

Research Article



Journal of Current Trends in Clinical Case Reports

Evaluation of Lipid Profile and Prostate Specific Antigen on Cannabis Smokers in Owerri

Nnodim Johnkennedy^{1*}, Ikpa Chimaobi Ikenna¹, Edward Ukamaka¹ and Nwanguma Eberechi¹

¹Department of Medical Laboratory Science, Faculty of Health Science, Imo State University, Owerri Nigeria

*Corresponding author: Nnodim Johnkennedy Department of Medical Laboratory Science, Faculty of Health Science, Imo State University, Owerri Nigeria; E-Mail: johnkennedy23@yahoo.com

Received Date: 26 August, 2020 Accepted Date: 02 September, 2020 Published Date: 18 September, 2020

Citation: Nnodim Johnkennedy, Ikpa Chimaobi Ikenna, Edward Ukamaka, Nwanguma Eberechi (2020) Cultivating Engagement in Research Using Social Media Networks to Recruit Participants for Health Related Research . J Cur Tre Clin Case Rep 1(1): 103

Abstract

This study was done to evaluate the levels of lipid profile and prostate specific antigen on cannabis smokers in Owerri. Forty students of Imo State University who used cannabis for less than three years between the ages of 18 and 35 years were used as test subject, while 20 students who do not use cannabis within the ages of 18 to 35 years were used as control. The levels of lipid profile (total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein) were determined by spectrophotometric method, while PSA level was determined by enzyme linked immunoassay (ELIZA) method. The results were analyzed using students t-test at P<0.05. The results obtained showed that the level of total cholesterol (163.51±30.31mg/dl) in cannabis smokers was not significantly increased when compared with the control (151.20±33.36mg/dl) at P<0.05. On the other hand, the level of PSA (3.58±0.4ng/ml) in cannabis smokers was significantly increased when compared with the control (2.74±0.21ng/ml) at P<0.05. This observation may probably imply that cannabis smoking could be a risk factor in the development of prostate cancer. Hence, smoking cannabis should be avoided.

Keywords: lipid profile, prostate specific antigen, cannabis, smokers

Introduction

Marijuana is a consist of shredded leaves, stems and flower buds of the Cannabis sativa plant [1]. It can be smoked, eaten, vaporized, brewed and even taken topically. The hallucinogenic effects of cannabis are mainly due to a chemical in cannabis called tetrahydrocannabinol. The other important component in cannabis is cannabidiol [2].

Cannabis can make some people giggly and chatty, and other people paranoid, confused and anxious [3]. Some people may experience mild hallucinations if they take particularly strong cannabis or may have problems concentrating and learning new information. It is observed that it can lead to students perform badly in exams. This is because cannabis impacts the part of the brain we use for learning and remembering things [4, 5].

In the same vein, smoking cannabis can lead to wheeze and out of breath and coughing uncomfortably or painfully [6]. Smoking cannabis has also been associated with increase in the risk of lung cancer and heart rate. Hence affecting blood pressure, which makes it particularly harmful for people with heart disease [7]. Cannabis smoking can affect sperm count and ovulation [8, 9].

Smoking cannabis may affect lipid profile. Lipid profile constitute a lipid panel of test including: total cholesterol, Highdensity lipoprotein cholesterol, triglycerides and very low-density lipoproteins. It's unclear how this lipid profile is affected among cannabis smokers [10, 11].

Prostate cancer is an established public health concern in modern society and has been for decades. It is the most common cancer in men (asides from non-melanoma skin cancer) and the second most common cause of cancer death [12]. Even with widespread screening with prostate-specific antigen (PSA), still 5% of cases present with metastatic lesions at the time of diagnosis [13]. In light of the above, there is a fundamental necessity to search for and find new and novel treatments to this common pathology. Cannabis and cannabinoids have often been an issue of much polemics in the realm of science, but since the discovery of cannabinoid receptors in rat brain in the late 1980s, there has been a growing interest in the research of these compounds and the knowledge continues to expand [12].

Cannabis is the preferred name of the plant Cannabis sativa, Cannabis indica, and Cannabis ruderalis. The Cannabis plant is a known potent psychoactive substance and can cause addiction in users. It is partly known that marijuana, the commonest recreational drug of abuse, may have adverse effects on cardiovascular disease. While some data suggest cannabis use to confer cardio metabolic benefits such as reductions in Low Density Lipoprotein (LDL) [14]. Some studies show that cannabis users have a lower plasma High Density Lipoprotein (HDL), which are important risk factors for cardiovascular disease [15].

Prostate cancer is the most common cancer and the second leading cause of cancer-related deaths affecting men in the Owerri Nigeria. Various studies have examined the effects of canabis

smoking on PSA, but were mostly carried out in the older men [12].

Due to the paucity of information, it will be of great importance to evaluate the implications of canmabis smoking on some biochemical parameters especially lipid profile and prostate specific antigen.

Materials and Methods Experimental Design

Forty students from Imo state university who used cannabis for not less than three year within the age of 18-35 years and twenty students who do not use cannabis but were age matched with the test subjects were used as control.

Sample Collection

Five (5) milliliters of blood sample was collected by standard venopuncture methodfrom each participant and was dispensed into

dry bottle. This was centrifuged to get the serum for the analysis of lipid profile and prostate specific antigen.

Biochemical assay: The serum lipid profile total cholesterol, triglyceride, high density lipoprotein cholesterol and low density lipoprotein cholesterol were determined by standard method [16]. While determination of serum prostate specific antigen was by enzyme linked immunoabsorbent assay (ELIZA).

Statistical Analysis

Results were presented in mean \pm standard deviation (SD). All data obtained in the study were analyzed using the student t—test (spss.20). The level of significance was set at p <0.05.

Results

Table 4.1: Mean value of Triglyceride, Total cholesterol, High density lipoprotein, Low density lipoprotein and PSA in Cannabis smokers Vs Non cannabis smokers (Controls).

Table 4.1: Mean value of Triglyceride, Total cholesterol, High density lipoprotein, Low density lipoprotein and PSA in Cannabis smokers Vs Non cannabis smokers (Controls)

Biochemical parameters	Cannabis smokers	Non cannabis smokers	t-value	p-value
TG (mg/dl)	75.73+ 10.6	72.12+ 10.66	0.54	0.606
TC (mg/dl)	163.51+ 30.31	151.20+ 33.36	0.61	0.558
HDL (mg/dl)	54.73+ 12.7	51.74+ 12.29	0.38	0.715
LDL (mg/dl)	82.03+ 12.32	74.89+ 16.02	0.79	0.452
PSA (ng/ml)	3.58+ 0.49	2.74+ 0.21	3.5	0.008

P<0.05 (Significant); P>0.05 (Not significant)

Discussion

Some health issues are associated with cannabis smoking particularly cardiovascular diseases as well as psychological problems [17, 18].

The current study reveals that there was no statistical significant difference (p>0.05) in the mean value of triglyceride in cannabis smokers when compared to non-cannabis smokers. This result confirms the work carried out by Muniyappa et al., which revealed that cannabis consumption did not affect the triglyceride in both subjects [19]. The result therefore proves that cannabis use doesn't affect triglyceride level.

There was no statistical significant difference (p>0.05) in the mean value of total cholesterol, HDL and LDL in cannabis smokers when compared to non-cannabis smokers. This is in line with the study carried out by Hossein et al., which investigated the hypercholesterolemic effect of drug-type Cannabis sativa seed in guinea pig, and found that serum high density lipoprotein cholesterol (HDL-c) level was not affected by the consumption of cannabis seed [20].

HDL-c has preventive role in coronary artery disease and some studies in animal models of atherosclerosis support the cardio protective role of HDL-c [21]. The HDL level of cannabis smokers was not significantly raised but there was a trend for HDL-c to increase, this means that cannabis could be beneficial to cardiovascular health. Some studies have also established that appetite is modulated by the cannabinoid [22].

The present study reveals that there was a statistical significant increase (p<0.05) in the mean value of serum PSA in cannabis smokers was when compared to non-cannabis smokers. Prostate specific antigen (PSA) is a protein that is produced by the cells of the prostate gland and enters the bloodstream. Studies by Smith et al., reveals that excessive use of Cannabis disrupts the normal structure of the prostate cells, resulting in increased amounts of active PSA entering the bloodstream before being inactivated [23]. Thus, the percentage of free (inactivated) PSA in the circulation is reduced, and there is a higher amount of bound PSA. The possible mechanism by which this occurs is not known.

It is observed from the study that there was significant increase in the PSA Level of cannabis smokers when compared to the control subjects while no effect was observed in the lipid profile. This may imply that cannabis smoking could be a risk factor for prostrate cancer.

References

- 1. Soleiman H and Mahmoud J (2010) Blood lipids in middleaged British men. British Heart Journal 49: 205-209.
- 2. Bauman A and Phongsavan P (2017) Epidemiology of substance use in adolescence: prevalence, trends and policy implications. Drug and Alcohol Dependence 55:187-207.
- 3. Samsha (2010) National Household Survey on Drug Use and Health (2011); NIDA, Monitoring the Future.
- 4. Baggio S, Deline S, Studer J, Mohler Kuo M, Daeppen JB and Gmel G (2014) Routes of administration of cannabis used for nonmedical purposes and associations with patterns of

- drug use. Journal Adolescence Health 54:235-240.
- Azorlosa JL, Greenwald MK and Stitzer ML (2015) Marijuana smoking: Effects of varying puff volume and breath hold duration. Journal Pharmacology and Experimental Therapy 272:560-569.
- 6. Moir D, Rickert WS, Levasseur G, Larose Y, Maertens R, White P and Desjardins S (2018) A comparison of mainstream and sidestream marijuana and tobacco cigarette smoke produced under two machine smoking conditions. Chemical Research Toxicology 21: 494-502.
- Adam DG, Heather M, Timothy B and James H (2011) Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. American Heart Journal 16:145-151.
- Brousseau ME and Schaefer EJ (2010) Diet and Coronary Heart Disease: Clinical Trials on Current Atherosclerosis 2: 487-493.
- 9. Thomas G, Kloner RA and Rezkalla S (2014) Adverse cardiovascular, cerebrovascular, and peripheral vascular effects of marijuana inhalation: what cardiologists need to know. American Journal Cardiology 113:187-90.
- 10. Fagerström K (2012) The epidemiology of smoking: health consequences and benefits of cessation. Drugs 62: 1-9.
- Hall W and Degenhard L (2009) Adverse health effects of non-medical cannabis use. Lancet 374: 1383-1391.
- 12. Aboul-Enein BH, Bernstein J and Ross MW (2016) Evidence for Masturbation and Prostate Cancer Risk: Do We Have a Verdict. Sexual Medicine Reviews 4: 229-234.
- 13. Alan J, Louis R, Alan W and Craig A (2015) Campbell-Walsh Urology (Eleventh ed.). Elsevier Health Sciences 1005.
- 14. Alexander DD, Mink PJ, Cushing CA and Sceurman B (2010)

- A review and meta-analysis of prospective studies of red and processed meat intake and prostate cancer Nutrition journal 9: 50-56.
- 15. Issa JS, Santos PC, Vieira LP, Abe TO, Kuperszmidt CS, et al. (2014) Smoking cessation and weight gain in patients with cardiovascular disease or risk factor. International Journal Cardiology 172:485-487.
- Chessbrough M (2007) District Laboratory practice in tropical countries ECedition. Cambridge University Press 2:80-85.
- 17. Penner EA, Buettner H and Mittleman MA (2013) The impact of marijuana use on glucose, insulin, and insulin resistance among US adults. American Journal of Medicine 12:583-589.
- 18. Cooney M, Dudina A, De Bacquer D, Wilhelmsen L, Sans S, et al. (2009) Athersclerosis 206:611-616.
- 19. Muniyappa R, Sable S, Ouwerkerk R, Mari A, Gharib AM, et al. (2013) Metabolic effects of chronic cannabis smoking. Diabetes Care 36: 2415-2422.
- 20. Hossein H and Isaac KI (2007) Hypercholesterolemic Effect of Drug-Type Cannabis sativa L. Seed (Marijuana Seed) in Guinea Pig. Pakistan Journal of Nutrition 6:59-62.
- 21. Kawahiri MC, Maugeais A and Rader D (2010) High density lipoprotein metabolism: Molecular target for new therapies for atherosclerosis. Current Atherosclerosis Report 2: 363-372.
- 22. Kirkham TC and Williams MC (2011 Endogenous cannabinoids and appetite. Nutrition. Research Reviews 14:65-86.
- 23. Smith RA, von Eschenbach AC and Wender R (2011) American Cancer Society guidelines for the early detection of cancer: Update of early detection guidelines for prostate, colorectal, and endometrial cancers. Also: Update 2001 Testing for early lung cancer detection. CA Cancer Journal of Clinical Chemistry 51:38-75.