

Obesity: An emerging disease

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Abstract

Obesity is rapidly becoming an emerging disease in developing countries due to the increasing westernization of societies and change in the lifestyle. The etiology of obesity is said to be multifactorial, with a combination of genetic and environmental factors. Literature has been extensively reviewed to provide a broad overview of obesity. Data for this review were obtained from original articles, review articles and textbooks. Internet search engines were also employed. The years searched were from 1993 to 2008. Obesity, classified in terms of the body mass index and the waist-hip ratio, has several associated co-morbidities such as diabetes mellitus, hypertension, degenerative osteoarthritis and infertility. In Nigeria, there is limited information on obesity. A literature review on obesity is necessary to improve the knowledge about obesity in developing countries, its prevention and its management.

Key words: Disease, emerging, obesity

Date of Acceptance: 16-Mar-2011

Introduction

Obesity is a non-communicable disease which is gaining increasing importance globally and is a rapidly emerging disease in the developed world. It is a chronic condition characterized by an accumulation of body fat.^[1] Obesity is one of the most important preventable diseases in developed countries. The prevalence of the disease is increasing in both industrialized nations and in those undergoing alterations in diet and activity patterns as a consequence of adoption of the western culture.^[2]

Etiopathogenesis of obesity

The origin of obesity is unclear, but it appears to be multifactorial and requires the continuous differentiation of new adipocytes throughout life.^[3] This process of adipocyte differentiation from preadipocytes has been shown to be controlled by members of the peroxisome proliferator-activated receptor (PPAR γ). There is said to be a genetic basis for the development of obesity.^[2,4] Polymorphisms in various genes controlling appetite, metabolism, and adipocytokine release, such as adiponectin, predispose to obesity. Some genetic conditions that feature obesity have been identified (such as Prader-Willi syndrome and

melanocortin receptor mutations), but known single-locus mutations have been found in only about 7% of obese individuals.^[5,6]

There may be other factors influencing the expression of genetic characteristics that induce weight gain or allow weight loss. Leptin is a protein hormone that is produced by adipocytes.^[6] It regulates body weight, metabolism and reproductive function.^[7] It seems to provide the brain with needed feedback concerned with the dynamic stability of body fat mass. A loss of body fat leads to a decrease in leptin, which in turn leads to a state of positive energy balance, that is, food intake exceeds energy expenditure.^[8]

In obesity, there is excessive adipose tissue mass.^[3] Obesity results when the body's intake is greater than output over a period of time due to a sedentary lifestyle.^[9] Thus, dietary factors and physical activity patterns play a strong role in obesity, and this is called exogenous obesity.^[5] Obesity is positively associated with dietary factors such as increased

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Access this article online

Quick Response Code:



Website: www.njcponline.com

DOI: 10.4103/1119-3077.91741

PMID: 22248935

fat intake, low fiber consumption, increased hidden sugars in prepared foods, reduced amounts of unrefined sugars, and inadequate fruit and vegetable intake.^[9] The daily eating pattern also seems to be associated with weight change, especially high calorie diet, which can be measured in several ways:^[9] actual weighing of food through direct observation, 24-h our dietary recall using food models to estimate calories, and daily eating patterns. The most objective is the direct observation method. In the absence of the highly objective methods, the dietary eating pattern could be used to have a rough guide in a clinic setting, for rapid assessment of the patient's diet.^[10]

A physical activity pattern, such as a sedentary lifestyle, is also associated with weight change. The physical activity pattern has important influence on physiological regulation of body weight. Decreased physical activity plays an important role in obesity. In South West Nigeria, there is no exercise culture, especially among women. However, women are engaged in some physical activity such as walking long distances to hawk their wares, which are strenuous, and even at times deleterious to health. Other lifestyle factors which could cause obesity include insufficient sleep, endocrine disruptors—food substances which interfere with lipid metabolism, decreased variability in ambient temperature and decreased rates of smoking which suppress appetite. Increased use of medication that leads to weight gain such as steroids and some antidepressants may cause weight gain. Increased distribution of ethnic and age groups that tend to be heavier, pregnancy at a later age and intrauterine and intergenerational effects also contribute to the etiology of obesity.^[5] Psychological factors as well as menopause also predispose to weight gain.^[11] Endocrine problems such as Cushing's disease and polycystic ovary disease may lead to obesity. Positive natural selection of people with a higher body mass index (BMI), assortative mating, and heavier people forming relationships with each other are other factors which have been implicated in the etiology of obesity. Other social determinants to obesity include the income differential, marital status, and BMI change in friends, siblings or spouse irrespective of geographical distance.^[5] Childhood and adolescent obesity have also been identified as risk factors for obesity in adulthood.^[12]

Cultural attitudes also influence obesity.^[4] For example, in a part of Nigeria, pubescent daughters of the wealthy are sent to fattening huts before marriage.

Classification of obesity

Obesity can be classified into central or peripheral obesity. In central obesity, otherwise called "android" obesity, the distribution of fat is commonly on the upper part of the trunk (the chest and abdomen) and is more common in the males.^[2] Android obesity is more clearly associated with disordered lipid and glucose metabolism and diseases like diabetes mellitus, gout, atherosclerosis, osteoarthritis, cardiovascular disease

especially hypertension, and some cancers.^[2,13] However, in the peripheral or "gynecoid" type of obesity, the distribution of fat is mainly on the hip and thighs and is more common in females.^[2,14] Before the menopause, lipid assimilation is favored in the abdominal and femoral depots. However, after the menopause, these differences in fat metabolism between the abdominal and femoral sites disappear. The differences seen in the metabolism of fat suggest that female hormones are responsible before the menopause.^[2]

There are various measures of obesity, and the BMI is a very useful and common one. It is a mathematical formula that is highly correlated with the body fat.^[15] BMI is calculated as weight in kilograms divided by the square of the height in meters.^[16] The BMI takes into account both frame size and body composition and is considered to provide a realistically achievable range of healthy weights and is a predictor of dangers associated with obesity.^[2,17-24] A BMI less than 18.5 kg/m² is underweight. Normal BMI ranges between 18.5 and 24.9 kg/m². A BMI greater than or equal to 25 kg/m² is overweight. Obesity is defined as having a BMI of 30 kg/m² or more. There are three grades of obesity:^[1] grade I, which is a BMI equal to or greater than 30 kg/m²; grade II, which is moderate obesity with a BMI of 35–39.9 kg/m²; and grade III, which is extreme or morbid obesity, and the BMI is greater than or equal to 40 kg/m².

Waist–hip ratio (WHR) is another means of knowing if a person is obese and is calculated by using the ratio of waist circumference to hip circumference.^[16] Waist and hip measurements are simple and their expression as a ratio (WHR) makes it possible to do studies on a large number of obese individuals. Using this method of calculation of WHR, extreme gynecoid distribution has a WHR of about 0.5, whilst WHR is 1.0 in the android type.^[2,25-28] Another classification of WHR is 0.76–0.80 which is normal, 0.81–0.86 which is moderate obesity, while above 0.86 is severe obesity.^[16]

Prevention of obesity

Primary and secondary prevention should be emphasized in the prevention of obesity. The healthcare costs of obesity are considerable, especially when complicated by associated conditions such as diabetes mellitus, osteoarthritis,^[18] hypertension, gall stone disease, post-menopausal breast cancer, and colon cancer.^[29] Prevention of obesity is thus cost effective and should begin in early childhood by instilling healthy patterns of exercise and diet.^[9]

It is important to begin prevention of obesity early because prevalence of childhood obesity is increasing throughout the world. In some developing countries, there is an increasing trend toward the western lifestyle such as adoption of the dietary pattern of eating "fast foods", which are high in carbohydrate and fat. Within the United Kingdom, estimates of obesity range from 6% in preschool children to

17% by the age of 15 years. Schools provide an opportunity for preventing and treating obesity and can be employed for primary prevention through early detection of obesity. The use of health promotion, intended to influence dietary and physical activity behavior as a method of prevention of obesity, can also be employed.^[30]

Co-morbidities of obesity

There are several medical consequences of obesity.^[5,6,19,31] These co-morbidities affect various systems of the body.^[32-36] Cardiovascular system complications seen in obesity include hypertension. It is known that there is an increase of 3 mmHg and 2 mmHg in systolic and diastolic pressures, with every 10 kg rise in body weight.^[15,25] Cerebrovascular disease, ischemic heart disease and congestive cardiac disease are also the cardiovascular complications of obesity.^[37] Other complications include varicose veins and pulmonary embolism. There is a three times increased risk of premature death with obesity.^[9] In the endocrine system, type 2 diabetes mellitus is a co-morbidity which is commoner in obese patients.^[5,31] Obese patients are also prone to dyslipemia.^[38] Obesity increases the incidence of and mortality rate in type 2 diabetics.^[37,39] Obesity also leads to infertility, menstrual irregularities and polycystic ovarian syndrome (PCOS).^[2] Respiratory complications also occur in obese people, which include obstructive sleep apnea, central sleep apnea, or a combination of the two.^[40] Asthma and obesity hypoventilation syndrome could also occur in obese people.^[5]

The renal and genitourinary system in obesity could be affected in several ways: urinary incontinence,^[9] erectile dysfunction, chronic renal failure, prostate cancer and hypogonadism in males, breast,^[25] cervical, ovarian and uterine cancer in females, and still birth.^[31] Gastrointestinal system complications include gastroesophageal reflux disease (GERD), fatty liver disease, and hernia. Colorectal and gall bladder cancers can also develop.^[25] Cholesterol supersaturation of the bile in obese individuals causes cholelithiasis.^[25,35]

In the dermatological system, skin diseases such as intertrigo and cutaneous moniliasis increase.^[28,38] Obese people may also have stretch marks, cellulitis and carbuncles. Complications in the musculoskeletal system include osteoarthritis,^[41-43] immobility, low back pain, and hyperuricemia which predisposes to gout.^[5]

The neurological system is affected by cerebrovascular disease and carpal tunnel syndrome.^[5,37] There are also several psychological problems of obesity. Depression, low self-esteem,^[2,9] body dysmorphic disorder and social stigmatization are some of these psychological problems faced by the obese individual. Obesity is a cause of significant mortality and morbidity and generates great social and financial costs.^[44]

Management of obesity

It is important to emphasize to the individual that management of obesity is lifelong.^[45] Health education and counseling have a remarkable role in encouraging adoption of lifestyle modifications. Health education should be offered to the individual about the etiology and treatment options for obesity. Referral may be necessary to other members of the multidisciplinary team, such as the physiotherapist, occupational therapist, the social worker, the dietician, and the clinical psychologist.^[41]

Lifestyle modification

Lifestyle modification in the management of the patient as a whole focuses on dietary modification and exercise. Weight reduction can be done through dietary modification and exercise. Dietary modification follows satisfactory counseling and involves a 24-hour activity associated dietary recall. This recall includes detailed information about the meal schedule, and the quantity and quality of food intake. The dietary recall is a verbal report of food intake over the past 24 hours, or "yesterday", and may help in nutritional intervention.^[45] The role of the physician is to set reasonable weight loss goals. A realistic goal is a weight loss of 0.5–1 kg per week. This weight loss is achievable using diets of 1000–1500 kcal/day, which is known as low calorie diet. All obese people lose weight on a low energy intake.^[38] The physician should aim for a BMI of 25 kg/m² for the patient who wants to undergo dietary modification. After 6 months of therapy, the rate of weight loss on this regimen usually declines and then plateaus.^[45] The second phase of weight loss therapy is the maintenance phase and this second stage should take priority. The physician also helps the patient select an appropriate weight loss program, and if simple diet sheets are not effective for dietary modification, the Family Physician refers the patient to a dietitian.^[30,45] Very low calorie diets that are less than 800 kcal/day can be used to treat morbid obesity. Very low calorie diets, however, generally have a limited place in the management of obesity as the pattern of eating cannot be maintained and rebound weight gain is seen on stopping this type of diet.^[45] Once a patient has lost weight, diet still needs to be monitored by the physician, and also by behavioral and group therapy. There is, however, little evidence to show that dietary advice by physicians is heeded.^[9] Most influence on diet comes from national food policy, price of food, advertising, general education and cultural influences.^[4,9]

Behavioral therapy is effective individually and in groups, when combined with low calorie diets.^[45] Behavior change strategies involve techniques in stimulus control. The simplest form of behavior therapy involves advice to avoid situations that tempt overeating. Other forms of advice are keeping food out of sight or eating only at the table.^[9,45] Practice eating management such as eating slowly is also included under behavior therapy. Behavior substitution can also be done, such as exercising instead of eating when angry.

Self-monitoring could be done by keeping food and exercise diaries and rewards can be given as a form of behavioral therapy. Prevention of relapse in obesity may require group therapy. Cognitive restructuring could also be included such as use of personal self-talk that focuses on progress rather than failure.^[45]

Group activities such as weight watchers, who offer weekly meetings focused on nutrition, behavior modification and relapse prevention, can be joined. Exercise is also encouraged in these weight loss groups. There is a one-time registration fee for the weight watchers and a weekly fee, and when participant reaches goal weight, he or she becomes a lifetime member and may attend monthly meetings without charge as an incentive and encouragement to the new members.^[9,45]

Exercise is an integral part in lifestyle modification of obesity.^[9,46] Exercise is a useful treatment for many other conditions managed by physicians. Regular exercise reduces the risk of coronary heart disease, diabetes mellitus, obesity, colon cancer and osteoporosis.^[45,46] In hypertension, exercise can result in a reduction of 10 mmHg in systolic and 8 mmHg in diastolic blood pressure. In hypercholesterolemia, exercise causes high-density lipoproteins (HDL) to increase and low-density lipoproteins (LDL) to decrease, therefore reducing the risk of atheroma formation. Exercise also helps to reduce anxiety and the intensity of depression.^[46]

Assessment of physical activity includes asking about regular exercise such as walking to work, to the market and whether in active employment. The Allied Dunbar National Fitness Survey Activity levels are graded from 0 to 5. It measures an activity of 20 min on each occasion in the previous 4 weeks: 5 stands for greater than or equal to 12 occasions of vigorous activity; 4 stands for greater than or equal to 12 occasions of a mix of moderate and vigorous activity; 3 stands for greater than or equal to 12 occasions of moderate activity; 2 stands for 5–11 occasions of a mix of vigorous and moderate activity; 1 stands for 1–2 occasions of a mix of moderate and vigorous activity; and 0 stands for no moderate or vigorous activity of greater than 20 min duration. The target physical activity level as per the age group is 5 for 16–34 years, 4 for 35–54 years, and 3 for 55–74 years.^[46]

Negotiating change in lifestyle is possible by encouraging sedentary people to increase their activity levels. As with all lifestyle interventions, the patients must want to change if they are to alter their lifestyle. If exercise levels are satisfactory, congratulate and inform the patient about the benefits of exercise. If the levels of exercise are unsatisfactory, encourage the patient to increase the physical activity and support the individual with health education leaflets. There is more success if exercise recommended is moderate, if the exercise does not require a special facility, and if the type of exercise can be incorporated into daily life routines such as walking to work.^[46] Regular aerobic exercise also helps to reduce weight and improve health. The Family Physician should tailor advice to

suit the individual and local facilities available. Light exercise, such as aerobics, is as effective as walking to some.^[9] Swimming and cycling are recommended for obese women due to lack of weight bearing during the activity. Other adjuncts to lifestyle modification for obesity include drug therapy.

Drug therapy

Medications for treatment of obesity are formulated to reduce energy intake, increase energy output or decrease the absorption of nutrients.^[46] There are several types of drugs that can be used in the management of obesity.^[9,46] Methylcellulose, which works by decreasing absorption of nutrients, has been tried but found ineffective for treatment of obesity. Phenteramine, a catecholaminergic drug, is used as an appetite suppressant and is only licensed for a 12-week use in patients with moderate to severe obesity. Phenteramine is, however, not recommended for the routine management of severe obesity.^[9] Rapid weight relapse usually occurs after short-term use of an appetite suppressant and Phenteramine also carries the added risk of pulmonary hypertension, which is rare and insidious in onset.

Orlistat (Xenical) is a gastric and pancreas lipase inhibitor. Orlistat decreases fat absorption in the intestines. Patients taking this drug are also started on a medical action plan, which includes diet and exercise. Orlistat is expensive and is only used for motivated patients (those who have lost over 2.5 kg in the previous month and have a body mass index over 30 kg/m²).^[9] Anti-depressants may also be needed as an adjunct therapy for obesity, as some of these obese patients may be depressed because of their weight and some may end up eating more to ease their depression.^[45] However, it is to be noted that some of these anti-depressants, especially Amitriptyline, could cause increased appetite and worsen obesity.

Surgery

The physician should only consider referral for obesity as a last resort, if behavioral and dietary modification have failed and BMI is over 40 kg/m². Wiring of the jaws together so that only liquid diet can be taken through a straw can be done as a surgical modality while managing morbid obesity.^[2] Gastroplasty is the most common procedure done surgically for managing obesity in developed countries.^[9] Mortality following gastroplasty is high.^[9] Other surgical modalities for management of obesity are gastric bypass,^[47] adjustable silicone gastric banding (ASGB), and vertical gastric banding (VGB).^[35] Surgical excision of excess fat from various sites has also been advocated and tried, but usually fat reappears at other sites and the scars produced following the excision of fat may be uncosmetic.^[20]

Follow-up

The patient should be followed-up on a regular basis to encourage compliance with lifestyle modification and other forms of treatment modalities for obesity. It is essential to

maintain the patient's motivation throughout the course of therapy and even as a lifestyle change.^[9] Ongoing follow-up has been shown to help sustain weight loss in obesity.

Conclusion

Obesity, like other emerging diseases requires more systematic investigation to establish facts on it in developing countries, especially in view of its increasing importance globally.

References

- Azinge EC. Obesity and its complications in 30 Nigerian patients in Lagos. *Niger Q J Hosp Med* 1997;7:49-52.
- Anate M, Olatinwo AW, Omesina AP. Obesity an overview. *West Afr J Med* 1998;17:248-54.
- Antonio JV, Robert VC, Mercedes J, Ariel W, Walter JP, Jose FC, *et al.* Peroxisome proliferator-activated receptor gene expression in human tissues: Effects of obesity, weight loss, and regulation by insulin and glucocorticoids. *J Clin Invest* 1997;99:2416-22.
- Brian Y. Does food advertising make children obese?. *Advertising and Marketing to Children*; 2003. p. 19-26.
- Ishola AG. Obesity: As a disease. *Niger Clin Rev J* 2008;1:5-12.
- Okubadejo NU, Fasanmade OA. Concomitant hypertension and type 2 diabetes mellitus in Nigerians: Prevalence of obesity and its indices compared to normotensive diabetics. *Niger Med J* 2004;45:79-83.
- Oyekan AO. Genetics of food intake, body weight and obesity-role of Leptin. *Niger Med Pract* 2005;47:108-12.
- Paul FD. Weight management and obesity. In: Sloane P, Slatt L, Curtis P, editors. *Essentials of Family Medicine*. 3rd ed. William and Wilkins; 2004. p. 679-97.
- Chantel S, Everitt H, Birtwistle J, Stevenson B. Obesity. In: Simon C, *et al.*, editors. *Oxford Handbook of General Practice*. 1st ed. Oxford: Oxford University Press; 2002. p. 166-7.
- Bakare FA. The prevalence of overweight and obesity among adult market women in Ibadan, Oyo State. A project submitted in partial fulfillment of the Master Of Science (M.Sc) degree in Human Nutrition. Department of Human Nutrition, College of Medicine, Ibadan, Nigeria: University of Ibadan; 1998. p. 13, 14, 67.
- Payne P, Curtis P. Promoting health for women at menopause. In: Sloane P, Slatt L, Curtis P, editors. *Essentials of Family Medicine*. 3rd ed. William and Wilkins; 2004. p. 179-83.
- Abdulbari B. Prevalence of obesity, overweight, and underweight in Qatari adolescents. *Food Nutr Bull* 2006;27:39-45.
- Clark DO, Mungai SM. Distribution and association of chronic disease and mobility across four body mass index categories of African-American women. *Am J Epidemiol* 1997;145:865-75.
- Ogunranti JO. Central and appendicular girth anthropometric measurements in Nigerian women. *Afr J Med Sci* 1994;23:315-8.
- Luke A, Darazo-Arvizu R, Rotimi C, Prewitt TE, Forrester T, Ogunbiyi OJ, *et al.* Relation between Body Mass Index and Body Fat in Black Population Samples from Nigeria, Jamaica, and the United States. *Am J Epidemiol* 1997;145:620-8.
- Ojoawo AO. Anthropometric indices in patients with knee osteoarthritis as observed in Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife. *J Niger Med Rehabil Ther* 2002;7:26-30.
- Wardle J, Johnson F. Weight and dieting: Examining levels of concern in British adults. *Int J Obesit* 2002;26:1144-9.
- Seidell JC. Time Trends in obesity: An epidemiological perspective. *Hormone Metab Res* 1997;29:155-8.
- Philbin EF, Ries MD, Groff GD, Sheesley KA, French TS, Pearson TA. Osteoarthritis as a determinant of an adverse heart disease risk profile. *J Cardiovasc Risk* 1996;3:529-33.
- Alessi MC, Henry M, Morange P, Nalbou G, Peiretti F, Juhan-Vague I. Production of Plasminogen activator inhibitor 1 by human adipose tissue: Possible link between visceral fat accumulation and vascular disease. *Diabetes* 1997;46:860-7.
- David FW. Descriptive epidemiology of body weight and weight change in US adults. *Ann Intern Med* 1993;119:646-9.
- Pereira MA, Jacobs DR, Van HL, Slaterry ML, Kartashov AI, Ludwig DS. Dairy consumption, obesity and the insulin resistance syndrome in young adults. *JAMA* 2002;287:2081-8.
- Zachary-Jennings C. Obesity and infertility. *Postgrad Doct Afr* 2000;22:34-8.
- Van De Walle P, Van O. Hand assisted laparoscopic vertical banded gastroplasty: Technique and analysis of the first 140 cases. *Obesit Surg* 2002;12:628-33.
- Pi-Sunyer FX, Xavier F. Medical hazards of obesity. *Ann Intern Med* 1993;119:655-60.
- Shiriki KK. Special issues regarding obesity in minority populations. *Ann Intern Med* 1993;119:650-54.
- Wilks R, McFarlane-Anderson N, Bennet F, Fraser H, McGee D, Cooper R, *et al.* Obesity in peoples of the African diaspora. *Ciba Foundation Symp* 1996;201:37-48; discussion 48-53, 188-93.
- Okosun IS, Forrester TE, Rotimi CN, Osotimehin BO, Muna WF, Cooper RS. Abdominal adiposity in 6 populations of West African descent: Prevalence and population attributable fraction of hypertension. *Obesit Res* 1999;7:453-62.
- Swinburn B, Ashton T, Gillespie J, Cox B, Menon A, Simmons D, *et al.* Healthcare costs of obesity in New Zealand. *Int J Obesit Relat Metab Disord* 1997;21:891-6.
- Sahota P, Rudolf MC, Dixey R, Hill AJ, Barth JH, Cade J. Evaluation of implementation and effect of primary school based intervention to reduce risk factors for obesity. *BMJ* 2001;323:1027-32.
- Fadupin GT, Joseph EU, Keshinro OO. Prevalence of obesity among type 2 diabetics in Nigeria: A case study of patients in Ibadan, Oyo State, Nigeria. *Afr J Med Sci* 2004;33:381-4.
- Fries JF, McShane D. Reducing need and demand for medical services in high-risk persons: A health education approach. *West J Med* 1998;169:201-7.
- Himes CL. Obesity, disease, and functional limitation in later life. *Demography* 2000;37:73-82.
- Cottam DR, Schaefer PA, Fahmy D, Shaftan GW, Angus LD. The effect of obesity on neutrophil Fc receptors and adhesion molecules (CD16, CD11b, CD62L). *Obesit Surg* 2002;12:230-5.
- Albrecht RJ, Pories WJ. Surgical intervention for the severely obese. *Best Pract Res Clin Endocrinol Metab* 1999;13:149-72.
- Pi-Sunyer FX. Comorbidities of overweight and obesity: Current evidence and research issues. *Med Sci Sports Exerc* 1999;31(Suppl 1):S602-8.
- Fasanmade OA, Okubadejo NU. Magnitude and gender distribution of obesity and abdominal adiposity in Nigerians with type 2 diabetes mellitus. *Niger J Clin Pract* 2007;10:52-7.
- National Task Force on the Prevention and Treatment of Obesity. *Medical Care for Obese Patients: Advice for Health Care Professionals*. *Am Fam Physician* 2002;65:81-8.
- Orluwene CG, Ojule AC. Plasma cholinesterase activity in obese type 2 diabetics. *Port Harcourt Med J* 2006;1:39-43.
- Chantel S, Everitt H, Birtwistle J, Stevenson B. Exercise. In: Chantel Simon, *et al.* *Oxford Handbook of General Practice*. 1st ed. Oxford: Oxford University Press; 2002. p. 168-9.
- Chantel S, Everitt H, Birtwistle J, Stevenson B. Osteoarthritis. In: Chantel Simon *et al.* *Oxford Handbook of General Practice*. 1st ed. Oxford: Oxford University Press; 2002. p. 358-9.
- Hinton R, Moody RL, Thomas SF. Osteoarthritis: Diagnosis and therapeutic considerations. *Am Fam Physician* 2002;65:841-8.
- Sahyoun NR, Hochberg MC, Helmick CG, Harris T, Pamuk ER. Body mass index, weight change, and incidence of self-reported physician diagnosed arthritis. *Am J Pub Health* 1999;89:391-4.
- Rodin J. Cultural and psychological determinants of weight concerns. *Ann Intern Med* 1993;119:643-5.
- Rakel ER. Nutrition and Family Medicine. In: Rakel RE, editor. *Textbook of Family Practice*. 6th ed. Philadelphia: Saunders; 2002. p. 1107-58.
- Berke EM, Morden NE. Medical management of obesity. *Am Fam Physician* 2000;62:419-26.
- Ghasseman AJ, MacDonald KG, Cunningham PG, Swanson M, Brown BM, Morris PG, *et al.* The workup for bariatric surgery does not require a routine upper gastrointestinal series. *Obesit Surg* 1997;7:16-8.

How to cite this article: Ogunbode AM, Ladipo M, Ajayi IO, Fatiregun AA. Obesity: An emerging disease. *Niger J Clin Pract* 2011;14:390-4.

Source of Support: Nil, **Conflict of Interest:** None declared.