e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2023; 15(8); 1467-1470

Original Research Article

Studying the Connection between Tobacco Chewing and the Quality of Semen in Patients Undergoing Infertility Evaluation

Vanisha Dhaka[1], Deepali Chauhan[2], V. Pawar[3], Harpreet Kaur[4]

[1]Assistant Professor, Department of Pathology, Maharishi Markandeshwar College of Medical Sciences and Research, Sadopur, Ambala, Haryana, India

[2]Assistant Professor, Department of Obstetrics and Gynaecology, Maharishi Markandeshwar College of Medical Sciences and Research, Sadopur, Ambala, Haryana, India

[3]Professor, Department of Pathology, Bharati Vidyapeeth Medical College & Hospital, Sangli, Maharashtra, India

[4]Assistant Professor, Department of Obstetrics and Gynaecology, Maharishi Markandeshwar College of Medical Sciences and Research, Sadopur, Ambala, Haryana, India

Received: 30-5-2023 / Revised: 30-06-2023 / Accepted: 30-07-2023

Corresponding author: Dr. Harpreet Kaur

Conflict of interest: Nil

Abstract:

Objective: The use of tobacco is one of several lifestyle choices that have an impact on male fertility. Chewing tobacco is a common addiction among Indian males. Our research's goal was to evaluate the connection between Indian men who are habitual tobacco chewers and the quality of their sperm.

Patient(s): Three categories of tobacco chewing frequency were used to classify 300 male patients having infertility evaluations: mild (3 times/day, n = 100), moderate (3–6 times/day, n = 100), and severe (>6 times/day, n = 100).

Results: The mild group had considerably greater sperm concentration, percentage motility, morphology, and percentage viability than the moderate group than the severe group. Although the differences were not statistically significant, the percentage of men with azoospermia is increased with the severity of addiction.

Conclusion: In our study, chewing tobacco was substantially linked to a decline in sperm quality and more deranged with increase in quantity of tobacco usage. Chewing tobacco therefore may have a negative impact on couple's social and mental wellbeing. So, timely advice for quitting tobacco and couple's counselling for fertility can help society in a better way.

Keywords: Male Infertility, Tobacco Chewing, Sperm Quality.

Introduction

Chewable tobacco is one of the smokeless tobacco products. The United States Department of Agriculture stated in 1993 that consumption of smokeless tobacco had tripled while manufacturing production had grown for eight years in a row. There are between 6 million and 22 million users of smokeless tobacco in the US, according to estimates [1]. According to a nationwide study of 5,894 college and university students from various parts of the country, 8% to 15% of them used smokeless tobacco [2]. Furthermore, a recent survey found that in 2022, 21.3 % of American male high school students were active users [3].

The behaviour seems to be particularly widespread among particular demographic groupings. For instance, lower income society males chew tobacco more frequently than higher income groups [4]. Young amateur and professional athletes appear to be particularly susceptible to the habit [5] as do people in countries including India, China, and South-east Asia [6]. Chewing tobacco usage is consistently linked to socioeconomic indicators in India, both at the individual and household levels. People without a formal education are 2.69 times more likely to smoke and chew tobacco than people who are educated. [7].

Smoking is also dangerous along with smokeless tobacco and other forms [8]. Chewing tobacco also minimally raises the incidence of oral and respiratory tract malignancies, according to a meta-analysis [9]. Though, chewing tobacco is linked to a decreased risk of cardiovascular morbidity than smoking [10]. The practise is regarded as the most significant risk factor for several oral premalignant lesions, while it has been firmly linked to oral malignant illnesses [11]. Smokeless tobacco contains a number of carcinogens, but the most significant ones are N-nitrosamine (TSNA), N-

nitrosonornicotine (NNN), and 4-(methylnitrosamino) -1- (3-pyridyl) -1- butanone (NNK). Nicotine is converted to NNN and NNK during fermentation, ageing, and curing [12]. Other substances, such 3-methyl nitrosamine propion aldehyde (MNPA), harm DNA by breaking its single strand and creating cross-links in proteins [13-21].

According to a growing number of investigations, environmental chemicals and physical factors may have an impact on human male fertility. The possible implications of tobacco consumption on reproductive health have drawn both scientific and public attention. The impact of tobacco chewing on male fertility is not well understood. As a result, the goal of our study was to assess the connection between smoking and sperm quality in male partners of infertile couples who were undergoing infertility assessment and advise them to quit tobacco chewing.

Materials and Methods

Individuals who attended the infertility clinic in Gynae outpatient Department were examined. 300 male partners of infertile couples who had infertility evaluations over the period of one and half years were included in our research. Each participant gave their free, informed consent the patients were between of 21 and 55 years of age. All had a history of tobacco chewing for average four to ten years, but no one had any other physical disease. Patients were divided into three categories based on how frequently they chewed tobacco: mild (3 times per day, n = 100), moderate (3- 6 times per day, n = 100), and severe (>6 times per day, n = 100). Semen analysis was done in accordance with the standards set out by the World Health Organisation (WHO) 2021. Azoospermia is when there is no sperms in the ejaculate, Asthenozoospermia, if motility is decreased, oligozoospermia, if there are less than $15x10^6$ sperm per millilitre, teratozoospermia if there were >04% abnormal sperm morphology. Total sperm motility should be more than or equal to 40%, sperm vitality should be more than or equal to 58%. [23]. One-way analysis of variance (ANOVA) was used to compare the three groups, and an unpaired t test was utilised to compare two groups. The significance threshold for each two-tailed hypothesis test was.05. Calculations were carried out using the statistical programme Graph Pad in Stat version 3.00 (Graph Pad Software, San Diego, CA).

Results

Men in the moderately addicted group had normal sperm parameters that were considerably greater than those in the highly addicted group, and vice versa (Table 1). According to WHO criteria, the sperm count, motility, morphology, and vitality of sperms were near normal in mild group (23). On the other hand, teratozoospermia was seen in samples from males in the moderate and severe categories.

The incidence of azoospermia increased in the severely addicted group, with rates of azoospermia in the mild, moderate, and severe tobacco chewing groups being 1%, 3%, and 14%, respectively. From mild to moderate to severe addiction, oligoasthenoteratozoospermia (OAT) incidence exhibited an upward tendency. We found decreased sperm motility and morphology values in men with a severe tobacco chewing habit compared to those with a moderate habit in semen samples characterised by OAT (Table 2). Due to the small number of samples characterised by OAT in the light tobacco chewing group, no comparisons were done with this group. When males from the three study groups were examined who had normal sperm count, motility, and vitality, the values of the normal sperm parameters were considerably lower in the men with a severe tobacco chewing habit compared to the moderate group and in the moderate compared to the mild group. In the group that are in mild tobacco group, normal sperm morphology was noticed as well as normal sperm count, motility, and vitality. In contrast, samples from the moderate and severe tobacco chewing groups had teratozoospermia, which was distinguished from samples with normal sperm count, motility, and vitality (Table 3).

Table 1: Comparison of sperm parameters in all semen samples collected from mild, moderate, and severe tobacco chewing groups

Parameter	Mild group (n=100)	Moderate group (n=100)	Severe group (n=100)
Sperm count(10^6/mL)	73.1±22.2	41.1±21.2	22.4±19.1
Motility (%)	56.3±6.2	50.2±11.2	46.4±14.3
Morphology (%)	26.7±8.6	22.5±9.3	15.3±7.8
Viability (%)	56.3±4.6	53.2±8.6	47.3±12.1

Table 2: Comparison of sperm parameters in samples characterized with oligoastheno-terato zoospermia from moderate and severe tobacco chewing groups

Parameter	Moderate group	Severe group	P value
Sperm count (10^6/mL)	3.8±1.6	3.88±1.34	0.4
Motility(%)	26.11±6.1	22.3±11.34	0.02
Morphology(%normal)	6.4±1.2	6.22±2.2	0.04
Viability (Eosin stain) (%)	32.3±5.4	29.3±9.4	0.2

Table 3: Comparison of sperm parameters in samples characterized with normal sperm count from mild, moderate, and severe tobacco chewing groups

Parameter	Mild group(n=100)	Moderate group(n=100)	Severe group(n=100)
Sperm count (10^6/mL)	76.3±37.2	47.33±22.1	35.1±22.16
Motility (%)	60.3±5.1	58.1±6.11	55.3±2.2
Morphology (%)	32.1±9.1	26.6±8.11	21.34±2.2
Viability (%)	63.5±6.1	60.2±6.34	60.22±4.11

Discussion

Regardless of the mode of ingestion, tobacco usage exerts a detrimental influence on morbidity and mortality rates. The deleterious consequences encompass an array of oral health issues, including gum recession and leukoplakia, compounded by the emergence of nicotine addiction and an augmented susceptibility to mortality from cardiovascular diseases and malignancies within the oral cavity, larynx, and pharynx [21]. This association also extends to smokeless tobacco products, which can be equally enslaving [22]. In a distinct context, our study focuses on males undergoing infertility assessment, a cohort often overlooked in the realm of tobacco's repercussions. Intriguingly, our investigation underscores a marked deterioration in semen quality parameters, namely sperm count, motility, morphology, and viability, mirroring the habit of chewing tobacco.

Inevitably, our findings appear in contrast to those reported by another study, one encompassing 119 tobacco chewers. Surprisingly, this prior research did not divulge any noteworthy fluctuations in sperm parameters when comparing tobacco users and nonusers [20]. Nonetheless, the pivotal divergence resides in the scale of our study, boasting a significantly larger sample size of n = 300, thereby augmenting our sensitivity in identifying significant discrepancies. Furthermore, we adopted a unique approach of categorizing patients based on the frequency of tobacco usage, thus permitting a more discerning assessment of chewing tobacco's impact.

The implications of chronic systemic nicotine exposure are multifaceted and extend beyond the domain of infertility. Its potential to expedite coronary artery disease and precipitate episodes of acute myocardial ischemia, coupled with a propensity to elevate blood pressure, has been well-documented [20]. Chewed tobacco not only affords a direct route for nicotine absorption but may also compound the repercussions of this habit in the context of male infertility.

Animal models subjected to nicotine exposure showcased pronounced inflammatory responses and distinct ultrastructural modifications within their testes [19,20]. Furthermore, seminal plasma analysis in humans has revealed the presence of cotinine and trans-3-hydroxycotinine, prominent nicotine metabolites, with their levels inversely correlated to sperm quality [5]. This intriguing correlation between nicotine metabolites and semen parameters underscores the intricate interplay between nicotine exposure and spermatogenesis. As these complexities emerge, it becomes increasingly evident that further research endeavours are imperative to unravel the full spectrum of nicotine's influence on male reproductive health.

Furthermore, the relationship between nicotine and male fertility extends to molecular and cellular levels. Emerging evidence suggests that nicotine may impact spermatogenesis through various mechanisms, including disruption of oxidative balance, induction of DNA damage, and modulation of hormone levels. Oxidative stress induced by nicotine can perturb the delicate equilibrium of reactive oxygen species and antioxidants in the testicular microenvironment, potentially compromising sperm viability and function. Concurrently, nicotine's capacity to induce DNA damage poses a significant concern, as DNA integrity is paramount for the transmission of genetic information to offspring. Additionally, nicotine's influence on hormonal regulation, including alterations in testosterone levels and hypothalamic-pituitary-gonadal axis function, could contribute to the observed decline in semen quality.

Despite the strides made in our comprehension of nicotine's implications on male fertility, numerous facets remain enigmatic. The orchestration of factors influencing spermatogenesis, the interactions between nicotine and spermatozoa at molecular levels, and the long-term consequences of nicotine exposure on the reproductive health of

males warrant further exploration. A holistic understanding of these multifaceted associations necessitates more comprehensive studies encompassing diverse populations, rigorous experimental designs, and sophisticated analytical techniques. Such endeavours hold the potential to illuminate the mechanistic underpinnings of nicotine's impact on male reproductive health and to inform strategies aimed at mitigating its adverse effects.

Conclusion

The least number of men who chew tobacco are also able to receive medical care for infertility. Therefore, targeted high-risk populations should be the focus of preventative and cessation programmes. Strategies should be created to draw the public's attention to the potential link between tobacco chewing and the prevalence of male infertility.

Additionally, tobacco chewers who are infertile should be warned about the adverse consequences tobacco chewing habit may have on the quality of their sperm.

References

1. Lando HA, Haddock CK, Klesges RC, Talcott GW, Jensen J. Smoke- less tobacco use in a population of young adults. Addict Behav. 1999; 24:431–7.
2. Glover ED, Laflin M, Flannery D, Albritton DL. Smokeless tobacco use among American college students. J Am Coll Health. 1989; 38:81–5.
3. Adam AL. Trends and patterns of tobacco use in the United States. Am J Med Sci. 2022; 123:248 –54.
4. Howard-Pitney B, Winkleby MA. Chewing tobacco: who uses and who quits? Findings from NHANES III, 1988 –1994. National Health and Nutrition Examination Survey III. Am J Public Health. 2002; 92:250 – 6.
5. Ernster VL, Grady DG, Greene JC, Walsh M, Robertson P, Daniels TE, et al. Smokeless tobacco use and health effects among baseball players. JAMA. 1990; 264:218 –24.
6. Nair U, Bartsch H, Nair J. Alert for an epidemic of oral cancer due to use of the betel quid substitute's gutkha and pan masala: a review of agents and causative mechanisms. Mutagenesis. 2004; 19:251– 62.
7. Subramanian SV, Nandy S, Kelly M, Gordon D, Davey Smith G. Patterns and distribution of tobacco consumption in India: cross sectional multilevel evidence from the 1998 –9 national family health survey. BMJ. 2004; 328:801– 6.
8. Bates C, Fagerstrom K, Jarvis MJ, Kunze M, McNeill A, Ramstrom L. European Union policy on smokeless tobacco: a statement in favour of evidence based regulation for public health. Tob Control. 2003; 12:360 –7.
9. Rodu B, Cole P. Smokeless tobacco use and cancer of the upper respiratory tract. Oral Surg Oral Med Oral Pathol Oral RadiolEndod2002; 93:511–5.
10. Asplund K. Smokeless tobacco and cardiovascular disease. Prog Cardiovasc Dis. 2003; 45:383–94.
11. Thomas G, Hashibe M, Jacob BJ, Ramadas K, Mathew B, Sankaranarayanan R, et al. Risk factors for multiple oral premalignant lesions. Int J Cancer 2003; 107:285–91.
12. Hoffmann D, Djordjevic MV. Chemical composition and carcinogenicity of smokeless tobacco. Adv Dent Res. 1997; 11:322–9.
13. Sundqvist K, Grafstrom RC. Effects of areca nut on growth, differentiation and formation of DNA damage in cultured human buccal epithelial cells. Int J Cancer. 1992; 52:305–10.
14. Kunzle R, Mueller MD, Hanggi W, Birkhauser MH, Drescher H, Bersinger NA. Semen quality of male smokers and nonsmokers in infertile couples. Fertil Steril. 2003; 79:287–91.
15. Saleh RA, Agarwal A, Sharma RK, Nelson DR, Thomas AJ Jr. Effect of cigarette smoking on levels of seminal oxidative stress in infertile men: a prospective study. Fertil Steril. 2002; 78:491–9.
16. Stillman RJ, Rosenberg MJ, Sachs BP. Smoking and reproduction. Fertil Steril. 1986; 46:545– 66.
17. Vine MF, Tse CK, Hu P, Truong KY. Cigarette smoking and semen quality. Fertil Steril. 1996; 65:835– 42.
18. Close CE, Roberts PL, Berger RE. Cigarettes, alcohol and marijuana are related to pyospermia in infertile men. J Urol1990; 144:900 –3.
19. Sofikitis N, Miyagawa I, Dimitriadis D, Zavos P, Sikka S, Hellstrom W. Effects of smoking on testicular function, semen quality and sperm fertilizing capacity. J Urol.1995; 154:1030 – 4.
20. Dikshit RK, Buch JG, Mansuri SM. Effect of tobacco consumption on semen quality of a population of hypofertile males. Fertil Steril. 1987; 48:334 – 6.
21. Vogt HJ, Heller WD, Borelli S. Sperm quality of healthy smokers, ex-smokers, and never-smokers. FertilSteril1986; 45:106 –10.
22. Lewin A, Gonen O, Orvieto R, Schenker JG. Effect of smoking on concentration, motility and zona-free hamster test on human sperm. Arch Androl.1991; 27:51– 4.
23. World Health Organization. Laboratory manual for the examination of human semen and sperm-cervical mucus interaction. 4th ed. Cambridge/New York: Cambridge University Press, 1999.