Prevalence of hypertension in obese and non-obese Saudis

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ABSTRACT

Objective: Obesity occurs at a high prevalence in the Saudi population. Studies in literature show that hypertension occurs more frequently in obese individuals. This study was designed to determine the prevalence of hypertension in obese Saudis in comparison with results obtained in non-obese individuals.

Methods: The screening involved a statistically designed household screening program. Only adults 14-70 years of age were included in the study. Blood pressure (systolic and diastolic) was measured when the individuals were in sitting position and height and weight were used to calculate Body Mass Index. All individuals with Body Mass Index > 30 were classified as obese and hypertension was measured as systolic blood pressure > 140 and diastolic blood pressure > 90 or both. The prevalence of hypertension was calculated in the obese and non-obese group. Chi square analysis was carried out to determine the significance of the difference in prevalence in different groups.

Results: In the non-obese males and females the prevalence of hypertension was 4.8% and 2.8%. While in the obese group the prevalence was almost 1.6 times higher in the males (8%) and 3.52 times higher (8%) in the female obese. The results were separated on the basis of the province to which the population belonged and hypertension prevalence was calculated in the obese and non-obese. In each region the prevalence of hypertension was higher in the obese group compared to the non-obese group. Non-obeses females had significantly lower hypertension prevalence than the male in the same province but the hypertension prevalence was higher in the females compared to the male in the obese group. Male in the Eastern, Southern and Western provinces did not show an increased hypertension prevalence in the obese.

Conclusion: Since the prevalence of obesity is high in Saudis and since obesity and hypertension occur together and cause serious complications, it is strongly suggested that measures are adopted to decrease prevalence of obesity and its underlying complications. Awareness programs are required at the level of the general public for successful implication of preventive programs.

Keywords: Obesity, hypertension, cardiovascular disease.

Saudi Medical Journal 2001; Vol. 22 (1): 44-48

Obesity is one of the most frequently encountered nutritional disorders in the developed countries and is assuming a significant status in the developing ones.[1] It has been considered as a health problem due to associated complications which include increased risk of cardiovascular diseases, cerebral and peripheral vascular diseases, hypertension, hyperlipidaemias, biliary tract diseases, osteoarthritis, gout, cancers of the gastro-intestinal tract, uterus and ovary and diseases of the female reproductive tract.[2-7] In addition, obesity has effects on longevity and general health in both young and old individuals and it has been shown that the overall mortality rate is increased in the obese compared to the normal weight individuals.[8]

Obese subjects are at an increased risk of becoming hypertensive and several studies have shown a significantly higher prevalence of

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Received 21st June 2000. Accepted for publication in final form 11th September 2000.

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Table 1 - Prevalence of hypertension in obese and non-obese Saudis.

Sex	No	Obese			Non-Obese		
		No	No. with HT	Prevalence of HT (%)	No	No with HT	Prevalence of HT (%)
Male*	6225	896	70	7.8	5329	256	4.8
Female*	8580	1685	146	8.7	6895	174	2.5
Total	14805	2581	216	8.4	12224	430	3.5

*Statistical significance of the difference in the prevalence of hypertension in obese and non-obese individuals
Male: χ^2=13.4 df=1 p<0.002
Female: χ^2=140.5 df=1 p<0.0001
Total: χ^2=119.0 df=1 p<0.0001
HT: Hypertension

hypertension in obese individuals.[9-24] This has a special implication as it predisposes to cardiovascular damage and atherogenic cardiovascular diseases.[25,26] Hence obesity is a major risk factor for hypertension, that itself is a direct cause of premature cardiovascular morbidity and mortality.[10,27] In an extensive investigation conducted in different regions of Saudi Arabia we observed that 13% of Saudi males and 20% of Saudi females were in the obese group with a Body Mass Index (BMI) of 30 or more.[28] We also reported the prevalence of hypertension in the overall Saudi population as 5.3% and 3.65% in the males and females. These findings in Saudis served as a basis of the present study to determine the prevalence of hypertension in obese and non-obese Saudis. This paper presents our findings in different regions of Saudi Arabia and discusses the urgent need to control obesity in Saudi Arabia, in order to control associated complications.

Methods. This study was conducted as a component of a major National Project to investigate the prevalence of diabetes mellitus in Saudi population. The screening was based on a statistically designed "Household Screening Programme", the details of which have been published elsewhere.[29] The population screened were Saudi males and females living in different provinces of Saudi Arabia. Randomly selected areas in each province were divided into sectors and in selected sector, every tenth street and on every street, every tenth house was selected for screening. The family was contacted and the purpose of the screening was explained. Only those families who volunteered were included in the study. An early morning visit was made to the family house and essential details were recorded for each member above the age of 14 years. Blood pressure (systolic and diastolic) was recorded by an experienced and trained nurse using mercury sphygmomanometer using standard method, when the individual was in sitting position. The height (in meters) and weight (in kilograms) were recorded. Height was recorded using a measuring tape, with the individual standing without shoes, next to the wall, with the heels, buttocks, shoulders and occiput touching the wall. The head was kept erect and the measuring tape was stretched slightly to measure the height to the nearest 0.1 cm. Weight was recorded on a measuring scale calibrated daily at the beginning of each working day. Each individual was requested to wear a light dress and the weight was recorded by taking two successive readings to the nearest 100g. For each individual the Quetelet index or the Body Mass Index (BMI) was calculated using the formula: weight (kg)/height^2(m^2). All individuals with a BMI > 30 were grouped as obese and those below 30 were grouped as non-obese. Using systolic pressure > 140 mmHg and/or diastolic pressure > 90 mmHg, the individuals were classified as hypertensive. Chi square analysis using 2 x 2 contingency tables were used to obtain the significance of difference between the results of any two groups.

Results. The study group comprised of 14,805 adult individuals (males = 6225; females = 8580) with ages ranging from 14-70 years. There were 2581 adults (males = 896 and females = 1685) who were classified in the obese group and 12224 (males = 5329; females = 6895) who were in the non-obese group). The prevalence of hypertension was calculated in the obese and non-obese males and females and the results in the total group are presented in Table 1. A significant increase in the prevalence of hypertension was observed in the obese, where the females had almost 3.5 times higher prevalence of hypertension compared to their non-obese counterpart, while the obese males had about 1.6 times higher prevalence than the non-obese males.

The total population was separated into five groups depending on the province to which they belonged and the number of males and females screened in

each province are presented in Table 2. In each area the prevalence of hypertension was calculated in the obese and non-obese males and females. In each area the prevalence of hypertension was higher in the obese group except in the male in the Eastern, Southern and Western provinces. The females had lower hypertension prevalence in the non-obese group and a prevalence significantly higher than the male in the obese group. The male obese had a higher prevalence of hypertension than the non-obese male (Table 2).

Discussion. Obesity and hypertension are closely associated multifactorial disorders and studies in different population groups have confirmed that the prevalence of hypertension increases significantly in the obese.[9-12,27,30] The results of this study confirm the same trend in the Saudi population, where the prevalence of hypertension is higher in the obese than the non-obese and the prevalence in the obese females is more than the obese males. The only exception are the male groups in the Eastern, Southern and Western provinces, who have a non-significant difference in the HT prevalence in the non-obese and the obese group. This suggests that not only obesity, but other factors, possibly genetic and environmental also play a role in increasing the prevalence of hypertension in the obese group. In general, weight gain in adults is a potent risk factor for the later development of hypertension and several studies have shown that weight reduction in the obese hypertensive often reduces arterial blood pressure.[11,13,31,32] It has also been suggested that birth-weight may also be linked to adult blood pressure, possibly as a result of its relationship with later obesity.[32] The mechanism of development of hypertension in obese individuals is shown to be due to hyperinsulinemia, a characteristic of obesity, and insulin resistance.[27,33-35] This is believed to contribute to the probability of developing hypertension by activating the sympathetic nervous system and by causing sodium retention.[27] In addition, the pressor effect of insulin in obesity may be further enhanced by the observation that its vasodilator action can be blunted in obese subjects. Furthermore, Leptin, a hormone, which is often increased in obese individuals, may contribute to development of hypertension, through its effects on insulin, sympathetic nervous system and sodium retention. In addition, the kidney may also play a role in producing hypertension by abnormal renal sodium handling coupled with structural changes in the kidney of an obese patient and these can raise blood pressure.[27]

Obesity and hypertension are major risk factors for cardiovascular system. Arterial hypertension

Table 2 - Prevalence of hypertension in obese and non-obese Saudis in different provinces of Saudi Arabia.

			Obese			Non-Obese		
Province*	**Sex**	**Total No.**	**No.**	**No. with HT**	**Prevalence of HT**	**No.**	**No. with HT**	**Prevalence of HT**
Central	M	1913	295	23	7.82	1619	50	3.90
	F	2616	595	48	8.07	2021	50	2.47
Eastern	M	251	33	3	9.1	218	29	13.3
	F	360	71	17	23.9	289	22	7.6
Southern	M	1926	203	12	5.9	1723	63	3.66
	F	2768	428	30	7.01	2340	39	1.70
Nothern	M	1127	151	17	11.26	976	51	5.22
	F	1518	313	26	8.30	1205	36	3.00
Western	M	1008	215	15	7.0	793	63	7.94
	F	1318	278	25	9.0	1040	27	2.60

M: Male, F: Female, HT: Hypertension
*Significance of the difference in the prevalence of HT in obese and non-obese.

Central: M: χ^2=13.82 df=1 p<0.01, F: χ^2=38.34, df=1 p<0.0001
Eastern: M:χ^2=0.156 df=1, p=0.7, F: χ^2=14.1, df=1, p<0.0001
Southern: M: χ^2=1.9, df=1, p=0.167, χ^2=40.3, df=1, P<0.0001
Nothern: M: χ^2=7.36, df=1, p<0.005, F: χ^2=4.69, df=1, p<0.05
Western: M: χ^2=0.107, df=1, p=0.743, F: χ^2=22.0, df=1, p<0.0001

increases afterload to the left ventricle, while obesity produces an increase in stroke volume and increases preload. Hence, as a result of this double burden, the heart adapts with eccentric left ventricular hypertrophy. Contractility becomes impaired early in the course of obesity hypertension and ventricular ectopy is observed. Thus, as a consequence, the obese hypertensive patient is at a high risk for congestive heart failure and sudden death.[23,26,36] It has been shown that childhood obesity is the single marker of the child at risk for development of cardiovascular disease later in life.[37] Obesity and a high dietary intake of carbohydrates, salts etc. or both can induce several potential pressor mechanisms: (1) higher plasma norepinephrine and epinephrine levels; (2) a tendency to hyperaldosteronism; (3) enhanced sensitivity of blood pressure to salt; (4) increased total blood volume leading to increased cardiac output and eventually eccentric left ventricular hypertropy, and finally (5) increased cystolic free Ca^{++} levels and reduced intracellular Mg^{++} levels.[38] These factors play a role in predisposing to the development of hypertension in the obese individuals. Since several studies have shown that weight reduction in obese patients not only reduces blood pressure, but also reduces the cardiovascular changes,[22,24,31] it is of utmost importance to adopt measures to decrease childhood and adult obesity in the Saudi population. This requires awareness programs for the general public, in order to educate them about the causes, consequences and ways and means of prevention of obesity. The importance of physical exercise and dietary restriction of calories, plays an important role in weight reduction and the awareness about those need to be spread to all including children and adults. These steps are essential in Saudi population who have a high prevalence of obesity and these prevention steps will play an essential role to reduce the complications associated with obesity.

References

1. Lara-Pantin E. Obesity in developing countries. In: Berry E, Blondheim SH, Eliahou HE, et al editors. Recent Advances in Obesity Research. V. London: John Libbey & Co; 1987. p. 5-8.
2. Bierman EL, Bagdade JD, Porte D Jr. Obesity and diabetes: the odd couple. Am J Clin Nutr 1968; 21: 1434-1437.
3. Kannel WB, Bran N, Skinner JJ, Dauber TR, McNamara PM. The relation of adiposity to blood pressure and development of hypertension. The Framingham Study. Ann Intern Med 1967; 67: 48-59.
4. Raison JM, Safar ME, Cambien FA, London GM. Forearm hemodynamics in obese normotensive and hypertensive subjects. J Hypertension 1988; 6: 299-303
5. Report of the Hypertension Task Force. Vol. 9. NIH Publication 79-1631: 59-77. Washington DC: USA; US Department of Health, Education and Welfare, 1979.
6. Krothiewski M, Bjorntorp P, Sjostrom L, Smith U. Impact of obesity on metabolism in men and women: importance of regional adipose distribution. J Clin Invest 1983; 72: 1150-1162.
7. Assmann G, Schutte H. Obesity and hyperlipidaemia: results for the prospective cardiovascular Munster (PROCAM) study. In: Bjorntorp P, Brodoff BN, editors. Obesity. Philadelphia: Lippincott Co; 1992. p. 502-511.
8. Bray GA. Obesity: definition, diagnosis and disadvantages. Med J Aust 1985; 142: 52.
9. Dustan HP. Hypertension and obesity. Prim Care 1991; 18: 495-507.
10. Dustan HP. Obesity and hypertension. Diabetes Care 1991; 14: 488-504.
11. Dustan HP. Obesity and hypertension. Ann Intern Med 1985; 103: 1047-1049.
12. Heyden S, Schneider KA. Obesity and hypertension: epidemiological aspects of the relationship. J Hum Hypertens 1990; 4: 431-435.
13. Kaplan NM. Obesity in hypertension: effects on prognosis and treatment. J Hypertens Suppl 1998; 16: S35-S37.
14. Jones DW. What is the role of obesity in hypertension and target organ injury in African Americans. Am J Med Sci 1999; 317: 147-151.
15. Kumanyika SK. The association between obesity and hypertension in blacks. Clin Cardiol 1989; 12: IV72-77.
16. Landsberg L. Obesity and hypertension: experimental data. J Hypertens Suppl 1992; 10: S195-S201.
17. Bjorntorp P. Hypertension and other complications in human obesity. J Clin Hypertens 1986; 2: 163-165.
18. Frohlich ED. Obesity and hypertension. Hemodynamic aspects. Ann Epidemiol 1991; 1: 287-293.
19. Schmieder RE, Rockstroh JK. Obesity and hypertension. Curr Opin Nephrol Hypertens 1994; 3: 546-549.
20. Rocchini AP. Adolescent obesity and hypertension. Pediatr Clin North Am 1993; 40: 81-92.
21. Richards RJ, Thakur V, Reisin E. Obesity-related hypertension: its physiological basis and pharmacological approaches to its treatment. J Hum Hypertens 1996; 10: S59-S64.
22. MacMahon S, Cutler J, Brittain E, Higgins M. Obesity and hypertension: epidemiological and clinical issues. Eur Heart J 1987; 8 Suppl B: 57-70.
23. Schmieder RE, Messerli FH. Obesity hypertension. Med Clin North Am 1987; 71: 991-1001.
24. Staessen J, Fagard R, Amery A. Obesity and hypertension. Acta Cardiol Suppl 1988; 29: 37-44.
25. Kannel WB. Fifty years of Framingham Study contributions to understanding hypertension. J Hum Hypertens 2000; 14: 83-90.
26. Licata G, Scaglione R, Dominguez LJ. Early markers of cardiovascular damage in obese subjects. Nutr Metab Cardiovasc Dis 1999; 9: 78-86.
27. Mikhail N, Golub MS, Tuck ML. Obesity and Hypertension. Prog Cardiovasc Dis 1999; 42: 39-58.
28. El-Hazmi MAF and Warsy AS. Prevalence of obesity in the Saudi population. Ann Saudi Med 1997; 17: 302-306.
29. El-Hazmi MAF, Warsy AS, Al-Swailem AR, Al-Swailem Am, Sulaimani R, Al-Meshari AA. Diabetes mellitus and impaired glucose tolerance in Saudi Arabia. Ann Saudi Med 1996; 16: 381-385.
30. Kastarinen MJ, Nissinen AM, Vartiainen EA, Jousilahti PJ, Korhonen HJ, Puska PM, Tuomilehto. Blood pressure levels and obesity trends in hypertensive and normotensive Finnish population from 1982 to 1997. J Hypertens 2000; 18: 255-262.
31. Reisin E, Frohlich ED, Messerli FH, Dreslinski GR, Dunn FG, Jones MM, Batson HM Jr. Cardiovascular changes after weight reduction in obesity hypertension. Ann Intern Med 1983; 98: 315-319.
32. Huang Z, Reddy A. Weight change, ideal weight and hypertension. Curr Opin Nephrol Hypertens 1999; 8: 343-346.
33. Hall JE, Brands MW, Hildebrandt DA, Mizelle HL. Obesity-associated hyperten-sion. Hyperinsulinemia and renal mechanisms. Hypertension 1992; 19 Suppl (1): 145-155.

34. Landsberg L. Obesity and the insulin resistance syndrome. Hypertens Res 1996; 19 Suppl (1): S51-55.
35. Rocchini AP. Insulin resistance, obesity and hypertension. J Nutr 1995; 125: 1718S-1724S.
36. Dustan HP. Mechanisms of hypertension associated with obesity. Ann Intern Med 1983; 98: 860-864.
37. Rocchini AP. Hemodynamic and cardiac consequences of childhood obesity. Ann NY Acad Sci 1993; 699: 446-456.
38. Weidmann P, de Courten Bohlen L. Insulin resistance, hyperinsulinemia and hypertension. J Hypertens Suppl 1993; 5: S27-S38.