around 12 : 30 p.m., fell asleep watching TV in his living room, and next remembered being interviewed by his superiors. It was the first time he had experienced complete memory loss after consuming alcohol. He was perplexed and could

not understand his behavior.

Data from the driving recorder on his car showed dangerous driving for around 25 km from 3 : 38 p.m. to 4 : 12 p.m. on that day. He had crossed the center line and the roadside line 3 times each, driven through a red light, hit a pole while making a left turn, drove in the opposite lane against oncoming traffic, and finally, spent about 10 min parking the car in his office parking lot.

Two days later, the suspect had a medical examination and was diagnosed with transient global amnesia (TGA) because the episode occurred suddenly with memory loss within 24 h and was accompanied by no symptoms before or after the episode, and it could not be attributed to other neurological conditions, such as epilepsy or stroke, based on electroencephalography (EEG), magnetic resonance imaging (MRI), and magnetic resonance angiography (MRA). Neither arrhythmia nor cardiac abnormality was seen on the electrocardiogram or echocardiogram. The blood tests showed diabetes mellitus, hyperlipidemia, and hyperuricemia, but each condition was mild or moderate. The suspect did not take medications or supplements and had no history of head trauma or family history of parasomnia. He lived alone and worked only the day shift. He usually drank two glasses of an alcoholic beverage with 25% ethanol content with dinner every evening and slept from 10 : 00 p.m. to 5 : 00 a.m.

The police consulted us regarding the association between the alcohol consumed, BAC, and the behavior of the drunk driving suspect with complete memory loss.

Pharmacokinetic analyses of BAC. Widmark's formula was used to estimate the BAC; this formula relates to the one-compartment model with zero-order elimination kinetics [10]. The BACs were estimated from a noninvasive BrAC measurement. A plasma/breath ratio of 2000 : 1 is typically used in Japan [3]. The β value, *i.e.*, the zero-order elimination rate constant, used was 0.16 or 0.20 [4].

Our simulation presumed that the suspect had drunk the alcoholic beverages at 12 : 15 p.m., which is the intermediate time between 12 : 00 p.m. and 12 : 30 p.m., because the precise drinking start time was unknown. First, we estimated the forward time-course of the BAC based on the amount of alcohol consumed because the theoretical initial BAC (C_0) was 1.35 mg/mL, which is sufficiently high that it cannot be

considered to be due to the influence of the absorption phase or due to the first pass effect. Consequently, when the β_{60} values were 0.16 and 0.20, the calculated BACs at 7:07 p.m. were 0.25 and 0 mg/mL, respectively, which are markedly lower than the BAC of 1.1 mg/mL that was derived from the actual measured BrAC of 0.55 mg/mL (Fig. 1). The β_{60} value calculated based on this forward time-course of BAC was 0.04 mg/mL/h, which is too low to be realistic for a living human without a serious illness. These results suggested that the suspect incorrectly estimated the amount of alcohol consumed.

Then, we estimated the backward time-course of the BAC from the actual measured BrAC. When the β_{60} values were 0.16 and 0.20, the calculated BAC values during the time the suspect was driving (3:38 p.m. to 4:12 p.m.) were 1.66-1.57 and 1.80-1.68 mg/mL, respectively, which suggested that he committed a drunk driving offense. When the β_{60} values were 0.16 and 0.20, the calculated BACs at 12:15 p.m. were 2.20 and 2.47 mg/mL, respectively, which are markedly higher than the C_0 value of 1.35 mg/mL calculated based on the amount of alcohol consumed (Fig. 1). These results again indicated that the suspect incorrectly esti-

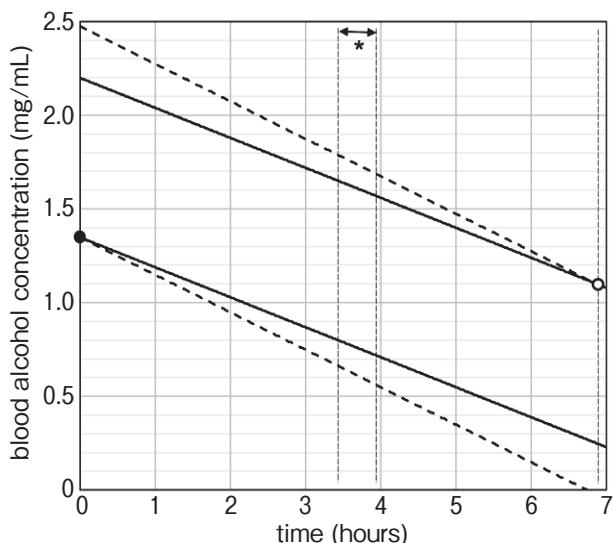


Fig. 1 Estimation of the time-course of BAC when the β_{60} value was 0.16 (—) and 0.20 (- -) using Widmark's formula. The upper two lines show the time-course calculated backward from the BAC based on the actual measured BrAC; the white circle (\circ) shows 1.1 mg/mL at 7:07 p.m. The lower two lines show the time-course calculated from the amount of drinking; the black circle (\bullet) shows a C_0 value of 1.35 mg/mL at 12:15 p.m. The asterisk (*) shows the period when the suspect was driving.

mated the amount of alcohol consumed. The estimated amounts of alcohol consumed were 546 and 615 mL of an alcoholic beverage with a 25% ethanol content when the β_{60} values were 0.16 and 0.20, respectively.

Discussion

In the present case, we describe an unusual case of a Japanese drunk driver with complete memory loss. We evaluated the case based on the pharmacokinetic analyses of BrAC, the testimonies of the witnesses, driving recorder data, medical records, and statements of the suspect. The suspect actually had consumed approximately 1.6 to 1.8 times the amount of alcohol mentioned in his statement because the peak BAC value was calculated to be >2 mg/mL; we judged that he incorrectly estimated the amount of alcohol consumed due to alcohol-induced amnesia. He then fell asleep due to alcohol-induced decreased sleep latency. Finally, he drove his car to work "as usual." The major limitation of this case, however, is the lack of objective evidence that the suspect fell asleep after drinking because he was alone.

There is no consensus among sleep experts regarding the association between alcohol and SW. Some authors have argued that there is no direct experimental evidence that alcohol predisposes or triggers SW due to increases in slow-wave sleep, from which SW results due to incomplete arousal; hence, SW should not be diagnosed in the presence of severe alcohol intoxication [8, 11]. However, other authors have declared that precluding the relationship based on a lack of evidence is inappropriate and may have unwarranted medico-legal implications for defendants [12-15]. In the present case, no evidence based on a sleep-expert clinical interview, polysomnographic sleep study with video monitoring, or polysomnographic sleep study with video monitoring and alcohol ingestion was used to elucidate the cause of the subject's behavior; however other evidence was suggestive of alcohol-related SW.

According to the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition, the diagnostic criteria for SW in non-rapid eye movement (NREM) sleep arousal disorders are the following: (1) experiencing recurrent episodes of incomplete awakening from sleep, usually occurring during the first third of the major sleep episode, accompanied by SW; (2) recalling little or no dream imagery; (3) showing amnesia regarding

the episodes; (4) experiencing clinically significant distress and impairment in social, occupational, or other important areas of functioning owing to the episodes; (5) experiencing disturbances not attributable to the physiological effects of a substance; and (6) showing coexisting mental and medical disorders that do not justify the episodes of SW. The definition of SW within the criteria is as follows: repeated episodes of rising from bed during sleep and walking about; the individual has a blank, staring face during SW; the individual is relatively unresponsive to the efforts of others to communicate with him or her; and the individual can be awakened only with great difficulty [5]. In the present case, the suspect's episode at least partially met all the criteria except the fifth one. The onset seemed to occur approximately 2 h after falling asleep; this it occurred during approximately the first third of his usual major sleep episode. While the suspect had not used other substances and had no coexisting mental or medical disorders, it is unknown whether the suspect had experienced recurrent episodes, although he was not aware of a similar previous experience. His appearance and his condition at his office were consistent with the definitions of SW, and amnesia regarding his driving to work was present. He followed his usual routine, including such activities as changing into his work clothes and shaving his face, but the activities of subjects with SW may range from walking to conducting an involved sequence of semipurposive actions [5].

Conversely, alcohol consumption can be related to other types of parasomnia such as REM sleep behavior disorder (RBD) and nightmares. Subjects with RBD literally enact their dreams due to a loss of atonia during REM sleep. Actions such as punching, kicking, leaping, running from bed, and even diving through a window have been correlated with dream imagery. Unlike with SW, the subject with RBD seems unaware of the actual environment but acts on the dream sensorium and typically remembers the dream. RBD may occur during withdrawal from alcohol, and it is associated with a variety of neurological conditions including Parkinson's disease, dementia, and others [5]. In the present case, the suspect's episode did not match the characteristics of RBD, and he was not undergoing withdrawal from alcohol. Furthermore, his episode differs from the immediate biochemical effects of alcohol, such as alcohol-induced blackout, which is defined as amnesia or memory loss, for all or part of a drinking

episode, during which the subject can actively engage with and respond to their environment [16,17]. There is also another differential diagnosis related to the immediate biochemical effects of alcohol. In idiosyncratic alcohol intoxication (IAI), which has been variously called pathogenic, complicated, atypical, and paranoid alcohol intoxication, a severe behavioral syndrome develops rapidly after the subject drinks a small amount of alcohol. A person with IAI experiences confusion, disorientation, illusions, transitory delusions, and/or visual hallucinations, and may display greatly increased psychomotor activity and impulsive, aggressive behavior. IAI, usually described as lasting a few hours, terminates during prolonged sleep, and patients cannot recall the episodes on awakening [5]. However, our suspect's episodes and the amount of alcohol consumed do not coincide with the characteristics of IAI.

In Japan, the maximum alcohol concentration permitted by the Road Traffic Act is 0.15 mg/L when measured by breath, so a BAC of 0.3 mg/mL is considered the legal limit [3]. In the present case, the pharmacokinetic analyses of BAC based on the actual measured BrAC when the β_{60} values were 0.16-0.20 showed levels in the range of 1.66-1.80 mg/mL during driving. The immediate biochemical effects of alcohol on the brain are either depressing or stimulating in nature, depending on the BAC resulting from the amount of alcohol consumed. Voluntary motor actions usually become perceptibly clumsy at 1.0 mg/mL; at 2.0 mg/mL, the function of the entire motor area of the brain is measurably depressed and the parts of the brain that control emotional behavior are also affected [2,5]. Alcohol consumption increases the risk of crash because of effects such as poor judgment, increased reaction time, lower vigilance, decreased visual acuity, and depressed consciousness [2]. The suspect in the present case drove abnormally while under the influence of alcohol as the driving recorder data showed, but fortuitously did not cause a traffic accident.

The suspect was clinically diagnosed with TGA. The main criterion for TGA is heteroanamnestically confirmed anterograde amnesia. Additionally, the amnesia is commonly accompanied by headache, dizziness, and nausea. TGA is also characterized by the absence of clouding of consciousness, focal neurological symptoms, epileptic features, recent head injury or active epilepsy as well as a reduced ability to identify oneself. Most patients also engage in repetitive questioning

during the episode because of anxiety or agitation [18]. We think that the criteria do not match the episode experienced by the present suspect because he was simply unresponsive without any associated symptoms, such as repetitive questioning, although he had amnesia. Moreover, he had no neurological findings on EEG, MRI, or MRA.

We conclude that it is highly possible that the suspect experienced SW as a type of parasomnia due to alcohol, so it should be judged that he was non compos mentis while driving drunk, since he might not have been able to control his behavior while in a state of SW. However, if he were to commit criminal acts during SW under the influence of alcohol in the future, those crimes should not be excusable because he has now been made aware of his tendency to exhibit alcohol-related complications. It is our opinion that he must abstain from alcohol in the future. The occurrence of sleep-related criminal acts performed while a suspect experiences complete memory loss under the influence of alcohol has not been sufficiently recognized. Awareness of this phenomenon should be raised among the police, public prosecutors, and the general public in Japan. It is desirable that sleep- and alcohol-related criminal acts should be carefully interpreted by experts in both alcohol-related medicine and sleep medicine.

References

1. Saukko P and Knight B: Knight's forensic pathology. 3rd Ed. Arnold, Great Britain (2004) pp 552-559.
2. Global Road Safety Partnership, World Health Organization, FIA Foundation for the Automobile and Society: Drinking and Driving: a road safety manual for decision-makers and practitioners. Geneva, Switzerland (2007) pp 3-21.
3. Desapriya E, Shimizu S, Pike I, Subzwari S and Scime G: Impact of lowering the legal blood alcohol concentration limit to 0.03 on male, female and teenage drivers involved alcohol-related crashes in Japan. *Int J Inj Contr Saf Promot* (2007) 14: 181-187.
4. Himemiya-Hakucho A and Fujimiya T: Pharmacokinetic analyses using absorption kinetics in low-alcohol dose cases of drunken driving. *Leg Med (Tokyo)* (2017) 26: 98-101.
5. Sadock BJ, Sadock VA and Ruiz P: KAPLAN & SADOCK'S Synopsis of psychiatry. 11th Ed. Wolters Kluwer, Philadelphia (2014) pp 1343-1364.
6. Cohen De and Cock V: Sleepwalking. *Curr Treat Options Neurol* (2016) 18: 6.
7. Stallman HM, Kohler M and White J: Medication induced sleepwalking: A systematic review. *Sleep Med Rev* (2018) 37: 105-113.
8. Pressman MR, Mahowald MW, Schenck CH and Bornemann MC: Alcohol-induced sleepwalking or confusional arousal as a defense to criminal behavior: a review of scientific evidence, methods and forensic considerations. *J Sleep Res* (2007) 16: 198-212.
9. Siclari F, Khatami R, Urbanik F, Nobili L, Mahowald MW, Schenck CH, Cramer Bornemann MA and Bassetti CL: Violence in sleep. *Brain* (2010) 133: 3494-3509.
10. Widmark EMP. Verleilung und unwandlung des ethyl alcohols in organismus des hundes (Disposition and constant of ethyl alcohol in dog). *Biochems Z* (1993) 267: 128-134.
11. Pressman MR, Mahowald MW, Schenck CH, Cramer Bornemann MA, Banerjee D, Buchanan P and Zadra A: Alcohol, sleepwalking and violence: Lack of reliable scientific evidence. *Brain* (2013) 136: e229.
12. Ebrahim I and Fenwick P: Letter to the Editor re: Pressman et al. Alcohol-induced sleepwalking or confusional arousal as a defense to criminal behavior: a review of scientific evidence, methods and forensic considerations, *J. Sleep Res* (2007) 16, 198-212. *J Sleep Res* (2008) 17: 470-472.
13. Ebrahim I and Fenwick P: Sleep related violence, alcohol and sleepwalking. *Brain* (2012) 135: e219.
14. Cartwright RD: Alcohol and NREM Parasomnias: Evidence versus Opinions in the International Classification of Sleep Disorders 3rd Edition. *J Clin Sleep Med* (2014) 10: 1039-1040.
15. Rumbold JM, Riha RL and Morrison I: Alcohol and non-rapid eye movement parasomnias: where is the evidence? *J Clin Sleep Med* (2014) 10: 345.
16. Perry PJ, Argo TR, Barnett MJ, Liesveld JL, Liskow B, Hernan JM, Trnka MG and Brabson MA: The association of alcohol-induced blackouts and grayouts to blood alcohol concentrations. *J Forensic Sci* (2006) 51: 896-899.
17. Wetherill RR and Fromme K: Alcohol-induced blackouts: A review of recent clinical research with practical implications and recommendations for future studies. *Alcohol Clin Exp Res* (2016) 40: 922-935.
18. Spiegel DR, Smith J, Wade RR, Cherukuru N, Ursani A, Dobruskina Y, Crist T, Busch RF, Dhanani RM and Dreyer N: Transient global amnesia: current perspectives. *Neuropsychiatr Dis Treat* (2017) 13: 2691-2703.