Coll. Antropol. **29** (2005) 2: 567–572
UDC 616.711-007.5-053.9
Original scientific paper

Effects of Smoking and Alcohol Consumption on Vertebral Deformity in the Elderly – An Epidemiological Study

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A B S T R A C T

The aim of the study was to assess the role of smoking and alcohol consumption as possible risk factors for vertebral deformities in an elderly Croatian population sample. Data on smoking habit, alcohol consumption, body mass index, and overall life activity were collected in 425 randomly chosen community dwelling subjects. Radiographic morphometric method was used to assess vertebral deformities of thoracic and lumbar spine. Men smoked and drank significantly more than women. There was no association of either smoking status or number of cigarettes, or frequency of alcohol intake with prevalent vertebral deformities. There was a tendency of an increased risk of vertebral deformities in heavy drinkers (OR=1.69; 95% CI=0.98–2.91), and a reduced risk of these deformities in female regular drinkers (OR=0.72; 95% CI=0.14–3.66). Further studies in the Croatian population are needed to establish the association of smoking and alcohol consumption with vertebral deformities.

Key words: *alcohol, epidemiology, fractures, osteoporosis, smoking, spine*

Introduction

Osteoporosis is the most common metabolic bone disease. There are many genetic and environmental risk factors for osteoporosis, of which those causing fractures as most severe osteoporosis sequels are of particular interest[1]. A positive association between smoking and rate of non-vertebral fractures was found in most but not all studies[2,3], whereas long term, heavy alcohol consumption substantially increased the risk of hip fracture[4]. Data on vertebral fractures as one of the hallmarks of osteoporosis are scarce[5,6]. Two main reasons for these considerations are the lack of a universally accepted definition of vertebral fractures and the fact that a substantial proportion of these fractures escape clinical diagnosis[1]. It has therefore become conventional to use the term vertebral deformity, reserving the term fracture for clinically apparent deformities.

The aim of the study was to assess the effect of smoking and alcohol consumption as the possible risk factors for vertebral deformities in an elderly Croatian population sample, using data from the population-based epidemiological study. As due to genetic and environmental factors, each population has its own peculiarities, this would enable us to compare our results with similar results obtained elsewhere.

Subjects and Methods

Subjects

Community-dwelling subjects aged 50 and above were recruited for a random sample. Every fourth patient of that age from the register of two outpatient clinics in Zagreb (Croatia) was invited by a letter of invitation for an interview and lateral thoracolumbar radiography. Non-responders were sent a repeat letter of invitation or called by phone. Out of 600 subjects invited to participate in the study, there were 365 (60.1%) first time responders, and 60 more subjects were recruited upon the second call. Thus, the overall response rate was 70.1% (425/600). For practical and ethical reasons it was not possible to conduct a radiographic survey of non-responders. Instead, 40 non-responders answered a short-

Received for publication September 24, 2004

TABLE 1
AGE AND SEX DISTRIBUTION OF STUDY SUBJECTS (N=425)

Age (yrs)	Men		Women		Men + Women	
	N	%	N	%	N	%
50–54	40	25.6	53	19.7	93	21.9
55–59	39	25.0	55	20.4	94	22.1
60–64	28	17.9	60	22.3	88	20.7
65–69	23	14.7	56	20.8	79	18.6
70–74	22	14.1	28	10.4	50	11.8
≥75	4	2.6	17	6.3	21	4.9
Total	156	100.0	269	100.0	425	100.0

ened version of the questionnaire by phone. The results of the non-responders survey suggested the differences in demographic data and lifestyle characteristics between those who participated and those who declined participation in the study to be small. These findings argue against a serious non-response bias.

There were 156 (36.7%) men and 269 (63.3%) women. Median age was 61.0 years (range 50–81 years), with no sex difference (p=0.128), (Table 1).

Methods

Responders were interviewed by an investigator experienced in epidemiological research. The information was obtained on whether a subject smoked cigarettes or used other forms of tobacco, was a past smoker, or had never smoked at all. If he/she had ever smoked, the life period (in years) and average number of cigarettes *per* day were also recorded. As for alcohol consumption, the aim of the interview was to determine the frequency of taking any alcoholic beverage in the past year, offering choice among the following options: every day, 5–6 days a week, 3–4 days a week, 1–2 days a week, less than once a week, or not at all. Data were also obtained regarding possible confounding factors than can be associated with smoking and drinking, and could influence the prevalence of vertebral deformities, such as anthropometric data (height in meters, weight in kilograms) and physical activity score (added numbers for level of physical activity as: 1-light, 2-moderate, 3-heavy, or 4-very heavy, in three life periods: age 15–25, 25–50 and >50 years). The questionnaire was phrased to ascertain the most strenuous level of activity carried out daily during each of these three age period. The definitions for each grade were: light (1) – predominantly secretarial, office or similar work; moderate (2) – activities involving standing, walking; heavy (3) – activities involving lifting heavy loads; very heavy (4) – activities involving continuous heavy work such as agricultural or construction works as well as professional sports.

X-ray measurements and definition of vertebral deformity

Lateral thoracic and lumbar spinal radiographs were taken according to a standard protocol. Tube-film distance was 1.2 m, with thoracic films centered at T7 and lumbar films centered at L2. Radiographs were taken with the subject in the left lateral position, and for thoracic films the breathing technique was used (allowing blurring of the overlying ribs and lung detail by motion). The films were evaluated using a translucent digitizer and cursor. Six points were marked in a standardized fashion for each vertebral body from thoracic 4 to lumbar 4, to describe vertebral shape. The co-ordinates were recorded on an electronic grid. From these co-ordinates the anterior (Ha), central (Hc) and posterior (Hp) heights were determined for each vertebral body from T4 to L4.

Reference values for vertebral height ratios were derived using an iterative algorithm. The method was proposed by Black et al.[7], and the improved modification described by Melton et al. was used in this study[8]. It is based on the assumption that those vertebrae that are fractured have a measurement that lies at one extreme of the distribution. For each vertebral height ratio, the iteration begins by removing all observed values more than 1.5 interquartile ranges above the 75th percentile or below the 25th percentile. After removing these observations, the percentiles and interquartile range are recalculated for the remaining sample and the process is repeated until no more observations qualify for removal. The mean and standard deviation (SD) of the trimmed sample are then used as estimates of the mean and SD in the unfractured vertebrae.

For defining vertebral deformity the method proposed by McCloskey et al. was used[9]. According to the method, a predicted posterior height (Hpred) is calculated for each vertebra from posterior heights of up to four adjacent vertebrae. Vertebral deformity is present if any of the following criteria is met: 1) Ha/Hp decreased and Ha/Hpred <3 SD below the reference mean; 2) Hm/Hp decreased and Hm/Hpred <3 SD below the reference mean; or 3) Ha/Hpred decreased and Hp/Hpred <3 SD below the reference mean.

Statistics

The prevalence of deformity was calculated based on the number of individuals with at least one vertebral deformity. Data were analyzed for either sex in separate.

Apart from descriptive statistics, *t*-test for independent samples, Mann-Whitney U test, χ^2-test and logistic regression were used in statistical analysis of the results. The analysis was done by use of the SPSS version 10.0 for Windows and STATA 3.1 computer programs.

Results

Prevalence of vertebral deformities

X-ray analysis was available for all 425 subjects. Vertebral deformities were found in 50 (11.8%) subjects, 24 (15.4%) men and 26 (9.7%) women. χ^2-test yielded no sex difference in the prevalence of vertebral deformities (χ^2=3.111, p=0.078). The prevalence of vertebral deformity slightly rose with age (Mann-Whitney U=6995.0, p=0.019). Considering sex distribution, logistic regression confirmed a small effect of age on the prevalence of vertebral deformities (OR=1.06; 95% CI=1.02–1.10).

Smoking

Distribution of subjects according to smoking status showed that 81 (19.1%) subjects were current smokers, 99 (23.3%) were ex-smokers and 245 (57.6%) never smoked. Respective numbers for men were 42 (27.0%), 62 (39.7%) and 52 (33.3%) and for women 39 (14.6%), 37 (13.8%) and 193 (71.7%), showing statistically significant sex difference with an obvious male preponderance (Mann-Whitney U=13354.0, p=0.000). The same was found when the subjects were divided into groups of ever-smokers and never-smokers (p<0.001). The ever-smokers started smoking at the age of 21.4±7.5, and those who stopped did it at the age of 47.5±11.1. There was no association between smoking status and prevalence of vertebral deformities, either for each sex separately or taken together (Table 2).

When the subjects were divided into groups of ever- and never-smokers, the results on vertebral deformities were similar (Table 3).

Although there was a tendency of some positive association between ever-smokers and vertebral deformities expressed as relative risk, the confidence interval embraced unity.

The number of cigarettes *per* day highly varied (from 2 to 100), and was unevenly distributed. For every subject the number of daily cigarettes was multiplied by years of smoking, thus yielding a score for further analysis. As the resulting numbers showed great dispersion, the subjects were arbitrarily divided into two groups according to median value (25.856). The group of lower consumption included 107 (38 men and 69 women) subjects, and the group of higher consumption 73 (51 men and 22 women) subjects (p<0.001). The distribution of subjects in these two groups showed no statistically significant association between heavier cigarette smoking and prevalence of vertebral fractures (Table 4).

Crude relative risk and relative risk adjusted for confounders of age, body mass index, alcohol consumption (see below), and throughout life activity score for vertebral deformity were calculated for heavier smokers. The relative risk of vertebral deformity was not statistically significant in heavier smokers, even when adjusted for these confounders.

TABLE 2
DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO SMOKING STATUS AND VERTEBRAL DEFORMITIES (N=425)

Smoking	Men		Women		Men + Women	
	Vertebral deformity					
	Yes	No	Yes	No	Yes	No
Yes, now	9	33	3	36	12	69
Ex-smokers	8	54	6	31	14	85
Never	7	45	17	176	24	221
Total	24	132	26	243	50	375

Men – Mann-Whitney = 1395.0, p=0.442, Women – Mann-Whitney = 2998.0, p=0.337, Men + Women – Mann-Whitney = 8323.0, p=0.344

TABLE 3
DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO PAST SMOKING STATUS AND VERTEBRAL DEFORMITIES (N=425)

Vertebral deformity	Past smoking habit					
	Men – N (%)		Women – N (%)		Men + Women – N (%)	
	Yes	No	Yes	No	Yes	No
Yes	17 (16.4)	7 (13.5)	9 (11.8)	17 (8.8)	26 (14.5)	24 (9.8)
No	87 (83.6)	45 (86.5)	67 (88.2)	176 (91.2)	154 (85.5)	221 (90.2)
Total	104 (100.0)	52 (100.0)	76 (100.0)	193 (100.0)	180 (100.0)	245 (100.0)

Men – OR=1.26, 95% CI=0.48–3.2, Women – OR=1.39, 95%CI=0.59–3.27, Men + Women – OR=1.55, 95% CI=0.86–2.81

TABLE 4
DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO SMOKING SCORE (PRODUCT OF MULTIPLICATION OF CIGARETTES SMOKED DAILY AND YEARS OF SMOKING) AND BY VERTEBRAL DEFORMITIES (N=425)

Vertebral deformity	Smoking score								
	Men – N (%)			Women – N (%)			Men + Women – N (%)		
	Group 1	Group 2	Total	Group 1	Group 2	Total	Group 1	Group 2	Total
Yes	9 (23.7)	10 (14.5)	19 (17.8)	6 (11.8)	2 (9.1)	8 (11.0)	15 (16.9)	12 (13.2)	27 (15.0)
No	29 (76.3)	59 (85.5)	88 (82.2)	45 (88.2)	20 (90.9)	65 (89.0)	74 (83.1)	79 (86.8)	153 (85.0)
Total	38 (100.0)	69 (100.0)	107 (100.0)	51 (100.0)	22 (100.0)	73 (100.0)	89 (100.0)	91 (100.0)	180 (100.0)

Group 1 ≤25.856 (number of cigarettes x number of years smoking), Group 2 >25.856 (number of cigarettes x number of years smoking), Men – OR=1.01, 95% CI=0.60–1.72, Women – OR=1.33, 95% CI=0.69–2.57, Men + Women – OR=1.21, 95%CI=0.83–1.77, Adjusted for confounders: Men – OR=0.98, 95% CI=0.59–1.61, Women – OR=1.12, 95% CI=0.60–2.10, Men + Women – OR=1.18, 95 %CI=0.83–1.68

Alcohol

Concerning the frequency of consuming alcoholic beverages in the past year, 141 subjects were not drinking at all, 99 subjects reported alcohol consumption less than once a week, 86 subjects on 1–2 days, 29 on 3–4 days, 14 on 5–6 days a week, and 56 subjects drank every day. So, the majority of subjects were not drinking at all or were moderate drinkers. There was a significant difference between men and women, men being those who were drinking more (Mann-Whitney U=7247.5, p= 0.000).

As there was a pretty clear distinction between heavier drinkers and a category of moderate and no drinkers, the subjects were divided into two groups: group 1 consisting of subjects who were not drinking at all and those who were drinking 1–2 times a week at the most, and group 2 including those who were drinking at least 3–4 times a week. There were 326 (76.7%) subjects in group 1 and 99 (23.3%) subjects in group 2. The difference between men and women was statistically significant (p<0.001). The distribution by vertebral deformity showed no association with the frequency of drinking alcoholic beverages (Table 5).

On calculating relative risk we found a tendency of positive association between heavy drinkers (group 2) and prevalence of vertebral deformities for all subjects, although it did not reach statistical significance.

Adjustment for confounders (age, smoking, body mass index and throughout life activity score) produced some changes; however, no statistically significant association between alcohol consumption and vertebral deformities could be confirmed.

Discussion

Vertebral fracture epidemiology should be considered separately in relation to fractures that become clinically apparent from those that are detected on population screening. Study of the former provides data on the health service burden, whereas investigation of the latter may be more valuable in studying the etiology and identifying the predictors of further fractures. We used the latter approach to assess the effect of smoking and alcohol consumption on vertebral deformities in the elderly.

Our population-based study was designed in a standardized manner in an attempt to reduce the likelihood of the findings being due to methodological artifacts. The study was conducted over a relatively short period of time (within one year), thus any difference in prevalence between subjects due to secular change in the occurrence of deformity is likely to be small.

Although there are some conflicting results, a recent meta-analysis of the effects of cigarette smoking on bone

TABLE 5
DISTRIBUTION OF STUDY SUBJECTS ACCORDING TO FREQUENCY OF ALCOHOL INTAKE AND VERTEBRAL DEFORMITIES (N=425)

Vertebral deformity	Frequency of alcohol intake								
	Men – N (%)			Women – N (%)			Men + Women – N (%)		
	Group 1	Group 2	Total	Group 1	Group 2	Total	Group 1	Group 2	Total
Yes	9 (11.8)	15 (18.7)	24 (15.4)	24 (9.6)	2 (10.6)	26 (9.7)	33 (10.1)	17 (17.2)	50 (11.8)
No	67 (88.2)	65 (81.3)	132 (84.6)	226 (90.4)	17 (89.4)	243 (90.3)	293 (89.9)	82 (82.8)	375 (88.2)
Total	76 (100.0)	80 (100.0)	156 (100.0)	250 (100.0)	19 (100.0)	269 (100.0)	326 (100.0)	99 (100.0)	425 (100.0)

Group 1 – subjects with alcohol intake 1–2 times a week at the most, Group 2 – subjects with alcohol intake at least 3–4 times a week, Men – 1.83, 95 %CI=0.55–6.08, Women – OR=0.72, 95% CI=0.14–3.66, Men + Women – OR=3.77, 95% CI=0.48–2.95, Adjusted for confounders: Men – OR=1.58, 95% CI=0.73–3.40, Women – OR=1.10, 95% CI=0.28–4.29, Men + Women – OR=1.69, 95% CI= 0.98–2.91

mineral density (86 studies with 40,753 subjects) has shown that smokers have a significantly reduced bone mass at all bone sites, especially at hip, compared with non-smokers (never and former smokers)[10].

There are several mechanisms that might explain the adverse effect of smoking on skeleton, which include altered metabolic pathway of estradiol, lower fractional calcium absorption, and reduced calcium absorption efficiency (probably due to suppression of the PTH-calcitriol axis), altered osteoblast activity, and interference with the ability of bone cells to participate in repair and remodeling events[11,12].

Indirect fracture estimates indicate an increased risk of hip fracture of 31% in women and 40% in men, and of vertebral fractures of 13% in women and 32% in men[10]. In their meta-analysis of 19 cohort and case-control studies, Law and Hackshaw found that in current smokers relative to non-smokers the risk of hip fracture was similar at the age of 50, but greater thereafter by 17% at age 60, 41% at age 70, 71% at age 80 and 108% at age 90[13]. Results from a large cross-sectional population-based study revealed that current smoking was not associated with a significantly higher incidence of fractures as compared with current non-smoking, although current long-term smokers (more than 35 years) had a significantly higher rate of all prevalence fractures[14]. The relation between smoking and hip fractures could be partly mediated by the association of low body weight with smoking[10,15].

Although using a different type of data (i.e. comparable method of data collection but different measures used for smoking and alcohol consumption), smoking and alcohol consumption in our subjects did not seem to differ significantly from the data reported from a population study in Croatia[16].

Our study showed men to smoke more and to be heavier smokers than women, as expected considering the sociocultural habits in Croatia. Also, the age at starting smoking was obviously older in the past. We could not confirm any association between smoking and prevalence of vertebral deformities. Nevertheless, we found a tendency to an increased risk of vertebral deformities in ever-smokers compared to never-smokers, although the result did not reach statistical significance. To estimate the strength of any possible association between smoking and vertebral deformities we considered the effect of important confounders, however, the results did not alter significantly. Results from prospective studies in postmenopausal women (OFELY study)[17], and in men and women aged 50–79 (EPOS study)[6] failed to show any consistent association with the incidence of all osteoporosis-related and vertebral fractures. Similarly, in a large, multicenter, cross-sectional, population-based study of risk factors on bone density and vertebral deformity, Lunt et al. found adverse effects of life-time smoking on bone mineral density[18]. However, it could not be confirmed for vertebral deformities, suggesting that bone mineral density may have acted as an intermediate variable.

The association of alcohol consumption with osteoporosis and osteoporotic fractures can be explained by its direct toxic effects or possible interference with bone metabolism[19]. In our study, men were found to take alcohol drinks significantly more often than women. We could not find any association between alcohol intake and prevalence of vertebral deformities, even when adjusted for important confounders. A limitation of our study was that the questions on alcohol intake were only based on the frequency of alcohol consumption, whilst no information was obtained on the amount of alcohol consumed. It also referred only to alcohol consumption during the previous year. The extent to which alcohol intake in that period reflected the intake in the past (and in the presumed biologically relevant time period with respect to bone mass) is unclear, although the similarity in the pattern of consumption in the subjects when they were divided into two age groups (above and below 65) suggested some consistency in the level of alcohol intake. In the literature there are few data on alcohol as a fracture risk factor. Seeman et al. found a small excess risk of vertebral deformity in men[20]. However, the bias of that study was that subjects were recruited from a metabolic clinic. Felson reported no excess of hip fractures in subjects aged ≥65 with alcohol intake of 207 ml or more *per* week[21]. A few epidemiologic studies indicated a beneficial effect of moderate alcohol consumption on skeletal health in postmenopausal women. In a population-based prospective cohort study, Bainbridge et al. found modest alcohol consumption to be a protective factor for greater femoral neck bone mineral density over time in premenopausal and postmenopausal women[22]. The MEDOS study has suggested moderate alcohol consumption (spirits) to be associated with a decrease in hip fractures[23]. Naves-Diaz et al. showed that elderly women (aged ≥ 65) who had been taking alcohol on more than 5 days a week had a reduced risk of vertebral deformity[5]. The same could also be inferred from the results of our study, where there was a tendency to a lower risk of vertebral deformities in women, when adjusted for confounders. However, the results were not statistically significant, and no definite conclusion could be made because of the wide confidence interval. In contrast to the study by Naves-Diaz et al., it was not observed in men. The reasons for this might be that men generally drink more than women, and a higher number of chronic abusers with an increased risk of osteoporotic fractures are likely to be found among men. Finally, in a recent twin study, Williams *et al.* found that women who drank moderately (8 units *per* week on an average) had higher bone mineral density at the hip and lumbar spine than women who drank minimal amounts of alcohol[24]. Additional studies are needed to confirm the amount of alcohol consumed that might be beneficial for bones, and to identify the level at which this benefit is outweighed by the increased risk from excess alcohol and the possible underlying mechanisms.

In conclusion, in a Croatian population sample of subjects aged ≥50 we confirmed that generally men smoke and drank more than women. We found no statistically significant association between smoking and pre-

valence of vertebral deformities. This also held for alcohol consumption, with the possible exception of women in whom regular moderate alcohol intake might have a protective effect against the risk of vertebral deformity. The statistically non-significant effects of smoking status and alcohol intake on vertebral deformity in the Croatian population might be the consequence of small sample size, but are generally consistent with the direction and magnitude of effects observed in larger studies in other populations. Additional data are needed to confirm or contradict our results.

Acknowledgements

The authors wish to thank Alan J. Silman and Terry W. O'Neill (ARC Epidemiology Research Unit, Manchester, UK) for their valuable assistance.

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UTJECAJ PUŠENJA I UNOSA ALKOHOLA NA DEFORMACIJE KRALJEŽAKA U STARIJIH OSOBA – EPIDEMIOLOŠKA STUDIJA

SAŽETAK

Cilj istraživanja bio je procijeniti ulogu pušenja i pijenja alkohola kao mogućih čimbenika rizika za deformacije kralježaka na uzorku starije hrvatske populacije. U 425 randomizirana neinstitucionalizirana ispitanika prikupljeni su podatci o pušenju, uporabi alkohola, indeksu tjelesne mase i ukupnoj tjelesnoj aktivnosti. U određivanju deformacija torakalnih i lumbarnih kralježaka upotrebljena je radiografska morfometrijska metoda. Muškarci puše i piju alkohol više nego žene. Nije nađena povezanost pušačkog statusa i broja popušenih cigareta ili učestalosti pijenja alkoholnih pića s prevalentnim deformacijama kralježaka. Postoji tendencija povišenog rizika za deformacije kralježaka u osoba koji više piju (OR=1.69; 95% CI=0.98–2.91) i smanjenja tog rizika u žena koje redovito piju (OR=0.72; 95% CI=0.14–3.66). Potrebna su daljnja istraživanja u hrvatskoj populaciji kako bi se utvrdila povezanost pušenja i konzumacije alkohola s deformacijama kralježaka.