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Case Report: Catastrophic Effects of Using Cannabis Via Bucket Bong in Top End Northern Territory of Australia

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Abstract. The prevalence of cannabis usage is increasing worldwide, including among both Indigenous and non-Indigenous Australians. The long-term effects of cannabis use on the lungs are well-known. However, the acute adverse effects on the lungs are sparsely reported. There are different ways in which cannabis can be inhaled, such as smoking or through a water vaporizing method known as a "bong." An improvised innovative bong device that is commonly used in Northern Australia, called a "bucket bong," uses water and air pressure to assist in cannabis inhalation. In this report, we describe three patients from remote and rural Northern Australian communities presenting with near–life-threatening events (acute pneumonitis and massive pneumothorax) immediately after the use of cannabis via bucket bong.

INTRODUCTION

According to a United Nations' report, cannabis is one of the most widely used illicit drugs worldwide, and it is reported that 4% of the global population aged 15 to 64 years has used cannabis during their life course.[1] The prevalence of cannabis use in Australia is reported to be significantly higher than the global average.[1,2] Moreover, consumption of cannabis is quite prevalent among those residing in remote and rural communities of Australia.[3] There are different ways in which cannabis can be inhaled, such as smoking or through a water vaporizing method known as a "bong." A "bucket bong" is an innovative bong device that uses water and air pressure to assist in cannabis inhalation. The long-term effects of cannabis on the lungs are well-known.[4,5] However, there is little evidence in the literature regarding acute lung injury after inhalation of cannabis, more specifically via a bucket bong. In this report, we describe three patients who presented with near catastrophic acute lung injury after the use of cannabis through a bucket bong, a common mode of bong use among the remote and rural communities of Northern Australia.

CASE REPORT

Case 1. A 40-year-old male from a remote community in the Northern Territory (NT) of Australia initially presented to a local community health center with sudden onset of cough, shortness of breath, and fever. He was noted to be febrile with a temperature of 39.5°C and an oxygen saturation value down to 83% on room air. His symptoms started approximately 1 hour after smoking cannabis through a bucket bong. He was transferred (airlifted) to the Royal Darwin Hospital (RDH), a tertiary care university-affiliated teaching hospital in the capital city, Darwin, NT of Australia. Upon presentation, a chest x-ray and high-resolution computed tomography (CT) of the patient's chest showed bilateral midzone alveolar opacity, particularly in the perihilar distribution, alongside ground glass opacities (Figure 1). He had raised inflammatory markers with a C-reactive protein level of 222 mg/L and a white cell count of 31.5×10^9/L with neutrophil predominance. The patient required admission to the intensive care unit because of persistent hypoxemia and required supplemental oxygen therapy. He was treated with a course of empirical antibiotics in view of being febrile at presentation and blood tests demonstrating raised inflammatory markers and high white cell count. No pathogenic organisms were cultured, and atypical pneumonia serology was subsequently negative. Otherwise, his connective tissue disease screen was unremarkable. The patient clinically improved, and after he recovered from his acute illness, pulmonary function tests revealed normal spirometry, normal total lung capacity, and mildly reduced diffusing capacity for carbon monoxide. It was concluded that this patient's presentation was likely secondary to acute pneumonitis in the context of cannabis use through a bucket bong.

Case 2. A 31-year-old male, again from a remote community in the NT of Australia, was transferred to RDH with a spontaneous right-sided pneumothorax requiring intercostal catheter (ICC) insertion (Figure 2A and B). The patient had no significant documented past medical comorbidities. However, he had a notable significant smoking history and cannabis use. It was noted that the current presentation occurred almost immediately after the consumption of cannabis through a bucket bong. A chest CT scan revealed background emphysematous changes with large bullous disease in the right lung apex (Figure 2C and D). The majority of the blood investigations were within normal ranges, including serum alpha-1 antitrypsin level. The patient had recurrence of his pneumothorax shortly after his ICC removal. Hence, he subsequently proceeded on to video-assisted thoracic surgery and talc pleurodesis and bullectomy. The cause of his pneumothorax was determined to be secondary to apical bullous lung disease, likely in the context of smoking cannabis via bucket bong.

Case 3. A 22-year-old female patient originally presented to a remote community health clinic with complaints of pleuritic chest pain and shortness of breath immediately after smoking cannabis via a bucket bong. She was initially misdiagnosed as having an exacerbation of airway disease and was treated with oral antibiotics. However, the astute reporting radiologist at the RDH alerted the community clinic about the presence of a large pneumothorax (Figure 3A), and she was eventually transferred to RDH by air after an ICC was

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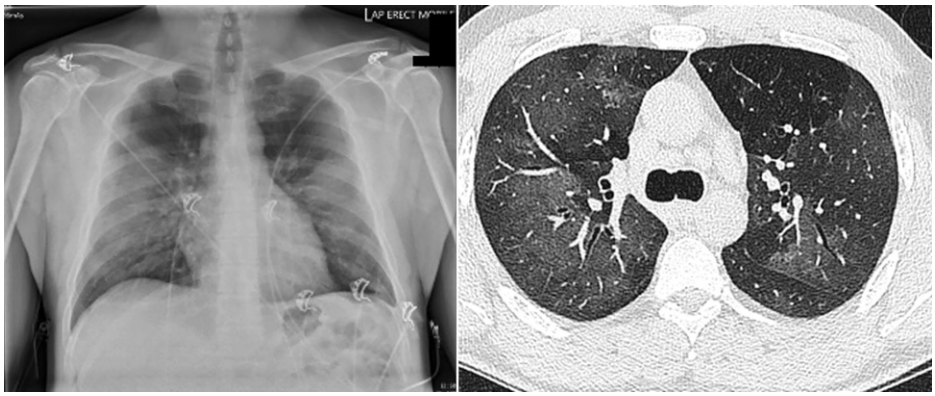

FIGURE 1. Chest x-ray and high-resolution CT of the chest showing bilateral pulmonary opacity. CT = computed tomography.

inserted in the remote community health clinic by the medical retrieval team (Figure 3B). A chest CT scan subsequently demonstrated a moderate to large right-sided pneumothorax. Several right apical, tiny, subpleural bullae were also noted (Figure 3C). The ICC was subsequently replaced with a large-bore chest drain owing to minimal improvement (Figure 3D). The pneumothorax gradually improved, and the chest drain was removed 6 days later. She was discharged from the hospital with a plan for ongoing close follow-up and advice/education provided on the ill effects of using cannabis through a bucket bong. The patient's spontaneous pneumothorax was deemed secondary to apical bullous lung disease in the setting of smoking cannabis via bucket bong.

DISCUSSION

To the best of the authors' knowledge, this is the first report of near-acute life-threatening lung conditions after

FIGURE 2. (**A**) Right-sided pneumothorax. (**B**) Intercostal catheter on the right side. (**C** and **D**) Chest CT showing emphysematous changes with bullae in the right lung apex. CT = computed tomography.

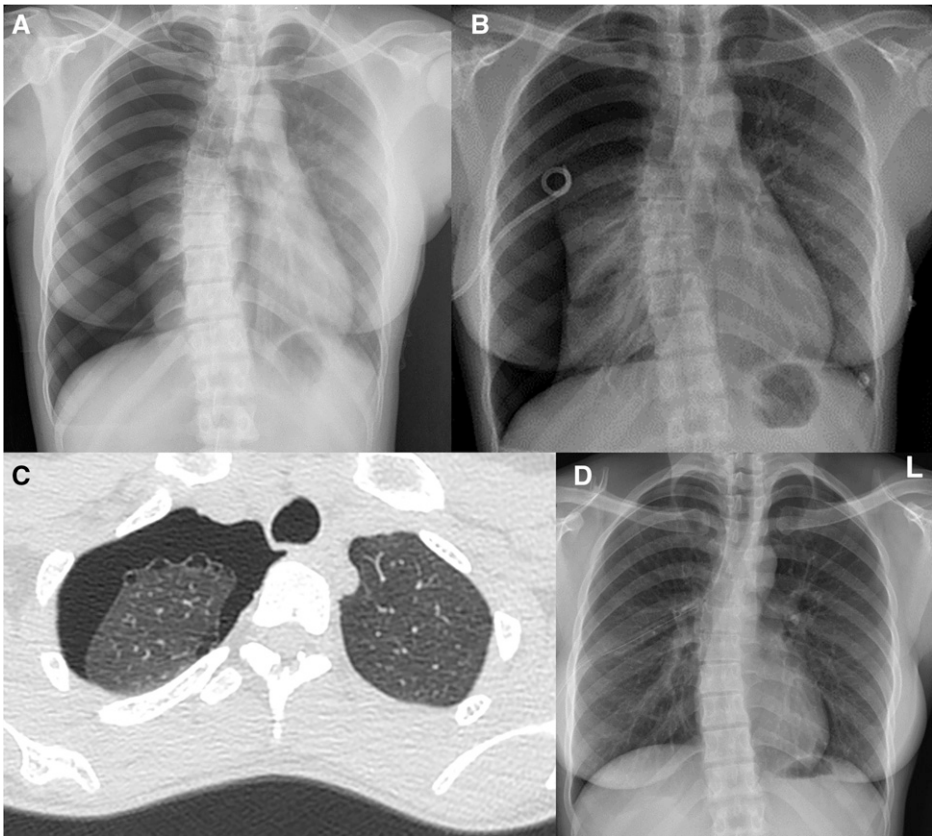

FIGURE 3. (**A**) Chest x-ray shows right-sided pneumothorax. (**B**) Intercostal catheter on the right side. (**C**) Chest CT shows large right-sided pneumothorax and bullae in the right lung apex. (**D**) Chest x-ray shows large-bore chest drain. CT = computed tomography.

consumption of cannabis via bucket bong, more specifically among people residing in the remote and rural communities of the Top End, NT region of Australia. There are different ways in which cannabis can be consumed.[6] The most common method of consumption is via smoking cannabis mixed with tobacco. However, it may also be inhaled through a vaporizing device or added to food and eaten or drunk.[6,7] Another typical method of use is through a bong, which uses water to assist in inhalation of the cannabis.[6] There are different types of bongs, including the standard and most common bong that draws cannabis smoke from a metal cone through water before being inhaled into the lungs.[6,7] Conversely, a bucket bong is an innovative method that uses water to create a suction force. The assembly of a bucket bong is indeed very simple. Conventionally, it is created by assembling a plastic bottle and bucket filled with water together as shown in Figure 4 (left image).[8] The mechanism behind its use is that the water pressure draws air through the bottle outlet by altering the water and air pressure inside the bottle. As the water rises inside the bottle, it forces smoke out of the bottle outlet, which can be inhaled into the lungs in high concentration/volume and pressure.[8,9]

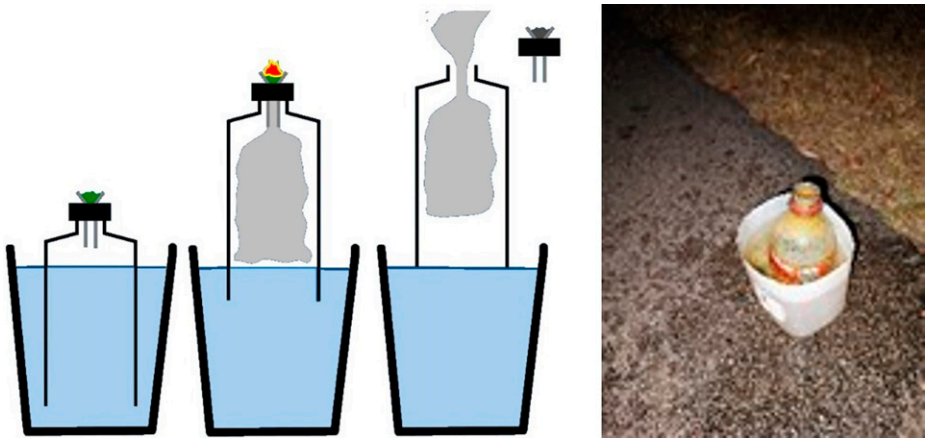

FIGURE 4. Diagram showing bucket bong in operation and picture of an actual bucket bong abandoned after use.

Anecdotally, this method allows users to get the maximum effects of cannabis instantly.

There is comprehensive literature describing the effects of cannabis on lungs among recreational inhaled illicit drug users,[10–12] including reports of patients diagnosed as having hypersensitive pneumonitis,[13,14] pneumothorax, and pneumomediastinum.[15,16] Moreover, lung function parameters among cannabis users in particular are noted to display higher forced vital capacity (FVC) and hyperinflation.[17–20] The reason for this increase in FVC is unclear, but it is theorized that it is due to the training of respiratory muscles by the characteristic inhalation techniques used by cannabis users. Furthermore, cannabis is proposed to have an acute bronchodilator effect. Because bronchodilatation of the small airways can increase FVC, this could also be the reason for the increased FVC in cannabis users.[21] Chest radiology details published in previous reports indicate that cannabis users could demonstrate the presence of emphysema and bullous disease.[22–24] Although it is postulated that this could be related to prolonged cannabis use, the evidence surrounding this linkage is less convincing, as it is confounded by a significant proportion of cannabis users also having a history of smoking tobacco.

In the context of the patients presented in this report, alongside the current evidence mentioned above, we hypothesize that for the patient with acute pneumonitis, rapid inhalation of cannabis mixed with smoke in high concentrations and volume, in addition to the possible inhalation of toxic plastic chemical substances in a potentially unhygienic condition (Figure 4, right image) through the bucket bong likely contributed to acute pneumonitis. For the other two patients with pneumothorax, the same phenomena could be responsible. Moreover, the Valsalva maneuver associated with the inhalational technique of cannabis could have led to overdistension of the lungs, in turn dramatically increasing the total lung volume. For these patients, the presence of prior bullous disease would further increase the risk of pneumothorax.

In addition, the altered physiological mechanism of pulmonary gas flow and volume could explain the occurrence of pneumothorax among bucket bong users. Consider Bernoulli's field equation for calculating pressure and flow rates:

$$P_a + {}^1\!/_2\rho(v_a)^2 + \rho g h_a = P_b + {}^1\!/_2\rho(v_b)^2 + \rho g h_b,$$

where P is pressure, ρ is fluid density, v is fluid velocity, g is gravity, and h is the height of the fluid column. Subsets a and b are two points along the flow path, namely the oral cavity (a) and the end alveolus (b).

By making several calculation assumptions, such as 1) velocity at the end alveolus is zero—zeroing term v_b, 2) the depth is constant (i.e., the weight of a column of air is negligible)—cancelling terms h_a and h_b on each side, 3) the pressure of each system at the start of inhalation (oral intake) is atmospheric—turning P_a into P_{atm}, then Bernoulli's equation could be simplified to $P_{atm} + \frac{1}{2}\rho {v_a}^2 = P_b$. Setting up two equations 1) for normal inspiration and 2) for the bucket bong and dividing the equations gives $\frac{P_{b_2}}{P_{b_1}} = \frac{\rho {V_{a_2}}^2}{\rho {V_{a_1}}^2} + 1$.

We can simplify this once more by assuming that the densities of air and marijuana smoke are comparable (a generous assumption given there is evidence that marijuana smoke is denser than air, albeit variably dependent on factors such as packing density and relative humidity)—removing the ρ in each equation and recognizing that the geometry (or airways topology) of both circumstances is identical—allowing "velocity" to be substituted with "flow rate," Q, giving $\frac{P_{b_2}}{P_{b_1}} = \frac{{Q_{a_2}}^2}{{Q_{a_1}}^2} + 1$, or $\frac{P_{bong}}{P_{insp}} = \frac{{Q_{bong}}^2}{{Q_{insp}}^2} + 1$. This could be interpreted as indicating that the relative pressure produced by a bucket bong compared to deep inspiration at the end alveolus is directly related to the relative squares of the initial volumetric flow rates. Volumetric flow rate, Q_1, is measured directly during spirometry as the inspiratory flow rate, and Q_2 depends on the build of the bucket bong; for example, a 2-L bong could be inhaled in approximately 1/4 of a second, giving a flow rate of 8 L/second.

The flow rate of a bucket bong (using 8 L/second), being significantly higher than deep inspiration (using 5 L/second), leads to a relative pressure of $\frac{P_{b_2}}{P_{b_1}} \geq 3.5$.

Considering that the bucket bong continues to force air inward, this becomes a plateau inspiratory pressure 3.5× higher than that achieved by maximum inspiratory pressure (normally ranging between 80 and 120 cmH_2O depending on body habitus). The plateau pressure achieved by a bucket bong can therefore be estimated at 300 cmH_2O, which is indeed several times higher than during conventional mechanical ventilation, which could produce barotrauma at pressures as low as 40 cmH_2O.[25,26]

Nevertheless, the adult population residing in the Top End, NT of Australia are reported to have a much higher burden of chronic respiratory disorders, including complex and advanced respiratory conditions, giving rise to significant morbidity and mortality.[27–45] The true prevalence of bucket bong consumption within NT remote communities is not exactly known. However, it is reasonable to speculate that what is represented in this report is only a glimpse of a potentially significant underexplored or underreported problem. In a population with an already higher burden of chronic respiratory disorders,[46,47] use of a bucket bong may further add not only to morbidity and mortality but also to healthcare cost and utilization. Hence, the patients presented in this report could be considered a wake-up call to explore avenues to implement patient educational strategies.[48,49]

CONCLUSION

The cases represented in this report illustrate an acute lung injury that could be caused by utilizing a bucket bong for cannabis consumption and the potential physiological mechanism involved in the development of pneumothorax. We hope that this report prompts future research/reports that can provide further information on the usage patterns and other health-related effects of cannabis use via bucket bong worldwide, with an ultimate goal of facilitating educational interventions to prevent catastrophic consequences.

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