National Journal of Physiology, Pharmacy and Pharmacology

RESEARCH ARTICLE

A comparative study of age of menarche in private and government school-going girls

Smriti Sinha[1], Mohd Asghar Ali[2]

[1]Department of Physiology, School of Medical Sciences and Research, Sharda University, Greater Noida, Uttar Pradesh, India, [2]Department of Physiology, Mamata Academy of Medical Sciences, Hyderabad, Telangana, India

Correspondence to: Smriti Sinha, E-mail: drsmritisinha@gmail.com

Received: October 21, 2019; **Accepted:** November 13, 2019

ABSTRACT

Background: The menarche is known to assess the integrity of a woman's circuitry from brain to ovary to the uterus and its timing is determined by multiple factors, one of which is attainment of a particular waist-to-hip (W/H) ratio, suggesting that leptin conveys information about the fat distribution to hypothalamus to initiate the process of menarche. **Aims and Objectives:** This study aims to assess the mean age of menarche in this region (AOM) and to find if there is any difference between the AOM of government and private school-going girls and to find the relationship between AOM and various anthropometric measurements. **Materials and Methods:** A total of 250 randomly selected adolescent girls were asked to fill a pretested pre-structured questionnaire regarding anthropometric measurements, sociodemographic profile, and menstrual history. **Results:** The mean AOM was found to be 12.94 ± 0.74 years and the difference between the AOM of government and private schools is highly significant ($P < 0.001$) and W/H ratio is the single best predictor of the AOM. **Conclusion:** As childhood obesity and mental stress are on a rise worldwide, both these puberty accelerators are producing a new generation of girls who are sexually mature at a very tender age and this pubertal maturity should be matched by social maturity.

KEY WORDS: Age of Menarche; Waist-to-Hip Ratio; Body Mass Index; Government and Private School-going Girls

INTRODUCTION

Adolescence is characterized by tremendous pace of growth and change that is second only to infancy and is marked by an important milestone called puberty.[1] In a female, although menarche is a late marker of puberty, it is a well-validated indicator and an easily remembered event when compared to others in the process of female sexual maturity. The timing of menarche is different for each individual and is influenced by female biology, genetic, as well as environmental factors during childhood. Caren Craig said that age of menarche (AOM) is known to assess the integrity of a woman's circuitry from brain to ovary to the uterus. The average AOM has declined over the years at a rate of 4 months per decade till it stabilized at 13 years from an average of 17 years in 1830, leading to a changed definition of precocious puberty.[2] This early menarche leads to an extended lifetime exposure to ovarian hormones which ultimately predisposes to breast and ovarian carcinoma, polycystic ovarian syndrome, short adult stature, increased cardiovascular risks, and type 2 diabetes mellitus, but on the desirable side has been reported to increase the bone mineral density as well as bone microstructure.

Access this article online	
Website: www.njppp.com	Quick Response code
DOI: 10.5455/njppp.2020.10.1035613112019	

Neuroendocrinology of Menarche

Gonadotropin-releasing hormone (GnRH) stimulates the release of gonadotropins by 12–14 weeks of gestation and

the levels peak around midgestation and subsequently decline toward birth finally suppressed at term, due to increased production of fetal estrogen toward the end of gestation (negative feedback inhibition). This is required for masculinization or feminization of brain. After birth, the placental estrogens reduce, causing a lack of negative feedback and increase in gonadotropin levels again after a week of neonatal life. This is called "MINI PUBERTY" which is supposedly for the development of genital organs. This GnRH pulse generator which is a group of scattered neurons around the preoptic and rostral hypothalamus again undergoes a modicum of suppression to a mid-childhood nadir. According to GONADOSTAT THEORY, this quiescent period is due to a high sensitivity of GnRH neurons to low levels of estradiol released by prepubertal ovaries, exerted through gamma-aminobutyric acid (GABA). 1–3 years before onset of clinically evident puberty, low serum levels of luteinizing hormone (LH) during sleep become demonstrable which is pulsatile in fashion and reflect endogenous episodic discharge of GnRH, supposedly due to estradiol-kisspeptin positive feedback relationship. This nocturnal pulsatile secretion increases in amplitude and frequency and is later also evident during daytime and occurs at 90–120 min interval followed by a follicle-stimulating hormone pulse within 30 min. With passage of time, the daytime pulses increase while the sleep time pulses decrease, with eventual loss of diurnal variation. Now, this GnRH pulse generator being the epicenter of pubertal development is influenced by multiple neurotransmitters such as glutamic acid, kisspeptin, GABA, preproenkephalin, and factors produced by glial cells like transforming growth factor β. Kisspeptin neurons present in the anteroventral periventricular nucleus and arcuate nucleus of hypothalamus receives information from leptin which, in turn, activates the G protein-coupled receptor 54, which accelerates the pulsatility of GnRH pulse generator. Apart from leptin, kisspeptin gene also receives information from mammalian target of rapamycin, a serine kinase that operates as a sensor for cellular energy status and also from circulating estradiol.[3] This leptin secreted by adipose tissue is strongly related to the gluteofemoral fat than the upper body or the abdominal fat content during puberty.[4] Many studies indicate that leptin exerts a permissive action on onset of puberty by reducing the gonadal feedback suppression of LH, which was responsible for the prepubertal suppression of GnRH. Estradiol is essential for emergence of kisspeptin expression on GnRH neurons in prepubertal period. Leptin also causes a significant increase in 17,20 lyase without a sustained influence on 17α-hydroxylase increasing the production of estradiol, which has a positive effect on kisspeptin neurons. Thus, leptin represents a metabolic gate for puberty and reduction in plasma leptin level causes inhibition of the onset of pubertal changes. It is a known fact that serum leptin levels rise in both sexes at the start of puberty, which also causes a reduction in sex hormone-binding globulin. Excess body fat increases the levels of hormones such as insulin, leptin, and estrogen which accelerates the pubertal process.[5]

MATERIALS AND METHODS

The study was conducted on a total of randomly selected 250 adolescent girls of different schools in the city of Hyderabad. Institutional ethical clearance was obtained. Out of total subjects 200 underwent the study, 100 each from different government and private schools. Informed consent was taken from their families along with taking the head of the institution under confidence. A self-tested, pre-structured, self-explanatory questionnaire in English as well as in the local language was prepared and provided to each subject according to their preference. Each subject was asked to fill the questionnaire maintaining full privacy. The questionnaire comprised three sections, namely, anthropometric measurements, family background including the sociodemographic profile and menstrual history. The filled in questionnaire was collected back quickly to avoid subject bias. The birth dates were confirmed from the school records.

Inclusion Criterion

All apparently healthy adolescent girls preferably of the age group of 10–16 years were included in the study.

Exclusion Criterion

All girls who did not attain menarche till date, were undergoing evident physical or mental health problems or had one in the past, were under regular medication for chronic illnesses, had undergone recent surgeries, belonged to a broken family or who have suffered a recent demise of a family member.

Research Instrument

1. Standard portable weighing machine with calibrated scale of 0.5 kg marked from 0 kg to 130 kg.
2. Standard portable stadiometer
3. Stretch-resistant measuring tape.

Each subject's height, weight, hip, and waist circumference were taken using anthropometric measurement standards (Centers for Disease Control and Prevention 2009). Body mass index (BMI)

Table 1: Age-wise distribution of subjects based on attainment of menarche

AOM (years)	Number of subjects		Total percentage (*n*)
	Government school	Private school	
<10	0	1	0.5 (1)
10.1≥11	2	5	3.5 (7)
11.1≥12	5	23	14.0 (28)
12.1≥13	35	63	49.0 (98)
13.1≥14	57	8	32.5 (65)
14.1≥15	1	0	0.5 (1)
Total	100	100	100 (200)

AOM: Age of menarche

Table 2: Mean AOM with respect to BMI

BMI range	Government school	Private school	Total percentage (*n*)	Nutritional level	Mean AOM (years)
15≥16	5	3	4.0 (08)	Severely underweight	14.06±0.18
16.1>18.5	28	19	23.5 (47)	Underweight	13.60±0.39
18.5≥22.9	65	55	60.0 (120)	Normal	12.86±0.40
23≥24.9	2	21	11.5 (23)	Overweight	11.76±0.47
≥25.0	0	2	1 (2)	Obese	10.25±0.35
Total	100	100	100 (200)		12.94±0.74

AOM: Age of menarche, BMI: Body mass index

and waist-to-hip (W/H) ratio were calculated. The subjects were asked to fill the AOM on recall basis (nearest month and year).

Statistical Analysis

Statistical analysis was done using the SPSS software and the Student's *t*-test results revealed that the difference between AOM of government and private schools is highly significant ($P < 0.001$).

RESULTS

The mean age of subjects was 12.74 ± 2.34 years. The mean BMI of all the subjects was 20.88 ± 3.53, while the mean BMI of government school-going girls was 19.76 ± 2.42 and private school-going girls was 21.02 ± 3.16 ($P < 0.15$). The mean W/H ratio was 0.69 ± 0.14 while for government school was 0.68 ± 0.02 and private school was 0.70 ± 0.23 ($P < 0.05$) which is a significant change observed.

Table 1 clearly shows that majority of the government school girls attained menarche around 13–14 years while the private school girls attained around 12–13 years. Table 2 clearly shows that girls having a normal BMI attained menarche around 12 years while the AOM declined with obesity and increased with leanness. Table 3 shows that majority of the respondents achieved menarche around a W/H ratio of 0.7 and the AOM was inversely proportional to W/H ratio. Table 4 depicts that the difference between government and private schools was significant and W/H ratio is the most significant variable deciding the AOM.

The study clearly revealed that the AOM was negatively correlated with the BMI, W/H ratio, and the waist and hip circumference, but W/H ratio can be taken as a single best predictor of AOM. The girls from government schools attained delayed menarche as compared to the private school girls as they were thin built while private school girls had a higher obesity index.

DISCUSSION

The present study is aimed to relate the AOM with the other anthropometric and physiological factors affecting the girl child. The overall mean AOM observed in our study is 12.94 ± 0.74 years which is comparable to that of girls from other urban centers, for example, Kolkata (12.3 years) and Mumbai (12.9 years), but lower than that of the girls of Chandigarh (13.2 years) and Delhi (13.34 years). The AOM in Nellore (a nearby district of the erstwhile Andhra Pradesh state) was found to be 13.83 ± 0.87 years. This difference may be attributed to the minor changes in environment, dietary habits, and lifestyle at different places. The lowest AOM recorded in the study was 10 years while the highest was 14.5 years.

Table 3: Mean AOM with respect to W/H ratio

W/H ratio	Total number of subjects	Total percentage	Mean AOM (years)
0.68–0.72	41	20.5	13.71±0.46
0.72–0.76	112	56.0	12.92±0.56
0.76–0.80	40	20.0	12.49±0.47
0.80–0.84	7	3.5	11.21±0.81

W/H: Waist to hip, AOM: Age of menarche

Comparison of AOM for Government and Private Schools

Referring to Table 5, the mean AOM for government schools was calculated to be 13.25 ± 0.68 years while that of private schools was 12.62 ± 0.67 years which was found to be significant ($P < 0.05$). This difference may be attributed to better socioeconomic conditions leading to higher BMI, higher prevalence of obesity, and lower physical activity in private schools girls as compared to the government schools. Easy access to adult content online due to the easy and rampant availability of personal mobiles and tablets can also be a cause of early AOM in private school girls.

AOM versus BMI

BMI has been used by the WHO as a standard for recording obesity statistics since the early 1980s. From Table 2, we can observe that girls having a high BMI had an early menarche ($r = 0.91$). The underweight girls had a mean AOM of 14.06±0.18 years. whereas the girls who had a normal bmi had an AOM of 12.86±0.40 years[6] as well as by Goon *et al*. (2009). Acharya and Reddaiah concluded that for each

Table 4: Comparison of the variables

Variables	Government school	Private school	*P*	Correlation coefficient	Significance
BMI	19.75±2.42	22.01±3.16	<0.010	−0.91	Significant
W/H ratio	0.70±0.02	0.72±0.02	<0.001	−0.96	Significant
Waist circumference	54.11±1.95	56.03±1.76	<0.002	−0.89	Significant
Hip circumference	78.21±1.12	79.01±0.15	<0.006	−0.78	Non-significant

BMI: Body mass index, W/H: Waist to hip

Table 5: Mean AOM in government and private schools

Variables	Mean AOM (years)
Government school-going girls	13.25±0.68
Private school-going girls	12.62±0.67
Overall	12.94±0.74
P	<0.001

AOM: Age of menarche

unit increases in BMI, there is 24% chance of attaining early menarche.[7] Higher total calorie intake is associated with early AOM as these extra calories are converted to adipose tissue, which releases leptin and also increases the circulating estradiol, thus preponing the GnRH pulse generator. A study has shown a positive correlation between leptin and BMI ($r = 0.88$).[8] Verma (2010) also concluded that girls with high BMI had an early AOM (11.97 ± 1.68 years) as compared to girls with a normal BMI (12.67 ± 1.37 years). Not only with the total caloric intake but also there is a positive correlation between AOM and animal protein intake, after controlling for BMI as depicted by studies by Berkey *et al*. as well as Roger *et al*. Recent worldwide increased consumption of animal protein and saturated fat has increased the childhood obesity, resulting in falling ages of puberty mainly in the developing countries. Roger *et al*. (2010) showed that increased total and animal protein intake as well as increased fat intake at 3–7 years were positively associated with early AOM and the same was concluded by Berkey *et al*.[9] Thus although obesity is not the only factor contributing to early AOM but is a major cause.

AOM versus W/H Ratio and Hip Circumference

W/H ratio is found to be a better correlation of obesity than BMI. From Table 3, it can be deduced that the W/H ratio and the hip circumference are inversely proportional ($r = 0.96$ and $r = 0.78$, respectively) to the AOM. Laseek and Gaulin have expressed that fat distribution rather than the total fat, in particular, the gluteofemoral fat versus the upper body fat to be a better predictor of menarche,[10] suggesting that leptin conveys information about the fat distribution to hypothalamus rather than the total fat. It is emphasized that the W/H ratio of 0.7 was optimum to attain menarche as they have an exact level of estrogen in the body. The Medical Research Council and National Survey of Health and Development have also emphasized that increased birth weight, rapid growth in infancy, rapid prepubertal weight gain, childhood obesity, and physical inactivity are predictive of early menarche, as also approved by the Ellis Energetic theory.[11]

Limitations

Measurement of the AOM was based on recall method which can be influenced by errors of poor memory. The AOM can also be influenced by genetics, environmental factors, family disputes, stress, age of attainment of menarche of the mother, physical abuse, levels of exercise, and exposure to exogenous hormones in which this study did not incorporate.

CONCLUSION

This study found that the average AOM was lesser for private school-going girls mainly due to the higher fat content in the body, preferably the higher percentage of gluteofemoral fat as compared to the upper body fat. As childhood obesity and mental stress are on a rise worldwide, both these puberty accelerators are producing a new generation of girls who are sexually mature at a very tender age. However, this early pubertal maturity should be matched by efforts to socially develop the young girls to reduce the gap between the physical and social puberty. As puberty increases the interest in sexual activity, causing an early sexual debut, unprotected sex, sexually transmitted diseases, sexual abuse, and teenage pregnancy, it is highly recommended to strictly initiate sexual classes at all primary school levels as well a healthy discussion by the parents at an early age. It is also highly imperative to inculcate a habit of healthy eating and regular physical activity both at school and at home in the young minds, to reduce the gap between physical and social puberty. Looking at the early sexual maturation, there should be amendments in the law for the right age of consensual sex.

REFERENCES

1. Available from: http://www.who.int/maternal_child_adolescent/topics/adolescence/dev/en. [Last accessed on 2019 Jul 22].
2. Study of Age of Menarche, Secular Trends and Factors Affecting it. Available from: http://www.ispub.com/ijba/3/2/7469. [Last accessed on 2019 Jul 22].
3. Karapanou O, Papadimitriou A. Determinants of menarche. Reprod Boil Endocrinol 2018;8:115.
4. Lassek WD, Gaulin SJ. Brief communication: Menarche

is related to fat distribution. Am J Phys Anthropol 2007;133:1147-51.
5. Steingraber S, Solorzano CM. The falling age of puberty in US girls: Obesity and pubertal transitions in girls and boys. Reproduction 2010;140:399-410.
6. Ayatollahi SM. Age at menarche in Iran. Ann Hum Biol 2002;29:355-62.
7. Acharya A, Reddaiah VP. Nutritional status and menarche in adolescent girls in an urban resettlement, New Delhi. Indian J Community Med 2006;31:302-3.
8. Hassink SG Sheslow DV, de Lancey E, Opentanova I, Considine RV, Caro JF, *et al.* Serum leptin in children with obesity: Relationship to gender and development. Paediatrics 1996;98:201-3.
9. Berkey CS, Gardner JD, Frazier AL, Colditz GA. Relation of childhood diet and body size to menarche and adolescent growth in girls. Am J Epidemiol 2000;152:446-52.
10. Laseek WD, Gaulin SJ. Menarche is related to fat distribution. Am J Phys Anthropol 2007;133:1147-51.
11. Ellis BJ. Timing of pubertal maturation in girls: An integrated life history approach. Psychol Bull 2004;130:920-58.

How to cite this article: Sinha S, Ali MA. A comparative study of age of menarche in private and government school-going girls. Natl J Physiol Pharm Pharmacol 2020;10(01):49-53.

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