Cannabinoid Hyperemesis: An Erratic Syndrome Linked with Cannabis Abuse

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Editorial

Cannabinoid hyperemesis syndrome (CHS) is characterized by chronic cannabis use, recurrent episodes of intractable nausea and vomiting, frequent hot bathing and abdominal pain [1]. The syndrome was first described by Allen et al. [2], and later Sontineni et al. [3] who proposed abridged clinical diagnostic criteria. Simonetto et al. by appraising all PubMed indexed journals with case reports and case series on CHS, reported a case series of 98 patients [4]. This study confirmed the previously stated outcomes. The researchers avowed that CHS should be measured in younger patients with chronic cannabis use and repeated nausea, vomiting, as well as pain in the abdominal. Amid illicit drug, cannabis is the utmost typically used drug in the world [5].

In line with the 2015 National Survey on Drug Use and Health, 22.2 million people have used cannabinoids in the past month [6]. The prevalence of use is higher in men than women, a gender gap that extended in the years 2007 to 2014 (Figure 1) [7]. The number of people affected is not diaphanous as of 2015 [8]. Cannabis use is linked with copious acute and chronic adverse effects as stated earlier such as vomiting, followed by cyclic vomiting syndrome, intense feelings of nausea and accompanying symptoms, abdominal discomfort and compulsive hot bathing behaviour [3]. Habboushe and Sedor, reported that CHS can lead to snags, for example, acute renal failure (ARF) [9].

The pathogenesis of CHS is covert, several mechanistic theories attempting to explain the exact pathology. These notions fall into two themes: dose reliant accumulation of cannabinoids and associated effects of cannabinoid toxicity; and the functionality of cannabinoid receptors in the brain and predominantly in the hypothalamus that controls body temperature and the digestive system [10]. Chang and Windish offer an outline of substantiating proof for these theories; however, the mechanisms by which cannabis engender controls nausea and the adverse magnitudes of chronic cannabis toxicity remain cryptic [11].

Cannabinoid binds to two kinds of G-protein coupled cannabinoid receptors, CB1 and CB2, that act by inhibiting adenylate cyclase [12]. In conjunction with the discovery of the CB, and CB receptors has been the identification of endogenous arachidonic acid derivatives that bind to these receptors called endogenous cannabinoids, or endocannabinoids [13]. The therapeutic potential of cannabinoids has been identified and these compounds are utilized as antiemetics for controlling nausea and vomiting as well as in the progressive phases of ailments such as cancer and acquired immune deficiency syndrome [14].

The acute incidents of CHS typically last for 24 to 48 h and the cessation of use is effective. Generally, 1 to 3 months is required for perfect recovery [15]. Numerous medications for nausea and vomiting are ineffective for this syndrome. Chen and McCarron stated that relief has been reported with lorazepam and haloperidol [16]. Furthermore, evaluating for dehydration owing to vomiting and hot showers is imperative as it can lead to ARF and resolved with intravenous hydration [16].

CHS is a novel and under-documented clinical entity. Advanced studies are obligatory to control this disease prevalence and its other epidemiological features, natural antiquity as well as pathophysiology. Further treatments are enforced and exertions to stop cannabis abuse are supreme.

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Competing Interests

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