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THE EFFECT OF CIGARETTE SMOKING ON HUMAN SEMEN BOUNDARIES AND HORMONES

Shubham

Dyal Singh College , Karnal (132001) , Haryana
Mobile no.-8708075152
E-mail:- bhattishubham25@gmail.com

Abstract

Foundation: In this planned examination, semen boundaries and hormone centralizations of fruitless smokers were contrasted and barren non-and ex-smokers. We likewise decided the number of men with idiopathic barrenness would quit smoking trying to improve their ripeness. Techniques: 1104 men (517 non-smokers, 109 ex-smokers and 478 smokers) with fruitlessness for in any event 1 year were assessed. Assessment included clinical history, physical assessment, hormone investigation and two semen examinations. Preceding the second semen investigation, smokers were asked to stop smoking. RESULTS: Smokers were fundamentally more youthful (P < 0.001), had essentially more round cells in their discharges (P = 0.003), and the level of discharges with >1×106/ml leukocytes was higher in smokers (P < 0.001). Expanded free and absolute serum testosterone (P < 0.001) and diminished prolactin levels (P < 0.001) were found in smokers. No distinctions were found between non-smokers and ex-smokers. Just 23.1% of the smokers versus 46% non-smokers (P < 0.001) returned for a second semen examination, 14 of whom diminished and 15 of whom quit smoking totally. Testosterone levels were fundamentally lower in the individuals who had the option to stop or diminish smoking (P < 0.001). Ends: Smoking doesn't influence traditional semen boundaries, yet altogether increments round cells and leukocytes. Just a couple of idiopathic barren smokers had the option to stop smoking.

INTRODUCTION

In spite of overall enemy of smoking efforts, cigarette smoking is normal. The most noteworthy pervasiveness of smoking is seen in youthful grown-up guys during their regenerative period (46% smokers somewhere in the range of 20 and 39 years) (Langgassner, 1999).

About 30% of the Austrian male populace matured 15 and more seasoned are smokers. Smoking among men is expanding in Focal and Eastern Europe. Generally 35% of European men smoke, with a pervasiveness of 44% or considerably higher in the Eastern parts (Bulgaria, Greece, Turkey) and 30% in the Western parts (UK, Sweden, Finland) of Europe (Corrao et al., 2000).

Cigarette smoking might be related with sub-fruitfulness in guys and may bring about diminished sperm focus, lower sperm motility, and a decreased level of morphologically ordinary sperm separately (Lewin et al., 1991; Sofikitis et al., 1995; Zinaman et al., 2000).

Nineteen investigations assessing the impact of smoking on semen boundaries in barren men and nine examinations in rich men have been distributed up until now (Plant, 1996; Zinaman et al., 2000). The significant inadequacy of these investigations is a little by and large patient number (just two examinations included >500 men, and >200 smokers) (Dikshit et al., 1987; Lewin et al., 1991).

In an ongoing meta-examination (Plant, 1996), remembering 27 investigations for the relationship between cigarette smoking and semen quality, a mean decrease in sperm convergence of 13%, a mean decrease of sperm motility of 10%, and a mean decrease of morphologically typical sperm of 3% was accounted for in smokers. The greater part of the investigations, nonetheless, which announced a noteworthy distinction in semen quality were acted in ordinary, non-fruitlessness center men. Lamentably, in 25 out of 27 investigations in this meta-examination, the quantity of smokers was <200 men. Another significant weakness is the absence of precise smoking portion data.

Smoking may cause sub-richness by impacting hormone levels (Vogt et al., 1986). Testosterone levels might be unaltered, raised, or diminished and estradiol levels are fundamentally discovered to be raised in smokers (Plant, 1996).

Smoking may affect on richness, as detailed in an ongoing report enlisting 200 men (Zinaman et al., 2000). In this investigation it was noticed that cigarette smoking was fundamentally connected with a diminished pregnancy rate and impeded semen boundaries. Men with azoospermia were rejected and the creators didn't report men with genital sickness. In this examination just 6% (n = 12) were smokers. Despite the fact that there were just six smokers in both the pregnant and the non-pregnant gathering, a factual hugeness (P = 0.02) was determined.

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So as to defeat the inadequacies distinguished in different examinations (for example low member number, and absence of smoking portion information), we looked at semen boundaries and hormone levels of an enormous number of barren smokers with non-smokers and ex-smokers and assessed the smoking portion.

It was as of late closed (Plant, 1996), that men with negligible semen quality who wish to have kids may profit by halting smoking. Moreover, there are just restricted information on whether men would quit smoking for the possibility of recuperating from fruitlessness (Pusch et al., 1989). Along these lines, we decided the number of men would quit smoking on the off chance that they figured it would expand their fruitfulness.

MATERIALS AND METHODS

Patients

This imminent investigation was directed between January 1993 and September 2000 in the fruitlessness unit of the Branch of Urology, College of Graz. Men with a background marked by fruitlessness for at any rate 1 year, who had the option to give a discharge, were sequentially assessed. Work-up for barrenness incorporated a clinical history, physical assessment, just as the evaluation of hormone and semen boundaries.

Clinical history and especially any history of past genital illness was evaluated utilizing a poll including the quantity of cigarettes every day and the length of smoking just as the smoking status of the female accomplice.

Men who had quit smoking ≥6 months before the assessment for barrenness were named ex-smokers and men who had never smoked as non-smokers. Each man who had smoked cigarettes for >6 months was all the while smoking was delegated a smoker. Smokers were sorted as gentle (≤10 cigarettes every day), moderate (>10 and ≤20 cigarettes every day) and hefty smokers (>20 cigarettes every day).

Physical assessment was performed by a uro-andrologist. All men were inspected first in an upstanding situation to preclude the presence of a varicocele. A varicocele was analyzed by touching the spermatic line during the Valsalva move. Venous reflux was affirmed by Doppler ultrasound. Any anomaly of the testicles, epididymis or ductus deferens was recorded. The testicular size was estimated with an orchidometer (Connection, Hamburg, Germany) and considered typical if the volume was >14 ml.

Ultrasound of both testicles was performed utilizing a direct 7.5 MHz ultrasound test (Sono Layer-J, Toshiba, Tokyo, Japan or SSD-1700, Aloka, Tokyo, Japan).

Hormone investigation

Sexual hormone investigation included estimation of LH [immunoradiometricassay (IRMA); ICN, High Wycombe, Bucks, UK], FSH (IRMA; ICN), testosterone (radioimmunoassay; Immunotec, Marseille, France) and prolactin (Cobas Core® chemical immunoassay; Hoffmann–La Roche, Basel, Switzerland) in all men, and in 433 men free testosterone (radioimmunoassay; DPC, Los Angeles, CA, USA), and estradiol (E2, radioimmunoassay; Immunotec) were estimated moreover. All blood tests were drawn somewhere in the range of 08.00 and 10.00.

Semen examination

Semen tests were gathered by masturbation in a perfect example holder after a sexual restraint for 3–6 days, permitted to condense and assessed quickly from that point as indicated by WHO rules (World Wellbeing Association, 1992). Discharge volume, fructose, pH, and time to liquefaction were estimated. Sperm fixation and the centralization of round cells were resolved utilizing a haemocytometer twice for each example (Thoma; Assistent Sondheim/Rhoen, Germany).

Within the sight of >106 round cells/ml these cells were additionally separated utilizing histo-substance recoloring to identify peroxidase positive cells (Endtz, 1972). The level of peroxidase positive round cells recoloring earthy colored was dictated by checking ≥100 round cells under the magnifying instrument (Axiolab; Carl Zeiss GmbH, Oberkochen, Germany) at 10×40 amplification. From that point the centralization of peroxidase positive cells was determined by duplicating the level of peroxidase positive cells by the all out convergence of round cells.

Morphology and motility assessment

For assessment of sperm morphology, prestained slides (two for every semen test), which are typically utilized for platelet separation, were spread with a little volume of semen and permitted to air dry (Testsimplets®; Roche Diagnostics, Mannheim, Germany). Sperm morphology was resolved utilizing the WHO measures (World Wellbeing Association, 1992). Other than the level of morphologically irregular sperm, the sperm head, neck and mid-piece, tail abandons, just as the presence of cytoplasmic beads were evaluated. Numerous imperfections per spermatozoon were noted, if present, by methods for a research facility cell counter (Earth Adams, Inc., New York, NY, USA) The complete number of deformities was tallied and the teratozoospermic list was determined (all out number of imperfections/number of sperm with deserts).

Motility was controlled by assessing 200 sperm for each example, 60 min after semen assortment. Motility was reviewed as 'a', 'b' or 'c and d' as indicated by the WHO measures (World Wellbeing Association, 1992).

The consequences of semen examinations were arranged by the classification of semen factors (World Wellbeing Association, 1992). Normozoospermia was analyzed when sperm focus, motility and morphology were inside the reference esteems. The reference an incentive for 'sperm focus' was ≥20×106 sperm/ml, for 'motility' ≥50% sperm with forward movement (classifications 'an' and 'b') or ≥25% sperm with classification

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'a' development, and for 'morphology' ≥30% sperm with ordinary morphology individually. Oligozoospermia was resolved when sperm focus was not exactly the reference esteem. Moreover, asthenozoospermia was analyzed when motility, and teratozoospermia when morphology, were underneath the reference esteems. An oligoasthenoteratozoospermia was analyzed when every one of the three factors (fixation, motility, morphology) were upset. Mixes (oligoasthenozoospermia, oligoteratozoospermia and asthenoteratozoospermia) were utilized when two factors were upset. Azoospermia was analyzed when, even in the residue after centrifugation at >3000 g for 15 min, no sperm were recognized.

Following assessment, all chose factors were gone into a mechanized information the board framework (FileMaker; FileMaker, Inc., Santa Clause Clara, CA, USA).

After the essential assessment for fruitlessness, all men without a background marked by, or flow, genital illness just as men who had neither azoospermia nor extreme oligozoospermia were welcomed for a second semen examination 3 months after the fact. The motivation to prohibit these men is that men with non-idiopathic barrenness got causative treatment as quickly as time permits, and men with a high evaluation oligozoospermia or azoospermia were offered helped conceptive methods, if proper.

Extreme oligozoospermia was analyzed when the sperm fixation was <5×106/ml and included men who had oligozoospermia alone or in blend with asthenozoospermia and additionally teratozoospermia.

Smokers with no history of, or momentum, genital illness and smokers who had neither azoospermia nor extreme oligozoospermia were educated about the conceivable antagonistic impacts of cigarette smoking on semen boundaries or potentially fruitfulness and that no other explanation for barrenness could be recognized. They were encouraged to quit smoking promptly so as to improve the semen quality and were encouraged to return for a semen examination 3 months in the wake of having quit smoking. Notwithstanding verbal advising, composed data was sent to each member of the examination.

The rest of the smokers with either genital infection or a sperm grouping of <5×106/ml were encouraged to quit smoking too.

INSIGHTS

Men were assembled into smokers, ex-smokers and non-smokers. A spellbinding examination of the information was performed and the factors were additionally investigated with a t-test and investigation of change (ANOVA), or with the Wilcoxon–Mann–Whitney test and the Kruskal–Wallis test (FSH, LH, testosterone, free testosterone, E2, prolactin) contingent upon the ordinariness supposition.

Factors were sorted at whatever point conceivable and investigated with cross-tables utilizing $\chi 2$-test or Fisher's definite test (aftereffects of semen examinations, hormones, female smoking status, dispersion of non-smokers, ex-smokers and smokers at development). Numerous examinations of non-smokers with gentle, moderate and weighty smokers were finished utilizing ANOVA followed by Dunnett's test (Dunnett, 1955). The correlation of the first and the second semen investigation was finished with a matched t-test. Measurable investigation was performed by a bio-analyst utilizing SPSS factual programming (SPSS Inc., Chicago, IL, USA).

RESULTS

Taking all things together, 1104 men were assessed for barrenness. Of these, 517 were non-smokers, 478 smokers and 109 ex-smokers separately. None was avoided for including began smoking inside the past a half year.

No critical contrasts in the aftereffects of semen examinations were seen between non-smokers and smokers, though with ex-smokers azoospermia was watched essentially less regularly (P = 0.035). The consequences of semen investigations are given in Table I.

An aggregate of 426 (38.6%) men gave a background marked by, or momentum, genital malady, the sort and level of which are recorded in Table II. The appropriation of genital infection among non-smokers and smokers was the equivalent in the two gatherings; interestingly, ex-smokers had essentially less genital malady (P = 0.0056, Table III).

Serious oligozoospermia (sperm fixation <5×106/ml) and azoospermia were analyzed in 232 men (21%) and were found in 104 (20.1%) non-smokers, 23 (21.1%) ex-smokers and 105 (22%) smokers separately.

Serious oligozoospermia and azoospermia in blend with a genital illness were more normal in smokers than in non-smokers and ex-smokers (15.5, 13.2 and 11% separately). In any case, the contrast among smokers and non-smokers was not factually noteworthy (P = 0.241).

Mean age, weight file (BMI), just as semen and hormone boundaries for non-smokers, smokers and ex-smokers are appeared in Table IV.

Contrasted and non-and ex-smokers, smokers were altogether more youthful (P < 0.01), had fundamentally more round cells in their discharges (P = 0.012), higher LH (P = 0.035), higher testosterone (P < 0.001), free testosterone (P = 0.001) and had lower prolactin levels (P < 0.001). The level of discharges with >1×106/ml

peroxidase positive round cells was additionally essentially higher in smokers than non-or ex-smokers (11.8 versus 13.1 versus 23.5% individually; P < 0.001).
Conversely, ex-smokers had an essentially higher BMI (P = 0.031) contrasted and non-smokers and smokers.
Out of 478 smokers, 124 were named gentle, 244 as moderate and 110 as weighty smokers. Ordering smokers as mellow, moderate and hefty, just BMI (24.9, 25.5, 26.1 kg/m2; P = 0.05), the mean number of cigarettes every day (6.5, 18.5, 32.4; P < 0.001) and the term of smoking (10.5, 12, 13.9 years; P < 0.001) were essentially extraordinary between gentle, moderate, and substantial smokers.
A sum of 350 (31.7%) female accomplices smoked. Out of 478 male smokers, 239 (half) had female accomplices who were additionally smokers, while just 91 (17.6%) non-smokers and 20 (22.5%) ex-smokers revealed that their female accomplices were smokers (P < 0.001).
Men with any history of, or flow, genital ailment, azoospermia or extreme oligozoospermia were additionally prohibited. Men who denied their endorsement to return for assessment following 3 months (n = 12) were likewise avoided, subsequently leaving 588 qualified men for additional assessment (258 non-smokers, 70 ex-smokers and 260 smokers). Of these, 211 men (36%) returned for a subsequent semen investigation.
Fundamentally more non-smokers (n = 119, 46.1%) and ex-smokers (n = 32, 45.7%) returned for the second semen examination. Interestingly, just 60 (23.1%) smokers returned for a subsequent semen investigation (P < 0.001), 14 of whom had diminished smoking and 15 had totally halted. Out of 29 smokers, who had either diminished or quit smoking, just six (20.7%) men had a smoking accomplice.
The mean testosterone levels of men who had either halted or diminished smoking (4.3 ng/ml) were altogether lower contrasted and smokers who didn't quit smoking or didn't return for additional assessment (5.0 ng/ml) (P < 0.001).
Non-smokers and ex-smokers returned for a second semen examination after an interim of 14 weeks. Mean chance to second semen investigation for men who had halted, diminished or kept on smoking was 18, 22 and 43 weeks separately (P = 0.148). No noteworthy contrasts between the first and second semen examination were taken note.

DISCUSSION

The level of smokers in our investigation of barren men was 43.3% and was in this way not the same as the Austrian male populace somewhere in the range of 18 and 50 years, which was accounted for to be 44.2% during the examination time frame (Langgassner, 1999). Guys matured 20–24 years have the most elevated pace of smoking (47.9%) (Langgassner, 1999). This is as per our discovering in light of the fact that smokers looking for work-up for fruitlessness were altogether more youthful than non-and ex-smokers.
In an ongoing meta-investigation (Plant, 1996) of 27 examinations tending to the relationship between cigarette smoking and semen quality, it was noticed that the majority of the examinations report a noteworthy distinction in semen quality were acted in typical, non-fruitlessness center men. Seven out of nine investigations in rich and just six out of 19 examinations in barren men revealed a measurably noteworthy contrast in semen quality. The biggest examination in this meta-investigation (Lewin et al., 1991) included 662 fruitless men (382 non-smokers, 280 smokers) and announced a factually noteworthy distinction in sperm fixation (55 versus 46.9×106/ml). In any case, in our enormous examination on 1104 fruitless men, including 478 smokers, no distinctions regarding ordinary semen boundaries (sperm focus, motility and morphology) between non-smokers and smokers were watched.
A potential inclusion of round cells and leukocytes, which were fundamentally raised in our examination in smokers contrasted and non-smokers, was likewise revealed in a past examination (Close et al., 1990). The creators watched a pattern (P = 0.12) towards higher leukocyte numbers in a little report assessing the discharges of 22 fruitless smokers. Essentially raised leukocytes have likewise been accounted for in the fringe blood of smokers (Repel et al., 1997). Cigarette smoking appears to initiate bone marrow, and it is hypothesized that blood leukocytosis adds to the incessant lung irritation related with cigarette smoking (van Eeden and Hogg, 2000). The component, nonetheless, which enacts leukocytes in the semen of smokers, is indistinct.
Leukocytes are the significant wellspring of receptive oxygen species (ROS) in the discharge (Sharma and Agarwal, 1996). Raised leukocytes may hinder fruitfulness by development of ROS (Ochsendorf, 1999). ROS are hurtful to sperm DNA (Shen et al., 1999) and layer phospholipids (Kim and Parthasarathy, 1998) as a result of oxidation. The impacts of unreasonable oxidation on sperm work have been recommended as impeding. The part of ROS, notwithstanding, and whether ROS focuses were raised in the semen of smokers, has not been concentrated at this point.
Wolff found that 20% of men with raised leukocytes in their discharge had genital plot contaminations (positive societies) (Wolff, 1995).
The way that ex-smokers had essentially less genital ailment and an equivalent level of men with a sperm tally of <5×106/ml, recommends that ex-smokers may include an uncommon gathering of barren men. The

assessment of potential impacts of smoking, in any case, on sperm focus by contrasting smokers with ex-smokers ought to be done carefully, if by any means.

It was recently detailed that smoking is a co-factor along with genital infection, for example, varicocele, and can impede human semen quality (Klaiber et al., 1987). In this little investigation, smokers with a varicocele had an excessively high frequency of oligozoospermia. In our investigation this perception couldn't be affirmed. In spite of the fact that we found a higher level of smokers with genital infection and a sperm centralization of <5×106/ml, factual essentialness was not accomplished.

Diminished prolactin levels have as of late been accounted for in female smokers (Weigert et al., 1999), like our discoveries in male smokers. In an examination utilizing the GH3 rodent pituitary cell line, it was demonstrated that nicotine can down-manage prolactin quality articulation (Coleman and Bancroft, 1995). This may clarify why prolactin is fundamentally diminished in smokers. In rams it was noticed that hypo-prolactinaemia may influence LH discharge and impact testicular capacity by straightforwardly influencing testosterone and semen creation (Regisford and Katz, 1993). In any case, rams are occasional reproducers and responded diversely during hypo-prolactinaemic periods in spring and harvest time. The effect of diminished prolactin levels on human semen quality, in this manner, stays muddled.

Fundamentally expanded, diminished, and unaltered degrees of testosterone were accounted for in past examinations (Plant, 1996). In our gathering of smokers, testosterone levels were altogether expanded, which is in accordance with the bigger investigations (Vogt et al., 1986; Field et al., 1994). The essentially raised LH in smokers proposes a focal actuation of Leydig cells, which clarifies raised testosterone and free testosterone levels. No portion reliance of cigarettes smoked and span of smoking on testosterone levels was found in our examination. A potential clarification is that smoking may, after some time, lead to a degeneration of Leydig cells, which thus diminishes testosterone creation. This theory is upheld by an ongoing report on rodents that were presented to tobacco smoke and demonstrated diminished testosterone levels (Yardimci et al., 1997). The histological assessment of the rodent testicles in this investigation indicated less and deteriorated Leydig cells.

The inquiry, be that as it may, of in the case of smoking expands LH, testosterone and free testosterone without anyone else or whether men with raised hormone levels are more inclined to getting dependent on cigarette smoking stays muddled. Men with higher testosterone levels are supposedly more regularly occupied with wellbeing hazard conduct than men with lower levels (Stall et al., 1999). In our investigation, men who had the option to lessen or quit smoking, nonetheless, had altogether lower testosterone levels after entering the examination analyzed wiht the entire gathering of smokers. This may uphold the speculations that a high testosterone level improves a wellbeing hazard conduct, for example, smoking, and then again may make it simpler for those with lower testosterone levels to shun smoking.

Tobacco smoke is a cell mutagen and cancer-causing agent and may antagonistically influence fruitfulness. Each smoker ought to be urged to quit smoking, particularly if a pregnancy is arranged. Tobacco smoke contains a great deal of known poisons, which may effectsly affect richness in both genders. Basically halting smoking, in any case, could forestall the poisons contained in tobacco smoke.

So as to decide the level of men ready to diminish or quit smoking for the possibility of improved ripeness, just men with idiopathic barrenness and a sperm convergence of >5×106/ml were surveyed in our examination. The purpose behind this was men with non-idiopathic barrenness got causative treatment, and men with a high-grade oligozoospermia or azoospermia were offered helped conceptive methods, if suitable.

Altogether more non-smokers than smokers returned for a second semen examination. As a potential clarification, we speculate that solitary a couple of smokers quit smoking, and that a large number of the individuals who didn't stop decided not to go to a subsequent semen investigation. In this specific circumstance, the female smoking status appeared to assume a significant job, on the grounds that >79% of the subjects who had either decreased or quit smoking had non-smoking accomplices. The quantity of smokers who quit smoking, notwithstanding, was shockingly low in our investigation. This is as opposed to a formerly announced high acknowledgment of fruitless smokers to quit smoking (Pusch et al., 1989). In this investigation, 63% chose to quit smoking, yet the quantity of smokers isn't accounted for.

Altogether more smokers had accomplices who smoked as well. Smoking in female accomplices has expanded during the most recent decade (Haidinger et al., 1998), which may have a considerably more prominent impact on the ripeness of a couple than male smoking (Bolumar et al., 1996; Augood et al., 1998). Practically 80% of men who had the option to lessen or quit smoking had a non-smoking accomplice.

In a past examination, sperm motility and morphology improved following a half year of follow-up in nine men who quit smoking (Sofikitis et al., 1995). The aftereffects of semen examinations in the wake of pulling back or lessening smoking are not announced in detail in our investigation. They are of restricted importance and should be deciphered with alert in view of the modest number of men in these gatherings and the short development. Further examinations are expected to research the drawn out impacts of pulling back from smoking on customary semen boundaries, round cells and leukocytes.

All in all, in our enormous investigation with a sum of 1104 fruitless men including 571 non-smokers, 109 ex-smokers and 478 smokers, no noteworthy contrasts in regular discharge boundaries (sperm fixation, morphology and motility) between non-smokers, ex-smokers and smokers were watched, in spite of the fact

that azoospermia was more predominant among ex-smokers than the other two gatherings. Round cells and leukocytes were altogether expanded in the discharges of smokers contrasted and non-and ex-smokers. Since leukocytes produce ROS, this may add to fruitlessness in smokers.

We additionally watched raised serum levels of testosterone, free testosterone, LH and diminished prolactin levels in smokers, yet the mechanism(s) of these changes, assuming any, remaining parts indistinct.

At last, just a couple of idiopathic barren smokers had the option to stop smoking.

Table I.

Results of semen analyses

	Total	**Non-smokers**	**Smokers**	**Ex-smokers**
	***n* = 1104**	***n* = 517 (46.8)**	***n* = 478 (43.3)**	***n* = 109 (9.9)**
Values in parentheses are percentages.				
**P* = 0.035.				
Normozoospermia	404 (36.6)	184 (35.6)	183 (38.3)	37 (33.9)
Asthenozoospermia	206 (18.7)	94 (18.2)	90 (18.8)	22 (20.2)
Oligozoospermia	113 (10.2)	50 (9.7)	51 (10.7)	12 (11.0)
Teratozoospermia	29 (2.6)	15 (2.9)	10 (2.1)	4 (3.7)
Asthenoteratozoospermia	69 (6.3)	34 (6.6)	27 (5.6)	8 (7.3)
Oligoasthenozoospermia	68 (6.2)	32 (6.2)	26 (5.4)	10 (9.2)
Oligoteratozoospermia	20 (1.8)	10 (1.9)	9 (1.9)	1 (0.9)
Oligoasthenoteratozoospermia	95 (8.6)	47 (9.1)	37 (7.7)	11 (10.1)
Azoospermia	100 (9.1)	51 (9.9)	45 (9.4)	4 (3.7)*

Table II.

Number and percentage of men with genital disorders (*n* = 426)

Genital disease	**No.**	**% of total (1104)**
Varicocele	199	18.0
Pathology of the epididymis and	71	6.4

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Genital disease	No.	% of total (1104)
ductus		
deferens, history of genital infection		
Hypogonadism (testis volume <14 ml)	58	5.3
History of orchidopexy	44	4.0
Cryptorchidism	8	0.7
Testicular cancer (presently found)	5	0.5
History of testicular cancer	20	1.8
Snow-storm in testis ultrasound	13	1.2
(testicular microlithiasis)		
Others	8	0.7

Table III.
Number and percentage of non-smokers, smokers and ex-smokers with or without genital disease

	Genital disease		
	No (%)	**Yes (%)**	**Total**
*P = 0.0056.			
Non-smokers	304 (58.8)	213 (41.2)	517
Smokers	292 (61.1)	186 (38.9)	478
Ex-smokers	82 (75.2)	27 (24.8)*	109
Total	678	426	1104

Table IV.
Mean age, body mass index and the results of the semen and hormone analyses of non-smokers, smokers and ex-smokers

	Non-smokers	**Smokers**	**Ex-smokers**	***P***
	(*n* = 517)	**(*n* = 478)**	**(*n* = 109)**	

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	Non-smokers	Smokers	Ex-smokers	*P*
	(*n* = 517)	(*n* = 478)	(*n* = 109)	
Values are means (SD).				
[a]Significantly different from non-smokers.				
[b]Significantly different from ex-smokers.				
[c]Significantly different from smokers.				
[d]Teratozoospermic index = total number of defects/number of sperm with defects.				
Age	33.4 (6.4)	31.5 (5.1)[a,b]	33.7 (5.7)	<0.001
Body mass index (kg/m^2)	25.8 (3.5)	25.5 (3.3)	26.5 (3.6)[a,c]	0.031
Volume (ml)	3.6 (1.8)	3.7 (1.8)	3.7 (1.7)	0.803
Liquefaction time (min)	38.9 (36.1)	39.4 (34.4)	38.6 (35.1)	0.874
Fructose (µg/ml)	2758 (1379)	2638 (1242)	2918 (1301)	0.191
Morphologically abnormal (%)	57.8 (17.6)	56.1 (17.9)	57.6 (17.6)	0.207
Head defects (%)	39.1 (16.5)	39.2 (15.7)	38.5 (15.2)	0.924
Mid-piece defects (%)	11.3 (6.5)	11.2 (7.1)	12.3 (7.0)	0.401
Tail defects (%)	14.3 (12.0)	14.2 (9.5)	14.1 (9.2)	0.213
Cytoplasmic droplets (%)	7.5 (4.4)	7.7 (4.3)	7.6 (4.5)	0.800
Teratozoospermic index[d]	1.47 (0.16)	1.48 (0.16)	1.50 (0.17)	0.338
Sperm concentration (×10^6/ml)	57.9 (70.8)	58.8 (63.9)	59.1 (67.2)	0.730

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	Non-smokers	Smokers	Ex-smokers	*P*
	(*n* = 517)	**(*n* = 478)**	**(*n* = 109)**	
Grade `a' motility (%)	24.4 (9.4)	24.8 (8.6)	23.4 (10.7)	0.569
Grade `b' motility (%)	19.2 (5.1)	19.9 (4.6)	18.4 (4.8)	0.12
Round cells ($\times 10^6$/ml)	2.7 (3.38)	3.5 (5.3)[a,b]	2.6 (3.4)	0.012
FSH (mIE/ml)	7.1 (7.9)	7.5 (9.8)	7.4 (6.3)	0.122
LH (mIE/ml)	4.4 (2.8)	5.1 (3.9)[a,b]	4.8 (3.1)	0.035
Testosterone (ng/ml)	4.2 (2.1)	5.0 (2.7)[a,b]	4.4 (2.5)	< 0.001
Free testosterone (pg/ml)	15.2 (5.3)	17.6 (5.6)[a,b]	14.9 (4.1)	< 0.001
Estradiol (pg/ml)	27.4 (42.8)	26.2 (13.2)	26.0 (11.0)	0.335
Prolactin (ng/ml)	13.1 (7.7)	11.2 (4.9)[a,b]	12.1 (5.5)	< 0.001

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