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From the Editor

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I would like to take this opportunity to thanks the authors who support the journal and send us their valuable papers. In addition I would like to announce the second middle east congress of ageing and alzheimer's of which the MEJFM is one of the official organisers of this conference. The conference website is: http://www.meama.com/secondconference/index.htm. We would like to encourage our readers to attend and to submit abstracts for the conference.

In this edition a paper from Jordan looked at Pattern of inflammatory markers in children with asthma and allergic rhinitis. The study included 42 healthy children (mean age 9±5 years) and 70 pediatric patients during regular control of respiratory allergic diseases, asthma (n=47) and rhinitis (n=23), mean age 7±4 years. The author concluded that children with respiratory allergic diseases had greater concentrations of hsCRP in serum compared with healthy children.

A paper from An Najah University looked at Prevalence of Allergic Rhinitis & Its Risk Factors Among An-Najah University Students. The study sample consisted of around 1000 (52% females, & 48% males) randomly selected students from all colleges of the University. Data were collected through structured interview including questionnaire filling. The Results show relatively lower allergic rhinitis prevalence in Palestine compared to some neighbouring countries, but were consistent with studies done in Turkey. The results confirmed the strong relationship of Allergic Rhinitis and respiratory infections and Asthma.

A paper from Iran looked at the concept of disease clustering for public health specialists. The examination of disease clustering has become a flourishing area in medical research during recent decades. The term cluster usually refers to uncommon diseases of non-infectious origin such as leukaemia, spontaneous abortion and suicides, which are repeatedly supposed to be due to environmental exposures. The aim of the present article is to discuss some of the most important fundamental issues surrounding this concept for the public health specialists within the Middle East region.

A paper from Turkey looked at Peripheral Giant Cell Granuloma using a Case Report. The author reports that a 30-year-old female patient admitted to their clinic with the complaint of painless swelling and gingival growth underwent total excision of an exophytic lesion causing purplish-red colored gingival growth found during clinical examination in the upper jaw in the region of right vestibular 1st molar tooth. Histopathological examination revealed PGCG. The author stressed that effective treatment of PGCG requires not only complete excision of the lesion, but also elimination of irritating factors.

Zeid AA and Dahabrah M looked at the Pattern of inflammatory markers in children with asthma and allergic rhinitis. The study included 42 healthy children (mean age 9±5 years) and 70 pediatric patients during regular control of respiratory allergic diseases, asthma (n=47) and rhinitis (n=23), mean age 7±4 years. The study results demonstrated that children with respiratory allergic diseases had greater concentrations of hsCRP in serum compared with healthy children.

Dr. Mazen Ahmad Asayreh from Jordan has submitted a paper on chest pain in women. The author found that in women referred with chest pain, a diagnosis of normal coronary arteries was common.

Pattern of Inflammatory Markers in Children with Asthma and Allergic Rhinitis

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Keywords: C-reactive protein, asthma, allergic rhinitis.

ABSTRACT

The aim of the study was to estimate whether determination of C-reactive protein (CRP) concentration could be used as an inflammation marker in children with asthma and allergic rhinitis.

Materials and methods:
The study included 42 healthy children (mean age 9±5 years) and 70 pediatric patients during regular control of respiratory allergic diseases, asthma (n=47) and rhinitis (n=23), mean age 7±4 years. Highsensitive CRP (hsCRP) concentration was determined by immunoturbidimetric method on latex particles. The concentrations of C3, C4 and al pha1-antitrypsin were determined by immunoturbidimetric method on an Olympus AU 400 biochemistry analyzer, whereas leukocyte and platelet counts were determined on a Sysmex XT-1800i counter.

Results:
The concentration of hsCRP was statistically significantly higher in patients with asthma and allergic rhinitis than in healthy children. These patients also had statistically significantly higher levels of C3, C4, al pha1-antitrypsin and leukocyte count as compared with healthy subjects. Platelet count was significantly greater in asthma (but not rhinitis) patients as compared with the group of healthy children.

Conclusion:
Study results demonstrated that children with respiratory allergic diseases had greater concentrations of hsCRP in serum compared with healthy children.

Introduction

Allergic respiratory diseases, which are among the most common chronic diseases in children may manifest with symptoms in the upper (allergic rhinitis and sinusitis) and lower (allergic asthma) airways. As the histology, allergic disease epidemiology, mechanisms of inflammation, triggers for allergic disease clinical manifestation, diagnostic procedures and treatment are common to both upper and lower airways, they can be referred to as an integral respiratory system[1]. Accordingly, asthma and rhinitis can be considered as manifestations of a single chronic allergic respiratory syndrome[2]. Chronic airway inflammation as one of the major features of asthma and allergic rhinitis involves many cell types, of which mastocytes, eosinophilic granulocytes and T-lymphocytes, play most important roles. In sensitive individuals, the inflammation induced by environmental allergens leads to the symptoms of asthma (bronchoconstriction, cough and chest tightness, frequently overnight or at dawn)[3,4] and rhinitis (nasal congestion, rhinorrhea, sneezing, nose itching)[5]. The discomforts are usually reversible and resolve spontaneously or with therapy.

C-reactive protein (CRP) is a well-known inflammation marker. Serum concentration of CRP is generally determined to assess a systemic inflammation[6], e.g. pneumonia, rheumatic disease, intestinal disease, etc.[7]. It has recently been observed that CRP, even in the reference interval, i.e. determination of high sensitive CRP (hsCRP), can serve as a relevant prognostic marker in patients with cardiovascular disease[8] or diabetes mellitus[9]. Determination of hsCRP concentration implies determination of CRP concentration by the established turbidimetric method on latex particles but adjusted to the low measurement area. So, hsCRP can also be used to assess the grade of inflammation in asthma patients[10].

As inflammation is one of the major characteristics of respiratory allergic diseases, the aim of this study was to estimate whether determination of CRP concentration

would be of use as a marker of inflammation in children with asthma and allergic rhinitis.

Materials and Methods

The study included 42 healthy children (control group), mean (x±SD) age 9±5 years, and 70 pediatric patients during regular control of respiratory diseases, asthma (n=47) and rhinitis (n=23), mean (x±SD) age 7±4 years. Control group consisted of clinically healthy children without information of atopic status referred for systematic medical check-up at King Hussein Medical Center. The diagnosis of allergic disease was based on clinical criteria (personal and family history, physical examination, pulmonary function measurement, provocation skin tests) and laboratory testing (increased concentration of total and specific Ig E antibodies, blood and nasal swab eosinophilic granulocyte count). The study groups included children without diabetes mellitus. Body mass index was uniform

in both study groups (between 5th and 85th centile values for age). Children with acute viral or bacterial infection of the airways were excluded. All patients were referred from primary health care offices to King Hussein Medical Center in Amman-Jordan between January and June 2007. Diagnostic work-up was performed according to standardized procedure, and in line with ethical principles (approved by the Hospital Ethics Board) and Declaration on Human Rights from Helsinki 1975 and Tokyo amendments 2004[11]. Blood sampling was done upon clinical examination at outpatient clinics of allergology and pulmonology, between 8.00 a.m. and 4.00 p.m.

Methods

CRP concentration was determined by immunoturbidimetric method on latex particles[12], on an Olympus AU 400 biochemistry analyzer, using reagents from the same manufacturer. CRP concentration was determined in two ways:

(a) a method with linearity of 0.2 to 480 mg/L, and

(b) a method in low measurement area (linearity of 0.08 to160 mg/L; hsCRP). The concentrations of complement components C3 and C4 and of al pha1-antitrypsin (AAT) were determined by immunoturbidimetric method on an Olympus AU 400 biochemistry analyzer, using reagents from the same manufacturer. Leukocyte and platelet counts were measured on a Sysmex XT-1800i blood counter.

Results

The group of children with allergic diseases were presented in two modes: in total, irrespective of diagnosis, and in subgroups according to diagnosis (asthma and allergic rhinitis), for statistical analysis to be performed for the group as a whole and for each subgroup separately. Results obtained on the concentrations of hsCRP, CRP, C3, C4, AAT, leukocyte count and platelet count in the control group of healthy children and the group of children with respiratory allergic diseases (asthma and rhinitis) are presented in Table 1. As between-group differences were statistically significant for C3, C4, AAT and leukocyte count, and borderline for hsCRP, CRP and platelet count, between group analysis was performed. The concentration of CRP was statistically significantly higher in patients with asthma and rhinitis than in the control group, irrespective of the method of determination. The levels of C3, C4, AAT and leukocyte count were also statistically significantly higher in the patient group, either in total or in groups according to diagnosis. Platelet count was statistically significantly higher in asthma patients but not in rhinitis patients as compared with the control group of healthy children. The mean hsCRP concentration was statistically significantly higher in children with allergic diseases (0.65±0.55 mg/L) than in control group children (0.28±0.16 mg/L). The patients with asthma showed higher values of the upper range limit for hsCRP (2.75 mg/L) than patients with allergic rhinitis (1.57 mg/L). Only the concentration of CRP measured by the conventional

Procedure was statistically significantly lower in rhinitis patients as compared with asthma patients, whereas the values of other tests did not differ significantly between these two subgroups. Percentile values revealed the patients with allergic rhinitis to have an hsCRP concentration of = 1.57 mg/L, whereas 5% of asthma patients had an hsCRP concentration greater than 1.57 mg/L. In some 40% of asthma patients, the concentration of the complement components C3 and C4 exceeded the concentration recorded in patients with allergic rhinitis. Platelet count was found to be =363x109/L in patients with allergic rhinitis and >450x109/L in 5% of asthma patients. Percentile values of AAT were by 8% on an average greater in asthma children than in those with allergic rhinitis.

Discussion

The present study indicated the children with asthma and allergic rhinitis to have a higher concentration of hsCRP than healthy children. The search of the available literature revealed only one group of Israeli authors to have presented results of hsCRP determination in 63 asthma children. These authors compared hsCRP concentration in acute exacerbation of asthma and upon therapy administration, and found it to be significantly higher in acute disease as compared with post-therapeutic state (14.28±8.45 mg/L vs. 1.92±3.16 mg/L). They also report on the correlation between hsCRP concentration and forced expiratory volume in 1 second (FE V1)[15]. In our study, both the children with asthma and those with allergic rhinitis had the mean hsCRP concentration lower than the concentration from the above-mentioned report (0.71±0.58 mg/L and 0.53±0.50 mg/L, respectively). Takemura et al.[10] determined hsCRP concentration in adult asthmatic patients and showed it to be higher in patients without therapy with inhalation corticosteroids (1.33±1.48mg/L) than either in healthy subjects (0.21±0.30 mg/L) or in patients receiving therapy (0.9±1.0 mg/L). The hsCRP levels recorded in our control group of healthy children (0.28±0.16 mg/L) were comparable to those reported by Takemura et al.[10] in healthy adults. According to some authors[16], systemic inflammation could also be verified in asthma patients, since these patients had an elevated concentration of acute phase proteins. Our study demonstrated the children with asthma and allergic rhinitis to have a higher leukocyte count and A1-AT concentration than healthy children, supporting the existence of mild systemic inflammation in patients with respiratory allergic diseases. In adult patients, it is not asthma alone that is the key factor to increase the concentration of hsCRP, as it can be influenced by other factors such as the risk of cardio vascular disease[8], diabetes mellitus[9], obesity[17], atherosclerosis and atherothrombosis[18]. The prevalence of these risk factors is by far lower in children; therefore, the elevated concentration of CRP in our children could have been ascribed to inflammation due to respiratory allergic diseases. It was demonstrated that complement also plays a role in allergic inflammation, as the C3 and C4 levels were greater in children with respiratory allergic diseases than in healthy controls. CRP is known to be able to activate complement components[19]. Platelet

count was also increased in patients with asthma but not in those with allergic rhinitis. Future studies should therefore investigate the causes of this difference between asthma and rhinitis because platelets may have a varying role in allergic reactions[20]. We are aware of the limitations of the present study due to the lack of information on the lipid status that may influence the hsCRP concentration. Study results (one of the first in this area) demonstrated that children with respiratory allergic diseases had greater concentrations of hsCRP in serum as compared with healthy children. Further studies are needed to demonstrate whether determination of hsCRP concentration could be useful in therapeutic monitoring of children with respiratory allergic diseases.

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Table 1. HsCRP, CRP, C3, C4, A1-AT concentrations, and platelet and leukocyte counts in healthy subjects and patients with respiratory allergic diseases asthma and rhinitis

		HSCRP (MG/L)	CRP (MG/L)	C3 (G/L)	C4 (G/L)	AAT (G/L)	PLT (× 109/L)	LKC (× 109/L)
Control N=42	Range	0.08- 0.79		0.90-1.30				
	M	0.23		1.10				
	X±SD		0.4 ±0.2		0.22±0.05	1.30 ±0.27	299 ±57	7.0 ±1.6
	p	0.064 (5)	0.074 (5)	<0.001 (5)	0.011 (5)	0.020 (5)	0.054 (5)	0.006 (5)
Asthma/ Rhinitis N=70	Range	0.10 - 2.75	0.2 - 3.4					
	M	0.50	0.7					
	X±SD			1.64 ±0.25	0.32 ±0.09	1.79 ±0.30	326 ±91	8.3 ±1.9
	p	<0.001(1)	<0.001(1)	<0.001(1)	<0.001(1)	<0.001(1)	<0.001(1)	<0.001(1)
Asthma N=47	Range	0.13 - 2.75	0.3 - 3.4					
	M	0.55	0.7					
	X±SD			1.67 ±0.23	0.33 ±0.08	1.83 ±0.33	338 ±103	8.4 ±2.1
	p	<0.001(2)	<0.001(2)	<0.001(2)	<0.001(2)	<0.001(2)	0.042 (2)	<0.001(2)
Rhinitis N=23	X±SD	0.53 ±0.50	0.7± 0.6	1.56 ±0.28	0.29 ±0.09	1.69 ±0.20	298 ±47	8.1 ±1.5
	p	0.003(3)	0.0048 (3) 0.045 (4)	<0.001 (3)	<0.001 (3)	<0.001 (3)	0.479 (6)	0.017(3)

(1) Asthma/rhinitis vs. control; (2) Asthma vs. control; (3) Rhinitis vs. control; (4) Rhinitis vs. asthma; (5) ANOVA or Kruskal-Wallis for asthma, rhinitis and control.

The Effect of The ALCAT Test Diet Therapy for Food Sensitivity in Patient's With Obesity

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Key Words: Obesity, Body Mass Index, Body Fat, Elimination Diet, Food Sensitivity, ALCAT Test.

ABSTRACT

Investigations were performed on 27 patients (14 males,13 females)with mean age of 42.77± 6.23 Years and with mean height of 168.66 ± 2.09 cm with obesity who had difficulty losing weight when they adhered to a reduced calorie diet were evaluated for specific white blood cell food induced reactions. Twelve weeks after following the ALCAT diet plans, we observed a significant decrease in Body weight, Total Body fat percent and Body Mass Index. Body weight was decreased significantly from 91.37 ± 10.56to 74.6 ± 6.76 kg, Total Body fat % was decreased significantly from 37.1 ± 7.16to 27.66 ± 6.52 % and Body Mass Index was significantly decreased from 32.1 ± 3.8to 26.1 ± 2.63 kg/m2. Thus our results confirmed the value of ALCAT test and the elimination diet in alleviating symptoms such as obesity, gastrointestinal reflux, chronic fatigue, headache and other chronic disorders associated with food hyper sensitivities.

Introduction

Obesity is an increasing health problem worldwide. The World Health Organization predicts that by 2015, 2.5 billion people will be overweight (body mass index (BMI) ([weight (kg)/ (height (m))^2]) >25, and 700 million adults will be obese (>30).[1] No universally accepted definition for obesity exists but there is general consensus that BMI levels above 25% for adults are considered overweight and over 30% considered obese. Adult men and women should maintain BMI levels between 18% and 24.5%.[2] Excess weight is a major risk factor for a wide range of chronic diseases and exacerbates hypertension, dyslipoproteinemia, osteoarthritis and other musculoskeletal problems.[3]

According to the world Health Organization (WHO) instances of obesity are on the increase in the United Arab Emirates.

In 2005, over 75% of women over 30 in the United Arab Emirates were classified as overweight, with similar estimates for men.[4] The adverse health outcome of excess weight places an increasing burden on our health care system.[5,6] Being overweight is highly resistant to intervention[7] and it is doubtful that we are any closer to a solution today than we were decades ago. In fact, we appear to be moving away from, rather than towards, the national objectives according to data from National Health and National Examination Survey, NHANES III.[8] Until now, no intervention has been shown consistently to achieve true weight control. Although the precise reason for the high relapse rate is not known, the stunning uniformity of these findings, which now extend over nearly five decades, should give pause to anyone who proposes to treat, much less cure, obesity.[9] However, recent advances in technology show that obesity is associated with a low-grade inflammation of white adipose tissue stemming from the chronic activation of the innate immune system leads to further weight gain, insulin resistance and diabetes.[10] As food intolerance/ sensitivity is one possible cause of this low-grade inflammation, we decided to investigate the hypothesis that the adherence to a food intolerance elimination diet improves weight

in refractory patients. The method used for determining the diet related triggers of this inflammation in this study is the ALCAT test.

The ALCAT Test

The ALCAT test uses a specially designed particle counter (hematology analyzer with an automated assay sampler) and food test agents to semi-qualitatively measure white blood cell reactivity, if any, to each agent analyzed. The degree of reactivity is determined by comparing a baseline distribution curve (of the white cells) against the distribution curve generated by the analysis of each test agent/blood sample, and calculating the absolute differences between the curves and the standard deviation (SD). Any reactivity under SD1 will be considered as non-reactive (NEG); reactivity

between SD1 and SD2 will be considered as marginally reactive (RANGE 1+); reactivity between SD2 and SD3 will be considered reactive (RANGE 2+); and finally, reactivity above or equal to SD3 will be considered markedly reactive (RANGE MPOS).

There is evidence demonstrating the ALCAT test to be effective in improving body mass index (BMI) and/ or scale weight. According to a Baylor University study, "As compared to

following a plan of their own choosing, participants who followed the ALCAT plan achieved rather dramatic changes in their body composition." This experiment showed that 98% of the subjects following the ALCAT plan either lost scale weight or improved their body composition.[11] Dr. J.R. Cabo-Soler, Chief of Biochemistry at the University of Valencia, reported that iso-caloric food elimination diets, based on ALCAT test results, promoted enhanced weight loss, comprised more of adipose tissue, rather than muscle mass, as determined by DEXA studies in a population of refractory weight loss subjects.[12] The ALCAT test has demonstrated a reproducibility of 94.94%, according to a trial by Steinman et. al at the University of Cape Town.[13] 92.0678% reproducibility was reported by Neetling et. al. at the

University of the Free Orange State, also in South Africa, which makes it an acceptable screening model for intolerance testing in humans. In Addition, a Norwegian study reported the ALCAT test to be >90% reproducible.[16,14] Fell et. al reported an 83.4% correlation between ALCAT test results and double blind oral challenges as determined by careful clinical evaluation in statistically significant number of patients exhibiting food sensitivity related symptoms, such as migraines, irritable bowel syndrome, eczema and other conditions, that are often observed as co-morbidities in obese patients.[15]

Despite mounting evidence of the efficacy of the ALCAT test in reducing obesity and the overall activation of the immune system, there have been no studies of the weight loss benefits of the ALCAT test reported for an Arab population. The purpose of this study was, therefore, to determine the effectiveness of the ALCAT test as a weight loss tool in Arab patients who had experienced difficulty achieving goal weight by calorie restriction.

Patients, Materials and Methods

This study was designed to determine whether people who could not lose weight on a low calorie diet could achieve their weight loss using the results from ALCAT test. A group of 27 patients with 14 males and 13 females with mean age of 42.77± 6.23 Years and with mean height of 168.66 ± 2.09 cm with obesity who had difficulty losing weight when they adhered to a reduced calorie diet were evaluated for specific white blood cell food induced reactions. (Table 1) Patients were exhibiting multiple symptoms including: obesity, gastrointestinal reflux, chronic fatigue, headache and other chronic disorders associated with food sensitivities.

Citrated blood is diluted 1 in 5 with buffer and approximately 90µl is added to added to each test agent well. The test agents are diluted preparations of food extracts (standardized for potency) bonded to the bottom of the well. Following 45 minutes incubation at 37 ° C with constant agitation, the test agents are incubated for a further 30 minutes at room temperature. Red cells are lysed by adding an azide free, electrolytic solution containing an lytic reagent, "ALCALyse", supplied by Cell

Science Systems, Ltd. Each test agent is then analyzed in sequence using the ROBOCat II particle counter (also manufactured by Cell Science Systems, Ltd.) with one control for every 10 food items tested. The 100 foods tested are shown in Table 2.

The basis of the ALCAT test is the measurement of changes in white blood cell size/volume following incubation with food and other test agents using the ROBOCat II linked to a computer. Contact between foreign entities and whole blood can cause autolysis, a phenomenon known as autocytotoxicity, and other cellular reactions.

There are three mechanisms which cause this phenomenon, and one does not require the priming of cells in vitro by either antibody or antigen[17].

It was on this basis that an automated method was sought which would not only be reproducible and objective but would directly correlate with in vivo food challenge.

The computer is programmed to compare cell cultures incubated in the presence of food agents and measure a shift in cell volumes related to exposure to the test agent.

In keeping with standard laboratory practices, deviation in the test histograms, when compared with a control (identically treated but lacking the test agent) that exceeds one standard deviation (SD) is regarded as a positive reaction, and the patient is advised to avoid that food.

Results

Twelve weeks after following the ALCAT diet plans, we observed a significant decrease in Body weight, Total Body fat percent and Body Mass Index. Body weight was decreased significantly from 91.37 ± 10.56to 74.6 ± 6.76 kg, Total Body fat % was decreased significantly from 37.1 ± 7.16to 27.66 ± 6.52 % and Body Mass Index was significantly decreased from 32.1 ± 3.8to 26.1 ± 2.63 kg/m2.

Discussion

In this study, we demonstrated the beneficial role of ALCAT test in obesity.

Correlations between obesity and ALCAT test results were positive and significant in these patients. All these patients had difficulty losing weight when they adhered to a reduced calorie diet; this study confirmed a greater weight loss in patients when they were placed on a diet plan according to the ALCAT test results. Also, the weight loss was mostly fat. Other interesting observations included, a better sense of well being and improved physical performance, improvement in abdominal bloating and digestive problems. These findings are significant because many overweight patients find it difficult to lose weight by cutting calories alone. This study suggests that delayed food hyper sensitivities may interfere with weight loss regardless of calorie restrictions. Also the beneficial effects of improved sense of well being and improvement of gastrointestinal conditions were observed collateral benefits consistent with an overall normalization of immune activity.

Conclusion

Obesity is a major public health problem among local Arab societies and more recently, among Asian countries as well. The associated health risks and diseases present a

tremendous drain on the economy and affect the quality of life. The most effective program's for losing and maintaining a desirable body weight with careful monitoring of proper diet should be implemented holistically to ensure a successful weight reduction programme. Individuals need to be aware of the many myths and misconceptions surrounding weight control. Most 'miracle agents' for weight loss do not have a scientific basis. However, these data provide compelling evidence for the effectiveness of the ALCAT test diet plan in producing a positive change in body weight, body Fat and BMI and self reported disease symptoms. Thus our results confirmed the value of ALCAT test and the elimination diet in alleviating symptoms such as obesity, gastrointestinal reflux, chronic fatigue, headache and other chronic disorders associated with food hyper sensitivities.

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Table1: Partial list of symptoms associated with food intolerance.

Characteristics	Number of cases	Total percentage %
Depression	ADHD	Urticaria
Perennial rhinitis	CFS	Fibromyalgia
Asthma	Inflammatory Bowel	Arthritis
Eczema	Irritable Bowel	Diarrhea
Dermatitis	Migraine Headache	Infertility
Autism	Otitis Media	GI Ulcer

Table 2: List of Food agents used by the ALCAT test in 27 patients with obesity.

Food allergens	Food allergens	Food allergens	Food allergens
Almond	Corn	Lemon	Rice
Apple	Courgette	Lentil bean	Salmon
apricot	Cow milk	Lettuce	Sardine
Banana	Crab	Lima bean	Sesame
Barley	Cucumber	Lobster	Shrimp
Basil	Curry	Mango	Soybean
Bay leaf	Date	Mint	Spinach
Beef	Dill mix	Mushroom	Strawberry
Beef sugar	Duck	Mustard	Sting bean
Black pepper	Egg white	Nutmeg	Sunflower
Broccoli	Egg yolk	Oat	Sweet potato
Cabbage	Eggplant	Olive	Tea
Cane sugar	Garlic	Onion	Thyme
Cantaloupe	Ginger	Orange	Tomato
Carrot	Gliadin	Oregano	Tuna
Cashew	Gluten	Papaya	Turkey
Cauliflower	Goat milk	Parsley	Turmeric
Cayenne pepper	Grape	Peach	Turnip
Celery	Grapefruit	Peanut	Vanilla
Chick pea	Green pepper	Pear	Walnut
Chicken	Green pea	Pineapple	Watermelon
Cinnamon	Haddock	Plum	Wheat
Cocoa	Hazelnut	Rabbit	White potato
Coconut	Honey	Radish	Yeast (Baker)
Coffee	Lamb	Raspberry	Yeast (Brewer)

Table 3: List of Food agents used by the ALCAT test in 27 patients with obesity.

Patient No	Sex	Age (years)	Height (cm)	Body Weight (kg)	
				Before	After
1	M	46	165	90	75
2	F	40	167	80	70
3	F	41	169	83	70
4	M	40	171	103	84
5	F	43	170	106	76
6	F	39	170	96	78
7	F	48	172	76	60
8	M	42	169	71	64
9	M	40	168	86	70
10	M	33	165	89	70
11	M	43	167	93	77
12	F	40	170	84	68
13	F	45	168	90	74
14	M	49	173	85	73
15	M	51	167	96	78
16	F	53	171	112	85
17	M	48	170	103	86
18	M	56	169	96	80
19	M	40	170	81	67
20	F	43	165	82	70
21	F	34	169	86	71
22	M	30	170	89	76
23	F	46	168	93	80

24	M	50	167	104	85
25	M	43	166	110	80
26	F	38	168	100	80
27	F	34	170	80	67
Mean + SD		42.77 + 6.23	168.66 + 2.09	91.37 + 10.56	74.6 + 67.6
The p-values derived from Student 't'-tests. P < 0.001 is highly significant					

Table 4: List of Food agents used by the ALCAT test in 27 patients with obesity.

Patient No	Sex	Age (years)	Height (cm)	Total Body Fat (%)	
				Before	After
1	M	46	165	38	29
2	F	40	167	30	24
3	F	41	169	26	20
4	M	40	171	43	26
5	F	43	170	36	24
6	F	39	170	33	21
7	F	48	172	26	19
8	M	42	169	25	20
9	M	40	168	30	21
10	M	33	165	31	20
11	M	43	167	40	31
12	F	40	170	38	29
13	F	45	168	43	32
14	M	49	173	38	30
15	M	51	167	45	34
16	F	53	171	53	40
17	M	48	170	49	40
18	M	56	169	43	32
19	M	40	170	30	20
20	F	43	165	33	22
21	F	34	169	31	23
22	M	30	170	39	26
23	F	46	168	40	32
24	M	50	167	42	34
25	M	43	166	45	39
26	F	38	168	40	33
27	F	34	170	36	26
Mean + SD		42.77 + 6.23	168.66 + 2.09	37.1 + 7.16	27.66 + 6.52
The p-values derived from Student 't'-tests. P < 0.001 is highly significant					

Table 5: The Body Mass Index of 27 patients before and after 12 weeks of following the ALCAT diet plan.

Patient No	Sex	Age (years)	Height (cm)	BMI {weight (kg)/ height (m^2)	
				Before	After
1	M	46	165	33.1	27.5
2	F	40	167	28.7	21.5
3	F	41	169	29.1	24.5
4	M	40	171	35.2	28.7
5	F	43	170	36.7	26.3
6	F	39	170	33.2	27.0
7	F	48	172	25.7	20.3
8	M	42	169	24.9	22.4
9	M	40	168	30.5	24.8
10	M	33	165	32.7	25.7
11	M	43	167	33.3	27.6
12	F	40	170	29.1	23.5
13	F	45	168	31.9	26.2
14	M	49	173	28.4	24.4
15	M	51	167	34.4	28.0
16	F	53	171	38.3	29.1
17	M	48	170	35.6	29.8
18	M	56	169	33.6	28.0

19	M	40	170	28.0	29.1
20	F	43	165	30.1	29.8
21	F	34	169	30.1	28.0
22	M	30	170	30.8	23.2
23	F	46	168	33.0	25.7
24	M	50	167	37.3	24.9
25	M	43	166	39.9	26.3
26	F	38	168	35.4	28.3
27	F	34	170	27.7	23.2
Mean + SD		42.77 + 6.23	168.66 + 2.09	32.1 + 3.8	26.1 + 2.63
The p-values derived from Student 't'-tests. P < 0.05 is significant					

Chest Pain in Women

ABSTRACT

Objective:
To characterize the clinical features, investigations, and prognosis of women referred with chest pain who subsequently underwent coronary angiography.

Material and methods:
A retrospective analysis of 500 women with chest pain seen in emergency or outpatient clinic in three hospitals belonging to Royal Medical.
Services in Jordan between (Jan 2000-jan 2004) who subsequently underwent coronary angiography. Women were divided according to angiography results as: division with normal coronaries, and other with coronary artery disease.

Results:
195/500 women have had normal coronary angiograms, and 305 of them have had coronary artery disease. Diabetes mellitus was the biggest risk factor that was encountered in women with coronary artery disease (P=0.001). The specificity and positive predictive value of exercise testing before angiography were 68%, and 73% respectively.
Revascularization procedures were 216/305 (71%). Many patients with normal coronaries had symptoms during follow up 146/195 (75%) and 44/195 (23%) required readmission for severe symptoms.

Conclusions:
In women referred with chest pain, a diagnosis of normal coronary arteries was common. Risk factor analysis and exercise stress testing were of limited value in predicting coronary artery disease in women. A diagnosis of non-cardiac chest pain in patients with normal coronaries is of little benefit regarding the morbidity.

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Key words: chest, pain, women

Introduction

Epidemiologic studies of acute myocardial infarction have described gender differences in the time of death after infarction, with greater numbers of men dying before hospitalization than women. Chest pain in women is a commonly encountered condition which accounts for an appreciable number of referrals to cardiologists for further evaluation. Psychiatric illnesses are present in up to 50% of new patients attending the cardiac clinic with chest pain. Patients with recurrent chest pain who are free of significant coronary artery disease (CAD) account for 10% to 30% of patients who undergo coronary angiography.

The accurate diagnosis of chest pain is often difficult. Myocardial ischemia, aortic dissection, pulmonary embolism, pericarditis, and gastroenterological sources of chest pain are the most common differentials. The symptom of chest pain has many causes; some of them are cardiac and others are not.

The presence of common symptoms, such as heartburn and regurgitation, usually make the diagnosis of gastroesophageal reflux disease fairly straightforward. Coronary angiography is the criterion for establishing a diagnosis of CAD. Patients with positive results on an exercise test are more likely to be further investigated, but ST segment shift with exercise is a less specific marker of CAD in women.

However, coronary angiography carries a small but well documented risk of complications and consequently should be for those patients most likely to have chest pain of cardiac origin. For many years research in CAD has been focused on men, yet CAD is also the major cause of death and an important cause of disability in women.

We studied the files of 500 women referred to the cardiac center in King Hussein Medical Center (KHMC) with chest pain for further investigation over a four year period, in addition to comparing the characteristics of women with CAD and women with normal coronary arteries.

Materials and Methods

Patients were divided into two groups according to the presence or absence of CAD identified by coronary angiography. A diagnosis of CAD, based on the cardiologist's reports, was made if the diameter of stenosis in any coronary artery exceeded 40%.

Patients were excluded if they were found to have cardiac disease other than CAD. The presence of recognized risk factors for CAD, which included a family history (first degree relative with CAD), hypercholesterolemia (random total cholesterol more or equal to 6.5mmol/l or patient receiving lipid lowering agent), hypertension requiring specific treatment, history of smoking (current or previous cigarette smoker), and diabetes mellitus (requiring treatment by diet, oral hypoglycemic, or insulin, were recorded).

The exercise test was analyzed as positive or negative. Patient details were obtained from the clinical

notes, with follow up to present day. Events during follow up including MI, hospital readmission and death were recorded. Chi –square was used for statistical analysis.

Results

Of the 500 females who underwent coronary angiography, 305 (61%) had CAD and 195 (39%) had normal coronary arteries. Women with CAD were older than women with normal coronary arteries (mean SD 58.7(9.1) year v 53.6(9.3); P 0.001). Only diabetes mellitus was more frequently encountered in women with CAD than women with normal coronary arteries (39/305(13%) v 4/195(2%); P=0.01) (Table 1). Hypertension and positive family history for CAD were more frequently encountered in women with CAD than in those with normal coronaries (hypertension 156/305(51%) v 106/305 (35%), P=0.003; family history, 216/305 (71%) v 155/195(51%), P= 0.01.

Smoking was not a common risk factor for CAD in both groups (70/305(23%) v 33/195(17%)). Exercise test results were correlated with the presence or absence of CAD. The test was positive in 50/195 (26%) in women with normal coronary arteries and 201/305 (66%) in women with CAD. The sensitivity of exercise testing was (63%) and the specificity was (74%). In 103/305(34%) had single vessel disease, 88/305 (29%) had two vessel disease, and 118/305 (39%) had triple vessel disease .No correlation was found between the results of exercise test and numbers of diseased vessels.

Follow up details were obtained in 91% of patients. Mean follow up time was 3.6 years for patients with normal coronary arteries. Table II shows the outcome in the 195 women with normal coronary arteries. Three women died from non-cardiac causes, and three women died suddenly of an unknown cause. Table III shows the outcome in the 305 women found to have CAD.

Discussion

There is growing interest in research into women with suspected or documented coronary artery disease, which until recently, has been little studied. Coronary artery disease is the main cause of death in women in the Western world; whether the results from these studies can be applied to all women is unknown.

Consequently, the aim of our study was to characterise women referred with chest pain to a cardiac centre since they represent an important clinical problem. Standard risk factors for coronary artery disease and the results of exercise testing were of limited value in distinguishing women with coronary artery disease from those with chest pain from non-cardiac causes. Despite a diagnosis of noncardiac chest pain, many patients continued to have symptoms and seemed to have derived little benefit from cardiac investigation. Furthermore, cardiac events were no more frequent during the follow up period. Women represented the minority of patients referred with a clinical diagnosis of angina for further investigation. 39% of women referred with chest pain for further investigation, were subsequently found to have normal coronary arteries, which is in keeping with the coronary artery surgery study, in which 46%of women referred with chest pain for angiography had normal coronary arteries.

Studies examining the importance of risk factors in the development of coronary artery disease have shown that hypertension, smoking, raised serum concentrations of lipids, diabetes mellitus and a family history of coronary artery disease are all important in predicting the development of the disease . Other than diabetes mellitus, however, risk factors for coronary artery disease in women were poor discriminators in our study. The reason(s) why only diabetes mellitus discriminated between women with and without coronary artery disease is uncertain. Smoking is not a common risk factor for coronary artery disease in Jordan because of social restrictions. In one study the relative risk of fatal coronary artery disease in diabetics compared with non-diabetic patients was 3.3 in women after adjustment for age, systolic blood pressure, cholesterol, body mass index, and cigarette smoking. Positive results on the exercise test were found in 26% women subsequently shown on angiography to have normal coronary arteries, which is comparable with other studies. Some patients may have abnormalities of coronary flow reserve, which could account for their symptoms.

Our data indicates that the vast majority of patients with normal coronary arteries continue to experience chest pain. Perhaps this is not surprising since the cause of the patient's symptoms may remain undiagnosed, despite further non-cradiological investigation. Alternatively, patients may continue to believe that their pain is cardiac in origin, a possible explanation in some, since about a third continued antianginal treatment during follow up. Although these findings are not new, the implication is that doctors communicate poorly with patients and reassurance is inadequate. Furthermore, the situation is perpetuated by the continued prescription of antianginal drugs in the knowledge that the patient does not have coronary artery disease. Perhaps cardiologists spend disproportionately little time counseling patients with normal coronary arteries compared with patients with coronary artery disease.

The results of this study indicate that chest pain in women referred for coronary angiography is often non-cardiac in origin, and standard criteria used to determine the likelihood of coronary artery disease in men are of limited value in women .Current limitations on health care resources emphasise the need for better identification of those women most likely to have coronary artery disease before referral for invasive assessment.

Although establishing a diagnosis of normal coronary arteries may be reassuring for the patient's physician, such a diagnosis does little to relieve the symptoms experienced by these patients, who, in the absence of an alternative diagnosis, continue to place a considerable drain on health care resources.

Conclusions

Chest pain in women is common and may or may not have a cardiac cause. In this study 39% of women referred with chest pain who subsequently underwent coronary angiography were found to have normal coronary arteries. Despite a diagnosis of normal coronary arteries morbidity was considerable; an appreciable proportion continued to have chest pain and to take anti-anginal drugs.

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Table 1: risk factor profile in patient with chest pain:

Patient group	Family history of(CAD)	Hypercholesterolaemia	Hypertension	Smoking	Diabetes mellitus
Coronary artery disease (CAD)	216 (71%)	146 (48%)	156 (51%)	70 (23%)	39 (13%)
Normal coronary arteries	155 (51%)	79 (41%)	106 (35%)	33 (17%)	4 (2%)

Table 2: Outcome in 195 patients referred to hospital with chest pain and found to have normal coronary arteries:

Outcome	Women number and percent
Continued chest pain	146(75%)
Further treatment for angina	56(29%)
Readmission to hospital due to chest pain	87(45%)
Myocardial infarction	3(1.5%)
Death from non-cardiac cause	3(1.5%)
Sudden death	3(1.5%)

Prevalence of Allergic Rhinitis & Its Risk Factors Among An-Najah University Students - Nablus/Palestin

ABSTRACT

Background: Inhalant allergic conditions such as seasonal and perennial allergic rhinitis is becoming quite common. The effect of allergy on an individual's quality of life and the extent to which it may restrict daily activities is often overlooked.
Controlling allergies can significantly decrease health care costs. The purpose of this study is to estimate the prevalence of allergic rhinitis among young adults in Palestine represented by An-Najah University students.

Methods: The study sample consisted of around 1,000 (52% females, & 48% males) randomly selected students from all colleges of the University. Data were collected through structured interview including questionnaire filling. All data were analysed using SPSS program applying Chi-square test, with 95 % level of significant (P value = 0.05).

Results: Allergic rhinitis prevalence rate was 3.1 and the percentage of patients who reported to have allergic rhinitis was 38.1%; there was no statistically significant association between allergic rhinitis and gender, smoking, place of living, and other housing conditions. On the other hand the relationship between allergic rhinitis and weight loss, deep sleeping, chronic respiratory infections, nasal polyps, anxiety, and sleep apnea was a statistically significant relationship (p value < 0.05). The triggers that have large effects on the health of the population sample for allergic rhinitis were respiratory infections, tyre burning and war gases, house dust, strong odours, auto exhaust, smoke and weather changes (49.7%, 49 .1 %, 46.7%, 40.6 %, 33.9%, 33.8%, 34.2%), respectively.

Conclusion: Results show relatively lower allergic rhinitis prevalence in Palestine compared to some neighbouring countries, but were consistent with studies done in Turkey. The results confirmed the strong relationship of Allergic Rhinitis and respiratory infections and Asthma.

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Key words: allergic Rhinitis, prevalence, Palestine

Introduction

Allergic rhinitis, especially in adolescents and children, has become a major public-health problem in developed and developing countries[1]. A large - scale cohort study in the UK revealed a rising trend of allergic rhinitis, from 12 to 23% during 1958-1970[2]. Several large surveys in Switzerland also showed increasing prevalence of self-reported hay fever from 9.6% in 1985 to 13.5% in 1992[3]. Similar results have also been reported all around the world[4,5,6].

Inhalant allergic conditions such as seasonal and perennial allergic rhinitis are becoming quite common[7]. The effect of allergy on an individual's quality of life and the extent to which it may restrict daily activities is often overlooked.

Controlling allergies can significantly decrease health care costs[8,9]. The purpose of this study is to estimate the prevalence of allergic rhinitis among young adults in Palestine represented by An-Najah University students.

Methodology

Nablus district is located in the northern part of the West bank. It is bounded by Jenin from the north; Tulkarm from the west; Ramallah and Jericho from the south and the Jordan river from the east[10]. The geographical position of Nablus district in the northern part of west bank gives it a comparatively low temperature range. Located in Nablus, An-Najah National University, is currently the largest University in the West Bank, with 16 colleges and around 13,000 enrolled students[11].

Population of the Study

The study population was chosen from An-Najah university in Nablus. The study sample consisted of a total of 1000 randomly selected students from all colleges of the University, whether scientific, humanitarian, or community college. The percentage of students in the sample represented the percentage of students in each college.

The age of the students was at a range of (18-27). Both males and females were included in almost equal percentage.

Data Collection

Data were collected during the period of the first of September 2004 to the end of December 2004, using structured interview. A questionnaire was designed, evaluated, and reviewed by an expert statistician. A pilot test was carried out on 30 students to find the capacity of students to understand the questionnaire wording then the questionnaire layout was modified accordingly. A total of 1,116 questionnaire forms were distributed; the total response rate in this study was 90 %, 1007)\ questionnaires were returned.

Questionnaire Component

The questionnaire spotlighted several parts that play important roles in triggering allergy. The following are important components of the questionnaire:

Sociodemographic factors including age, sex, college, weight, sport, and smoking.

Environmental history including residence, trees, allergens, inside the home, type of cooling, type of heating, indoor animal and type of pillow.

Triggers that cause or worsen the subject's symptoms including exercise, respiratory infections, weather changes, foods. The symptoms included nasal, sinus, eyes, chest, eczema, asthma and allergy problem (frequency and severity), and health problems other than asthma and allergy.

Data Analysis

All data from questionnaires for the 1007 student sample were entered into the computer and computed using SPSS program and applying Chi - square test, with 95 % level of significant (P value = 0.05).

Analysis of Descriptive Studies

Tables containing descriptive studies were obtained, such as sex, age, residence, environmental, social and living factors.

Analysis of Relationship

Relationship between risk factors, triggers, and some disease with allergic rhinitis among An-Najah University students was obtained; also relationships between sex, age, residence, smoking, sport practicing and allergic rhinitis were obtained.

Results

Table 1 describes the demographic and anthropometric characteristics of The study sample. Males and females were almost equal. Most of the study sample were single (94.9%), not working (93.6%), and non smokers (81.1%).

Table (2) describes the place of living whether it is dormitory or own house; it also describes some environmental factors of the place of residence. More than half the sample live in the city, and about half live in the university dormitory. 60% live in relatively new houses, which were either stone buildings or brick buildings. Using fan was the major cooling method, with only 1.3% using air conditioning.

Table 3 shows the prevalence rate of allergic rhinitis in this study and the distribution of allergic rhinitis according to gender and place of residence.

Prevalence rate of allergic rhinitis was calculated as follows:

$$P = \frac{\text{Number of people with the disease or condition at a specific time } (\times 10)\ n}{\text{Number of people in the population at risk at the specified time*}}$$

*The number of An-Najah University students in the year 2004 was 12,500 students.

Table 4 shows the percentage of triggers in relation to allergic rhinitis in our study sample. The triggers that have a large effect on health of population sample for rhinitis were respiratory infections (49.7%), tyre burning and war gases 49.1%, house dust 46.7%, strong odours 40.6%, auto exhaust 33.9%, smoke 33.8%, weather changes 34.2%, grass and trees 20.9%, and cosmetics 20.1%. All other triggers appear to have less effect.

Table 5 shows that there was a statistically significant relationship (p value < 0.05) between allergic rhinitis and weight loss, deep sleeping, chronic respiratory infections, chronic abdominal pain, nasal polyps, anxiety, sleep apnea, chronic diarrhea, migraines and anemia. However there was no statistically significant relationship (p value > 0.05) between allergic rhinitis and gender, smoking, smoker at home, living place, the tree and grass around the house, kind of building, kind of heating source, kind of cooling source, kind of animals in house, kind of animal around the house, kind of pillow, practice of sports, heart problems, diabetes, thyroid disorders, skin allergy, and glaucoma.

Discussion

Table 1 gives a comprehensive demographic and anthropometric description to the study sample. Our study sample, represents the typical profile of university students in Palestine, where we have a fair mix of males and females; most of them were single and around 20 years of age. All colleges of the university were well represented in this study sample.

Exposure to smoking whether directly and indirectly affects more than half of the sample study, and about half of this sample practice sports.

When the target population were distributed according to their residence, 46.1% of the target population were living in dormitories, which can be explained by the political situation after AL Aqsa Intifada in which closure and checkpoints make transportation between Palestinian cities very difficult. In regards to house conditions, the results show that majority of students live in relatively new stone buildings with quiet lay out in the city. Although the percentage of asbestos buildings was low, it indicates an important need for raising awareness among students about asbestos and its hazardous effects on the lungs.

The triggers that have a large effect on the health of population study sample for allergic rhinitis were respiratory infections, tyre burning and war gases, house dust, strong odours, auto exhaust, smoke and weather changes (Table 4)

These results indicate that war gases and tyre burning play an important role in worsening allergic rhinitis symptoms which points to the effect of political conflict and the use of war gases and tyre burning on the health of Palestinian society.

Furthermore our results indicate that triggers in this study are mainly non allergic in nature. Previous studies show that, AR triggers can be allergic or non allergic in nature[12]. The allergic triggers are house dust mite, pollen, animals, such as dogs and cats, fungal spores and cockroaches, particles. The non-allergic triggers

include smoke and pollution from cooking fuels, wood smoke, smog, viral respiratory tract infections and weather changes. All the above triggers are found in urban, camp and rural environments albeit to different extents[12].

Our results show a statistically significant relationship between allergic rhinitis and weight loss, deep sleeping, chronic respiratory infections, chronic abdominal pain, nasal polyps, anxiety, sleep apnea, chronic diarrhea and migraines at p value < 0.05(Table 5).

Several studies in other parts of the world have shown similar relationship results[13,14]. The gender relationship with Allergic Rhinitis was not statistically significant in our study (Table 5). A Swedish study also did not find a difference between men and women in the general population regarding allergic rhinitis[15]. However a study in Tehran, found a significant relationship between gender and Allergic Rhinitis[16].

The same study in Tehran and another study in Finland[17] also found that environmental and social factors are important risk factors in the incidence of Allergic Rhinitis[16]. On the contrary this relationship in our study was not statistically significant, (Table 5).

This difference in prevalence, triggers and risk factors for allergic rhinitis among different countries has been demonstrated repeatedly in the epidemiological studies.

The international study of asthma and allergies in childhood (ISAAC)[18] steering committee, which conducted a study to investigate worldwide prevalence of asthma, allergic rhino conjunctivitis, and atopic eczema was a very obvious example.

The multifactorial factors and the presence of several types of allergic rhinitis are possible explanations.

Conclusion

Palestine, as a country in transition shifting from a traditional to a modern society, has several unique features that put the population at risk of developing allergic conditions.

This is the first study to determine the prevalence of allergic rhinitis and its risk factors among young adults in Palestine. Our results show relatively lower allergic rhinitis prevalence in Palestine compared to some neighbouring countries, but were consistent with studies done in Turkey.

The results also show a statistically significant relationship between allergic rhinitis and weight loss, deep sleep, chronic respiratory infections, nasal polyps, anxiety, sleep apnea, and migraines but neither gender nor residence and environmental factors have a statistically significant relationship with Allergic Rhinitis.

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Table (1) Demographic and Anthropometric characteristics of study sample

Gender	**Frequency**	**Percent%**
Male	448	44.8
Female	511	51.1
Missing system	41	4.1
Total	**1000**	**100**
Age	**Frequency**	**Percent %**
17.5-20	546	54.6
20.5-22	333	33.3
22.5-24	66	6.6
More than 24	25	2.5
Missing system	30	3
Total	**1000**	**100**
College	**Frequency**	**Percent%**
Scientific	348	34.8
Humanitarian	649	64.9
Missing system	3	0. 3
Total	**1000**	**100**
Sport practice	**Frequency**	**Percent%**
Yes	514	51.4
No	428	42.8
Missing system	58	5.8
Total	**1000**	**100**
M. Status	**Frequency**	**Percent%**
Married	43	4.3
Single	949	94.9
Missing system	8	0.8
Total	**1000**	**100**
Job	**Frequency**	**Percent%**
Employed	18	1.8
Worker	10	1
Not working	936	93.6
Missing system	36	3.6
Total	**1000**	**100**
Smoker	**Frequency**	**Percent%**
Yes	176	17.6
No	811	81.1
Missing system	13	1.3
Total	**1000**	**100**
Smoker in house	**Frequency**	**Percent%**
Yes	554	55.4
No	366	36.6
Missing system	80	8
Total	**1000**	**100**

Table (2) Residence,housing and environment description of study sample

Place of Living	Frequency	Percent%
Camps	38	3.8
Village	550	55
City	410	41
Missing system	2	0.2
Total	1000	100
Living in Univ. dormitories	Frequency	Percent%
Yes	461	46.1
No	511	51.1
Missing system	28	2.8
Total	1000	100
How old is my house	Frequency	Percent%
Less than 20	602	60.2
20-50	324	32.4
More than 50	63	6.3
Missing system	11	1.1
Total	1000	100
The lay out of house	Frequency	Percent%
Factories	19	1.9
Fields	172	17.2
Crowded population	160	16
Quiet place	926	62.9
Missing system	20	2
Total	1000	100
Kind of building	Frequency	Percent%
Stone building	534	53.4
Asbestos building	31	3.1
Block building	410	41
Missing system	25	2.5
Total	1000	100
Type of Heating	Frequency	Percent%
Electricity	194	19.4
kerosene	119	11.9
Gas	418	41.8
Coal	208	20.8
Central heating	30	3
Conditioner	13	1.3
Space heaters	8	0.8
Missing system	10	1
Total	1000	100
Cooling system	Frequency	Percent%
Fan	671	67.1
Conditioner	58	5.8
Windows of the house	265	26.5
Missing system	6	0.6
Total	1000	100
Animals living in a house	Frequency	Percent%
Cat	113	11.3
Dog	16	1.6
Birds	90	9
Chicken	108	10.8
Goats	52	5.2
Pigeons	128	12.8
Other	23	2.3
Nothing	451	45.1
Missing system	19	1.9
Total	1000	100
Animals living near the house	Frequency	Percent%
Cat	224	22.4
Dog	58	5.8
Horses	41	4.1
Chicken	126	12.6
Cows And Goats	228	22.8
Other	25	2.5
Nothing	165	16.5
Missing system	23	2.3
Total	1000	100
Kind of Pillow	Frequency	Percent%
Cotton	382	38.2
Spring	274	27.4
Feathers	48	4.8
Wool	243	24.3
Other	42	4.2
Missing system	11	1.1
Total	1000	100
No. of family in house	Frequency	Percent%
3 and less	56	5.6
4-6	291	29.1
7and more	694	69.4
Missing system	9	0.9
Total	1000	100
Cover of the floor	Frequency	Percent%
Carpets	751	75.1
Moqutte	141	14.1
Tile uncovered	101	10.1
Missing system	7	0.7
Total	1000	100

Table (3) Prevalence rate and distribution of allergic rhinitis among study sample

Prevalence rate	**Percentage**	**Frequency**	
3.1	38.1%	381	Allergic Rhinitis
Allergic Rhinitis/ Gender			
Total	**No**	**Yes**	**Gender**
448 100.0%	272 60.7%	176 39.3%	Male Count %
511 100.0%	319 62.4%	192 37.6%	Female Count %
959 100.0%	591 61.6%	368 38.4%	Total Count %
Allergic Rhinitis/Residence			
Total	No	Yes	Residence
38 100.0%	21 55.3%	17 44.7%	Camp Count %
550 100.0%	335 60.9%	215 39.1%	Village Count %
410 100.0%	261 63.7%	149 36.3%	City Count %
998 100.0%	617 61.8	381 38.2%	Total Count %

Table (4) Triggers that worsen or cause symptoms of the study sample.

Triggers	**Frequency**	**Percent % ***
Grass, trees	209	20.9
House dust	467	46.7
Animals	95	9.5
Respiratory infections	497	49.7
Exercise	68	6.8
Night time	119	11.9
Strong odors	406	40.6
Cosmetics	201	20.1
Emotional upset	145	14.5
Smoke	338	33.8
Tire burning , war gases	491	49.1
Cold air	148	14.8
Weather changes	342	34.2
Drugs	99	9.9
Foods, food additives	39	3.9
Latex	40	4.0
Menstrual cycle, pregnancy	11	1.1
Morning time	116	11.6
Auto exhaust	339	33.9
Insect. Mold	183	18.3

*These percentages of triggers apply for only those who have symptoms.

Table (5) Allergic rhinitis& social, environmental, health profile relationships

Variables Name	Chi Value	P Value
Gender	0.296	0.587
Are you smoker	0.225	0.635
Are their smokers in your house	1.06	0.303
Living place	1.473	0.47
The tree and grass around the house	0.957	0.81
Kind of building that I live in it	3.4	0.18
Kind of heating source	5.97	0.42
Kind of cooling source	1.74	0.41
Kind of animals in house	6.75	0.455
Kind of animal around the house	5.07	0.53
Kind of pillow I use	4.97	0.29
# of family in my house	24.9	0.07
Sports practice	4.65	0.3
Heart problems	0.17	0.67
Diabetes	0.022	0.881
Weight loss	7.2	0.007
Deep sleeping	10.4	0.001
Chronic respiratory infections	27.9	0.000
Chronic abdominal pain	7.6	0.006
Nasal polyps	25.7	0.000
Anxiety	10.37	0.001
Thyroid disorder	0.01	0.9
Skin disorder	1.37	0.24
Sleep apnea	5.1	0.023
Chronic diarrhea	8.5	0.004
Migraines	8.09	0.000
Anemia	5.47	0.019

Environment and Our Health

Lesley Pocock
Managing Director, medi+WORLD International
Caroline Pembroke
United Nations Environment Committee.

www.planetarysurvival.net

Most doctors are aware of the adverse impact on human health that a polluted and degraded natural environment has. Additionally much of current big business is polluting, destroying finite resources that belong to humanity for eternity, and selling us unnecessary goods. Add to this, warfare, organised crime, inequities, global warming, depleted resources and dramatic weather and climate events and we end up with a planet that according to all reputable scientists has lost the ability to support life as we know it.

As we are past the 'tipping point' we are told there is no longer any hope for us whatsoever. That is of course, unless we start to actively repair the planet, which has to be the aim of all thinking people globally.

A global group of concerned citizens, have started a global campaign to restore the ownership of the planet back to the people of the world and to demand a world where all people are treated equitably and given equal opportunity. Most importantly however all affairs of mankind need to be sustainable.

Doctors in the Middle East have been most supportive of this project and have joined the global "Save the Planet" team. We also have alliances with Climate Change groups from Middle East countries. We simply ask all doctors of the Middle East to support our project by clicking on the following URL, and all are invited to register as a supporter. (See www.planetarysurvival.net)

In remembrance of Professor Rob Pierce, Lost in The Victorian Bushfires, February 2009

Professor Rob Pierce was a respected colleague and one of our esteemed authors and by way of tribute I copy a worthy testimonial from one of his former PhD students, as published in the Melbourne Age.

Lesley Pocock,
Publisher & Managing Director,
medi+WORLD International

A good man lost

Please take a good look at the face of Professor Robert Pierce (feared) dead in Kinglake. I knew him for just a short time as PhD supervisor at the Austin Hospital.

Professor Pierce was a wise, noble, brilliant man who cared for the future of humanity and his patients with a rare genius as well as warmth, humour, humility and grace. He had a mind like a steel trap and a heart of gold. He clearly loved his family, his staff and his students and was always available despite his busy international schedule. His contribution to medicine and the science of sleep and breathing was immeasurable. As a doctor, he cared for his patients with deep humanity. I suspect he might have died in a similar way, trying to help in some way.

Professor Pierce was one of those rare individuals who make the world a better place, so please take great care in looking at his face. He was a very good man.

Paul Read,
Menzies Creek,
Victoria, Australia

The Concept of Disease Clustering for Public Health Specialists

ABSTRACT

The examination of disease clustering has become a flourishing area in medical research during recent decades. The term cluster usually refers to uncommon diseases of non-infectious origin such as leukaemia, spontaneous abortion and suicides, which are repeatedly supposed to be due to environmental exposures. The aim of the present article is to discuss some of the most important fundamental issues surrounding this concept for the public health specialists within the Middle East region.

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Key words: General cluster, specific cluster, geographical epidemiology

Introduction

The examination of disease clustering has become a flourishing area in medical research during recent decades[1]. Generally speaking, the search for disease clusters is one of the branches of geographical epidemiology[2]. The term cluster usually refers to "uncommon diseases of non-infectious origin (e.g., leukaemia, spontaneous abortion, suicides), which are often perceived to be due to environmental exposures"[3].

Clusters of health events are often reported to health authorities especially within developed countries[4]. Although evidence suggests that only a small fraction of such reports are likely to lead to the identification of a real disease cluster[5], health authorities should investigates cautiously such reported clusters further for two obvious reasons: Firstly, time and space clustering may suggest that there would be some social, economic, cultural, etc. predisposing factors, which affect the occurrence of disease. Secondly, it can also guide an appropriate response for relieving communities from the fear of a perceived or real disease cluster.

Given the importance of cluster studies in both developed and developing countries, the aim of the present article is to discuss some of the most important fundamental issues surrounding this concept for the public health specialists especially within the Middle East region. To fulfil this demand, the article begins with the existing definitions of cluster, and then it moves to discuss different types of cluster and their related statistical issues, and also diverse scenarios in cluster investigations. Finally, it ends by providing some guidelines for dealing with a cluster more appropriately.

Definition of Cluster

There are different definitions of cluster. For instance, Knox (1989) defined a cluster as: "a geographically bounded group of occurrences of sufficient size and concentration to be unlikely to have occurred by chance[6]." Whilst spatial cluster is the main focus of this definition, Last (1995) tries to define this term more generally to include both temporal and spatial aspects of a cluster. He defined a cluster as: "aggregation of relatively uncommon events or diseases in space and/or time in amounts that are believed or perceived to be greater than could be expected by chance[7]."

Similarly, Gerstman (1998) defined cluster as: "A close grouping of disease or disease-related events in space, time, or both space and time and is usually reserved to describe the aggregation of rare diseases such as specific forms of cancer[8]." Rothman (1990) also states that: "Clustering in both space and time, or space-time clustering means that the incidence rates are temporarily higher in some places than in others, with the places that have a high incidence rate changing with time[9]." Furthermore, the definition which refers to it earlier in the first paragraph of the introduction, focuses on the non-infectious origin of a cluster[3].

Different types of cluster and their related statistical issues

It would be possible to classify types of clustering studies into general and specific. General or non-specific clustering is the analysis of the overall clustering tendency of the disease incidence in a study region. It

should be noted that the investigators of the general clustering do not seek to determine the exact locations of clusters but simply to assess if clustering is noticeable in the study region. On the contrary, specific studies are designed to determine the precise location of the clusters[10].

During recent years there has been a rapid expansion in the number of statistical tests for detecting of both general and specific diseases clusters. These tests have become more specially designed to encompass the particular disease-environment interactions[11&12]. However, caution is required in the application of such tests in order to avoid 'false positives' results i.e. detecting an unreal cluster as a real one[13].

The types of statistical tests for both kinds of disease clusters are dependent on the types of data which might include point and area data[1]. Each item of health data, such as population or environmental exposure, may be connected with a point e.g. a home or an area e.g. a district[14].

General cluster in area data implies that given an event e.g. suicide, the rates of it within neighbouring areas are likely to be more similar than those in distant ones[15&16]. In such situations detecting a cluster is accomplished by the use of spatial autocorrelation statistics[17]. The two most commonly used spatial auto correlation statistics for detecting general clustering in area data are the *I* statistic, developed by Moran[18] and Geary's c statistic[19].

On the contrary, specific clusters in area data search for local clustering e.g. hot spots of high or low values by finding any association between a value at a specific area and values of neighbouring areas[1]. There is also a number of spatial autocorrelation statistics available e.g. Getis and Ord's G* statistic for detecting such specific clusters[20&21].

Tests for the clusters detection in point format data are more frequent than those for area data[1]. To name a few, Cuzick and Edwards'[22] method examines the k nearest neighbours of each case in order to determine global clustering. The geographical analysis machine[23] and the spatial scan statistic[24] also try to detect the localised clustering by drawing predefined circles over the area of study and compare the risk of disease inside and outside of each circle.

Diverse scenarios in cluster investigations

It would also be possible to categorise diverse scenarios in cluster investigations into three situations as follows[25]:

Within the first scenario no clustering has been already detected within the population under study. Therefore, the question of whether or not a cluster is occurring is being approached a priori.

The second scenario is similar to the first in that nothing is known about the occurence of clusters in the population. However, there is a specific hypothesis to be investigated e.g. leukaemia risk is associated with closeness to a nuclear power plant.

In the third scenario, a disease cluster has already been detected within the population under study. Therefore, a posteriori or post hoc approach is selected in order to determine the realness of the cluster and/or to provide an explanation for it.

One should bear in mind that the problems of interpretation of each cluster, is crucially dependent on its scenario. For instance, it can be only possible to infer the conventional P value in relation to a priori hypotheses[5].

How to deal with a cluster?

In order to appropriately respond to the reports of the clusters a comprehensive approach is needed. For instance, the recommended approach by the US Centers for Disease Control and Preventions (CDC) consists of a four-stage process, which includes: primary response, evaluation, major feasibility study, and etiologic study. It should be noted that each step provides opportunities for collecting data and making informative decisions in order to stop or carry on the investigation[4].

It is also suggested that to implement such comprehensive approach successfully each health authority should have an interior management system. Such a system involves the establishment of a central point of responsibility and control. Furthermore, written working procedures and devoted resources might have immense value[4].

Conclusions

The investigations of suspected disease clusters due to environmental exposures are often originated in response to public anxiety within developed countries[4&5]. This makes public health specialists within such countries examine the alleged clusters from different perspectives in order to prepare a more appropriate plan for dealing with these events and their perceived risks among the community[26&27].

Given the recent environmental changes within developing countries including countries within the Middle East region, it seems that public health specialists in these countries also have to take the issue of cluster investigation more seriously. They should be aware that when such investigations become informative, that the following criteria are met: "chemical exposures are documented, routes of human exposure are traced, sub-populations at highest risk are identified, reliable denominator data are available, the diagnosis of the outcome has been consistent over time, and specific health outcomes are studied[8]."

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Peripheral Giant Cell Granuloma: A Case Report

ABSTRACT

Objective: Peripheral giant cell granuloma (PGCG), also known as osteoclastoma, giant cell reperative granuloma, or giant cell hyperplasia, is a reactive exophytic lesion observed in the oral cavity. We report an extremely rare case of PGCG.

Method: The case is discussed in light of the information in the literature.

Results: A 30-year-old female patient admitted to our clinic with the complaints of painless swelling and gingival growth underwent total excision of an exophytic lesion causing purplish-red colored gingival growth found during clinical examination in the upper jaw in the region of right vestibular 1st molar tooth. Histopathological examination revealed PGCG.

Conclusion: We wish to stress that effective treatment of PGCG requires not only complete excision of the lesion, but also elimination of irritating factors.

Keywords: Peripheral giant cell granuloma, gingival hyperplasia, gingival mass, benign tumor, aggressive periodontitis.

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Introduction

Giant cell granulomas of the jaw develop in two forms, namely the peripheral giant cell granulomas (PGCGs) and central giant cell granulomas. The peripheral type develops in the gingiva and alveolar process, while the central type originates from the bone. Both lesion types are observed most commonly in females, and in the mandible. Etiologically, they have been proposed to have hormonal, traumatic or neoplastic origins[16].

The PGCGs, also known as osteoclastomas, giant cell reperative granulomas, or giant cell hyperplasias, are reactive exophytic lesions found in the oral cavity. Although irritants or aggressive factors such as trauma, tooth extractions, badly finished fillings, plaques, dental calculi, chronic infections, or the effects of nutrients may be the causes, their etiology is not completely known[5,6,9,10]. The lesion, being more commonly encountered in adulthood, may recur following excisions unless the local traumatic factors and infections in its etiology are eliminated. The PGCGs are observed frequently in the oral mucosa, forming tumor-like masses. Rather than being genuine tumors, they are pathological reactive hyperplasic lesions developing as a result of abnormal repair[8]. They are also known as giant cell epulis or myeloid epulis[15].

In this case report, our aim is to describe a patient with gingival swelling originating in the upper jaw.

Case Presentation

A 30-year-old female patient was admitted to the Otorhinolaryngology Outpatient Clinic in Varto State Hospital with the complaints of painless swelling and gingival growth in the anterior region of upper jaw. The swelling had been present for 2 months, and had grown progressively.

Intraoral examination revealed a purplish-red colored, stalked exophytic lesion with a hard appearance extending from the midline of palate and alveolar crest in the region of the right vestibular 1st molar tooth in the upper jaw, accompanied by dense dental stones, while the gingiva were found to be hyperemic, edematous and inflamed. The mucosa overlying the lesion was found to be ulcerated due to trauma caused by the lower teeth (Figure 1). Palpation of the mass revealed no fluctuations. The patient had insufficient oral hygiene. Anamnesis of the patient revealed no systemic diseases. Examination of the lymph nodes in the head-and-neck region revealed no lymphadenopathy. Her blood tests including complete blood count, thyroid function tests, liver function tests, and serum calcium, phosphorus, ionized calcium and parathormone levels revealed normal results.

The patient was informed regarding the diagnosis and treatment plan, educated about oral hygiene, and recommended to use chlorhexidine (CHX) 0.02% for 7 days (Figure 2). At the end of the treatment period, the case advanced to the surgical

phase, in which the aforementioned mass was completely excised with the aid of scalpels and monopolar cauterising under local anesthesia (Figures 3 and 4). Postoperatively, the patient was recommended to use CHX 0.02% shaking solution and to apply regularly for control visits.

Four months later, the lesion region was found to have recovered. Biopsy of the lesion revealed the typical histological appearance of PGCG (Figure 5).

Discussion

Giant cell lesions have a wide distribution spectrum, ranging from slowly growing asymptomatic radiolucent lesions to rapidly growing aggressive lesions, which are characterized by pain and root resorption and have a high recurrence potential[1,7,16]. PGCGs are lesions of the gingiva and oral mucosa that are thought to arise from the periodontal ligament or periosteum. These lesions have been reported to have a rate of being encountered, ranging from 5.1% to 43.6% among all reactive growths[9]. Such lesions are observed at a rate of 40% between 40 and 60 years of age, and at a rate of 20-30% between 10 and 20 years of age. The PGCGs have been reported to be two times more common in females than males, and more frequent in the lower jaw than the upper[1,17]. In our case, the female sex and age of patient were compatible with the literature.

Although Pindborg[13] has described that giant cell lesions are localized in the premolar and molar regions, these lesions are observed generally in the gingiva and alveolar regions of incisor and canine teeth. In our case, the lesion was observed between the central and premolar-molar teeth in the upper jaw.

In our patient, absence of pain associated with the lesion may have arisen from the fact that the lesion might not have reached the level of occlusion and therefore might have not been affected by traumatic forces arising during chewing. Bodner et al[2] have found an inverse correlation between the level of oral hygiene and size of PGCG. The patient presented in this report had had complaints of gingival hemorrhage beginning in her thirties, and had not brushed her teeth at all. These factors might have rendered the periodontal picture more dramatic. Likewise, it may be proposed that insufficient oral hygiene of the case and the presence of periodontitis might have been effective in development of PGCG.

As PGCGs originate from soft tissues, it has been suggested that radiographic findings are not notable[17]. However, irritating factors in occasional cases may give significance to radiographic findings. Our PGCG case did not undergo X-ray examination as she was in the second month of pregnancy, and as the teeth in the region of lesion were not loose, the lesion had a stalk, and the patient attended at an early stage, suggesting that the lesion was benign.

The maximum capacity of PGCGs to enlarge is currently unknown; however, Kfir et al[10] have reported that the lesions may enlarge from 0.1 cm to 3 cm, and 94% of these lesions are smaller than 1.5 cm. In our case, the lesion had a size of 2.5-3 cm and was evaluated as giant cell epulis.

It has been reported that PGCGs may cause mobility and/or displacement in neighboring teeth[3]. As our case was encountered at an early stage, no displacement or luxation was observed in the teeth associated with the lesion.

Though the etiology of PGCGs is not precisely known, local irritating factors such as tooth expulsion, maladapting prostheses and restorations, plaques, calculi, and nutrient debris play significant roles[2,11,15]. In our patient, the oral hygiene level was considerably low, and in her personal history she had tooth expulsion 2 months prior to her initial admission. Also, she had local irritating factors such as debris, plaques, and calculi.

Lesions similar to PGCGs may rarely be encountered in patients with hyperparathyroidism. These lesions are named Brown tumors. However, Brown tumors associated with hyperparathyroidism are more commonly localized within bones, and display similarity to central giant cell granulomas[12]. The patient in our case has been found to be systemically healthy. In a few cases, giant cell granulomas have been reported to be oral findings of hyperparathyroidism. In their PGCG case studies, Giansanti et al[6] have found no association between hyperparathyroidism and PGCGs.

Overall, hyperparathyroidism has been observed in less than 10% of all cases[14]. Likewise, the tests in our case revealed no such disease.

On the other hand, Gunhan et al[8] have proposed that these lesions might be affected by sex hormones. According to these authors, giant cells are potential targets for estrogen activation. In our case, the patient was in the 2nd/3rd months of pregnancy, and the lesion had a progressive growth, both indicating a possibility of estrogen activation (comparison of figures 1 and 2).

The therapeutic approach has been reported in several cases to be excision of the PGCG with scalpels or CO2 lasers(4). Although treatment with laser has limited applications in lesions neighboring the bone structures, it has advantages such as less hemorrhage in the site of surgery, absence of pain, and increasing sight. Eversole et al[5] have reported that the risk of recurrence ranges from 5 to 11%. In our case, the priority in our therapeutic approach was towards eliminating the growth in the gingiva, and the lesion was surgically completely excised, followed by curetting the neighboring structures. As a result of a 4-month follow-up, no recurrence was observed.

Giant cell granuloma lesions should be carefully diagnosed and excised in an as early as possible stage, via a careful treatment plan, and should be histopathologically distinguished from similar lesions. Our case was caught at an early stage, and a successful result was obtained with performing the required tests, therapy, and follow-up. In conclusion, we wish to stress the importance of not only successful treatment of these lesions that can be encountered in the oral cavity, but also their follow-up regarding the risk of recurrence.

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Figure 1: The appearance of lesion on first admission in the 2nd month of pregnancy

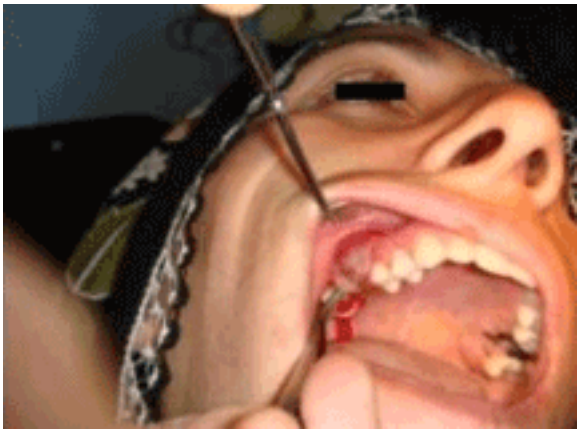

Figure 2: The preoperative appearance in the 3rd month of pregnancy

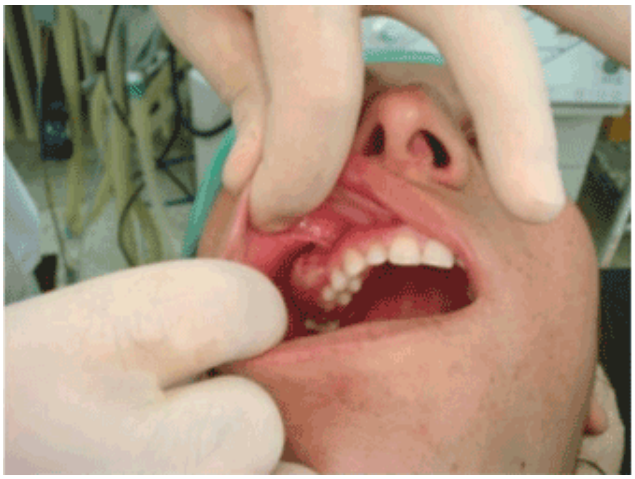

Figure 3: The appearance of the location of the mass following its removal

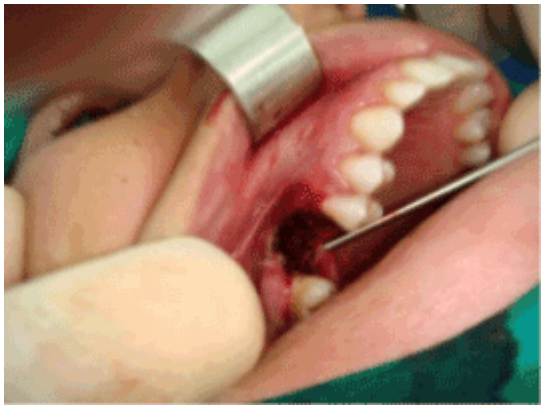

Figure 4: Macroscopic appearance of the lesion.

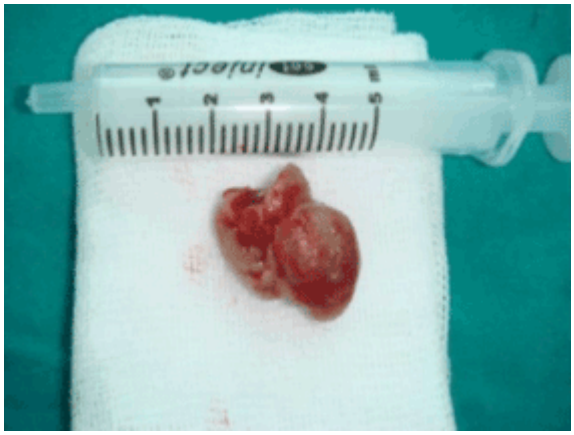

Figure 5: Numerous giant cells, some with osteoclast-like appearance, within the stroma that appear to be hemorrhagic and covered with squamous epithelium (H-E ×125).