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ORIGINAL ARTICLE

Gestational Trophoblastic Disease in Al-Thawra Hospital, Sana'a-Yemen

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Abstract:

Objective: To determine the incidence, types, complications, management and outcomes of gestational trophoblastic disease (GTD) in gynaecological and obstetric department in Al-Thawra Modern Hospital in Sana'a City during the period of one year.

Methodology: This is an observational descriptive study. All patients who diagnosed and registered as GTD during the period of the study (1st January to 31 of December 2011) were included in the study.

Results: During the study period, there were 13153 deliveries, 80 cases of them had gestational trophoblastic disease. The overall incidence of GTD was 1:164 deliveries. Forty women (50%) had partial mole, 35 women (43.75%) had complete mole, 3 women (3.75%) had choriocarcinoma and 2 women had invasive mole (2.5 %). With regard to methods of treatment 73cases (91.25%) Underwent suction evacuation, 5 cases (6.25%) underwent hysterectomy and 2 cases (2.5%) treated with hysterectomy. Of the 80 cases of GTD 9 cases (11.25%) developed persistent gestational trophoblast disease and received chemotherapy. Seven cases of them improved by receiving single agent chemotherapy (MTX), other two cases become resistant to (MTX), hysterectomy was done for both of them and received multi-agent chemotherapy, one of them improved and the other case not complete her follow up and came in advanced stage with lung and brain metastases then she died.

Conclusion: The study documented a hospital-based incidence of gestational trophoblast as 1:164. Partial hydatidiform mole was the most common type of GTD followed by complete hydatidiform mole chemotherapy is effective in treatment of persistent GTD.

1. Introduction

Gestational trophoblastic disease refers to a spectrum of abnormalities of trophoblast associated with pregnancy from benign hydatidiform mole through to the malignant invasive mole, choriocarcinoma, and the rare placental site/epithelioid trophoblastic tumor. Complete hydatidiform mole (CM) occurs in about one per 1000 pregnancies and partial hydatidiform mole (PM) three per 1000 pregnancies (1). Trophoblastic tissue normally shares certain characteristics with malignancies, such as the ability to divide rapidly, to invade locally, and occasionally to metastasize to distant site such lungs, so women diagnosed with hydatidiform mole should be registered with regional center for regular monitoring of human chorionic gonadotropin to check for onset of malignancy (2). They can be distinguished histological and genetically; CMS are diploid and nearly always androgenic in origin, where as PMS are triploid consisting of one maternal and two paternal sets of chromosomes (3). Both CMS and PMS secrete human chorionic gonadotropin (HCG), but their distinct histological and genetic identities lead to important differences in clinical behaviour, thus after termination of molar pregnancy, the risk of developing malignant disease, simply detected by a plateau or rising serum or urine concentration of (HCG), is 15% for a CMS and only 0.5% for PMS (1). All forms of GTD produce (HCG) (4). The amount of (HCG) correlates with disease volume and so monitoring this hormone is an accurate biomarker for screening, diagnosis, and therapeutic response and follow up of women with GTD (5).

Complications may end by hysterectomy and many cases become persistent received chemotherapy, hysterectomy may play several roles in the treatment of GTD, first it may be performed primarily to treat placental site trophoblastic tumor (6), epithelioid trophoblastic tumors, or chemotherapy-resistant disease .In addition, severe uncontrollable vaginal or intra-abdominal bleeding may necessitate hysterectomy as an emergency procedure (7). So, this descriptive study was conducted to investigate the proportion of GTD among obstetric cases, the presentations, the complications, and the different modalities of treatment.

2. Objectives

1) To determine the incidence of GTD in gynaecological and obstetric department in Al-Thawra Modern Hospital in Sana'a city during the period of one year.

2) To describe the GTD patients regarding the age, residence, parity, special habits, past history of similar disease and past history of abortion.

3) To estimate the percentage of patients developing persistent GTD and in turn in need further management with chemotherapy and follow up.

4) To determine the proportion of different categories of gestational trophoblastic (complete, partial moles, choriocarcinoma) in our hospital.

5) To determine the recurrence rate among the patient who presented with hydatidiform mole.

3. Patients and Methods

This observational, descriptive study was conducted at obstetric and gynaecological department in Al-Thawra Modern Hospital Sana'a city Yemen. Eighty patients who diagnosed and registered as GTD during the

period of the study (1st January to 31 of December 2011) were included in the study. All cases gave a verbal consent. Specially designed questionnaire form was filled for each patient by direct interview. The data requested includes demographic characteristic, obstetric and gynaecological history, history of prior molar pregnancy and vaginal bleeding then complete medical and gynaecological examination was done for each cases. For statistical analysis frequencies and percentage distribution was applied. The protocol of administering chemotherapy was as follows:

After discharge from hospital, the patients followed weekly by serum HCG till become negative, and then followed monthly by serum HCG for one year. Nine cases that developed persistent gestational trophoblast received chemotherapy. We started with monitoring hematologic, renal and hepatic indices. Before each cycle of chemotherapy we monitor serum HCG level weekly during therapy. All the 9 cases started with single chemotherapy, 4 days with methtrexate and 4 days with folonic acid, then rest for one week before started next course. Only two cases received multiagent chemotherapy, EMA/ CO:

First case was in single dose chemotherapy came with vaginal bleeding on examination metastatic in the vagina there was nodule in the vagina wall, and then hysterectomy was done. Multiagent chemotherapy EMA/CO continue till 3-time negative serum HCG and continue follow up for one year, she improved. The Second case did not complete her follow up and came in advance stage with lung and brain metastases and the she died.

Ethical Considerations

Ethical clearance was obtained for the study. Written informed consent was obtained from the participants. Confidentiality of data was preserved.

4. Results

During the study period from 1st January to December 2011, there were 13153 deliveries, 80 cases of them had gestational trophoblastic disease. The overall incidence of GTD was 1:164 deliveries. Distributed as the following: 40 women were diagnosed as have partial mole (50%), 35 cases (43.75%) were have complete mole, 3 cases choriocarcinoma (3.75%) and 2 cases invasive mole (2.5%) (Figure 1).

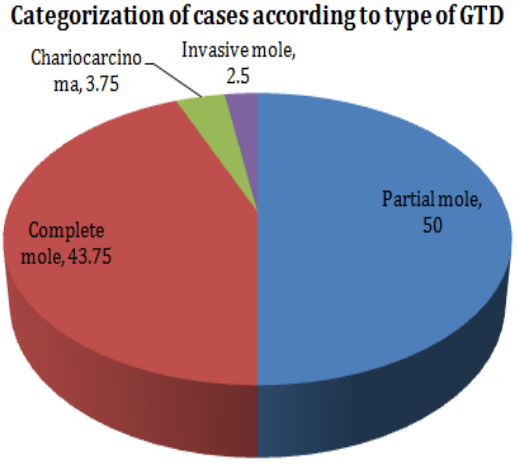

Figure 1. Categorization of cases according to types of GTD

According to residence, the highest group came from a rural area 44 cases (55%). That is because Al-Thawra Hospital is a referral facility and because of the limited health care services in rural medical centres, the remaining 36 cases (45%) were from urban areas either from Sana'a or other major cities in Yemen.

The age of the patients ranged from 16-50 years, an important percentage (36.25) of GTD was found among age group 21-30 years. The third group was between 31- 40 years, 20 cases (25%). The age group more than 40 years was 6 cases (7.5%) this age of less pregnancy but with significant morbidity regarding the GTD. With regard to the parity, the highest percentage (56.25 %) was in women who have 1-5 deliveries. With regard to the distribution of patients according to their special habits 37 of patients chew Qat (46.25%). Regarding the smoking habit, 20 cases (25%) use habble bubble (Table 1).

Table 1. Demographic characteristics and special habits of cases

Characteristic	Number (%)
Age	
<20	25(31.25)
21-30	29(36.25)
31-40	20(25%)
> 40	6(7.5%)
Parity	
Nulipara	18(22.5%)
1-5	45(56.25)
>5	17(21.25)
Special habits	
a- Smoking	
Yes	20(25%)
No	80(75%)
b-Chewing Qat	
Yes	37(46.25%0
No	43(53.75%)
Blood group	
O	40(50%)
A	33(41.25%)
B	5(6.25%)
AB	2(2.5%)
Total	80(100.0%)

During the study we note the highest percentage of women with GTD came complaining of vaginal bleeding (N: 58; 72.5%), while 12 women (15%) came for antenatal care, diagnosed during routine ultrasound scan and confirmed by histopathology. Six cases (7.5%) came with hyperemsis gravidarum and 3 cases (3.75%) have hyperthyroidism, which represent 8.6% of complete mole. Only one case during the study have preeclampsia at 18 weeks pregnancy there percentage was (1.25%) (Figure 2).

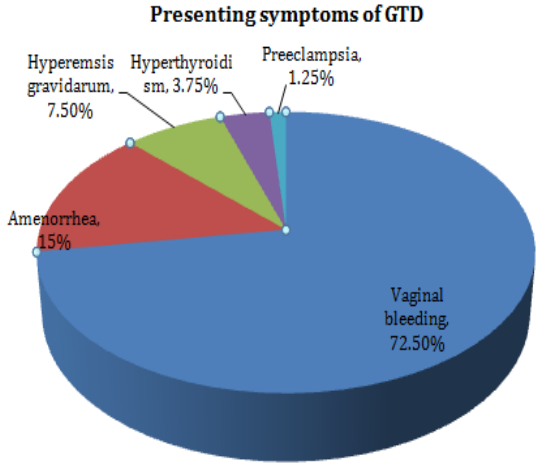

Figure 2. Presenting symptoms of GTD

There were 10 women who required blood transfusion (12.5%). The majority of cases (84%) have high level of HCG. According to history of abortion there were 24 cases (30%) have history of abortions .Out of 80 women with GTD there were 6 cases of them have past history of GTD (7.5%)two cases of them received chemotherapy in the past. The study show there were three cases had family history of GTD. Regarding the distribution of patients according to their blood group forty cases (50%)have blood group O, followed by blood group A 33 cases (41.25%), then 5 cases (6.25%) blood group B, and two cases (2.5%) have blood group AB (table 1). With regard to the treatment, the vast majority (N: 73, 91.25%) underwent suction evacuation. 5 cases (6.25%) underwent hysterectomy and 2 cases 2.5%

treated with hysterotomy one due to haemorrhage and the other one due to partial mole (table 2). Nine out of 80 cases of GTD (11.25%) developed persistent gestational trophoblast disease and received chemotherapy. Seven cases of them improved by receiving single agent chemotherapy (MTX), other two cases become resistant to (MTX), hysterectomy was done For both of them and received multi-agent chemotherapy, one of them improved and the other Case not complete her follow up and came in advanced stage with lung and brain metastases then she died.

Table 2. Distribution of patients according to the type of treatment

Type of treatment	No. of cases
Suction evacuations	73(91.25%0
Hysterectomy	5(6.25%0
Hysterotomy	2(2.5%)
Total	80(100%)

5. Discussion

The present study that was carried out at Al-Thawra modern general hospital Sana'a from Jan to Dec 2011 showed that there were 80 cases diagnosed as gestational trophoblastic disease admitted to hospital.

The incidence in this hospital-based study is 1 in 164, which was higher in comparison to other countries like Saudi Arabia and Iraq, which was 1 in 452, 1 in 318 respectively (8, 9). While considered approximately near the incidence in Asia 1 in 200 (10). The incidence has great geographic variability. In the USA it occurs in approximately 1 in 1500 to 1 in 2000 pregnancies, in Southeast Asia up to 13 in 1000(11, 4). This can be explained by the lower estimation of the vaginal deliveries, because most women delivered at home. According to residence we found the highest group came from rural area 44 cases (55%) because our hospital is referral hospital and there are no facilities in rural medical centers.

The remaining 36 cases (45%) from urban areas either from Sana'a or other big cities in Yemen, with regard special habits, higher incidence of molar pregnancy seen in patients chewing Qat (46.25%).In 1990 there were study done in Abu-Dhabi revealed that there is increase risk of molar pregnancy in two ethnic groups the Asian and Gulf Arabs mainly in Omanis and Yemeni, because many Gulf Arabs chew the leaves of cathaedulis (Qat or Khat), the Asian chew betel nut mixtures, it is possible that Qat release mutagens into the saliva (12).

More than one third of the patients percent (36.25%) were in the age group (21-30), followed by age group<20 yrs (31.25%), this result corresponding to study occurs in South Africa at King Edward V111 Hospital which reveled that the mean age of patients was 28.5 yrs (13).

There is other study in Pakistan found that molar pregnancy found in (32.6%) in age group (21.30), followed by age group less than 20 yrs (22%) the two age group is the age of high peak of childbirth (14).

We noticed that one fifth of the (22.5%) of cases were nullipara and more than half (56.25%) were multipara<5 and (21.25%) in cases of more than Para 5, this result correlate with study done in Iraq between (2008-2009) reveled that (52.5%) occur in multipara <5 (25%) occur in multipara>5, followed by (22.5%) in nullipara (15)

From our study we found that female who have blood group O and A are more common

among the gestational trophoblastic disease patients, which represent (50%), (41.25%) respectively.

In study in Pakistan at 2004 the most frequent blood group was O and B (16). Other study in Italy found that blood group A was more common among gestational trophoblastic disease patients (17).

According to clinical presentation most of patients with gestational trophoblastic disease in our study present with vaginal bleeding (72.5%), (12.5%) of them required blood transfusion this corresponding to study in Saudi Arabia at 2001 which level that vaginal bleeding was the common presenting symptoms in (84%) of Cases (18), we found some result in study done in Pakistan between 2000-2004 (19) and in other study done in India between 1995- 1999 (20), while only (15%) of women came for antenatal care and diagnosed during routine ultrasound scan and confirmed by histopathology.

There were 6 cases (7.5%) came with hyperemsis gravidarum, 3 cases (3.75%) have hyperthyroidism Which represent 8.6% of complete mole and only one case with preeclampsia at 18 weeks pregnancy (1.25%).

Regarding history of abortion there were 24 cases (30%) have positive history 6 cases of them (7.5%) have past history of gestational trophoblastic disease, two cases of them received chemotherapy in the past of that 6 cases have only one previous molar pregnancy, two cases have two previous molar pregnancy and one have more than two previous molar pregnancy.

In the study in UK at 2010 found that after molar pregnancy, the risk of further complete and partial mole rise to 1-2% and after two molar pregnancies the risk of the third mole is 15-20% (14). Also we found 3 patients have family history of GTD that may be due to genetic or environmental factors.

With regard to methods of treatment 73 cases (91.25%) underwent suction evacuation five cases (6.25%) underwent hysterectomy and two cases (2.5%) treated with hysterotomy of 80 cases of GTD nine cases (11.25%) developed persistent GTD. Seven cases of them improved by receiving single agent chemotherapy (Methotrexate) the other two cases became resistant of MTX, hysterectomy was done for both and both received multi agent chemotherapy one of them improved but the other patient did not complete her follow up and came in advanced stage with lung and brain metastases then she died.

6. Conclusion

The study documented a hospital bases incidence of gestational trophoblast is 1: 164. The high proportion was in age group 21-30, among patients came from rural area, multiparous women, Blood O patient and patients who had previous abortion. Partial hydatid mole was the most common type of GTD followed by complete hydatidiform mole chemotherapy is effective in treatment of persistent GTD. The management of gestational trophoblast disease in our hospital not differs from that protocol found in other centers in the world, However, Follow up of patients is the real problem for both patients and doctors because no special center for GTD and no registration to patients in proper way.

7. Recommendations

1) Establishment of a center for registration, management and follow up the patients with GTD.

2) Measurement of serum HCG level must be done and available for all patients.

3) Antenatal care services should be improved and become available for all mother.

4) Education of patients about hazards which can occur to both mother and baby due to very early or late child birth.

5) The drugs, which use for treatment of persistent GTD and choriocarcinoma, should be available and good financial support should be given for those patients.

6) Improvement of patient records and registration in the hospital.

References

1. Seckl MJ, Sebire NJ, Berkowitz RA. Gestational trophoblastic disease. Lancet 2010, 376:717- 7

2. Sebire NJ Seckl Mj. Gestational trophoblastic disease: current management of hydatidiform mole.BMJ 2008, 337:453

3. Paradinas FJ, Fisher RA. Pathology and molecular genetics of trophoblastic disease. Curr Obstet 1995, 5: 6-12.

4. World Health Organization (WHO), Gestational Trophoblastic Disease. Tech. Rep ser No 692. Geneva, 1983

5. Felmate CM, Batrofi J. Fulop V, Goldstein DP, et al: Human Chorionic Gonadotropin FOLLOW UP IN PATIENTS WITH MOLAR PREGNANCY: a time for Revolution Obstet Gynecol 2003, 101(4):733-6

6. Palmieri C, Fisher RA, Sebire NJ, Lindsay L, et al.: Placental site trophoblastic tumor arising from partial hydatidiform mole. Lancet 2005; 366 -688

7. Chao A, Lin CT, Chang TC, et al: Choriocarcinoma with diffuse intra-abdominal abscess and disseminated intravascular coagulopathy: a case report. J Repord. Med 2002, 47: 689

8. Al-Mulhim AA. Hydatidiform mole: a study of 90 cases. J Family Community Med.2000 Sept; 7(3): 57-61

9. Shahla Karim Al Alafi, Demon Ibrahim Omer. Prevalence and clinical Observation of Gestational Trophoblastic Diseases in Maternity Teaching Hospital in Erbil City. WSEAS TRNSACTION on BIOLOGY and BIOMEDICINE, 2010, 7(3): 190-9

10. Loukovara M, Pukkala E, Lehtovirta P, Leminen A. Epidemiology of hydatidiform mole in Finland, 1975 to 2001. Eur J Gynecol Oncol 2005; 26 (2): 207-8

11. Tham BW, Everard JE, Tidy JA, Drew D, Hancok BW, Gestational Trophoblastic Disease in the Asian population of Northern England and North Wales. BJOG 2003; 110:555-9

12. Graham I H, Fajardo AM, Richards RL. Epidemiological study of complete and partial hydatidiform mole in Abu Dhabi: influence of maternal age and ethnic group. J Clin Pathol 1990,43:661-4

13. Moodley M, Tunkyl K, Moodley J. Gestational Trophoblastic Syndrome: an audit of 112 patients. A South African Experience. Int J Gynecol Cancer 2003; 13 (2):234-9

14. Sadiq Saleem, Panjwani Suchita. Gestational trophpblastic disease experience at the basic medical sciences institute, JPMC, Karachi. Pak J med. Sci 2006, 22 (4): 483-5

15. Rauf B,Hassan L, Ahmed S. Management of Gestational Trophoblastic Tumors: A Five–Year Clinical Experience. J Coll Physician Surge. Pak. 2004,14 (9): 540-9

16. Lavacchia C, Francechi S, Parazzini S,Fasoli M, Decarli A, et al. Risk factors for gestational trophoblastic disease in Italy .Am J Epidiol .1985 , 121 (3): 457-64

17. Soto Wright V, Bernstein M, Golstein DP, Berkowitz RS.The changing clinical presentation of complete molar pregnancy. Obstetric Gynecology; 86 (5), 2001, 775-9

18. Qurban Ali Bugti, Nassema Baloch, Mohammed Aslam Baloch, Gestational Trophoblastic Disease in Quetta. Pak J Med Res 2005, 44 (2): 92-5

19. Kumar N, Saxena YK, Rathi AK,Chitra R ,Kumar P. Host and risk factors for gestational trophoblastic disease :A hospital-based analysis from India. Med. Sci Monit. 2003, 9 (10): 442-7

20. Seckl MJ, Fisher RA, Salerno GA, Rees H, et al: Choriocarcinoma and partial hydatidiform moles. Lancet 2000,356: 36-9

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ORIGINAL ARTICLE

Serum Testosterone Level in Hyperlipidemic Yemeni Individuals

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Abstract:

Background and Aim: Severe obesity may cause hypogonadism in men. Our study aims to study the relation between hyperlipidemia and serum level of total testosterone.

Methods: Serum concentrations of total testosterone, triglyceride and cholesterol were determined in thirty five healthy male individual which were divided into two groups the test group which includes 14 individuals with high lipid profile while the control group includes 21 individuals with normal lipid profile.

Results: Significant reduction ($p \leq 0.05$) in serum testosterone level in obese and hypertriglyceridemic individuals (4.98 ± 1.72) and (5.47 ± 1.85) when compared with control group (6.86 ± 2.30) respectively.

Conclusion: This study concludes that obesity and hypertriglyceridemia reduce serum testosterone level.

Key words: Serum testosterone, obesity, hyperlipidemia

1. Introduction

Severe obesity may cause hypogonadism in men (1-6).The first reports describing this phenomenon suggested that the reduction in total testosterone was mainly the result of a decrease in sex hormone-binding globulin (SHBG) (1-3). Later studies, however, demonstrated a significant decrease in total as well as free testosterone (4-7). Subsequently, adrenal androgens were also found to be decreased (8, 9). In addition to the decrease in androgen levels, there is an increase in serum estrogens. Both estrone and estradiol concentrations are significantly higher than in age-matched non-obese men (3, 5, 7). Our study objected to the relation between hyperlipidemia and serum level of total testosterone in healthy Yemeni individuals.

2. Subjects and Methods

Subjects: Thirty five healthy individual mean age was 28.5 ± 9.2 years, they were divided into two groups, the first group (test group) contain fourteen individuals had high lipid profile (plasma triglyceride and total cholesterol more than 200 mg/dl) and (twenty one) had normal lipid profile (control group) were included in the study. All subjects were asked for informed consent. Then fasting blood sample was collected for laboratory analysis from each subject.

Sample collection: All individual were subjected to collection of 5 ml fasting (12-h) blood sample and put into test tube without anticoagulant and left for 10 minutes after which the sample were centrifuged for 10 minutes at 4000 rpm for sera collection which stored at -21◦c for and used in essay of cholesterol, triglyceride and total testosterone.

Measurements of body mass index: Body mass index was calculated by the equation described by world health organization

$$BMI = \frac{\text{Weight (kg)}}{\text{Height}^2\,(\text{m}^2)}$$

A BMI between 25 and 29.9 is considered overweight, obesity start at 30>

Hormone measurements: A morning serum level of total testosterone was measured by enzyme immunoassay test which is based on the competitive binding between testosterone in test specimen and testosterone-horsedish peroxides (HRP), for a constant amount of rabbit anti-testosterone. In the incubation, goat anti-rabbit IgG-coated wells are incubated with testosterone standards, controls, patient sample, testosterone-HRP conjugate reagent and rabbit antitestosterone reagent for 90 min.

During the incubation, a fixed amount of HRP-labelled testosterone competes with the testosterone in the standard, sample or quality control serum for a fixed number of binding sites of the specific testosterone antibody. Thus, the amount of testosterone-HRP immunologically bound to the well progressively decreases as the concentration of testosterone in the specimen increase. Unbound testosterone-peroxidase conjugate is then removed and the wells washed, followed by addition of tetranethylbenzidine (TMB) solution resulting in development of blue color which is measured spectrophotometrically at 450 nm (10).

Lipid profile measurements: Serum triglycerides was measured according to the method described by Giovanni, et al. (11). Triglycerides in the sample incubated with lipoprotein lipase (LPL) liberate glycerol and free fatty acids. Glycerol is converted to glycerol-3-

phosphate (G3P) and adenosine -5- diphosphate (ADP) by glycerol kinase and ATP. Glycerol-3-phosphate (G3P) is then converted by glycerol phosphate dehydrogenase (GPD) to dihydroxyacetone phosphate (DAP) and hydrogen peroxide (H2O2). Hydrogen peroxide (H2O2) reacts with 4 aminophenazone (4-AP) and p-chlorophenol in presence of peroxidase enzyme (POD) to give a red colored dye. Then intensity of color formed is proportional to the triglyceride concentration in the sample.

Serum cholesterol was measured according to the method described by Franco et al. (12). Cholesterol esterase hydrolyzes cholesterol ester to free fatty acids and free cholesterol which oxidized by cholesterol oxidase to Cholest-4en-3one (ketone) and hydrogen peroxide (H2O2). Peroxidase enzyme catalyzes the reaction between hydrogen peroxide and 4-aminophenazone in the presence of phenol to produce the color dye quinonimine and water. The intensity of the color formed is proportional to cholesterol concentration in the sample.

Statistical analysis: Mean and standard deviations of all parameter in each groups was calculated by SPSS version 18. Data were compared using the independent samples t- Test with P value $\leq$ 0.05 considered statistically significant.

Ethical Considerations

Ethical clearance was obtained for the study. Written informed consent was obtained from the participants. Confidentiality of data was preserved.

3. Results

This study involved 35 male individuals. Their age arranged between 21 and 46 with main 28.5± 9.2 years and their BMI arranged between 17.8 and 43.2 with main 25.7 ± 4.5. Figure 1 and 2 show the BMI and lipid profile in the test and control group.

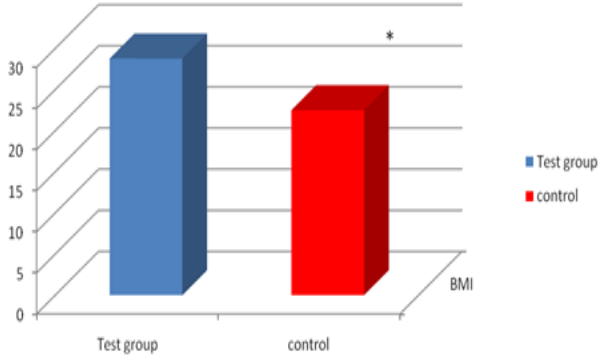

Figure 1. BMI of test group (hyperlipidemic group) and control group

*Significant difference at p value less than 0.05

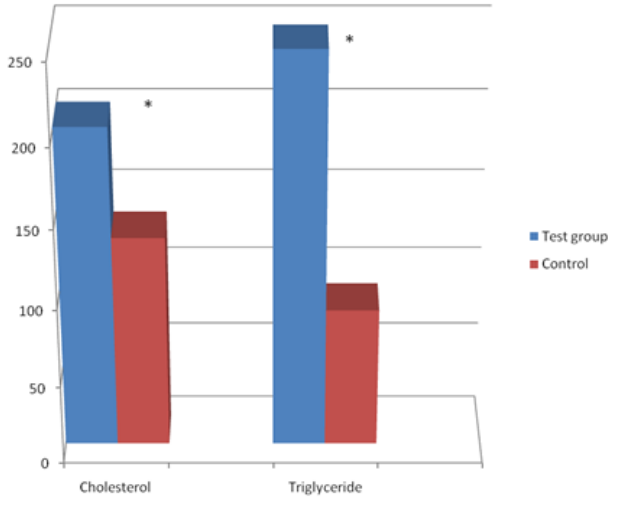

Figure 2. level of serum triglyceride and total cholestrol (mg/dl) in test group(hyperlipidemic group) and control group

*Significant difference at p value less than 0.05

The study Show reduction in serum level of total testosterone in hyperlipidemic group (5.47 ± 1.85) when compared with control group (7.04 ± 1.83) was statically significant ($p=0.026$) (Table 1). Furthermore, table 2 shows a negative correlation between serum levels of total testosterone and BMI, triglycerides and cholesterol. This correlation was statically significant with BMI and triglycerides while insignificant with cholesterol

Table 1. Serum level of total testosterone in control group and Hyperlipidemic group

	Hyperlipidemic group	Control group	P value
Serum level of total testosterone	5.47 ± 1.85	7.04 ± 1.83	0.026*

*Significant difference at p value less than 0.05

Table 2. Correlation between serum levels of total testosterone and BMI, triglycerides and cholesterol

	Serum level of total testosterone	
	R	P value
BMI	- 0.514	0.002*
Serum Triglyceride	- 0.445**	0.007*
Serum Cholesterol	- 0.255	0,139

*Significant difference at p value less than 0.05

4. Discussion

Infertility is a reproductive health problem which affects 10 to 15 percent of couples. Male infertility causes 50 percent of the cases (13-15) and it represents approximately 55% of infertility among Yemeni population (16). Obesity is one of the most important risk factor of infertility (17). This study indicates high significant negative correlation between BMI and serum total testosterone levels (table 2). This agrees with de Boer et al. (18) who reported that severe obesity decreases level of serum testosterone. Decrease of body weight increased in androgen level and experienced an improvement in semen parameters (19-21).

In our study, significant reduction in serum total testosterone in hyperlipidemic group was observed when compared with control group (table 1). Furthermore, table 2 shows significant negative correlation between serum total testosterone level and triglyceride. This agrees with Zmuda et al. (22) who showed that a decrease in endogenous testosterone is associated with an increase in triglycerides. Decreased serum triglyceride stimulated by lipoprotein and hepatic lipase increases serum total testosterone level (23).

The decrease in testosterone level may be due to conversion of testosterone in peripheral tissue to estrogens. de Boer et al (18) reported that sever obesity decrease level of serum testosterone and returns to the normal level after treatment with letrozole (aromatase inhibitor) for 6 weeks. Presence of estrogen receptors in the male hypothalamus has indicated that estrogen might contribute to low testosterone levels through a negative feedback mechanism. Estrogen acts on the hypothalamus to negatively regulate the release of pulses of gonadotropin releasing hormone (GnRH) as well as the release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the anterior pituitary gland, and estrogen agonists have been shown to have an inhibitory effect on androgen biosynthesis (24).

Hypercholesterolemia, a major risk factor for atherosclerosis, is a complex disorder associated with genetic and environmental factors. More and more epidemiological data has demonstrated that low testosterone concentrations in men are associated with a higher risk of atherosclerosis (25-29). Our results showed insignificant negative correlation between testosterone and total serum

cholesterol level (table 2). Feng et al. reported that testosterone levels in serum, testis, and liver were lower in hypercholesterolemic mice than those in normal mice. The decline was significant in testis and liver testosterone while insignificant in serum testosterone levels and suggests an adverse effect on testosterone biosynthesis (30).

5. Conclusion

In this study, evaluation of the serum testosterone level among hyperlipidemic Yemeni individuals and matched control group was carried out. Serum testosterone level significantly reduced among hypertriglyceridemic and overweight individual groups. More studies should evaluate the real effect of hypercholesterolemia on testosterone level.

Acknowledgment

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References

1. Amatruda J., Harman S., Pourmotabbed G. and Lockwood D. Depressed plasma testosterone and fractional binding of testosterone in obese males. J Clin Endocrinol Metab. 1978 (47):268 -71
2. Glass A., Swerdloff R., Bray G., Dahms W. and Atkinson R. Low serum testosterone and sex-hormone-bindingglobulin in massively obese men. J Clin Endocrinol Metab. 1977 (45):1211-9
3. Schneider G, Kirschner M., Berkowitz R. and Ertel N. Increased estrogen production in obese men. J Clin Endocrinol Metab. 1979 (48):633-8
4. Strain G., Zumoff B., Kream J. et al. Mild hypogonadotropic hypogonadism in obese men. Metabolism. 1982 (31):871-5
5. Zumoff B. Hormonal abnormalities in obesity. Acta Med Scand. 1988 (723):153-60
6. Zumoff B., Strain G., Miller L., Rosner W., Senie R., Seres D. and Rosenfeld R. Plasma free and non-sex-hormone-binding globulin-bound testosterone are decreased in obese men in proportion to their degree of obesity. J Clin Endocrinol Metab 1990 (71):929-31
7. Giagulli V., Kaufman J. M. and Vermeulen A. Pathogenesis of the decreased androgen levels in obese men. J Clin Endocrinol Metab. 1994 (79):997-1000
8. Tchernof A, Despre´s JP., Belanger A. et al. Reduced testosterone and adrenal C19 steroid levels in obese men. Metabolism. 1995 (44):513-9
9. Tchernof A, Labrie F., Belanger A. and Despre´s J. Obesity and metabolic complications: contribution of dehydroepiandrosterone and other steroid hormones. J Endocrinol. 1996 (150):S155-S64
10. Wilson K, and Walker J. practical biochemistery principles and techniques. 4th ed. Cambridage: Cambridage univeristy press 1995
11. Giovanni B, Juanlto Y. and Ting Y. Mechanized Enzymatic Determination of Triglycerid. Cli chem. 1975 (21):420-4
12. Franco G, Crescenzio I., Giuseppe M. and Enzo M. Enzymic Determination of Cholesterol in High-Density Lipoprotein Fractions with a Sensitive Reagent. Cli chem. 1981 (27):375-9
13. Dublin L. Amelar RD 1971. Etiologic factors in consecutive cases of male infertility. Fertil Steril 1294 (22): 469-74
14. Hendry WE, Sommerville IF, Hall RR, Pugh RC. Investigation and treatment of the subfertile male. Br J Urol 1973 (45): 684-92
15. Zhou-Cun A, Yuan Y, Si-Zhong Z, Zhang W, Lin L. Chromosomal abnormality and Y chromosome microdeletion in Chinese patients with azoospermia orsevere oligozoospermia. Acta Genetica Sinica, 2006 (33): 111-6
16. Jibrel SO. Is infertility preventable? Some of the preventable aspect of infertility in yemen. Yemeni Med Science J 2001; 1:64-71
17. Nguyen RH, Wilcox AJ, Skjaerven R, Baird DD. Men's body mass index and infertility. Hum Reprod. 2007 (22):2488-93
18. De Boer H, Verschoor L., Ruinemans-Koerts J. and Jansen, M. Letrozole normalizes serum testosterone in severely obese menwith hypogonadotropic hypogonadism. Diabetes, Obesity and Metabolism. 2005 (7): 211-5
19. Kasturi S. S., Tannir J. and Brannigan R. E. The metabolic syndrome and male infertility. J. Androl. 2008 (29):251-9
20. Isidori, A. M. et al. Leptin and androgens in male obesity: evidence for leptin contribution to reduced androgen levels. J. Clin. Endocrinol. Metab. 1999 (84):3673-80
21. Chavarro J. E., Toth T. L., wright D. L., Meeker J. D. and Hauser R. Body mass index in relation to semen quality, sperm DNA integrity, and serum reproductive hormone levels among men attending an infertility clinic. Fertil. Steril.doi:10.1016/j.fertnstert.2009.01.100

22. Zmuda JM, Cauley JA, Kriska A, Glynn NW, Gutai JP, Kuller LH Longitudinal relation between endogenous testosterone and cardiovascular disease risk factors in middle-aged men. A 13 year follow-up of former multiple risk factor intervention trial participants. Am J Epidemiol 1997 (146):609-17

23. Breier C, Drexel, H. et al. Essential role of post-heparin lipoprotein lipase activity and of plasma testosterone in coronaryheart disease. Lancet. 1985 (2):1242-4

24. Hammoud A. O., Gibson M., Peterson C. M., Hamilton B. D. and Carrell D. T. Obesity and male reproductive potential. J. Androl. 2006 (27):619-26

25. Foreman M. Cardiovascular disease: a men's health hazard. Nurs Clin. 1986 (21):65-73

26. Kalin MaZ, B. Sex hormones and coronary disease: a review of the clinical studies. Steroids. 1990 (55):30-52

27. Alexandersen P, Haarbo J. and Christiansen C. The relationship of natural androgens to coronary heart disease in males. Atherosclerosis. 1996 (125):1-13

28. Hak A, Witteman J., de Jong F. et al. Low levels of endogenous androgens increase the risk of atherosclerosis in elderly men. J Clin Endocrinol Metab 2002 (87):3632-9

29. Rosano G, Sheiban I., Massaro R. et al. Low testosterone levels are associated with coronary artery disease in male patients with angina. Int J Impot Res. 2007 (19):176-82

30. Feng Y, Zhu Y., Chen X. et al. Effects of diet-Induced hypercholesterolemia on testosterone-regulated protein expression in Mice Liver. Journal of Nanoscience and Nanotechnology. 2005 (5):1-4

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ORIGINAL ARTICLE

Relationship of Body Mass Index with Lipid Profile among Teaching Staff at the Higher Institute of Health Sciences, Sana'a

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Abstract:

Background: Obesity is a global disease; however there is a few documents about obesity, while there is no published data about the association of body mass index (BMI) with lipid profile among Yemeni people.

Aim: The aim of this study was to determine the association of BMI with lipid profile among teaching staff in the Higher Institute of Health Sciences, Sana'a city, Yemen.

Methods: Cross-sectional study was conducted in the Higher Institute of Health Sciences, Sana'a, 103 teaching staff aged 21-59 years (49 men and 54 women) were recruited in this study. The weight, height, waist and blood pressure of subjects were measured and BMI was calculated. The plasma lipid profile; Low-density lipoprotein cholesterol (LDLc), high-density lipoprotein cholesterol (HDLc), total cholesterol (TC) and triglycerides (TG) were measured. Informed consent was obtained from each subject.

Results: The BMI was positively correlated with TG, waist, systolic and diastolic blood pressure (P = 0.025; <0.001; 0.002; 0.002, respectively). This association was pronounced among women subjects than men particularly, the systolic and diastolic blood pressure (P= 0.012 and 0.008, respectively). The mean of waist, systolic and diastolic blood pressure of overweight, and obese subjects were higher than normal, (P = <0.001; 0.006; 0.009, respectively).

Conclusion: BMI was positively correlated with cardiovascular risk factors; TG, waist, and blood pressure, thus overweight and obese are more susceptible to cardiovascular diseases than normal BMI subjects.

Key words: Body mass index, blood pressure; triglyceride; overweight; obese

1. Introduction

Obesity is one of the most important metabolic diseases worldwide which has become widely prevalent in the recent two decades (1). Obesity has many-fold reasons and, considering genetic factors as the sole cause of obesity prevalence does not seem to be logical. Reduction in physical activity and using high-calorie foods increase excessive energy input of the body (2). The International Obesity Task Force estimates that at present at least 1.1 billion adults are overweight; including 312 million who are obese (3). The prevalence of overweight and obesity is in increasing. Obesity is now estimated to be the second leading cause of mortality and morbidity, causing an estimated 2.6 million deaths worldwide and 2.3% of the global burden of disease (4). There is an overall consensus that obesity poses a significant risk for the development of cardiovascular disease, alterations in glucose metabolism and reduces life expectancy (5).

In recent years, BMI has become the medical standard used to measure over weight and obesity. This is a measure of how appropriate person's weight is for his/her height (6). BMI was calculated as weight in kilograms divided by height in meters squared as indicated by the World Health Organization (7). Association of lipid profiles with obesity and BMI has been reported (8, 9). Waist circumference is increasingly being accepted as the best anthropometric indicator of abdominal adiposity and metabolic risk (10).

Heart disease is the number one cause of death; risk factors include high cholesterol, high triglycerides, low HDL-C, diabetes, hypertension, gender, and cigarette smoking (11). A higher risk of coronary heart disease (CHD) has been associated with higher LDLc levels among adults. Meanwhile, a high HDLc level is usually protective. Lowering lipids through diet and/or lipid lowering drugs has been shown to reduce the incidence of atherosclerotic events (12). There is no published data about the association of body mass index (BMI) with lipid profile among Yemeni people.

2. Study Aims

The aim of this study was to determine the association between body mass index and the lipid profile among teaching staff in the Higher Institute of Health Sciences, Sana'a city, Yemen.

3. Subjects and Methods

Subjects

A cross-sectional study conducted among staff at the Higher Institute of Health Sciences, Sana'a city. The registered teaching staffs at Higher Institute of Health Sciences were 200; all of staffs were invited to participate in the study. The number of responding people was 106 (50 men's and 56 women's) aged 21-59 years. A consent form for blood collection was obtained from all subjects.

Methods

The weight, height, and waist circumference were measured. Weight was measured to the nearest 0.1 kg in light clothing and standing barefoot using a well calibrated balance scale. Height was measured to the nearest using a wooden meter fixed on the wall while the subject was standing relaxed, barefoot and heels together touching the wall. Waist circumference (WC) was measured with a flexible measuring tape at the point halfway between the lower border of ribs and the iliac crest in a horizontal plane (13). BMI was calculated as weight in kilograms divided by height in squared meters as indicated by the World

Health Organization (7). Blood pressure was measured by a trained staff member on the right arm, with the subject in the sitting position, using a standard mercury manometer after at least 5 min of rest.

Laboratory investigations

Fasting blood samples were taken during the anthropometric measurements. Lipid profile, total cholesterol (TC), Low-density lipoprotein cholesterol (LDLc), high-density lipoprotein cholesterol (HDLc), and triglycerides (TG) were analyzed in Alolaqi Medical Lab Sana'a city by automated chemistry analyzer cobas c 311, (Roche Diagnostics, USA).

Statistical analysis

Statistical analysis was performed with the SPSS version 18. Pearson's correlation coefficient was used except in TG, the spearman correlation was used. The differences between groups were compared using one-way analysis of variance (ANOVA). TG was not normally distributed, therefore TG was log transformed and the results presented as geometric mean ± 95% confidence interval (CI). The statistical significance was set at the P value of less than 0.05.

Ethical Considerations

Ethical clearance was obtained for the study. Written informed consent was obtained from the participants. Confidentiality of data was preserved.

4. Results

The total participants were 106 teaching staff. Based on the value of BMI, subjects were classified as underweight (BMI < 18.5 kg/m2), normal weight (BMI 18.5–24.9 kg/m2), overweight (BMI 25–29.9 kg/m2) and obese (BMI ≥ 30 kg/m2) (7). However, there were 3 subjects were underweight thus were excluded from the study thus 103 (49 men's and 54 women's) aged 21-59 were included. Anthropometric and biochemical analysis of the subjects included in this study are shown in table 1.

Table 1. Anthropometric and biochemical analysis of the subjects

	Mean	Std. Deviation
Age (years)	35.6	8.0
Weight (kg)	66	11.8
Height (cm)	160.4	8.1
BMI (Kg/m2)	25.7	4.6
Cholesterol (mg/dl)	173	32.3
HDL (mg/dl)	45	12.6
LDL (mg/dl)	114	32
TG (mg/dl)	135	71
Waist (cm)	86.4	10.4
Systolic B.P	107	12
Diastolic B.P	73	10

BMI was positively correlated with TG, waist, systolic and diastolic blood pressure (P=0.025; <0.001; 0.002; 0.002, respectively), while BMI was not correlated with HDLc, TC, LDLc, and age (Table 2).

Table 2. Correlation of BMI with lipid profile, waist circumference, systolic and diastolic blood pressure and age

Parameters	R	*p* -value
Cholesterol (mg/dl)	0.168	0.089
HDL (mg/dl)	-.008	0.933
LDL (mg/dl)	.127	0.201
TG (mg/dl)	.221	0.025
Waist (cm)	.546	< 0.001
Systolic B.P (mm Hg)	.309	0.002
Diastolic B.P(mm Hg)	.299	0.002
Age	.059	0.557

R= Correlation coefficient

The results showed that BMI was positively correlated with waist in men and women subjects (P = <0.001; < 0.001, respectively) (Table 3). The association of BMI with systolic and diastolic

blood pressure was more pronounced among women than in men subjects (P = 0.012; 0.008, respectively). Furthermore, there was a borderline correlation of BMI with TG and age among women subjects (P = 0.084; 0.067, respectively).

Table 3. Correlation of BMI with lipid profile, waist circumference, systolic, diastolic blood pressure and age among men and women

	Men		Women	
Parameters	**r**	***p* -value**	**r**	***p* -value**
Cholesterol (mg/dl)	.208	0.151	.117	0.399
HDL (mg/dl)	-.151	0.299	.140	0.304
LDL (mg/dl)	.199	0.170	.031	0.822
TG (mg/dl)	.218	0.132	.237	0.084
Waist (cm)	.621	0.000	.492	0.000
Systolic blood pressure (mm Hg)	.268	0.063	.340	0.012
Diastolic blood pressure (mm Hg)	.291	0.130	.350	0.008
Age	-.113	0.376	.251	0.067

R= Correlation coefficient

The comparison of lipid profile, waist, systolic and diastolic blood pressure among subjects in relation to the BMI groups is shown in table 4. The mean of waist, systolic and diastolic blood pressure were higher in overweight and obese subjects compared to normal subjects (p= <0.001; 0.006; 0.009, respectively).

Table 4. Comparison of lipid profile, waist, systolic and diastolic blood pressure based on BMI

	Normal (18.5 - 24.9) n= 55	Overweight (25 - 29.9) n=32	Obesity(≥30) n= 16	P- value
Cholesterol (mg/dl)	167.5±31.3	179.9±35.4	179.4±27.2	0.161
HDL-C (mg/dl)	45.4±11.8	43.9±13.6	45.8±14.0	0.838
LDL-C (mg/dl)	111.0±31.5	116.7±35.2	121.9±27.4	0.443
Triglyceride (mg/dl)	107 (93-123)	132(109-159)	134(111-161)	0.108
Waist (cm)	81.5±8.0	89.9±8.0	96.4±12.2	0.000
Systolic blood pressure (mm Hg)	104.4±11.3	108.3±11.8	115.3±13.6	0.006
Diastolic blood pressure (mm Hg)	71.1±10.5	74.5±8.5	79.4±7.9	0.009

The data is presented as mean ± SD except TG was presented as geometric mean ± 95% confidence interval (CI).

5. Discussion

In the present study, the association of BMI with lipid profile, waist, systolic and diastolic blood pressure was evaluated among teaching staff at Higher Institute of Health Sciences, Sana'a city, Yemen. There was a positive correlation between BMI and TG, waist, systolic and diastolic blood pressure whereas there was no correlation between BMI and LDLc, HDLc, TC. This study is in agreement with previous studies (14, 15, 16). However, our results were in disagreement with Al-Ajlan study among Saudi people (17). This controversial data may be due to subjects characterization, in Al-Ajlan study, the subjects were men students in college aged 18-35 years and most of them are young (mean age 20.2±2.9) and he included the underweight subjects. While in our study the subjects were men and women aged 21-59 years (mean, 35.6 ±8.0) and the underweight subjects was excluded from our study due to a very small number (3 subjects). Being overweight or obese can lead to adverse metabolic effects on blood pressure, cholesterol and triglycerides (18). Free fatty acids (FFA) are released in abundance from adipose tissue mass. As a consequence, FFA increases the liver production of TG and secretion of VLDL. Hypertriglyceridaemia and VLDL reduce HDL cholesterol (19). Circulating FFA, may contribute to the induction of hypertension (19).

The association of BMI with lipid profile, age, and waist, systolic and diastolic blood pressure was further analysis among men and women subjects. The association of BMI with systolic and diastolic blood pressure was more pronounced in women than men. Women were more likely than men to have hypertension, low levels of HDL cholesterol and high levels of triglycerides (20).

Similar findings were also reported by Al-Ajlan among Saudi people (17) and Ali chehrei in Iranian adults (21). Our results were in contrast with that of Ugwuja, et al (22) and Abubakar et al (23) in Nigerian women. This contrast may be due to subjects characterizations, the Abubakar et al. subjects, were women and younger (19-32 years) while in our study the subjects both men and women and the age (35.6±8.0 years)

The mean of waist, systolic and diastolic blood pressure were statistically significant among different groups of BMI. Abubaker et al reported that, there were significant differences in systolic and diastolic blood pressure according BMI groups (23).

The limitation of this study was that the data were sampled from only one area, so there was some limitation in generalization of results.

6. Conclusion

BMI was positively correlated with cardiovascular risk factors, TG, waist, and blood pressure. This association was more pronounced among women than men particularly systolic and diastolic blood pressure. The systolic and diastolic blood pressure was higher among obese subjects compared to normal subjects.

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References

1. Garrow, JS. Obesity. In: Garrow J, James W, Ralph A. Human Nutrition and Dietetics, London, Churchill Livingston 2000.
2. Dai S, Eissa MA, Steffen LM, Fulton JE, Harrist RB, Labarthe DR. Clinical lipidology 2011;6: 235-44.
3. Haslam DW, James WP. Obesity. Lancet 2005; 366:1197-1209.
4. Ezzati M, Martin H, Skjold S, Vander Hoorn S, Murray CJ. Trends in National and State-Level Obesity in the USA after correction for self-report bias Analysis of Health Surveys. J R Soc Med 2006; 99:250-7.
5. Eyre H, Kahn R, Robertson RM. Preventing cancer, cardiovascular disease, and diabetes: a common agenda for the American Cancer Society, the American Diabetes Association, and the American Heart Association. Diabetes Care 2004; 27:1812-24.
6. Jafar TH Chaturvedi N and Papps G. Prevalence of Overweight and Obesity and their Association with Hypertension and Diabetes Mellitus in an Indo-Asian Population. CMAJ 2006; 175(9):1071-7.
7. World Health Organization. Report of a WHO Expert Committee. Physical status: the use and interpretation of anthropometry. Geneva 1995 (Technical Report Series No. 854).
8. Pihl E, Jurimae T. Relationship between body weight change and cardiovascular risk factors in men former athletes. Int J Obes Relat Metab Disord 2001; 25: 1057-62.
9. Bertolli A, Di-Daniele N, Ceccobelli M, Ficara A, Girasoli C, Lorenzoa, D. Lipid profile, BMI, body fat distribution and aerobic fitness in men with metabolic syndrome. Acta Diabetol 2003; 40 Suppl: 130-3.
10. Lemos-Santos MG, Valente JG, Goncalves-Silva RM, Sichieri R. Waist circumference and waist-to-hip ratio as predictors of serum concentration of lipids in Brazilian men. Nutrition 2004; 20: 857-62.
11. Syed Shahid Habib, Muhammad Aslam, Waqas Hameed. Gender differences in lipids and lipoprotein (a) profiles in healthy individuals and patients with type 2 diabetes mellitus. Pak J Physiol 2005; 1 (1-2).
12. Adams L. Hyperlipidemia. Minneapolis, MN center for leadership, Education and training in maternal and child nutrition, Division of epidemiology and community health, school of public health, university of Minnesota 2005; 10: 109-23.
13. Dalton M, Cameron AJ, Zimmet PZ, Shaw JE, Jolley D, Dunstan DW, Welborn TA, AusDiab Steering Committee. Waist circumference, waist-hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. J Intern Med 2003; 254:555-63.
14. Akpa Mr, Agomouh Dl, Alasia DD. Lipid profile of healthy adult Nigerians in Port Harcourt, Nigeria. Niger J Med 2006; 15: 137-40.

15. Kelishadi R, Alikhani S, Delavari A, Alaedini F, Safaie A, Hojatzadeh E. Obesity and associated lifestyle behaviors in Iran: Findings from the first national non-communicable disease risk factor surveillance survey. Public health Natr 2008; 11: 246-51.

16. Hajian-Tilaki KO, Heidari B. Prevalence of obesity, central obesity and associated factors in urban population aged 20-70 years, in the north of Iran. A population- based study and regression approach. Obes Rev 2007; 8:3-10.

17. Abdul Rahman Al-Ajlan. Lipid Profile in Relation to Anthropometric Measurements among College Men Students in Riyadh, Saudi Arabia. Int J Biomed Sci 2011; 7(2):112-9.

18. WHO. Diet, nutrition and the prevention of chronic diseases. Geneva 2003: World Health Organization.

19. Eckel, R. H., Grundy, S. M. & Zimmet, P. Z. The metabolic syndrome. Lancet 2005; 365 (9468): 1415-28.

20. Legato, M. J., Gelzer, A., Goland, R., Ebner, S. A., Rajan, S., Villagra, V, et al.. Gender-specific care of the patient with diabetes: review and recommendations. Gend Med 2006; 3(2):131-58.

21. Ali Chehrei, Saeid Sadrnia, Ammar Hassanzadeh Keshteli, Mohammad Ali Daneshmand , Jalal Rezaei. Correlation of dyslipidemia with waist to height ratio, waist circumference, and body mass index in Iranian adults. Asia Pac J Clin Nutr 2007; 16 (2):248-53.

22. Ugwuja EI, Ogbonna NC, Nwibo AN, Onimawo IA. Overweight and obesity, lipid profile and atherogenic indices among civil servants in abakaliki, south eastern Nigeria. Annals of Medical and Health Sciences Research I Jan-Mar 2013; Vol 3 Issue 1.

23. Abubakar AM, abruok MA, Gerie AB, Dikko AA, Aliyu S, Yusuf T, Magaji RA, Kabir MA, Adama UW. Relation of Body Mass Index with Lipid Profile and Blood pressure in Healthy Women of Lower Socioeconomic Group in Kaduna Northern Nigeria. AJMS 2009; 1(3): 94-6.

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ORIGINAL ARTICLE

Histopathological Profile of Colorectal Cancer in Yemen - An Eight Years Retrospective Study

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Abstract:

Objective: The aim of this retrospective study was to delineate the histopathologic profile of colorectal cancer (CRC) in Sana'a Yemen, and to investigate any changes in age, gender, and site of CRC and also attempt to find if there is a proximal or distal shift of CRC.

Methods: This is a descriptive record-based study of 437 cases of CRC diagnosed by 2 pathologists, in the Department of pathology, Sana'a University, Yemen, during the period from 1st January 2005 to the 30th December 2012. The diagnosis was made on hematoxylin and eosin stained sections and categorized the cases of colon malignancies according to histological type.

Results: Out of 437 cases of CRC, 178 (40.7%) were females and 259 (59.3%) were males. The commonest type of CRC was adenocarcinoma comprising 404 cases (92.4%). The female cases were 167 (41.3%) whereas the male cases were 237 (58.7%). Non-Hodgkin's lymphomas (NHL) accounted, for 25 (5.7%). The rest of the cases were three carcinoid, two undifferentiated, two leiomyosarcomas, and one gastrointestinal malignant epithelioid tumor. The mean age, for adenocarcinoma in females was 53.5 years and 55 years in males. In NHL the mean age was 29.9 years in females and 28 years in males. The maximum number of patients 103 (27%) were found in 51-60 years age group and the minimum number of patients 35 (9.3%) were seen in over 70 years age group. Patients aged below 41 years demonstrated a high frequency 78 (20.6%). Well-differentiated adenocarcinomas accounted for 65.3% followed by mucinous carcinoma 11.4%, and moderately differentiated 10.1%. The poorly differentiated and the signet ring cell carcinomas accounted for 3.5% each. Two hundred twenty-seven (56.3%) cases were located in the recto-sigmoid area. The least number 15 (3.5%) was found in transverse colon.

Conclusion: The present series clearly illustrated a significant resemblance of our data with these from developing countries regarding the early age of onset and left side subsite preponderance and less aggressiveness behaviour of cancer.

Key words: Colorectal cancer, adenocarcinoma, mucinous carcinoma, Yemen

1. Introduction

Colorectal cancer (CRC) has become one of the commonest malignancies and a major health concern worldwide. It is the third most common cancer in men and the second in women worldwide (1). According to the reports, every 9 minutes, someone dies from CRC (2). The frequency of CRC varies remarkably among different populations. The incidence is highest in developed countries and low in Asia, Middle East, South America, and Africa (3). Its incidence has decreased dramatically worldwide over the past decade due to implementation of screening programs. The causes for CRC range from germ line mutations of high penetrance genes such as the adenomas polyposis coli genes to a completely environmental risk factor such as excess body mass index at the other end. About 6% of all CRC are caused by the inheritance of muted genes with high penetrance (4). The environmental factors are largely dietary, particularly in terms of fats and animal protein, and their influence on the intestinal microflora and ultimately on the chemical composition of the intraluminal content (5). Life style and dietary factors are responsible for over two third of all CRC (4). The usual malignant tumor of the large bowel is a well-to-moderately differentiated adenocarcinoma secreting variable amounts of mucin (6). Approximately 50% of all carcinomas occur in the rectosigmoid area, although their relative incidence seems to be decreasing, in the sense that a shift in location toward the proximal colon during the past few decades has been noted (6). The recent global perception of a rising incidence of colorectal cancers necessitates detailed descriptions of the histolopathological patterns and variations. In Yemen no previous study has, been undertaken to document the histopathological patterns of colorectal cancer.

2. Study Aims

The aim of this retrospective study was to delineate the histopathological profile of CRC in Sana'a Yemen, and to find out any changes in age, gender, and site of CRC and also attempt to find if there is a proximal or distal shift of CRC.

3. Methods

A descriptive record-based study of 437 cases of CRC was carried out in the department of pathology, Faculty of Medicine and Health Sciences, Sana'a University, Sana'a Yemen, spanning a period of eight years from 1st January 2005 to the 30th December 2012. The diagnosis was made primarily in private laboratories of two consultant pathologists, in Sana'a which received the histopathologic biopsies from Sana'a and other Yemeni provinces. Most cancer patients are referred to Sana'a for further investigations and therapy, where most of the histopathologists and oncologists are practicing. The endoscopic biopsies and sections taken from the resected tumors were fixed in 10% formalin solution before being processed by manual and automatic tissue processor. These sections were stained with hematoxylin and eosin stain for routine histological diagnosis. During the histological study the sample was evaluated regarding the histological type, morphology and arrangement of cancer cells as well as grading of cancer. Finally the cancers were categorized into the main histological subtypes; adenocarcinoma, non-Hodgkin's lymphoma, and other rare types. The present study focuses on those cases subjected to colon and rectal endoscopic biopsies and total resection of cancer that revealed an invasive picture of cancer on light microscope, and excludes the doubtful cases of suggestive cancer and non-invasive neoplasms (carcinoma in situ). The clinical data needed for this study were

collected from the request forms and treated confidentially.

4. Results

A total number of 437 cases of CRC were studied. Two hundred and seventy eight cases were endoscopic biopsies and 159 cases were total resections of cancer. Out of the 437 cases, 178 (40.7%) were females and 258 (59.3%) were males with 1:1.45 female to male ratio. The commonest histological type of CRC encountered was adenocarcinoma comprising 404 (92.4%) of which female and male cases were 167 (41.3%) and 237 (58.7%) respectively; 1.4:1 male to female ratio. Non-Hodgkin's lymphomas account for 25 (5.7%). The rest of the cases were three carcinoid, 2 undifferentiated, 2 leiomyosarcomas, and one gastrointestinal malignant epithelioid tumor (Table 1).

Table 1. The gender distribution for CR malignancies based on histological subtypes

Types	Female		Male		Total	
	No	%	No	%	No	%
Adenocarcinoma	167	93.8	237	91.5	404	92.4
Non-Hodgkin's lymphoma	08	4.4	17	6.6	25	5.7
Carcinoid	01	0.6	02	0.8	03	0.7
Undifferentiated	-	-	02	0.8	02	0.5
Leiomyosarcoma	01	0.6	01	0.4	02	0.5
GIMET*	01	0.6	-	-	01	0.2
Total	178	100	259	100	437	100

*Gastrointestinal malignant epithelioid tumor
Female to male ratio: 1:1.5

The mean age for adenocarcinoma in females was 53.5 years with a median of 55 years whereas; the mean age in males was 58.4 years with a median of 55 years (age range in females was 18-85 and 17-95 in males). In NHL the mean age was 29.9 years in females and 28 years in males with a median of 33.5 years in females and 14 years in males (Table 2).

Table 2. Age distribution of adenocarcinoma and Non-Hodgkin's lymphoma

Type / Age years	Adenocarcinoma		Non Hodgkin's lymphoma	
	Female	Male	Female	Male
Mean	53.5	58.4	29.9	28
Median	55	55	33.5	14
Maximum	85	95	60	70
Minimum	18	17	03	02

Table 3 shows age distribution in five groups, the maximum number of patients (103 patients; 27%) were found in 51-60 years age group and the minimum number of patients (35 cases 9.3%) were seen in over 70 years age group. Patients aged below 41 years demonstrated high frequency 78 (20.6%). Well-differentiated adenocarcinomas accounted for 65.3% followed by mucinous carcinoma 11.4%, and moderately differentiated 10.1%. The poorly differentiated and the signet ring cell carcinomas accounted, for 3.5% for each one (Table 4).

Table 3. Differences in histological differentiation, for the adenocarcinoma (N= 404)

Gender / Degree of differentiation	Female		Male		Total	
	No	%	No	%	No	%
Well-differentiated	113	67.6	151	63.7	264	65.3
Moderately differentiated	19	11.4	22	09.3	41	10.1
Poorly differentiated	04	02.4	10	04.2	14	3.5
Mucinous	15	09	31	13	46	11.4
Signet ring cell	05	03	09	03.8	14	3.5
Papillary	03	1.8	01	0.4	04	1.0
Basaloid Carcinoma	-	-	01	0.4	01	0.2
Non-specified	08	04.8	12	5.1	20	5
Total	167	100	237	100	404	100

Female to male ratio: 1:1.4

Table 4. Age and sex distribution of patients with adenocarcinomas (N= 379)

Gender / Age group	Female		Male		Total	
	No	%	No	%	No	%
≤40	30	19.6	48	21.2	78	20.6
41-50	38	24.8	49	21.7	87	23
51-60	44	28.1	60	26.5	103	27
61-70	29	19	47	20.8	76	20
≥ 70	13	08.5	22	09.7	35	09.3
Total	153	40.4	226	59.6	379	100

* In twenty five cases, the ages were not mentioned

Table 5 depicts the topographic location of the CRC. Two hundred twenty-seven (56.3%) cases were located in the recto-sigmoid area. The least common site was transverse colon 15 (3.5%).

Table 5. Topographic distribution of adenocarcinoma and NHL

Type / Site	Adenocarcinoma		NHL*		Total	
	No	%	No	%	No	%
Rectum	161	40	2	8	164	38.1
Sigmoid	66	16.3	-	-	66	15.4
Descending colon	23	05.7	1	4	24	5.6
Transverse colon	15	03.7	-	-	15	3.5
Ascending colon	37	09.2	1	4	38	8.8
Cecum	26	06.4	15	60	41	9.5
Not-specified	76	18.7	6	24	82	19.1
Total	404	100	25	100	430	100

* NHL-Non-Hodgkin's lymphoma

5. Discussion

Colorectal cancer is a major cause of morbidity and mortality, being one of the most common malignant tumors in the world and the third leading cause of cancer related deaths in the US (7). Among both males and females the lowest rates of CRC incidence were observed for registries in Yemen (Age-specific incidence rates ASR: 4.3 per 100,000), India (ASR: 4.3), Egypt (ASR: 4.6), and Pakistan (ASR: 4.9) (1). The majority of registries with the high incidence rates of CRC were located in developed countries such as Canada (ASR; 45.4), Germany (ASR: 45.2) and Australia (ASR: 46) (1). In the less economically developed countries of the world, like Yemen, low CRC incidence rates may be due to the maintenance of a diet rich in fruits and vegetables as well as engaging of the people in occupations requiring greater physical activity. Colorectal cancer incidence has decreased dramatically worldwide over the past decade due to implementation of screening programs, but there is an observed variation in the age of diagnosis between different parts of the world (8). The adenocarcinoma arising from colonic epithelium was the commonest type of CRC in all age groups and in both sexes. It accounts, for 404 (92.4%) in this report. This result was compatible with several researches carried out in the Asian countries (9-10). Previous reports from the same continent and from Africa have showed lower figures (9, 12, 13). The authors demonstrated low percentages of adenocarcinoma were considered mucinous and signet ring cell carcinomas as separated entities. In our study adenocarcinoma includes all subtypes that arise from epithelial tissue. Our findings showed high frequency of NHL 5.7%, which nearly similar to that reported from Iran and Nigeria (10, 13). Involvement of the large bowel by lymphoma is rare in comparison to the small intestine and stomach. In Western countries primary NHL accounts, for 0.1% to 0.5% of all malignant tumors of the colon and rectum which make it the third most common large bowel malignancy after adenocarcinoma and carcinoid (14). The cecum is the most frequent location for NHL in our report and worldwide, probably due to the larger amount of lymphoid tissue in this

segment. The lower frequency of CRC in women compared to men in our report (M:F ratio 1.45:1) and worldwide may be due to slower reaction of certain risk factors associated with CRC such as smoking, alcohol consumption and obesity. However, CRCs are thought to be more heavily influenced by visceral abdominal fat that men tend to accumulate more if compared with women in whom subcutaneous fat is more common (15). Male patients were more likely to be older than the females, with mean ages of 58.4 and 53.5 years, respectively. One report from South of Yemen – Aden by Hamid GA et al. in 2012 showed a slight similar mean age at presentation, 48.8 years for females and 56.4 years for males (16). Cancer incidence report from Saudi Arabia documented a median age at diagnosis of 60 years for males and 55years for females (17). We observed that the mean age for adenocarcinoma was more than a decade earlier than that in the developed countries like US. As demonstrated in the current study, only 9.5% of carcinoma patients are over 70 year, whereas the corresponding figures for Western countries is much higher reaching up to 62% (18). This divergence can be due to differences in population structure and in life expectancies as well as early onset of the disease in developing countries. The important feature highlighted by this study is the resemblance of our data with those from developing countries regarding the early age of onset of CRC. In our report about, 20.6% of patients were in the younger age group, below the age of 41 year. Basaleem and Al-Sakkaf from Aden noted a relatively high proportion of early-onset, tumor (19.3% of cases were < 40 years) (19). The high incidence of the disease at ≤ 40 years are more than one third of cases in Egypt (20) and 21.4% in Saudi Arabia (21), compared to 3.6% in USA (22). This fact should be borne in mind when the physician examines a young Yemeni patient with large bowel symptoms. At the same time, a higher prevalence of CRC in younger age group may be due to a delay in screening based on age limitations in the existing recommendation; anal digital examination at the age of 40 years, fecal occult blood testing every year starting at 50 year, and flexible sigmoidoscopy every 3-5 years from 50 years onward (23). However this recommendation was suitable for developed countries, and should be modified according to the ages of onset for developing countries. The predominance of left colon (sigmoidorectal region) over right colon in our study mimics the situation from Middle Eastern countries as well as many developing and developed countries (11, 16, 20, 21). The underling mechanisms promoting the development of distal colon cancers are unknown but may be related to the interplay between environmental and constitutional factors that change with advancing age including sex hormones, effect of bile acid, bowel transit time, bacterial flora, fiber intake and fat intake (24). The patients with adenocarcinoma in current study tend to have 65.3%, well differentiated, 10.1% moderately differentiated and 3.5 poorly differentiated tumors. Previous, report from Aden demonstrated 66% well to moderately differentiated adenocarcinoma (16). Other figure from Nigeria revealed well-, moderately, and poorly differentiated forms as 52.3%, 32%, and 14% respectively (13). Abu Salem O. (25) from Jordan has reported that 69% of cases suffer moderately differentiated, 17% poorly differentiated and 14% well differentiated tumors. The highly percentage of well differentiated type could be explained by a biologically less aggressive disease (11). Although the colorectal carcinomas grading is a worthwhile exercise because of its correlation with prognosis, it suffers from all the drawbacks of a subjective evaluation, especially when performed in biopsy material. However, in our series 63.6% cases were endoscopic biopsies. The heterogeneity of colorectal carcinomas and the lack of firm

guidelines for the grading of tumors exhibiting areas with different degree of differentiation may explain why some authors have found widely varying proportions of histologic grades when they compared the results from different laboratories and it may partly explain why only slight to moderate inter-observer agreement has been observed in reproducibility tests (26). Very frequently the establishment of the diagnosis of colorectal cancer is significantly delayed in developing countries. Screening and early detection (secondary prevention) are important in influencing the CRC incidence rates and patients outcome. The screening tests including fecal occult blood test and structural screening tests (e.g. Sigmoidoscopy and colonoscopy) may help on increase rate of detection. In Yemen our patients are diagnosed late, because many people who have colon problems like constipation, change in the bowel habit, and rectal bleeding consult general physicians who prescribe medications for long term and subsequently, some of these patients whose cause is cancer are diagnosed late. In addition to this a lower health awareness and cancer phobia can be contributing factors leading to delay in seeking medical care. The total number of CRC during the period of study was 437 cases which were confirmed by microscopic diagnosis. Many cases were not microscopically confirmed and they were not notified. This country is facing many health problems; as there are no internationally adopted screening programs for cancers, no National Cancer Registry Center, limited medical insurance coverage, and very limited availabilities of the expensive medications.

The drawback of our study is that there may be underreporting of data, as all patients may not report to the doctor and many more would die without a histological proof of the disease. Also, patients with a histological proof of metastatic deposits may not be investigated for a primary tumor in the colon.

6. Conclusion

In conclusion, the present series clearly illustrated a significant resemblance of our data with these from developing countries regarding the early age of onset and left side subsite preponderance and well-differentiated grade of less aggressiveness behavior. Further research is needed to identify the cause of this observation, and this may be a reason to implement more strict guidelines for colon cancer screening and to consider starting this at a younger age. This calls for a more organized nationwide approach focused on patient education that encourages and illustrates the importance of CRC screening programs.

References

1. Ferlay J, Shin HR, Bray F, Foman D, Mathers C, Parkin DM. Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008. Int J Cancer 2010; 127:2893-917.
2. Benson AB. Epidemiology, disease progress, and economic burden of colorectal cancer. Journal of Managed Care Pharmacy 2007; 13: supplement 5-18.
3. Parkin DM, Whelan SL, Ferlay L, Youn RJ. Cancer incidence in five continents (IARC Sci.Publ.No.143) Series. Lyon, International Agency for Research on Cancer 1997; 143:566-7.
4. Cunningham D, Atkin W, Lenz HJ (2010). Colorectal Cancer. Lancet 2010; 375:1030-47.
5. Levin B. Nutrition and colorectal cancer. Cancer 1992; 70:1723-26.
6. Rosai J. Large bowel tumors. In: Akerman's Surgical Pathology. 9th ed. New York: Mosby; 2004, 776-825.
7. American Cancer Society. Colorectal cancer facts & figures 2008-2010 Atlanta: ACS .2008; 1.
8. Mosli M, Al-Ahwal M. Colorectal Cancer in the Kingdom of Saudi Arabia: Need for Screening. Asian Pacific J Cancer 2012; 13:3809-13.
9. Rasul KI, Awidi AS, Mubarak AA, Al-Homsi UM. Study of Colorectal Cancer in Qatar. Saudi Med J 2001; 22:705-7.
10. Mahmodlou R, Mohammadi Payvand, Sepehrvand N. Clinical study Colorectal Cancer in Northwestern Iran. International Scholarly Research Network ISRN Gastroenterology Volume 2012, Article ID 968560, 4 pages.

11. Zeenaden AA, Saber MM, Seif-El-din IA, Frag SA. Colorectal Carcinoma in gharbiah district, Egypt: Coparison between the elderly and non-elderly. Journal of Solid Tumors 2012; 2: 13-23.

12. Li M, Gu J. Changing patterns of colorectal cancer in China over a period of 20 years. World J Gastroenterol 2005; 11:4685-8.

13. Ibrahim KO, Anjorin AS, Afolayan AE, Badmos KB. Morphology of colorectal carcinoma among Nigerians: a 30-year review. Niger J Clin Pract. 2011; 14:432-5.

14. Stanojevic G, Nestorovic M, Brankovic B, Stojanovic M, Jovanovic M, Radojkovic M. Primary colorectal lymphoma: An overview. World J Gastrointest Oncology 2011; 13: 14-8.

15. Frezza EE, Wachtel MS, Chiriva-Internati M. Influence of obesity on the risk of developing colon cancer. Gut 2006; 55:285-91.

16. Hamid GA, Saeed NM, Ba-Ashen Y, Ba-Kubirah R. Colorectal carcinoma at Al-gamhouria teaching hospital, Aden, Yemen. Gulf J Oncolog. 2012; 1:16-9.

17. Cancer Incedence report. National Cancer Registry, Ministry of Health, Kingdom of Saudi Arabia, 1997-1998.p.46.

18. Altekruse SF, Kosary CL, Krapcho M, et al. (eds). SEER Cancer Statistics Review, 1975-2007, National Cancer Institute. Bethesda, MD, http://see.cancer.gov /csr/1975-2007/, based on November 2009SEERdata submission, posted to the SEER web site, 2010.

19. Basaleem HO, Al-Sakaf KA. Colorectal cancer among Yemeni patients. Characteristic and trends. Saudi Med J 2004; 25:1002-5.

20. Abo-Zaid AA, Khfagy W, Marzoul Om, Alaa A, Mostafa I. Colorectal cancer in Egypt. Dis Colon Rectum 2002; 45: 1255-60.

21. Mansoor I, Zahrani IHm Abdul Aziz S. Colorectal cancers in Saudi Arabia. Saudi Med J 2002; 23:322-7.

22. Keswani SG, Boyle MJ, Maramll JP, Mains L, Wilk SM, Hunt JP. Colorectal cancer in patients younger than 40 years of age. Am Surg 2002; 68:871-6.

23. Winawer SJ, Fletcher RH, Miller L. Colorectal cancer screening: clinical guidelines and rationale. Gasteroenterology 1997; 112:594-642.

24. Arai T, Takubo K. Clinicopathological and molecular characteristics of gastric and colorectal carcinoma in the elderly. Pathol Int. 2007; 57:303-14.

25. Abu Salem O. Colon cancer in North Jordan. Rawal Med J 2010; 35: 129-32.

26. Halvorsen TB, Seim E. Degree of differentiation in colorectal adenocarcinomas: a multivariate analysis of the influence on survival. J Clin Pathol 1988; 41:532-7.

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ORIGINAL ARTICLE

Frequency of Tongue Anomalies among Yemeni Children in Dental Clinics

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Abstract:

Objective: This study was designed to know the frequency of tongue anomalies and determine their relation with gender.

Methods: This study was conducted on five hundreds Yemeni children, (250) boys and (250) girls with an age ranged from 6 -12 years. The sample was collected during the 2nd semester of 2013 from dental clinics, College of Dentistry, University of Sciences & Technology, Sana'a, Yemen. Children were examined with a disposable mouth mirrors, using gloves and gauze pads.

Results: Fissured tongue (6.8%) was the most prevalent tongue anomalies, followed by geographic tongue (4.6%), then macroglossia (2.6%), after that ankyloglossia (2.2%), then hairy tongue (0.8%), and finally microglossia (0.4%).

Conclusion: The frequency of tongue anomalies among Yemeni children was in boys more than girls. Fissured tongue was the commonest tongue anomaly, while the lowest one was microglossia.

Key words: Tongue, Frequency, Anomalies, Yemeni, Children.

1. Introduction

Fissured tongue is a benign asymptomatic condition frequently seen in the general population that is characterized by grooves that vary in depth and are noted along the dorsal and lateral aspects of the tongue. Although a definitive etiology is unknown, it was seen in Melkerson-Resenthal syndrome and Down syndrome (1). It was stated that the frequency of fissured tongue was four to five times greater in institutionalized, mentally retarded children; a fact that can be partly explained by the frequency of fissured tongue in trisomy 21 (2).

Geographic tongue or benign migratory glossitis is a benign condition in which asymptomatic smooth erythematous patches with discrete borders appear on the dorsum of the tongue. The condition usually is discovered on routine clinical examination. This ulcer-like lesion may recur at different sites on the tongue, creating a migratory appearance and mostly resolves completely (3). The etiology of geographic tongue is unknown. Many factors have been suggested like familial condition with significant role of heredity, congenital anomaly, asthma, rhinitis, systemic diseases like psoriasis, anemia, gastrointestinal disturbances, candidiasis, lichen planus, hormona imbalance, psychological conditions (4-7).

Macroglossia, or enlarged tongue, is defined as a tongue that protrudes beyond the teeth during resting posture or if there is an impression of a tooth on the lingual border when the patients slightly open their mouths (8-10). Two classification systems are commonly used. The first classification subdivided macroglossia as generalized or localized, and the second classification subdivided macroglossia as true or relative (11,12). Vascular malformations and muscular hypertrophy are the most common etiologies of true macroglossia, with lingual lymphatic malformations being the most common vascular anomaly (12).

Macroglossia is a component of numerous syndromes, many caused by inherited metabolic anomalies in which the increase in tongue size is a manifestation of visceromegaly related to lysosomal storage diseases, such as Hurler syndrome, Hunter syndrome, and Maroteaux-Lamy syndrome (13). Other macroglossia-associated disorders include Beckwith-Wiedemann syndrome, neurofibromatosis type I, hemangiomatosis associated with Sturge-Weber syndrome, and congenital lymphangioma (cystic hygroma) (14). In a study, one hundred thirty-five children with macroglossia were identified. Macroglossia was the main reason for consultation in 84 children (15).

Macroglossia may be relative in patients who have a small mandible (micrognathia), which is seen in children with Pierre Robin syndrome; and can result in dysphagia, speech difficulty, and upper airway obstruction and may require surgical reduction of the volume of the tongue (16). It can be classified as either congenital (primary) or secondary. Primary macroglossia is due to over-development of the musculature, while the secondary form may result from a tumor of the tongue (such as diffuse lymphangioma or hemangioma), neurofibromatosis, or occasionally, blockage of the efferent lymphatic vessels, as in cases of malignant neoplasm of the tongue (17).

Ankyloglossia is a congenital oral anomaly characterized by an abnormally short lingual frenulum. Ankyloglossia can make breastfeeding difficult, causing sore nipples, poor infant weight gain, and early weaning in some infants with this condition (18). Severe degrees of ankyloglossia often cause a midline mandibular diastema, lingual mandibular periodontal defects, and speech impairment (2).

The diagnostic criteria of ankyloglossia are easy to detect. Usually, the patient is unable to protrude the tongue past the edge of the lower gingival or mandibular incisors. It said that "when ankylogloosia is noted at birth, one option is to leave it alone and let nature take its course, unless there are early feeding problems". If the child demonstrates any of the problems noted above, a frenulectomy (surgical release of the tongue) can be done (19).

Hairy tongue is a condition in which the heavily keratinized surface layer of the filiform papillae are not continuously desquamated through friction of the tongue with food, the palate, and the upper anterior teeth and replaced by new epithelial cells from below. The filiform papillae lengthen and become heavily coated with bacteria and fungi. The longer papillae give the tongue a hairy appearance (2).

Microglossia, or small tongue, is an anomalous uncommon condition causing tongue tip to be pressed to the incisal part of the lower alveolar bone not exerting physiological impact on the upper alveolar bone. It may be one of the factors for occlusion abnormalities in cleft lip and palate patients (20).

2. Study Aim

This study was designed to know the frequency of tongue anomalies and determine their relation with gender.

3. Methods

- **Sample**

This study was conducted on five hundreds Yemeni children, 250 boys and 250 girls with an age ranged from 6 -12 years. The sample was collected during the 2nd half of the year 2013 from dental clinics, College of Dentistry, University of Sciences & Technology, Sana'a, Yemen.

- **Examination of children**

Each child was examined to detect the tongue anomalies with a disposable mouth mirrors, using gloves and gauze pads. A data collecting chart was designed for recording the necessary information's for each child including personal data as name, age, sex and birth date. For each child who displayed apparent tongue anomaly, the medical condition was reported in order to get the information regarding the cause related to the tongue anomaly.

- **Ethical considerations:**

The process of the research was explained to the parents of the participating children, and a consent form was taken from them.

4. Results

This study was conducted to know the frequency of tongue anomalies among Yemeni children attended the dental clinics, College of Dentistry, University of Sciences & Technology, Sana'a, with an age ranged from 6-12 years. The sample size included 500 Yemeni children, 250 boys (50%) and 250 girls (50%) respectively.

Distribution of tongue anomalies according to gender is summarized in table 1 and figure 1, which show the following results:

- Tongue anomalies were seen in 87 child (17.4%). The number of tongue anomalies in boys was 62 (12.4%) and 25 (5%) in girls. They were more common in boys than girls.

Table 1. Distribution of tongue anomalies according to gender

Tongue anomalies \ Gender	Boys	Girls	Total
Fissured tongue	23 (4.6%)	11 (2.2%)	34 (6.8%)
Geographic tongue	16 (3.2%)	7 (1.4%)	23 (4.6%)
Macroglossia	9 (1.8%)	4 (0.8%)	13 (2.6%)
Ankyloglossia	8 (1.6%)	3 (0.6%)	11 (2.2%)
Hairy tongue	4 (0.8%)	0 (0%)	4 (0.8%)
Microglossia	2 (0.4%)	0 (0%)	2 (0.4%)
Total	62 (12.4%)	25 (5%)	87 (17.4%)

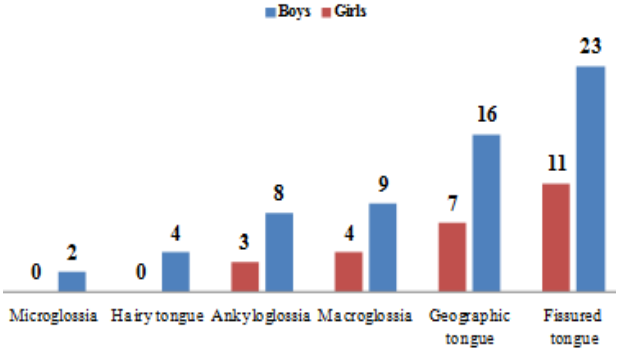

Figure 1. Distribution of tongue anomalies according to gender

▪ The total percentage of fissured tongue was (6.8%) with percentage (4.6%) in boys and (2.2%) in girls (figure 2). While the total percentage of geographic tongue was (4.6%) with percentage (3.2%) in boys and (1.4%) in girls (figure 3).

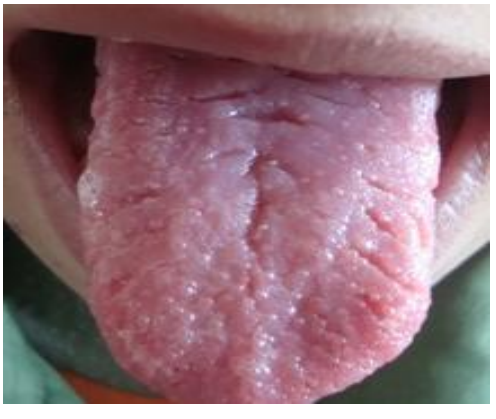

Figure 2. Photograph showing fissured tongue

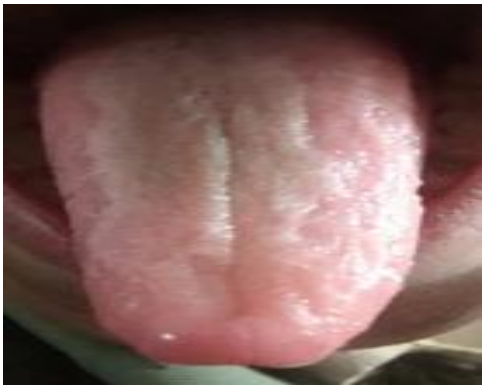

Figure 3. Photograph showing geographic tongue

▪ The total percentage of macroglossia was (2.6%) with percentage (1.8%) in boys and (0.8%) in girls (figure 4). While the total percentage of ankyloglossia was (2.2%) with a percentage (1.6%) in boys and (0.6%) in girls (figure 5).

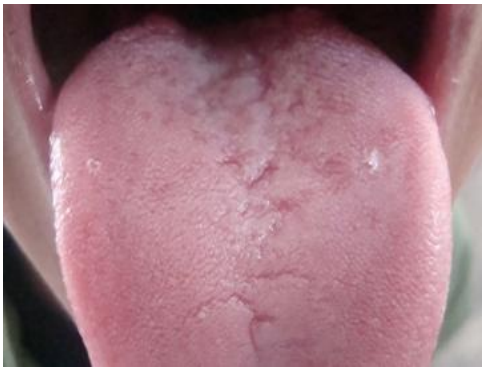

Figure 4. Photograph showing macroglossia

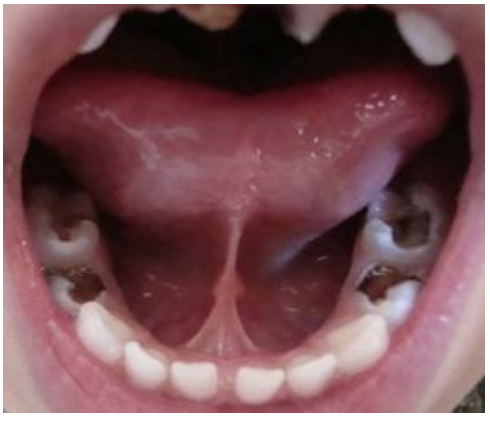

Figure 5. Photograph showing ankyloglossia

- In boys only, the percentage of hairy tongue was (0.8%). However, the percentage of microglossia was (0.4%).

5. Discussion

In the present study, there was a gender predilection of tongue anomalies, with the higher frequency in boys. The number of tongue anomalies was 62 (12.4%) in boys and 25 (5%) in girls. The reasons for this difference were related to hereditary and racial factors.

The frequency of fissured tongue in this study was 6.8% which is significantly less than the findings of Darwazeh and Pillai (1993) (21); Rabiei et al., (2003) (22); Khozeimeh and Rasti (2006) (2) who reported that the frequency of fissured tongue was 11%. It may be due to congenital anomalies or environmental factors such as chronic dry mouth and vitamin deficiency.

The present survey showed 4.6% of all children had geographic tongue. It is significantly higher than that of Shulman (2005) (23) which was (1.05%), but it is significantly lower than what was found by Darwazeh and Pillai (1993) (22) in Jordan; Mojarrad and Vaziri (2008) (24) in Iran, which were 6.8%, and 27% respectively. These differences may be related to sample size and hereditary factors.

The frequency of macroglossia in this study was 2.6%. It may be relative macroglossia due to micrognathia which is in agreement with Donnelly et al., (2000) (16), and may be primary macroglossia due to over-development of the musculature which is in agreement with Stewart and Boggs (1982) (17).

The present survey showed 2.2% of all children had ankyloglossia. It is significantly higher than what was found by Mojarrad and Vaziri (2008) (24) among 6-12 years old in Iran which was (0.8%). However, it is significantly lower than what was found by Rabiei et al., (2003) (22) among 1120 children in Lahidjan; Khozeimeh and Rasti (2006) (2) among 1540 children in Iran, which were 6.7% and 5% respectively. These differences may be related to sample size and hereditary factors.

Hairy tongue was observed in 0.8% of our subjects. This is in agreement with Khozeimeh and Rasti (2006) (2) in Iran, which was 0.8%. However, it is significantly lower than what was found by Darwazeh and Pillai (1993) (21) in Jordan which was 3.4%. These differences may be related to several predisposing factors such as oral use of certain drugs, dry mouth, and vitamin deficiency.

Finally, microglossia was observed in 0.4% of our subjects. It is the lowest tongue anomaly which is in agreement with Nadtochiĭ et al., (2012) (20), who reported that microglossia is an anomalous uncommon condition.

6. Conclusion

Tongue anomalies were seen in 17.4% of Yemeni children with a frequency in boys more than girls. Fissured tongue was the commonest tongue anomaly, while the least common one was microglossia.

References

1. Goswami M, Verma A, Verma M. Benign migratory glossitis with fissured tongue. J Indian Soc Pedod Prev Dent. 2012; 30(2):173-5.
2. Khozeimeh F, Rasti G. The prevalence of tongue abnormalities among the school children in Borazjan, Iran. Dental Research Journal 2006; 3: 1-2.
3. Shobha B, Barkha N. Benign migratory glossitis: Report of two cases. Indian J Dent Adv 2011; 3(4): 708-10
4. Weathers D, Baker G, Archard H, Burkes E. Psoriasis form lesions of the oral mucosa with emphasis on ectopic geographic tongue .Oral Surg 1974; 37:72-88.

5. Eidelman E, Chosack A, Cohen T. Scrotal tongue and geographic: Polygenic and associated traits. Oral surgery 1976; 42:591-6.

6. Mark R, Simons M. Geographic tongue-as manifestation of atrophy. Br j Dermatol 1979; 101:159-62.

7. Rajendran R, Sivapathasundaram B. Shafers Textbook of oral pathology, Philadelphia, 2006, 5th edition.

8. Weiss L, White J. Macroglossia: A review. J La State Med Soc.1990; 142(8):13-6.

9. Ueyama Y, Mano T, Nishiyama A, Tsukamoto G, Shintani S, Matsumura T. Effects of surgical reduction of the tongue. Br J Oral Maxillofac Surg. 1999; 37(6):490–5.

10. Ruscello D, Douglas C, Tyson T, Durkee M. Macroglossia: A case study. J Commun Disord. 2005; 38(2):109–22.

11. Myer C, Hotaling A, Reilly J. The diagnosis and treatment of macroglossia in children. Ear Nose Throat J. 1986; 65(10):444–8.

12. Vogel J, Mulliken J, Kaban L. Macroglossia: A review of the condition and a new classification. Plast Reconstr Surg. 1986; 78(6):715–23.

13. Cho H, Kim S, Song E, Lee J, Choi Y. Macroglossia secondary to lymphangioma of the deep neck space: Report of two cases. Korean Journal of Pediatrics 2010; 53(1):97-102.

14. Brightman V. Diseases of the tongue. In: Lynch HA. editor. Burket's oral medicine. 9th ed. Philadelphia: Lippincott-Raven, 1994; pages: 251-7.

15. Prada C, Zarate Y, Hopkin R. Genetic Causes of Macroglossia: Diagnostic Approach. Pediatrics, 2012; 129(2):431-7.

16. Donnelly L, Jones B, Strife J. Imaging of Pediatric Tongue Abnormalities. AJR 2000; 175:489-93.

17. Stewart R, Boggs W. Pathology of soft tissues and jaws. In: Stewart RE, Barber TK, Troutman KC, Wei SHY, editors. Pediatric dentistry. St. Louis: CV Mosby Co. 1982, page 184.

18. Messner A, Lalakea L, Aby J, Macmahon J, Bair E. Ankyloglossia: Incidence and associated feeding difficulties. Archives of Otolaryngology-Head and Neck Surgery 2000; 126(1): 36-9.

19. Kummer A. Ankyloglossia: to clip or not to clip? That's the question. ASHA Leader 2005; 10(17): 6-7.

20. Nadtochiĭ A, Starikova N, Fomina G. Morphofunctional tongue features revealed by multispiral CT in cleft lip and palate patients. Stomatologiia (Mosk). 2012; 91(4):54-9.

21. Darwazeh A, Pillai K. Prevalence of tongue lesions in 1013 Jordanian dental outpatients. Community Dent Oral Epidemiol 1993; 21(5):323-4.

22. Rabiei M, Mohtasam Z, Masoudirad H, Niazi M , Niazi H. Frequency of Tongue

23. Anomalies in Primary School Of Lahidjan. Journal of Guilan University of Medical Sciences 2003; 12(45):36-42.

24. Shulman J. Prevalence of oral mucosal lesions in children and youths in the USA. Int J Paediatr Dent 2005; 15(2):89-97.

25. Mojarrad F, Vaziri P. Prevalence of Tongue Anomalies in Hamadan, Iran. Iranian Journal of Public Health 2008; 37(2):101-5.

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ORIGINAL ARTICLE

Prevalence of Helicobacter Pylori Infection in Yemeni Patients

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Abstract:

Background: Helicobacter pylori (Hp), a common bacterial infection linked to disorders of the gastrointestinal tract, are found in half of the world population. In developing countries, Hp infection is a public-health issue (1). It is one of the world's most common human bacterial infections and associated with chronic gastritis, peptic ulceration and gastric cancer (2). Data on its prevalence in the Middle East is limited (3). There are limited data about H. pylori infection in Yemen.

Objective: The objective of this study is to determine the prevalence of H. pylori infection among patients undergoing upper GIT endoscopy with different symptoms at different endoscopic units at Different Hospitals and governorates in Yemen.

Subjects and Methods: All patients referred to the endoscopy units at Al-Wehdah Teaching Hospital-Thamar University, Police Model Hospital, Police General Hospital, endoscopic charity camps at Socatra Island, Mukalla, Dowaan and Seiyun Hospitals in Yemen, were enrolled in a prospective study. For each patient clinical and socioeconomic and environmental data were collected. Endoscopy was performed and two gastric biopsies were obtained from antrum and corpus. Helicobacter pylori infection was diagnosed at the time of endoscopy by using the rapid urease test (RUT).

Results: A total of 2300 patients ,1300 females (56.522%) with a mean age of 37.395 years (range 16-90 years) and 1000 males (43.478%) with a mean age of 39.168 Years (range 18-75 years) were included in this study. Abdominal pain was the most frequent symptom reported. Gastritis 99% and esophagitis 85% were the most frequent endoscopic findings. Helicobacter pylori were found in 2270 patients (98.7%). Thirty four patients (1.5%) patients were present with gastric cancer, 80 patients with gastric ulcer (3.48%), 1500 (65%) patients with duodenal erosions,150 (6.5%) patients with duodenal ulcer, all patients with duodenal and gastric ulcers have H.pylori positive test, by using rapid urease test (RUT).

Conclusions: The prevalence of Helicobacter pylori infection in hospital patients, who undergone upper gastrointestinal endoscopy is very high (98.7%). This study is confirming that Helicobacter pylori are significantly associated with oesophagitis, gastritis and peptic ulcer in Yemen.

Key words: H. Pylori, Yemen, RUT, YSALBG, YEMEN.

1. Introduction

Helicobacter pylori (H. pylori), a microaerophilic, flagellated, curved or spiral, gram-negative bacterium, selectively colonizes the human stomach (2). It is one of the most common infections that affect humans. H. pylori was identified in 1983, it is one of the most common infections that affects humans (4). It is associated with a wide range of digestive and other extra-gastrointestinal disorders (5).

Its infection is widespread throughout the world, and it is present in about 50% of the global human population (2). Its prevalence is highly variable in relation to geography, ethnicity, age, and socioeconomic factors. It is higher in developing countries and lower in the developed world. In general, however, there has been a decreasing trend in the prevalence of Hp infections in many parts of the world in the recent years (1). In developing countries the prevalence of H. pylori ranges between 70% - 90% (6), while in developed countries it is approximately 50% (7).

H. pylori prevalence in developed countries is slowly increasing during childhood, which continues through adolescence and early adulthood, with an abrupt increase around 50-60 years of age (8). In the non-industrialized countries, H. pylori prevalence increases more rapidly during childhood and most adolescents and adults are infected. Thus, differences in H. pylori prevalence between industrialized and non-industrialized countries are greater at younger ages and get smaller at older ages (9, 10, 11). It has been reported at Saudi Arabia, that infection which is acquired at an early age and reaches up to 36.9% as age advances (12). Differences in prevalence within populations are due to a variety of factors primarily relating to socioeconomic status and geographic origin (13).

The prevalence rate of H. pylori infection in patients undergoing endoscopy for upper gastrointestinal symptoms has now been reported from many Middle Eastern countries, including Egypt, Iran, Israel, Oman, Saudi Arabia, the United Arab Emirates and Yemen (14). It has been reported in Yemen that prevalence of H. pylori infection among patients underwent upper GIT endoscopy in Sana'a major hospitals was very high (99.6%) (15), while another study showed that patients with gastritis and peptic ulcer disease had similar rates of infection as reported from Europe, United States and Africa 71%-92%. However, patients with non-ulcer dyspepsia had higher rates of infection 61%- 89% (14). In 1998, 88.5 % of patients in Kuwait with dyspeptic symptoms who were referred for endoscopy proved H. pylori-positive (16), while at 2004, another study showed that 82.2% of dyspeptic patients in Yemen have H. pylori infection (17). It has been shown that H. pylori-positive patients tend to have dyspepsia, but the relationship between H. pylori and dyspepsia remains controversial (18).

The 2005 American College of Gastroenterology (ACG) guidelines for the management of dyspepsia recommended testing for H. pylori infection among dyspeptic patients even without alarming features (19). Helicobacter pylori is the major cause of gastritis, that plays a key role in the etiology of peptic ulcer and it is a risk factor for gastric carcinoma, however, detailed information on the prevalence of the bacteria in developing countries and on the factors that may influence the pattern of distribution remains scanty (3).

The mode of H. pylori transmission is unknown but it is thought to be mainly through the fecal-oral route, Oral-oral and waterborne transmissions are other modes (6). Helicobacter pylori (H. pylori); is a common bacterial infection

that is linked to disorders of the gastrointestinal tract (7).

Helicobacter pylori have a role in the multifactorial etiology of peptic ulcer disease. A link between H. pylori infection and duodenal ulcer disease is now established. The role of H. pylori as a gastric pathogen is dependent on virulence factors and pathogenic mechanisms. Virulence factors are those that allow H. pylori to survive in the hostile environment of the gastric lumen which includes its spiral shape, motility, adaptive enzymes, proteins, and ability to adhere to gastric mucosal cells and mucus (20). Pathogenic mechanisms are those that lead either directly to disruption of the gastric mucosal barrier including its toxins like Vac A and Cag A and mediators of inflammation. The prevalence of H. pylori infection in gastric and duodenal ulcers has consistently been found to be 80% and 90% -100% respectively (21).

Chewing Khat was not associated with a higher prevalence of oesophageal dysplasia, making it unlikely to be the cause of the perceived high incidence of oesophageal carcinoma in Yemen. There was a high prevalence of gastric H. pylori colonization (93%), this was not related to chewing Khat (22), while another study in Yemen showed that daily khat chewing was found associated with high prevalence of Helicobacter pylori and duodenal ulcer, this can be explained by khat chewing itself or by beverages consumed during the session or insecticides and chemicals used during the process of planting. All these factors together with the chemical constituents of the khat itself need further investigation to unveil the causation effect on duodenal ulcer (23).

A definite cure of peptic disease and prevention of ulcer complications, as well as cure of mucosa-associated lymphoid tissue (MALT) lymphoma, is dependent on successful eradication of H pylori (24). Recurrence of H pylori after a successful eradication is rare in developed countries and more frequent in developing countries (25). Recrudescence (re-colonization of the same strain) rather than reinfection (colonization with a new strain) is considered more likely to be responsible for most of the cases (26).

2. Study Aims

This study was designed to show the prevalence of H. pylori among Yemeni patients undergoing endoscopy for different reasons.

3. Patients and Methods

We conducted a prospective study during the period September 2008 to September 2012, involving patients who were referred from different governorates/Hospitals to the Endoscopy Units at Al-Wehdah Teaching Hospital-Thamar University, Police Typical Hospital, and Police General Hospital, endoscopic charity camps at Socatra Island, Mukalla, Dowaan and Seiyun hospitals. For each patient clinical and socioeconomic and environmental data were collected after informed consent was taken for every patient to be included in this study. With exclusion of patients who are complaining from upper GIT bleeding during the endoscopic procedure and those who are coming while they are under PPI and antibiotic therapy. Endoscopy was performed for all the patients and two gastric biopsies were obtained from antrum and corpus.

These patients were presented with upper gastrointestinal symptoms such as: Nausea, vomiting, heartburn, abdominal pain, and upper gastrointestinal bleeding. History of Chewing khat, non-steroidal anti-inflammatory drugs (NSAID)

ingestion, and smoking habit was documented. None of the patients had been taking anti-ulcer drugs or antibiotics for at least 4 weeks prior to endoscopy.

Upper gastrointestinal endoscopy was carried out with propofol sedation for some patients and local anesthetic spray for the others. The procedure was fully explained to all patients which was included in the informed consent. During endoscopy, the endoscopic findings such as hiatus hernia, oesophagitis and its grade, gastritis, gastric or duodenal ulcers and any gross esophageal, gastric or duodenal pathology were documented. Two biopsies were taken from the antrum and corpus of the stomach, and then we crushed on the RUT slide, the presence of H. pylori was determined by rapid urease test (RUT).

Ethical considerations

An informed consent was taken from the participants after the nature of the study have been expalined clearly.

4. Results

A total of 2300 patients (1300 females) with a mean age of 37.395 years (range 16-90 years) and (1000 males) with a mean age of 39.168 Years (range 18-75 years) were studied (Table 1).

Table 1. Age and sex distribution

Age group	Male	Female
16-29	235	345
30-44	450	465
45-59	210	315
60-64	56	76
65-74	25	55
>74	24	44

Age range for Males: 18-75; Mean age of Males: 29.368 years; Median age of Males: 44 years.
Age range for Females: 16-90; Mean age of Females: 37.922 years; Median age of Females: 49 years.

Abdominal pain was the highest presenting symptom. Gastritis 99% and esophagitis 85% were the most frequent endoscopic findings. Helicobacter pylori were found in 2270 patients (98.696%), (Table 2).

Table 2. Number of patients with Positive Rapid Urease Test (RUT)

H.pylori positive	No. of patients 2300	Percentage
Male	990/1000	99%
Female	1280/1300	98.462%
Total numbers/percentages	2270/2300	98.696%

Thirty four patients (1.5%) were present with gastric cancer, 80 patients with gastric ulcer (3.48%), 1500 (65%) patients with duodenal erosions, and 150 (6.5%) patients with duodenal ulcer. Patients showed Helicobacter pylori in their biopsies by using RUT are shown in table 3.

Table 3. Endoscopic findings of patients

Endoscopic findings	No. of patients/2300	Percentages
Oesophagitis	1955/2300	85%
Gastritis	2270/2300	99%
Gastric ulcer	80/2300	3.48%
Duodenal erosions	1500/2300	65.22%
Duodenal ulcer	150/2300	6.522%
Gastric cancer	34/2300	1.5%

NB: some patients are presenting with more than one of the above endoscopic findings

On the basis of Rapid Urease Test (RUT), the prevalence of H. pylori was 98.696% in Yemeni patients in the above hospitals. H. pylori infection was associated significantly with education level, residence, water supply and marital status but not for blood groups, age, sex, occupation, frequency of endoscopy, and household bets. Also H. pylori infection was significantly associated with mild

gastric inflammation grade, Peptic Ulcer Diseases (PUD), gastritis, and duodenitis as well as it is associated with some dyspeptic symptoms. The prevalence of H. pylori among patients with PUD (Gastric Ulcer and Duodenal Ulcer) was 100%.

5. Discussion

Since the prevalence of H. pylori infection is much higher in the less developed nations, where the socio-economic status, low living standards and poor sanitation may be implicated (2). This has stimulated us to conduct this prospective study to determine the prevalence of H. pylori; we collected the cases in the above mentioned hospitals, which covers almost all Yemen governorates.

The prevalence of H. pylori in Saudi Arabia is 80% of adults, while in Egypt is 90%, and in Lybia it is 94% of adults. The principal reasons for these variations involve socioeconomic differences between populations. Transmission of H. pylori is largely by the oral, oral or fecal, oral routes. A lack of proper sanitation, safe drinking water, and of basic hygiene, as well as poor diets and overcrowding represent major risk factors (27). These conditions resemble to a great extent the conditions of patients included in this study.

The prevalence of H. pylori at Sana'a and Thamar hospitals was 99 %, while at Hadramout Hospitals was 98% and at Socatra Hospital is 100%. The overall prevalence is approximately (98.696%), (Tables 4 and 5). These high percentages are due to lack of proper sanitation, of safe drinking water, and bad hygienic habits, like eating of vegetables in some area and chewing Khat without washing or with improper washing specially in the areas using water of sewerages to rinse these plants in agriculture as well as poor diets and overcrowding.

Table 4. Geographic distribution of patients

Sana'a			Thamar			Socatra			Hadramout								
									Mukalla			Dawaan			Seiyun		
No.	+	%	No.	+	%	No.	+	%	No.	+	%	No.	+	%	No.	+	%
300	297	99%	1300	1287	99%	40	40	100	45	44	98%	65	63	98%	550	539	98%

Table 5. Collective percentages of positive patients

Region/governorates	No. of Positive patients	percentages
Sana'a	297	99%
Thamar	1287	99%
Socatra	40	100%
Mukalla	44	98%
Dawaan	63	98%
Seiyun	539	98%
Total	2270	98.696%

We used rapid urease Test (RUT), because it is cheap, rapid result can be obtained and its sensitivity and specificity it reaches to 98 % and 99 % respectively (2).

6. Conclusion

The prevalence of Helicobacter pylori infection in patients subjected to an upper gastrointestinal endoscopy at the above hospitals, in Yemen is very high (98.696%). This study confirms that Helicobacter pylori is significantly associated with oesophagitis, gastritis and peptic ulcer. Further studies are needed to determine the types of Helicobacter pylori strains present in Yemen. The prevalence of H. pylori infection in dyspeptic patients in Yemen seems to be high. Duodenal ulcer disease was found to be significantly associated with H. pylori infection.

References

1. Helicobacter pylori in developing countries; WGO; 2010.
2. Howden CW. Clinical expressions of Helicobacter pylori infection. Am J Med 1996; 100: 27S, 32S.
3. Kamal E. Bani-Hani, Shadi M. Hammouri. Prevalence of Helicobacter pylori in Northern Jordan. Saudi Medical Journal 2001; Vol. 22 (10): 843-7.
4. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet. 1984; 1:1311–5.
5. Pakodi F, Abdel-Salam OM, Debreceni A, Mozsik G. Helicobacter pylori. One bacterium and a broad spectrum of human disease! An overview. J Physiol Paris 2000; 94: 139-52.
6. Al-Moagel MA, Evans DG, Abdulghani ME, Adam E, Evans DJ Jr, Malaty HM et al. Prevalence of Helicobacter Pylori (formerly Campylobacter) infection in Saudi Arabia, and comparison of those with and without upper gastrointestinal symptoms. Am J Gastroenterol 1990; 85: 944-8.
7. Graham DY, Malaty HM, Evans DG, Evans DJ Jr, Klein PD, Adam E. Epidemiology of Helicobacter Pylori in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. Gastroenterology 1991; 100: 1495-501.
8. Torres J, Perez-Perez G, Goodman KJ, Atherton JC, Gold BD, Harris PR, et al. A comprehensive review of the natural history of Helicobacter pylori infection in children. Arch Med Res. 2000; 31:431-9.
9. Pounder RE, Ng D. The prevalence of Helicobacter pylori infection in different countries. Aliment Pharmacol Ther. 1995; 9:S33-9.
10. Goodman KJ, Correa P. The transmission of Helicobacter pylori: A critical review of the evidence. Int J Epidemiol. 1995; 24:875-87.
11. Bardhan PK. Epidemiological features of Helicobacter pylori infection in developing countries. Clin Infect Dis. 1997; 25:973-8.
12. Hanafi MI, Mohamed AM. Helicobacter pylori infection: seroprevalence and predictors among healthy individuals in Al Madinah, Saudi Arabia. J Egypt Public Health Assoc. 2013 Apr; 88 (1):40-5.
13. Abdulaziz A. BinSaeed. Glimpse of the Epidemiological Research on Helicobacter Pylori in Saudi Arabia. Saudi J Gastroenterol. 2009; 15 (2): 85.
14. Novis BH, Gabay G, Naftali T. Helicobacter pylori: the Middle East scenario. Yale J Biol Med. 1998 Mar-Apr; 71 (2):135-41.
15. Huda Z. Al-Shami. Prevalence of Helicobacter pylori infection among patients underwent upper gastrointestinal tract endoscopy in Sana'a major hospitals. 2002; Thesis in MSc, Faculty oof Medicne, Sana'a University, Yemen.
16. Waleed M Alazmi, Iqbal Siddique, Nabeel Alateeqi and Basil al-Nakib. Prevalence of Helicobacter pylori infection among new outpatients with dyspepsia in Kuwait. BMC Gastroenterology 2010, 10:14.
17. Gunaid AA, Hassan NA, Murray-Lyon IM. Recurrence of Helicobacter pylori infection 1 year after successful treatment: prospective cohort study in the Republic of Yemen. Eur J Gastroenterol Hepatol. 2004 Nov; 16 (12):1309-14.
18. Rosenstock S, Kay L, Rosenstock C, Andersen L, Bonnevie O, Jørgensen T: Relation between Helicobacter pylori infection and gastrointestinal symptoms and syndromes. Gut 1997, 41 (2):169-76.
19. Talley N, Vakil N: Guidelines for the management of dyspepsia. Am J Gastroenterol 2005, 100 (10):2324-37.
20. Dunn B.E. "Pathogenic mechanisms of Helicobacter pylori". Gastroenterology Clinics of North America, 1993: vol. 22, no. 1, pp. 43-56.
21. Vikram Kate, N. Ananthakrishnan, Frank I. Tovey. Is Helicobacter Pylori Infection the Primary Cause of Duodenal Ulceration or a Secondary Factor? A Review of the Evidence. Gastroenterology Research and Practice; 2013.
22. El-Guneid A, el-Sherif AM, Murray-Lyon IM, Zureikat N, Shousha S. Effect of chewing Qat on mucosal histology and prevalence of Helicobacter pylori in the oesophagus, stomach and duodenum of Yemeni patients. Histopathology. 1991 Nov; 19 (5):437-43.
23. Yahia A. Raja'a, Tariq A. Noman, Abdul Karim M. Al Warafi, Nabeel A. Al Mashraki, Abdul Malik A. Al Yosofi. Saudi Medical Journal 2000; Vol. 21 (9) 887.
24. Yaron Niv. H pylori recurrence after successful eradication. World J Gastroenterol 2008 March 14; 14 (10): 1477-8.
25. Gisbert JP. The recurrence of Helicobacter pylori infection: incidence and variables influencing it. A critical review. Am J Gastroenterol 2005; 100: 2083-99.
26. Xia HX, Talley NJ, Keane CT, O'Morain CA. Recurrence of Helicobacter pylori infection after successful eradication: nature and possible causes. Dig Dis Sci 1997; 42: 1821-34.
27. World Gastroenterology organization guidelines: Helicobacter pylori in developing countries (2010): Updated on 2014.