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Hypertension and Cigarette Smoking: A Case-Control Study of Hypertension Patients in Yerevan

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Contents

ABSTRACT

Hypertension cause over half of all death in the world and at the end of the twenty century hypertension is supposed to pose as a national health problems all over the world.[4] In Armenia the mortality rate from hypertension rose from 52.8 out of 278.065 per 100000 population for all cardiovascular diseases to the 73.45 out of 351.17 per 100000 over the last ten years.[3] If hypertension untreated, about 50 percent of patients die of coronary heart disease, about 33 percent of stroke, and 15 percent of renal failure.[4] Commonly the role of hypertension is underestimated in causing the underlying vascular damage that leads to cardiovascular catastrophes. The death is usually attributed to stroke or infarction instead of hypertension which in fact is largely responsible. Besides, hypertension may not persist after a myocardial infarction or stroke. The immediate purpose of the present investigation is to reveal the strength of association between cigarette smoking and hypertension among men in Armenian. The obtained results show that there is no positive association between cigarette smoking and hypertension.. However it appears that former smokers are more prone to develop hypertension. The finding regarding the number of cigarettes shows that if one smokes more than 15 cigarettes per day he has an 1.2 times more risk to develop hypertension than those who smoke less than 15 cigarettes per day. It is also interesting to mention family history on smoking and hypertension, because the association is negative and there is no any research in literature regarding that. However, it can be expected that further research will develop stronger association between cigarette smoking and hypertension in terms of statistic significance. It has been found that physical exercise has a negative impact on hypertension development. It can also be analyzed from another point of view, because physical exercises are recommended for cardiovascular disease protection as a healthy lifestyle. It is obtained that especially morning running about 30 minutes more than 4 times a week shows a protective trend toward hypertension development. Regarding weight, it could be said that though the results are not statistically significant, they are correlated with data stated in literature. The blood pressure increases with weight gain and decreases with weight loss. However, there is no any specific data on factors that can be responsible for that change in blood pressure. In the present article it is obtained that weight over 65 kg can be a predictor for hypertension. It is worth mentioning that there is another interesting finding regarding alcohol consumption. According to literature, there are controversial ideas. The findings of present study correlate with ideas according to which drinking more than 250g per week can decrease the blood pressure, whereas the drinking of less than 250g can lead to some increase of blood pressure.

BACKGROUND

Hypertension is the major risk factor for coronary, cerebral, and renal vascular diseases, which cause over half of all death in the world and according to many authors at the end of the twenty century hypertension is supposed to pose as a national health problems all over the world.[4] The risk of developing coronary disease rise progressively with increase of systolic or diastolic pressure, both in the middle age and in the elderly. Besides, the number of persons identified as having hypertension continues to increase all over the world.[1] In the recent two decades in the United States hypertension has become the most frequent reason for visiting physicians as well as the indication for prescription drugs.[2]

In Armenia the mortality rate from hypertension rose from 52.8 out of 278.065 per 100000 population for all cardiovascular diseases to the 73.45 out of 351.17 per 100000. The cardiovascular diseases are still the leading cause of death in Armenia.[3]

The higher the level of blood pressure, the more likely different cardiovascular diseases will develop prematurely through acceleration of atherosclerosis, which is the pathological hallmark for uncontrolled hypertension. If hypertension untreated, about 50 percent of patients die of coronary heart disease, about 33 percent of stroke, and 15 percent of renal failure.[4] Commonly the role of hypertension is underestimated in causing the underlying vascular damage that leads to cardiovascular catastrophes. The death is usually attributed to stroke or infarction instead of hypertension that is largely responsible. Besides, hypertension may not persist after a myocardial infarction or stroke.

Since the measurement of blood pressure becomes widespread, the greater number of patient with hypertension is being identified. The question concerning the determination of the appropriate levels for prescription the diagnosis as having hypertension has increasingly become a problem. Blood pressure varies throughout the day and night, whether the person usually has normal or unusually high levels of blood pressure. The WHO's Expert Committee on Hypertension defines hypertension as systolic pressure of 160 mm Hg or higher and diastolic pressure of 95 mm Hg or higher. For comparison, normal adult blood pressure refers to systolic pressure of 140 mm Hg or lower and diastolic pressure of 90 mm Hg or lower. No single or specific cause is known for most hypertension. Since persistent hypertension can develop only in response to an increase in cardiac output or rise in peripheral resistance, defects may be present in one or more of the

multiple factors that affect two forces - cardiac output and peripheral resistance. The interplay of various factors affecting cardiac output and peripheral resistance may accelerate the disease. These factors may differ both in type and degree in different patients, because looking for a single defect may be a mistake.[5]

One of the major risk factors for hypertension is hemodynamic patterns Blood Pressure = Cardiac Output x Peripheral Resistance where if Increased Cardiac Output x Increased Peripheral Resistance than = Hypertension. The pathogenesis of disease is probably a slow and gradual process, so by the time the blood pressure becomes elevated, the initiating factors may no longer be apparent, because they may be normalized by the compensatory mechanisms already directed to that.[6]

The annual number of deaths from smoking is still increasing in the developed countries, but it will be increasing even faster elsewhere. Over the past few decades there has been a massive global increase in cigarette consumption, which will have its main effect on mortality in the next century. As well known cigarette smoking is one of the risk factor for vascular disease. There is also a strong association between cigarette smoking and all manifestations of coronary heart disease. At present, there are about 2 million deaths per year in developed countries from smoking. Framingham study demonstrated that cigarette smokers have a 2 – to 3 –fold increase in sudden death risk in each decade of life at entry between 30 and 59 years. [7] Cigarette smoking raises blood pressure, probably through the nicotine-induced release of norepinephrine from adrenergic nerve endings. The increased risk of stroke among cigarette smokers probably involves an acute fall in cerebral flow.[8] In 1990 in all developed countries for age 35-70, 318000 deaths were due to vascular diseases attributed to smoking from total 926000 deaths for male population. For Former Soviet Countries the mortality rate from hypertension was 25 per 100000 for men and 17 per 100000 for women out of all vascular disease 829 per 100000 and 386 per 100000 respectively. In Armenia for age 35-69 in 1990 from total 5.900 deaths for males, 2.200 were due to smoking, and mean years lost per death from smoking was 21 years. In Armenia, according to statistics of 1990, smoking attributed 919 for age 35-70 of all vascular deaths - 2562,.[9]

As authored by Japanese scientists the 24-hour ambulatory blood pressure was significantly lower in the nonsmoking period than in the smoking period.[10] Smokers have a greater seasonal variation in blood pressure than nonsmokers.[11] Smoking apparently potentiates the cardiovascular response to various climatic conditions and that factor should be taken into account in studies of blood pressure and in the diagnosis and treatment of

hypertension, particularly among cigarette smokers. It was documented that cigarette smoking was significant risk factors in people irrespective of the type of occupation and educational level.[12] While cigarette smoking is well established as a major risk factor for coronary heart disease in middle age population, some authors state diminished effect of cigarette smoking on coronary heart disease in elderly.[13] But it is argued that cigarette smoking has the same degree on cardiovascular disease development in elderly as in the middle age. It is therefore understandable that the cessation of cigarette smoking be promoted at all ages as part of an overall strategy directed to prevention of cardiovascular disease.[14] It is authorized that when smokers quit, a trivial rise in blood pressure may occur, probably reflecting a gain in weight.[4]

Smoking and lowering high blood pressure can help to prevent stroke. People with high blood pressure were twice under the risk of developing a stroke than those with normal blood pressure, meanwhile smokers had nearly twice the risk for having a stroke.

The first major study associating smoking with predicted risk of cardiovascular disease was published in 1958, and was confirmed by various epidemiological studies from all around the world. The association was strong and consistent that smoking is considered a cause of cardiovascular disease in the common sense of the world. However, the chain of causation that links specific components of cigarette smoking to mechanisms involved in smoking-associated cardiovascular disease has not identified.[15]

Atherosclerotic vascular disease (clogged arteries) is one of the chief contributor to hypertension. It is documented that smoke is an important risk factor for stroke and ishemic heart disease in hypertensive patients aged below 6o years, but less important in those aged over 60 years. The natural history of atherosclerosis and complexity of its ethiology and pathogenesis indicate that chronic exposure to a biologically active agent such as cigarette smoke could influence atherogenesis by many possible mechanisms. Comparison of smokers and nonsmokers has failed to show consistent differences in plasma cholesterol concentration, the major risk factor for atherosclerosis. Low-density lipoprotein (LDL) is associated with more severe atherosclerosis and greater risk of atherosclerotic disease, and high-density lipoprotein (HDL) with less severe atherosclerosis and lower risk of disease. Several studies show that smokers have lower HDL cholesterol concentration than nonsmokers. The difference is usually small, 5 mg/dl or less, but this difference represents a 10% decrease and would be expected to affect atherogenesis to a significant degree, particularly if it were maintained for 20 or 30 years between young adulthood and middle

age. And how smoking causes a depression of plasma HDL cholesterol concentration is not discovered, because the methabolism of HDL is much less studied than that of LDL.[15]

Regarding the gender, a higher percentage of men than women have high blood pressure until age 55. From age 55-74 the percentage of women having hypertension is somewhat higher than the percentage of men. Genetic alteration may initiate the cascade to hypertension as well. Although heredity plays a role in hypertension development, no discriminatory gene markers are currently available in determining the pathogenesis.[16] In studies of twins and family members in which the degree of familial aggregation of blood pressure levels is compared with the closeness of genetic sharing, the genetic contributions have been estimated to range from 30 to 60 per cent.[17] It is supposed that one of the hormones that is a prime candidate for a major role in hypertension development is insulin. This belief comes from the knowledge that hypertension is more common in obese and that hyperinsulinemia is a hallmark of obesity. The associations are most striking in people with upper body obesity, who have the highest prevalence of hypertension and the most pronounced hyperinsulinemia.[18]

Considerable circumstantial evidence support a causal role for sodium in genesis of hypertension. This evidence includes an increase in intracellular sodium in hypertensive animals and people, and increase that extends even to the hormotensive children of hypertensive parents.[4] The TONE study is a controlled trial demonstrating that reducing dietary sodium, and loosing weight result in a significant reduction of blood pressure in hypertensive men and women, aged 60-80 years. Further in obese the combined intervention of sodium reduction plus weight loss was more effective than either alone. It is recommended that the general public consume no more than 6 grams of sodium chloride per day.[7] This recommendation is based on the evidence for an association between dietary sodium chloride intake and blood pressure derived from a substantial number of epidemiological observations and clinical trials of salt restriction.

Both as a direct pressor and as a growth promoter, the renin-angiotensin mechanism may also be involved in the pathogenesis of hypertension. All functions of renin are mediated through the synthesis of angiotensin II. This system is the primary stimulus for the secretion of aldosterone and hence mediates the mineralocorticoid response to varying sodium intakes and volume loads. In addition to primary role in the preservation of normal fluid volume, the renin-angiotensin system participates in the control of blood pressure under circumstances of sodium depletion or volume contraction. When fluid volume is normal, blockade of renin-angiotensin system does little to the blood pressure, but during

volume contraction the increased levels of renin-angiotensin play an important role in maintaining the integrity of the circulation.[4]

Hypertension is more common among obese individuals and probably adds to their increased risk of developing ischemic heart disease. The findings corroborates the critical importance of the distribution of body fat, since blood pressure as well as blood lipids and glucose levels tend to be higher in those with central or upper body obesity.[19, 20] Risk estimates from population studies suggest that 75% of hypertension can be directly attributed to obesity.[7] It is well documented that blood pressure increases with weight gain and decreases with weight loss. In addition there is increasing evidence that obesity may provide the impetus for sympathetic nervous activation as well as for changes in renal structure and function. There is a considerable evidence that renal dysfunction, characterized by increased tubular sodium reabsorbtion and resetting of pressure natriuresis, plays a key role in increasing blood pressure in obese subjects. Physical fitness may help to prevent hypertension, and persons who are already hypertensive may lower their blood pressure by means of regular isotonic exercise.[21]

The role of alcohol in hypertension development deserves a special interest. In small quantities alcohol may raise blood pressure, in larger quantities alcohol may be responsible for a significant number of cases of hypertension. In all studies of this problem, the relationship between alcohol and blood pressure has been found to be independent of other known variables.[4] Incidence of heart disease in those who consume moderate amounts of alcohol – an average of one to two drinks per day for men and one drink per day for women is lower than that in nondrinkers. Alcohol intake of up about 20 g/day does not increase the risk of hypertension, but beyond this level, the risk increases progressively. However, with increasing consumption of alcohol there are increased public health dangers such as alcoholism, hypertension, stroke etc.[7] The ideas are controversial. Some have found a linear, progressively increasing level of blood pressure with increasing consumption of alcohol, whereas some found lower levers of blood pressure among those who drink one to two ounces of ethanol a day than among those who drink none at all.[22, 23]

The immediate purpose of the present investigation is to reveal the strength of association between cigarette smoking and hypertension among Armenian men. The association will be interpreted regarding the amount smoked per day, duration of smoking, type of cigarette – filter, non filter. Actually there is no any research done in Armenia regarding this problem, so the present research will give some points in understanding of the mechanisms of vascular disease development.

METHODS

The research question for this study is to reveal the strength of association between cigarette smoking and hypertension among men in Yerevan. The case-control method was used to investigate this research question because of its time efficiency and expense. The null hypothesis – there is no association between cigarette smoking and hypertension, the alternative hypothesis – there is association between cigarette smoking and hypertension. Hypertension is a dependent variable, cigarette smoking is independent variable.

Data collection:

Data collection was done by telephone interview and by face–to-face interview, it is less expensive, it can be completed more rapidly and with fewer staff than other interview methods. The anonymity of telephone interview may improve the response rate on sensitive questions[24]. The systematic random sample method was used to select cases, since the list of patients was available. The sample consisted of diagnosed hypertensive patients attending the Department of Hypertension, Institute of Cardiology, from 1998 up to now (patients received care at Institute of Cardiology on average for two weeks) and the patients attending the Department of Hypertension, Institute of Cardiology and Department of Cardiology of the Yerevan Municipal Hospital # 3 since September 1999. Controls were selected using random digit dialing method considering ratio one case to one control. The questionnaire was pretested in Cardiology Department of Yerevan Municipal Hospital # 3. After pilot survey, some changes were done in questionnaire (see appendix).

Definition of Cases:

Cases are men patients diagnosed with hypertension during time period 1998 – 1999 included in sample, living in Yerevan aged 40 - 80 obtained from medical register from Department of Hypertension, Institute of Cardiology and Department of Cardiology of Yerevan Municipal Hospital # 3.

Definition of Controls:

Controls are men who were selected using random digit dialing table never diagnosed with hypertension in their living in Yerevan aged 40-80.

Sample Size:

Sample size has been calculated assuming the equal number of cases and control for unmatched study. Specifying the values for $\alpha = 0.05$ and $\beta = 0.1$. Let p_0 denote the estimated exposure rate among controls and = 0.5, R is relative risk and = 2.8 [25].

$$n = [Z_\alpha \sqrt{2pq} + Z_\beta \sqrt{p_1 q_1 + p_o q_o}]^2/(p_1 - p_o)^2$$

Corresponding to $\alpha = 0.05$ and $\beta = 0.1$, than $Z_\alpha = 1.96$ and $Z_\beta = 1.28$.

$$p_1 = p_o R/ [1 + p_o (R - 1)]$$

$$p = 1/2 (p_1 + p_o) \qquad \text{and} \qquad q = 1 - p$$

After calculations *n =86. (two sided)*

Power Calculations:

Because of shortage of time it was impossible to conduct study using calculated sample size. Therefore the sample of cases and controls were those whom I managed to question. The final sample size consisted of 31 cases and 31 controls. Therefore, because of the shortage of time, sample size is smaller than it should have been. As a result, it is required to determine the resulting power 1 - β.

```
Estimated power for two-sample comparison of proportions
```

```
Test Ho: p1 = p2, where p1 is the proportion in population 1
                    and p2 is the proportion in population 2

                         Assumptions:

                    alpha =   0.0500  (two-sided)
                              p1 =   0.5000
                              p2 =   0.7360
                sample size n1 =       31
                              n2 =       31
                            n2/n1 =     1.00

                       Estimated power:

                            power =   0.3750
```

Thus, if the relative risk is = 2.8, a case – control study of n = 31 per each group has only a 37 percent chance of finding that the sample estimate will be significantly different from unity.

Analysis:

Analysis was done by creating the database in the Microsoft Access. After that the data was transformed to STATA Program (Statistics/Data Analysis) to determine whether there is a disease – exposure association by estimating the OR for a single 2 x 2 table.

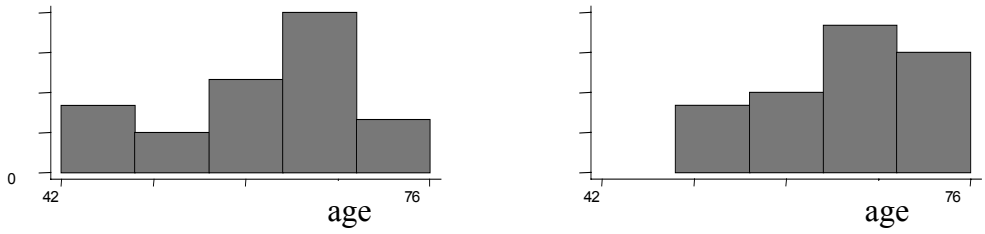
RESULTS

Age analysis shows that the youngest person in study was 42 years old man, and oldest one was 76 years old. The mean age for study was 62 years old with standard deviation 8.2846. The percentage of age distribution is the follow:

Table 1. Percentiles of age distribution of cases and controls

Age	Percentiles
42 - 50 years old	10 %
- 56 years old	25 %
- 64 years old	50 %
- 69 years old	75%
- 73 years old	90%

Figure 1. Histogram of age distribution



Control Cases

Calculating by 2 x 2 table the OR for association between age more and less 60 and the hypertension status was found that there is a positive association (OR = 2.3), but the association is not statistically significant.

Cornfield 95 % Confidence Interval .8239173 - 6.7849

Smoking:

It was found that from 31 cases 18 are smoker and from 31 controls 19 are smoker. 2 x 2 table calculations shows that there is no association between cigarette smoking and hypertension development. (OR=0,8), with a Cornfield 95 % Confidence Interval .3205945 2.385734

Table 2 Relationship of Cigarette Smoking to Hypertension

	Smokers	Nonsmokers	Total
Cases	18	13	31
Controls	19	12	31
Total	37	25	62

However the analysis regarding the former smoking status shows an opposite association with OR = 1.3, although the results are not statistically significant (Cornfield 95% Confidence Interval .3127456 6.069284)

```
                | former smoker          |        Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+------------------------+----------------------
          Cases |         4          27  |        31      0.1290
       Controls |         3          28  |        31      0.0968
----------------+------------------------+----------------------
          Total |         7          55  |        62      0.1129
                |                        |
                |      Point estimate    |  [95% Conf. Interval]
                |------------------------+----------------------
     Odds ratio |         1.382716       |.3127456    6.069284 (Cornfield)
 Attr. frac. ex. |         .2767857       |-2.197487  .8352359  (Cornfield)
 Attr. frac. pop |         .0357143       |
                +-------------------------------------------------
                           chi2(1) =  0.16  Pr>chi2 = 0.6882
```

Regarding number of cigarettes smoked per day in the average the following results are appeared

```
                | cig_nom                |         Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+------------------------+----------------------
          Cases |        15          16  |        31      0.4839
       Controls |        13          18  |        31      0.4194
----------------+------------------------+----------------------
          Total |        28          34  |        62      0.4516
                |                        |
                |      Point estimate    |  [95% Conf. Interval]
                |----------------------+---------------------
```

```
     Odds ratio |         1.298077       |.4813517  3.500312  (Cornfield)
 Attr. frac. ex. |         .2296296       |-1.077483 .7143112  (Cornfield)
 Attr. frac. pop |         .1111111       |
                 +-----------------------------------------------
                            chi2(1) =  0.26  Pr>chi2 = 0.6098
```

It was calculated from 2x2 table for case and control for number of cigarettes smoked more and less 15 per day. It is appeared the positive association between number of cigarettes smoked per day and hypertension OR = 1.29, although there is no any statistically significance association (Cornfield 95% Confidence interval .4813517 3.500312)

Physical Exercise:

The association between physical exercise and hypertension is negative, it appears from the table (OR=0.47, with Cornfield 95% Confidence Interval .1432029 1.556129)

```
                | exercise                |             Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+-------------------------+----------------------
           Cases |         5          26  |        31      0.1613
        Controls |         9          22  |        31      0.2903
----------------+-------------------------+----------------------
           Total |        14          48  |        62      0.2258
                 |                        |
                 |      Point estimate    |  [95% Conf. Interval]
                 |------------------------+----------------------
     Odds ratio |         .4700855       |.1432029  1.556129  (Cornfield)
 Prev. frac. ex. |         .5299145       |-.5561292 .8567971  (Cornfield)
 Prev. frac. pop |         .1538462       |
                 +-----------------------------------------------
                            chi2(1) =  1.48  Pr>chi2 = 0.2244
```

It was also found that from the 31 cases 5 have a morning running, but no one attend to gym and was not an sportsman, from controls only 9 have morning running and also no one attend to gym and was not an sportsman. The association between running and hypertension is negative (OR= 0.47, Cornfield 95% Confidence Interval .1432029 1.556129)

```
                  | Running             |       Proportion
                  | Exposed   Unexposed |     Total     Exposed
 -----------------+-------------------------+----------------------
             Cases |     5          26   |        31      0.1613
          Controls |     9          22   |        31      0.2903
 -----------------+-------------------------+----------------------
            Total |     14          48   |        62      0.2258
                  |                      |
                  |      Point estimate   |  [95% Conf. Interval]
                  |-----------------------+----------------------
     Odds ratio |         .4700855       | .1432029  1.556129  (Cornfield)
 Prev. frac. ex. |         .5299145       |-.5561292  .8567971  (Cornfield)
Prev. frac. Pop       |     .1538462             |
```

As can be seen there is an negative association between frequency of morning running and hypertension (OR= 0.1, and results are statistically significant P value 0.04)

```
                |      Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+------------------------+----------------------
          Cases |         1          30  |        31      0.0323
       Controls |         6          25  |        31      0.1935
----------------+------------------------+----------------------
          Total |         7          55  |        62      0.1129
                |                        |
```

Weight

By 2x2 table was calculated the association between weigh and hypertension for cases and controls for weight less and more 65 kg.

```
                |   Exposed   Unexposed  |     Total     Exposed
----------------+------------------------+----------------------
          Cases |        16          15  |        31      0.5161
       Controls |        14          17  |        31      0.4516
----------------+------------------------+----------------------
          Total |        30          32  |        62      0.4839
                |                        |
                |      Point estimate    |  [95% Conf. Interval]
                |------------------------+----------------------
     Odds ratio |         1.295238       |.4821689  3.479348  (Cornfield)
 Attr. frac. ex. |         .2279412       |-1.073962 .7125898  (Cornfield)
 Attr. frac. pop |         .1176471       |
                +-------------------------------------------------
                           chi2(1) =     0.26  Pr>chi2 = 0.6113
```

The positive association appears for weight more than 65 kg at OR = 1.29, and Cornfield 95% Confidence Interval .4821689 3.479348.

Family history:

```
                | family history/        |
                | hypertension           |        Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+------------------------+----------------------
          Cases |         8          23  |        31      0.2581
       Controls |        11          20  |        31      0.3548
----------------+------------------------+----------------------
          Total |        19          43  |        62      0.3065
                |                        |
                |      Point estimate    |  [95% Conf. Interval]
                |------------------------+----------------------
     Odds ratio |         .6324111       | 2171419  1.846225  (Cornfield)
 Prev. frac. ex. |         .3675889       |-.8462248 .7828581  (Cornfield)
 Prev. frac. pop |         .1304348       |
                +-------------------------------------------------
                           chi2(1) =     0.68  Pr>chi2 = 0.4086
```

It can be seen the negative association between family history on hypertension and hypertension (OR = 0.63, but there is no statistically significant association Cornfield 95% Confidence Interval .2171419 1.846225)

There is a negative association between family history on diabetes and hypertension OR = 0.61, and the results are not statistically significance (Cornfield 95% Confidence Interval is .1664981 2.307633)

	family history /diabetes		Proportion	
	Exposed	Unexposed	Total	Exposed
Cases	4	27	31	0.1290
Controls	6	25	31	0.1935
Total	10	52	62	0.1613

There is a positive association between heart disease and hypertension OR = 1.58.

	family history/ heart diseases		Proportion	
	Exposed	Unexposed	Total	Exposed
Cases	11	20	31	0.3548
Controls	8	23	31	0.2581
Total	19	43	62	0.3065

There is negative association between family history on smoking and hypertension OR= 0.65, but the results are not statistically significant (Cornfield 95% Confidence Interval .2370736 1.835381)

	family history /smoking		Proportion		
	Exposed	Unexposed	Total	Exposed	
Cases	18	13	31	0.5806	
Controls	21	10	31	0.6774	
Total	39	23	62	0.6290	
	Point estimate		[95% Conf. Interval]		
Odds ratio	.6593407		.2370736	1.835381	(Cornfield)
Prev. frac. ex.	.3406593		-.8353812	.7629264	(Cornfield)
Prev. frac. pop	.2307692				

chi2(1) = 0.62 Pr>chi2 = 0.4303

Diabetes:

Was analysed the association between diagnosed diabetes and hypertension. There is a association between diabetes and hypertension, OR = 1.44, and Cornfield 95% Confidence Interval .4506919 4.642051

	Diabetes		Proportion	
	Exposed	Unexposed	Total	Exposed
Cases	8	23	31	0.2581
Controls	6	25	31	0.1935
Total	14	48	62	0.2258

Drink:

	drink status		Proportion	
	Exposed	Unexposed	Total	Exposed
Cases	25	6	31	0.8065
Controls	26	5	31	0.8387
Total	51	11	62	0.8226
	Point estimate		[95% Conf. Interval]	
Odds ratio	.8012821		.2284091	2.820763 (Cornfield)
Prev. frac. ex.	.1987179		-1.820763	.7715909 (Cornfield)
Prev. frac. pop	.1666667			

chi2(1) = 0.11 Pr>chi2 = 0.7396

There was found no association between alcohol usage and hypertension OR = 0.8 and Cornfield 95% Confidence Interval .2284091 2.820763, association is not statistically significant.

However was found some paradoxical association between amount of alcohol drinking per week. In case of drinking more than 250g per week at average there is a protective effect, negative association OR = 0.2 and result is statistically significant, P value for test = 0.0157.

```
                | vol_nom                 |             Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+-------------------------+----------------------
          Cases |        16          15  |        31      0.5161
       Controls |        25           6  |        31      0.8065
----------------+-------------------------+----------------------
          Total |        41          21  |        62      0.6613
                |                         |
                |      Point estimate     |  [95% Conf. Interval]
                |-------------------------+----------------------
     Odds ratio |              .256       |.0844871  .7808893  (Cornfield)
 Prev. frac. ex. |              .744       |.2191107 .9155129  (Cornfield)
 Prev. frac. pop |                .6       |
                +-------------------------------------------------
                           chi2(1) =     5.83  Pr>chi2 = 0.0157
```

In case of less than 250g, there is a positive association between amount of alcohol and hypertension development.

```
                | vl_nom                  |        Proportion
                |   Exposed   Unexposed  |     Total     Exposed
----------------+-------------------------+----------------------
          Cases |        19          12  |        31      0.6129
       Controls |        15          16  |        31      0.4839
----------------+-------------------------+----------------------
          Total |        34          28  |        62      0.5484
                |                         |
                |      Point estimate     |  [95% Conf. Interval]
                |-------------------------+----------------------
     Odds ratio |         1.688889        | 6222406 4.583284  (Cornfield)
 Attr. frac. ex. |         .4078947        |-.6070955 .7818158  (Cornfield)
 Attr. frac. pop |              .25        |
                +-------------------------------------------------
                           chi2(1) =     1.04  Pr>chi2 = 0.3074
```

DISCUSSION

The present study has been done in order to find the association between hypertension and cigarette smoking, as one of the risk factors in hypertension development. The obtained results show that there is no positive association between cigarette smoking and hypertension, which is appeared as an opposite finding to those stated in literature. However, it can't be taken as a valid statement because of small power 0.3 and statistically insignificant association. The further research is needed to determine that association because it can be linked also with some factors related to Armenian population, which is not included in this study.

Despite, some findings will be interesting to discuss. It appears that former smokers are more prone to develop hypertension, and it correlates with data from literature. It is authorized that when smokers quit, the increasing of blood pressure may occur because of gain in weight.[4]

The finding regarding the number of cigarettes shows that if one smokes more than 15 cigarettes per day he/she has an 1.2 times more risk to develop hypertension than those who smoke less than 15 cigarettes per day. There is no any certain data regarding the number of cigarettes and hypertension development in literature. It is also interesting to speculate about family history on smoking and hypertension, because the association is negative and there is no any research in literature regarding that However it can be appeared that further research will develop more strong association between cigarette smoking and hypertension in terms of statistic significance.

Though the result are statistically insignificance there is some interesting findings that should be interpreted. It was found that physical exercise has a protective impact on hypertension development. It can be seen from another point of view, because physical exercise is recommended for cardiovascular disease protection as a healthy lifestyle. And it is obtained that especially morning running about 30 minutes more than 4 times a week shows a protective trend toward hypertension development. It would be interesting to investigate the hypertension and interaction of smoking status and physical exercises which are characteristic for Armenian population.

Although the findings are not statistically significant it should be mentioned that diabetes appears as a positive risk factor for hypertension. However, there are no clearly stated mechanisms of that association in literature, even though many authors claim that diabetes status is a predictor for hypertension development.

Regarding the weight it can be said that though the results are not statistically significant, they are correlated with data stated in literature. It is documented that about 75% of hypertension is associated with weight. The blood pressure increases with weight gain and decreases with weight loss. However, there is no any specific data that can be responsible for that change in blood pressure. In present article it is obtained that weight over 65 kg can be a predictor for hypertension.

The opposite correlation appears comparing the family history on hypertension and cardiovascular disease. In case of family history on hypertension there is a negative association with hypertension but in case of family history on cardiovascular disease there is a positive association, which is very difficult to interpret.

Another interesting finding should be mentioned regarding alcohol consumption. According to literature, there are controversial ideas. Some have found a linear, progressively increasing level of blood pressure with increasing consumption of alcohol, whereas some found lower levers of blood pressure among those who drink one to two ounces of ethanol a day than among those who drink none at all.[22, 23] The findings of present study correlate with ideas that drinking more than 250g per week can decrease the blood pressure, whereas drinking less than 250g can lead to some increase of blood pressure. However, there are no specific results which can approve marginal amount of alcohol which is responsible for high blood pressure.

Limitations

1. The major factor for the insignificant results is the small sample size instead of the needed. The small sample size can be a cause for some opposite results obtained through the analysis as well. It was impossible to have the needed sample size because of the shortage of time. The interview was conducted during September and beginning of October.
2. It should be mentioned that results are not generalizable, because only men population was involved in the study. Also it should be mentioned that the target population, both cases and controls, was only from Yerevan.
3. Another limitation is that analysis of patients' data taken from telephone interview was not done separately from analysis of patients from hospital. It would be interesting to have separate results, which could give some more information about association

between hypertension and risk factor. However, because of the shortage of time it has not been done.

4. As characteristic of any study it should be mentioned that information provided by interviewee can be not correct.
5. Besides, instrumental bias can have impact on results, though the questionnaire was designed to avoid that type of bias.
6. It should be mentioned that one of the limitations of the study is that the interaction is not defined, which also can have significant impact on findings.

Recommendations

1. It could be recommended that physical exercise especially running has a positive association with hypertension. The morning running more than 4 times per week has a protective effect on hypertension development.
2. The second can be that the weight less than 70kg can have protective effect on hypertension development.
3. It also could be recommended that absence of family history on hypertension has an protective effect on hypertension, although the results are not significant.

Conclusion

It is impossible from this study to conclude valid association between cigarette smoking and hypertension because of small power and insignificant results. However, it can be drawn that cigarette smoking has an impact on hypertension in terms of less or more 15 cigarette smoked per day. Also it could be supposed that family history on smoking has an impact on hypertension development.

Conclusion is that the physical exercise has a positive effect on hypertension especially the morning running over 4 times per week, which also can be recommended as a protector for hypertension development.

However, the more detailed analysis of association between cigarette smoking and hypertension in terms of confounding factors and interaction process is needed.

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Questionnaire

Introduction: I am a student of American University of Armenia, Department of Public Health. Currently I conduct research for my Master Thesis regarding to hypertension and other chronic disease. I would like to ask you a few questions about your lifestyle, habits and health. Your assistance will be very useful for my research. You can stop the interview whenever you want, and you can miss the questions whatever you want. The interview will least about 15 minutes. If you don't mind let me start the interview.

Thank you in advance for your cooperation.

1. Please your age _____?

2. Have you ever been diagnosed with hypertension?
Yes____
No____(go to question # 4)

3. For how many years have you suffered from hypertension?_____

4. Do you currently smoke?
Yes____
No____(go to question # 9)

5. How many cigarettes do you smoke per day? _____

6. When did you begin to smoke?

7. What type of cigarette do you smoke?
Filter____
Non filter____

8. How many persons smoke in your family? _____

9. Did you regularly smoke before?
Yes____
No____

10. Do you drink any alcohol?
Yes___
No ___ (go to questions # 13)

11. How many drinks do you drink on the average per week? 50gr

12. When did you start to drink it regularly?

13. Do you have family history on ? (check all possible answers)
1. cardiovascular disease
2. hypertension
3. diabetes
4. smoking

14. Do you do regularly any type of physical exercising?
Yes____
No_____(go to question # 17)

15. What type of exercise?
1. jog____
2. athletic gym___
3. sportsmen____

16. How many times did you exercise per week? _____

17. How much do you weigh in kg? _____

18. What kind of fat did prefer in your diet more frequently?

Animal fat_____
Vegetable fat_____

19. Have you ever been diagnosed as having diabetes?
Yes___
No____

Thank you for your time.